TREPONEMAL INFECTION IN ITS BIOSOCIAL CONTEXT AT LATE WOODLAND GARBACON CREEK, NORTH CAROLINA.

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Abstract

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Treponemal infection has been well documented in prehistoric settlements of the American Southeast; however, studies concentrating on the evidence for treponematosis may have not focused on signs of other conditions or overlooked other possible causes for the observed periosteal activity often taken as indicative of treponemal infection. The Late Woodland Garbacon Creek ossuary remains from the North Carolina coastal plain represent at least 29 individuals, and many of the elements show pathological activity. Of the adult tibiae alone, 75% present with periosteal lesions. The presence of at least one stellate lesion confirms the existence of treponemal infection in this population, but there are undoubtedly other diseases and conditions operating simultaneously. Macroscopic and radiographic analyses have revealed signs of possible scurvy, rickets, and anemia. This thesis discusses the interactions between these conditions which may be complicating the diagnosis, and the biosocial environment which contributed to the development of these conditions.

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The very fact that the children in the archaeological record died young informs us about the cultural and biological circumstances of a particular group. The ability of the adult population to keep the next generation, as vulnerable members of a community, alive and in good health is testament to their adaptability to their environment. (Lewis 2007, 186-7)

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Chapter 1:

Comorbidity and Garbacon Creek

Comorbidity and the Treponematoses

This thesis examines the context of treponemal infection in a comingled human skeletal sample of approximately 29 individuals from the Late Woodland (A.D. 800-1600) site of Garbacon Creek (31Cr86) in the North Carolina coastal plain. Infectious diseases never act on a population in a vacuum. The full effect of a specific disease organism on a population cannot be understood until the researcher considers the other pathogens invariably existing alongside it at both a population and individual level. A fully integrated biosocial system including the compounding factors of the cultural and physical environments completes the profile of a particular disease experience. Activity patterns, living conditions, means of subsistence, and cultural attitudes to disease place certain populations, or certain segments of a population, at higher risk of infection, and also shape the pathogen-host interaction following initial infection.

Nutrient deficiencies and infection have an unfortunate synergy: infections are likely to be more severe in malnourished individuals, and infections can turn borderline deficiencies into clinical cases (Chandra and Newberne 1977). The physical symptoms of infection vary with the immune response of the host, which is strongly determined by the host's nutritional status (Scrimshaw 1968). It is only when a disease process is studied in its full context that we know the extent to which internal and external conditions of the ill

individual impact the physical expression of a disease, and we can begin to understand the individual and population level cost of a pathological condition.

The specific pathogens of interest to this study are the spirochete bacteria of the genus *Treponema*. The *Treponema* have adapted to a wide variety of environments worldwide. The closely related quartet of diseases caused by these bacteria – pinta, yaws, nonvenereal syphilis (or bejel) and venereal syphilis – manifest with similar symptoms, and each is found in a distinct ecological niche (see ch. 2). Although the correlation between climate and specific treponemal disease has been well documented (Hackett 1967; May 1958), the effects of coincident pathological conditions on the physical symptoms of treponemal infection has yet to be fully explored (but see Buckley 2001; Smith 2008).

This thesis considers the effects of specific types of malnutrition on the acquisition and physical expression of treponemal infection. Not only does malnutrition compromise an individual's immune status, but it is also important to remember that a diet which is nutritionally deficient is usually lacking in more than one nutrient. Iron and vitamins C and D especially interact in ways which make it likely that a deficiency in one is accompanied by deficiencies in either or both of the others. Many other micronutrients are necessary for proper immune response, but the effects of deficiencies in these three are observable in the skeleton.

Ortner and coworkers (2001) remind us that the diagnosis of a metabolic condition or systemic disease generally requires consideration of the pattern of lesion distribution across the entire skeleton of an individual. The expression of any of the above metabolic conditions (anemia, scurvy, and rickets) in a single bone can be indistinguishable from a general reaction to any number of blood-borne pathogens. When healing skeletal lesions

from a prior infection or deficiency are visible next to those of a condition which was active at time of death, it is impossible to know whether the timing of the two conditions overlapped, and thus prove exact contemporaneous comorbidity in the older individuals of a sample. Rather, we are left with a general picture of the various conditions circulating in a population, which potentially existed in a given individual at the same time.

Garbacon Creek (31Cr86):

Geographical and Cultural Context

The Garbacon Creek site is located in the Northern Tidewater region of North Carolina's coastal plain, in Carteret County on a promontory of the Neuse River where it joins the Pamlico Sound (Fig. 1). The site was exposed by the passage of Hurricane Ginger in the autumn of 1971 and excavated on November 17-19, 1971 by Keith Egloff of the Research Laboratories of Archaeology at the University of North Carolina at Chapel Hill. The excavated portion of the site comprises a mass burial, or ossuary, which was dated based on the presence of a small globular pot from the Late Woodland White Oak/Oak Island period (Kakaliouras 1997). Although a confident assignment cannot be made, the site's location suggests that its inhabitants were most likely affiliated with either the Siouan or Algonkian cultural groups (Egloff 1971). Several post holes and refuse pits were noticed eroding out of the river bank during excavation, suggesting the presence of an associated settlement; however, the excavation did not extend far beyond the physical bounds of the

ossuary pit. Initially uncovered by erosion, the entire site has now completely worn away (Kakaliouras 1997).

Subsistence

The brackish estuarine environment of Pamlico Sound is typical of the North Carolina outer coastal plain (Hutchinson 2002). Perlman (1980) effectively summarized the local animal resources available to people living near estuaries:

> *The dependable location of some resources such as shellfish, the dependable cycling of some resources such as anadromous fish, the continual cycling of nonmigratory species within the estuary, and the movement of terrestrial prey animals into the coastal zone make estuaries highly productive and low risk environments for exploitation.*

John Lawson, in his 1709 survey of the Carolina interior, remarked that there were "no Savages living so well for Plenty, as those near the Sea." The Late Woodland period saw major shifts in subsistence patterns across the Eastern Woodlands as horticulture became a part of the subsistence strategy for many groups - but the archaeological evidence for horticulture in the outer coastal plain is erratic and scarce (Schaefer 2011).

Stable isotope analyses of skeletal remains combine with archaeobotanical evidence to suggest that maize, the staple crop of many North American agriculturalists, was adopted by peoples near the North Carolina coast later and to a lesser degree than by their inland neighbors (Hutchinson 2002). Stable isotope analysis has not been conducted on the Garbacon Creek sample due to the destructive nature of the process. However, Hutchinson and Norr (Hutchinson 2002) were able to conduct stable isotope analyses on remains from 11 other Late Woodland sites in the North Carolina coastal plain. Since the local physical

environment of Garbacon Creek is similar to that of these other neighboring sites, isotopic analysis of the Garbacon Creek remains would likely demonstrate, consistent with the results of Hutchinson's samples, that C4 plants comprised a minimal part of the diet.

The archaeobotanical evidence supports this conclusion, and indicates that prehistoric groups in the area relied mostly on foraging, subsisting on marine and terrestrial animals which included fish, shellfish, and deer, as well as plant foods including hickory nuts, acorns, walnuts, and a range of seeds (Scarry and Scarry 1997; Schaefer 2011). Although his account generalizes, describing the characteristics of all Indians in North and South Carolina together, Lawson adds to this list:

> *Venison, and Fawns in the Bags, cut out of the Doe's Belly; Fish of all sorts, the Lamprey-Eel excepted, and the Sturgeon our Salt-Water Indians will not touch; Bear and Bever; Panther; Pole-cat; Wild-Cat; Possum; Raccoon; Hares, and Squirrels, roasted with their Guts in;… All wild Fruits that are palatable, some of which they dry and keep against Winter, as all sort of Fruits, and Peaches, which they dry, and make Quiddonies, and Cakes, that are very pleasant, and a little tartish; young Wasps, when they are white in the Combs, before they can fly, this is esteemed a Dainty; All sorts of Tortois and Terebins; Shell-Fish, and Stingray, or Scate, dry'd; Gourds; Melons; Cucumbers; Squashes; Pulse of all sorts;… Fowl of all sorts, that are eatable; Ground-Nuts, or wild Potato's; Acorns and Acorn Oil…*

Within this cultural and environmental context, this thesis aims to explore the nature of the pathological evidence in these skeletal remains: what each of the conditions observed means for the expression of the others, and what cultural practices may have predisposed the Late Woodland inhabitants of Garbacon Creek to each of these conditions.

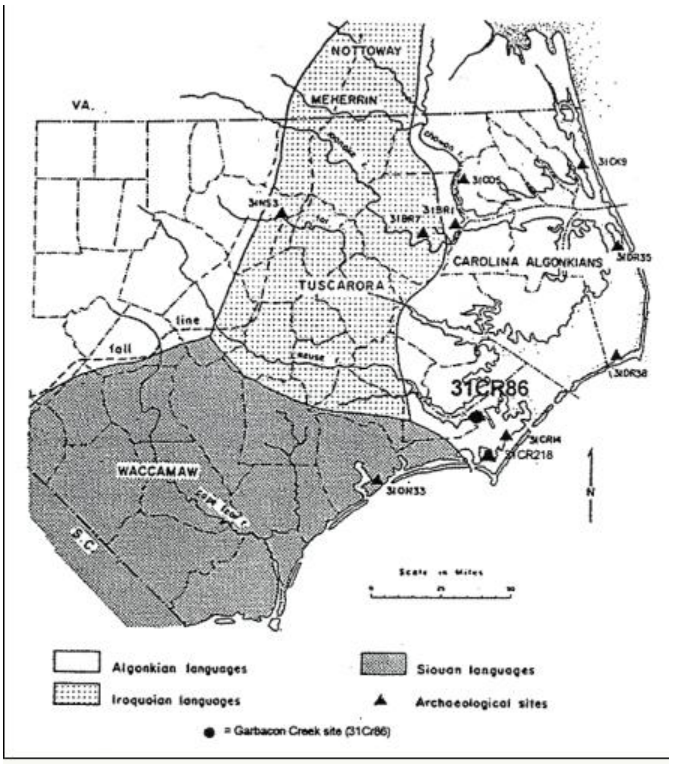


Fig. 1, Garbacon Creek (31Cr86) and the North Carolina Coastal Plain (from Phelps 1983:37)

Treponematosis at Garbacon Creek

There is a growing body of evidence supporting the existence of treponemal infection on the prehistoric North Carolina coast (Bogdan 1989; Bogdan and Weaver 1992; Monahan 1995; Truesdell 1995; Hutchinson and Weaver 1998; Hutchinson 2002; Reichs 2005). The signs of treponemal infection in the Garbacon Creek sample were previously explored by Ann Kakaliouras (1997). I re-examined the remains in order to better determine the age distribution and severity of the lesions, as well as the other pathological conditions potentially indicated by the skeletal evidence. A more accurate approximation of these

variables will allow us a more complete understanding of the treponemal experience in the Garbacon Creek population.

The high proportion of subadult remains retrieved from this site (41% of the sample) allows for a more nuanced study of the stressors affecting the Garbacon population. Children's bones respond more rapidly to systemic imbalances because they remodel much more quickly than the bones of adults. In addition to signs of systemic infection, the bones of the sample were examined for evidence of nutritional deficiencies, most notably in iron and vitamins C and D, each of which plays a crucial role in immune function. A broader investigation of the complex interactions between nutrition and infection would have been desirable, but this inquiry was necessarily constrained by the skeletal nature of the data.

The main cultural factors which determine the risk of infectious disease are subsistence base, because of its influence on population density and nutrition, and specialized activities which place different segments of a population at higher or lower risk of acquiring infection (although these activities may be connected with subsistence activities). I could not assess the impact of gendered behaviors in this sample because it was not possible to correlate individual elements with pelves and crania from the comingled burial. Despite this shortcoming, I will discuss age-related behaviors that may have contributed to the health differences observed between adults and subadults. Garbacon Creek is relatively unusual among sites in this area in that it contains a high proportion of subadults (41% of the individuals accounted for), and the percentage of the subadult remains showing signs of infection is also high (23% of the subadult long bones). This frailest portion of the population can present us with a magnified view of the disease environment in which these people likely lived.

Despite the archaeological and historic evidence of the adequate availability of edible resources, there are signs of possible nutritional deficiencies at Garbacon Creek, especially among the subadult remains. Due to the commingled state of the burial, lesion frequency by skeletal element is determined in lieu of intra-individual lesion distribution. In this case study, we are constructing an inexact model of the disease experience in the Garbacon Creek population based on the physical evidence available. While comorbidity is difficult to prove in a comingled sample, it is likely that if, for instance, high numbers of the postcranial elements present treponemal lesions and high numbers of the crania present signs of anemia, then these conditions co-existed in some individuals.

Materials and Methods:

The human remains and associated artifacts from Garbacon Creek are curated by the Research Laboratories of Archaeology at the University of North Carolina at Chapel Hill. Reconstruction of all elements was attempted in order to minimize the risk of inflating the minimum number of individuals present (MNI). Elements which contributed to the MNI count were assigned a number and recorded with information on specific element, portion of element present, side, provenience, age (for subadults), and pathological symptoms (see Appendices I, II, and III). The extant skeletal elements represent the remains of at least 29 individuals: 17 adults and 12 subadults. The minimum number of adults was determined from the crania. MNI for the subadult remains was only determined after the age of each subadult element had been estimated, creating more narrowly defined categories of cohorts. The most numerous skeletal element for each set of cohorts then determined the final MNI: one pre-term or low birth weight infant; ilia for <6 months of age; femora for 6 months-2

years of age; no more than one example of each element in the 2-5 year category; and ulnae for 5-10 years of age. The sample demography is summarized in Table 1.

Table 1: Garbacon Creek Demography

Age	Sex	Crania	Innominates
Adult	Female	5	4
Adult	Male	5	7
Adult	Indeterminate	7	7
Total Adults:		**17**	-
			Determining Element
< pre-term infant	Indeterminate	1	ilia
< 6 months	Indeterminate	2	ilia
6 mo. - 2 yrs	Indeterminate	3	femora
2-5 yrs	Indeterminate	1	humeri
5-10 yrs	Indeterminate	3	ulnae
10-20 yrs	Indeterminate	2	epiphyses
Total Subadults:		**12**	-

Age was estimated by visual comparisons of size against specimens of known age, and from maximum length of long bones (Johnston 1962). Nutritional stress can inhibit normal growth, but we have no way of correcting for this bias. Dental ages were estimated based on stage of development and eruption using the standard method developed by Ubelaker (1989). Adults were not sorted into narrower age ranges because the crania were often not complete enough to estimate age from cranial suture closure, and in the innominates post-depositional diagenesis made the state of auricular surfaces and pubic symphyses unreliable indicators. The sex of each adult cranium was estimated by visual observation of robusticity of the sexually dimorphic morphological characteristics (Buikstra and Ubelaker 1994). Of the 10 adult crania for which sex could be estimated, three were female, two possibly female, three were male, and two possibly male. The poor preservation

of the remaining 7 (41%) precluded sex estimates. It would have been preferable to estimate sex using a sample-specific range of robusticity, but the small size of the sample made this an unreliable method. Sex estimation of the innominates yielded a count of six males, one possible male, one possible female, and three females (Phenice 1969). The remaining seven innominates with intact auricular surfaces and sciatic notches were too fragmentary for sex estimation.

All bones were examined macroscopically for evidence of infection or specific metabolic stress. Bone changes which were judged to be pathological were recorded, noting position and extent of reactive bone. Bony changes were described as porosity (usually on the cranium), striations (Fig. 1), bone deposition (Figs. 2, 3), destruction (Fig. 9), or sclerotic bone (Fig. 4) (Ragsdale 1993). Lesions which exhibited characteristic treponemal morphology were described using terms defined by Hackett (1967). Select pathological elements were radiographed to determine the extent of new bone apposition and the involvement of the medullary cavity. A list of pathological elements in the adults is given in Table 2, and the pathological subadult elements are listed in Table 3.

Fig. 2 – Striations (tibia)

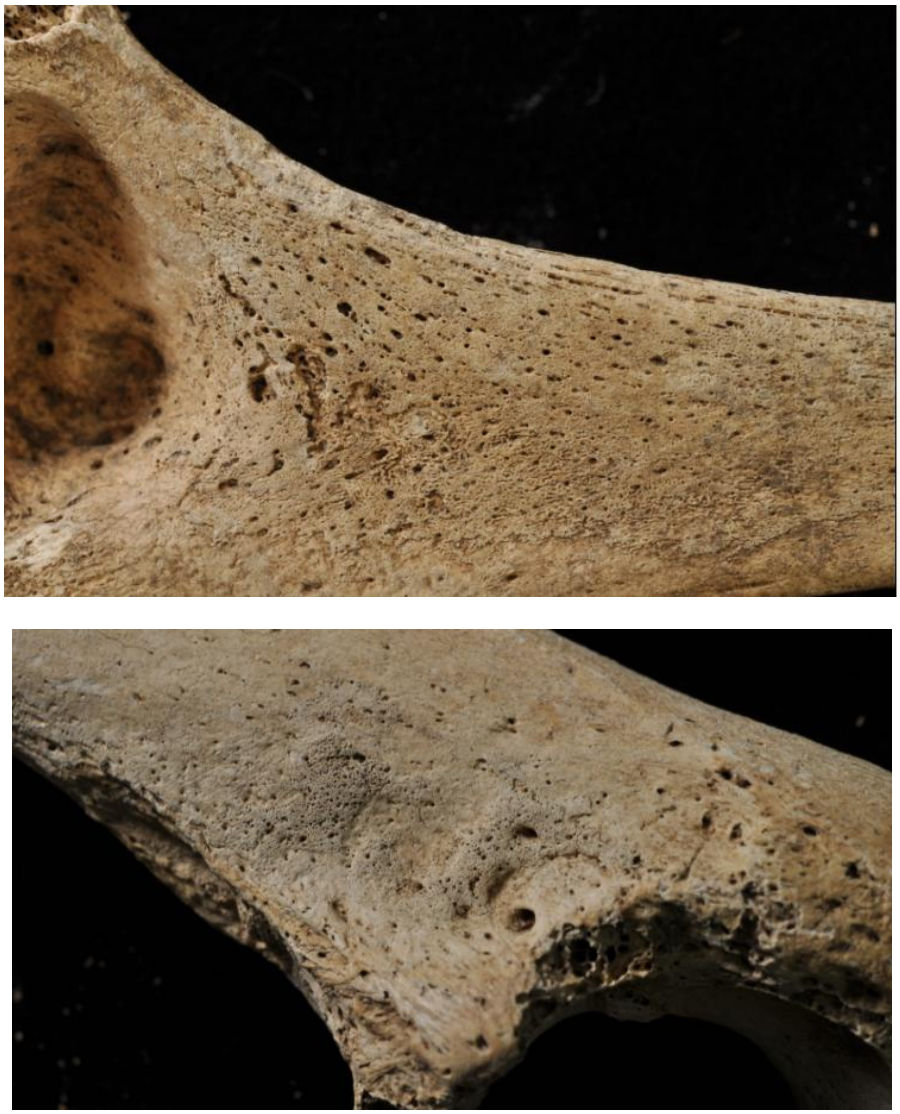


Fig. 3 – Examples of bone deposition (Distal humerus above and proximal ulna below)

Fig. 4 – Sclerotic bone (fibula)

Chapter 2:

Clinical and Archaeological Evidence of Treponemal Infection

Approaches to the Treponematoses:

The four diseases caused by the spirochete *Treponema pallidum* are collectively known as the treponematoses, but individually known as pinta, yaws, bejel (nonvenereal syphilis), and venereal syphilis. Although all four begin as skin infections, pinta does not cause skeletal changes (and so shall not be discussed further). The latter three, in their advanced stages, cause skeletal lesions that vary in severity but, nonetheless, manifest in frustratingly similar ways from the viewpoint of the palaeopathologist. The similarities and broad geographical spread of these diseases have led to a debate on the originating point of the morally charged treponeme of venereal syphilis. The arguments, which started in the sixteenth century, have resulted in three competing hypotheses which attempt to explain the origin and spread of the treponematoses.

The Columbian hypothesis postulates that venereal syphilis was present in the New World when the first European explorers arrived. They carried it back to Europe where it swiftly became prominent in the disease landscape of the 16th century. Proponents cite the remarkable virulence with which the disease struck what must have been an immunologically naïve, or "virgin," population (Dennie 1962). The Columbian hypothesis is supported by the fact that there are no documented medical case descriptions or skeletal samples from

before this time that can unambiguously be designated as cases of venereal syphilis (Harper et al. 2011).

The Pre-Columbian hypothesis contends that venereal syphilis was present in the Old World before contact with the Americas. Supporters of this theory point to disease descriptions as ancient as Classical Greece and Rome which seem to match the symptoms of venereal syphilis. Diagnostic accuracy was limited before the 16th century, and mentions of "venereal leprosy" in the Middle Ages suggest a conflation of the two disfiguring illnesses, since modern leprosy is not spread through sexual contact (although the causative mycobacterium may have evolved since the Middle Ages). Steinbock (1976) further points out that the fear of contagion which led to the foundation of leprosy asylums would make more sense if syphilis were being misidentified under this label, since leprosy has an incubation period of three to ten years while the signs of syphilis are evident after a few days. One might suggest a possible counter-argument to the idea that a disease with such a long incubation period and such low infectivity could inspire fear of contagion and measures of quarantine: the equally long incubation period and low infectivity of tuberculosis did not prevent the social seclusion of its victims in sanatoria in the late 19th and early 20th centuries.

Finally, the Unitarian hypothesis posits that the four conditions caused by *T. pallidum* are not separate diseases but syndromes of a single condition which manifests differently based on various environmental factors. The venereal expression is better suited to colder Europe where cultural and climatic considerations dictate full attire, thus largely limiting transfer of the spirochete during casual skin-to-skin contact. In warmer climates where there is typically less clothing worn, *T. pallidum* passes by direct skin-to-skin contact between children, and remains mostly a disease of childhood (Steinbock 1976). According to the

Unitarian hypothesis, nonvenereal syphilis likely came to the Americas with the first settlers to cross the Bering Strait, and then reverted back to yaws when the settlers migrated far enough south to allow the human living conditions which favor it (Bogdan and Weaver 1992).

Until recently, the different strains of the Treponema spirochete responsible for these four diseases have been morphologically indistinguishable. The molecular difference between venereal syphilis and the nonvenereal treponemes is a single base which creates a restriction in the tpp15 region of *T. pallidum pallidum* (the binding site of thiamine pyrophosphate (tpp), or vitamin B1). However, the treponemes *T. palladium pertenue*, which causes yaws, and *T. pallidum endemicum*, which causes endemic syphilis, are genetically identical (Centurion-Lara et al 1998). The clinical manifestations of yaws, endemic syphilis, and venereal syphilis are quite variable and overlap considerably. It is not clear that a confident distinction can be made between yaws and endemic syphilis in a skeletal sample; however, the bone lesions of a nonvenereal treponemal infection will affect a different age demographic – children old enough to interact with their cohort and adults with tertiary manifestations - than will the lesions of a venereal treponemal infection – infants and adults, but very few children – allowing for a distinction to be made there.

Yaws

Yaws is primarily a childhood disease found in humid tropical environments, characterized by early contagious skin lesions and eventually late destructive, non-contagious lesions (Aufderheide and Rodriguez-Martin 1998). Because of its long infectious stage it can remain endemic in small, isolated tropical populations. The risk of infection is greater when

children are old enough to be mobile, but skin-to-skin transmission can still be achieved in infancy by direct contact with infected family members, or may be spread by flies which have come into contact with the lesions. Following infection there is a three week incubation period, after which a skin lesion, or 'mother yaw,' appears, closely followed by others (Aufderheide and Rodriguez-Martin 1998). Healing is spontaneous, but the symptoms may recur periodically throughout life. Patients in the secondary stage of yaws suffer from fever, headaches, and widespread painful skin lesions. Only 3-5% of those who contract the disease will develop bony changes, which primarily affect the hands, feet, wrists, and tibiae. The tertiary stage of the disease, which appears between five and ten years after initial infection, afflicts its victims with gummatous lesions of the skin, soft tissue, and bone.

Nonvenereal Syphilis (Bejel)

Nonvenereal syphilis resembles yaws very closely in its stages of development and skeletal lesion distribution, but whereas yaws is found in humid tropical environments, endemic syphilis is found in regions of arid heat. Unlike yaws, the initial cutaneous symptom of nonvenereal syphilis is a contagious rash without a visible primary lesion, and its incubation period varies between two weeks and three months (Aufderheide and Rodriguez-Martin 1998). In addition to the skin and bone lesions of nonvenereal syphilis which are common to yaws, individuals with nonvenereal syphilis may develop cardiovascular lesions (Hudson 1958). Patients have also reported ostalgia, or bone pain, even before the development of radiographically visible lesions (Hurtzler, 1930, cited in Cook 1976). The clinical symptoms of nonvenereal syphilis fall somewhere in between those of yaws and those of venereal syphilis.

Venereal Syphilis

Venereal syphilis is not limited to any climatic range, but it is spread primarily through sexual contact and has a higher prevalence in urban areas where individuals are more likely to have multiple partners. The course of the disease is divided into three stages, with a potential for long periods of latency. The first sign of syphilis is the primary skin lesion, or chancre, which appears on the genitals between ten days and ten weeks after initial infection. The lesions usually disappear within a few weeks, causing many syphilitic individuals to mistakenly believe themselves cured. In this phase the spirochetes multiply and enter the lymph nodes. In the secondary stage the organism spreads hematogenously through the body, causing symptoms of the bones and soft tissue. Tertiary syphilis involves the skin, cartilage, and liver, among other organs. If the nervous system is involved the afflicted individual may suffer from partial paralysis, dementia, meningitis, nerve degeneration, or a combination of these symptoms (Musher and Knox 1983).

Congenital Syphilis

The best way to document the presence of venereal syphilis in an archaeological population is by identifying congenital syphilis, which is passed across the placenta from mother to child. Steinbock (1976) says that the infection is transmitted in 84% of syphilitic pregnancies, but the likelihood of transmission is higher if the mother is newly syphilitic.

The first sign of congenital syphilis is usually sniffles – or rhinitis – appearing between two and six weeks after birth and resulting from the involvement of the mucus

membranes. Chronic sniffles can lead to the development of a "saddle nose", or collapsed nasal bridge. Chawla et al. (1988), in a study of Zimbabwean neonates, found that low birth weight, hepatosplenomegaly, anemia, jaundice, and peeling of the skin on palms and feet were highly suggestive of congenital syphilis. Radiographs of stillborn syphilitic infants have shown periostitis and osteochondritis (Bogdan 1989). According to Murray and coworkers (1990), 75% of infected infants show syphilitic symptoms at birth, but in fewer than 10% of cases, it may first appear during early adolescence, when it will resemble far more the expression of bejel (persistent periosteal reaction and subsequent cortical thickening of long bones) (Ortner and Putschar 1985).

Osseous Lesion Morphology

In the secondary stage of treponemal infection, when bone involvement first appears, it generally occurs as widespread symmetric periosteal bone apposition. Lesions of the tertiary stage are both destructive and appositional, and are usually more confined in scope. This difference in distribution appears to be linked to the age differential in the disease stages, with earlier stages manifesting in younger individuals, whose higher tissue turnover rates favor a more systemic reaction (Cook, 1976).

In yaws, the appearance of secondary stage lesions has been documented between two weeks and four months after initial infection (Hunter et al., 1956). The bones most often affected by yaws, in order of decreasing frequency, are the tibia, fibula, femur, ulna, radius, spine, clavicle, hand, foot, skull, and pelvis (Ortner and Putschar, 1985). Localized periosteal apposition on the tibial mid-shaft is characteristic, and creates an anterior-posterior bowing of the bone, known as 'sabre tibia' (see Fig. 8). This symptom is

uncommon before age five. Diffuse periosteal apposition of bone causes thickening in the long bone cortices, making the affected elements appear swollen. In time, the medullary cavity may be narrowed by the involvement of endosteal bone. The cranium is very rarely involved, but destruction of the naso-palatal area may be pronounced. Destruction of the nasopharynx is not pathognomonic, however, as it can also be caused by leishmaniasis and leprosy, but the distinct postcranial symptoms of these conditions should allow for a clear differential diagnosis (Steinbock 1976).

The secondary stage lesions of endemic syphilis are variable and can sometimes resemble those of late congenital syphilis, with periosteal apposition that creates an irregularly thickened cortex, but almost no involvement of the medullary cavity. When localized rarefaction occurs as part of this reaction, the lesions have a recognizable "moth-eaten" appearance (Rost 1942; Ragsdale 1993). Musher and Knox (1983) determined that periosteal inflammation was clinically apparent in 25% of cases in the pretreatment era.

Gummatous osteomyelitis characterizes the long bone lesions in the tertiary phase of treponemal infection. A gumma, or granulomatous swelling, is caused by an insufficient blood supply from blood vessels damaged by the byproducts of the treponeme (Steinbock 1976). The bone surrounding the gumma presents as a hypervascularized periosteal ring built up around a "scooped-out" area in the cortex (Bogdan and Weaver 1992). Gummata which extend into the medullary cavity are surrounded by reactive sclerotic bone (Ortner and Putschar 1985). Spicules of subperiosteal new bone can lie perpendicular to the shaft, especially in the fibula (see Fig. 11). Gummatous periostitis rarely forms sequestra, but when present the sequestra are found near the metaphyses and epiphyses. Radiologically these symptoms are visible as a thickened cortex that is more irregular on the external surface,

with radiolucent foci of gummatous destruction (Steinbock 1976). Yaws can cause crater-like depressions on the frontal bone, but these are much less destructive than the cranial lesions of venereal syphilis.

Hackett and others have noted that, with the exception of the dental stigmata and osteochondritis seen in congenital syphilis, the lesions of yaws do not differ qualitatively from those of syphilis, and several long-term studies (Bruusgaard 1929; Rosahn 1946) have determined that roughly 65% of syphilitic patients die during a period of remission, leaving no anatomical evidence of the disease (Steinbock 1976), which further complicates attempts to locate it in the archaeological record. Diagnostic bone lesions of tertiary syphilis appear between two and ten years after primary infection, although few individuals will develop tertiary symptoms. Of those individuals who do present with tertiary symptoms, even fewer will develop bone lesions (Bogdan and Weaver 1992; Cook 1976). As with the other treponematoses, the tibia is the most common site of syphilitic lesions. Cranial involvement is much more common in venereal syphilis than in reactions to the non-venereal treponemes (Ortner and Putschar 1985; Steinbock 1976). Both venereal and congenital syphilis can affect the cervical spine, although the bone changes are slight and do not tend to involve more than two vertebrae. Inflammation of the periosteum covering the vertebral body may cause the vertebral body to become rounder and larger than normal, with occasional osteophytic spurs between vertebrae (Steinbock 1976).

C. J. Hackett (1976) has described and seriated the cranial reactions to the treponematoses, which he named the *caries sicca* sequence. Discrete cranial changes begin with clustered 1 mm pits on the ectocranial surface of the frontal and parietal bones, which then become focal superficial cavitations, which may perforate the endocranial surface.

During healing, a rounded margin of bone surrounds the lesions. This stage is described as 'circumvallate cavitation.' Continued healing fills in the cavity with new bone and leaves only a set of radial scars, the pathognomonic stellate (star-shaped) lesions.

In infants, the spirochetes tend to gather in places of rapid growth and development, so bone changes due to their presence are generally found at the growth plates, or metaphyses, of long bones, especially the distal femur and proximal tibia. The spirochetes cause a trophic disturbance by blocking nutrient pathways to the bone, resulting in necrosis (Buckley 2000). Congenital syphilis can cause pathognomonic perinatal growth arrests in the tooth buds of the developing permanent dentition. These are referred to colloquially as Hutchinson's incisors, mulberry molars and Moon's molars. Bauer (1944) reviews the possibility that the dental stigmata often appear when congenital syphilis is complicated by a case of rickets or other interruption of calcium and phosphorus metabolism. Nonetheless, Putkonen (1962) observed a high rate of dental stigmata in a series of syphilitic patients to whom he had given vitamin D supplements specifically to prevent rachitic complications.

The inflammatory changes of osteochondritis in congenital syphilis resolve after the first six months of life and diffuse periosteal new bone may be deposited as part of the remodeling of the metaphyses. This new bone is generally irregular and can cover entire diaphyses of multiple bones (Steinbock 1976). Osteochondritis is visible on radiographs as a wide band of increased calcification in the metaphysis (although this may not be preserved in archaeological specimens). Deposition of granulomatous tissue creates irregular lytic lesions at the metaphyses, most commonly found at the distal femur and proximal tibia, the fastest growing metaphyseal plates, and a lytic lesion such as this at the knee is known as Wimberger's sign (Buckley 2000). Wimberger's sign is considered diagnostic, although

osteochondritis could also be attributed to tuberculosis or pyogenic osteomyelitis (Steinbock 1976; Bogdan and Weaver 1992; Jaffe 1972).

Cranial involvement in congenital syphilis presents as either necrotizing osteitis or hypertrophic periostitis. Necrotizing osteitis may involve the endocranial and ectocranial surface with sequestra formation. Hypertrophic periostitis is hardly ever found in syphilitic individuals without other complicating conditions such as rickets. Parrot (1879) described the protruding bosses on the frontal and parietal bones resulting from subperiosteal bone apposition, which he ascribed solely to congenital syphilis, but which more likely result from rickets or anemia (Steinbock 1976). A number of authors (e.g. Taylor 1876, Jeans and Cooke 1930) have favored rickets as the aetiology of these lesions.

In the reparative phase of osteochondritis, diffuse active bone can envelop entire diaphyses of multiple long bones (Buckley 2000). This woven bone gradually organizes itself into lamellar bone wrapped around the original bone, creating a double cortex. This process is known as "periosteal cloaking," and McLean (1931) observed it in eight of 102 cases of congenital syphilis, although Hill believes the phenomenon may be connected instead to anemia (see p.38). In each case, the cloaking was observed to resolve itself unassisted in living individuals. Diaphyseal osteomyelitis is seen in about 50% of congenital syphilis cases, which may result in cortical thickening as new periosteal bone forms and joins to the cortex (Steinbock 1976).

The tibiae of individuals affected by congenital syphilis may exhibit antero-posterior bowing, a condition sometimes referred to as a "sabre shin of Fournier," as a result of the increased growth stimulated by epiphyseal irritation of the tibia while the unaffected fibulae hold the proximal and distal ends of the adjoining tibia in place (Jaffe 1972; Lewis 2007).

The bowing of rickets, caused by weight-bearing strain on unmineralized, structurally weak bones, generally warps the tibia in a lateral direction rather than an antero-posterior one (Steinbock 1976).

Osteological Evidence for the Treponematoses at Garbacon Creek

Crania: None of the crania from Garbacon Creek had facial bones that were intact enough to allow for observation of naso-palatal destruction, however two of the adult crania showed small areas of pitting which may be the initial stage of treponemal cranial involvement: one on the parietal of cranium #2, and two on the frontal of cranium #14 (Fig. 5). Many of the crania were present in very fragmentary states, but on the calvaria which were present, no other active *caries sicca* was observed.

Two frontals and parietals in the subadult sample were complete enough to examine for the bossing of Parrot's swellings; yet none were observed. Some of the subadult parietals showed signs of increased endocranial vascularity (Fig. 6), and one frontal had an ectocranial lesion with vascular patterning which perforated the cranial table (Figs. 7, 8). None of the dental stigmata pathognomonic of congenital syphilis were found.

Fig. 5, confluent clustered pits (Cranium #14), perhaps the beginning of a caries sicca sequence?

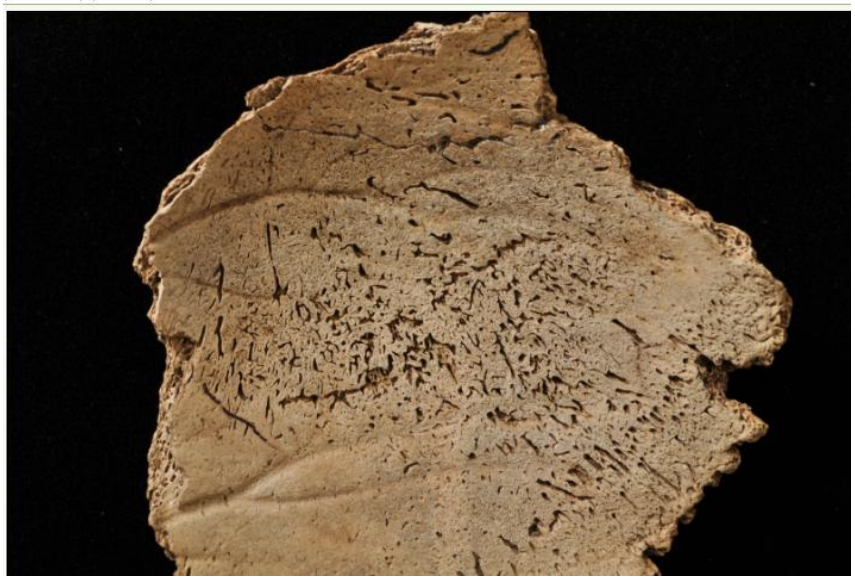


Fig. 6, endocranial vascularity

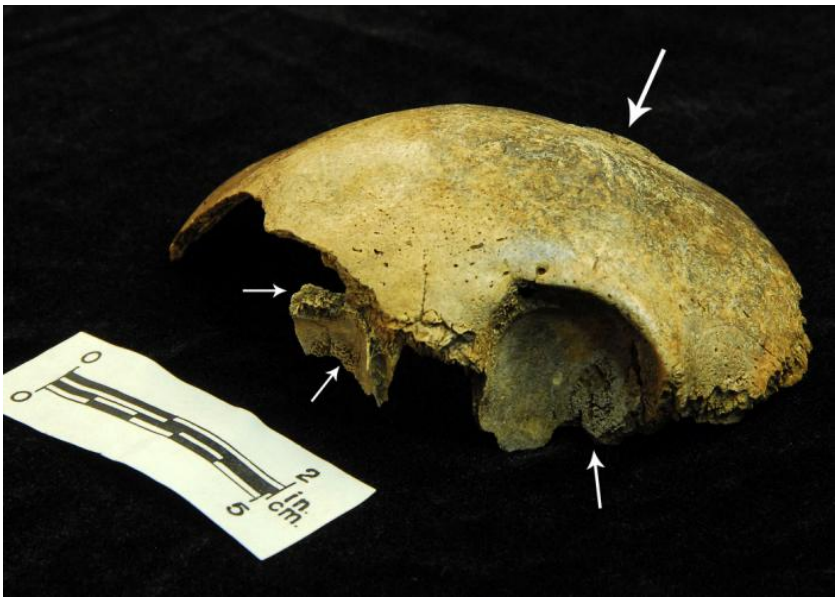


Fig. 7, ectocranial lesion, cranium 7a

Fig. 8, ectocranial lesion, cranium 7a. Note vascular patterning.

Postcrania: In the adult sample, two tibiae and one fibula have lytic foci with surrounding bone destruction and apposition and a combination of active and sclerotic bone which may represent bony involvement with gummata (Figs. 9, 10, 11, i). Eighteen of the twenty-four adult tibiae had areas of reactive bone (mostly striations – see Fig. 2) and four showed signs of anterior-posterior bowing (Figs. 12, iii). In both adults and children, all bowed tibiae also showed active bone deposition. When the active bone was not present over the entire diaphysis of a sabre tibia, it was found on the medial portion. The frequencies of long bone periosteal apposition align well with a diagnosis of non-venereal treponematosis (Cook 1976). Table 2 summarizes the data on postcranial lesion frequency and location for the adult remains. It is worth noting that the periosteal reactions observed on the forearms were often found at the elbow. All four humeri with periosteal reactive bone had it near the distal epiphysis, although it could be found either on the anterior or posterior portion of the bone. On both ulnae on which periosteal apposition was observed, it was found on the medial proximal section of the bone, by the coronoid process.

Fig. 9 - Tibiae (CR86.29 above; CR86.45 below) with lytic foci, sclerotic and active remodeling

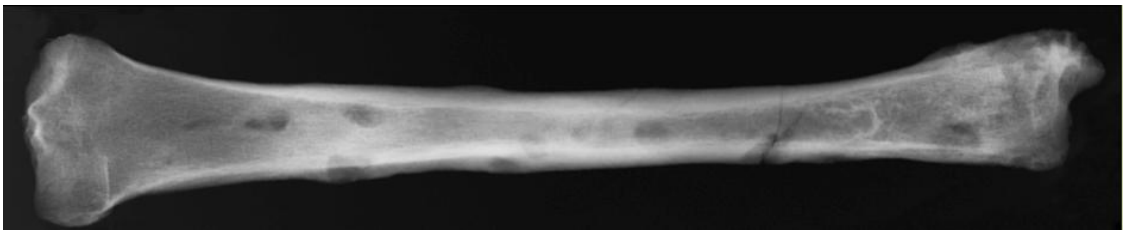


Fig. i. - x-ray of CR86.45– Note endosteal involvement, extensive periosteal remodeling

Fig. 10 - Fibula (CR86.38) with lytic foci, sclerotic and active remodeling (lateral view above, medial view below)

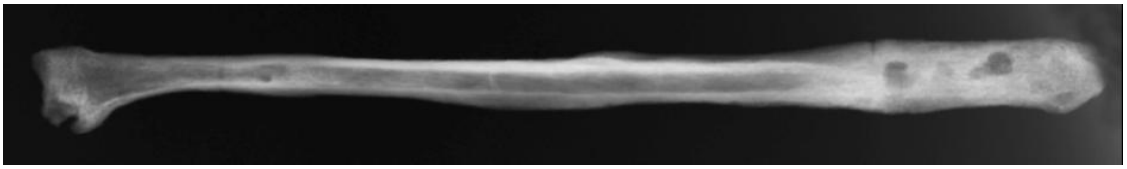


Fig. ii. - x-ray of fibula CR86.38 – note obstruction of distal medullary cavity

Fig. 11 – (CR86.45) close-ups, two views of distal end, middle diaphysis

Fig. 12 - Tibia(CR86.34) – Sabre (?) tibia, periosteal apposition on the anterior midshaft

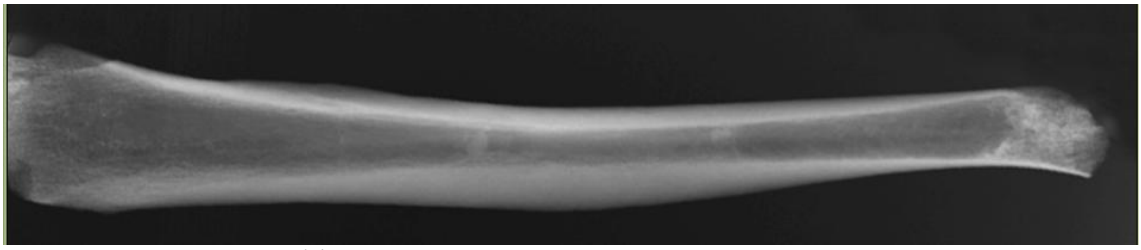


Fig. iii. - x-ray of Tibia CR86.34 –bone apposition on anterior midshaft visible

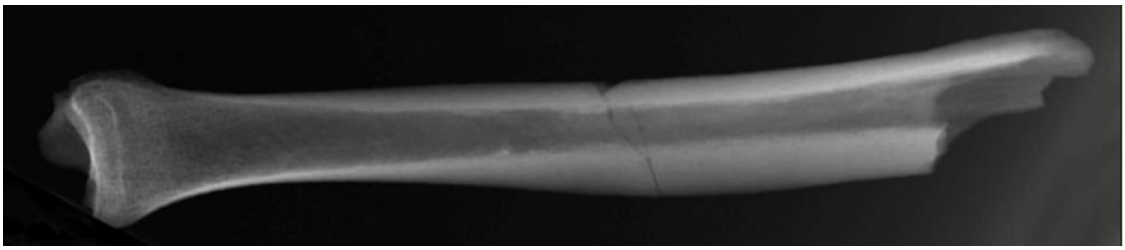


Fig. iv. Tibia CR86.40 - another possible sabre tibia

Table 2: Frequency of Periosteal Reaction in Cr86 Adults				
Elements, from most to least frequently affected by Yaws (Ortner & Putschar, 1985)	**Number of Elements Present**	**Number of Affected Elements**	**Percentage of Affected Elements**	**Location of Lesion**
Tibia	24	18	75	Either diffuse or along medial diaphysis
Fibula	19	4	21	entire element
Femur	26	6	23	entire length of diaphysis
Ulna	25	4	16	proximal
Humerus	27	4	15	distal
Radius	17	1	6	distal
Spine	220	0	0	--
Clavicle	19	3	16	variable
Hand	387	18	5	proximal
Foot	190	23	12	proximal
Skull	22	0	0	--
Ribs	123	11	9	variable, but not at costo-chondral junction
Pelvis	13	2	22	variable

Of the 13 metaphyses observable in this subadult sample, none showed osteomyelitic foci or bands of increased calcification. Table 3 summarizes the frequencies and locations of long bone periosteal apposition in the subadults. This periosteal reaction presents itself either as woven bone apposition (Fig. 14) or active periosteal remodeling (Fig. 15). The frequencies are consistent with a diagnosis of non-venereal treponematosis; however, two of the nine subadult tibiae in this collection were bowed slightly anterior-posterior, and both were aged to between two and five years (Figs. 16, vi). Sabre tibiae are not expected in the non-venereal treponemes in children under five. The presence of sabre tibiae in such young

individuals might suggest congenital syphilis, but the most diagnostic symptoms – the dental stigmata and osteochondritis – were not present in the surviving material.

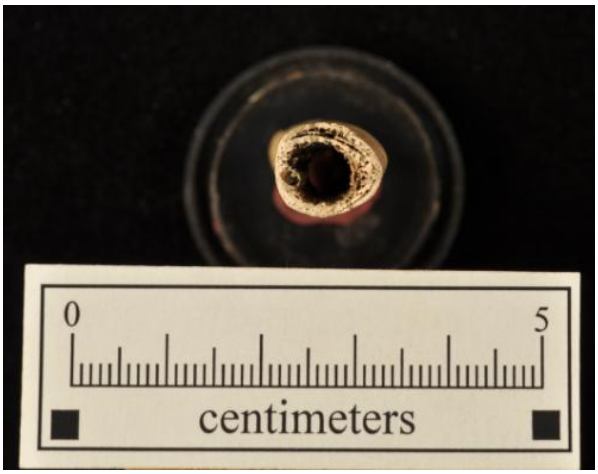


Fig. 13 - Periosteal cloaking

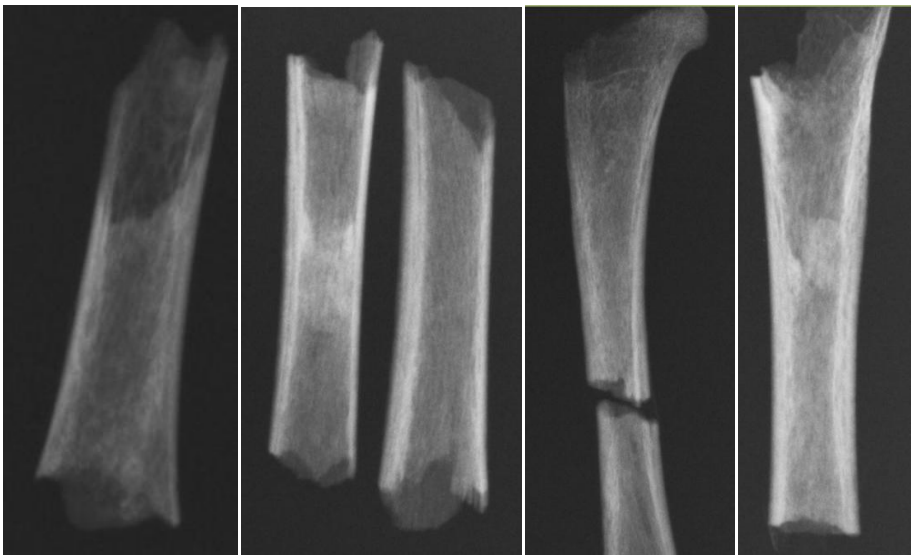


Fig. v. - Radiographs of periosteal cloaking. The double layer of cortical bone is clearly visible. (from left to right: CR86s7, s9, s5, s17, s16)

Fig. 14 – Femur (s13) with woven bone apposition

Fig. 15 – Tibia (s9) with active periosteal remodeling

Table 3: Frequency of Periosteal Reaction in Cr86 Subadults					
Elements, from most frequent to least (Ortner & Putschar, 1985)	**Number of Elements Present**	**Number of Affected Elements in Sample**	**Percentage of Affected Elements**	**Position of Periostitis on Diaphysis**	**Clarifying Notes**
Tibia	9	6	67	5 medial; 1 posterior	2 <2 yrs old; 3 from 2 to 5 yrs.
Fibula	indeterminate - too fragmentary	0	0	--	too fragmentary to determine count
Femur	10	3	30	2 lateral; 1 circumventing diaphysis	all <2 yrs old
Ulna	6	1	17	medial	<2 yrs old
Humerus	10	0	0	--	10 humeri, mostly distal epiphyses
Radius	5	0	0	--	5 radii
Spine	15	0	0	--	--
Clavicle	4	0	0	--	4 clavicles
Hand	10	0	0	--	--
Foot	5	0	0	--	--
Skull	6	0	0	--	MNI=6, mostly fragmentary
Ribs	30	0	0	--	--
Ilium	8	2	25	bordering acetabulum	0.5-2 yrs old; probably from same individual

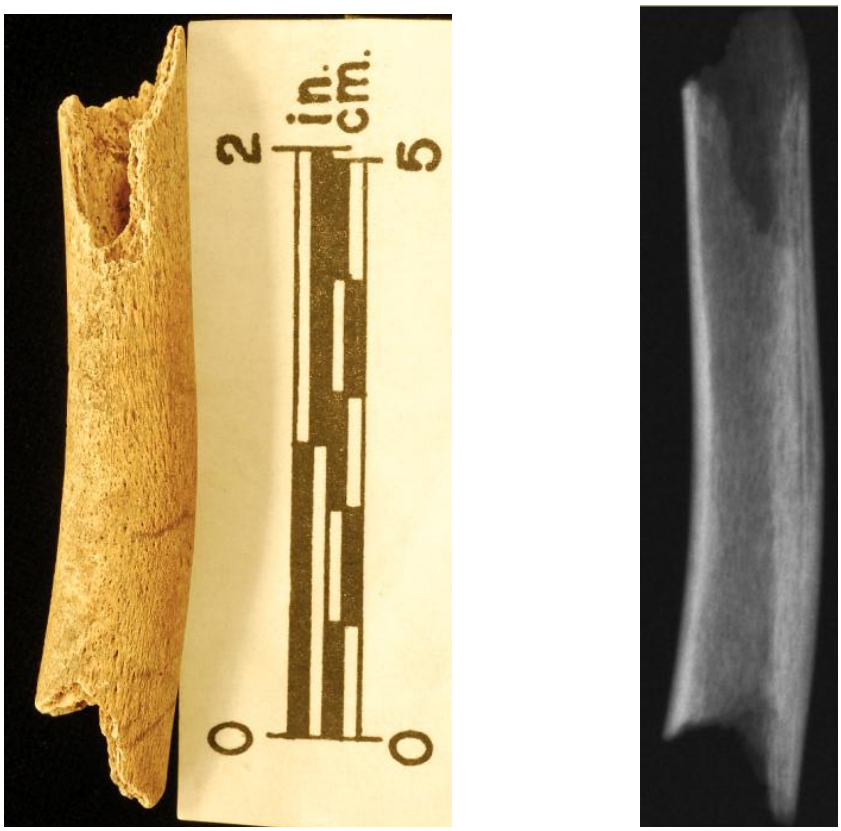


Fig. 16 (left) – tibia (s5), aged 2-5 years, bowed from anterior periosteal apposition
Fig. vi (right) – radiograph of same tibia (s5), anterior periosteal apposition clearly visible

Chapter 3:

Beyond Treponematosis: Other Pathological Conditions at Garbacon Creek

The following chapter is a discussion of the conditions most likely affecting the individuals of the Garbacon Creek ossuary sample, the clinical symptoms in living individuals, and the expected skeletal manifestations in archaeological specimens. The skeletal evidence for each of these conditions at Garbacon Creek is discussed after a review of the disease processes and the skeletal reactions each is expected to cause.

Non-specific Infection

Osseous reactions to tissue inflammation look very similar, regardless of the original cause of the inflammatory response, although clinical symptoms of various aetiologies may be differentiated in life. In a comingled burial, every skeletal element that shows signs of associated tissue inflammation cannot, and should not, be ascribed to a single pathological cause.

Periostitis describes new bone formation on the external cortex resulting from inflammation of the periosteum, which is caused by infection or trauma of immensely various types. Diagnosis of a specific cause is aided by analysis of the distribution of lesions across the entire skeleton, but most underlying causes of this response cannot be identified in this way. Although periostitis is a symptom of treponemal infection, most periostitis observed in any given sample is probably due to a number of other causes, and may not even be evidence of infectious disease (see Cook, in Powell and Cook 2005).

Osteomyelitis is the term applied to general osseous inflammation that extends into the marrow cavity. The term *osteomyelitis* is technically descriptive and not diagnostic, however the causative microorganism behind 90% of osteomyelitis cases is *Staphylococcus aureus* (Steinbock 1976; Scully et al. 1989). Other aetiologies, in order of descending frequency, are *Streptococcus*, *Salmonella*, fungi, viruses, tuberculosis, and the treponematoses (Ortner and Putschar 1985). The condition usually begins with a primary site of infection, at a skin lesion or subcutaneous abscess, from which the infection spreads hematogenously. Occasionally the bone can be directly infected following a compound fracture, which may then be followed by hematogenous dissemination of the pathogen (Steinbock 1976).

There are two forms of osteomyelitic reaction. Acute osteomyelitis is caused by pyogenic bacteria which invade the bone, periosteum, and marrow. It is rare in adults, and is seen most commonly in children between three and fifteen years of age (Steinbock 1976). It may affect any part of the skeleton, but generally favors long bone metaphyses, with sites of faster growth at higher risk. The instigating infection elicits an inflammatory response, and the resulting edema in the medullary cavity causes the swelling cortex of the bone to lay down new bone directly beneath the periosteum. Proteolytic enzymes involved in the immune response contribute to the resorption of trabeculae and the rarefaction of the internal bone structure (Steinbock 1976; Buckley 2000). Subperiosteal bone apposition eventually encloses the infected areas and forms an involucrum. Deprived of blood, the surrounded bone becomes necrotic, creating a sequestrum. The involucrum may be perforated at its weakest points by one or several cloacae, which allow the pus of the necrotic tissue to drain out of the marrow space (Steinbock 1976). Because infant bone remodels very quickly, osteomyelitis in an infant forms profuse involucra and sequestra. The femur and tibia, being the most rapidly growing bones, are the most commonly affected. In

children, only 15% of cases involve more than one bone, but in infants this percentage is higher (Buckley 2000).

When sequestra fail to drain, pyogenic osteomyelitis can become chronic osteomyelitis, which results in continuous osseous destruction and apposition around multiple nodes of infection. In 5-25% of cases, the hematogenous spread of the pathogenic organism may cause osteomyelitic foci in more than one bone, although usually no more than two. The balance of activity is appositional and a bone thus afflicted appears swollen (Ortner and Putschar 1985; Steinbock 1976).

Conditions which can be confused with osteomyelitis include skeletal tuberculosis, benign bone cysts, Paget's disease, and superperiosteal ossifying hematomas caused by trauma or scurvy (Truesdell 1995). The early skeletal manifestations of treponematosis may resemble pyogenic osteomyelitis, but usually more bones are involved in the systemic attack of *treponema*. The later sclerotic changes of treponemal infection can also be confused with osteomyelitis, but the pattern of treponemal new bone formation is more regular than that of most osteomyelitis-causing pathogens. Additional distinguishing points between the two are the rarity of cranial involvement in osteomyelitis, and the rarity of suppurative cloacae in treponematosis. Paget's disease can be identified by the involvement of multiple bones, the characteristic thickening of the cranium, and possible bowing of the long bones (Steinbock 1976; Bogdan 1989).

Osteological Evidence for Non-specific Infection at Garbacon Creek

The information on periosteal reaction in the adults is summarized in Table 2, and the information on the subadults is shown in Table 3. The subadults of Garbacon Creek presented many instances of periosteal woven bone apposition (Fig. 14) and active bone

remodeling (Fig. 15), but no sequestra or other evidence of osteomyelitis were found in the radiographs. Young children often acquire bacterial infections through the gastrointestinal tract when their diet begins to be supplemented with solid food in addition to breast milk during the weaning process. These infections can be spread hematogenously and cause widespread periosteal reaction in the skeleton. Any of the pathological subadult postcranial elements in this sample might be attributed to an infection of this type but, as Table 3 (p. 32) shows, the general pattern of periosteal reaction is consistent with a diagnosis of nonvenereal treponematosis (yaws or bejel).

In the adult sample, several elements showed medullary involvement in the radiographs (Fig. i). Two tibiae and one fibula had extensive remodeling with a swollen appearance in parts of the diaphyses and lytic foci which might indicate chronic osteomyelitis. The tibia (Figs. 9, i) which was paired with the fibula1 (Figs. 11, ii) had gummatous osteomyelitis and far more remodeling than any other adult skeletal element in the sample. Such extensive remodeling may be the result of a different disease process, perhaps a *Staphylococcus* infection with a primary focus of infection in the leg. But gummatous osteomyelitis is also a symptom in the tertiary stage of treponematosis, and variation in individual frailty could account for the pronounced symptoms in this particular case.

1 the bone around the articular facets of each had remodeled such that the pairing could be made with confidence

Nutrient Deficiencies which may complicate infection:

Iron and B12 Deficiency

Anemia is a term used to describe several different conditions in which the blood is lacking in either red blood cells, hemoglobin, or total volume (Hill 2001). The most notable clinical manifestations of anemia are fatigue, weakness, and memory loss; anemic children experience increased morbidity, decreased growth, and hindered psychomotor development (Ryan 1997). Anemias can be hereditary or environmental in cause. The anemias considered in this project are limited to nutritional anemias caused either by a deficiency in iron or in B12 because hereditary anemias such as sickle cell and thalassemia are most likely not indigenous to the Americas (Rucknagel 1966). Iron is the oxygen-binding component in hemoglobin, and a lack of iron results in the production of fewer red blood cells with decreased oxygen carrying capacity. Adults obtain 95% of the iron needed for hemoglobin production by recycling the iron from dying red blood cells, but infants in their first year of life depend on dietary iron for approximately 30% of their iron requirements (Dallman 1986). B12 is a water-soluble vitamin which aids in red blood cell production, metabolism, and nervous system functioning (Walker et al 2009).

Walker and coworkers (2009) have argued that iron-deficiency anemia (IDA) does not cause the skeletal lesions associated with anemia (porotic hyperostosis and cribra orbitalia, see below), since iron deficiency inhibits marrow hypertrophy rather than encouraging it. Iron deficiency results in a hemoglobin shortage and consequent lower levels of mature red blood cell production. Megaloblastic anemias, on the other hand, produce red blood cells with a shortened life span, and the body compensates by increasing the space devoted to red blood cell production. B12 and folic acid deficiency are common causes of megaloblastic anemia and have many of the same risk factors.

Any nutritional deficiency can be caused by a diet chronically lacking in sufficient amounts of the nutrient in question. B12 and heme iron, the more biologically available form of iron, are found only in animal-derived foods. Non-heme iron is found in a variety of plant foods, usually dark leafy greens, but the nutritional value of a diet can only be considered in context – a diet high in calcium, polyphenols (e.g. tannins), the proteins in dairy and eggs, excessive fiber, and phytates (found in cereals) can impair non-heme iron absorption, while it is absorbed more effectively when eaten in combination with vitamin C (Garn, in Stuart-Macadam 1992; Ryan 1997). Conversely, those suffering from scurvy may be at higher risk for developing IDA, and scorbutic individuals have shown a decreased ability to mobilize iron from storage in reticuloendothelial cells (Ryan 1997). While deficiency in vitamin A (a major dietary source of which is liver) does not inhibit absorption, it does inhibit the mobilization of iron stores, for reasons which are not well understood (Ryan 1997).

In addition to dietary insufficiency, upper respiratory infections, ear infections, and gastroenteritis can stimulate iron withholding, a mechanism by which the reticuloendothelial cells of the body refuse to mobilize their iron stores and, in doing so, deprive parasitic organisms of the iron necessary for their reproduction (Ryan, 1997). But this "anemia of inflammation" (WHO 2007) interrupts the bone marrow's usual response to erythropoietin, decreases red blood cell production, and sequesters iron in storage forms such as ferritin, which are less bioavailable. The prevention of marrow hyperplasia in this type of anemia means that it too should possibly not be considered responsible for porotic hyperostosis.

In opposition to Ryan, Hill (2001) believes that "rather than contributing to the adaptive fitness of the population, anemia is more likely an indication of physiological exhaustion and metabolic failure in most instances." A body fighting infection is less adept

at absorbing ingested nutrients, and iron deficiency can also cause mild atrophy of the lymphoid tissues and depletion of lymphocytes (Chandra and Newberne 1977). Certain parasites (e.g. hookworm) also cause direct blood loss which can contribute to the depletion of the body's stores of iron and B12. Although neonatal iron stores can ward off iron deficiency for the first months of life, Walker and coworkers (2009) suggest that B12 deficiency in infants begins in utero – infants are born with low B12 levels inherited from their asymptomatically B12-deficient mothers, whose breast milk is also low in the essential nutrient. An infant's iron requirements double within its first year, but adequate prenatal iron stores should provide a cushion for the first four to six months of life. Children are at their highest risk of developing IDA between six months and three years of age (Ryan 1997).

Porotic Hyperostosis

Marrow hypertrophy of the cranium, which manifests macroscopically as porotic hyperostosis, has been taken as an osteological indicator of anemia since Angel's work in the 1960s (Angel 1966). In children, the primary sites of red blood cell production are the spongy tissues of the skull and the marrow cavities of the long bones (Walker et al. 2009). The skeletal reaction to anemia is to increase the tissue space devoted to haemopoiesis, the production of red blood cells. In the cranium, this new haemopoietic tissue expands the spongy diploe and causes the cortex of the skull to become thinner, a change most readily apparent in the eye orbits, where the disappearance of the cortex reveals the underlying spongiosa, a symptom referred to as *cribra orbitalia* (Buckley 2000). Unless the trabeculae are enlarged, this change resembles the initial reaction of the bone to the orbital hematomas of scurvy or to non-specific infection (Ortner & Mays 1998). The endocranial surface of the involved cranial bone becomes thinned but is not perforated like the ectocranial surface,

while the enlarged trabeculae of the diploe are visible on the ectocranium, particularly on the frontal and parietals. Hill (2001) cautions that signs of periosteal reaction on the endocranium cannot be attributed to anemia and should signal to the palaeopathologist the presence of a separate pathological condition.

Hill (2001) also states that postcranial lesions on subadults under five years old caused by anemia or infection are indistinguishable from each other: both cause periosteal woven bone apposition on the long bone diaphyses. In adults the osseous lesions of anemia, when observed, are always remodeled, and are probably evidence of a childhood anemic episode. In the final stages of osseous healing the lesions may only be visible as irregular lumps of osteoid on the ectocranium of the frontal and parietal bones (Hill 2001).

Periosteal Cloaking

Angel (1967), Tayles (1996), and Hill (2001) have all recorded pathological changes in subadult long bones, particularly the tibiae, which appear as acute periostitis and look like a "bone-within-bone" in cross section and radiograph (see Figs. 13, v). It frequently involves the long bone diaphyses exclusively. Tayles (1996: 17) describes it as "reactive radial subperiosteal bone," and Nabarro describes a "layer of bone and red marrow, or rarely granulation tissue, being deposited outside the original corticalis" in cases of both congenital syphilis and rickets. I will continue to refer to this phenomenon as "periosteal cloaking."

Hill (2001), who believes periosteal cloaking to be connected with anemia, says that it should not be observed in children over age five, since the long bones cease to be involved in red blood cell production at this time. Indeed it is not observed in the Garbacon Creek sample in subadult postcrania older than this. It is, however, seen in various long bones, mainly tibiae, in the remains of subadults younger than five years. In support of its

connection to anemia, Hill cites the notable absence of woven bone apposition in a series of clinical radiographs which showed periosteal cloaking and the common co-occurrence of anemia in patients with periosteal cloaking. Indeed, in the Garbacon Creek sample, five of the seven instances of periosteal cloaking occur without any external evidence of periosteal reaction.

It is undetermined as of yet whether periosteal cloaking is, in fact, pathological. Glaser (1949) believed it to be part of the normal growth process of neonate long bones. In a radiographic study of 335 healthy newborns, Shopfner (1966) noted this double contour in long bones of 35% of the infants and saw that the second layer began to be incorporated into the underlying cortex in the older individuals of the study.

Osteological Evidence for Anemia at Garbacon Creek

Despite their ambiguous and multifactorial aetiology, I will consider cribra orbitalia, porotic hyperostosis, and periosteal cloaking as potential signs of anemia. Of the 17 adult crania from Garbacon Creek, 8 (47%) showed signs of porotic hyperostosis (Fig. 20) and 3 (18%) showed signs of cribra orbitalia. In the subadult crania, only one parietal fragment was observed with active porotic hyperostosis (Fig. 17). Three orbital roofs, two belonging to the same individual (cranium 7a), had pathological lesions; however, the orbital lesions on 7a consist of spiculated new bone formation and the bony destruction seems to trace a vascular pattern (Fig. 18). These lesions do not exhibit the characteristic expansion of the trabeculae in the anemic response, and it is likely that they are instead a response to orbital hematomas in a scorbutic individual (see below) rather than marrow hypertrophy occurring as a response to anemia. Hill (2001), however, would consider this kind of blastic hyperostosis to be within the normal range of cribra incarnations. The eye orbit of the

second individual, on the other hand, (Fig. 19) shows slightly enlarged trabeculae that may be indicative of marrow hypertrophy.

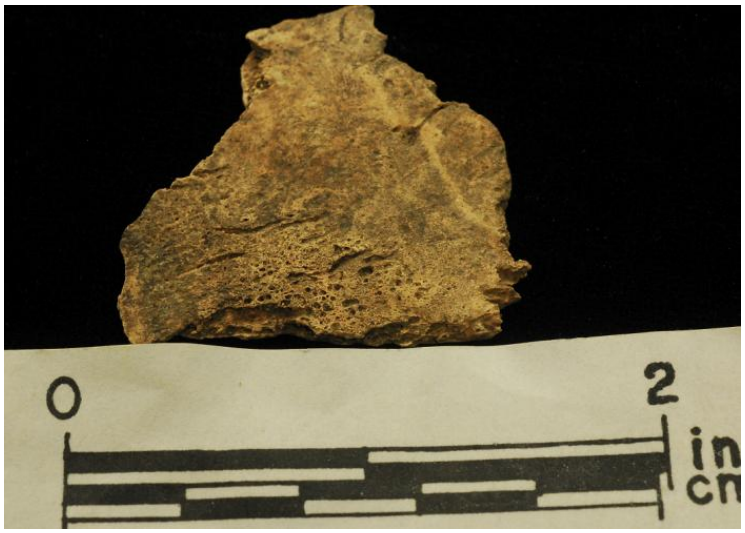


Fig. 17 - Subadult parietal fragment with hypertrophic porotic hyperostosis

Fig. 18 - Cranium 7a frontal – symmetric hypertrophic lesions in eye orbits

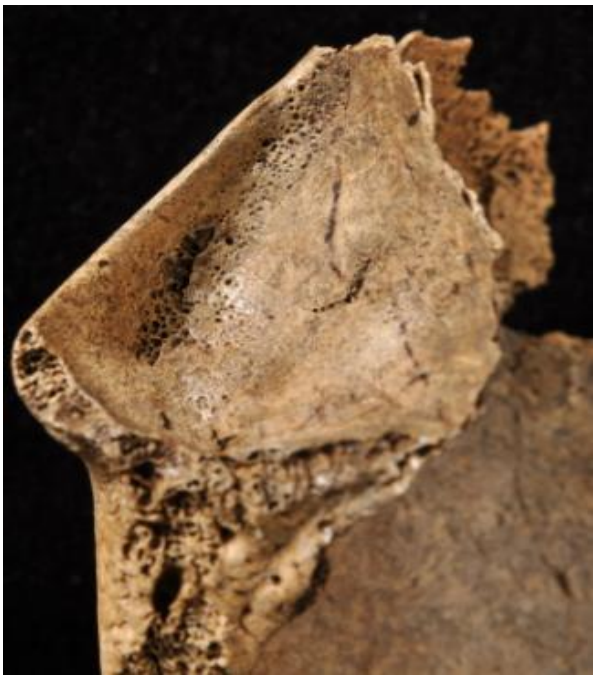


Fig. 19 - Subadult eye orbit – cribra orbitalia

Fig. 20 - Porotic Hyperostosis (cranium #6)

Scurvy

Scurvy (Vitamin C deficiency) is caused by a lack of vitamin C in the diet, and is most commonly seen in infants between five and twenty-four months of age (Buckley & Ives, 2006). Even if an infant's diet completely lacks vitamin C from birth, prenatal stores will meet the child's need until sometime in this age range (Buckley 2000). In their survey of scurvy in 22 of 557 subadults examined from North American archaeological sites, Ortner et al. found that 55% of observed scurvy cases occurred in individuals under 3 years of age, and 32% occurred in individuals between 3 and 7 years of age (2001). Tamura and colleagues (2000) suggested that it could develop in infants after only two to four months of inadequate vitamin C consumption, whereas healthy adults would have to be deprived for a longer period before clinical symptoms would be expected to appear, due to the slower rate of adult tissue turnover. The clinical symptoms of scurvy include lethargy, tiredness, diminished growth in children, hematomas, and musculoskeletal pain and weakness. Vitamin C is necessary for the formation of collagen and the cement-like substance on the endothelium of blood vessels which anchors them in place; a deficiency therefore results in depressed bone formation and fragile blood vessels which are liable to hemorrhage with minor trauma (Buckley 2000; Jaffe 1972).

Osseous Lesion Morphology

Scorbutic bone changes are typically symmetric and involve multiple elements in a single individual. Most of the skeletal symptoms of scurvy are the result of the body's response to localized hemorrhage. Subperiosteal hemorrhage, usually due to slight mechanical trauma, lifts the periosteum off the bone, an event known as periosteal stripping. This is more common in subadults, as the periosteum is less securely attached to the

underlying bone during this period of growth and development. The vascular response to hemorrhage is to create a proliferation of capillaries at the site of the spill in order to draw away the pooling blood, and the localized bone becomes more porous, filled with the tracks of additional capillaries (Buckley 2000). This reaction has been reported most often on the diaphyses of the lower limb bones, but may also occur on the ectocranial bosses of the parietals, creating Parrot's swellings (Ortner and Ericksen 1997). Similar cranial lesions are known to occur in cases of rickets (Fig. 30) (Ortner and Mays 1998; Ortner and Putschar 1985). Ortner and Ericksen (1997) note that, when porosity is observed on the skull, the porosity that results from the response to hemorrhage should be differentiated from marrow hypertrophy, a symptom of anemia which is caused by expansion of haemopoietic cells in the cranial vault (see above).

Common sites of hemorrhage in scorbutic individuals are generally places that experience mechanical stress where the associated arteries lie between the muscle and bone, the *temporalis* muscle being the most notable example of this in the skull. Areas associated with the *temporalis* muscle are the posterior maxillae and temporal fossa, which includes the greater wing of the sphenoid. Ortner and Ericksen (1997) consider porosity on the greater wing of the sphenoid to be a particularly diagnostic symptom. Signs of hemorrhage are common in the roofs of the eye orbits, the result of minor eye movements which are enough to rupture the weakened ocular vessels of scorbutic individuals. In a series of 34 autopsies on scorbutic patients, Fraenkel (1929) found four with evidence of porotic and hypertrophic lesions of the orbital roofs. Buckley and Ives (2006) maintain that the cranial lesions of scurvy are unlikely to appear without accompanying postcranial lesions, and despite the distinctive anatomical patterning of cranial lesions, a diagnosis of scurvy should not be made based on the presence of orbital and cranial lesions alone.

In the postcrania, the muscles with vascular supplies lying next to the bone are the *supraspinatus*, *infraspinatus*, and *subscapularis*. Signs of hemorrhage involving these muscles would be found on the scapula at the supraspinous fossa, infraspinous fossa, and subscapular fossa respectively, and have diagnostic force nearly as strong as signs of hemorrhage on the greater wing of the sphenoid. Scurvy may cause the cancellous bone to atrophy and the cortical bone to grow thinner across the entire skeleton. Buckley (2000) notes that "shell-like" periosteal apposition on the diaphyses of the limbs is sometimes seen as part of the post-hemorrhagic healing process – her description does not clarify whether this is describing "cortical cloaking" or "bone-within-bone", or merely localized woven bone apposition. When continual minor trauma results in chronic bleeding at the joints, the metaphyses become widened and cup-shaped, but Buckley and Ives (2008) suggest that the absence of this feature may merely imply a lower level of scurvy than is generally observed in clinical cases.

Although scurvy impairs bone formation, an individual who recovers from the condition resumes normal osteoblastic activity and will form new bone where hemorrhage has separated the periosteum from the underlying cortex (Ortner & Putschar, 1985). An individual who dies without recovering from scurvy will not show proliferative bone reactions to hemorrhage; an individual who survives significantly past a scorbutic episode will have all skeletal evidence of the deficiency erased. Following the reintroduction of adequate levels of vitamin C into the diet clinical symptoms improve rapidly; Follis and coworkers (1950) demonstrated the complete disappearance of histological signs of infantile scurvy after three months on a vitamin C-rich diet.

Osteological Evidence for Scurvy at Garbacon Creek

In the postcrania of the Garbacon Creek subadult sample, periosteal reactions presented either as woven bone apposition (Fig. 14), which was always localized, or as active periosteal remodeling (Fig. 15), which tended to extend along entire diaphyses (although not necessarily around the full circumference). I am not sure whether the distinction between the two reflects a difference in the stage of osseous reaction, or whether they represent reactions to two distinct aetiologies: the woven bone apposition being a response to periosteal stripping and the periosteal remodeling a response to hematogenous infection. Each type of bone change occurred both in concert with periosteal cloaking and without it, and periosteal cloaking was sometimes visible in cross section on elements without any signs of periosteal reaction. For a summary of periosteal reactions in the subadults, see Table 3. None of the metaphyses present showed any visible signs of pathology, although only 37.5% of subadult long bones had intact metaphyses.

Of the 26 adult scapulae, the two pathological specimens had active bone deposition on the subscapular fossa of the ventral body, and one also had active bone on the spine and acromion process. Of the seven subadult scapulae, two (aged 5-10) had pathological activity: both with porosity on the infraspinous fossa, and one with active bone deposition on the supraspinous fossa, along the axillary border, and also on the subscapular fossa. The sites of these lesions align with the expected pattern of scorbutic hemorrhaging associated with the *infraspinatus*, *supraspinatus,* and *subscapularis* muscles.

The frontal bone of cranium 7a, aged 5-10 years, has hypertrophic lesions in the eye orbits and on the superior frontal, and porosity on the lateral border which may be attributed to scurvy (Figs. 18, 24). Two mandibular halves, which may belong to the same individual, aged 5-9 years, show porosity along the condyles and the rami (Fig. 25), possibly

the result of scorbutic hemorrhage as a result of mastication. A fragment of probable subadult sphenoid was identified (Fig. 26), with porosity on what should be the greater wing[2], and two of the three zygomatics had marked porosity on their orbital rims (Figs., 27, 28). The scorbutic symptoms of the Garbacon Creek subadults are summarized in Table 4.

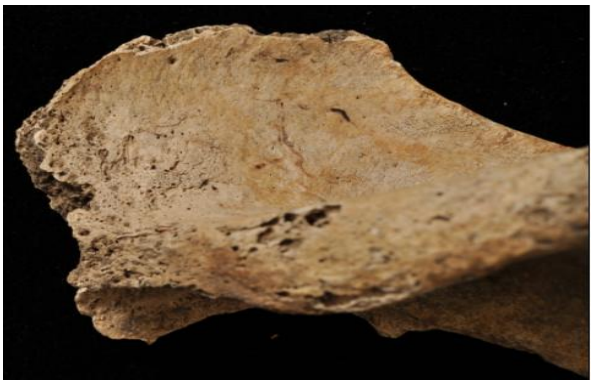


Fig. 21 - Periosteal reaction on adult scapula, infraspinous and supraspinous fossae

Fig. 22 - Periosteal reaction on adult scapula, acromion process

[2] If the fragment is not sphenoid then it is part of the lateral border of the frontal and is still part of the temporal fossa, which may be affected by hemorrhaging associated with the *temporalis* muscle.

Table 4: Scorbutic Symptoms in the Cr86 Subadults

(modified from Ortner and Ericksen, 2007; Brickley & Ives, 2008)

	Pathology by Element	Pathology Observed	Total Observable Elements Present	Clarifying notes
Cranium	abnormal porosity of cortex			
	- calvarium	0	6	--
	- sphenoid	0	0	--
	- maxilla	0	0	--
	- orbits	3	7	2 from a single individual
	- internal zygomatic	2	3	--
	- coronoid process of mandible	2	5	L/R fragments, both age 5-9; possibly a single individual
	new bone formation			
	- orbits	2	7	single individual, age 5-10
	- cranial vault	2	6	Cranial MNI = 6; pathological elements may be from same individual
Ribs	enlargement at costochondral junction - scorbutic rosary	0	approx. 30	may also indicate rickets
Scapulae	abnormal porosity of cortex			
	- supraspinous fossa	1	7	--
	- infraspinous fossa	2	4	one scapula (CR86s47) showed both supra/infraspinous
	- subscapular fossa	1	4	same scapula (CR86s47)
Long Bones	new bone formation	11	40	--

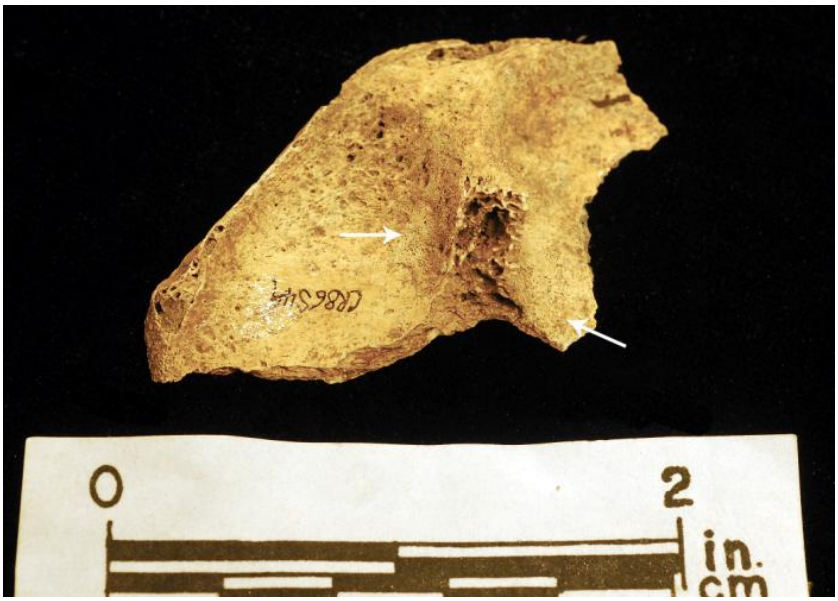


Fig. 23 - Periosteal reaction on subadult scapula, infraspinous and supraspinous fossae

Fig. 24 - Cranium 7a frontal – porosity on lateral border

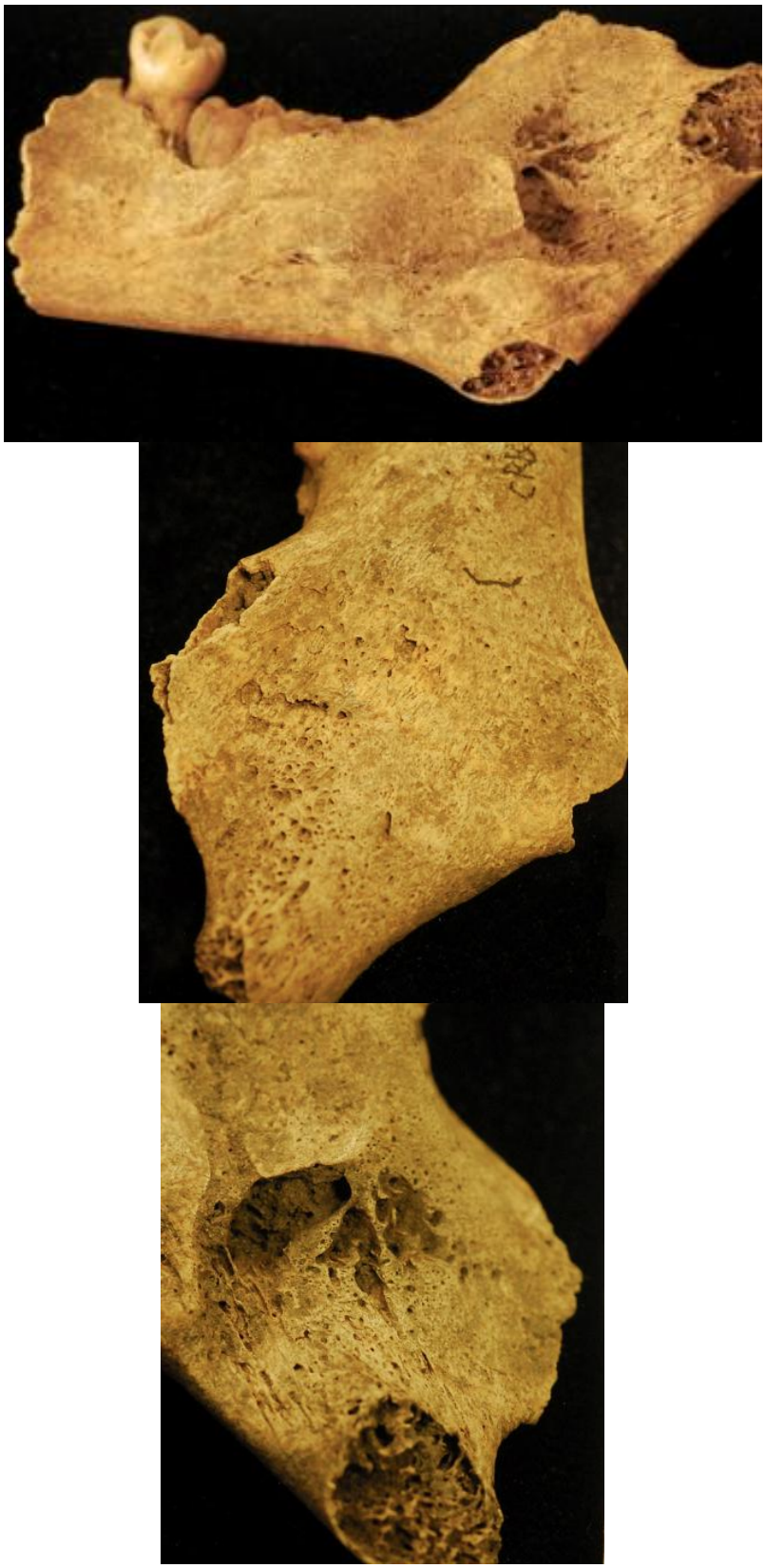
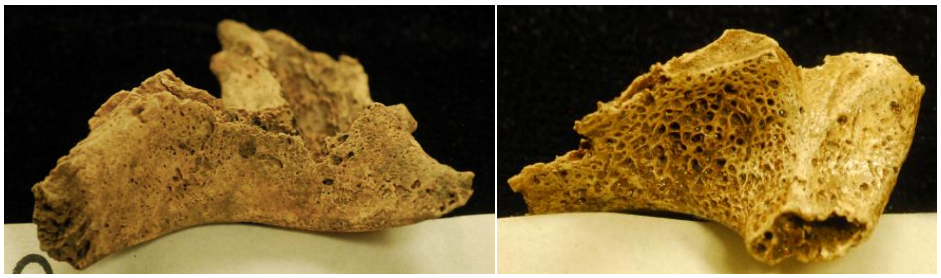


Fig. 25 - Subadult mandible (aged 5-9 years), porosity along ramus (multiple views)

Fig. 26 - probable sphenoid (two views), with porosity on both visible surfaces



Figs. 27 (left) and 28 (right) - orbital rims of subadult zygomatics. Note porosity.

Rickets

Rickets is the term applied to improper bone mineralization in children caused by vitamin D deficiency. Vitamin D aids intestinal mineral absorption and regulates the levels of calcium and phosphorus available in the bloodstream to support bone mineralization. Vitamin D is found in dietary sources such as eggs and oily fish, and exposure to sunlight is necessary for synthesis of its metabolically active form (Holick, 2003, cited in Brickley & Ives, 2008). The most common cause of rickets is insufficient sunlight exposure, but a diet low in protein and high in the phytates found in cereals can make calcium unavailable to bone growth and interfere with vitamin D metabolism (Pettifor & Daniels 1997:673). Rickets occurs most often in infants between three and eighteen months of age (Pettifor, 2003, cited in Brickley & Ives, 2008).

Osseous Lesion Morphology

The most characteristic symptom of rickets is the abnormal curvature of weight-bearing bones whose structure has been compromised by a failure to follow cartilage development with mineralized bone. The cartilage continues developing regardless of osteoid deposition and becomes disorganized; the disorganization is exacerbated by mechanical forces. When vitamin D once again becomes available, the bone mineralizes onto the misshapen cartilage template. In individuals who die before the condition reaches this stage, it is manifest as fraying and flaring at the metaphyseal rim and porosity in the areas which were filled with unmineralized osteoid at time of death (Brickley & Ives 2008). The bony changes of rickets are most commonly observed in the distal metaphyses of the femur, radius, ulna, and the proximal humerus, as well as the osteocartilagenous junctions of the ribs (the ragged flaring of the 'rachitic rosary,' which closely resembles that of the

scorbutic rosary). The metaphyses of the long bones tend to flare outwards, and the ribs become rounded and nodular, with a flattening of the natural pleural curve (Ortner and Putschar 1985). Vertebrae may also be compressed.

The expression of the condition depends in part on the age of the affected child. If the child is crawling but not yet walking, then the bones of the arm, especially the ulna, are likely to show deformity; if the bones of the leg are subject to the mechanical forces associated with walking, then they will be the bones most affected. The nutritional status of the child also determines rachitic manifestation: malnourished children develop porotic rickets, with concomitant stress fractures and abnormal curvature in axial weight-bearing bones; well-nourished children develop hyperplastic rickets, which creates plump bones with narrowed medullary cavities (Ortner and Putschar 1985). A summary of the characteristics in the subadult sample which are diagnostic of rickets is presented in Table 5.

Vitamin D deficiency in adults results in osteomalacia, which can also be brought on by general malnutrition, but is most commonly caused by deficiencies in protein, fats, calcium, and phosphorus. Women of child-bearing age who have endured multiple closely-spaced pregnancies are at highest risk. Changes are most apparent in the bones containing the most cancellous bone, namely the ribs, sternum, vertebrae, and pelvis (Ortner and Putschar 1985).

Table 5: Macroscopic Features of Rickets Observed in the Cr86 Subadults

(modified from Brickley & Ives, 2008)

	Pathology by Element	Pathology Observed	Total ObservableElements Present	Clarifying Notes
Cranium	layers of irregular, porous bone formation	1	6	may also indicate scurvy or anemia
	medial angulation of mandibular ramus	0	5	--
Ribs	change in rib neck angle	0	approx. 30	--
	enlargement of costochondral junctions – 'rachitic rosary'	0	approx. 30	may also indicate scurvy
Pelvis	exaggerated curvature of ilium	0	9	--
Long bones	flaring/swelling of distal metaphyses	0	6	may also indicate scurvy
	fraying at growth plate margins	0	30	may just be post-mortem damage
	cupping' of metaphyses	0	30	may also indicate post-mortem damage or scurvy
	bending (valgus and varus)	2	23	'observable' elements considered >1/2 diaphysis
	cortical thickening	9	40	may also indicate scurvy, congenital syphilis, non-specific infection, trauma

Osteological Evidence for Rickets at Garbacon Creek

The evidence for vitamin D deficiency at Garbacon Creek seems to indicate a sub-clinical level of deficiency which may have exacerbated the symptoms of other conditions. The lesion on cranium 7a (Figs. 7, 8) closely resembles the lesion on the cranium of a clinical case of 7 month old child with rickets and scarlet fever (Fig. 30) (Ortner and Putschar 1985). A right and left femur which, based on their nearly identical morphology, appear to be from the same individual, both showed a borderline abnormal degree of remodeling along the posterior border in x-ray, potentially cortical thickening correcting early anterior-posterior bowing of the diaphyses (Fig. vii). A few tibiae and ulnae showed slight valgus-varus curvature which may be outside the normal range (Fig. 29), but no lateral bowing was apparent in the limbs of the subadults.

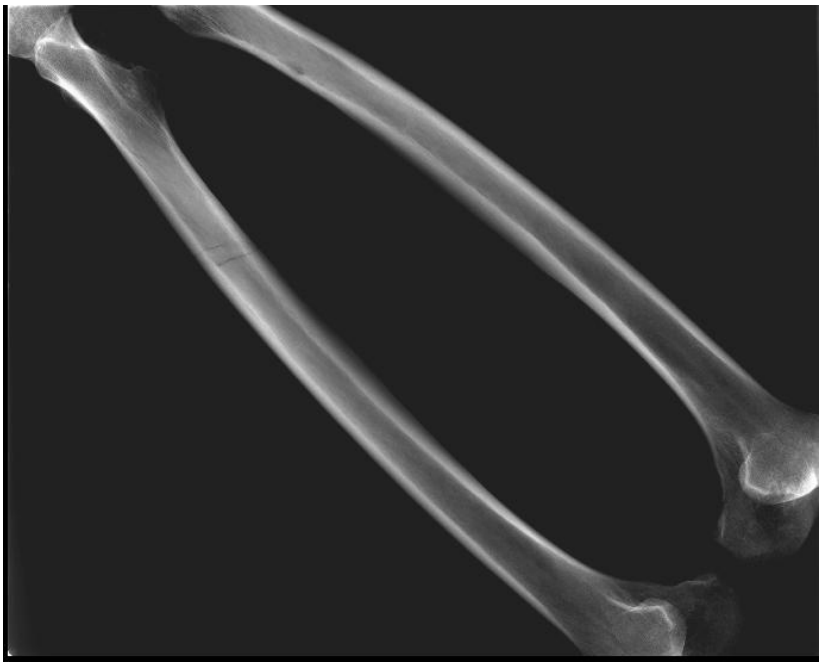


Fig. vii. – Posterior bone apposition to correct bowed diaphysis – possible healing rickets?

Fig. 29, bowed adult ulna below, with normal specimen above for comparison

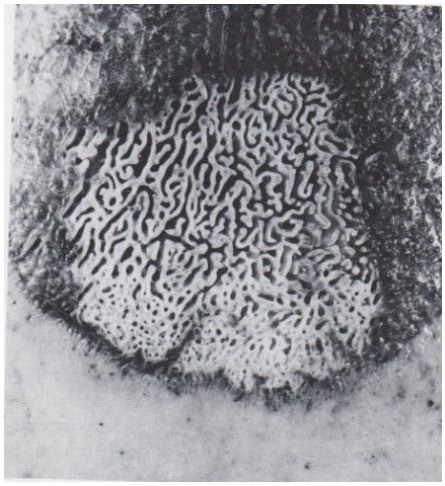


Fig. 30 - Cranial lesion from a 7 month old child with rickets and scarlet fever (Ortner and Putschar 1985)

Chapter 4:

Infection, Nutrition, and Immunity at Garbacon Creek

The Picture of Health

Table 6: Features with Multiple Diagnostic Possibilities

Feature	Rickets	Anemia	Scurvy	Treponematosis
costochondral swelling of the ribs	X	o	X	o
widened metaphyses	X	o	X	o
porosity of the upper orbitals	o	X	X	o
marrow hyperplasia of the skull	X	X	X	o
periosteal cloaking	X	X	o	X
periostitis	o	o	X	X

Key:	
X	**feature diagnostic of condition**
o	**feature not seen in condition**

None of the pathological features observed in the Garbacon Creek skeletal remains were pathognomonic for a specific infectious or nutritional condition (see Table 6), but a careful consideration of the evidence does suggest a probable picture of the disease landscape. Buckley (2001) has theorized that children living with a background of endemic

disease and periodic nutritional deficiency will develop bones that are very susceptible to slight trauma, and suggests that this might be the ultimate cause of the diffuse periostitis observed in her study sample of Polynesian subadult remains from Tonga. The same might be true of the children at Garbacon Creek.

There is strong evidence of treponemal infection among the Garbacon Creek adults, and the pattern of periosteal reaction in the subadults suggests a similar cause. Since no dental stigmata or osteochondritis were observed in the infants, and cranial involvement among the adults was scarce, it is probably a nonvenereal form of treponematosis (either yaws or bejel). Curiously though, some of the young subadults (2-5 years) have extensive periosteal apposition on the anterior tibiae, which might suggest the sabre shin of Fournier characteristically found in congenital syphilis. Periosteal involvement of the anterior tibia is, however, not an unusual phenomenon, since the bone's anterior crest lies close to the surface of the skin and is often implicated in skin infections of the lower extremities and daily minor trauma, although trauma in this bone is less expected in non-ambulant subadults. Additionally, we should keep in mind that pathogens do evolve, and the variant of treponemal infection experienced by peoples of Pre-Columbian North America might not necessarily correlate precisely to one of the four diseases known today (Powell and Cook 2005).

If porotic hyperostosis and cribra orbitalia are taken to be evidence of anemia, then anemia was present in the Garbacon Creek population. Anemia is one of the most common health conditions in the world, but the potential causes of anemia in the Late Woodland Garbacon Creek population nevertheless merit some discussion. The possible types of anemia in this part of the world are non-genetic anemias: iron-deficiency anemia and megaloblastic anemia caused by B12 deficiency. Both can be attributed to consumption of a

plant-based diet which does not include enough bioavailable iron or B12. Seasonal or unforeseen variation in availability of local foods makes this kind of limited diet a possibility. Women are generally more heavily affected by anemia, due to blood loss during menses and heightened iron requirements during pregnancy. Infants and small children are the group most at risk, since neonates born to anemic mothers will be born with lower-than-optimal iron stores and the iron-deficient breast milk of mothers will soon result in the iron-deficiency anemia of their infants. Iron-poor weaning foods could exacerbate this situation. The World Health Organization (2011) estimates that the iron needs of a rapidly growing child are more than ten times the need of an adult male, when calculated per kilogram of body weight.

However, insufficient ingestion of iron is not the only possible explanation for anemia at Garbacon Creek. Coastal populations are at risk of acquiring helminthic parasites such as fish tapeworm from consuming undercooked seafood, or hookworm by traveling barefoot. These parasites cause intestinal bleeding, and the resulting blood loss may contribute to the development of anemia (Albonjco et al. 1998). Fish tapeworm also competes directly with the host for B12 (Herbert 1959). In a high mobility population relying heavily on estuarine resources, this kind of parasitism is a distinct possibility. In addition, scurvy can indirectly cause megaloblastic pernicious anemia, since vitamin C is closely linked to folate formation (Herbert 1959). An insufficiency of vitamin C can inhibit proper iron metabolism, and blood loss from scorbutic hemorrhaging may contribute significantly to anemic potential.

The periosteal lesions on a number of the scapulae, the porosity on parts of some of the subadult mandibles, and cribra orbitalia in the subadults without convincing trabecular enlargement all point to the presence of scurvy. The cause of scurvy is usually a

monotonous diet devoid of fresh fruits and vegetables. This is the misfortune of those relegated to eating stored provisions for months on end, a winter situation common to some extent in most groups subsisting in non-tropical environments. In the northern hemisphere the months of February and March may be particularly scarce for provisions. However, the first foods to become available at this time of year are young green shoots which are abundant in vitamin C, and so the problem was likely one which arose and resolved itself with the turn of the seasons. John Lawson (1701) believed that the Indians of Carolina, "are never troubled by the Scurvy," but they may often have been affected at a sub-clinical level which did not manifest with symptoms that Lawson would have recognized.

Cultural and Biological Factors

In these signs of malnutrition we are perhaps seeing the effects of a culturally prescribed limitation on 'appropriate' foods. Unfortunately, ethnographers in the past did not ask all the questions that interest anthropologists today, so we are left lacking evidence on this topic. Such a limitation might apply to sick individuals, unfortunately complicating another disease condition with the addition of scurvy. Native American remedies often followed the principle of "like cures like," and this might not always result in the proper nutritional support for an ailing individual (Brett Riggs, personal communication, March 2012). A diet restricted for medical reasons would likely not affect the skeletons of adults, whose systems take longer to react to deficiencies; Tamura and colleagues (2000), on the other hand, have suggested that an infant may develop scurvy in as short a time as two months.

A dietary limitation might apply instead to all children in a certain age group. Most societies have their own ideas about what constitutes a proper weaning diet. In agricultural

societies it is often some form of cooked grain – high in carbohydrates, but lacking in protein and other essential nutrients. There is no ethnographic evidence for types of weaning foods among contact-period Native Americans, but if local availability dictated food choices, then acorn gruel may have been an option in this area (M. Scarry, personal communication, March 2012). Acorns provide carbohydrates and fats, and are a good source of magnesium, potassium, copper, and folate, but contain few other micronutrients and little protein (USDA 2012).

Included in the new, limited diet of a weanling are, inevitably, a whole host of pathogens with which the child's immunologically naïve gastrointestinal system must cope. We may be seeing the underlying osseous reaction to tissue inflammation from any of a slew of infectious microorganisms that enter a child's system via supplementary food during the weaning process. A low-grade infection can cause a cycle of nutrient malabsorption and direct nutrient wastage via diarrheal stools at the same time as the febrile response heightens nutrient requirements, and the ensuing immune suppression can leave a child open to further infection. A child being fed an inadequate diet is more susceptible to infection by the pathogens he or she encounters and, starting at a disadvantage, is worn down even further as the infection inhibits nutrient absorption via various pathways.

Vitamin C plays an important role in the immune response, being involved both in the production of antioxidants and the active destruction of pathogens. Vitamin deficiency results in a general suppression of immune function. Vitamin B12 is also implicated in general immune function, although the mechanism involved is not well understood (Chandra and Newberne 1977). Anemia inhibits the intestinal absorption of nutrients, including vitamin D and calcium, although no connection has been found between deficiencies in vitamin D or calcium and depressed immunity. Vitamin C is involved in the

production of collagen, so our view into coincident nutritional deficits is hampered because scurvy prevents the deposition of osteoid, so that vitamin C deficiency may mask the typical rachitic expression in an individual with deficiencies in both C and D. Although not directly linked to an increased susceptibility to infection, vitamin D deficiency or another interruption of calcium and phosphorus metabolism often magnifies the evidence of growth interruptions caused by other diseases, such as congenital syphilis (Nabarro 1954). If an individual suffers concurrently from rickets, scurvy, and anemia, he or she may not manifest rachitic bowing, but will be very susceptible to infection and to hemorrhage from slight trauma, both of which are possible causes of the periosteal reaction we see on the long bone diaphyses in the Garbacon Creek subadults.

The evidence for vitamin D deficiency in this sample is slight, but a subclinical level of vitamin D deficiency may have been responsible for exacerbating the skeletal symptoms of other conditions. Native Americans approached indoor space as an area mainly for storage and shelter against inclement weather, and it would have been highly unusual for a child to be sequestered indoors long enough to develop vitamin D deficiency (Brett Riggs, personal communication, March 2012). Nevertheless, the lesion on the frontal from subadult cranium 7a (aged 5-10 years) very closely resembles that of a 7-year-old English child who died in hospital with both rickets and scarlet fever (Fig. 30) (Ortner and Putschar 1985). The individual represented by cranium 7a probably suffered from a combination of nutritional disadvantage (very likely scurvy) and infection (potentially treponematosis, but equally possibly a non-specific childhood illness), possibly causing a severe febrile response and disruption of calcium metabolism. A subclinical level of vitamin D deficiency could explain the severity of the lesion, and it is possible that a child with scurvy would have

remained sedentary, since one of the primary symptoms of scurvy is pain when moving the joints.

Chandra and Newbern (1977: 38) remind us that "mild and subclinical deficits are not easy to pick up or quantitate but are likely to be functionally important," and in a population of seasonal hunter-gatherers, subclinical levels of nutrient deficiency are seasonal occurrences, which can weaken the immune response, raising the likelihood of contracting endemic chronic infections. The presence of nutritionally-linked anemia may have predisposed the younger individuals of the population to acquiring treponemal infection. Both incidence and prevalence of treponemal infection may have been highest during the last months of winter, when children crowded together for warmth and were nutritionally disadvantaged by dwindling provisions and may have contracted the infection more easily, and those with the disease may have relapsed under the same late winter conditions of deprivation.

The signs of pathology seen in the adults from the site are much the same as those in the children: nonvenereal treponematosis, healing porotic hyperostosis and cribra orbitalia, and slight bowing in some long bones, all of which potentially reflect conditions of childhood. The adult scapulae with possible signs of hemorrhage might represent adult individuals suffering from scurvy, but this would likely have been a seasonal hardship. The children in the archaeological record of Garbacon Creek might not have experienced any conditions of nutritional stress or infection that were out of the ordinary for this population; they may simply represent the part of the population born with a higher level of innate frailty (Wood et al. 1992), since it seems that many of the adults in the population survived similar conditions.

This study of the Garbacon Creek skeletal remains examines the context of treponemal infection in the Late Woodland population of this site. We can clearly see that nutrition and infection have an inescapable synergy, and that the presence of one disease condition can predispose an individual to other conditions or block the physical symptoms of a co-existing condition. Hopefully this work has contributed to our understanding of how such interactions may manifest themselves on the body, and how skeletal symptoms can reconstruct a picture of nutrition and activity, although the complex exchanges between human biology, environment, and culture form a web we have far to go to untangle.

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Appendix I

Adult Postcranial Inventory, 31Cr86*						
Label	**Element**	**Side**	**Portion**	**Completeness**	**Pathology**	**Notes**
CR86.126	clavicle	left	M, S	2	-	-
CR86.127	clavicle	left	M, S	1	-	-
CR86.128	clavicle	left	S, L	1	-	-
CR86.129	clavicle	left	L	3	-	-
CR86.130	clavicle	left	M, S	1	-	-
CR86.131	clavicle	left	S, L	1	-	-
CR86.132	clavicle	left	S,	2	active bone deposition - entire element	x-rayed
CR86.136	clavicle	left	S	2	healed fracture?	-
CR86.137	clavicle	left	S, L	1	-	-
CR86.119	clavicle	right	M, S, L	1	-	-
CR86.120	clavicle	right	M, S	1	-	-
CR86.121	clavicle	right	M, S	1	abscess? - inferior medial	medial epiphysis unfused; x-rayed
CR86.122	clavicle	right	S	2	-	-
CR86.123	clavicle	right	M, S, L	1	-	-
CR86.124	clavicle	right	S, L	2	-	-
CR86.125	clavicle	right	S, L	1	-	-
CR86.133	clavicle	right	M, S, L	1	-	-
CR86.134	clavicle	right	S	2	active and sclerotic bone deposition	x-rayed
CR86.135	clavicle	right	M, S, L	1	active bone deposition	-
CR86.50	humerus	left	P, S, D	1	-	-
CR86.51	humerus	left	P, S, D	1	-	-
CR86.52	humerus	left	S, D	1	-	-
CR86.53	humerus	left	P, S, D	1	-	-

Label	Element	Side	Portion	Completeness	Pathology	Notes
CR86.54	humerus	left	P, S, D	1	-	-
CR86.55	humerus	left	P, S, D	1	healed fracture?	x-rayed
CR86.56	humerus	left	P, S, D	1	-	-
CR86.57	humerus	left	S, D	1	-	-
CR86.58	humerus	left	D	3	slight active bone deposition - anterior and posterior distal	-
CR86.59	humerus	left	S, D	3	-	-
CR86.60	humerus	left	S, D	2	-	-
CR86.61	humerus	left	S, D	2	-	-
CR86.74	humerus	left	S, D	2	-	-
CR86.75	humerus	left	S, D	1	-	-
CR86.62	humerus	right	P, S, D	1	-	-
CR86.63	humerus	right	P, S, D	1	-	-
CR86.64	humerus	right	S, D	1	-	-
CR86.65	humerus	right	S, D	1	-	-
CR86.66	humerus	right	S, D	1	active bone deposition - anterior and posterior distal	x-rayed
CR86.67	humerus	right	S, D	1	-	-
CR86.68	humerus	right	P, S, D	1	-	-
CR86.69	humerus	right	P, S, D	1	sclerotic bone deposition - posterior distal	x-rayed
CR86.70	humerus	right	P, S, D	1	-	-
CR86.71	humerus	right	S, D	1	-	-
CR86.72	humerus	right	P, S, D	1	-	-
CR86.73	humerus	right	P, S, D	1	-	-
CR86.76	humerus	right	S, D	3	active bone deposition - anterior distal	-

Label	Element	Side	Portion	Completeness	Pathology	Notes
CR86.109	radius	left	S, D	1	-	-
CR86.110	radius	left	P, S	1	-	-
CR86.111	radius	left	S	1	-	-
CR86.112	radius	left	P, S, D	1	-	-
CR86.113	radius	left	P, S	2	-	-
CR86.114	radius	left	P, S, D	1	active bone deposition - distal metaphysis	-
CR86.115	radius	left	P, S, D	1	-	-
CR86.116	radius	left	P, S, D	1	-	-
CR86.117	radius	left	P, S, D	1	-	-
CR86.118	radius	left	S, D	2	-	-
CR86.102	radius	right	S, D	1	-	note: line of fusion vsible on distal epiphysis
CR86.103	radius	right	P, S	1	-	-
CR86.104	radius	right	P, S, D	1	-	-
CR86.105	radius	right	P, S, D	1	-	-
CR86.106	radius	right	S, D	1	-	-
CR86.107	radius	right	P, S	1	-	-
CR86.108	radius	right	P, S, D	1	-	-
CR86.77	ulna	left	P, S, D	1	-	-
CR86.78	ulna	left	P, S	1	-	-
CR86.79	ulna	left	S, D	1	-	-
CR86.80	ulna	left	P, S	2	active bone deposition - anterior, posterior, and medial proximal, anterior midshaft. Sclerotic bone deposition posterior midshaft	-

Label	Element	Side	Portion	Completeness	Pathology	Notes
CR86.81	ulna	left	P, S, D	1	combination sclerotic and active bone deposition - posterior and lateral, entire length of diaphysis	x-rayed
CR86.82	ulna	left	P, S	2	-	-
CR86.83	ulna	left	P, S, D	1	-	-
CR86.85	ulna	left	P, S	2	-	-
CR86.86	ulna	left	P, S	1	-	-
CR86.87	ulna	left	P, S	2	-	-
CR86.88	ulna	left	P, S	2	-	-
CR86.89	ulna	left	P, S, D	1	sclerotic bone deposition - medial proximal	-
CR86.84	ulna	right	P, S, D	1	-	-
CR86.90	ulna	right	P, S	1	-	-
CR86.91	ulna	right	P, S, D	1	-	-
CR86.92	ulna	right	P, S	1	-	-
CR86.93	ulna	right	P, S	2	-	-
CR86.94	ulna	right	P, S, D	1	lateral curvature?	-
CR86.95	ulna	right	P, S	2	-	-
CR86.96	ulna	right	P, S	1	lateral curvature? (s shape)	x-rayed
CR86.97	ulna	right	P, S	1	-	-
CR86.98	ulna	right	P, S	2	-	-
CR86.99	ulna	right	P, S	3	active bone deposition - anterior and medial proximal	-
CR86.100	ulna	right	P, S	1	-	-
CR86.101	ulna	right	P	3	-	-
CR86.195	scapula	right	glenoid, coracoid	1	-	-

Label	Element	Side	Portion	Completeness	Pathology	Notes
CR86.196	scapula	right	glenoid, axillary border	2	-	-
CR86.197	scapula	right	glenoid, coracoid, acromion	2	-	-
CR86.198	scapula	left	glenoid, axillary border	2	-	-
CR86.199	scapula	left	coracoid, acromion	1	active bone deposition on ventral body	-
CR86.200	scapula	left	glenoid, acromion	1	-	-
CR86.201	scapula	right	glenoid, acromion, coracoid	2	active bone deposition on ventral body, spine, acromion	-
CR86.202	scapula	right	glenoid	1	-	-
CR86.203	scapula	left	glenoid, coracoid	1	-	-
CR86.204	scapula	left	glenoid	1	osteophytes on glenoid rim, active bone deposition ventral	-
CR86.205	scapula	left	glenoid	1	osteophytes on glenoid rim	-
CR86.206	scapula	left	glenoid, coracoid	1	osteophytes on glenoid rim	-
CR86.207	scapula	left	glenoid, axillary border	1	-	-
CR86.208	scapula	left	acromion	1	-	-
CR86.209	scapula	left	glenoid, acromion	2	-	-
CR86.210	scapula	left	glenoid, acromion	2	-	-
CR86.211	scapula	right	glenoid, axillary border	2	-	-

Label	Element	Side	Portion	Completeness	Pathology	Notes
CR86.212	scapula	right	glenoid, acromion	1	-	-
CR86.213	scapula	right	glenoid, coracoid, acromion	2	-	-
CR86.214	scapula	right	acromion	1	-	-
CR86.215	scapula	right	acromion	2	-	-
CR86.216	scapula	left	glenoid, coracoid, acromion, axillary border	3	-	-
CR86.217	scapula	left	glenoid, acromion, axillary border	2	-	-
CR86.218	scapula	left	glenoid, axillary border	1	-	-
CR86.219	scapula	right	glenoid	1	osteophytes on glenoid rim	-
CR86.220	scapula	right	glenoid, acromion, axillary border	2	-	-
CR86.187	ribs	-	-	-	-	5 frags, none pathological
CR86.188	ribs	-	-	-	-	6 frags, none pathological
CR86.189	ribs	-	-	-	-	17 frags, 2 with active bone
CR86.190	ribs	-	-	-	-	37 frags, 1 with healed lesion
CR86.191	ribs	-	-	-	-	approx. 166 frags, none pathological

Label	Element	Side	Portion	Completeness	Pathology	Notes
CR86.192	ribs	-	-	-	-	68 frags, 2 with periosteal reaction
CR86.193	ribs	-	-	-	-	approx. 213 frags, 5 with periosteal reaction
CR86.194	ribs	-	-	-	-	approx. 180 frags, 2 with periosteal reaction
CR86.221	innominate	left	AS, SN (partial)	1	-	young adult
CR86.223	innominate	left	AS, , SN, acetabulum (partial)	2	-	female
CR86.224	innominate	left	AS, SN	1	-	male
CR86.228	innominate	left	AS (partial), SN (partial)	1	-	-
CR86.229	innominate	left	SN	1	active bone deposition - edge above ischial spine	-
CR86.230	innominate	left	AS, SN, acetabulum (partial)	2	-	Indeterminate
CR86.235	innominate	left	AS, SN, acetabulum, ilium, IS	3	-	male
CR86.236	innominate	left	AS (partial), SN, acetabulum	2	-	female, young adult
CR86.237	innominate	left	AS (partial), SN, acetabulum (partial)	2	-	male
CR86.238	innominate	left	AS (partial), SC, ilium, acetabulum, IT	3	-	male?

Label	Element	Side	Portion	Completeness	Pathology	Notes
CR86.240	innominate	left	AS, SN, acetabulum, ilium (partial), IT, pubic ramus	3	-	male
CR86.242	innominate	left	SN (partial), acetabulum, ilium (partial), IT (partial)	2	-	-
CR86.243	innominate	left	AS (partial), SN (partial), acetabulum (partial)	1	-	Indeterminate
CR86.222	innominate	right	SN (partial), acetabulum (partial)	1	-	-
CR86.225	innominate	right	AS, SN (partial)	2	active bone deposition	Indeterminate
CR86.226	innominate	right	AS, SN (partial), ilium (partial)	2	-	female?
CR86.227	innominate	right	AS, SN, ilium (partial), acetabulum, IT	3	-	male
CR86.231	innominate	right	acetabulum (partial), ilium (partial)	1	-	-
CR86.232	innominate	right	AS (partial), SN (partial), acetabulum (partial)	2	-	male

Label	Element	Side	Portion	Completeness	Pathology	Notes
CR86.233	innominate	right	AS (partial), SN (partial), acetabulum (partial)	2	-	Indeterminate
CR86.234	innominate	right	AS, SN	1	-	Indeterminate, Young adult
CR86.239	innominate	right	AS, SN, acetabulum, IT	3	-	female
CR86.241	innominate	right	AS, ilium (partial)	2	-	Indeterminate
CR86.247	innominate	right	AS, SN, IT, acetabulum	2	-	-
CR86.1	femur	right	P, S, D	1	-	-
CR86.2	femur	right	P, S, D	1	-	-
CR86.3	femur	right	P, S, D	1	-	-
CR86.4	femur	right	P, S	1	slight active bone deposition - entire length of diaphysis	-
CR86.5	femur	right	P, S	1	-	-
CR86.6	femur	right	P, S	1	striations - posterior, entire length of diaphysis. Active bone deposition - posterior, midshaft.	-
CR86.7	femur	right	P, S	1	-	-
CR86.8	femur	right	P, S	2	-	-
CR86.9	femur	right	P, S, D	1	anterior-posterior curvature	x-rayed
CR86.10	femur	right	P, S, D	1	osteophyte on head	-
CR86.11	femur	right	P, S, D	1	-	-

Label	Element	Side	Portion	Completeness	Pathology	Notes
CR86.12	femur	right	S, D	2	slight striations - entire length of diaphysis. Slight active bone deposition medial.	-
CR86.13	femur	right	S	1	-	-
CR86.14	femur	right	S	2	active bone deposition - entire length of diaphysis. Cortical thickening, medullary narrowing.	-
CR86.15	femur	left	P, S, D	1	-	-
CR86.16	femur	left	P, S, D	1	-	-
CR86.17	femur	left	P, S, D	1	anterior-posterior curvature,	x-rayed
CR86.18	femur	left	P, S	1	slight active bone deposition - entire length of diaphysis	-
CR86.19	femur	left	P, S, D	1	periostitis	-
CR86.20	femur	left	P, S, D	1	-	-
CR86.21	femur	left	P, S, D	1	slight anterior-posterior curvature	-
CR86.22	femur	left	S, D	1	-	-
CR86.23	femur	left	S	2	-	-
CR86.24	femur	left	P	3	-	-
CR86.25	femur	left	P	3	-	-
CR86.26	femur	left	P, S	2	-	-

Label	Element	Side	Portion	Completeness	Pathology	Notes
CR86.138	fibula	right	P, S, D	1	lytic foci - anterior proximal head, two disto-lateral. Active bone deposition - proximal head, entire length of diaphysis. Sclerotic bone deposition midshaft. Swelling - distal	x-rayed
CR86.139	fibula	left	S, D	1	-	-
CR86.140	fibula	right	S, D	1	-	-
CR86.141	fibula	indeterminate	S	2	active bone deposition - entire length of diaphysis.	-
CR86.142	fibula	right	S, D	1	-	-
CR86.143	fibula	indeterminate	S	2	-	-
CR86.144	fibula	left	S, D	1	-	-
CR86.145	fibula	right	S, D	1	-	-
CR86.146	fibula	indeterminate	S	2	-	-
CR86.147	fibula	indeterminate	S	2	-	-
CR86.148	fibula	right	S, D	1	active bone deposition - proximal diaphysis, lateral and medial; distal diaphysis, medial	-
CR86.149	fibula	right	P, S, D	1	-	-
CR86.150	fibula	indeterminate	S	2	swelling, active bone deposition, sclerotic bone deposition, radiating spicules - entire length of diaphysis	-
CR86.151	fibula	indeterminate	S	2	-	-

Label	Element	Side	Portion	Completeness	Pathology	Notes
CR86.152	fibula	indeterminate	S	1	-	-
CR86.153	fibula	indeterminate	P	3	-	-
CR86.154	fibula	indeterminate	P	3	-	-
CR86.155	fibula	indeterminate	S	2	-	-
CR86.156	fibula	indeterminate	S	2	-	-
CR86.27	tibia	left	P, S	1	slight striations - medial-posterior, entire length of diaphysis. Valgus curvature.	x-rayed
CR86.28	tibia	left	P, S, D	1	-	-
CR86.29	tibia	left	P, S, D	1	lytic foci proximal and distal, with localized swelling; striations along medial diaphysis; sclerotic bone distal posterior; active bone deposition in localized areas along entire diaphysis.	x-rayed
CR86.30	tibia	left	S	2	striations - medial, entire length of diaphysis; distal	subadult?
CR86.31	tibia	left	P, S, D	1	slight striations - medial, entire length of diaphysis. Sabering.	-
CR86.32	tibia	left	P, S, D	1	striations - lateral, proximal half of diaphysis (sclerotic); medial, entire length of diaphysis	-
CR86.33	tibia	left	S	2	striations - medial, entire length of diaphysis;	-

Label	Element	Side	Portion	Completeness	Pathology	Notes
CR86.34	tibia	left	P, S	1	slight striations - medial, entire length of diaphysis. Sabering.	x-rayed
CR86.35	tibia	left	P, S	1	-	-
CR86.36	tibia	left	S	2	slight striations - medial-posterior, entire length of diaphysis	-
CR86.37	tibia	right	S	2	-	-
CR86.38	tibia	left	S, D	3	striations and active bone deposition above medial malleolus	-
CR86.39	tibia	right	P, S	1	slight striations - medial-posterior, entire length of diaphysis.	-
CR86.40	tibia	right	S, D	2	slight striations - medial-posterior, entire length of diaphysis	x-rayed
CR86.41	tibia	right	P, S, D	1	-	-
CR86.42	tibia	right	P, S, D	1	striations - medial-posterior, entire length of diaphysis; distal	-
CR86.43	tibia	right	S, D	1	slight striations - medial, entire length of diaphysis. Sabering.	-
CR86.44	tibia	right	P, S, D	1	-	-

Label	Element	Side	Portion	Completeness	Pathology	Notes
CR86.45	tibia	right	P, S, D	1	multiple lytic foci along entire element, anterior and medial, various stages of healing; striations and active bone deposition along entire diaphysis; swelling distal with disorganized active bone	x-rayed
CR86.46	tibia	right	S	2	slight striations - medial, entire length of diaphysis.	-
CR86.47	tibia	right	P, S, D	1	-	-
CR86.48	tibia	right	D	3	striations and slight active bone deposition above medial malleolus	-
CR86.49	tibia	right	S	2	slight active bone deposition - anterior crest; medial-posterior	-
CR86.251	tibia	indeterminate	S	1	active bone deposition - entire length of diaphysis	-

*Elements not included: hyoids, sterna, hands, feet, vertebrae.

Portion Key: M=medial; S=shaft (diaphysis); L=lateral; P=proximal; D=distal; AS=auricular surface; SN=sciatic notch; IT=ischial tuberosity
Completeness Key: 1=more than 2/3 of element present; 2=more than 1/3-2/3 of element present; 3=less than 1/3 of element present

Appendix II

Adult Cranial Inventory, 31Cr86*								
	Sex Estimate					Pathology		
Label	**Mastoid Processes (R/L)**	**Nuchal Crest**	**Supra-orbital Margin**	**Supra-orbital Ridge**	**Sex Estimate**	**Porosity**	**Cranial Lesions**	**Notes**
#10	1/2	2	1	1	Female	R TMJ	osteoma (2-3 mm) posterior on L parietal;	parietal bossing; gouges along sagittal suture from scavenger activity
2236-11	3/3	1	3	1	Female	slight porosity along sagittal suture	periosteal apposition on external surface of L zygomatic	face almost intact
2236 #5	N/A	N/A	1	1	Female	slight porosity on frontal and along sagittal suture; slight cribra orbitalia	None observable	sutures almost obliterated
#6	2/ N/A	2	3	3	Female?	along sagittal suture	stellate lesion on R parietal? (right on fracture line - hard to tell); focal pitting on posterior parietals?	-

Label	Mastoid Processes (R/L)	Nuchal Crest	Supra-orbital Margin	Supra-orbital Ridge	Sex Estimate	Porosity	Cranial Lesions	Notes
#12	1/ N/A	1	N/A	N/A	Female?	None observable	osteoma (2-3 mm) posterior on L parietal	-
#8	5/5	3	5	3	Male	vascularity on supra-orbital ridges and posterior parietals	None observable	face almost intact
#3	N/A	1	5	5	Male	porosity/vascularity on supra-orbital ridge; porosity along sagittal suture; slight cribra orbitalia	raised porous area on central frontal and R parietal	-
#4	4/ N/A	1	5	4	Male	frontal and parietals	Pitting on parietals	bony ridge along left Masseter attachment; beautiful mat marks; signs of sinus infection
#18	2/ N/A	N/A	5	4	Male?		None observable	-
#15	5/5	2	N/A	N/A	Male?	parietals	osteoma (20 x 25 mm) posterior R parietal	Wormian bones, evidence of mechanical stress?

Label	Mastoid Processes (R/L)	Nuchal Crest	Supra-orbital Margin	Supra-orbital Ridge	Sex Estimate	Porosity	Cranial Lesions	Notes
#2	3/3	3	3	2	Indeterminate	cribra orbitalia	vascular lines on R frontal (?)	signs of sinus infection
#16	N/A	N/A	N/A	N/A	Indeterminate	porosity/ vascularity on pairetals/upper occipital	None observable	-
#14	2/ N/A	N/A	3	3	Indeterminate	porosity on supra-orbital ridges; R TMJ	2 areas of confluent clustered pitting on frontal	-
#9	3/3	3	3	3	Indeterminate	vascularity on supra-orbital ridges; diffuse porosity on frontal; porosity along sagittal suture	blunt trauma (13 x 7 mm) on L frontal;	mat marks on parietals
2236-13	3/2	1	3	1	Indeterminate	cribra orbitalia; slight porosity along sagittal suture	None observable	-
2236-10	3/2	2	3	1	Indeterminate	None observable	None observable	face almost intact
#17	N/A	N/A	N/A	N/A	Indeterminate	None observable	None observable	single parietal. Thin - may be subadult

*crania 7, 19, 20, 21, and 22 are fragmentary subadult crania. Cranium 1, and 11 could not be located.

Appendix III

Subadult Inventory, 31Cr86*

Pre-term Infant

Label	Element	Side	Portion Present	Age	Pathological Description	Notes
CR86s19	ulna	L	Prox. Meta./Diaph (1/2)	fetal-birth	periostitis along medial diaph.?	-
CR86s25	radius	Ind.	Diaph (<1/2)	fetal-birth	periostitis?	-
CR86s38	humerus	L	Diaph. (<1/2)/Dist. Meta.	fetal-birth	woven bone; periostitis?	-
CR86s43	scapula	R	complete	fetal-birth	-	-
CR86s57	ilium	R	acetabulum, partial body, AS	fetal-birth	-	-
CR86s58	ilium	L	complete	fetal-birth	-	-
CR86s66	pubis	R	complete	fetal-birth	-	-

Birth - 6 months

Label	Element	Side	Portion Present	Age	Pathological Description	Notes
CR86s37	humerus	L	Prox. Meta/Diaph. (<1/2)	birth-0.5	-	-
CR86s44	scapula	R	glenoid, axillary border	birth-0.5	-	-
CR86s54	ilium	L	SN, partial body	birth-0.5	-	-
CR86s55	ilium	L	acetabulum, SN	birth-0.5	-	-
CR86s60	ischium	L	>3/4	birth-0.5	-	-
CR86s65	pubis	L	<1/2	birth-0.5	-	-

Label	Element	Side	Portion Present	Age	Pathological Description	Notes

CR86s6	tibia	R	Prox. Meta./Diaph.	birth-0.5	periostitis along medial diaph.; cortical thickening and cloaking	x-rayed
CR86s7	tibia	Ind.	Diaph. (<1/2)	birth-0.5	cortical cloaking	x-rayed
CR86s15	femur	R	Diaph (<1/2)	birth-0.5	diffuse slight periostitis	x-rayed
CR86s17	femur	L?	partial Prox. Meta./Diaph	birth-0.5	cortical cloaking and thickening	x-rayed
6 months - 2 years						
Label	**Element**	**Side**	**Portion Present**	**Age**	**Pathological Description**	**Notes**
CR86s75	mandible	L	alveoli for C - DP4; only DP4 present	0.5-1.5	-	-
CR86s72	mandible	R/L	alveoli for R M1 - L DP4; only R M1, C present	1.5-3.5	-	-
CR86s26	radius	L	Dist. Meta.	0.5-2	woven bone along posterior diaph.	-
CR86s27	radius	L	Prox. Meta.	0.5-2	-	possible prox. To s26's dist.
CR86s36	humerus	R	Dist. Meta.	0.5-2	cortical cloaking	-
CR86s40	clavicle	R	Lateral	0.5-2	-	-
CR86s42	clavicle	L	Lateral, most of Prox. Portion	0.5-2	-	-
CR86s45	scapula	L	glenoid	0.5-2	-	-
CR86s46	scapula	R	glenoid, axillary border, acromion	0.5-2	-	-
CR86s52	ilium	R	acetabulum	0.5-2	slight periostosis bordering acetabulum	x-rayed
Label	**Element**	**Side**	**Portion Present**	**Age**	**Pathological Description**	**Notes**

CR86s53	ilium	L	acetabulum, partial body, AS	0.5-2	slight periostosis bordering acetabulum; complement to s52?	x-rayed
CR86s59	ischium	R	3/4	0.5-2	-	-
CR86s10	ulna	R	Prox. Meta./Diaph. (1/2)	0.5-2	-	-
CR86s8	tibia	L	Diaph. (1/2)	0.5-2	periostitis along medial diaph.; cortical thickening	x-rayed
CR86s13	femur	L	Diaph. (1/2)	0.5-2	woven bone along lateral diaph.	x-rayed
CR86s14	femur	L	partial Prox. Meta./Diaph	0.5-2	cortical cloaking?	x-rayed
CR86s16	femur	R	Diaph (3/4)	0.5-2	-	x-rayed
CR86s18	femur	R	Diaph.	0.5-2	-	x-rayed
CR86s20	femur	R	Prox. Meta./Diaph.	0.5-2	woven bone along lateral diaph.	complement w/s13?; x-rayed
CR86s68	femur	Ind.	Diaph. (dist. 1/2)	0.5-2	woven bone on dist. Diaph	possible match for s15; x-rayed
2-5 years						
Label	**Element**	**Side**	**Portion Present**	**Age**	**Pathological Description**	**Notes**
CR86s32	humerus	R	Diaph. (<1/2)/partial Dist. Meta.	2-5/5-10?	-	-
CR86s33	humerus	L	Dist. Meta.	2-5/5-10?	-	-
CR86s3	tibia	L	Diaph.	2-5	A-P curvature; slight periostitis medial diaph.	x-rayed
Label	**Element**	**Side**	**Portion Present**	**Age**	**Pathological Description**	**Notes**

CR86s9	tibia	Ind.	Diaph.	2-5	periostitis/woven bone along posterior diaph.; cortical cloaking	x-rayed
5-10 years						
Label	**Element**	**Side**	**Portion Present**	**Age**	**Pathological Description**	**Notes**
CR86s21	ulna	L	Prox. Meta/Diaph (<1/2)	5-10	-	-
CR86s22	ulna	R	Prox. Meta/Diaph (<1/2)	5-10	swelling at proximal end?; cortical cloaking	x-rayed
CR86s67	ulna	R	partial Prox. Meta/Diaph. (<1/2)	5-10	-	x-rayed
CR86s23	ulna	R	Prox. Meta./Diaph (3/4)	5-10	-	3 other ulna frags., age and side ind., possibly associated with s21-23
CR86s24	radius	R	Diaph./Dist. Meta.	5-10	-	-
CR86s28	radius	L	Prox. Meta/Diaph (1/2)	5-10	-	7 other radius frags., age and side ind., possibly associated w/s26-28; 1 w/ woven bone, 1 w/possible lytic lesion
CR86s29	humerus	R	Diaph.	5-10	-	-
CR86s30	humerus	L	Diaph. (<1/2)	5-10	-	complement w/s29?
Label	**Element**	**Side**	**Portion Present**	**Age**	**Pathological Description**	**Notes**

CR86s31	humerus	L	Prox. Meta/Diaph./partial Dist. Meta.	5-10	-	-
CR86s34	humerus	R	Diaph./partial Dist. Meta.	5-10	-	-
CR86s2	tibia	R	Diaph./Dist. Meta.	5-10	-	7.5-8.5; x-rayed
CR86s4	tibia	R	Diaph.	5-10?	slight porosity medial diaph.	x-rayed
CR86s1	tibia	L	Prox. Meta./Diaph.	5-10	-	7.5-8.5; x-rayed
CR86s11	femur	R	Prox. Meta./Diaph./Dist. Meta.	5-10	-	7.5-8.5; x-rayed
CR86s12	femur	L	Prox. Meta./Diaph./Dist. Meta.	5-10	-	complement w/s11?; x-rayed

*fragmentary epiphyses of adolescents not included

Appendix IV

Glossary of osteological terms

Cloaca: A smooth-walled drainage hole formed in osteomyelitic bone in order to provide an outlet for pus and necrotic byproducts.

Cortex: the solid, dense external portion of bone, comprised of compact, or cortical, bone.

Edema: the localized or generalized accumulation of excessive fluid in tissues or body cavities.

Endosteum: an ill-defined and largely cellular membrane lining the inner surface of bones which contains bone-forming cells.

Gummatous: refers in this case to bone lesions which are associated with skin manifestations which are literally 'gummy.'

Involucrum: a sheath of new bone which forms to enclose an infected, necrotic portion of bone.

Osteochondritis: inflammation of bone and cartilage which results in the production of small, focal epiphyseal areas of necrosis on the convex sufaces of diarthrodial joints.

Osteomyelitis: Infection or inflammation of bone involving the medullary cavity.

Acute osteomyelitis: Usually caused by pyogenic bacteria, this acute infection results rapidly in the formation of a sequestrum, involucrum, and potentially multiple cloacae.

Chronic osteomyelitis: the result either of a sequestrum that acts as a foreign body, perpetuating infection, or the persistence of a low grade infection in the bone and adjacent soft tissuesl It can recur as a series of acute episodes and remissions over a period of months or years.

Periosteum: a tough, vascularized membrane which covers the outer surface of bones during life, nourishing them and anchoring them to tendons.

Periostitis: formation of new bone on the cortex as a result of inflammation of the periosteum, which can be caused by a wide range of stimuli, including infection and trauma.

Pyogenic: pus-producing.

Sequestrum: an area of necrotic bone surrounded by living bone.

Spongiosa: cancellous or spongy bone tissue.

Suppurative: inducing the formation of pus.

Trabeculae: thin bony spicules that form trabecular, or cancellous, bone tissue, which has a spongy honeycomb structure and is found in the ends of long bones, in vertebral bodies, and in short and flat bones.