Metabolic Disease in Subadult Skeletal Remains from Late Ottoman-Era Tell Hisban, Jordan

by

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The site of Tell Hisban in Jordan was seasonally occupied by nomadic agropastoral tribes for over a thousand years. In the latter half of the 1800s, the Ottoman Empire instituted the Tanzimat, a series of reforms intended to solidify control over the region, including a new system of private land ownership. This new land law conflicted with traditional tribal-based land rights and resulted in intensification of agricultural production and diminished pastoralism in the regional economy. During this period of economic change, at least 62 individuals were interred in ruins on Tell Hisban, of which 55% were non-adults. Many long bones and cranial elements of non-adults within these commingled remains display evidence of vitamin C (scurvy) and D (rickets) deficiencies at a greater frequency than pre-Tanzimat or earlier regional cemeteries. Increased agricultural production may have impacted the availability of traditional foods high in ascorbic acid that prevented scurvy in past groups, and increased reliance on cereals, which lack key macronutrients. The resulting shift in diet would have disproportionately affected individuals more susceptible to nutritional stressors, such as pregnant women, infants, and weaning children. In the case of rickets, these nutritional stressors may have been exacerbated by cultural barriers

which limited an individual's exposure to sunlight and may have resulted in the surprising presence of rickets in this high-ambient UV radiation environment. Together with genetic predispositions to scurvy or rickets, these biocultural changes likely contributed to increased frailty in the form of metabolic disease for infants and young children within this population compared to earlier groups at Tell Hisban and contemporary populations in other areas of Jordan and Israel.

Metabolic Disease in Subadult Skeletal Remains from Late Ottoman-Era Tell Hisban, Jordan

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CHAPTER ONE

Introduction

Tell Hisban is a site in modern Jordan that has been inhabited for approximately 3000 years, albeit not continuously. During this time, numerous foreign powers held control over Tell Hisban, but this study focused only on the Late Ottoman (1808-1918 AD) period, which marks substantial land use changes in the region with the introduction of the *Tanzimat* reforms and adoption of large-scale agriculture of grains, particularly wheat. Although some semi-nomadic tribes were able to come together to purchase land in the name of some of their more influential members under this new system of land ownership, the reforms left others landless.

During this period, at least 64 bedouin individuals were interred in the ruins of an earlier building. Of these commingled remains, at least 29 individuals were under the age of 15 years, and preliminary analysis revealed skeletal indicators of the metabolic diseases, scurvy (vitamin C deficiency) and rickets (vitamin D deficiency). Contemporary skeletal assemblages from nearby sites in Jordan and Israel do not exhibit similar evidence of metabolic disease, and thus Tell Hesban offers a unique insight into the occurrence of scurvy and rickets in this region during the Late Ottoman period (Eakins 1993; Mitchell 2006; Rose and Kwaleh 2012; Smith and Horowitz 2009). The purpose of this study is to investigate the extent of these two diseases in the juveniles at Tell Hisban and explore the cultural factors which may have contributed to the prevalence of scurvy and rickets in the population.

The study of human skeletal remains provides unique insight into the health of past populations. Skeletal analyses can offer individual profiles in health, particularly for populations who may have been overlooked in historical records or lived before historical documentation. Commingled skeletal remains provide limited insight into individual health, but may illuminate

conditions affecting the population as a whole. By calculating the frequencies of bones exhibiting key, diagnostic pathologies, anthropologists may understand the morbidity of the population, or how many individuals were suffering from a particular disease. Additionally, age estimations from dental eruption or long bone measurements, may shed light on the overall mortality of the population, and highlight age ranges when individuals are particularly at risk of illness or even death.

In the commingled skeletal remains from Tell Hisban, the presence of lesions associated with metabolic diseases such as scurvy or rickets were assessed to understand population health. Scurvy results from a dietary deficiency in vitamin C. Rickets typically results from a deficiency in vitamin D, which is synthesized in the skin cells with adequate exposure to UV rays from the sun or obtained through diet, but can result from insufficient dietary calcium or genetic diseases as well. Both vitamins C and D are crucial to proper skeletal development and maintenance, and individuals who are deficient in these nutrients typically exhibit skeletal lesions indicative of the diseases. These lesions are most apparent in the bones of subadults, as the bony changes associated with normal growth are impaired during periods of disease. Because the mechanisms of both scurvy and rickets are well understood, and symptoms persist in the skeleton, these metabolic diseases were ideal for gaining insight into juvenile mortality rates and survivorship in the Ottoman-era population at Tell Hisban.

CHAPTER 2

Background

Tell Hisban is a site in modern-day Jordan located approximately 22km southwest of Amman. The site is in the fertile Madaba plain that is characterized by an open plateau crossed with wadis, or valleys, carrying runoff from seasonal rains and sometimes containing springs for year-round water access. The ruins of the ancient city occupy one of the highest tells in the area, and in the 19th century these earlier architectural remains we reused as storage spaces and animal pens, and as a cemetery for semi-nomadic pastoralists in the area.

Although the site has been excavated extensively since the late 1960s, graves from only two periods have been uncovered. Excavations at Hisban in 1971 through 1976 revealed 29 rock-cut chamber tombs dating between the Early Roman and Late Byzantine periods (63 BC-491AD) cut into exposed bedrock below the ancient city and in nearby hills (Waterhouse 1998). The skeletal remains from these tombs (MNI=191) were studied by Grauer and Armelagos (1998). Later excavations from 1998 through 2012 revealed a collection of commingled remains (MNI=62) dating to the Late Ottoman (1808-1918) period based on material cultural evidence (Walker 2001). During a preliminary analysis of these remains, possible indicators of metabolic disease were observed in the juvenile skeletons, but these were not the primary focus of that study and thus not explored in detail (Perry and Edwards 2018).

In 1516 A.D., Hisban came under Ottoman rule, shifting the city's role from a political and economic center under the Mamluks to an imperial hinterland, the Belqa'. The area remained largely agro-pastoral, with land use established by agreements between regional semi-nomadic tribes. However, beginning in 1858, the Ottoman caliphate instituted the *Tanzimat*, a system of

institutional changes meant to modernize and reconsolidate control of the empire, particularly in marginal areas containing predominantly nomadic populations like the Balqa', and increase the lucrative production of grain for global trade. Under the *Tanzimat*, the Ottomans adopted a system of land ownership, which made individuals easier to tax (and be conscripted into the military), but also disenfranchised poorer tribal members who could not afford land (Abujaber 1989).

Historical travelers report that two tribes occupied the lands surrounding Tell Hesban at the start of the 19th century (Russell 1989). The smaller of the two, the 'Ajarma, was already practicing small scale cultivation of some lands at this time (Abujaber 1989). The larger tribe, the 'Adwan, once exerted considerable influence over the Belqa' but by 1810 their influence had been surpassed by the Beni Sakhr, who traditionally occupied lands further north (Rogan 1999). In 1867, the Ottomans launched a military campaign to subdue the bedouin, who were resistant to paying taxes to the Ottoman government and were themselves collecting protection taxes (*khuwa)* from the people living in the area (Rogan 1999). The 'Adwan, allied with the Beni Sakhr, resisted this incursion of Ottoman authority, but were ultimately unsuccessful and the 'Adwan shaykh, 'Ali Dhiyab, was imprisoned (Rogan 1999). In 1869, the 'Adwan and Beni Sakhr made one more attempt to reassert their authority and collect *khuwa* but Ottoman forces maintained control over the area (Rogan 1999). Based on this history, the Hisban sample theoretically could belong to any of these three tribes, discussed in greater detail below.

Beni Sakhr

The Beni Sakhr were traditionally primarily camel herders but kept herds of sheep and goat as well (Lewis 1987). Following the institution of the new Ottoman land laws in the latter half of the 19th century, the Beni Sakhr were wealthy enough to purchase large tracts of land for

both collective use by the tribe and for personal use by the tribe's wealthiest members (Lewis 1987). Some of this land was devoted to large scale agriculture, and the Beni Sakhr often hired workers from weaker tribes to cultivate the lands or rented the land out to merchants from the nearby city, Salt (Lewis 1987). The Beni Sakhr used other portions of their land holdings to continue their pastoralist lifestyle, maintaining sizable herds of camels, sheep, and goats into the early 1930s (Lewis 1987; Rogan and Tell 1994).

‘Adwan

As the Beni Sakhr gained prominence in al-Salt and the surrounding regions towards the end of the 18th century, the ‘Adwan, the people of the city of al-Salt, and another tribe, the ‘Abad, worried about losing power in the region, are said to have divided the Balqa’ amongst themselves (Abujaber 1989: 70). In this agreement, Hisban fell under the influence of the ‘Adwan, who had been the most powerful tribe in the area before the encroachment of the Beni Sakhr (Abujaber 1989). Following their losses to the Ottoman armies, the ‘Adwan obtained more official ownership of some of the lands surrounding Hisban, mostly in the form of small individual plots (Rogan 1999). Like the Beni Sakhr, the ‘Adwan largely did not cultivate their own land but relied on *fellahin*, or agricultural laborers, to maintain the wheat and barley crops (LaBianca 1990). The ‘Adwan kept herds of sheep and goat during this time, but likely in smaller numbers than their pre-*Tanzimat* herd sizes (LaBianca 1990).

‘Ajarma

Of the three tribes living in and around Tell Hisban during the Late Ottoman period, the ‘Ajarma (‘Ajarmeh) were the smallest. The ‘Ajarma adopted seasonal agriculture earlier than the Beni Sakhr and ‘Adwan, and were cultivating barley and wheat by the late 18th century

(Abujaber 1989). Typically, the ‘Ajarma herded sheep, goats, and cattle year-round and cultivated wheat only during the winter months (LaBianca 1990). Following the land ownership laws, records indicate that the wealthier tribe members collected taxes for large wheat plantations, although these plantations were not necessarily owned by the ‘Ajarma, and may have been managed by them instead (Abujaber 1989). The fields were typically cultivated by *fellahin* however the plantations provided the food and clothing for a number of bedouin who did not necessarily work on the plantations (Abujaber 1989). These individuals still maintained herds of sheep and goats for additional income (Abujaber 1989).

The tribal identity and status of individuals buried at Hisban during this period is not known, but the lack of permanent settlement in the environs of the site suggest they were semi-nomadic, perhaps working as agricultural laborers on a seasonal basis or pastoral nomadic tribes who increasingly were losing access to their traditional lands (Abujaber 1989). The governmental interference, land ownership, and subsistence patterns during the Late Ottoman period could have impacted the morbidity and mortality patterns of local communities in the Madaba Plain. To understand the physiological consequences of these political and economic shifts, this study focused on two indicators of nutritional and physiological well-being that manifest in human skeletal remains, deficiencies in vitamin C (scurvy) and vitamin D (rickets) in subadults (i.e. individuals under 15 years of age).

Scurvy

Metabolic diseases are any disease or disorder which affects the body’s ability to metabolize nutrients. This includes deficiencies in ‘vital amines’ or vitamins (McCollum et al. 1922: 298). Scurvy is the result of a deficiency in vitamin C, or ascorbic acid. Humans are one of the few mammals incapable of creating their own vitamin C, and thus are completely reliant

on dietary sources of the nutrients, but exact daily requirements vary depending on factors such as age and sex (Table 1). The quantity of vitamin C between plant species varies dramatically, but in general green leafy vegetables and citrus fruits are rich in the nutrient. However, the ascorbic acid they contain can be reduced by processing (which releases ascorbic acid oxidase from vesicles in the cells) and cooking (which catalyzes the destruction of ascorbic acid by phenolase) (Davies et al. 1991).

Table 1: Daily Vitamin C Requirements

Infants	40-50mg/day
Children (1-3 years)	15mg/day
Pregnant women	85mg/day
Breastfeeding women	120mg/day

(Popovich et al 2009)

Recent studies suggest that vitamin C deficiency may be partially influenced by genetic factors. The human plasma protein haptoglobin (Hp) is a polymorphic trait with three main phenotypes (Hp1-1, Hp2-1, and Hp2-2) (Delanghe et al. 2007). When blood cells rupture, Hp binds with the resultant free hemoglobin to prevent oxidative damage to surrounding cells (Carter & Worwood 2007). Hp2 is less efficient at binding to these free radicals than Hp1, and thus Hp2-2 individuals must contribute a larger portion of their vitamin C stores to binding to these free radicals in the place of Hp2 (Delanghe et al. 2007). The frequencies of the codominant Hp^1 and Hp^2 alleles vary among populations across the world, but fixation of a single allele has not yet been found in any population studied (Carter & Worwood 2007).

Vitamin C is a key component in the synthesis of collagen, a connective fiber found in every tissue, including bone. Inadequate levels of vitamin C result in compromised blood vessels

that rupture easily, and hemorrhaging, which is apparent on the skin as 'spots.' Motion at joints exacerbates the degradation, resulting in swelling, which can make additional movement painful. The gums can also swell and even ulcerate, and the teeth may fall out as the periodontal ligaments deteriorate (Brickley and Ives 2008).

Changes in soft tissue due to vitamin C deficiency can cause reactions in the skeleton. As more blood vessels break down, less blood is delivered to the body. This can cause sufferers of scurvy to develop iron-deficiency anemia as well. To compensate for this, the body manufactures more vascular tissue to replace the compromised blood vessels, particularly trabecular bone (Klaus 2017). The expanded trabecular bone causes existing cortical bone to be resorbed, resulting in the bones having a porous appearance. The craniofacial region in children is particularly susceptible to such remodeling in response to vitamin C deficiency, such as the bones forming the eye orbits (frontal, zygomas, and maxilla) and those containing teeth (maxilla and mandible) (Ortner & Ericksen 1997). Associated porosity has also been observed in the sphenoid (Ortner 2003) and basilar portion of the occipital (Moore and Koon 2017). This porosity can also be observed in bones serving as attachment sites for major muscle groups, such as the scapula (Brickley and Ives 2006) and pelvis (Ortner 2003), which are involved in a majority of movement and more likely to be damaged.

In addition to tissue degradation, the bone itself can be affected by the compromised collagen resulting from vitamin C deficiency. In both juveniles and adults, bone continually remodels throughout life, but in individuals with vitamin C deficiency, the new bone will be structurally compromised due to lack of functional collagen. As a result, this weaker bone can develop incomplete 'pseudofractures' affecting layers of the cortical bone. Ribs are especially prone to such damage, particularly near the costochondral joints. Damage to ribs can even result

in steeper angles and a ‘depressed’ appearance of the chest at the sternum (Brickley and Ives 2008).

Juveniles are particularly susceptible to the effects of dysfunctional collagen as new bone is replacing cartilage cells rather than old bone cells. In juveniles suffering from vitamin C deficiency, this new bone growth is slowed considerably. When new bone is mineralized along the metaphyseal ends of the long bones, it is structurally weakened and more prone to microfractures. These microfractures may cause the metaphyses to appear slightly ‘flared’ after vitamin C is ingested and healing begins (Tamura et al. 2000).

Skeletal evidence of scurvy has been found in a diversity of environments across the world and in different time periods. In a Neolithic Lapita site on the island of Vanautu in the South Pacific, porosity and abnormal subperiosteal bone was observed in the long bones, clavicle, scapula, pelvis, ribs, and cranial bones (such as the sphenoid) of 15 subadults, of whom only two were over the age of 6 months. These lesions were attributed to scurvy, possibly resulting from maternal malnutrition or seasonal food insecurities (Buckley et al. 2014). A study of 25 individuals under the age of 4 years from a prehistoric site in Tonga found similar abnormal porosity and subperiosteal new bone on the long bones. In this instance, porosity was not observed on the sphenoid, orbits, or maxilla, and thus scurvy was suggested as one possible etiology for the lesions (Buckley 2000). In Phnom Knang, Cambodia, porosity in the sphenoid and palate coupled with ‘vascular impressions’ on the endocranial surface in a skull of an individual approximately 6 years in age were attributed to scurvy as well (Halcrow et al. 2014).

While compiling his list of diagnostic criteria for scurvy, Donald Ortner and colleagues analyzed 363 subadult skulls from various sites across Peru which had since been stored at the Smithsonian Museum of Natural History. Ortner et al concluded that 10% of the individuals in

the collection suffered from scurvy based on abnormal porosity of the cortex and irregular new bone growth, particularly on the greater wing of the sphenoid (Ortner, Kimmerle, and Diez 1999). More recently, Klaus analyzed the skeletal remains of 641 individuals from the Lambayeque Valley in northern Peru and found only five individuals with porosity and abnormal bone growth in the sphenoid, orbits, maxilla, zygomatics, and mandible which could be attributed to scurvy (Klaus 2014). Scurvy has also been reported in skeletal remains from a site (3600-3200BP) in the Atacama desert in northern Chile. Of the 12 subadults uncovered, all four perinates exhibited multiple cranial lesions consistent with scurvy, a possible result of the agricultural transition (Snoddy et al. 2017).

In a comparative analysis of individuals from the desert of the American southwest, juveniles were said to have scurvy if porosity was observed on the sphenoid and in two other cranial bones that are diagnostic for scurvy. This study found that scurvy was most prevalent before 1200CE and after 1500CE, and predominantly affected the more rural areas rather than the political centers which presumably had access to a greater diversity of foodstuffs (Crandall 2014). In the historic United States, porosity was observed in the maxillae, sphenoids, and orbits of at least nine subadults in a commingled skeletal assemblage from an historic African-American church in Philadelphia. Scurvy was proposed as the most likely cause of the lesions, but other diseases could not be ruled out (Ellis 2016).

Some studies have identified scurvy in skeletal remains from Europe as well. In a Middle Byzantine Greek skeletal collection, four subadults were observed to have porosity and abnormal new bone growth on aspects of the cranium and ilium which were interpreted as scurvy (Bourbou 2014). However, a comparative analysis study of scurvy in Europe found generally very few reports of scurvy in Greece and Italy. This was attributed to the relatively higher

availability of fresh vegetables compared to countries in Western and Northern Europe (Mays 2014). Scurvy was considerably more frequent in northern European countries and was observed in skeletal remains from a winter hunting post in Russia, as well as from Dutch and British sailors (Mays 2014). Unfortunately, in these three cases, diagnosis of scurvy occurred before the standardization of methods, and thus these studies are not necessarily reliable evidence (Mays 2014). Scurvy has also been proposed as an etiology for the porous cranial elements observed in the commingled skeletal remains of 950 individuals from a 19th century workhouse in Kilkeny City, Ireland (Geber and Murphy 2012). The authors considered definitive examples of scurvy to be porosity in the greater wing of the sphenoid, the coronoid process of the mandible, and the posterior portion of the maxilla, while porosity on the frontal bone, scapula, femur, tibia, and lesser wing of the sphenoid were considered indicative of scurvy (Geber and Murphy 2012). Using this method, they estimated that approximately 22% of subadults in the population were affected with scurvy, likely caused by the sudden shortage of potatoes that are rich in vitamin C (Geber and Murphy 2012).

Paleopathological reports of scurvy in the Middle East are relatively scarce. In a Predynastic site from Egypt (c.3800-3600BCE), 11 individuals were excavated from a cemetery. In one individual, an infant aged approximately 1 year, porosity was observed in the sphenoid, maxilla, orbits, and zygomas, and interpreted as scurvy (Pitre et al. 2016). In a Bronze Age tomb in the United Arab Emirates, periosteal reactions that could have resulted from scurvy were observed in 41.4% of subadult right femora and also inflamed linea aspera in 24.5% of individuals but these were interpreted as likely resulting from malaria or a bacterial infection. However, only the long bones were analyzed, which are less diagnostic than the cranial elements (Baustian 2010).

Several skeletal assemblages dating to the Middle to Late Islamic period have been excavated in both Jordan and Israel. Analysis of these individuals showed indicators of diseases such as osteomyelitis, as well as nonspecific stress indicators such as linear enamel hypoplasias, but possible scorbutic lesions are mentioned infrequently (Eakins 1993; Mitchell 2006; Smith and Zegerson 1999). In a collection from Dor, on Israel's coastal plain, 84% of juveniles exhibited cribra orbitalia and scurvy was suggested as a possible etiology but could not be confirmed for want of more diagnostic skeletal indicators (Smith and Horowitz 2009). In a small (n=16) 12th century possible migrant population buried in Al-Wu'ayra in southern Jordan, porosity was observed in 54% of sphenoids, 64% of maxillae, and 45% of mandibles (Rose and Khwaleh 2012). A deficiency in vitamin C or folic acid was proposed as the possible cause of these pathologies, but this skeletal assemblage likely represents individuals of European rather than Near Eastern origin (Rose and Khwaleh 2012). Another Middle-Islamic site near Tel Jezreel contained 21 infants, of whom 14% had intracranial bone formation possibly linked to scurvy (Mitchell 2006). However, scurvy is not generally linked to bone formation on the internal surface of the occipital (Brickley and Ives 2008) and is not a likely candidate here. The Late Ottoman era bedouin skeletal assemblages from Tell el-Hesse, on Israel's coastal plain, and Khirbet al-Mudayna, just south of Hisban, did not display skeletal indicators of scurvy (Eakins 1993; Sadvari 2009). In addition, while cranial "pitting and thickening" was found in a Middle to Late Islamic cemetery at Caesarea (Smith and Zegerson 1999), and "osteoporosis/pitting" in the crania of children at Tel Harif and Tel Sheva (Goldstein et al. 1976), these were not specifically attributed to scurvy.

Rickets

The second metabolic disease studied in this population is vitamin D deficiency, commonly called rickets in juveniles or osteomalacia in adults. Primarily, vitamin D is

synthesized after exposure to UV-B rays but may obtained in small amounts from diet as well (Lockau and Atkinson 2017). Several environmental barriers, such as reduced daylight during winter in higher latitudes or particulate matter in the air creating smoke or smog can limit an individual's access to sunlight. Biological barriers, such as increased melanin content in the skin, and cultural barriers such as clothing or limited outdoor activities, can limit this exposure as well. In these instances, humans require supplements to maintain healthy vitamin D levels. Compared to vitamin C, the dietary availability of vitamin D is limited, as the nutrient exists in relatively small quantities in fish, liver, and eggs (Mattila et al. 1995). However, vitamin D compounds are less likely to break down during cooking and processing (Jakobsen & Knuthsen 2014). In addition, congenital anomalies can affect the body's ability to synthesize vitamin D or properly allocate minerals to the skeleton, resulting in similar effects on the body as rickets caused by vitamin D deficiency (Brickley and Ives 2008). The primary role of vitamin D in the body is to metabolize calcium. Calcium is typically found primarily in the skeleton, and to a lesser extent in the bloodstream. When vitamin D levels are low, as in vitamin D deficient rickets, the body scavenges calcium from the skeleton to maintain blood calcium levels. This allocation of calcium from bones to the rest of the body compromises the structural integrity of the bone, which is then prone to fracturing.

Nutritional rickets occurs when individuals have adequate levels of vitamin D but still express symptoms of rickets or osteomalacia, typically due to insufficient intake of dietary calcium combined with an unusually high intake of phytic acid. In individuals with low serum calcium levels, the body triggers an increase in parathyroid hormone, which in turn may cause Secondary Hyperparathyroidism (Jamal & Miller 2013). Parathyroid hormone converts existing vitamin D in the body into a less accessible form [$1,25(OH)_2$ D] while the more accessible form

[25(OH)D] degrades more quickly, resulting in vitamin D deficiency and eventually rickets or osteomalacia in severe cases (Clements 1989; Thacher et al 2010).

Hereditary rickets due to a genetic mutation is characterized by a deficiency in phosphate (PO_4), rather than vitamin D. The primary mineral in bones and teeth is hydroxyapatite ($Ca_{10}(PO_4)_6(OH)_2$) (Lin and Chang 2015). In vitamin D deficiency rickets, the individual is unable to metabolize calcium (Ca) to mineralize new bone with hydroxyapatite. Conversely, individuals with hypophosphatemic rickets are unable to absorb sufficient levels of phosphate from the gut or kidneys, and this insufficiency results in similarly unmineralized bones. The most common of these diseases is X-linked dominant Hypophosphatemia (XLH) which affects 1 in 20,000 individuals and comprises 80% of all heritable types of rickets (Pavone et al. 2014). Affected individuals are born with normal levels of phosphate, provided the mother also had normal levels during fetal development. However, because the infant is unable to absorb additional phosphate, their osteoblasts are likewise unable to form hydroxyapatite and mineralize new bone (Bitzan & Goodyer 2019).

Among the remaining 20% of heritable rickets cases is Hereditary Hypophosphatemic Rickets with Hypercalciuria (HHRH), which is notable for this study as it was first observed among a "Bedouin kindred" near Tel Aviv (Tieder 1985). HHRH is an autosomal recessive trait that typically occurs very rarely in the general population but has been observed in higher frequencies among populations with inbreeding, such as in offspring from marriage between first cousins (Tieder et al. 1987; Lorenz-Depiereux et al. 2006). Like those with XLH, individuals with HHRH are unable to absorb phosphate. However, these individuals also have unusually high levels of calcitriol which is responsible for transporting calcium from the gut and bones to the blood. As the body scavenges phosphate from the hydroxyapatite in the bones, the excess

calcium is taken into the individual's blood and results in hypercalciuria and demineralized bones (Bitzan & Goodyer 2019).

Genetic predispositions have been linked to nutritional rickets, though the mechanism is not yet well understood. A series of genes code for vitamin D receptors, but only polymorphisms at the *Fok1* locus have been extensively studied (Fischer et al 2000; Lu et al 2003; Wu et al 2006). Vitamin D receptors are responsible for delivering calcium to bone for mineralization. The dominant F allele has been linked to a mutation in the start codon so that the vitamin D receptor protein is never produced. In populations in Nigeria and China, higher frequencies of this FF genotype have been observed among rachitic individuals than among the non-rachitic population (Fischer et al 2000; Lu et al 2003; Wu et al 2006). However, it is important to note that the FF genotype was observed in 18-22% of nonrachitic individuals from each of these populations, leading one author to theorize that low levels of dietary calcium contribute to rickets in FF individuals (Fischer et al 2009). This theory is supported by a comparison of serum calcium levels and the F allele in rachitic individuals from Egypt and Turkey. Individuals from both countries had lower than normal serum calcium levels, but the Egyptian individuals had much lower levels than the Turkish individuals. However, instances of rickets were comparable between both populations, which the author attributed to the higher frequency of the F allele among Turkish individuals (Baroncelli et al 2008).

Regardless of cause, the loss of calcium from the bones of rachitic individuals can leave lasting skeletal evidence of Vitamin D deficiency. As with vitamin C deficiency, juveniles are especially susceptible to the effects of vitamin D deficiency. Typically, cartilaginous plates develop in juveniles at the end of the metaphyses on long bones. In normal growth, calcium is deposited in these cartilage cells (chondrocytes), and this mineralization results in their

apoptosis, or programmed cell death (Brickley and Ives 2008). However, when the individual is vitamin D deficient, calcium is not deposited into the chondrocytes, which then continue to grow and multiply. Because cartilage is more malleable than bone, these chondrocytes spread out of their typical tight arrangements as pressure is put on the bone, especially in long bones like the tibia and radius, which bear most of the body's weight during walking or crawling (Brickley and Ives 2008). Once vitamin D is ingested, these irregularly arranged chondrocytes are mineralized as usually occurs in normal development, but here resulting in tibias and radii with distal ends which appear 'flared' or 'cupped' (Brickley and Ives 2008; Ortner and Mays 1998). Long bones may also appear thicker in cases of healing rickets, as layers of unmineralized osteons accumulated on the surface of the diaphyses during deficiency become mineralized. In the cranium, the sudden mineralization of these cells can result in "spiculated" bone formation on the cortical bone of the cranial vault (Brickley et al. 2018; Brickley and Ives 2008).

Both adults and juveniles are subject to having calcium stripped from their bones during vitamin D deficiency. This is seen as porosity in the long bones, vertebrae, and cranium (Brickley and Ives 2008). Furthermore, the structure of these bones is compromised, leaving the bones more susceptible to deformations. This is most apparent in the tibiae, which tend to bow under the pressure of the juvenile's body weight (Brickley and Ives 2008). Newly porous vertebrae may also compress under body weight, resulting in abnormal curvature of the spine (Brickley and Ives 2008). In juveniles, these deformities are also visible in the ribs, which, as in vitamin C deficiency, swell at the costochondral joints, producing the 'rachitic rosary' (Brickley and Ives 2008). Further remodeling of the rib angles can cause the sternum to protrude more than normal, so that the individual appears 'pigeon chested' (Brickley and Ives 2008; Ortner and

Mays 1998). Additionally, the pelvis may be affected as the acetabulum is irregularly angled forward (Brickley and Ives 1998).

There is not a clear (timeline) for the progression of rickets as the disease severity may be affected by a myriad of factors, including the cause of rickets, diet, and exposure to sunlight. However, the healing process is better understood. After reintroduction of vitamin D or calcium, many of the aforementioned skeletal changes associated with rickets are reversed. As the body continues to remodel bones through growth and development and into adulthood, childhood indicators of rickets, save for the most severe deformities, may no longer be evident in the skeleton upon reaching adulthood. The rate at which these indicators are obliterated from the skeleton is dependent upon the remodeling rates of different bones. For example, the adult femur has a bone turnover rate of between 10-20 years, so any changes made to the skeleton during childhood would still be visible for a time into adulthood (Brickley et al. 2018).

Teeth are not immune to the effects of vitamin D deficiency. In juveniles, it can cause delayed eruption of the deciduous teeth, and increased prevalence of cavities. Most remarkably, because teeth do not remodel during life, they contain a 'history' of the individual's health. Although non-specific stress indicators like linear enamel hypoplasias have notably been used to assess individual health in the past, the unique role of vitamin D in bone mineralization makes a deficiency of this nutrient much more visible in the skeleton. In teeth, mineralization can be interrupted by defects in pathways involving vitamin D, calcium, or phosphorous (D'Ortenzio et al. 2016). In typical tooth formation, dentine is laid down and then calcospherites grow slowly outward to fuse with one another, thus calcifying the dentine. However, in individuals suffering from vitamin D deficiency, these calcospherites will fail to grow sufficiently and will not fuse (D'Ortenzio et al. 2016). This results in the formation of 'interglobular dentine' or spaces of

unmineralized matrix in the teeth, which are visible during histological analysis (D'Ortenzio et al. 2016). While other areas of the bone are constantly remodeled during an individual's lifetime, the teeth are not. Thus, this interglobular dentine can illuminate periods of vitamin D deficiency in an individual's past when traces of the disease elsewhere in the skeleton have been erased.

Because rickets is most often caused by vitamin D deficiency which is usually preventable with exposure to sunlight, the disease is uncommon but not unknown in the archaeological record prior to the Industrial Revolution. In a Mid-Holocene site in South Africa (c. 3000 BCE), the skeletal remains of an infant aged between 3.5-5 months were uncovered. The infant exhibited flared sternal ends, a bowed left tibia, cupping in the proximal end of the right ulna, and the shafts of the long bone had unusually thick cortical bone. These pathologies were attributed to rickets, but likely not nutritional rickets as isotope values were suggestive of good nourishment (Pfeiffer and Crowder 2004). In an infant aged approximately 2.5 years at death from a 4th century site in France, Blondiaux and colleagues observed flared sternal rib ends, lateral straightening of ribs, flared and cupped metaphyses on the long bones, and cranial porosity which were concluded to be rickets (2002). This case of rickets was attributed to a type of hereditary vitamin D-resistant rickets, such as X-Linked Hypophosphatemia (Blondiaux et al 2002).

The first known use of 'rickets' in the English-speaking world was as a cause of death in the 1636 Annual Bill of Mortality for the City of London, followed in the next few decades by several papers about the new disease and its symptoms, though the link to sunlight exposure would not be realized until centuries later (Mays 2017; O'Riordan 2006). Thus it is not surprising that the majority of rickets cases in the archaeological record date to the period of smoke-darkened skies and increased urbanization which characterized industrialization in the

mid-17th century or later Europe and the U.S. A comparative study used bowing in the long bones to diagnose rickets in two different cemeteries from East London, Broadgate (1569-1720 CE) and Spitalfields (1729-1859 CE). Of the subadults in Broadgate, 20% showed evidence of rickets, compared to 14.7% of subadults in Spitalfields. The difference in these frequencies was attributed to differences in social class (Pinhasi et al. 2006). In a 19th century church in Birmingham, England, 21 of 164 juveniles were determined to have rickets based on flared long bone metaphyses, flared sternal rib ends, and bowed long bones (Mays et al. 2007). Subadult individuals from 19th century Surrey seem to be more affected, with 14 of 79 subadults exhibiting at least 3 of the indicators of rickets, including flaring at the sternal ends of ribs, flaring or cupping of the long bone metaphyses, leg bone deformities, and arm bone deformities (Watts and Valme 2018).

Rickets affected individuals living outside of England as well. Nine children aged birth to five years from the prominent Medici family were uncovered in a family tomb in Italy, eight of whose remains were commingled but lived sometime between 1671-1737 and another who lived between 1598-1602. Of these individuals, four were found to have probable cases of rickets, exhibiting bending deformities in the leg and arm bones, as well as flaring in the sternal ends of the ribs (Giuffra et al. 2013). In a cemetery from North Holland in the Netherlands (1617-1866 CE), nine of 95 subadults (neonate-15 years) were determined to be rachitic based on deformed arm and leg bones, costochondral rib flaring, and irregular mandible angle. In this instance, rickets was proposed to be a result of nutritionally deficient weaning foods (cereals and goat's milk) in combination with swaddling during the first year (Veselka et al 2013). Rachitic long bone bowing and metaphyseal flaring was also observed in at least 18 subadults from a

commingled burial in an early 19th century African-American church in Philadelphia, Pennsylvania (Ellis 2010).

In the majority of Late Ottoman era sites in both Israel and Jordan, no skeletal indicators of rickets were observed (Eakins 1993; Mitchell 2006; Rose and Khwaleh 2012; Smith and Horowitz 2009). Bending deformities were observed in 5.3% of tibiae from the Late Islamic period in Caesarea, Israel, but the ages of individuals affected were not specified (Smith and Zegerson 1999). Bowing, swelling of the diaphysis, and flared metaphyseal ends were reported in long bones of bedouin burials in two sites (Tel Harif and Tel Sheva) in Late Ottoman era Israel (~1850CE), which are likely indicators of rickets. However, these conditions were combined with several others and reported as simply "pathologies" for each bone, and thus it is not possible to further explore the implications of these findings (Goldstein, Arensburg, and Nathan 1976).

When two or more diseases are present and active in the same individual, they may interact with each other and manifest differently than when they occur alone. Scurvy inhibits new bone formation while rickets inhibits mineralization of new bone so that in instances of co-occurrence, only the effects of one disease may be visible in the skeleton (Schattmann et al. 2016). Therefore, instances of co-occurrence in the archaeological record are rare. In a child approximately 6 years of age from a Chalcolithic site in Spain (2400 BCE), bowed ulnas and tibiae were observed as well as porosity in the sphenoid, orbits, maxilla, and mandible. The authors suggest that co-occurrence of rickets and scurvy are responsible for these pathologies (Castilla et al. 2014). Two studies have found possible cases of co-occurrence in skeletal remains from Roman-era Britain. In Dorset, an infant (aged 0-3 years) was diagnosed with scurvy and rickets based on criteria published by Brickley and Ives (2008) but the specific pathologies

prompting this diagnosis were not provided (Redfern et al. 2012). In a 'Romanized' Dorchester population of 364 juveniles (<17 years), just 7 individuals had possible cases of co-occurrence of rickets and scurvy. In this study, rickets was diagnosed using criteria proposed by Ortner and Ericksen (1997) while scurvy was diagnosed when individuals had both cranial porosity and sclerotic new bone. Again, the specific pathologies found in each individual diagnosed with co-occurring rickets and scurvy were not included (Lewis 2010).

The most comprehensive study to date of the co-occurrence of rickets and scurvy used the skeletal remains of 48 juveniles under the age of 5 years from 16th-18th century Douai, France (Schattmann et al. 2016). Of these, 12 individuals had possible cases of co-occurring rickets and scurvy, with at least half of the individuals exhibiting scorbutic porosity in the maxillae, mandibles, and orbits, and the abnormal growth plate porosity, thickened long bones, and femoral *coxa vara* typical of rickets. Interestingly, porosity of the greater wing of the sphenoid, often considered a diagnostic feature of scurvy, was only observed in 2 of the individuals. Likewise, bending deformities, typically associated with rickets, were only observed in the arm bones of 2 individuals and the leg bones of 4 individuals (Schattmann et al. 2016). These data suggest that in instances of co-occurrence, scurvy and rickets interfere with each other enough to sufficiently affect expression in the skeletal remains, even limiting the expression of the most common pathologies.

Both scurvy and rickets are influenced by diet. Traditionally the bedouin of Jordan herded goats and sheep, with wealthier tribes such as the Beni Sakhr also maintaining herds of camels (Lewis 1987). The milk from these animals was consumed either raw or in the form of cheeses and yogurt (Hobbs 1989). The animals were only killed and consumed directly for special occasions (Groen et al 1964). Additionally, historical sources report that the Bedouin

supplemented their diet seasonally with small-scale cultivation of crops, such as melons and cucumbers (Smith 1896). Of these, cantaloupe has the highest amount of vitamin C (Table 2).

Table 2: Vitamin C content of known vegetables consumed by the bedouin

Vegetable	Ascorbic Acid
Cucumber	10 mg/100g
Cantaloupe	53 mg/100g
Watermelon	7 mg/100g

(Stone 1937)

The bedouin are known to use *Heliotropium ramossimum* as a treatment for mouth sores, including those caused by scurvy. The leaves of the plant were often boiled into a paste which was then applied directly to the sore (Abu-Rabia 2015). Although the vitamin C content of *H. ramossimum* has not yet been quantified, the closely related *H. somalense* was found to have an average of 39mg of vitamin C in every 100g of edible plant portions, approximately 130% of the daily value recommended for adult males (Mwajumwa, Kahangi, and Imungi 2010). The bedouin also eat either the whole plants or simply the leaves of *Malva nicaeensis*, or 'khubeza' (Groen et al. 1964). The sister species, *M. sylvestris,* was reported to have approximately 10mg of ascorbic acid in every 100g of leaves, much lower than that of the *Heliotropium* genus. However, the *M. sylvestris* flowers were found to be significantly more nutritious, with over 100mg of vitamin C for every 100g of flower mass (Barros, Carvalho, and Ferreira 2010).

Milk is an excellent source of nutrition which would have been available for most of the year. The Damascus goat, one of the most common breeds in Jordan, produces an average of 2.5 kg of milk per day for 281-336 days of the year, between March and November (Teleb et al 2003; Zaitoun et al 2005). Goat milk has between 88-116 mg of calcium per 100 g of milk but

only about 0.89 mg per 100g of ascorbic acid (Sawaya et al 1984). Camels provide even more nutrition over longer periods of time. Though milk production varies drastically by breed, Saudi camels were found to produce an average of 1,970 liters of milk per year (Musaad et al 2012). Milk production peaks during the colder months (November to January) and can last as long as 18 months (Musaad et al 2012; Mohamed et al 2005). The average calcium content of camel milk is 114 mg per 100g which is comparable to goat milk (Al-haj & Alkanhal 2010). However, camel milk is rich in vitamin C, with between 37.4-47.8 mg per 100g, more than three times the amount of ascorbic acid found in cow milk (Mohamed et al 2005; Musaad et al 2012).

Because camels produce large quantities of nutritious milk for longer periods of time than goats, individuals who could afford to keep camels were likely less susceptible to nutritional and dietary deficiencies. Those individuals who only had access to goat milk supplemented their vitamin C content during the fall and winter with "khubeza" (Groen et al 1964). However, with the adoption of large-scale agriculture, production of cereals would have replaced the cultivation of diverse crops. Furthermore, as wheat grew in economic importance, it grew in dietary significance as well, becoming a staple among the modern bedouin (Groen et al 1964).

Modern bedouin tribes in Israel primarily consume wheat in the form of "rarif", an unleavened bread, but also as a flour porridge ("kuskusu") and crushed wheat ("burgul") (Groen et al 1964). Historically, barley and millet were grown and likely consumed in some form as well (Burckhardt 1822). While all cereals have a high caloric content, they lack important micronutrients (Yu and Tian 2018). Furthermore, cereals contain phytates which can interfere with the absorption of other micronutrients (Reddy et al 1982). Phytic acid is negatively charged and binds to positively charged elements such as zinc, iron, and most importantly for the study of

rickets, calcium (Konietzny & Greiner 2003). The unleavened bread consumed by the bedouin is extremely high in phytates, with 125mg of phytic acid/100g of dry matter (Berlyne et al 1973).

The high phytic acid content in bread has been implicated in cases of rickets and osteomalacia in Indian immigrants in the United Kingdom. Because the darker-skinned Nigerian immigrants included in the study were not vitamin D deficient, the authors concluded that regular consumption of the high-phytate chapati bread rather than biological barriers to UV-B exposure due to melanin production caused rickets and osteomalacia in the Indian immigrants (Wills et al 1972). Additionally, one study of rachitic and non-rachitic children in Pakistan found that while vitamin D and calcium levels were similar between the two groups, the rachitic children had significantly higher phytase levels, suggesting phytic acid limited the absorption of calcium and consequently impaired bone mineralization in the rachitic children (Dunnigan & Smith 1965).

During the Late Ottoman era, the people of Tell Hisban experienced shifts in political power, and likely a shift in diet for some portion of the bedouin population. Inadequate nutrition can result in the development of metabolic diseases, such as vitamin C and D deficiencies. Because subadults are still growing, they are the most at risk of developing metabolic diseases, and subsequently are more likely to exhibit skeletal indicators of such diseases. Studying evidence of scurvy and rickets in the juvenile skeletal remains from Tell Hisban can illuminate the physiological consequences of these changes for this group of people.

CHAPTER 3

Materials & Methods

Materials:

The Hisban sample consists of a commingled sample comprised of at least 62 individuals. Preservation was generally excellent, with only a few bones incomplete or exhibiting taphonomic damage. Currently the remains are housed in a storage facility in Madaba associated with the Hisban Archaeological Project and were studied at the American Center of Oriental Research in Amman for this project in 2018. In addition, 68 long bones exported for isotope analysis and held in the bioarchaeology laboratory at East Carolina University were included in this study.

Although the total sample included both adult and subadult individuals, for this study, only the 29 individuals aged 15 years and younger were included. Juveniles were selected because individuals who are still developing are more susceptible to metabolic disease, and these diseases are evident as deformations in the bone. Furthermore, this somewhat atypical age range was picked for convenience, as all the individuals in the population were either younger than 15 years, or older than 18 years. Age was established based on rates of dental eruption in the maxilla and mandible and estimated using long bone length and rates of epiphyseal fusion bones not pathologically altered (Schaefer et al. 2009). The subadult MNI was calculated using both the left and right femora. In total, more than 1,400 bones belonging to juvenile individuals were studied for evidence of vitamin C or D deficiency (Table 3).

Table 3: List of all elements analyzed and included in the study

	Element	# Analyzed
Cranial	Frontal	47
	Mandible	30
	Maxilla	52
	Occipital (vault)	31
	Occipital (basilar)	21
	Occipital (lateral)	43
	Parietal	78
	Sphenoid (greater wing)	24
	Sphenoid (sella turcica)	27
	Temporal	52
	Zygomatic	42
Ribs	Rib 1	42
	Rib 2	49
	Ribs 3-10	451
	Rib 11	31
	Rib 12	13
Long Bones	Femur	59
	Fibula	41
	Humerus	58
	Radius	51
	Tibia	50
	Ulna	46
	Scapula	54
	Ilium	41
	Total	1431

Methods:

The bones were analyzed macroscopically for diagnostic and associated indicators of vitamin C and D deficiencies, as specified in current literature (i.e. Brickley and Ives 2008; Brickley et al. 2017; Moore and Koon 2017; Ortner 2003; Ortner and Mays 1998). Observations were recorded using the standards specified by Buikstra and Ubelaker (1994), with some modifications for bending deformities not adequately described by existing codes. Differential diagnosis of these metabolic deficiencies usually involves the identification of a suite of skeletal changes seen in an individual (Brickley and Ives 2008, Ortner and Ericksen 1997). Because this sample is commingled, observations of lesions and other changes related to these conditions in different elements cannot be linked. Because some skeletal anomalies are more diagnostic of vitamin C and D deficiencies than others, only the specific pathologies considered indicative of either scurvy or rickets were included in this study. The bones included in this study are the cranial elements (frontal, occipital, sphenoid, maxilla, mandible, zygomatic), the long bones (tibia, fibula, femur, humerus, radius, ulna), the ribs, the scapula, and the ilium. Although rachitic lesions can be manifest in the vertebral column, these are typically diagnosed using the entirety of the individual's vertebrae (Ortner 2003). The commingled nature of the sample makes this feat impossible, so this indicator was excluded from the study. Additionally, scorbutic porosity may be found in the clavicle but it is not considered diagnostic and thus was excluded from this study as well (Brickley and Ives 2008).

Scorbutic and rachitic lesions in human bone take the form of porosity, abnormal bone formation, and shape anomalies (Table 4). As part of data collection, the side, aspect, and either the extent of bone affected (<1/3, 1/3-2/3, or >2/3) or the extent of expression (barely discernible or clearly discernible) of specific lesions were recorded.

Scurvy most commonly manifests in the skeleton as porosity, which is often accompanied by abnormal new bone formation (ANB), which may affect only the outer cortex or the inner trabeculae as well (Figures 1-5). To be considered indicative of scurvy, this porosity had to be less than 1mm in diameter and had to consist of at least 10 foci. The ANB had to be sclerotic in nature to be associated with scurvy.

Figure 1: A subadult left zygomatic with scorbutic porosity and ANB (H98 FLDL Sq2 Loc3 B.21 #140b)

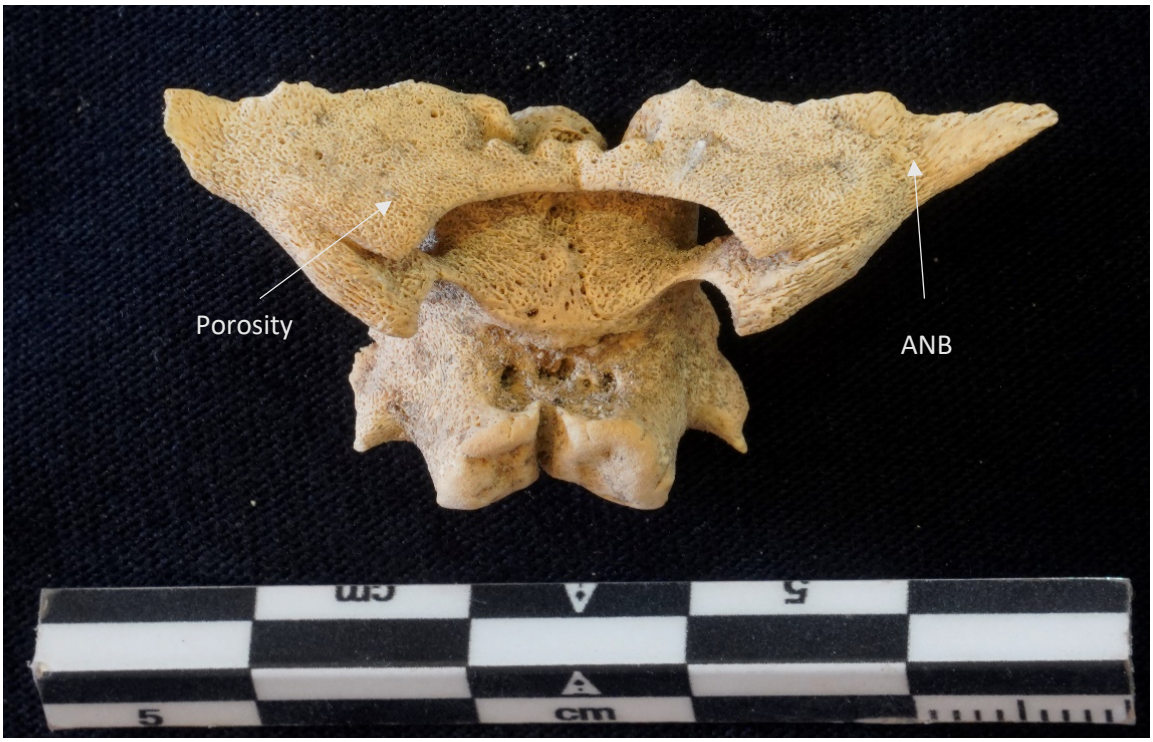

Figure 2: A subadult sphenoid with scorbutic porosity and ANB (H98 FLDL Sq2 Loc3 PB17 B.23 #218a)

Figure 3: The basilar portion of a subadult occipital bone with scorbutic porosity and ANB (H98 FLDL Sq2 Loc3 B.2 #89)

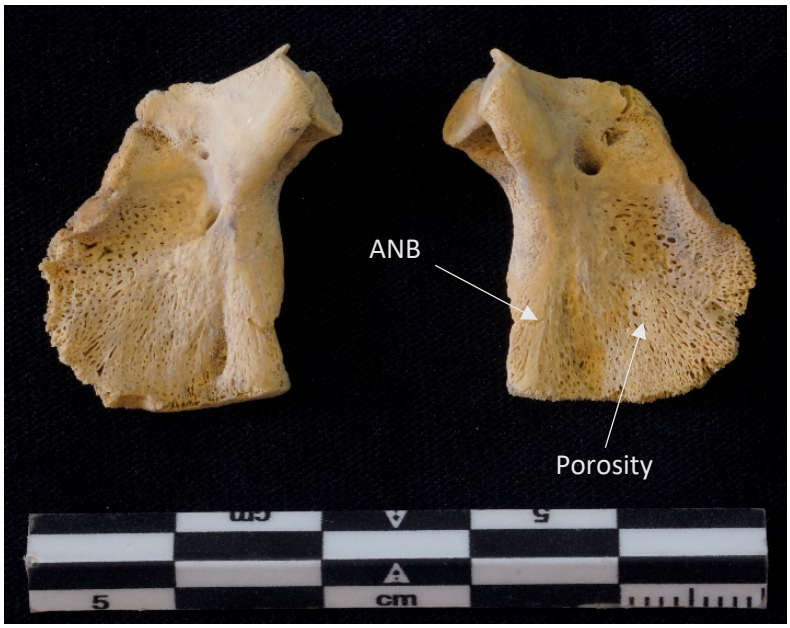

Figure 4: The basilar portions of a subadult occipital with scorbutic porosity and ANB (H98 FLDL Sq2 Loc3 B.2 #87 & 88)

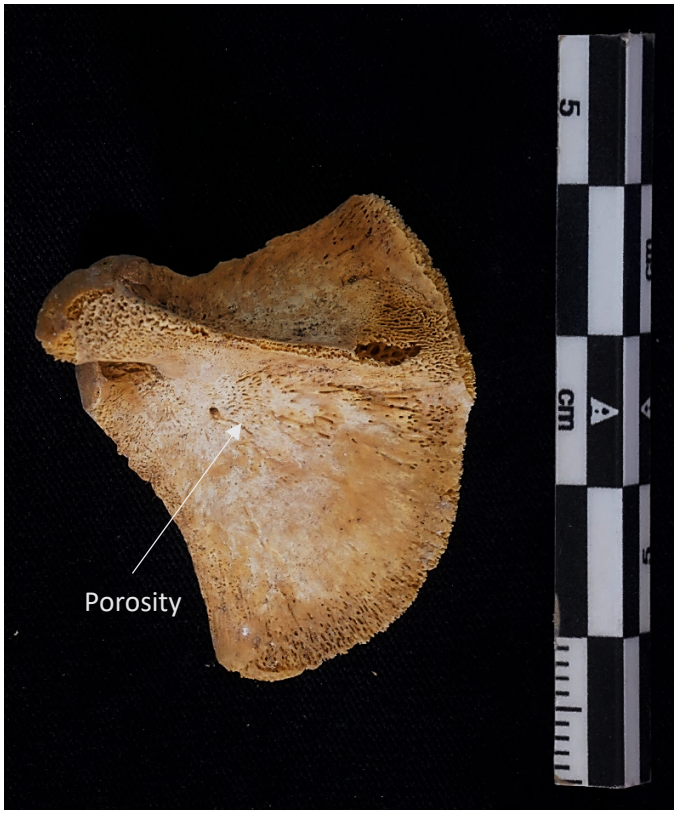

Figure 5: A subadult left scapula with scorbutic porosity, particularly in the infraspinous fossa (H01 FLDL Sq2 Loc3 B.1 #247f)

Rickets results in a greater variety of pathologies in the skeleton than scurvy. Bending deformities in the long bones, as well as the mandible and ilium, and lateral, 'J'-shaped straightening of the ribs were considered indicative of rickets and were scored as either 'barely' or 'clearly' bowed (Figures 6-8). Additionally, 'thickening' of the epiphyseal shafts and cupping, tilting, or flaring of the metaphyseal ends of long bones were recorded as indicative of rickets (Figures 9-11). When available the metaphyseal ends of long bones under the growth plate were observed for texture which was scored as either 'normal', 'fine grained and slightly rough', 'rough with pitting', 'extreme pitting and porosity', or 'frayed' according to Ortner and Mays (1998). Finally, 'beading' or anteroposterior expansion of the sternal ends of the ribs was considered indicative of rickets and likewise scored as 'barely' or 'clearly' affected (Figure 12).

Figure 6: A subadult right fibula with bowing indicative of rickets (H98 FLDL Sq2 Loc3 B.1B #136c)

Figure 7: Top: A subadult mandible with bending deformities in the ramus and condyles (H98 FLDL Sq2 Loc3 B.23 #65)

and Bottom: a normal subadult mandible for comparison (Coquerelle et al 2011)

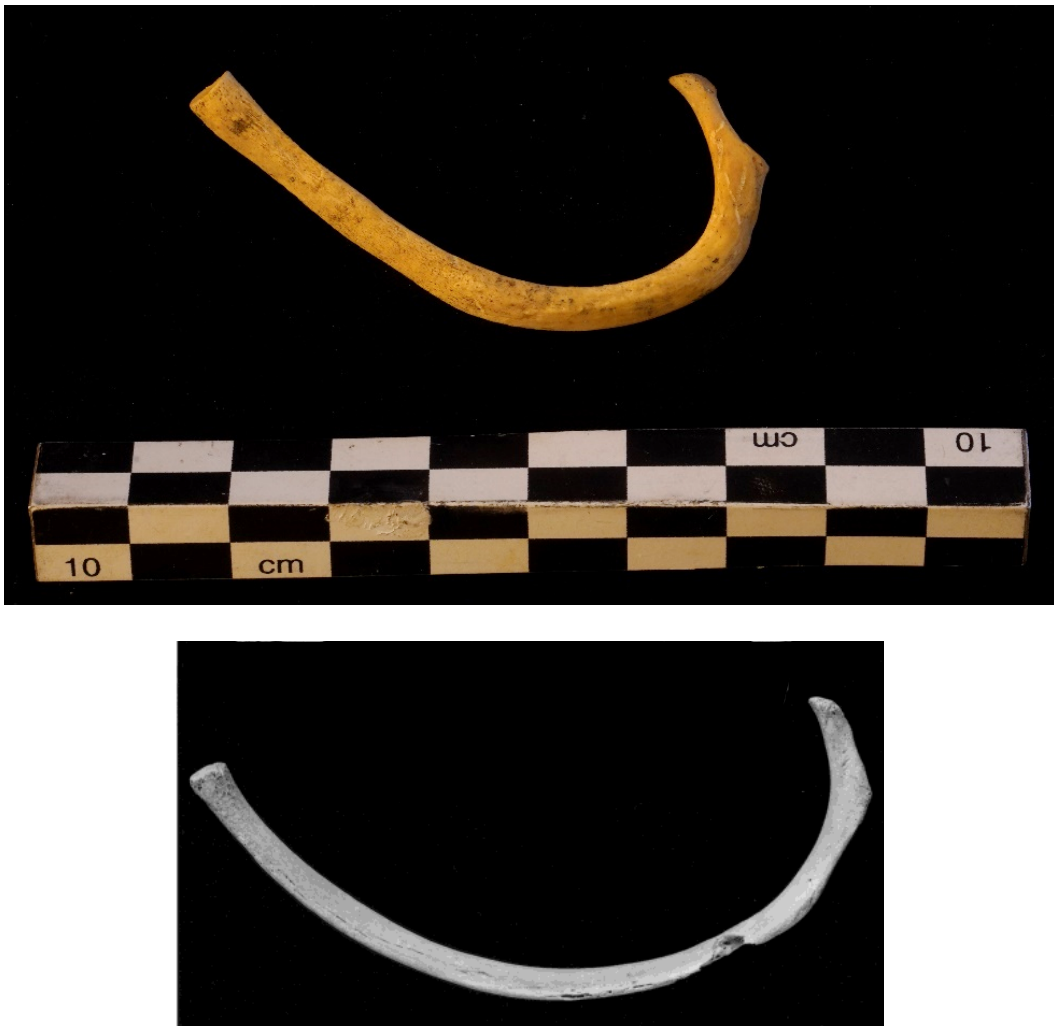

Figure 8: Top: A subadult left rib with lateral 'J-shaped' straightening (H98 FLDL Sq2 Loc3 B.1D #3a) and Bottom: normal 'C-shaped' right rib for comparison (Ortner and Mays 1998)

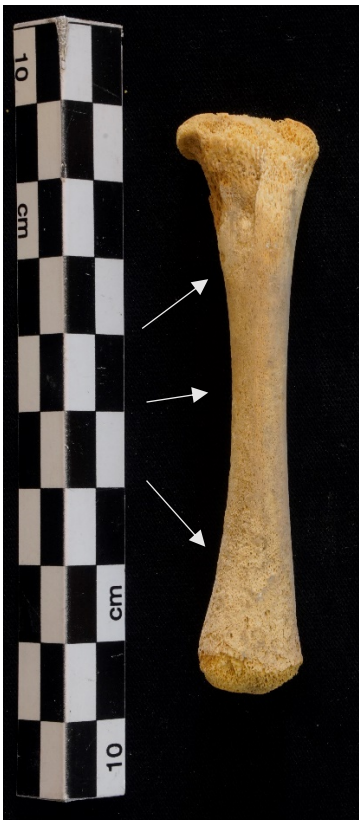

Figure 9: A subadult right tibia with 'thickening' of the diaphysis (H01 FLDL Sq2 Loc3 B.1 #236d)

Figure 10: A subadult left radius with metaphyseal flaring on the distal end (H98 FLDL Sq2 Loc3 B.1D #200d)

Figure 11: A pair of subadult femora with metaphyseal flaring on the distal ends (H98 FLDL Sq2 Loc3 PB13 B.11 #97b & 98b)

Figure 12: A subadult right rib sternal end exhibiting the 'beading' deformity (H98 FLDL Sq2 Loc3 B.1D #15a)

Rickets and scurvy may also produce similar pathologies that were recorded without attempting to differentiate between possible etiology. Both diseases may produce porosity along the ends of the long bone diaphyses. To be considered indicative of metabolic disease rather than typical bone growth, this porosity had to extend more than 10mm from the metaphyseal ends (Figure 13). Both diseases may produce 'flaring' or superioinferior expansion of the sternal ends of the ribs, which was noted but not otherwise scored (Figure 14). Additionally, both rickets and scurvy may result in porosity in the bones of the cranial vault. This pathology was recorded following the same methods as the more diagnostic scorbutic cranial porosity found in the sphenoid, maxilla, and mandible. However, again due to the commingled nature of the sample, because this porosity alone is not diagnostic of metabolic disease, it will not be discussed further.

Although the results of several indicators of metabolic disease are presented in this study, not all are considered in the discussion. Only porosity in those bones considered most diagnostic for scurvy (the sphenoid, mandible, and maxilla) were used as these bones are most commonly used in other studies and thus most convenient for comparative purposes. Likewise, only bending deformities of the long bone shafts and shape abnormalities of the long bone growth plates aree discussed in detail as indicators of rickets, as these are the pathologies most often reported in other studies of the disease and thus the pathologies most easily compared. When possible, the relationship between two pathologies in a single type of bone was explored, such as with bending deformities and metaphyseal flaring in the long bones. However, because of the commingled nature of the Tell Hisban sample, it was not possible to compare frequencies of pathologies on an individual basis. Rather, the ratios of bones affected by these pathologies were calculated and compared to other studies. This allowed a better understanding of which bones may be most

affected in the course of a disease, and facilitates the comparison of pathologies between a commingled and non-commingled sample.

Figure 13: A subadult left ulna with porosity extended over 10mm from the metaphyseal ends (H98 FLDL Sq2 Loc3 B.1D #182c)

Figure 14: Two subadult left ribs exhibiting flaring of the sternal ends (H98 FLDL Sq2 Loc3 B.1D #5a & 9a)

Table 4: Specific Bones and Pathologies Analyzed

Bone: Location/Pathology	*Scurvy/Rickets/Both*	*Diagnostic?*	*Source*
Frontal: Vault porosity	Both	Non-diagnostic	Ortner & Ericksen 1997
Frontal: Vault ANB	Both	Non-diagnostic	Ortner & Ericksen 1997
Frontal: Orbit porosity	Both	Non-diagnostic	Ortner & Ericksen 1997
Frontal: Orbit ANB	Both	Non-diagnostic	Ortner & Ericksen 1997
Parietal: Vault porosity	Both	Non-diagnostic	Ortner & Ericksen 1997
Parietal: Vault ANB	Both	Non-diagnostic	Ortner & Ericksen 1997
Parietal: Inferior aspect porosity	Scurvy	Probable	Stark 2014
Parietal: Inferior aspect ANB	Scurvy	Non-diagnostic	Stark 2014
Occipital: Vault porosity	Both	Non-diagnostic	Ortner & Mays 1998
Occipital: Vault ANB	Both	Non-diagnostic	Ortner & Mays 1998

Bone: Location/Pathology	*Scurvy/Rickets/Both*	*Diagnostic?*	*Source*
Occipital: Basilar portion porosity	Scurvy	Probable	Moore & Koon 2017
Occipital: Basilar portion ANB	Scurvy	Possible	Moore & Koon 2017
Occipital: Lateral portion porosity	Scurvy	Probable	Moore & Koon 2017
Occipital: Lateral portion ANB	Scurvy	Possible	Moore & Koon 2017
Sphenoid: Greater wing porosity	Scurvy	Probable	Ortner et al. 1999
Sphenoid: Greater wing ANB	Scurvy	Probable	Ortner et al. 1999
Sphenoid: Body porosity	Scurvy	Possible	Brickley & Ives 2008
Sphenoid: Body ANB	Scurvy	Possible	Brickley & Ives 2008
Temporal: Posterior aspect porosity	Scurvy	Non-diagnostic	Brickley & Ives 2008
Temporal: Posterior aspect ANB	Scurvy	Non-diagnostic	Brickley & Ives 2008
Zygomatic: Interior aspect porosity	Scurvy	Possible	Ortner & Ericksen 1997
Zygomatic: Interior aspect ANB	Scurvy	Non-diagnostic	Ortner & Ericksen 1997
Maxilla: Anterior portion porosity	Scurvy	Possible	Ortner & Ericksen 1997
Maxilla: Anterior portion ANB	Scurvy	Possible	Ortner & Ericksen 1997

Bone: Location/Pathology	*Scurvy/Rickets/Both*	*Diagnostic?*	*Source*
Maxilla: Posterior portion porosity	Scurvy	Probable	Ortner & Ericksen 1997
Maxilla: Posterior portion ANB	Scurvy	Non-diagnostic	Ortner & Ericksen 1997
Mandible: Coracoid process porosity	Scurvy	Possible	Stark 2014
Mandible: Coracoid process ANB	Scurvy	Non-diagnostic	Stark 2014
Mandible: Ramus porosity	Scurvy	Possible	Stark 2014
Mandible: Ramus ANB	Scurvy	Non-diagnostic	Stark 2014
Mandible: Abnormal condyle angle	Rickets	Possible	Ortner & Mays 1998
Long Bones: Metaphyseal Porosity	Both	Non-Diagnostic	Brickley et al. 2018
Long Bones: Metaphyseal ANB	Both	Non-Diagnostic	Ortner & Mays 1998
Long Bones: Epiphyseal Plate Porosity	Rickets	Probable	Ortner & Mays 1998
Long Bones: Porosity on Concave Side	Rickets	Possible	Brickley et al. 2018
Long Bones: Bowing	Rickets	Probable	Ortner & Mays 1998

Bone: Location/Pathology	*Scurvy/Rickets/Both*	*Diagnostic?*	*Source*
Long Bones: Flaring Metaphyses	Both	Possible	Ortner & Mays 1998
Long Bones: Thickening	Rickets	Probable	Ortner & Mays 1998
Long Bones: Cupping at growth plate	Rickets	Probable	Brickley & Ives 2008
Long Bones: Growth plate tilting	Rickets	Probable	Watts & Valme 2018
Femur: Femoral coxa vara	Rickets	Probable	Ortner & Mays 2018
Scapula: Infraorbital fossa Porosity	Scurvy	Probable	Brickley & Ives 2008
Scapula: Supraorbital fossa Porosity	Scurvy	Probable	Brickley & Ives 2008
Ilium: Internal aspect Porosity	Scurvy	Possible	Brickley & Ives 2008
Ilium: External aspect Porosity	Scurvy	Possible	Brickley & Ives 2008
Ilium: Exaggerated medio-lateral curvature	Rickets	Possible	Ortner & Mays 1998
Ribs: Sternal End porosity	Both	Non-diagnostic	Brickley et al. 2018
Ribs: Abnormal rib neck angle	Both	Possible	Ortner & Mays 1998
Ribs: Lateral straightening	Both	Possible	Ortner & Mays 1998
Ribs: Flaring sternal ends	Both	Possible	Brickley et al. 2018

Bone: Location/Pathology	*Scurvy/Rickets/Both*	*Diagnostic?*	*Source*
Ribs: Beading	Both	Possible	Ortner & Mays 1998
Ribs: Costochondral Fracture	Both	Possible	Brickley & Ives 2008

CHAPTER FOUR

Results

The results were grouped by type of abnormality observed, which was reintegrated in terms of differential diagnosis and frequency of vitamin C and D deficiencies within the Hisban sample.

No Observable Pathologies

The 9 juvenile skulls exhibited no observable pathologies. Of these skulls, 6 were entirely complete, and 3 were missing portions of the occipital. The estimated ages at death ranged from 18 months ± 6 months to 12 years ± 3 years, and over half (55%) of the skulls represented older children (aged 5-10 years).

Shape Abnormalities

Frequency rates for shape abnormalities by bone are recorded in Tables 5 and 6. Of the shape abnormalities associated with rickets, the majority are found in the long bones with only a few in the mandibles and ilia. Among long bones, 39.7% exhibit bending deformities, 50.2% show metaphyseal flaring, and only 8.2% have an abnormally wide diaphysis. Bending deformities were found in 55% of lower limb bones and 24.5% of upper limb bones. Metaphyseal flaring was observed in 65% of lower limb bones and 34% of upper limb bones overall. Of the lower limb bones, 7.3% were observed to be abnormally wide, and 9% of upper limb bones exhibited the same pathology. A strong association exists between the presence of metaphyseal flaring and bowing in both the upper limbs (χ^2=35.632, p<0.0001) and lower limbs (χ^2=30.274, p<0.0001) Most of the abnormally wide upper limb bones also have flared metaphyses (χ^2=6.096, p=0.0136). No strong relationship is seen in the lower limbs (χ^2=1.853,

p=0.1734). On the other hand, upper and lower limb bones with bowing tend to have abnormal widths (upper limb χ^2=6.866, p=0.0088; lower limb χ^2=6.622, p=0.0101.

Abnormalities affecting the growth plates are reported in Tables 6 and 7. A larger percentage of upper limb bones (4.5%) than lower limb bones (1.3%) exhibited growth plate cupping. Growth plate tilting was observed in 11% of upper limb bones and 14.6% of lower limb bones. There was no relationship between metaphyseal cupping and tilting in either the upper limbs (χ^2=1.734, p=0.1879) or the lower limbs (χ^2=0.644, p=0.4224). Most of the changes to the epiphyseal plate surface appear as "fine grained and rough", although in many cases this feature was not observable due to preservation of the metaphyses. In general, bones showing bowing (χ^2=4.052, p=0.3991) and flared metaphyses (χ^2=7.294, p=0.1211), were more likely to have some pitting and fraying of the metaphyseal ends, although the associations were not strong. On the other hand, bones that were not abnormally wide (χ^2=5.821, p=0.2129), or had uncupped (χ^2=6.585, p=0.1595) or untilted (χ^2=2.330, p=0.6754) metaphyseal ends were more likely to have pitting and fraying of the metaphyseal ends, but again these relationships are not strong.

Table 5: Frequencies of Shape Abnormalities in Long Bones, Ilia, and Mandibles

Element	**Total Number**	**Bending**			**Metaphyseal Flaring**			**Uniformly Abnormally Wide**		
		Barely	**Clearly**	**Total %**	**Barely**	**Clearly**	**Total %**	**Barely**	**Clearly**	**Total %**
Humerus	58	5	0	**8.6**	4	3	**12.1**	2	0	**3.4**
Radius	51	9	10	**37.3**	13	12	**49.0**	2	2	**7.8**
Ulna	46	9	5	**30.4**	17	4	**45.7**	5	3	**17.4**
Femur	59	19	16	**59.3**	20	19	**66.0**	3	1	**6.8**
Tibia	50	13	12	**50.0**	23	12	**70.0**	1	4	**10.0**
Fibula	41	13	10	**56.1**	14	9	**56.1**	0	2	**4.9**
Ilium	41	5	0	**12.2**						
Mandible	30	8	4	**40.0**						

Table 6: Frequencies of Growth Plate Deformities by Long Bone

Element	**Total Number**	**Growth Plate Cupping**			**Growth Plate Tilting**		
		Barely	**Clearly**	**Total %**	**Barely**	**Clearly**	**Total %**
Humerus	58	0	0	**0.0**	0	0	**0.0**
Radius	51	1	4	**9.8**	6	6	**23.5**
Ulna	46	2	0	**4.3**	4	1	**10.9**
Femur	59	0	0	**0.0**	0	1	**1.7**
Tibia	50	0	1	**2.0**	10	5	**30.0**
Fibula	41	1	0	**2.4**	4	2	**14.6**

Table 7: Long Bone Epiphyseal Growth Plate Abnormalities

Element	**Total with Epiphyseal Plates**	**Normal**	**Fine Grained and Rough**	**Rough with Pitting**	**Extreme Pitting and Porosity**	**Fraying**	**Total % Affected**
Humerus	4	4	0	0	0	0	**0.0**
Radius	9	5	3	1	0	0	**44.4**
Ulna	10	4	6	0	0	0	**60.0**
Femur	20	15	5	0	0	0	**20.0**
Tibia	23	14	6	2	1	0	**39.1**
Fibula	15	14	1	0	0	0	**6.7**

Porosity

Porosity was observed in several elements and affected either the periosteum or the periosteum and the cortex of the bone (Table 8).

The long bones were observed for porosity extending greater than 10 mm from the end of the metaphysis. The leg bones exhibited the highest percentages of porosity which mostly affected the periosteal layer. However, in instances of porosity which affected both the periosteal layer and the cortex, the ulna was more severely affected (27.5%) than the humerus (8.6%). Long bone metaphyseal end porosity is more likely to be seen in upper and lower limb bones that are bowed (upper χ^2=12.214, p=0.0005; lower χ^2=16.463, p=0.0003), and have flared metaphyses (upper χ^2=22.679, p<0.0001; lower χ^2=18.486, p<0.0001), epiphyseal plate tilting (upper χ^2=15.918, p<0.0001; lower χ^2=4.873, p=0.0273), and abnormal epiphyseal plate texture (upper χ^2=22.275, p<0.0001; lower χ^2=32.803, p<0.0639) but not be seen in bones that are abnormally wide (upper χ^2=6.675, p=0.0098; lower χ^2=3.434, p=0.0018). No strong relationship was seen between bone loss and upper and lower limb cupping (upper χ^2=0.360, p=0.5482; lower χ^2=2.314, p=0.1283).

The unspecialized ribs 3-10 have the highest percentage of elements affected by periosteal porosity. Ribs 3-10 are also significantly more affected by porosity of the periosteum and cortex (17.8%) than other ribs. Interestingly, not a single rib 12 showed any evidence of porosity.

Among the cranial bones, the lateral portion of the frontal bone had the highest percentage (55%) of elements exhibiting porosity in the periosteal layer, followed by the temporals (53.8%) and the basilar portion of the occipital (47.1%). The temporal bone had the highest percentage of elements affected by combination periosteal and cortex porosity (12.8%), followed distantly by the zygomatic (4.8%). The relatively high frequency of temporal bones affected with porosity is interesting because porosity on the temporal bone is not considered diagnostic for rickets or scurvy. Unfortunately, because most of the cranial bones could not be associated with each other, correlations of porosity frequencies between elements were not computed.

Table 8: Type of Porosity and Extent of Element Affected

Element	**Total Number**	**Porosity**							
		Periosteum				**Periosteum + Cortex**			
		< 1/3	**1/3-2/3**	**> 2/3**	**Total %**	**< 1/3**	**1/3-2/3**	**> 2/3**	**Total %**
Humerus	58	3	4	1	**13.8**	5	0	0	**8.6**
Radius	51	2	3	1	**11.8**	11	2	1	**27.5**
Ulna	46	9	4	4	**37.0**	0	1	1	**4.3**
Femur	59	13	8	1	**54.2**	1	2	1	**6.8**
Tibia	50	14	14	4	**64.0**	0	0	2	**4.0**
Fibula	41	15	5	1	**51.2**	0	0	2	**4.9**
Scapula	54	12	5	2	**35.2**	2	0	1	**5.6**
Ilium	41	13	3	4	**48.8**	0	0	1	**2.4**
Rib 1	42	7	3	1	**26.2**	0	0	0	**0.0**
Rib 2	49	6	10	1	**34.7**	0	0	0	**0.0**
Ribs 3-10	451	84	87	13	**40.8**	3	5	0	**17.8**
Rib 11	31	4	7	1	**38.7**	0	1	0	**3.2**
Rib 12	13	0	0	0	**0.0**	0	0	0	**0.0**
Sphenoid	58	4	2	2	**13.8**	0	0	0	**0.0**
Zygomatic	42	3	4	0	**16.7**	1	0	1	**4.8**
Maxilla	45	5	3	6	**31.1**	0	0	0	**0.0**
Mandible	43	5	3	3	**25.6**	0	0	0	**0.0**
Basal	28	3	5	1	**32.1**	0	0	0	**0.0**
Lateral	51	5	13	6	**47.1**	0	1	1	**3.9**
Vault	35	4	3	0	**20.0**	1	0	0	**2.9**
Frontal	40	15	5	2	**55.0**	0	0	0	**0.0**
Parietal	66	11	11	4	**39.4**	0	1	1	**3.0**
Temporal	39	5	9	7	**53.8**	0	3	2	**12.8**

Abnormal New Bone Formation

Several different elements exhibited abnormal new bone formation which resulted in new periosteal bone or abnormal matrix (Table 9). Among the long bones, those of the leg exhibited the highest percentages of abnormal new periosteal bone, all in the form of woven (new, loosely arranged) bone. However, the radius and ulna were the long bones with the highest percentage of combination woven and sclerotic (dense and irregular) new periosteal bone. Among the long bones, the tibia was the most affected by abnormal matrix all expressed as woven, immature bone (4.0%), but the difference between this bone and the others is slight. Almost all the long

bones had a co-occurrence of ANB and metaphyseal end porosity (humerus χ^2=6.057, p=0.0138; radius χ^2=9.574, p=0.0020; ulna χ^2=26.787, p<0.0001; femur χ^2=19.414, p<0.0001; tibia χ^2=12.206, p=0.0005; fibula χ^2=17.658, p<0.0001)

Ribs 2, 11, and the 3-10 category had the highest percentages within the rib sample of new woven bone (38.8%, 38.7%, and 36.4%, respectively). These three groups are the only ribs to exhibit sclerotic new periosteal bone, or combination woven and sclerotic new bone. Abnormal matrix in the form of cancellous expansion, or expansion of the internal cortical bone was only recorded on three ribs, one in each of the rib 2, 11, and 3-10 categories. There was no evidence of pathology on any twelfth rib. Porosity and ANB were positively associated in Rib 1 (χ^2=15.700, p<0.0001), Rib 2 (χ^2=20.242, p<0.0001), Ribs 3-10 (χ^2=272.785, p<0.0001), and Rib 11 (χ^2=27.557, p<0.0001)

Of the cranial bones, the temporal had the highest percentage affected by new woven bone (48.7%), followed by the lateral portion of the occipital (37.3%) and frontal (35%) bone. In the combination woven and sclerotic new bone category, the temporal (28.2%) and lateral portions of the occipital (9.8%) continue this trend. However, there were no instances of combination woven and sclerotic bone observed on the basilar part of the occipital. Instead, the maxilla follows closely behind the lateral portion of the occipital, with 8.9% of elements exhibiting combination woven and sclerotic bone.

For every element except the basilar and lateral portions of the occipital, sclerotic new bone was rarely observed unless accompanied by woven bone. Almost all of the cranial bones observed for porosity also had ANB (frontal χ^2=25.946, p<0.0001; mandible χ^2=10.664, p=0.0011; maxilla χ^2=14.650, p=0.0007; basilar portion of occipital χ^2=5.786, p=0.0162; lateral portions of occipital χ^2=10.012, p=0.0016; squamosal portions of the occipital χ^2=21.324,

$p<0.0001$; greater wing of the sphenoid $\chi^2=6.235$; $p=0.0125$; sella turcica of the sphenoid $\chi^2=13.917$, $p=0.0002$, the zygomatic $\chi^2=3.845$, $p=0.0499$; and the temporal $\chi^2=26.902$, $p<0.0001$).

Table 9: Distribution of Abnormal New Periosteal Bone Across All Elements

Element	Total Number	Abnormal New Periosteal Bone											
		Woven Bone				Sclerotic Bone				Woven + Sclerotic Bone			
		< 1/3	1/3 - 2/3	> 2/3	Total %	< 1/3	1/3 - 2/3	> 2/3	Total %	< 1/3	1/3 - 2/3	> 2/3	Total %
Humerus	58	5	12	5	**37.9**	0	0	0	**0.0**	0	1	0	**1.7**
Radius	51	8	3	2	**25.5**	0	0	0	**0.0**	1	2	2	**9.8**
Ulna	46	8	2	3	**28.3**	0	0	0	**0.0**	1	0	2	**6.5**
Femur	59	18	3	4	**42.4**	1	0	0	**1.7**	2	1	0	**5.1**
Tibia	50	14	8	5	**54.0**	0	0	0	**0.0**	0	0	2	**4.0**
Fibula	41	12	5	0	**41.5**	0	0	0	**0.0**	0	0	2	**4.9**
Scapula	54	12	3	0	**27.8**	0	0	0	**0.0**	1	0	2	**5.6**
Ilium	41	13	4	0	**41.5**	0	0	0	**0.0**	0	0	3	**7.3**
Rib 1	42	6	5	1	**28.6**	0	0	0	**0.0**	0	0	0	**0.0**
Rib 2	49	10	9	0	**38.8**	0	1	0	**2.0**	0	0	1	**2.0**
Ribs 3-10	451	68	87	9	**36.4**	4	0	0	**0.9**	8	7	1	**3.5**
Rib 11	31	4	7	1	**38.7**	0	0	0	**0.0**	0	2	0	**6.5**
Rib 12	13	0	0	0	**0.0**	0	0	0	**0.0**	0	0	0	**0.0**
Sphenoid	58	5	1	1	**12.1**	0	0	0	**0.0**	0	1	3	**6.9**
Zygomatic	42	6	0	0	**14.3**	1	0	0	**2.4**	0	0	2	**4.8**
Maxilla	45	5	1	2	**17.8**	0	0	0	**0.0**	1	2	1	**8.9**
Mandible	43	5	4	0	**20.9**	1	0	0	**2.3**	1	0	1	**4.7**
Occ.-Basal	28	3	2	1	**21.4**	0	1	0	**3.6**	0	0	0	**0.0**
Occ.-Lateral	51	7	8	4	**37.3**	2	0	0	**3.9**	1	2	2	**9.8**
Occ.-Vault	35	6	2	0	**22.9**	0	0	0	**0.0**	0	0	0	**0.0**
Frontal	40	7	5	2	**35.0**	0	0	0	**0.0**	0	2	1	**7.5**
Parietal	66	9	7	4	**30.3**	2	0	0	**3.0**	1	4	0	**7.6**
Temporal	39	9	9	1	**48.7**	0	0	0	**0.0**	1	4	6	**28.2**

Table 10: Distribution of Abnormal Bone Matrix Across All Elements

Element	Total Number	Abnormal Matrix							
		Woven, Immature Bone				Cancellous Expansion			
		< 1/3	1/3 - 2/3	> 2/3	Total %	< 1/3	1/3 - 2/3	> 2/3	Total %
Humerus	58	0	0	1	**1.7**	0	0	0	**0.0**
Radius	51	0	0	0	**0.0**	0	0	1	**2.0**
Ulna	46	0	0	0	**0.0**	0	0	1	**2.2**
Femur	59	0	1	0	**1.7**	0	0	0	**0.0**
Tibia	50	1	0	1	**4.0**	0	0	0	**0.0**
Fibula	41	0	0	0	**0.0**	0	0	2	**4.9**
Scapula	54	0	0	0	**0.0**	0	0	0	**0.0**
Ilium	41	0	0	0	**0.0**	0	0	0	**0.0**
Rib 1	42	0	0	0	**0.0**	0	0	0	**0.0**
Rib 2	49	0	0	0	**0.0**	0	1	0	**2.0**
Ribs 3-10	451	0	0	0	**0.0**	0	1	0	**0.2**
Rib 11	31	0	0	0	**0.0**	0	1	0	**3.2**
Rib 12	13	0	0	0	**0.0**	0	0	0	**0.0**

Rib shape abnormalities

The prevalence of rib pathologies is listed in Table 11. Ribs 3-10 were most affected by both lateral ('J'-like) straightening (12.6%) and flared sternal ends (33.5%). These unspecialized ribs were the only ribs to exhibit beading or thickening of the sternal end (antero-posterior expansion different from the more superior-inferior expansion of 'flared' sternal ends). Healed microfractures of the sternal ends were observed in ribs 2, 11, and the 3-10 group, but microfractures of the rib neck were only seen in ribs 3-10. It is interesting to note that no eleventh ribs exhibited shape abnormalities, and that no pathologies were observed on any twelfth rib. Ribs that showed lateral straightening tended to also have a flared rib end (χ^2=195.957, p<0.0001), but having a flared sternal end was not necessarily predictive of lateral straightening.

Table 11: Frequencies of Rib Pathologies, including Shape Abnormalities and Fractures

Element	**Total Number**	**Flaring Sternal Ends**			**Lateral Straightening**			**Beading**		**Fracture**		
		Barely	**Clearly**	**Total %**	**Barely**	**Clearly**	**Total %**	**#**	**%**	**Sternal**	**Neck**	**Total %**
Rib 1	42	6	2	**19.0**	0	0	**0.0**	0	**0.0**	0	0	**0.0**
Rib 2	49	8	0	**16.3**	2	0	**4.1**	0	**0.0**	1	0	**2.0**
Ribs 3-10	451	113	38	**33.5**	43	14	**12.6**	49	**10.9**	8	2	**2.2**
Rib 11	31	0	0	**0.0**	0	0	**0.0**	0	**0.0**	1	0	**3.2**
Rib 12	13	0	0	**0.0**	0	0	**0.0**	0	**0.0**	0	0	**0.0**

CHAPTER FIVE

Discussion

Differential Diagnosis

The suite of skeletal lesions seen in the infants and children at Hisban contains a high frequency of diagnostic indicators of vitamin D and C deficiencies. However, other conditions leading to similar skeletal responses and patterning should be ruled out before making a definitive diagnosis. Attempting to identify diseases based on skeletal remains is not without limitations. Clinical identification of diseases such as scurvy and rickets relies on testing levels of these nutrients in the blood or urine (Hodges et al 1971; Spence and Serwint 2004). Scurvy in particular can be difficult to diagnose based on skeletal remains, as a number of diseases result in porosity of the bone, such as hypertrophic osteoarthropathy, Caffey's disease, treponemal disease (such as syphilis or yaws), chronic infections, leukemia, inflammatory meningitis, trauma, and anemia (Buckley 2000, Halcrow et al. 2014, Klaus 2014).

Hypertrophic osteoarthropathy (HOA) is a relatively rare disease which is characterized by digital clubbing and periostitis, usually in the long bones (Shakya et al. 2018). The disease can be inherited genetically or acquired and has been associated with a host of other diseases, such as tuberculosis, congenital heart disease, and cancers (Loyer et al 2019; Shakya et al. 2018). HOA results in subperiosteal abnormal new bone formation, which is thought to occur as a result of increased blood flow (Flohr et al 2018). However, this new bone manifests in sheets or 'shells' around the cortical bone, and the trabecular bone is unaffected (Flohr et al. 2018). This is dissimilar from the porosity in the Tell Hisban sample which exhibits thinning of the existing

cortical bone on the long bones. Furthermore, there is no evidence of clubbing in the phalanges from this collection (Perry, personal communication).

Caffey's disease, also called infantile cortical hyperostosis, is a rare genetic inflammatory disease typically affecting infants in the first few months of life (Kamoun-Goldrat & le Merrer 2008; Nistala et al 2014). It is caused by a mutation that results in defective Type I collagen, a major building block for all tissues in the body (Nistala et al. 2014). Caffey's disease exhibits as thickening of the cortical bone which affects most often the mandible (in over 70% of cases), as well as the clavicle, ribs, scapula, and ulna (Kamoun-Goldrat & le Merrer 2008; Nistala et al. 2014). Although abnormal periosteal bone was observed in the mandible, ribs, scapula, and ulna at Tell Hisban, it was not particularly thick. Furthermore, the swelling associated with Caffey's disease typically lasts only a few months, and most radiographic traces of the disease have been erased with regular remodeling by the age of 2 (US Library of Medicine Genetics Home Reference). The age range of this disease is not compatible with the population at Tell Hisban, as active lesions were observed in children aged 2-3 years.

Treponemal diseases are caused by spirochete bacterial infections, and include venereal syphilis, and the endemic treponematoses yaws, pinta, and bejel (Farnsworth & Rosen 2006). The infections manifest differently, varying largely with the climate inhabited by the afflicted individual, despite few genetic differences in the offending bacteria (Farnsworth & Rosen 2006). The primary characteristic of these diseases is lesions on the skin but can also include periosteal reactions in the bone as the disease progresses, as observed in yaws, bejel, and venereal syphilis (Antal et al. 2002). Yaws is found only in tropical areas, as the bacteria need constant warmth and humidity to survive (Antal et al. 2002), and thus is not the likely cause of the periosteal reactions observed in the Tell Hisban individuals. Conversely, bejel is found in hot, dry regions

and has long been documented among the bedouin (Farsworth & Rosen 2006; Hudson 1937). However, periosteal lesions associated with bejel are non-focal, manifesting as 'bumps' rather than the thinning, apparently porous cortical bone observed at Tell Hisban (Rothschild et al. 2006). Furthermore, the disease typically affects children aged 2 to 15 years, whereas abnormal new bone was observed on children under the age of 2 at Tell Hisban (Farnsworth & Rosen 2006). Venereal syphilis can affect any tissue and can be vertically transmitted through the placenta of an infected mother to her fetus (Cooper & Sanchez 2018). During pregnancy, syphilis can cause spontaneous abortions, premature delivery, stillbirth, and perinatal death (Cooper & Sanchez 2018). In the first two years of life, children with congenital syphilis may display periostitis, osteochondritis, and bone demineralization which may result in microfractures (Cooper & Sanchez 2018). Healing periostitis in congenital syphilis results in several layers of new bone around the diaphysis, similar to the bone thickening observed at Tell Hisban, but does not include the porosity and surrounding reactive bone observed in the population (Cooper & Sanchez 2018). Furthermore, dental deformations such as Mulberry Molars or Hutchinson's Incisors provide evidence for congenital syphilis in the archaeological record (Gaul & Grossschmidt 2014), but these were absent in this sample.

Acute leukemia in children can also result in skeleton involvement in 50-70% of cases (Resnick & Haghighi 1995). Leukemia interferes with normal bone growth and tumors may reach the periosteum. As a result, bone along the metaphysis may be grooved or porous with large vascular foramina (Ortner 2003). In 50% of cases, this porosity is accompanied by lymphoma which results in lesions elsewhere in the skeleton (Resnick & Haghighi 1995). The porosity observed at Tell Hisban occasionally has large vascular foramina, but the associated

new bone is not grooved. Furthermore, the sarcomatic lesions typical of lymphoma were not observed in any of the individuals of Tell Hisban.

Trauma to the periosteum can cause abnormal subperiosteal bone growth and porosity. In children, the periosteum is less securely attached to the bone and thus more prone to damage (McKibbin 1978). Healing cases of trauma manifest as new bone which has a porous appearance and delineates sharply from the surrounding bone (Ortner 2003). However, the porosity observed in healing trauma generally is larger in diameter than that observed in the Tell Hisban population. Furthermore, accidental trauma is unusual in children under the age of 2, and child abuse typically includes bone fractures, particularly of the torso, head, and spine, which were absent at Tell Hisban (Buckley & Whittle 2008).

The presence of porosity in the roof of the eye orbits (cribra orbitalia) has long been attributed to iron-deficiency anemia (Walker et al. 2009). Recent studies have sought to challenge a unilateral diagnosis of this pathology and propose methods for differentiating between anemic orbital porosity and scorbutic orbital porosity (Klaus 2017; Walker et al. 2009; Zuckerman et al. 2014). Unfortunately, each of these methods requires multiple cranial bones from the same individual (Klaus 2017; Zuckerman et al. 2014), an impossibility in the commingled pathological remains from Tell Hisban. Therefore, anemia cannot be ruled out as a possible cause for the porosity observed in the eye orbits of the individuals from Tell Hisban. However, as ascorbic acid is key for the absorption of iron, anemia has been observed as a secondary condition in scorbutic individuals (Clark et al. 1992; Cox 1969), which may have been the case at Tell Hisban as well.

Unfortunately, because of the commingled nature of the remains, it is not possible to analyze a suite of pathologies belonging to one individual. However, the frequencies of

pathologies observed among different elements from Tell Hisban can be compared to the population-wide frequencies reported in other studies of scurvy, as discussed below.

Few diseases result in bending deformities similar to those observed in the subadult long bones at Tell Hisban. Blount's disease is characterized by bowed legs, and early onset of the disease occurs before the age of 10 (Janoyer 2019). The disease is caused by incorrect development of the metaphysis and epiphysis along the medial border of the tibia, causing mediolaterally tilted growth plates (Janoyer 2019). If the bending deformities observed at Tell Hisban were the result of Blount's disease, it is unlikely that bending deformities would be observed in the upper limb bones as well as the ilium and mandible.

Metaphyseal dysplasia is a rare hereditary disease resulting in abnormal cortical bone growth that may resemble rickets. It can cause delayed mineralization, flaring or cupping in the long bone metaphyses and the sternal ends of ribs, widening of the diaphyses, and bowing in the long bones, particularly the proximal femur (Brickley and Ives 2008: 116). However, in this disease, the dense cortical bone replaces the more porous trabecular bone, practically eliminating the marrow space in long bones (Ortner 2003). This does not resemble the normal or even expanded trabecular bone observed in the individuals from Tell Hisban.

Hypertrophic osteoarthropathy, Caffey's disease, treponemal disease (such as syphilis or yaws), chronic infections, leukemia, and trauma may all cause porosity in the diaphyses of the long bones (Brickley and Ives 2008). However, as discussed above, these types of porosity are dissimilar to the pathologies in the Tell Hisban population. Porosity extending >10mm from the metaphyses on the long bones, although typically associated with rickets, may also be caused by scurvy (Brickley and Ives 2008). Because this study explores both these diseases, this porosity

can contribute to the broader discussion of ill health in this subadult population without pathologies being specifically ascribed to either scurvy or rickets.

As noted above, scurvy typically is diagnosed in skeletal remains using a suite of lesions observed in a single individual. However, the commingled nature of the remains at Hisban necessitates calculating the frequencies of each affected element and comparing these frequencies to those observed in other studies of complete skeletons where scurvy has a more definitive diagnosis. Scurvy preferentially affects certain skeletal elements over others, so one means of supporting the diagnosis of scurvy is to compare the involvement of these elements at Hisban with populations with confirmed scurvy. Porosity in the sphenoid, maxilla, and mandible are considered particularly diagnostic for scurvy. At Tell Hisban, 24% of sphenoid bones, 41% of maxillae, and 37% of mandibles exhibited some degree of porosity with a sphenoid:mandible:maxilla ratio of 1:1.8:2.25. A study of scorbutic porosity in Irish famine victims had a ratio of 1:0.4:0.9 (Geber and Murphy 2012). A study of subadults in a Roman population had a ratio of 1:2.4:3.4 (Schattman et al 2016). Skeletal indicators of both rickets and scurvy were observed in this population, which is similar to the individuals at Tell Hisban. This may explain why the ratio observed at Tell Hisban more closely resembles that from the Roman population than that of the Irish population, which exhibited only indicators of scurvy. Furthermore, this cooccurrence of the two metabolic diseases may partially explain the difference between the Tell Hisban and Roman ratios. Because scurvy and rickets interact, symptoms of the two disease may manifest differently during cooccurrence depending on a variety of factors including severity and order of onset.

The sphenoid:mandible:maxilla ratio observed in a Crusader population from southern Jordan (1:0.8:1.2) is moderately similar to that of Tell Hisban (1:1.8:2.25), where Rose and

Khwaleh (2012) attribute the scurvy-like lesions to destruction of folic acid due to UV-B radiation in the presumably light-skinned population of European descent. The relative amount of melanin in the Hisban population compared to the Crusader sample obviously is not known, although photographic evidence of bedouin from the region during this period suggest they have more melanin than western Europeans potentially involved in the Crusades. Although the ratio of sphenoid:mandible:maxilla porosity does not exactly match those observed and attributed to scurvy in the skeletal remains from other archaeological sites, this is likely due to differences in individual expression of the disease. At Tell Hisban, porous lesions were observed in bones diagnostic for scurvy, as well as those considered general indicators of scurvy, and the appearance of these lesions most closely matched the appearance of scorbutic lesions. Thus, scurvy is the most likely etiology for the porosity observed in the cranial elements in the population from Tell Hisban.

Rickets is a more straight-forward diagnosis because skeletal indicators are more unique, but the commingling of remains at Tell Hisban still necessitates a population-wide interpretation of pathological frequencies. At Tell Hisban, in the lower limb long bones, the bowing: metaphyseal flaring ratio was 1:1.18 and in the upper limb long bones, the ratio was 1:1.39. In a rachitic medieval population from France, this ratio was 1:0.75 in the legs and 1:4.88 in the arms (Schattmann et al 2016). The unusually high percentage of flaring in the upper limb bones in the Medieval French population may be due to the small sample size, or the possible co-occurrence of scurvy in the population. Other studies do not report metaphyseal flaring or abnormal diaphyseal width separately in the upper and lower limb bones. The population at Tell Hisban had an overall long bone ratio of bowing:metaphyseal flaring:abnormal diaphyseal width of 1:1.26:0.207. This is dissimilar to a Roman population which had a ratio of 1:1.49:1 and a

rachitic Italian Renaissance population which had a ratio of 1:0.798:0.23 (Brickley et al 2018; Giuffra et al 2013). The differences in these ratios is likely due to combining the frequencies of metaphyseal flaring and abnormal diaphyseal width of the upper and lower limb bones. If the individuals from the Roman or Italian populations were affected by rickets at a different age, such as after learning to walk, the frequencies of deformities in the upper limb bones would be smaller than those seen in the upper limb bones from Tell Hisban.

Although the ratios of shape deformities seen at Tell Hisban do not closely resemble those seen in rachitic individuals in other populations, these pathologies are still likely the result of rickets. Few other diseases cause both bending and metaphyseal flaring in the long bones, and those that do manifest differently than is seen in the subadult population at Tell Hisban.

When scurvy and rickets co-occur in an individual, one disease typically presents more strongly in the skeletal system than the other (Schattmann et al. 2016). There is no clear consensus on which disease takes precedence, but a variety of factors such as order of onset and relative severity seem to influence which disease becomes dominant (Schattmann et al. 2016). It is therefore possible that the effects of one metabolic disease have been obscured by those of the other disease in the individuals at Tell Hisban. As a result, the actual frequencies of both rickets and scurvy may be higher than suggested by the frequencies of pathology observed in the skeletal remains.

Causes of Scurvy at Hisban

Scurvy primarily results from a diet deficient in vitamin C but can be influenced by other factors such as genetics and lifestyle. As noted above, daily vitamin C requirements vary depending on age, and are generally higher for mothers. Traditionally in the Late Ottoman period, the bedouin in the Balqa' herded sheep, goats, and sometimes camels (Lewis 1987).

These animals often were kept for their milk rather than their meat and were only slaughtered in special cases (Abu-Saad et al 2001; Al-Ani 1980; Groen et al 1964). Seasonally, in the 19th century, the bedouin also cultivated melons and cucumbers (Smith 1896). The vitamin C content of these food sources is listed below (Table 12).

Table 12: Vitamin C content of foods known to be consumed by the 19th century bedouin of the Negev

Food	Ascorbic Acid	Source
Goat milk	0.89 mg/100g	Sawaya et al. 1984
Camel milk	37.4-47.9 mg/100g	Mohamed et al. 2005; Musaad et al. 2012
Cucumber	10 mg/100g	Stone 1937
Cantaloupe	53 mg/100g	Stone 1937
Watermelon	7 mg/100g	Stone 1937
Wheat	0.0 mg/100g	Brickley and Ives 2008

The high frequency of scorbutic lesions in the subadult population from Tell Hisban suggests that the increased dependence on nutritionally deficient wheat observed in modern bedouin peoples (Groen et al 1964) may have occurred prior to, or during the Late Ottoman era as the other foods traditionally consumed by the bedouin would have provided sufficient vitamin C to meet daily requirements for both infants and young children. Furthermore, because camel milk is rich in ascorbic acid, it may not have been an accessible resource for this group. Rather, it is likely they were dependent on the more widely available goat milk which is lower in ascorbic acid.

In newborns and breastfeeding infants, scurvy can be indicative of maternal malnutrition, as the mother provides all nourishment for her infant. During both pregnancy and breastfeeding, a woman's daily vitamin C requirements increase to compensate for the needs of the fetus or infant (Table 1). The fetus is prioritized, and vitamin C levels in a newborn are higher at the time of birth than in the mother (Lund and Kimble 1943; Teel, Burke, and Draper 1938). However, in guinea pigs, who like humans are unable to synthesize vitamin C, the mothers no longer prioritized the pups when ascorbic acid levels were low for a prolonged period, instead maintained their own serum levels of vitamin C (Schjoldager, Tveden-Nyborg, and Lykkesfeldt 2013). It is possible that similar methods of self-preservation are present in humans, resulting in normal maternal but low fetal ascorbic acid levels. In fact, congenital scurvy has been observed in a modern bedouin infant from Israel who was just 5 days old and being breastfed. Although the serum vitamin C levels of the mother were not tested, a skeletal survey of the mother showed no indicators of scurvy, and the doctors concluded that the child's disease was due to an inadequate supply of ascorbic acid in utero(Hirsch, Mogle, and Barkli 1976).

Scurvy could have been a result of weaning practices in bedouin society. Among modern bedouin in the Negev, boy babies are breastfed for 1-2 years, while girls are weaned earlier, at 6 months to a year (Abu-Rabia 2010). This is consistent with an isotopic study of skeletal remains from the Late Islamic bedouin cemetery at Khirbet al-Mudayna which found that weaning likely occurred between 6 months to a year of age (Gregoricka and Judd 2016), an age range which is consistent with increased mortality in the Tell Hisban sample. In modern bedouin populations in Israel, weaning children are fed primarily unleavened bread and hard-boiled eggs, as well as camel milk when available (Groen et al 1964). This is not inconsistant with isotopic data from Khirbet al-Mudayna, which reveal that subadults and adults were consuming similar quantities of

C_4 plants, perhaps wheat or barley (Gregoricka and Judd 2016). If weaning children were fed primarily cereals, they would be susceptible to scurvy as wheat has no vitamin C (Brickley and Ives 2008). This would be especially true for those infants from families without access to camel milk, which would provide at least some vitamin C.

Although scurvy results from vitamin C deficiency in the diet, a recent study found that only 17% of variance in vitamin C serum levels are explained by variation in vitamin C intake (Hampl, Taylor, and Johnston 2004). The additional variation can be explained by genetic variation and cultural practices, such as smoking. The primary genetic factor, as noted earlier, is differences in the human haptoglobin protein (Hp). Human haptoglobin binds to hemoglobin following hemolysis (rupture of red blood cells) to prevent oxidative damage to surrounding cells. Of the three main phenotypes, Hp1, Hp2-1, and Hp2-2, Hp2-2 individuals are more susceptible to developing scurvy because they require additional vitamin C to bind excess hemoglobin (Delanghe et al. 2007).

One study found that the Hp^1 allele frequency is 0.27 among modern Jordanians (Carter & Worwood 2007). Although the study does not specify whether bedouin were included in the analysis, a previous study found no significant difference between the haptoglobin frequencies in bedouin and non-bedouin Jordanian males (Saha & Banerjee 1986). Using the Hardy-Weinberg equation for allelic equilibrium ($p^2 +2pq+q^2=1$), approximately 50% of Jordanians are expected to have the Hp2-2 genotype (Hardy 1908; Weinberg 1908). However, these equations assume no migration between populations and that all individuals mate randomly, two conditions which are rarely satisfied in human populations (Hardy 1908; Weinberg 1908). As these conditions are violated, allelic frequencies in the population change each generation, so identifying Hp^2 frequency in the population from Tell Hisban would require genetic testing. However, if allelic

frequencies of the population at Tell Hisban resemble those of modern Jordanians, over half the population would have an increased risk of developing scurvy during periods of nutritional stress.

Causes of Rickets at Hisban

Like scurvy, rickets may occur due to dietary deficiencies, but may also result from insufficient levels of vitamin D, or genetic mutations that limit an individual's ability to absorb or process vitamin D, calcium, or phosphate. In living people, vitamin D levels are estimated using measurements of serum 25(OH)D (calcifediol) levels, although it has been argued that $1,25(OH)_2D$ (calcitriol) levels are more representative as they better reflect the more active form of vitamin D (Thacher et al 2010). There is no clear consensus for what serum levels constitute vitamin D deficiency, with values for adults falling between 11ng/mL and 20 ng/mL as suggested parameters (Khuri-Bulos et al 2014). In practice, the 20 ng/mL value may be more accurate as stunted osteoid mineralization has been observed in the bones of individuals with serum levels over 11 ng/mL (Holick et al. 2012). Vitamin D insufficiency, which is characterized by soft tissue changes but normal bone mineralization, has been defined in adults with thresholds between 30 ng/mL and 50 ng/mL (Batieha et al 2011; Holick et al. 2012). These serum level thresholds are likely to vary drastically between individuals, as factors such as sex and age influence daily vitamin D requirements (Holick et al. 2012). Indeed, requisite calcifediol levels for infants and children are lower, with vitamin D deficiency and insufficiency defined as serum levels <15 ng/mL and 15-20 ng/mL, respectively (Gordon et al. 2008; Khuri-Bulos et al. 2014).

In cases of calcium-deficient rickets, the body converts calcifediol into the more active form of vitamin D, calcitriol, whose primary function is delivering calcium from the gut to the bones. In living individuals, this deficiency is clearly represented by an unusually high ratio of

serum calcitriol to calcifediol (Fischer et al. 2000). Unfortunately, this imbalance is not similarly reflected in dry bone. However, historical sources and studies of modern bedouin in Israel suggest that calcium-deficiency could have caused the rachitic lesions observed in the subadults from Tell Hisban (Groen et al 1964; Shany et al 1976). As the 19th century bedouin were still herding sheep, goats, and occasionally camels, the primary source of calcium in the bedouin diet was probably milk or milk byproducts such as yogurt or cheese (Groen et al. 1964; Rogan 1999). Among modern bedouin, eggs were also reported to be consumed, but primarily by men (Groen et al 1964). It is unlikely that chicken eggs were important to the diet of 19th century bedouin peoples. Faunal analysis from Tell Hesban found that while the domestic chicken represented 80% of all bird bones, it comprised only 4% of all faunal remains, and most of these avian bones dated to the Mamluk period (1260-1456 CE) (Boessneck 1995).The calcium content for these foods are reported below (Table 13).

Table 13: Calcium content for different foods consumed by bedouin

Type of Milk	Calcium content	Source
Cow Milk	104-128 mg/100g	Gaucheron 2005
Camel Milk	114 mg/100g	Al-haj and Alkanhal 2010
Goat Milk	88-116 mg/100g	Sawaya et al. 1984
Egg (chicken)	50 mg/100g	USDA 2018

For children under the age of 10, recommended daily calcium intake is between 700-900mg, and daily intakes below 450mg are considered low (Black et al. 2002). Individuals with low calcium levels and high-phytate diets are at an increased risk of developing rickets because phytates bind preferentially to calcium, impeding transport of the element to the bone for

mineralization (Clements 1989; Konietzny and Griener 2003). One phytate molecule can bind up to five calcium molecules and effectively neutralize up to one third of daily dietary calcium (Selle, Cowieson, and Ravindran 2009). The unleavened bread ("rarif") consumed by modern bedouin is exceptionally high in phytates (125mg/100g) (Berlyne et al. 1973; Groen et al 1964).

During weaning, modern bedouin children are given rarif in large quantities, supplemented by eggs, goat milk, or camel milk, when available (Groen et al 1964). If the subadult individuals were not provided sufficient quantities of calcium-rich milk to counteract the phytase binding, calcium-deficiency could easily occur. In a clinical study in Iran, three adult individuals were given 2.5g of phytic acid daily for 28 days in the form of unleavened bread. As a result, two of the individuals experienced a decrease in serum calcium levels (Reinhold et al. 1973). Because juveniles are still experiencing skeletal growth, such a depletion of available calcium levels would likely impair bone mineralization and possibly even result in rickets.

Maternal health may impact the prevalence and severity of rickets in newborns and infants. The presence of rachitic bending deformities in the radius and ulna as well as metaphyseal flaring on the distal end of the radius at Hisban indicates that rickets was active in infants who were still crawling. Infants typically crawl in the first 7-13 months of life, though this exact age range varies slightly between populations (Touwen 1976). This unusually young age of onset may be indicative of congenital rickets resulting from maternal osteomalacia or malnutrition during pregnancy (Elidrissy 2016). In such instances, the symptoms of rickets tend to worsen or become more obvious in the infant in the following months as the child is breastfed (Anatoliotaki et al 2007; Elidrissy 2016 Gordon et al 2008; Kensarah et al 2016). In a cohort of infants in the United Arab Emirates, 92% of the individuals born with rickets were found to be deficient in vitamin D. In addition, 97% of mothers of vitamin D-deficient rachitic infants were

likewise found to be deficient in vitamin D, likely due to inadequate UV-B exposure and insufficient vitamin D supplements (Dawodu et al. 2005). However, a clinical study from Israel found that while bedouin women had lower levels of calcitriol than Jewish women, they did not exhibit symptoms of osteomalacia (Shany et al 1976). This is consistent with the absence of pelvic deformities indicative of osteomalacia in the adult females from Tell Hisban (Brickley et al. 2008; Perry and Edwards 2018). As the presence of bending deformities and metaphyseal flaring in younger individuals from Tell Hisban is not inconsistent with congenital rickets, it was likely caused by maternal vitamin D insufficiency (soft tissue changes but normal bone mineralization) rather than deficiency (impaired bone mineralization) as bone mineralization of adult female skeletal remains do not appear affected.

It is possible that the rachitic lesions observed in this population were caused by inadequate subadult or maternal exposure to UV radiation due to clothing or other cultural factors. Bedouin women are known to weave textiles, often from the wool of their sheep, interspersed with goat or camel hairs and then sometimes dyed (Abdel-Kareem 2010; Shamir 1995). In the 19th century, these woolen textiles were used to make the black tents in which the bedouin reside (Smith 1896). Black wool attenuates 98.6% of UV-B radiation, so any individuals residing in a tent would be effectively shielded from sunlight (Matsuoka et al. 1992). Contemporaneously, these woolen textiles were also used in the manufacture of belts worn by bedouin women, but not necessarily other garments (Abdel-Kareem 2010). Historical documents report that bedouin women around Hisban wore long, loose blue garments made of linen (Smith 1896). Flaxen textiles have also been found in a bedouin cemetery at a nearby Late Islamic site, although these may be identified as burial shrouds, as they were not dyed (Müller 2003). Undyed linen has been found to block 93.3-95.8% of UV radiation, while linen which has been dyed

navy blue is even more effective, blocking over 97.5% of UV radiation (Das 2010; Tarbuk et al. 2010).

Although the bedouin garments would have blocked the majority of UV radiation, historical sources report that bedouin women sometimes walked outside with their faces uncovered, thus exposing the skin to UV-B radiation (Burckhardt 1822; Smith 1896). This limited exposure to UV radiation may have resulted in vitamin D insufficiency which would be evident in the soft tissue and blood serum levels but not the bones. During prolonged periods spent indoors, subclinical vitamin D deficiency can progress to active rickets or osteomalacia, as observed in patients who have been hospitalized for extensive periods of time (Matthews et al. 2012). In the 19th century, bedouin women reportedly traditionally stayed indoors for 40 days postpartum (Abu-Rabia 2010) potentially exacerbating already low vitamin D levels due to protection from UVB radiation due to clothing or their domicile. However, the mother's body typically prioritizes the infant, redirecting nutrients to the fetus during pregnancy and, later the breastmilk (Schjoldager, Tveden-Nyborg, and Lykkesfeldt 2013). Therefore the mother would have to be significantly deficient in vitamin D or neonates and nursing infants to express rickets or scurvy, as has been observed in clinical cases (Dawodu et al 2005; Mohapatra et al 2003).

Finally, the rachitic lesions observed in the subadults from Tell Hisban may not be the result of cultural or dietary factors but rather genetic defects in the absorption and processing of phosphate, which along with calcium is essential for maintaining skeletal rigidity. In X-linked dominant rickets, any individual possessing the gene (both hemizygous males and heterozygous females) express severe skeletal deformities (Bitzan and Goodyer 2017). Thus, if the bending deformities in this skeletal assemblage were the result of X-linked dominant rickets, the adult remains would be expected to show similar deformities (indicating an affected individual), or no

evidence of deformities at all (indicating a healthy individual). However, during previous analysis of the adult remains, no upper limb bones and only 2% of adult lower limb bones exhibited abnormal curvature indicating childhood rickets (Perry and Edwards 2018).

If the population were suffering from the autosomal recessive Hereditary Hypophosphatemic Rickets with Hypercalcemia (HHRH), only individuals who are homozygous recessive would show the extensive bending deformities resulting from rickets. Provided that the mother is healthy, infants with HHRH are born with normally mineralized skeletons, as nutrient absorption in utero bypasses the defunct intestines (Bitzan & Goodyer 2019). Within a month after birth, the serum phosphate level of the fetus will fall below normal and skeletal changes will begin within six months (Bitzan & Goodyer 2019). However, there is evidence that individuals who are heterozygous for the trait suffer a milder form of the disease and have an impaired ability to absorb phosphate (Tieder et al 1987). These heterozygous individuals would then suffer from subclinical rickets which would only manifest in the skeleton during times of extreme nutritional stress. This pattern of disease expression, with homozygous recessive infants suffering severe rickets within the first years of life and perishing, while some older (presumably heterozygous) individuals suffering only mildly from the disease does not conflict with the morbidity patterns observed in the skeletal assemblage from Tell Hisban and thus cannot be excluded as a possible cause for the rachitic lesions.

The frequency of rickets-related skeletal changes in children exceeds that seen at the site prior to the Late Islamic period or observed elsewhere in the region during the Late Islamic period. Evidence of metabolic disease was not observed in the skeletal remains from the Early Roman to Late Byzantine period at Tell Hisban (Grauer and Armelagos 1998). Additionally, at Khirbet al-Mudayna, a site 45km southwest of Amman, the Late Islamic-era bedouin skeletal

collection showed indicators of cribra orbitalia and porotic hyperostosis, but no scurvy or rickets (Sadvari 2009). Other Late Islamic-era bedouin skeleton collections from Jordan and Israel likewise show no indicators of either metabolic disease (Eakins 1993; Mitchell 2006). Possible cases of scurvy were observed in 50% of juvenile individuals in a 12th century skeletal assemblage from Al-Wu'ayra in southern Jordan (Rose and Khwaleh 2012), and in 84% of juveniles from a Late Ottoman era collection from the coastal plain of Israel (Smith and Horowitz 2009), but a differential diagnosis either was not applied (Smith and Horowitz 2009) or other etiologies such as folic acid deficiency could not be ruled out (Rose and Khwaleh 2012). In Late Islamic bedouin burials from Caesarea, Israel, 5.3% of tibiae had bending deformities but no other bones were affected. Furthermore, the study does not specify if the affected tibiae belonged to adults or subadults, and thus the possible extent of rickets or osteomalacia cannot be explored further (Smith and Zegerson 1999). Finally, bowing, diaphysis widening, and metaphyseal flaring were observed at two Late Ottoman-era bedouin cemeteries in Israel, but these pathologies were attributed to infection and insufficient data is provided to make a differential diagnosis (Goldstein, Arensburg, and Nathan 1976). The skeletal remains at Tell Hisban represents a group with a temporally and spatially-unique rate of scurvy and rickets, suggesting their access to dietary sources or cultural practices were unique for the period. It is possible that these individuals were economically marginalized and did not always have access to high-quality and nutritious foods, exacerbated by cultural practices inhibiting UV-B radiation especially for pregnant or nursing mothers and infants. The high rates of metabolic disease at Hisban coincides with the implementation of new land-use laws in the region by the Ottoman empire, which may have been a factor in this group's marginalization.

Establishing Identity

As noted earlier, the identity of the individuals buried in the partially collapsed room on Tell Hisban remains unclear. However, the historical background research necessary for this project in addition to the research results may provide some insight into the tribal identity of these individuals. The Ottoman empire kept meticulous records of taxes and land ownership, but this either rarely included specific information about pastoral nomadic inhabitants of the land and their burial practices, or the documents have yet to be translated from Ottoman Turkish. Historical travelers report that two tribes occupied the lands surrounding Tell Hisban at the start of the 19th century (Russell 1989). The smaller of the two, the ‘Ajarma, was already practicing small scale cultivation of some lands by this time (Abujaber 1989). The larger tribe, the ‘Adwan, adopted this practice later, but likewise cultivated crops on small individual plots of land (Jaussen 1908; Rogan 1999). The Beni Sakhr gained prominence in the area later, and primarily hired *fellahin* to grow cereals on large tribal plantations (Rogan 1999). A majority of the Beni Sakhr continued their nomadic herding lifestyle, only residing near villages in the spring and summer months (Abujaber 1989).

In addition to the historical presence of the tribes at Hisban during the late 19th century, access to resources by these three tribes may highlight their identity in light of the metabolic conditions from which they suffered. Although there is diversity within tribes, generally the Beni Sakhr were known as camel herders, a trait not shared by the ‘Adwan or the ‘Ajarma who therefore would not have had regular access to vitamin C-rich camel milk. Furthermore, historical sources describe the Beni Sakhr as having a stronger presence in the area to the south of Hisban (Burckhardt 1822). Thus, the individuals buried at Hisban likely were not part of the Beni Sakhr tribe. Rather, it is likely that the population interred at Tell Hisban is associated with

either the ‘Ajarma or the Adwan, both of whom are mentioned in historical traveler’s accounts of the region during this time (Conder 1889; Smith 1896).

Both the ‘Ajarma and ’Adwan had access to wheat, which if consumed as the high-phytase unleavened bread *rarif*, particularly at levels seen today, would have resulted in developing calcium-deficient rickets. It is not clear whether other cultural practices of the ‘Ajarma or ‘Adwan, such as clothing type and time spent exposed to UV radiation, would have resulted in one group suffering from scorbutic and rachitic lesions more than another as observed in the skeletal remains from Tell Hisban. However, of the two, the ‘Ajarma are the only tribe whose descendants still live in the area around Tell Hisban, although they do not have memory of their tribal use of Tell Hisban of a place of burial cemetery in the area (Walker, personal communication).

Summary

The porosity observed in the sphenoid, maxilla, and mandible at Hisban likely results from scurvy, as porosity resulting from other diseases manifest differently and in different location. Likewise, the combination of bending deformities and metaphyseal flaring observed in the long bones is unique to rickets. However, there are several possible causes of these two metabolic diseases. Scurvy results from a diet deficient in vitamin C and could be linked to the increased dependence of bedouin on nutritionally null wheat (Groen et al 1964). It could also have been due to a dietary deficiency of the infant, perhaps during the weaning process, or a congenital deficiency which developed in utero as a result of maternal vitamin C deficiency.

Rickets may have been caused by a juvenile or maternal deficiency in vitamin D or calcium, or by a genetic disorder impairing an individual’s ability to absorb phosphate. Vitamin

D deficiency is caused by limited exposure to UV-B rays, which would have been possible considering the attenuating properties of both woolen and flaxen textiles. Furthermore, vitamin D deficiency remains an issue among modern Jordanians due to inadequate exposure to UV-B radiation resulting from a combination of long clothing and spending a large amount of time indoors (Batieha et al 2011; Khuri-Bulos et al 2014). It is possible that bedouin women and consequently their infant children at Tell Hisban were likewise vitamin D deficient, as it is known that they wore long sleeves and stayed inside 40 days postpartum (Abu-Rabia 2010; Smith 1896). Rickets may also have resulted from a calcium deficiency which impeded the mineralization of bones. This calcium deficiency may have been exacerbated by a diet of high-phytic breads, such as *rarif*, which impede the absorption of calcium. Both these dietary deficiencies could have developed in the infant, likely during weaning, or may have been congenital, resulting from a uterine environment insufficient in either calcium or vitamin D. Finally, rickets may have resulted from genetic diseases, such as Hereditary Hypophosphatemic Rickets with Hypercalciuria, in which an individual is unable absorb phosphate and instead scavenges it from the skeleton, resulting in bone demineralization.

Finally, it is possible that the co-occurrence of rickets and scurvy in this population altered the expression of both diseases. Because rickets and scurvy interfere with each other, it is possible that the true occurrence of both diseases in this population is higher than indicated by skeletal lesions.

CHAPTER SIX

Conclusion

The individuals from Tell Hisban were interred during a period of economic and political change. As the Ottoman Empire adopted individual land ownership laws at the end of the 19th century, some individuals, especially those of those from the less wealthy tribes, were marginalized. As a result, members of these tribes could have lost access to a previous wide array of food sources, which could have put some individuals, particularly young children, at an increased risk of developing metabolic diseases, specifically scurvy and rickets.

Scurvy is the result of a diet deficient in vitamin C, and typically results in characteristic porosity and abnormal new bone formation in juvenile cranial bones, most diagnostically, the sphenoid, mandible, and maxilla, as well as portions of the postcranial skeleton. In the individuals from Tell Hisban, a possible cause for scurvy was an over-reliance on wheat and other cereals, which have little nutritional value. This would have been especially problematic for new and expectant mothers as the daily vitamin C recommendation increases for pregnant and breastfeeding women. Failing to meet these daily values for extended periods of time can result in infantile scurvy.

Rickets is most evident in the skeleton as bending deformities, diaphyseal thickening, and metaphyseal flaring in the long bones, and may result from several factors. In vitamin D deficient rickets, individuals have inadequate exposure to UV-B radiation, and are unable to synthesize vitamin D. This may have been the case for the individuals at Tell Hisban as dark woolen tents and linen clothing attenuate over 95% of UV rays, and inadequate UV-B exposure is a problem even in modern Jordan (Batieha et al. 2010). Additionally, rickets may result from a diet which is low in calcium and high in calcium-binding phytates, which consequently impairs

the mineralization of bones. Modern bedouin in Jordan and Israel have a high phytate diet in the form of unleavened bread, and likely experienced a reduction in herd size, and thus access to calcium-rich milk from goats, sheep, or even camels. Together, this high phytate and low calcium diet can result in nutritional rickets. Finally, rickets can also result from genetic diseases which impair the body's ability to process calcium or phosphate, both vital for proper bone mineralization. Most of these diseases are dominant and result in osteomalacia in the adult skeletons. However, the autosomal recessive Hereditary Hypophosphatemic Rickets with Hypercalciuria can result in increased mortality for homozygous recessive juveniles while leaving adults largely unaffected, which is not inconsistent with the Tell Hisban sample.

When considering the skeletal evidence for disease in a past population, it is important to remember that the morbidity expressed skeletally is not necessarily representative of the overall population morbidity. Morbidity and mortality statistics assume that the population demographics remain stationary, that there is no migration and that birth and death rates remain constant and equal, which is impractical for a human population (Wood et al. 1992). Furthermore, the individuals represented in the skeletal assemblage are only those who died with visible indicators of the disease; it does not include all individuals who are at risk, who contracted the disease and recovered, or who died before the effects of the disease were visible in their skeletons (Wood et al. 1992). Finally, the skeletal assemblage does not inform on "hidden heterogeneity", or the variations in frailty and susceptibility to a disease within population subgroups (Wood et al. 1992).

Future Research

To better understand the presence of metabolic disease in this subadult population, future research should consider indicators of active or healed scurvy and rickets in the adult remains

from Tell Hesban, as these individuals survived the probable nutritional stressors which increased the mortality in individuals under the age of 2 years.

Rickets interferes with the mineralization of all bone, including teeth. A recent study proposes that periods of vitamin D deficiency are recorded as irregular growth in the teeth (D'Ortenzio 2016). Using this method on teeth from Tell Hisban, future research could identify individuals who have suffered periods of vitamin D deficiency in childhood even when the skeletal evidence of rickets has been erased through time. Additionally, genetic testing could reveal predispositions for developing rickets. The presence of two F alleles at the *Fok1* loci can indicate a predisposition to calcium-deficient rickets (Pettifor et al. 2018). Mutations at the *SLC34A3* allele would reveal that the relatively high frequency of rickets observed in subadult individuals at Tell Hesban is the result of Hereditary Hypophosphatemic Rickets with Hypercalciuria (HHRH), rather than nutritional or cultural influences (Lorenz-Depiereux 2006).

Assessing scurvy in adult skeletons is more difficult than in subadult skeletons. Some studies (Maat 1982; Van der Merwe et al. 2010) have proposed a collection of criteria for identifying scurvy and scorbutic lesions in adult skeletons, but these studies have relied on the added presence of lesions across multiple parts of the body, which would be difficult to assess in the adults of this commingled sample. Currently there are no other methods for identifying healed scurvy in adult skeletal remains. However, genetic testing for the Hp2-2 genotype could reveal an increased risk for developing scurvy among some individuals at Tell Hesban (Carter & Worwood 2007).

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APPENDIX: DATA COLLECTION KEY

CRANIUM

Porosity

Location

3.1.1 (periosteal)

3.1.2 (Cortex/Trabeculae/Diploe)

3.1.3 (Endosteal surface)

3.1.4 (Mixed: 1&2)

3.1.5 (Mixed: 2&3)

3.1.6 (Mixed: 1, 2, &3)

Extent

3.2.1 (<1/3)

3.2.2 (1/3-2/3)

3.2.3 (>2/3)

Focal

Is it 3.3.5 (10+ foci)?

Size

Is it 3.4.8 (<1mm)?

Abnormal Bone Formation

Periosteal?

4.1.1 (Reactive woven bone)

4.1.2 (Sclerotic reaction)

4.1.3 (Both woven and sclerotic)

Abnormal Matrix?

4.5.1 (Deposition of woven, immature bone)

4.5.2 (Cancellous expansion)

4.5.3 (Trabecular coarsening)

4.5.4 (Small spicules)

Extent

4.6.1 (<1/3)

4.6.2 (1/3-2/3)

4.6.3 (>1/3)

Shape Abnormalities

1.4.1 (barely discernible)

1.4.2 (clearly discernible)

POSTCRANIA

Do same Extent/Focal/Size for each!!!

Ilium

1.3.7 (exaggerated medio/lateral curvature)

1.4.1 or 1.4.2

Porosity along external and internal ilium? Y/N

Scapula

Porosity on supraspinous/infraspinous fossae? Y/N

Long Bones

1.1.0 (Bowing)

1.3.1 (Flaring metaphyses)

1.3.2 (Uniformly abnormally wide)

1.3.8 (Cupping at growth plate)

1.3.9 (Growth plate tilting)

1.3.10 (Femoral coxa vara)

1.4.1 or 1.4.2

Porosity >5-10mm superiorly along metaphysis? Y/N

Porosity along concave side of bowed bone (new bone could be irregular)? Y/N

Ribs

1.2.0 (Angulated/alteration in the rib neck angle)

1.3.5 (Flaring sternal ends)

1.3.6 (Lateral straightening)

Beading? Y/N

Porosity along pleural border and near sternal end? Y/N

Costochondral fracture? Y/N

5.1.1 (Complete)

5.1.2 (Partial)

5.1.3 (Simple)

Fracture Sequelae

5.4.1 (Callus formation, woven bone only)

5.4.2 (Callus formation, sclerotic reaction)

5.4.3 (Healing/obliteration of cranial fracture)

Epiphyseal Plate Porosity

ALSO: Periosteal/Abnormal Matrix/Extent

Diffuse thinning at growth plate? Y/N

3.8.1 (Normal)

3.8.2 (Fine-grained and rough)

3.8.3 (Rough with pitting)

3.8.4 (Extreme pitting & porosity)

3.8.5 (Fraying at growth plate)