THE CO-OCCURRENCE OF SCURVY AND RICKETS IN 16TH TO 18TH CENTURY SKELETAL MATERIAL FROM DOUAI, FRANCE.

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Abstract

Disease is not a unique or singular phenomenon. The medical literature contains multiple reports discussing disease interactions and co-occurrence which remains an important issue. Despite this, there has been no systematic investigation of disease co-occurrence in paleopathology. This thesis will begin to fill the gap by producing a detailed analysis of the pathological indicators for scurvy, rickets, and their co-occurrence, focussing on features of co-occurrence and their identification.

The Collégiale Saint-Amé collection from 16th to 18th century Douai, France includes 48 individuals ranging from fetal to five years of age. Previous research indicated a large number of potential cases of scurvy and rickets in the juveniles (Devriendt et al. 2010). The current study identified 12 cases of possible co-occurrence based on macroscopic, radiographic, and microscopic techniques; biocultural and historical data supported disease presence.

Macroscopic results indicate that lesions associated with scurvy are identifiable and the vascular system is not known to be directly affected by rickets. Rickets features are present but changes are subtle and reduced in prevalence. Radiographs demonstrate features of both diseases but the presence or absence of the line of Fraenkel, a scurvy feature, was useful in identifying the likely dominant disease process. Diagenetic change significantly impacted microscopic investigations but the technique provided some supporting evidence for the presence of rickets.

The results clearly demonstrate that cases of co-occurrence of scurvy and rickets are present and identifiable in the archaeological record. Important factors for recognition include the sequence in which conditions develop and duration of illness. Presently only cases with moderately or better developed features of both scurvy and rickets can be identified. Use of multiple techniques was critical to observe subtle changes and build a case for disease presence. Further research on co-occurrence of any diseases is encouraged to create a fuller understanding of past disease.

Acknowledgements

I would like to thank Dr. Megan Brickley for helping me discover the interesting and fascinating topic of disease co-occurrence, for her assistance in the development of the project, and providing invaluable mentorship. Additional thanks are owed for inviting me to participate on other ongoing vitamin D deficiency projects which furthered my understanding of rickets and allowed me to visit interesting cities across Europe. I would also like to thank my committee member Dr. Hendrik Poinar for his contribution to the development of the research project and investigation into collagen use for diagnosing scurvy.

I cannot express the thanks I owe the members of the CAD, including Pierre Demolon, Benoît Bertrand, William Devriendt and Sophie Vatteoni, who graciously allowed me access to their collection and their facilities, as well as use of their data for the current research project. Many thanks to Dr. Snezana Popovic who assisted with the interpretation of the histology thin sections. I am grateful for the support from Bonnie Kahlon and Leila Watamaniuk who patiently taught me and assisted me in all my lab work.

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Declaration of Academic Achievement

The research contained in this dissertation was completed by Annabelle Schattmann, under the supervision of Dr. Megan Brickley and Dr. Hendrik Poinar. Research questions and methodology was developed in consultation with Dr. Brickley and Dr. Poinar. Permission to access to the skeletal collection was obtained from Pierre Demolon, Benoît Bertrand, William Devriendt, and Sophie Vatteoni. Data collection and analysis was independently completed by Annabelle Schattmann. Previously collected data on limb bone length for age estimates and a collection of radiograph images of the bones produced by Benoît Bertrand, William Devriendt, and Sophie Vatteoni was made available for re-analysis. The results of the re-analysis and some radiographs images are included in the text with proper citation and acknowledgement. Dr. Brickley graciously provided additionally funding for this project.

Chapter 1: Introduction

Paleopathology has a long standing history exploring a wide range of topics and diseases with particular attention to the identification of disease and understanding the interplay of disease, people, environment and culture in the past. One area which has received little attention is the co-occurrence of disease.

Co-occurrence of disease can be defined by the simultaneous presence of multiple diseases acting upon a single person. Disease weakens the body by inhibiting the optimal function of its various systems. Interaction with a variety of factors including biology, environment and culture can increase an individual's risk of acquiring a second condition. For example, in a weakened state the body cannot efficiently ward off other diseases and the affected individual may not be able to acquire enough essential nutrients for proper function. The boundaries and potential range of interaction between the two or more diseases is very variable and an important point of interest (e.g., Singer 2009). The interaction between the diseases can be minimal or non-existent, antagonistic or even mutualistic. The phenomenon of co-occurrence is well documented in the medical field and remains an important issue today, particularly with regards to frail members of society who are at greater risk of developing co-occurrences, the associated costs, managing chronic conditions, and increased mortality (e.g., Meigs 2003; Valderas et al. 2007; Lee et al. 2009).

A few cases of co-occurrence have been reported in paleopathology. For example, paleomolecular techniques have found DNA evidence of tuberculosis with *Leishmania infantum*, leprosy and hepatitis B (Donoghue et al. 2005; Matheson et al. 2009; Bianucci et al. 2012). Cases of possible infantile scurvy and rickets co-occurrence have also been identified using skeletal material (e.g., Devriendt et al. 2010; Lewis 2010; Redfern 2012; Geber & Murphy 2012; Klaus 2014). However, a systematic investigation on the topic of co-occurrence has not yet been pursued. The current project will attempt to begin addressing this gap in the knowledge. Scurvy and rickets co-occurrence was selected as the topic for further paleopathological analysis as scurvy (vitamin C deficiency) and rickets (vitamin D deficiency) are metabolic diseases which impact the skeleton and are well documented to co-occur in the clinical literature on infants and children by researchers from the 1800s onwards (e.g., Cheadle 1878; Barlow 1883; Follis et al. 1940; Bromer & Harvey 1948; Fouron & Chichoine 1962).

The aims of this thesis are to conduct a preliminary investigation on the co-occurrence of disease, specifically scurvy and rickets, from a paleopathological perspective to build a more realistic and nuanced perspective of disease in the past. The thesis will answer two questions. First, can cases of scurvy and rickets co-occurrence be identified in paleopathology? Second, how might co-occurrence affect the appearance of typical scorbutic and rachitic features used to identify the diseases in paleopathology? To answer

the central questions of this thesis, an archaeological collection containing infant and children remains with previously reported cases of scurvy and rickets was identified. The Collégiale Saint-Amé collection in Douai, France was selected and 48 juveniles were made available for study. The juveniles are dated to the early modern period, between 16th and 18th century, and range from fetal to five years of age at death (Devriendt et al. 2010). Numerous techniques, including macroscopy (visual examination), radiology (x-ray) and microscopy (histological thin sections and scanning electron microscopy) were used to identify the presence of disease and suggest possible co-occurrence of scurvy and rickets.

The medical literature suggests that the expression of disease is very variable in cases of scurvy and rickets co-occurrence due to the intricate and sometimes inhibitory relationship of the diseases (Cheadle 1878; Barlow 1883; Bromer & Harvey 1948). However, in most cases, scurvy was observed to supervene onto rickets. In reported cases, the features of scurvy are often clear while those of rickets are either well developed or nearly unobservable. Identification of co-occurrence should therefore be possible if features of both diseases are expressed. The observed paleopathological cases should also have a disease expression consistent with that observed in medical cases. Macroscopically, scorbutic and rachitic features should be observable, though in some cases the features of rickets may not be very clear (Cheadle 1878; Bromer & Harvey 1948). Scurvy is expressed by haemorrhages and inflammation which does not always result in bony changes. Ortner et al. (1998; 1999; 2001) have linked porosity with inflammation and haemorrhaging. Radiographically, some reports suggest features of rickets are clearly visible while other suggests scurvy should be present. Thus, features of both diseases should be present but results are variable (Bromer & Harvey 1948; Fouron & Chichoine 1962; Valentini et al. 2011). Many non-diagnostic features are shared between both conditions and high prevalences of these features is expected (Bromer & Harvey 1948). Microscopically, possible evidence of rickets may be observed in cases where the disease had a chance to develop. Any major changes to the typical expression of singular disease are unlikely but the identification of a features which had a high prevalence in cases of co-occurrence would be very interesting and helpful to identify future cases of scurvy and rickets co-occurrence.

The thesis is organised into eight chapters with additional appendices. The first chapter is the introduction. The subsequent three chapters contain important background information about the diseases with near exclusive focus on juvenile concerns. Chapter 2 explores vitamin C deficiency by defining infantile scurvy, briefly discussing the biochemical role of vitamin C and previous paleopathological research on infantile scurvy. A comprehensive description defining the expected features of scurvy in paleopathology from a macroscopic, radiographic, and microscopic perspective, with supporting information from clinical literature, is also provided. Chapter 3 uses the same format as Chapter 2 to explore vitamin D and rickets. Chapter 4 provides a literature review on the study of co-occurrence in paleopathology and medical literature on scurvy

and rickets co-occurrence from a clinical, radiographic, and microscopic perspective. Additionally, previous observations of scurvy and rickets co-occurrence in paleopathology and expectations of co-occurrence, specifically regarding presentation of disease, are explored. Chapter 5 provides site background on Saint-Amé and a thorough explanation of techniques used to age, sample, score preservation, and assess disease presence in the juveniles from Saint-Amé for the current research. Results of the techniques outlined in Chapter 5 are available in Chapter 6. Answers to the central questions of this thesis and subsequent discussion of their meanings are explored in Chapter 7. Limitations and issues affecting research are discussed. Additional biocultural and socioeconomic contextual evidence is detailed to support or refute the suggested presence of scurvy and rickets in Douai during the early modern period. Chapter 8 provides final conclusions on the results of the study, insights and provides suggestions for further research on the topic of disease co-occurrence.

Chapter 2: Background - Infantile Scurvy

2.1. Introduction

In this chapter, infantile scurvy will be defined. The clinical, radiographic, and microscopic appearance of the disease as documented and understood currently in paleopathology will be explored with supporting information from the clinical literature. The focus is exclusively on infantile and childhood scurvy as the current research only considers juvenile remains. The history of scurvy, the biochemical effects of vitamin C and its association with scorbutic symptoms will be briefly discussed.

2.2. Scurvy

Scurvy is the disease caused by a deficiency in vitamin C. Vitamin C, also called L-ascorbic acid or ascorbate, is a water soluble vitamin and an essential nutrient. The majority of animals can synthesise this vitamin. However, selection or neutral mutation processes have caused humans and some animals to lose this ability, requiring them to obtain vitamin C from their diet. Such species include guinea pigs, haplorhine primates (all primates except lemurs and lorises), teleostei fish (e.g., salmon and trout), most bat species, and some species of passerine birds (e.g., song birds) (Pauling 1970; Cueto et al. 2000; Drouin et al. 2011; Lachapelle & Drouin 2011). Vitamin C rich foods include fresh fruits, vegetables, and human breast milk, with small amounts in raw liver (Ingalls et al. 1938; Lee & Kader 2000; USDA 2014). A deficiency of this vitamin is caused by a deficient diet and/or poor nutrient absorption.

As a disease of essential nutrient deficiency, scurvy could occur at any point in the past as long as the conditions were conducive. Some of the earliest documented cases of disease attributed to scurvy come from ancient Greek and Roman authors Hippocrates, Galen, and Pliny (Still 1935). The vast majority of the early medical literature exclusively considers adults cases, particularly in relation to sailors and maritime voyages. In these contexts, scurvy was a common occurrence due to the lack of vitamin C rich foods in a sailor's diet and this issue was of great concern to the world powers of the 18th and 19th centuries. For example, James Lind's (1753) famous treatise on the curative properties of orange and lemon juice stems from these concerns. However, the disease does not discriminate on the basis of age.

Infantile scurvy, also known as Möller-Barlow's disease, is the focus of the present research on scurvy. It was first documented and discussed in the medical literature of the 16th and 17th centuries (e.g., Glisson 1650). The classic reference works were produced later by Möller (1859), Cheadle (1878), and Barlow (1883). The disease is identical in chemical and biology to the adult condition but the signs and symptoms are different as children are undergoing growth. Infantile scurvy is presently considered to be a rare

condition although certain periods in the past witnessed significant increases in the number of cases.

The risk of developing scurvy is variable and dependent upon socio-cultural, environmental, and physiological factors. Access to vitamin C rich food sources is the most important factor. Vitamin C sources for infants include breast milk and vitamin C rich formula. Prior to the development of formulas in 1800s and early 1900s, European infant feeding alternatives and weaning foods included pap or panado which were likely poor in vitamin C as they were composed of boiled milk or water, flour or bread with some added sugar (Drake 1930; Senoir 1983). Animal milk contains some vitamin C which would have helped prevent the development of scurvy (Grewar 1965; USDA 2014) but the vitamin can be leached from the originating product and destroyed if heated to high temperatures and/or cooked for extended periods of time (e.g., Zilva 1935; Rumm-Kreuter & Demmel 1990). Infant formulas became available during the industrial period but early recipes were deficient in vitamin C (Still 1935, Cheadle 1878). Infantile scurvy incidence increased at the time and researchers noted that cases of scurvy were rare in breast fed infants but nearly always occurred in those fed formula (Hess 1920). Early formula was an expensive milk replacement but the product was considered novel and purchased by individuals of higher social standing for their children, putting them at a greater risk for developing scurvy. During and after weaning, foods high in vitamin C are critical. Prior to the introduction of the potato and good access to fresh fruits and vegetables, such as the "market gardens" of 17th and 18th centuries England (Lomax 1986), foods high in vitamin C would have been difficult to access for the urban populations and to store for long winters (Lewis 2007). Cheadle (1878) remarks that due to mass production and easy storage of potatoes, which became widely available by late 18th century (Still 1935), the children of the poor were rarely affected by scurvy. Upper class children gained some protection as their families could afford wet nurses, sometimes in-house (Senoir 1983). Diets including vegetables and animal milk would have been available on a regular basis to supplement the diets of upper class families and provide some protection against scurvy (Cheadle 1878). However, animal milk is known to contribute to the development of scurvy if given as the sole food to a child (Grewar 1965; Brickley & Ives 2008; Saxholt et al. 2009; USDA. 2014). The children of the middle class were at increased risk as the families elected to not consume potatoes due to class standing and could not afford much milk. Storage of foods for extended periods of time also reduces the content of vitamin C (Lee & Kader 2000). Lastly, the body needs to be able to absorb the vitamin in the digestive system. Children are particularly vulnerable to develop disruptions of the digestive system including diarrhea and stomach illnesses which reduce the body's ability to absorb vitamin C (Hagmann 1937). Other conditions such as wasting, failure to thrive, dysentery, disease such as infectious conditions, and parasites can also reduce the guts ability to uptake ascorbic acid (Larralde 2007; Hess 1920).

2.3. Vitamin C: Biochemical Role

Ascorbic acid (vitamin C) has numerous functions and interacts with a variety of processes in the body (e.g., Bourne 1972; Peterkofsky 1991; Du et al. 2012). When ascorbic acid is unavailable, the processes are disrupted causing the clinical symptoms of scurvy to develop. A selection of the most relevant functions will be discussed here.

The major function of ascorbic acid is the creation and maintenance of connective tissues, particularly collagen. Ascorbic acid does not affect the formation of collagen per se but influences the post-translation stage, impacting the final product (Peterkofsky 1991). Collagen is formed of three polypeptide chains made of numerous amino acids. Once formed, the chains cross-link to form collagen's triple helix structure which confers strength and resistance capabilities. Ascorbic acid is a co-factor in the hydroxylation of critical amino acids proline and lysine which act to crosslink the chains together (Woodhead-Galloway 1980; Ramachandran & Ramakrishnan 1976; Peterkofsky 1991). If these amino acids are unable to hydroxylate, the collagen will be poor in quality, unstable, and form at a reduced rate (Peterkofsky 1991; Bourne 1972).

Poor quality collagen impairs the formation and calcification of bone, as it is a major component of the unmineralised bony matrix called osteoid (Chatterjee 1990). Osteoblasts, which deposit bone forming osteoid, show reduced activity, appear modified and secrete abnormal osteoid when scorbutic (Jaffe 1972). Although bone formation is reduced, sometimes very significantly, bone resorption continues as normal, leading to brittle and weak bone. Collagen is also an important component in connective tissues in vascular walls and scar tissue. Poor collagen production weakens vascular walls and increases their susceptibility to rupture under light pressure or trauma which causes hemorrhaging and delays in wound healing (Chatterjee 1990). Additionally in cases of severe scurvy, old wounds can sometimes re-open due to collagen breakdown without proper replacement (Bourne 1942). Guinea pig studies have demonstrated changes in teeth of animals affected by scurvy. The researchers observed degeneration of the pulp, cement and dentin, a halt in dentin deposition by odontoblasts, as well as the absence and disorganization of cells, nerves, nuclei, odontoblasts and blood vessels (Zilva & Wells 1919; Boyles 1938; Jaffe 1972). Enamel formation is unaffected but haemorrhaging can locally disrupt ameloblasts (enamel forming cells) (Boyle 1938). However, these changes have only been observed in animal studies, not in children (Jaffe 1972).

A common co-occurrence of infantile scurvy is anemia, although no specific type of anemia is more prevalently associated with the condition (Parsons & Smallwood 1935; Goldberg 1963; Grewar 1965; Jaffe 1972). Ascorbic acid increases folic to colinic acid conversion and promotes iron absorption in the digestive system (Grewar 1965). A deficiency in folic acid can cause megaloblastic anemia while a deficiency in iron causes iron-deficiency anemia (Grewar 1965). Therefore, a deficiency of vitamin C can increases

the risk of developing anemia, but only if a person is deficient in other components of blood pathways.

Ascorbic acid also acts as an important cofactor in numerous processes, for example energy production (carntine) and neurotransmitters (dopamine to norepinephrine). In the first case, a deficiency of vitamin C will cause lethargy, while in the second it can lead to depression and mood variability (Carr & Frei 1999; Kamien 2011). Other small roles include serotonin formation, maintenance of responsiveness to epinephrine, formation of bile acids, tyrosine metabolism, peptide amidation, copper metabolism, and various protein metabolic processes (Chatterjee 1990; Carr & Frei 1999).

Lastly, recent research has shown that vitamin C impacts the immune system with high concentrations of the vitamin located in leukocytes and its antioxidant properties (Field et al 2002). The vitamin can reduce the duration and severity of colds and infections but therapeutic use has demonstrated mixed results; although the vitamin may assist in disease prevention for conditions caused by oxidative stress (e.g., cancer, cardiovascular) (Chatterjee 1990; Carr & Frei 1999; Du et al. 2012; Hemilä & Chalker 2013). Early documented cases of infantile scurvy found the children usually died of related infectious diseases (Grewar 1965; Jaffe 1972). An increased risk of infection, due to a poor immune response, when scorbutic is documented (Field et al. 2002). However, vitamin C benefits against infection have not been proven. The association may therefore also reflect other variables such as overall health, nutrition, and sanitation amongst others.

2.4. Previous Paleopathological Research on Infantile Scurvy

Analyses of infantile scurvy in paleopathology are a recent development. The first systematic study of infantile scurvy was produced by Ortner and Ericksen in 1997. Subsequent works include Ortner et al. (1999; 2001), Brickley and Ives (2006), Mays (2008), Devriendt et al. (2010), Stark (2010; 2014), Brown and Ortner (2011), Geber and Murphy (2012), Lovász et al. (2013), Bourbou (2014), and Klaus (2014). Cases of infantile scurvy have been diagnosed in infants aged between birth and 18 years, from diverse backgrounds including Native American and European, and from a variety of time periods, from prehistoric to historic. The paleopathological studies have identified a variety of features caused by the disease using an array of techniques including macroscopy, radiography, and microscopy. The following sections will discuss the documented paleopathological features with additional perspectives from clinical research to provide additional context.

2.5. Clinical and Macroscopic Descriptions

Scurvy can occur at any age but risk of developing the disease is largely determined by socio-cultural and physiological factors. The majority of reported infantile scurvy cases in

the last century occurred between the ages of six months to one year but in the 1800s the documented average was closer to one year of age (Barlow 1883; Still 1935; Grewar 1965). Signs and symptoms of scurvy only appear once the body pool of vitamin C is critically low. The time required to reach this level is usually six to nine months but can take as little as three months (Hess 1920; Tienboon 2012). The time required to reach critical levels is dependent on the initial body pool and amount of trace vitamin C intake during the period of deficiency. Congenital scurvy is exceedingly rare in the medical literature as infant stores of the vitamin usually last up to 5 month post-partum and breast milk provides additional vitamin C (Ingalls et al. 1938; Woodruff 1956). Vitamin C is stored throughout the body in various tissues, particularly those with high metabolic activity (e.g., adrenal glands, pituitary gland, liver, and kidney) (King 1938; Hediger 2002). Although, cases can occur when the mother is very ill and her body pool of vitamin C is exceedingly low during both pregnancy and breastfeeding, or if the infant is fed a diet poor in vitamin C (Hess 1920; Jackson & Park 1935; Hirsch et al. 1976; Bhat & Srinivasan 1989). The weaning period can be a vulnerable time due to particular diets which may not meet vitamin C requirements. Scurvy is unusual in children aged 1 ½ to 2 years of age or older as they are usually weaned and are consuming a wide range of foods (Hess 1920). In this age group, cases occur as a result of narrow diets for particular reasons such as food preferences or inaccessibility (Noordin et al. 2012). In young infants and children, scurvy has been known to co-occur with anemia (e.g., Barlow 1883; Parsons & Smallwood 1935; Goldberg 1963) and can produce clinical signs (e.g., osteopenia, subperiosteal new bone) similar to congenital syphilis (e.g., Barlow 1883; Jackson & Park 1935; Park et al. 1935).

Tests for ascorbic acid levels are standardised in the literature with most clinicians using the levels found in blood plasma. Recent literature defined the normal serum range for ascorbic acid to be 26.1-84.6 μmol/l in children, classifying values under the minimum number as deficiency (Valentini et al. 2011; Besbes et al. 2010) or a level below 8mg% ascorbic acid when measuring the buffy coat, the anticoagulant portion of a blood sample (Grewar 1965). A low plasma level may be reached up to 100 days prior to the appearance of the first clinical signs of scurvy while the delay is only 30-40 days when measuring the buffy coat (Grewar 1965; Jaffe 1972). Today, infantile scurvy is readily treatable.

Scurvy has no pathognomonic features. In the past, some researchers included early disease as a distinctive stage such as “latent” scurvy or “subacute” scurvy (Hess 1920; Frölich 1935; Jaffe 1972; Noordin et al. 2012). The stage were qualified by delayed growth, irritability, slight increase in the fragility of connective tissues, decreased ascorbic acid values in the plasma and buffy coat, and decreased appetite with a failure to display the classic signs of scurvy. However, this stage is not always recognised as the symptoms are vague. A typical clinical picture of infantile scurvy consists of tender and swollen bleeding gums, hemorrhages, debilitating pain, tender limbs (particularly the

legs) and swelling of the joints (Evans 1983; Tienboon et al. 2012). Trauma is the driving force behind the development of hemorrhages (Park et al. 1935). Hemorrhaging typically occurs in the skin (subcutaneous) or under the periosteum (subperiosteal) of bone. Hemorrhage size ranges from a small, localised bleed to an injury extending the whole length of the diaphyseal limb bone shaft (Hess 1920). Hemorrhages also lead to inflammation and swelling in the location they appear.

The majority of clinically recognised scorbutic signs and symptoms in infants are associated with soft tissues and are unobservable on the skeleton and in paleopathology. However, chronic inflammation and hemorrhages can cause porosity and occasionally hypertrophic lesions on the skeleton (Ortner & Ericksen 1997; Ortner et al. 1999; Brown & Ortner 2001). The paleopathological literature places great emphasis on the porosity aspect when studying scurvy. Once inflammation sets in or a hemorrhage occurs, the body increases the volume of vascular tissue in the area to remove any excess blood or liquid, resulting in increased porosity of the bone (Ortner & Ericksen 1997). Ortner and Ericksen (1997) define porosity as local, abnormal bone with visible pores under 1mm in diameter which penetrates the cortex of normal or hypertrophic lamellar bone. Scorbutic porosity is contrasted to 'normal' porosity which is sparse and variable in size (Ortner et al. 2001). Locations prone to develop porosity are where haemorrhages and blood can come into direct contact with the bone such as under the periosteum or where vessels have direct contact with bone (Ortner & Ericksen 1997). Once the disease begins to resolve or the child receives trace amounts of vitamin C, bony calluses of hemorrhages can develop as plaques of new bone superimposed onto the lamellar bone (Ortner et al. 2001). The following list consists of the major macroscopic features used to identify scurvy in paleopathology.

The skull and its features are well described in the paleopathological literature and were the focus of the first research papers. Some of these features were confirmed with a preserved skull from a case diagnosed during life by Barlow and through autopsy reports (Ortner & Ericksen 1997). Unfortunately, post cranial bones of the known skeleton were not preserved for comparative analysis. Following is a list of the different scurvy features which can be observed on the skull.

1. *Porous lesions of the sphenoid*: Porosity is observed bilaterally on the external surface of the sphenoid's greater wing, see Figure 2.1. The feature was first identified by Ortner and Ericksen (1997) and considered to be "virtually pathognomonic" in paleopathology but not required for a diagnosis (Ortner et al. 2001:214). However, this feature has not been documented clinically (Noordin et al. 2012). Ortner and Ericksen (1997) correlate this feature to thickening observed by Barlow (1883) in the zygomatic region of some diagnosed cases. Also, the temporalis muscle attaches to the sphenoid in this region and the vascular system has two arteries which graze the bone and could cause inflammation in the region if hemorrhagic (Ortner & Ericksen

1997). Occasionally, hypertrophic bone is present on the greater wing, lesser wings and sphenoid body (Brickley & Ives 2006; Mays 2008). Lastly, porosity can be observed around the foramen rotundum, internal aspect (Brown & Ortner 2011), see Figure 2.1.

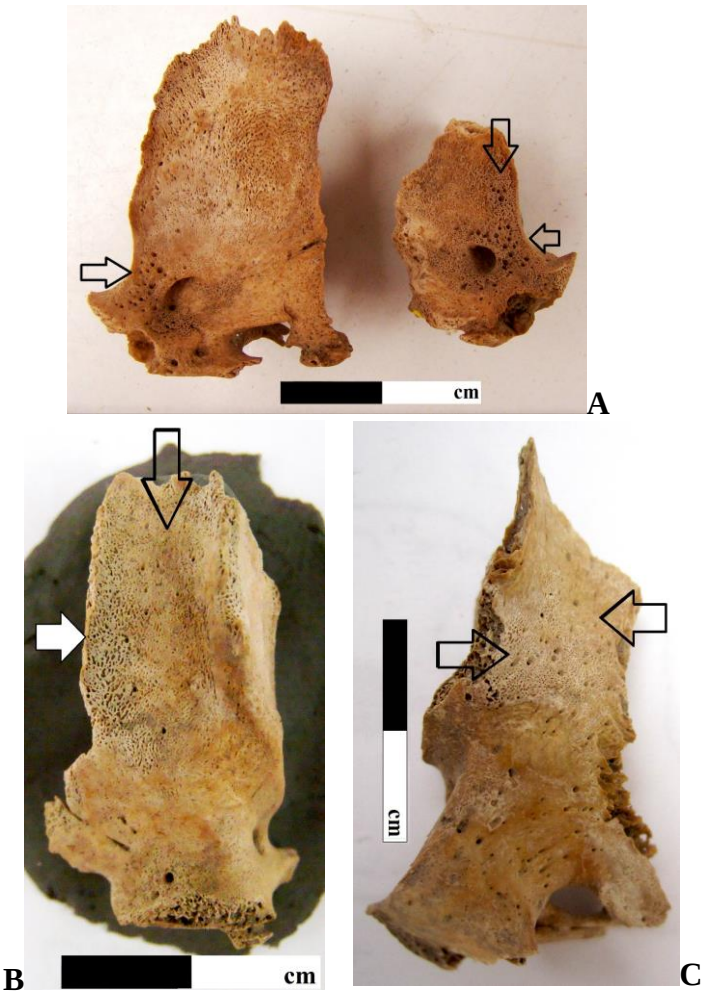


Figure 2.1. Scurvy: porosity and new bone on the sphenoid. (A) Juvenile 95, abnormal porosity around the foramen rotundum (arrow). (B) Juvenile 125, new bone formation (solid arrow) and abnormal porosity (open arrow). (C) Juvenile 56, arrows point to abnormal porosity.

2. *Orbit*: Clinically, hemorrhaging on the orbital plate is a rare feature. The feature is usually unilateral and, if the reaction is large enough, can displace the eye (*exophthalmos*) (Hess 1920; Jaffe 1972). In paleopathology, slight porosity in the orbit is normal and, in young infants, new bone can be deposited in layered sheets (Brickley & Ives 2006). However, significant porosity or thick bone hypertrophy located on the roof (frontal bone) and the lateral (zygomatic) portions of the orbit,

usually bilateral, should be considered abnormal (Ortner & Ericksen 1997). Although accepted as a major feature, it is uncommon in diagnosed cases of scurvy (Ortner & Ericksen 1997; Ortner et al. 1999). It is also important to not confuse this feature with what has been described as cribra orbitalia.

3. *Porosity of the maxilla and mandible.* Clinically, inflammation and hemorrhaging of the gums is a near diagnostic sign of scurvy. However, porosity only occurs after teeth have erupted, which is at earliest around 4 months of age (Barlow 1883; Hess 1920; Grewar 1965; Gustafson & Koch 1974). In absence of hemorrhaging, the gums can still react by appearing irritated, tender, puffy, and coloured red to purple (Hess 1920). In very advanced cases of scurvy, periodontitis and ante-mortem tooth loss can ensue. Clinically, a foul breath is often noted and is likely associated with these changes.

    a. *Infraorbital foramen:* Increased porosity around the foramen of the maxilla is commonly observed in paleopathology but new bone is rare (Ortner et al. 1999), see Figure 2.2A. Arteries, veins and nerves pass through the foramen and rupture or inflammation of these vessels could be associated with the bleeding gums phenomenon (Ortner et al. 1999).

    b. *Alveolar processes*: Tooth eruption is normally accompanied by porosity. However, if the porosity extends far from the alveolar process then it is considered pathological in paleopathology (Ortner et al. 1999; Brickley & Ives 2006), see Figure 2.2A and 2.2D.

    c. *Posterior maxilla*: Porosity and occasional new bone are located at the posterior, around the molars. This feature is considered important by Ortner and Ericksen (1997).

    d. *Palate*: Hemorrhages and petechiae have been recorded in clinical cases on the hard palate (Hess 1920; Harris 1928). In paleopathology, porosity has been observed on the palate of the maxillary and palatine bones. Porosity is a normal feature of this area, typically in a u-shape along the alveolar process and thickest at the anterior of the mouth (Ortner et al. 1999). Porosity is pathological when the expression is excessive and extends posteriorly or antero-medially to the palatine suture (intermaxillary suture) (Ortner & Ericksen 1997; Brickley & Ives 2006), see Figure 2.2B.

    e. *Coronoid process:* On the mandible, abnormal, increased porosity is located on the medial aspect of the coronoid process above, and sometimes around, the mandibular foramen (Ortner et al. 1999), see Figure 2.2C. The foramen is the insertion location of the temporalis muscle. The porosity can be significant but it

does not extend much anteriorly to this, never reaching the alveolar margin (Brickley & Ives 2006). Ortner et al. (1999) found the feature was uncommon.

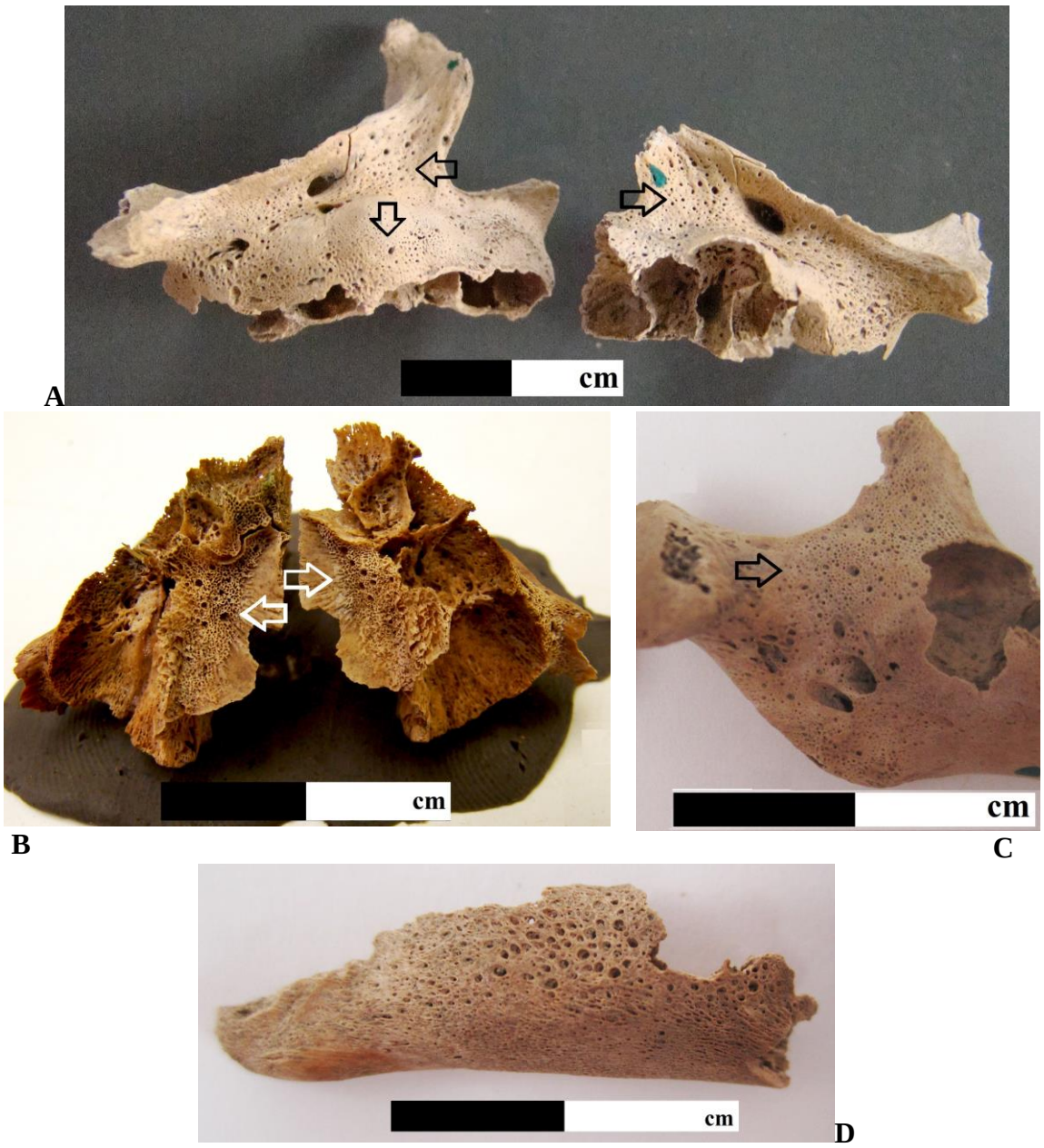


Figure 2.2. Scurvy: porosity and new bone on the maxilla and mandible. (A) Juvenile 124, abnormal porosity around the infraorbital foramen of the maxilla and porosity along the alveolar process (arrows). (B) Juvenile 208, porosity and new bone formation on the palatal surface of the maxilla (arrows). (C) Juvenile 124, abnormal porosity on the coronoid process (left mandible) (arrow) (D) Juvenile 219, porosity on the alveolar process of the mandible.

f. *Cranium*: Clinically, hematomas and porosity have been observed on cranial bones at autopsy (e.g., Barlow 1883 on the frontal and parietal bones). Swelling and thickening of the cranial bones leading to bossing is called '*Parrot's swellings*' in infants. The feature can be observed on Barlow's diagnosed scurvy skull (Barlow 1883; Ortner & Ericksen 1997). Swelling and thickening of cranial bones can also be observed in cases of rickets and congenital syphilis (Jaffe 1972; Ortner 2003). In paleopathological cases of scurvy, porosity, sometimes accompanied by thickened new bone, occurs in particular but non-specific locations across the cranial vault. The lambdoid region was the most common location observed by Ortner et al. (1999). Fontanels may also remain open, a feature observed clinically at autopsy and in paleopathologically (Barlow 1883; Mays 2008).

g. *Ectocranial*: In paleopathology, porosity is considered abnormal if it is dense, irregular in shape, and penetrates the cortex. This feature has been observed on most cranial bones, with specific reference to the parietals, glabella and temporal bones in the literature (Brickley & Ives 2006; Mays 2008). Vascular impressions, either branched or star shaped, were observed on the frontal, parietal, and occipital bones (Brown & Ortner 2011).

h. *Endrocranial*: Large porosity and plaques of new bone are abnormal features when considering the endocranial surface is usually undergoing a resorptive process in young, growing infants and children. These features have been observed on the occipital bone and temporal squama (Brickley & Ives 2006; Mays 2008). Vascular impressions, called '*branched lysis*' on the endocranial surface, are observed on most bones but particularly the parietal and occipital. Impressions were observed at autopsy of known scurvy cases (Mays 2008; Brown & Ortner 2011).

4. *Internal surface of the zygomatic bone*: This aspect of the zygomatic bone can show extensive abnormal porosity (Ortner et al. 1999).

Postcranial feature for infantile scurvy were first described by Ortner et al. (2001) after diagnosing individuals with scurvy on the basis of cranial features. Further studies (e.g., Brickley & Ives 2006; Mays 2008; Brown & Ortner 2001) have refined the findings.

1. *Long bone metaphyses*: During growth, osteoclasts reshape the ends of the bones through resorption. The process reveals vascular channels, appearing as open pores, which are quickly filled-in by osteoid deposited by osteoblasts. However, as bone formation is impeded in scurvy, the pores are left open, leaving substantial porosity (Ortner et al. 2001). In typical circumstances, the porosity doesn't extend over 5-10mm from the physis of the bone with variability caused by age and type of bone involved (Ortner et al. 2001), see Figure 2.3B. Brown and Ortner (2011) felt the feature was uncommon in paleopathological cases, with their own observation of

porosity often exceeding the minimum but were under the maximum distance. A review of this feature should be conducted with greater attention to age and bone variability.

2. *Long bone shaft*: In juveniles affected by scurvy, subperiosteal hemorrhages are common as the periosteum is loosely attached and can easily be lifted from the bone (Gray 1959), see Figure 2.3. Clinically, hemorrhages are most common in the lower limbs (knees and ankles) and the inner thigh in infants (Barlow 1883; Hess 1920). The size of subperiosteal hemorrhages range from a small, localised bleed to an injury extending the whole length of the diaphyseal limb bone shaft (Hess 1920; Evans 1945). In paleopathology, subperiosteal hemorrhages are inferred from the presence of porosity and hypertrophic new bone formation caused by the lifting of the periosteum (Ortner 2003). However, Kwon et al. (2002) report that infants between the ages of one and four months can develop subperiosteal new bone on any limb bone as a normal feature of growth. Above 4 months, normal growth can cause the development of subperiosteal new bone but his feature is uncommon. Lower limb bones, particularly the tibia and femur were most common and often bilateral (66% of the time for the tibia and 79% of the time for the femur) though not exclusively. New bone was found to be a rare occurrence on the ulna and radius (Kwon et al. 2002).

3. *Scapulae*: Subperiosteal hemorrhages have been observed at autopsy on the scapulae, specifically swelling on the dorsal aspect and blood on the ventral aspect (Barlow 1883; Hess 1920). Barlow (1883). This is logical as numerous arteries (e.g., the subscapular and thoracodorsal) have direct contact with the bone (Ortner & Ericksen 1997). Slight porosity and new bone formation is observed bilaterally on the supra- and infraspinous fossae, as well as the anterior (or ventral) portions of the scapulae in paleopathology. The supraspinous fossa is considered the most common location (Brickley & Ives 2006).

4. *Sternal end of the ribs*: Clinically, the sternal end of the ribs can swell and, in advanced scurvy, the costochondral junction cartilage can become displaced behind the rib bone ("*sub-luxation*"), causing an acute angle to form known as the '*scorbutic rosary*' (Grewar 1965; Hess 1920). The feature is typically observed on ribs one to eight in clinical cases (Noordin et al. 2012). Grewar (1965) claims the feature is different from rachitic beading as it is created by a different mechanism. The difference may be challenging to see paleopathologically as the cartilage is rarely preserved. Nonetheless, the sternal ends appear enlarged (Brickley & Ives 2008), see Figure 3.1A.

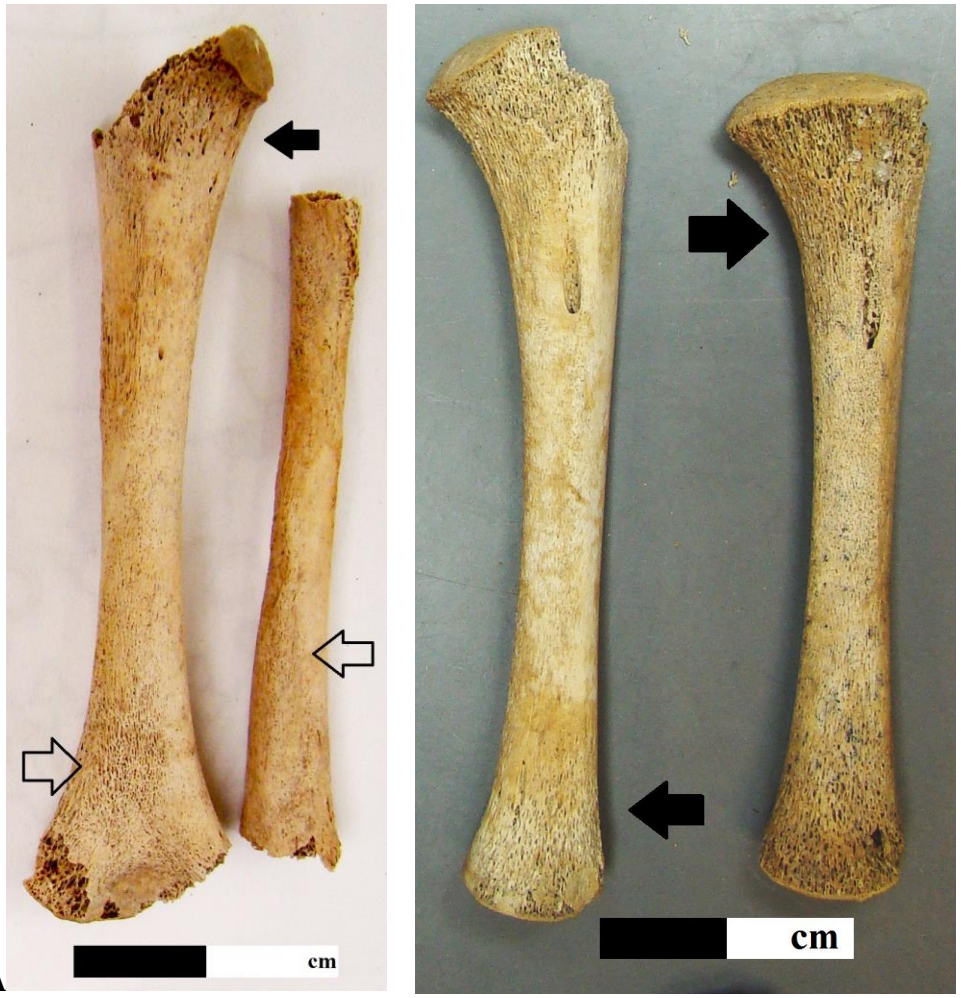


Figure 2.3. Scurvy: reactive new bone on the diaphysis and metaphyseal porosity of limb bones (A) Juvenile 231, left humerus and left radius. Reactive new bone (open arrows) and metaphyseal porosity (solid arrow). (B) Tibiae (right) juvenile 125 and (left) Juvenile 220. Metaphyseal porosity (arrows).

5. *Ilium*: Subperiosteal haemorrhaging is a rare feature on the pelvis (Brickley & Ives 2008). Barlow (1883) observed blood on the internal aspect of the ilium during only one autopsy. Recently, Brown and Ortner (2011) observed bilateral porosity on the ilium potentially caused by scurvy. The porosity was located on the exterior of the bone, central portion, and the internal surface "between the anterior borders of the ilium and auricular surfaces" (Brown & Ortner 2011:202). The internal surface porosity was accompanied by vascular canals and plaques of new bone (Brown & Ortner 2011).

6. *Fractures*: Fractures occur due to build-up of weak and brittle bone and have not been recorded in association with cases of scurvy in the paleopathological literature but it is

> a known clinical symptom and one used in radiography to identify scurvy. Fractures are associated with severe disease (Park et al. 1935). They are observed clinically at the metaphyses of limb bones and adjacent to the sternal end of the ribs (e.g., Barlow 1883; Brickley & Ives 2008; Noordin et al. 2012). Barlow (1883) noted at autopsy that the epiphyses could become separated from the shaft resulting in possible fracture.

Clinical symptomatology concerns all aspects of the body which a physician or surgeon can observe. Although most of these observations cannot be assessed in paleopathological cases, the general changes remain important to consider when assessing the impact of disease on the individual. Edema and anarsarca (swelling) are observed on various structures including the limbs, breast, and belly amongst other locations (Hess 1920). The swelling is often caused by hemorrhages and poor bone formation. The joints of limbs commonly appear swollen, including the knee and ankle (Barlow 1883; Hess 1920). If edemas occur at the ends of the bones, epiphyses are sometimes displaced away from their respective shafts (Hess 1920). Scorbutic children are very irritable due to pain from swelling and fractures (Grewar 1965; Jaffe 1972; Carr & Frei 1999; Kamien 2011). Limbs become tender to the touch from swelling and cause discomfort, making it difficult for the infants and children to walk or sit upright. Often, the children twist their legs into a "frog" position but if the pain is too great, it may lead to pseudo paralysis (Grewar 1965; Jaffe 1972). Tenderness and swelling are most common in the lower limbs but tenderness is usually more prominent on one side (Grewar 1965). Subcutaneous hemorrhages in the form of bruises occur anywhere on the body but are most common in the lower extremities except in cases under six months of age, where it is just as common in the upper as lower limbs (Hess 1920). Other clinical symptoms are occasionally noted but are not only exclusively associated with scurvy. Individuals often appear pale and suffer from fevers (Grewar 1965). Young children can appear small for their age (Tienboon 2012). Of 66 cases observed in Grewar's (1965) clinical study, a significant number of children were classified under the 3rd percentile for their age in growth. However, it is unclear if this symptom is due to scurvy alone. Additionally, untreated scurvy can be fatal (Grewar 1965; Valentini et al. 2011). For example, seven of Barlow's (1883) 31 cases died, or a mortality of roughly 1:4.3 individuals but mortality rate is variable and determined by the extent and severity of disease. Enlarged organs including the heart (e.g., myocardial hypertrophy) and spleen have also been observed at autopsy (Hess 1920; Grewar 1965).

Important limitations accompany the previous paleopathological works. First, the early studies (e.g., Ortner & Ericksen 1997; Ortner et al. 1999) only considered the skull of individuals. This is in conflict with the medical literature which focuses on changes observed in the limbs, particularly the lower limbs. Second, individuals which did not display abnormal porosity on the sphenoid were eliminated from the studies (Ortner et al. 1999; Ortner et al. 2001), potentially biasing some of the results. The medical literature

does not report any changes at this spot. Although, the location is challenging to observe macroscopically by a physician and there is a good physiological reasoning behind the observation of porosity at the location (Ortner & Ericksen 1997). Third the studies are biased towards older age categories. For example, all the skulls were from individuals aged over 2 years in Ortner and Ericksen's (1997) study while only 2 fell in the category between birth and 2 years in Ortner et al. (1999). A systematic undertaking of anatomical reference collections with scorbutic bone has not yet been completed and may provide additional support for observed paleopathological features. However, the anatomical reference collections are often less than ideal as the specimens often have little accompanying medical information and selection of specimens may be biased towards severe or abnormal cases which do not reflect the possible range of features and expression potentially encountered in paleopathology.

2.6. Radiographic Features

Radiography is an additional tool used clinically to diagnose scurvy. Scurvy has no pathognomonic features but a combination of at least two or more radiographic signs can be considered highly suggestive of scurvy in a clinical setting (Grewar 1965). The majority of radiographic features are tied to the process of continued bone resorption coupled with a failure to form new bone. These processes result in overall bone loss for both trabecular and cortical bone (Shore 2008). Early signs of scurvy can be found in the metaphyses of bones undergoing growth, particularly in the lower limbs (ankles and knees), proximal humerus and the wrist area (Jaffe 1972; Noordin et al. 2012; Shore 2008). Clinicians typically only radiograph the lower limbs. The majority of clinically documented radiograph features should be observable in paleopathology though poor preservation may damage or mask fragile features such as the '*corner sign*' or '*Pelkan's spur*' (Stark 2010; Stark 2014). Researchers have devised a number of radiographic features which can be used to diagnose scurvy. Following is a list of these features.

1. *White line of Fraenkel*: Scurvy halts the production of chondrocytes (cartilage cells) but differentiation and calcification of chondrocytes continues as normal (Shore 2008). Coupled with poor cartilage resorption after mineralization, the zone of provisional calcification at the epiphyseal junction of the metaphysis develops a thick, opaque, white band of dense bone (Grewar 1965; Shore 2008), see Figure 2.4. A white line at the growth plate is normal but is thickened in scurvy (Valentini et al. 2011). The feature is characteristic but not diagnostic of scurvy when it appears in conjunction with the '*scurvy line*' feature and '*ground glass trabeculae*' (Valentini et al. 2011; Shore 2008).

2. *Scurvy line or Trümmerfeld zone*: Just underneath the '*white line of Fraenkel'*, towards the diaphysis, there is an area or band of increased radiolucency (also called rarefraction) (Grewar 1965), see Figure 2.4. This area is located in the proliferative

cartilage zone and is the area most affected by continued resorption, leaving an appearance of rarefraction. As there is reduced tissue, the bone becomes brittle, weak, and prone to fractures from pressure (Shore 2008; Noordin et al. 2012).

3. *Generalized osteopenia and ground glass trabeculae*: Continued bone resorption coupled with a halt in bone formation results in a thin cortex, reduced amount of trabecular bone, and greater radiolucency (Park et al. 1935), see Figure 2.4. Resorption is first observed in the spongiosa of the bone (Grewar 1965; Park et al. 1935). Due to the loss of bone, the remaining trabeculae develop a blurred (called '*ground-glass*') appearance (Shore 2008). In the metaphysis, the trabeculae may also lose their typical longitude axis causing the trabeculae to appear disorganised (Shore 2008). Over time, the whole bone will develop generalised osteopenia.

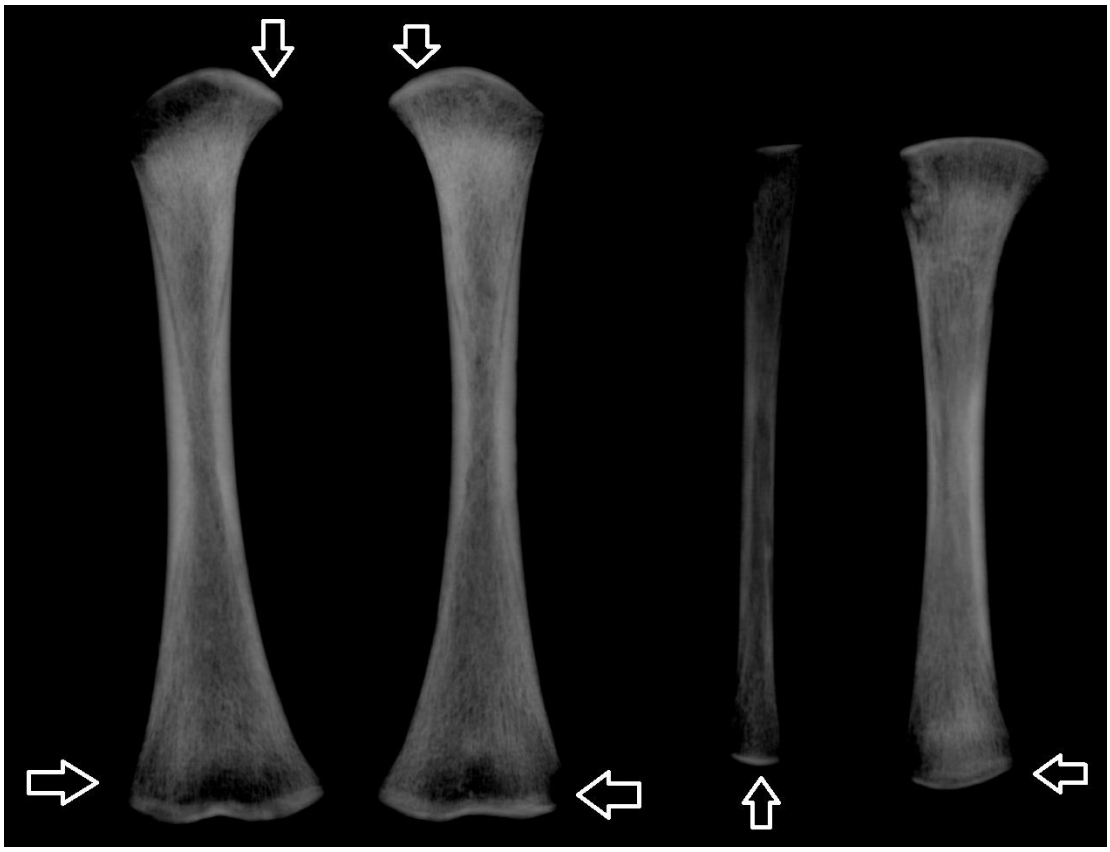


Figure 2.4. Scurvy: radiographic features. Juvenile 6, radiograph of the femora, right fibula and right tibia displaying scorbutic features. Open arrow point to the white line of Fraenkel with the scurvy line towards the shaft. The fibula displays some bone loss (osteopenia) and a thin cortex. Radiograph taken by the CAD at the Laboratoire d'Analyses Physiques et de Caractérisation des Matériaux, Direction de l'Archéologie, Communauté d'Agglomération du Douaisis.

4. *Wimberger's ring*: The feature is characteristic of scurvy and is restricted to the epiphyses of long bones. The ring appears as a distinctive white border around the margins of the epiphyses, similar to the '*white line of Fraenkel*,' and is accompanied by rarefaction of the trabeculae at the center of the bone (Shore 2008; Noordin et al. 2012).

5. *Thinned cortex*: The feature is common in advanced cases of scurvy and caused by the continued resorption of bone (Shore 2008). The thin cortex appears '*pencilled*' in or to have a '*pencil-point*' effect (Grewar 1965; Hess 1920; Noordin et al. 2012). Epiphyses can also have thin but distinctive cortices (Noordin et al. 2012).
6. *Metaphyseal fractures*: Fractures can occur in the thickened area of the metaphysis and is considered nearly diagnostic of scurvy when observed (Shore 2008). The feature is typical of advanced cases of scurvy as risk of fracturing increases with greater bone loss.

7. *Corner sign:* Corner signs are characteristic of scurvy and appear as clefts of broken bone occurring between the zone of proliferation and the shaft (Shore 2008; Valentini et al. 2011). As the metaphyses undergo greater bone loss, the structure becomes prone to fractures. Fractures, particularly transverse fractures, occurring on the edges of the bone cause corner signs. On radiographs, fractures appear as lines of decreased radiolucency (Noordin et al. 2012).

8. *Pelkan's or lateral spurs*: As noted above, the metaphysis becomes highly susceptible to fractures. Fractures can also displace or separate the epiphysis and zone of provisional calcification away from the metaphysis leading to misalignment (Shore 2008; Grewar 1965). When this occurs, the thickened '*white line of Fraenkel*' is also pulled away beyond its limit causing the formation of a spur, a typical feature of scurvy (Shore 2008). The process can also result in a cupping effect when fractures occur towards the center of the bone. Grewar (1965) suggests it is distinctive from rickets as scorbutic cupping will appear sharp while a rickets origin will appear ragged. Differentiating the two conditions may be challenging in paleopathology as typically only ossified bone remains and the bone will probably have undergone taphonomic processes since death.

Additional features can be observed clinically. When radiographs of the chest are made, the '*scorbutic rosary*' may be observable. The sternal end of the rib will appears enlarged and round (Noordin et al. 2012). In the skull, a "hair-on-end" bone formation, porotic hyperostosis and marrow hyperplasia can be present, although these features may be due to anemia (Noordin et al. 2012). Healing, or receiving trace amounts of vitamin C will resume normal bone and collagen formation, allowing the calcification of subperiosteal hemorrhages along the long bone shafts into bony calluses (Evans 1945; Jaffe 1972). Clinically, the bony calluses are recorded from radiographs but the method of

investigation is unnecessary in paleopathology as the bone is available for direct study. Additionally, features such as fractures and areas of rarefraction will repair and remodel in the healing stage. In some cases, a residual line of dense bone will remain at the location of the metaphysis-epiphysis junction active during disease once growth resumes (Noordin et al. 2012). The area of rarefaction, as seen when disease was active, may also persist in the center of the epiphyses after healing (Shore 2008).

2.7. Microscopic Features

Investigations into the microscopic appearance of scurvy have been produced with controlled animal studies (e.g., Delf & Tozer 1918) and autopsy on children (e.g., Harris 1928; Park et al. 1935). Microscopic investigations are typically used for post-mortem diagnosis of scurvy (e.g., Clarke et al. 1980) and to understand the processes which result in radiographic and macroscopic features. As scurvy affects new bone formation in children, the literature focuses on the changes occurring at the metaphyseal ends of the long bone limbs or the sternal end of the ribs (Hess 1920). In most investigations, the junction is cut longitudinally for study.

Microscopic changes are some of the first to appear and have been observed in guinea pig studies of subclinical scurvy (e.g., Clarke et al. 1980). However, no pathognomonic features can be associated with scurvy. When scurvy is latent, microscopic changes are less severe and most or all features are absent based on findings in guinea pigs (Delf & Tozer 1918). Although, an uneven sternal rib end growth plate, some irregular columnar cartilage formation, smaller cartilage cells, shorter and fewer trabeculae, and increased of blood in marrow cavity have been reported to appear (Delf & Tozer 1918). When scurvy is chronic and severity of disease is variable, a band of ossified bone (observed on radiographs as the '*white line of Fraenkel*') develops at the junction and is hypothesized to stabilise and strengthen the junction (Delf & Tozer 1918).

The marrow in the metaphysis becomes altered, turning yellow and developing a "gelatinous consistency", called '*Gerüstmark*' or '*framework marrow*' (Hess 1920; Harris 1928; Bourne 1972). Osteoblasts, marrow cells, blood vessels and trabeculae in the metaphysis and epiphyseal junction area are significantly reduced in number (Bourne 1972). The trabeculae become shortened, isolated, disoriented, and very brittle which results in the development of the '*scurvy line*' (or Trümmerfeld zone) and generalised osteopenia radiographic features (Bourne 1972). The surrounding cortical bone of the metaphysis becomes thin and subperiosteal hemorrhaging can be observed (Bourne 1972).

The scorbutic lattice is a characteristic feature of scurvy which develops in the metaphyseal growth plate and sternal ends of the ribs (Follis et al. 1940). In the 'provisional calcification' zone of the cartilage column, calcium salts are placed between

the cartilage cells creating ossified cartilage. Osteoid is normally deposited and the cartilage is removed to develop new bone. However, scurvy causes a reduction in osteoblasts which results in a reduced amount of osteoid secreted and non-removal of the cartilage. The build-up of calcified cartilage, called scorbutic lattice, weakens the metaphysis junction by increasing the brittleness (Bourne 1972). The feature is observed on radiographs as the '*white line of Frankel*'. Developing cartilage cells, which are typically organised in a column and grow larger in size towards the bone, become reduced in size and the column distorts in shape into a "zigzag" when scorbutic (Hess 1920; Harris 1928; Bourne 1972). Collectively, the changes cause the junction to weaken and distort in shape. In severe cases, the metaphyseal end can fracture, be crushed by the surrounding compact cartilage and, in conjunction with the brittleness, lead to fractures, breakdown of the junction, and macroscopic changes such as flaring of the ends (Hess 1920; Bourne 1972). The breakdown and subsequent increase in osteoblasts at the fracture is also very characteristic of scurvy (Follis et al. 1940).

The majority of microscopic features are soft tissue features which are unlikely to survive in archaeological bone. However, ossified subperiosteal hemorrhages can be confirmed when studying cross sections of archaeological bone (e.g., Van Der Merwe et al. 2010; Lovász et al. 2013). The new bone appears as added layers of bones which do not disturb the underlying cortex. The appearance can assist in a differential diagnosis by ruling out conditions which affect the underlying cortex (e.g., inflammation, rickets) (Lovász et al. 2013). Further investigations into the possibility of observing the scorbutic lattice in archaeological bone should be pursued as, in theory, the ossified portions of the cartilage could be observed. Fractures in the metaphyses and trabecular bone should also be observable in paleopathology (Brickley & Ives 2008). However, fractures are non-diagnostic and can be caused by a variety of diseases, including from pressure and application of force, connective tissues diseases (e.g., osteogenesis imperfecta), metabolic diseases (e.g., rickets, juvenile osteoporosis), neoplastic conditions (e.g., leukemia) and abuse. Lastly, the reduction in bone of longstanding or severe scurvy is observable but non-specific (Brickley & Ives 2008).

2.8. Conclusion

Scurvy, a vitamin C deficiency, is characterised by impeded bone formation and weakened connective tissue resulting in hemorrhages. These two major processes leave many traces, whether observed clinically, macroscopically, radiographically or microscopically. For the most part, the features described in clinical cases can be observed in paleopathological cases. However, paleopathologists can only see bones devoid of tissue and therefore must use other clues, in this case the presence of porosity and new bone, to determine areas affected by hemorrhages and inflammation in life.

Chapter 3: Background – Rickets

3.1. Introduction

The chapter will define rickets and discuss the clinical, radiographic, and microscopic appearance of the disease as seen in clinical settings with special focus on features that are observable in skeletal paleopathology. The complex pathway of vitamin D and its interactions with bone, calcium, parathyroid hormone and phosphorus will be briefly touched upon. The focus is on rickets as the current research only considers juvenile remains.

3.2. Rickets

Rickets is a disease resulting from bone mineralization failure occurring during linear growth and affecting active growth plate areas (Jaworski 1972; Milgram 1990; Pitt 2002). Bone mineralization can also fail during bone maintenance and is called osteomalacia (Jaworski 1972; Pitt 2002). Both conditions can occur in children. Adults, however, are only affected by osteomalacia. The causes of mineralization failure are manifold. Vitamin D deficiency (called nutritional rickets) is the most common and typically due to a lack of ultraviolet B (UVB) ray exposure, dietary deficiency and malabsorption in the gut of vitamin D (Pai & Shaw 2011). Vitamin D interacts with other elements and organs which, if imbalanced or malfunctioning, can also cause mineralization failure. Elements include calcium and phosphorus (Mankin 1974a; Marie et al. 1982; Pitt 2002). Chronic liver or renal disease impacts the process of vitamin D production and activation, resulting in vitamin D deficiency and other conditions, e.g., renal osteodystrophy (Adams 2005; Pai & Shaw 2011). Genetic disorders such as X-linked hypophosphatemia (also called vitamin-D-resistant rickets), acquired conditions (e.g., neoplasms), and some drugs can also disrupt vitamin D metabolism resulting in rickets (Milgram 1990; Pitt 2002; Papadopoulou et al. 2013). For further details, Mankin (1974b), Glorieux et al. (1998) and Pai and Shaw (2011) provide extensive discussions on different causes and related diseases.

The first clinical description of rickets was produced in 1645 by Whistler, quickly followed by Glisson's (1650) famous treatise, entitled "De Rachitide" (Cousins & DeLuca 1972). At the time, rickets typically affected individuals of upper class, likely because children were kept indoors, swaddled and had little exposure to sunlight (Fildes 1986; Mays 2003). The number of children affected by rickets surged to a prevalence of over 90% in infants and children living in industrial Northern European cities during the Industrial Revolution (19^{th} to 20^{th} centuries) (Mays 2003; Lewis 2007). Britain's children were particularly affected, resulting in rickets being called the 'English disease'. Causes for the high prevalence include urbanisation, crowding, and pollution. Prior to understanding the cause of rickets, cod liver oil was used as traditional medicine for

rheumatisms along the coasts of Northern Europe and found, through observations by doctors, to have curative properties in cases of rickets by the late 18th century, early 19th century (Guy 1923; Fildes 1986). Subsequently, cod liver oil was used as the main treatment for rickets as it contains very high amounts of vitamin D, though this was not known at the time. Findlay (1908) first demonstrated that exercise (access to sunlight and 'pure' air) were critical to the etiology of rickets, though the idea was suggested previously by Dudgeon and Lucas (cited by Findlay 1908:14). Mellaby's research between 1919 and 1925 demonstrated rickets as a disease of dietary deficiency while McCollum and colleagues (1922) identified vitamin D (Pitt 2002). Continued research on vitamin D, rickets, and osteomalacia continues today as the populations of most northern latitude countries are affected by low vitamin D levels (e.g., Absoud et al. 2011).

3.3. Vitamin D and Bone

Vitamin D is a steroid, fat-soluble hormone which comes in two types, vitamin D_3 (cholecalciferol) and Vitamin D_2 (ergocalciferol) (Milgram 1990; Mankin 1974a; Tenenhouse 1990). Vitamin D_3 is produced by the human body from UVB radiation (e.g., sun radiation) that penetrates the skin and converts 7-dehydrocholesterol to vitamin D_3. Bodily production of vitamin D accounts for over 80% of our vitamin supply and is regulated by the endocrine system in a complex metabolic pathway (Mankin 1974a; Pettifor 2003; Pitt 2002). The vitamin can also be absorbed from our diet. Vitamin D_2 is acquired from plants, yeast and fungi while vitamin D_3 can be procured from fish (e.g., fish liver oil) and some animal products (e.g., eggs) (Wagner & Greer 2008; USDA 2014). Absorption of the various forms is dependent on the bile composition of the small intestine (Tenenhouse 1990).

The majority of circulating serum vitamin D is in its inactive form, 25-hydroxyvitamin D_3 [$25(OH)D_3$] (Pitt 2002). Activation converts inactive vitamin D to calcitriol, known as 1α,25-dihydroxyvitamin D_3 [$1,25(OH)_2D_3$] in the kidneys (Mankin 1974a; Pitt 2002). Conversion is dependent upon need as calcitriol is quickly metabolised, not stored (Pitt 2002). Active vitamin D can perform biochemical roles and bind to tissues (St.-Arnaud & Demay 2003). Major roles include regulating calcium and phosphate homeostasis, and bone mineralization (Adams 2005). Vitamin D has also recently been found to have influence on the immune system through interaction with receptors and genes. Decreased risk of acquiring disease, changes in progression of the disease course, and rapid treatment of some diseases are a few of the observed effects. Respiratory bacteria or viral diseases, such as tuberculosis and influenza, and chronic conditions are documented examples of diseases affected by vitamin D levels but much more work is required to fully understand the complex relationship between vitamin levels and the immune system (e.g., Borges et al. 2011; White 2012).

The homeostasis of calcium, phosphorus and bone are regulated by vitamin D, in conjunction with the parathyroid hormone (PTH) and the hormone calcitonin (Pitt 2002). If calcium and/or phosphorus are imbalanced, homeostasis is restored by either increasing intestinal absorption or retrieving the minerals from bone. When calcium is low (hypocalcemia), PTH is activated to stimulate production of active vitamin D. Low serum phosphate (hypophosphatemia) undergoes the same process but can directly stimulates vitamin D production independent of PTH (Pitt 2002; Tenenhouse 1990). Elevated active vitamin D promotes calcium and phosphorus absorption in the gut (Pitt 2002). Simultaneously, elevated active vitamin D inhibits PTH to prevent hypercalcemia (high calcium*)* (Mankin 1974a; Pitt 2002). PTH can also increase renal excretion of phosphorus, preventing hyperphosphatemia (Tenenhouse 1990; Pitt 2002:7). If hypercalcemia occurs, active vitamin D can suppress PTH, inhibiting active vitamin D production which results in decreased calcitonin serum levels and reduced mineral absorption (Mankin 1974a; Pitt 2002). To release calcium and phosphorus from bone, PTH and active vitamin D trigger osteoblasts which in turn prompt osteoclasts to breakdown bone (osteocytic osteolysis) resulting in the release of the minerals (Pitt 2002; Stern 2005). Clearly, calcium, phosphate, vitamin D and PTH are tightly intertwined. Vitamin D deficiency can destabilise the response systems by reducing absorption of calcium and phosphorus from the gut (Boyde 1993). The body will then react by releasing the important minerals from bone. Calcium, phosphorus, alkaline phosphatase and a normal PH are required for proper bone mineralization (Adams 2005).

3.4. Clinical and Macroscopic Descriptions

Vitamin D deficiency can occur at any age. Modern clinical studies have found that rickets tends to develop between three to 18 months of age (Jaffe 1972; Pettifor 2003). Except in extreme conditions, the minimum age rickets develops in a child is between three to six months after birth. Infants do not store vitamin D. Instead, throughout pregnancy vitamin D is passed from the mother to the child via the placenta (Pettifor 2003). Once born, the infant relies on remaining circulating inactive vitamin D which has a half-life of three to four weeks (Pettifor 2003). Once this supply is used up rickets can develop but clinical symptoms require time to be perceivable. Cases of congenital rickets have been documented (e.g., Moncrieff & Fadahunsi 1974; Anatoliotaki et al. 2003) but prenatal deficiencies are rare as the mother's body, even if deficient, will compensate by funnelling available vitamin D [25(OH)D] to the foetus at the detriment of the mother (Shore 2008). Neonatal rickets is historically rare as breast milk contains some vitamin D, varying between 4 to 60IU/L depending on the mother's intake (Lammi-Keefe 1995; Pettifor 2004). Daily vitamin D requirement for infants and children is roughly 300IU (birth to months of age) or 400IU (over 6 months of age) which cannot be met from breast milk alone (Lammi-Keefe 1995; Pettifor 2004). Supplementation from UVB ray induced vitamin D production is necessary (Specker et al. 1985; Pettifor 2003).

The risk of developing rickets is variable and depends upon socio-cultural, environmental, and physiological factors. Sociocultural factors influence the diet and exposure to UV rays. Examples include duration of breastfeeding, weaning foods, amount of vitamin D and calcium rich foods consumed. Length of exposure to sunlight is affected by the amount and types of clothing worn (cover the skin), urbanism, and time spent indoors among others. Environmental factors influence the availability of vitamin D and calcium rich foods as well as the amount of UV rays penetrating the atmosphere. Examples include the location's geographical latitude (less UV rays towards the poles), the season (lower UV in late winter), amount of available sunlight, and amount of atmospheric pollution (blocks UV rays) (Pettifor 2003). Physiological factors include the ability to produce and absorb vitamin D as well as other critical components of calcium and phosphorus homeostasis. Examples include skin pigmentation, bile composition, calcium, phosphate and pH levels (Tenenhouse 1990). In the past, rickets was more common in cities than in the countryside (Chick 1976). Children are also particularly vulnerable as their requirements of vitamin D and calcium are high (Boyde 1993).

Clinical identification of vitamin D deficiency relies on serum levels of circulating 25(OH)D. Definitions of deficiency are variable and calcium can impact the development of features associated with the disease. Serum levels under 50 nmol/L is considered insufficient or deficient, under 30-25 nmol/L as deficient or severe, and under 12.5 nmol/L as extreme (Ross et al. 2011; Braegger et al. 2013; Davit-Béal et al. 2014). Characteristic bone changes of rickets only appear once the body pool of vitamin D is critically low (under 30-25nmol/L) and small amounts of vitamin D can prevent rickets development as long as the individual's levels do not become critically low (Pitt 2002; Braegger et al. 2013; Davit-Béal et al. 2014). Clinical symptoms manifest due to increased unmineralised osteoid and tissue, poor quality bone formed, continued bone resorption, and development of fractures. Increased flexibility and reduced strength of the bones results in bending (Jaffe 1972; Stern 2005). Longstanding and severe vitamin D deficiency will lead to the development of hypocalcemia and potentially secondary hyperparathyroidism if hypocalcemia is severe and sustained (Jaffe 1940; Boyde 1993; Pettifor 2003). Hypophosphatemia is also noted to accompany vitamin D deficiency, and is an important factor in genetic and acquired rickets (Pitt 2002).

The identification of rickets in paleopathology is longstanding but recently Ortner and Mays (1998) produced a macroscopic definition and feature list for paleopathological study of the disease. Following the publication, a number of further studies were conducted on the topic including Blondiaux et al. (2002), Schamall et al. (2003), Mays et al. (2006), Ellis (2010), and Giuffra et al. (2013). An interesting thesis on post-medieval rural rickets was produced by Veselka (2012) but it only assessed the individuals using macroscopic features within a biocultural perspective. Other studies have focused on a particular aspect of rickets, for example Pinhasi et al. (2006) looked at the effect of rickets on growth while Mays et al. (2007) looked at hyperparathyroidism in a case of

rickets. The features of rickets used to identify cases in paleopathology include those utilised in clinical and radiological investigations as bone can be directly observed. The major identifying features of rickets include bending and changes at the metaphyses. A list of important paleopathological features is as follows.

1. *Bone deformities*

    a. *Mandible*: Some juveniles display medial and/or posterior bending of the mandibular ramus and condyle (Ortner & Mays 1998). The feature is considered rare. Ortner and Mays (1998) attribute the feature to chewing action. Clinically, the feature has not been reported.

    b. *Long bones*: Bending is most explicit in bones undergoing rapid development at the onset of rickets, see Figure 3.3B. To develop, pressure from weight bearing or tension from muscles and tendons is required to be exerted on the bone(s) but bending can occur at any point in the structure (Shore 2008; Shore & Chesney 2013). Children commonly learn to crawl between the ages of six to eight months and to walk at earliest eight months (Jaffe 1972; Guiffra et al. 2013). It is at these times that bending of the limb bones begins to be observed (Jaffe 1972; Guiffra et al. 2013). Bending in the upper limbs and tibia has been documented. Clinically, bending of the tibia is associated with pressure from the calcaneal tendon while the upper arms are associated with weight bearing during creeping or to support a seated position (Shore & Chesney 2013). Paleopathologically, Ortner and Mays (1998) have also associated upper arm bending with weight bearing during crawling. When walking, the lower limbs are weight bearing to support the body which results in bending as observed clinically. Bending of the knees is known as '*genu valgum'* (or knock-knee) when the knee is bent towards the midline of the body, or '*genu varum'* when the knee is bent away from the body. Along with '*coxa vara'* (bending of the femoral head), bending of the knee can result in significant difficulties to a child's ability to maintain a standing position and walk (Jaffe 1972). *Flattening of the femoral head* has also been documented (Mays et al. 2006). If the bending is very severe, the changes can become permanent (Mankin 1974a).

    c. *Ribs, ilium and other bones*: Bending is most obvious in the limbs but can occur in any bone if force is continuously applied. Ortner and Mays (1998) recorded deformities of the ribs and ilium. *Rib* bending appears as a more acute angle in their curvature (Ortner & Mays 1998; Mays et al. 2006). This change is a sign of severe disease (Jaffe 1972) and can cause respiratory difficulties (Adams 2005). *Pelvis* deformities, if extreme, can affect a woman's ability to give birth (Adams 2005). Clinically, the *spine* is a documented area with deformity typically

occurring in the thoracic (‘*kyphosis’*, often related to rib deformities) or lumbar vertebrae (Jaffe 1972; Shore 2008).

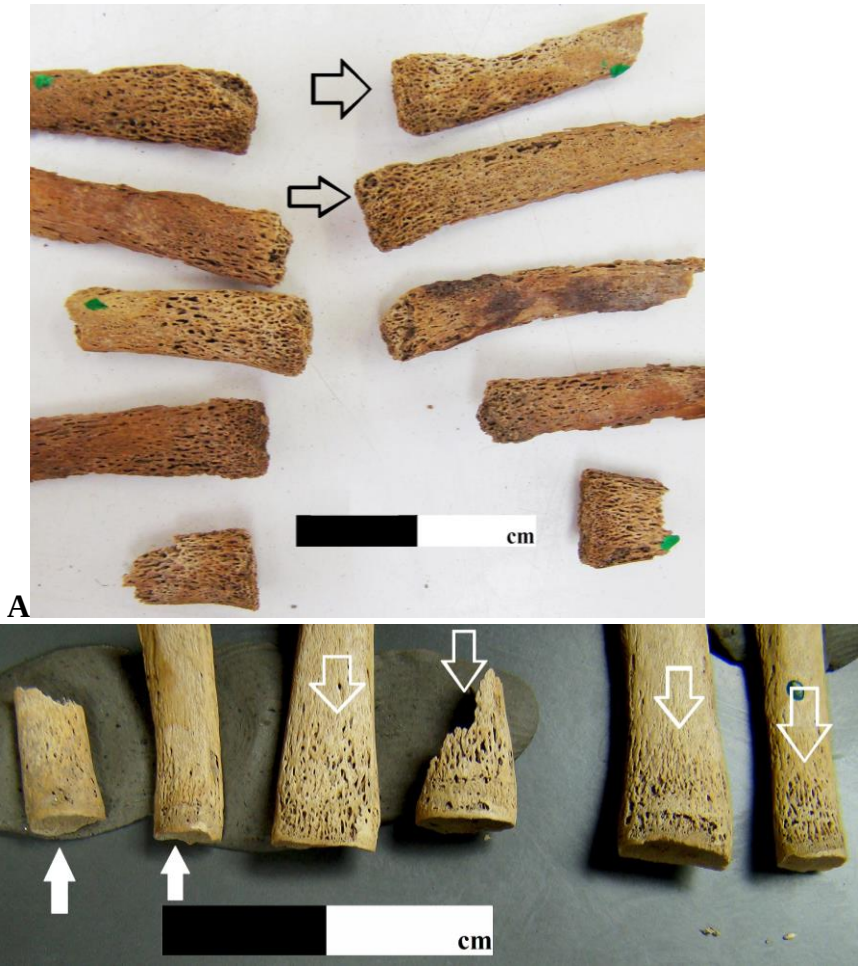


Figure 3.1. Rickets: flaring and porosity at the sternal rib ends. (A) Juvenile 624, significant porosity and flaring (arrow). (B)Juvenile 124, some porosity at the sternal ends (open arrows) and some normal ribs (solid arrows).

2. *Metaphyses of long bones and ribs*:

    a. *Flaring and cupping:* As with bending, flaring is most explicit in bones undergoing rapid development at the onset of rickets and, clinically, flaring is an early sign of rickets (Adams 2005). Widening, thickening, and flaring are caused by an accumulation of disorganised un-mineralised cartilage and cells in the metaphyses. Eventual collapse of poorly mineralised trabecular bone results in *‘cupping’* (Shore 2008; Shore & Chesney 2013). The extent of change is dependent on the amount of accumulated un-mineralised cartilage (Shore &

Chesney 2013). The feature can develop on any limb bone metaphysis and at the sternal end of ribs, called '*rachitic rosary'*, see Figure 3.1. In young infants, enlargement of the wrist (distal ends of the radius and ulna) as well as ankle occasionally occur while common sites in older children include the wrist (radius, ulna), knee (femur, tibia) and ankle (tibia, fibula) (Jaffe 1972; Ortner & Mays 1998). As for ribs, in early disease the sternal rib ends appear enlarged and assume the appearance of a bead with deformities as disease worsens (Jaffe 1972).

b. *Porosity of metaphyseal and sternal rib ends:* In paleopathology areas of abnormal porosity have been observed as bone can be directly studied. A pattern of pores amongst strands of bones, called '*slit/strut'* by Ortner and Mays (1998), appear on the dorsal aspect of the sternal end of ribs (see Figure 3.1.) and on the metaphyses of long bones, close to the growth plates. The pattern is similar to mesh but irregular. The porosity is explained as areas where unmineralised osteoid was once present in life and the strands of bone are the body's attempt to increase the overall strength of the bone (Ortner & Mays 1998). Porosity has not been investigated from a clinical perspective.

c. *Abnormal growth plates (fraying)*: Growth plates appear porous with unusual new bone formed on its surface as a result of continued endochondral growth and poor mineralization. Clinically, projections made of cartilage are visible in radiographs due to accumulation of material at the growth plate (Shore & Chesney 2013). Ortner and Mays (1998) devised a three step sequence to score the severity of change with appearance ranging from fine-grained to a coarse, roughened surface with porosity. The method was improved by Mays et al. (2006) who added a first step to the sequence which reflects early disease and describes the surface as having a 've*lvet'* appearance.

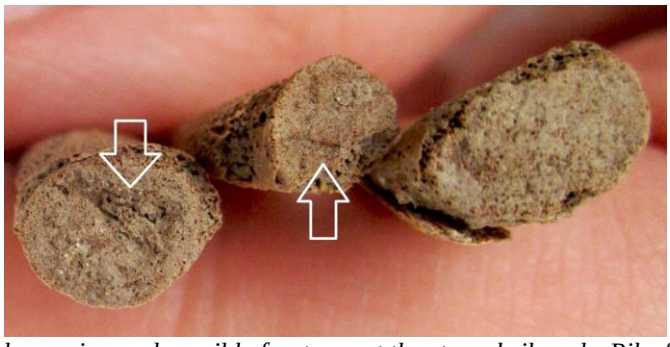


Figure 3.2. Rickets: depression and possible fractures at the sternal rib ends. Ribs from juvenile 37.

d. *Fractures*: Ellis (2010) documented compression fractures on the growth plates of lower limbs in archaeological skeleton from 19th century New York City.

Insufficiency fractures have been observed clinically in radiographs and are discussed below in Section 3.5. See Figure 3.2. for an example from Saint-Amé.

3. *Thickening*: Long bones and cranial bones may develop an enlarged, also described as thickened, appearance in paleopathology (Orther & Mays 1998).

4. *Porosity and hypertrophy of cranial bones*: In paleopathology, 'o*rbital porosity'* has been documented (Ortner & Mays 1998). Porosity, thickening and bossing are also observed on the bones of the cranium in both paleopathological and clinical settings (Jaffe 1972; Ortner & Mays 1998; Shore 2008). The frontal bone, typically the superior portion, and parietals becomes bossed. Deformities may also develop as the bones soften. For some children, the cranial bone becomes so soft they cave in, called *'craniotabes'*, when touched but return to normal once pressure is removed (Shore 2008).

5. *Impaired growth*: Growth can become impaired when rickets is severe and chronic, potentially masking rachitic symptoms (Adams 2005; Mays et al. 2006; Shore 2008).

6. *Porosity within curvature of bones*: Porosity may develop within the concave portion of a bent bone as a unique healing reaction to rickets. The reaction represents the body's attempt to increase a bone's strength and return it to normal (Mays et al. 2006).

A number of additional features can be observed in paleopathology. If a bone is broken, the trabeculae may appear thickened and coarse (Ortner & Mays 1998). Patches of woven bone and porosity around growth areas may be observed on nearly any bone due to the systemic nature of the disease (Ortner & Mays 1998; Giuffra et al. 2013). An interesting series of features which could be further explored in paleopathology regard dental development issues. Dentition usually begins to erupt at six months of age but rickets may delay eruption (Jaffe 1972; Gustafson & Koch 1974). Additionally, rickets can result in poor quality enamel formation, appearing as enamel hypoplasia and increase the risk of caries (Davit-Béal et al. 2014). Both deciduous and permanent dentition can be affected but changes develop during crown mineralization. Crown mineralization occurs between fetal and 12 months of age for deciduous dentition while permanent dentition mineralises between birth and eight years of age (Davit-Béal et al. 2014). Cases of healing rickets have been identified in paleopathology using macroscopic features including the absence of porosity and growth plate fraying, presence of porosity in the curvature of the bones, and additional supporting radiographic evidence (Mays et al. 2006).

Clinicians use many other features to identify rickets in children which cannot be used in paleopathology. Bending in the ribs may result in the protrusion of the sternum and Harrison grooves (Brodkin 1956; Jaffe 1972; Adams 2005). Fontanelles and cranial

sutures of young children may be widened, though the feature disappears once the openings close (Jaffe 1972; Guiffra et al. 2013). Muscle weakness may also develop (Root 1990).

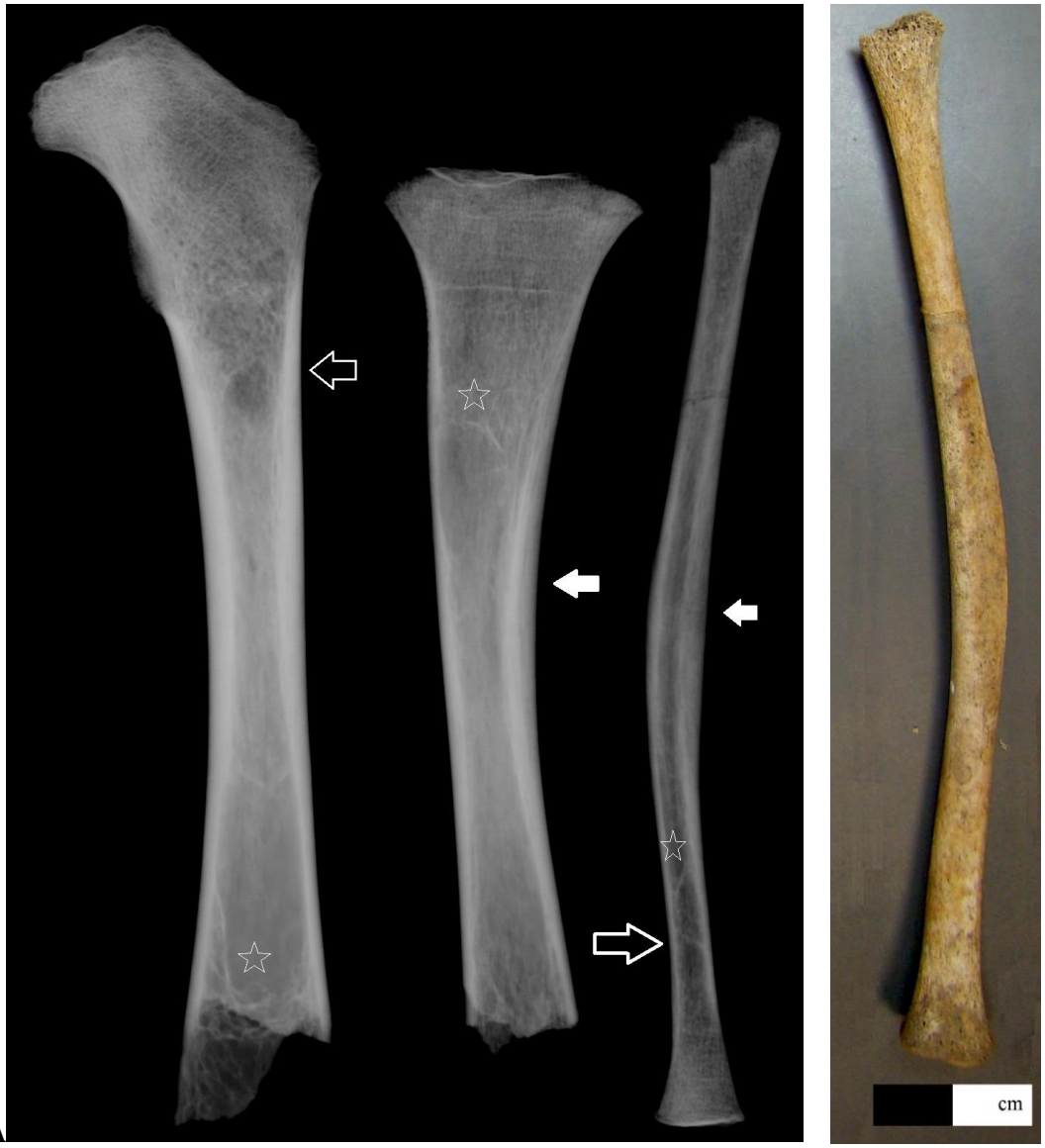
Treatments include oral or injected administration of vitamin D, calcium, phosphate and/or ultraviolet light exposure treatment to stimulate vitamin D synthesis. Exact values used are variable and complications such as renal disease may require very large dosages (Adams 2005). Vitamin D administration will activate the processing pathways and increase demand for calcium and phosphorus therefore these minerals may also be supplemented (Root 1990). Improvement is rapid with treatment. Serum levels should resolve within two weeks but radiographic features require two to three months (Adams 2005).

3.5. Radiographic Features

Bones undergoing the most growth at disease onset will present with the most pronounced alterations (Adams 2005; Shore & Chesney 2013). The most common areas affected and targeted for radiography in clinical research include the knee (distal femur and proximal tibia), the wrist (distal ulna and radius), sternal ends of the ribs, hip (proximal femur), ankle (distal tibia), and proximal humerus (Adams 2005; Shore & Chesney 2013). In clinical contexts, changes including bowing of long bones, widening and cupping of the metaphyses, and '*fraying'* are studied radiographically due to the inability to directly observe a patient's bones. The first radiographic change to occur within bone is diffuse osteopenia, followed by changes in the metaphyses, then by shaft changes (Shore & Chesney 2013). The metaphyseal ends is the typical focal point of clinical radiographic investigations (Shore 2008). In addition, clinicians can use the Thacher scale to evaluate severity of rickets and objectively quantify the amount of change occurring (Thacher et al. 2000). The focus of the evaluation is placed on changes observed at metaphyses, specifically fraying and cupping (Thacher et al. 2000).

The use of radiography to assist in identifying cases of rickets in a paleopathological context was established by Mays et al. (2006) though the technique had been used prior but relied on extensive clinical knowledge. Other paleopathological examples include Blondiaux et al. (2002), Schamall et al. (2003), Ellis (2010) and Giuffra et al. (2013). Nearly all radiographic features observed in a clinical setting can be observed in archaeological bone after accounting for diagenesis. Though, some such as cupping, metaphyseal enlargement and fraying can be better observed macroscopically in archaeological bone. Care should be taken when diagnosing disease with radiographs as diagenesis of the bone and soil infiltration can hinder the observer's ability to score the radiographic features in archaeological, potentially resulting in misdiagnosis. Diffuse osteopenia and trabecular coarsening are the most common radiographic features of

rickets in paleopathology (Mays et al. 2006). Following is a list of common paleopathological features.



A **B**

Figure 3.3. Rickets: radiographic features, Part 1. Juvenile 190. (A) Radiograph of the left femur, left tibia and left fibula. Features include coarse trabeculae (stars), areas of osteopenia (open arrows) and thickened cortex with periosteal apposition (solid arrow). (B) Macroscopic image of left fibula. Bending is clear and remnant of possible flaring at the distal metaphysis. Radiograph taken by the CAD at the Laboratoire d'Analyses Physiques et de Caractérisation des Matériaux, Direction de l'Archéologie, Communauté d'Agglomération du Douaisis.

1. *Diffuse osteopenia*: Appears as increased radiolucency in any bone but most commonly in limb bone diaphyses, metaphyses and epiphyses, see Figure 3.3. and 3.4. for slight examples. The overall effect is called "*moth-eaten*" to distinguish from the "*ground-glass*" appearance of scurvy (Bromer & Harvey 1948). The feature is caused by the built up of non-mineralised mature chondrocytes and osteoid as well as trabecular bone resorption and loss of distinct margins (including the growth plate) (Shore & Chesney 2013). It is one of the first features to develop though it is non-diagnostic. Changes in the epiphyses take longer to develop as growth is reduced (Shore 2008).

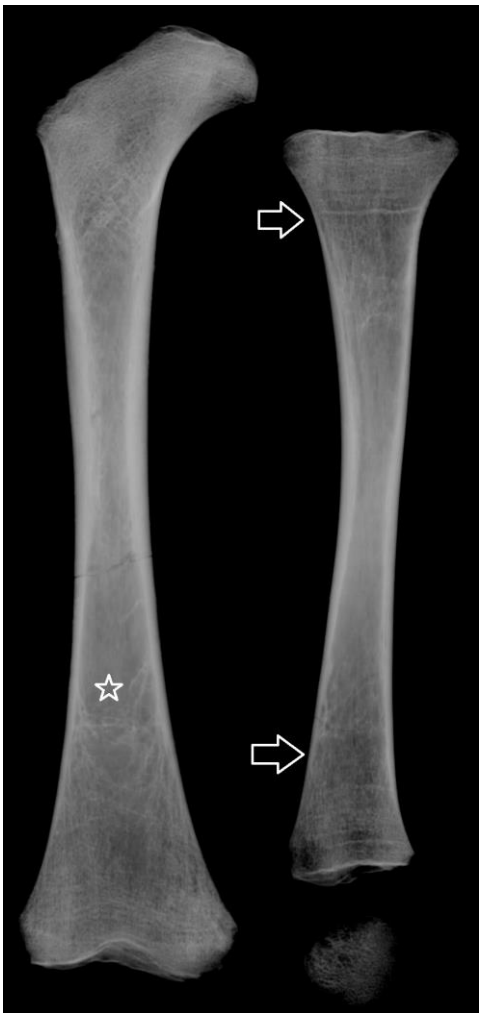


Figure 3.4. Rickets: radiographic features, Part 2. Juvenile 190. Radiograph of the right femur and right tibia. Features include coarse trabeculae (star), thickened cortical bone on the left side of the tibia. The epiphysis (lower right corner) is poorly preserved. Radiograph taken by the CAD at the Laboratoire d'Analyses Physiques et de Caractérisation des Matériaux, Direction de l'Archéologie, Communauté d'Agglomération du Douaisis.

2. *Cortical thickening*: New bone formation is a sign of healing and develops within the bends of the bones to increase support and strength (Mays et al. 2006; Shore & Chesney 2013), see Figure 3.3. and 3.4. Mays et al. (2006) also observed trabecular thickening within curvatures when bending was extreme.

3. *Coarsening of the trabecular bone:* Within the shaft, the small secondary trabeculae, oriented perpendicular to the loading forces, become radiolucent, emphasizing the appearance of primary trabeculae, oriented with the loading forces (Bonakdarpour 2010), see Figure 3.3. and 3.4. The change produces the impression of coarsening in the remaining trabeculae.

4. *Fraying*: Clinically, the feature is assessed radiographically but in paleopathology the feature can be scored macroscopically.

5. *Fractures:* Fractures are not extensively documented in paleopathological radiography but noted clinically. Decreased amount of mineralised bone, decreased rigidity, and abnormally high amount of

osteoid increases the risk of fractures, particularly insufficiency fractures (Shore & Chesney 2013). Insufficiency fractures are a result of normal physiological stress on abnormal bone (Pentecost et al. 1964). Looser's zone (pseudofractures) is a feature of severe osteomalacia and can be observed, though rare, in juveniles (Adams 2005). When fractures occur, new bone will develop at the site in an attempt to repair the fracture but will appear very poorly formed. However, Shore and Chesney (2013:168-169) discuss that the argument for vitamin D deficiency causing fractures is debated is currently being debated in the literature and a number of studies did not find an increase in fractures or severe bone loss. One study mentioned, Chapman et al. (2010), found fractures only in their sample's most severe, clear cases of rickets but the fractures did not occur around the growth plate, instead further along the bone in the metaphysis and diaphysis. Fractures may therefore help support a diagnosis of rickets but alone may indicate other ongoing processes.

Healing can also be observed radiographically by a reversal from diseased state to normal. Normal trabeculae appear at the ends of the metaphyses, the cortex thickens, and definition of the margins return (Brickley & Ives 2008).

3.6. Microscopic Features

Microscopy is a longstanding technique used to study rickets in clinical medicine, but with biochemical innovations the technique is now less frequently used in diagnosis of infants and young children (Koo 1996). Sections of bone from ribs (typically the sternal ends) and/or the metaphyses of long bones were historically used to study histological changes in experimental animal research and patients (e.g., Sherman & Pappenheimer 1921; Maxwell & Turnbull 1932; Ranström & Sydow 1949). The use of these areas allows researchers and clinicians to directly observe rachitic changes related to growth. A limited number of studies employ diaphyseal bone to assess changes in already formed juvenile bone (e.g., Villanueva et al. 1963). Today, microscopic studies of rickets use standard hip biopsies instead of rib or metaphyseal bone as sampling procedure are less invasive, the bone is easy to access, and it is less susceptible to changes from stress (e.g., Marie et al. 1982; Raubenheimer et al. 1997). Histomorphometry is also routinely used in clinical research. The technique allows researchers to quantify the amount of change present and over time, using static and dynamic measures on thin sections of bone (e.g., Parfitt 1988; Glorieux et al. 1991; Beyers et al. 1994; Priemel 2010; Dempster et al. 2013).

Microscopy has been used in paleopathological investigations since the 1920s (Garland 1993) and a detailed review of paleohistology history, use and terminology was produced by Schultz (2001) and de Boer et al. (2013). Recent paleopathological studies on rickets from a microscopic perspective includes Schamall et al. (2003) who examined samples of vertebrae from individuals with rickets and osteomalacia held in medico-historical

collections and Mays et al. (2006) who investigated the appearance of the metaphyseal growth plates and confirm a diagnosis of secondary hyperparathyroidism later reported in detail by Mays et al. (2007). The studies have used a variety of microscopic techniques including light microscopy (Schamall et al. 2003), scanning electron microscopy (SEM) (Schamall et al. 2003; Mays et al. 2006; Mays et al. 2007), and histomorphometry (Schamall et al. 2003). SEM back-scattered electrons imaging (BSE-SEM) is particularly useful in assessing rickets as changes in mineral density of bone can be directly observed. Static histomorphometry could be utilised in paleopathological studies but dynamic histomorphometry is impossible as the technique requires live bone to absorb tetracycline labelling which is then measured at a later time point. Taphonomy and diagenesis are important limiting factors in microscopic analysis. Discussion on the topic is provided in Section 7.2.

Paleopathological study of rickets can assess change at both the metaphysis and diaphysis. A detailed discussion on metaphyseal specific features will not be provided here as it falls beyond the scope of the project but readers may refer to works such as Jaffe (1972) and Mankin (1974a) for additional details. Briefly, rachitic changes at the metaphyseal ends or growth plates of bones focus on the cellular organization and structure of the area. Much emphasis is placed on tissues and cells which do not preserve well in the archaeological record. Important changes include alterations in cartilage cells (increased in size, failure to differentiate and mineralise; disorganised columnar structure, cells extend towards the diaphysis, accumulation of cells due to failure to proceed with apoptosis, and un-mineralised cartilage in the zone of preparatory calcification), vascular penetration of bone becomes disorganised, marrow may appear fibrous, and other observations on osteoblast cells (Sherman & Pappenheimer 1921; Jaffe 1972; Oppenheimer & Snodgrass 1980; Shore & Chesney 2013). The build-up of cells and tissues results in the widening of the metaphyses. In the current study, diaphyseal bone was employed. An explanation for the selection can be reviewed in Chapter 5, Section 5.3.3.3. The use of diaphyseal bone indicates that osteomalacia was reviewed following the definitions discussed in Section 3.2. However, any observed evidence of osteomalacia can still be used to support a diagnosis for vitamin D deficiency as the disease is present in juveniles affected by rickets. Below is a list of features expected when investigating diaphyseal bone for evidence of osteomalacia.

1. *Increased osteoid:* Clinically, the principal microscopic feature of rickets and osteomalacia is an increase in volume of osteoid due to failure of the mineralization process (Rauch 2003). Increased osteoid is not pathognomonic as osteoid naturally occurs in areas undergoing bone formation or remodelling. Additionally, diseases which increase bone turnover or affect mineralization can result in increased osteoid (Villanueva et al. 1963; Pitt 2002). Unmineralised osteoid is unlikely to preserve in the archaeological record (Mays et al. 2007) but remnants of buried osteoid seams (trapped within bone) can be recorded. Buried osteoid seams will appear as poorly

mineralised bone or open slits (See Figure 3.5., 7.1.). Mankin (1974a) believes thickened osteoid seams (non-buried) are a cardinal feature of bone changes in osteomalacia. The number of seams can indicate disease severity (Mankin 1974a, Pitt 2002).

a. *Osteoid seams and trabecular bone*: When affected by rickets and osteomalacia, thickened osteoid seams along the trabeculae can be observed (Mankin 1974a; Pitt 2002). Figure 5.4. shows the location of osteoid along trabecular bone. The change is most prominent in trabecular bone as this type of bone develops faster than cortical bone (e.g., Raubenheimer et al. 1997). The feature is very commonly used to diagnose rickets or osteomalacia. Rauch (2003) states a minimum osteoid thickness of 9μm is required to diagnose a child. Archaeologically, un-mineralised osteoid along the trabecular bone is unlikely to preserve as it remains exposed.

b. *Osteoid seams and cortical bone*: Similar to trabecular bone, osteoid along cortical bone has been noted in neonatal rickets (Oppenheimer & Snodgrass 1980). Seams can also occur in haversian systems of cortical bone (Frost 1962; Mankin 1974a). These seams can be classified as either active or resting. Active seams are biologically active, undergoing mineralization, contain osteoblasts, and the surrounding bone is usually well, but not completely, mineralised (Frost 1962). *Active osteoid seams* occur naturally and the number is highest in infancy, declining with age. Villanueva et al. (1963) found that in rib diaphysis of juveniles under two years of age have on average 5.43 active seams per mm^2 of cortical bone with a standard deviation of 3.29. Between two and ten years of age, the values decrease to an average of 2.38 seams with a standard deviation of 1.40. Increase in width of active seams is suggestive of osteomalacia (Pitt 2002). *Resting seams* are biologically inactive, have been quiescent for many weeks, have no osteoblasts, and cannot be labelled with tetracycline labelling (Frost 1962; Schen et al. 1965; Haas et al. 1967). Frost (1962) suggests inactive seams develop as a result of "great physiological stress" or disrupted mineralization processes. Inactive seams can become '*buried* ' when the seams becomes surrounded by bone, see Figure 7.1. Buried osteoid seams are usually observed to be surrounded by well mineralised bone suggesting the individual is undergoing healing from rickets and osteomalacia, or a previous episode of such a condition occurred in the past (Teitelbaum et al. 1976; Priemel et al. 2010). Frost (1962) also found that osteoid seams along circumferential lamellae are unusual in 'normal' children and adults. Buried osteoid seams are typically long and wide but the exact length and width defining a buried osteoid seams is uncertain.

2. *Osteopenia and areas of poor mineralization:* Any bone formed during disease will be poorly mineralised in both cortical and trabecular bone (Brickley & Ives 2008) (see Figure 3.2.).

a. *Irregular haversian channels*: Cortical bone reacts to vitamin D deficiency by becoming thin and porous. Haversian channels become larger and both the cavity and organisation of the haversian system may become irregular in shape, losing the typical circular appearance (Engfeldt et al. 1956; Jaffe 1972; Pitt 2002; Brickley & Ives 2008). However, some irregularity is expected in infants and children as the bone is rapidly growing and microscopically shifting from cortical to trabecular.

b. *Thin and fewer mineralised trabeculae*: Uninterrupted resorption processes without proper mineralisation of new bone results in thin, irregular shaped and organised trabecular bone (Oppenheimer & Snodgrass 1980; Marie et al. 1982; Pitt 2002). However, trabecular bone can appear thick clinically due to the development of thick osteoid seams. In paleopathology, osteoid is unlikely to be preserved therefore thin trabeculae should be expected.

c. *Howship's lacunae*: Osteoclastic resorption of bone leads to the development of Howship's lacunae which appear as multiple, irregular semi-circular indents into bone or 'bite marks' along the bone (See Figure 3.5 and 3.7.). If rickets or osteomalacia is severe and thick osteoid covers the bone surface, resorption by osteoclasts is thought to halt or be reduced (Raubenheimer 1997). Oppenheimer and Snodgess (1980) also found a decrease in number or absence of osteoclast cells present in neonatal rickets. Nonetheless, Raubenheimer's (1997) case study of children (3 to 15 years of age) affected by rickets and osteomalacia found an increased number of osteoclasts and amount of resorption despite significant osteoid accumulation. The author ascribes the increase to the development of secondary hyperparathyroidism, linked to severe rickets, which promotes osteoclasts and increases the manifestation of the Howship's lacunae (Brickley & Ives 2008).

d. *Bearded osteocytes*: Similar to the buried osteoid seams, poor mineralization of the cortical bone around osteocyte lacunae and canaliculi is observed in cases of vitamin D resistant rickets (Steendijk et al. 1965; Steendijk & Boyde 1973). The poorly mineralised bone, called 'beards' by Steendijk and Boyde (1973) will develop in globular shapes and be oriented to where new bone development occurs. Size was normal for both structures (Steendijk et al. 1965).

e. *Cement lines*: Poorly formed bone has also been commonly observed along cement lines (Brickley & Ives 2008).

f. *Cloudy zones and separation of new bone from mature bones*: Bonucci et al.'s (1969) study shows that some calcification occurs in osteomalacic bone as cloudy,

granular areas of bone were observed in microscopic sections, see Figure 3.5. and 3.7. The granular and cloudy appearance is caused by incomplete deposition of minerals on osteoid. Similar to osteoid seams, if this feature is present at the end of well calcified matrix or is '*buried*' it should be preserved archaeologically. Bone is formed by the addition of layers over time. Poorly formed bone, as in rickets and osteomalacia, may improperly fuse to the older bone (Bonucci et al. 1969; Brickley & Ives 2008). The new bone will appear as bony projections and may fail to fuse with other strips of bone further along the formation front, giving the impression that mineralization has "skipped" ahead (see Figure 3.6. and 3.7.).

3. *Insufficiency fractures*: Experimental research found spontaneous fracturing near the costochondral junctions of lower ribs in rachitic rats (Sherman & Pappenheimer 1921; Shore & Chesney 2013) and has been observed in metaphyses of long bones in archaeological remains (Ellis 2010). Fracturing may be observed histologically if sections of the metaphyses are studied. Pseudofractures (also called Milkman's fractures and Looser's lines) are considered near pathognomonic of osteomalacia (Mankin 1974a).

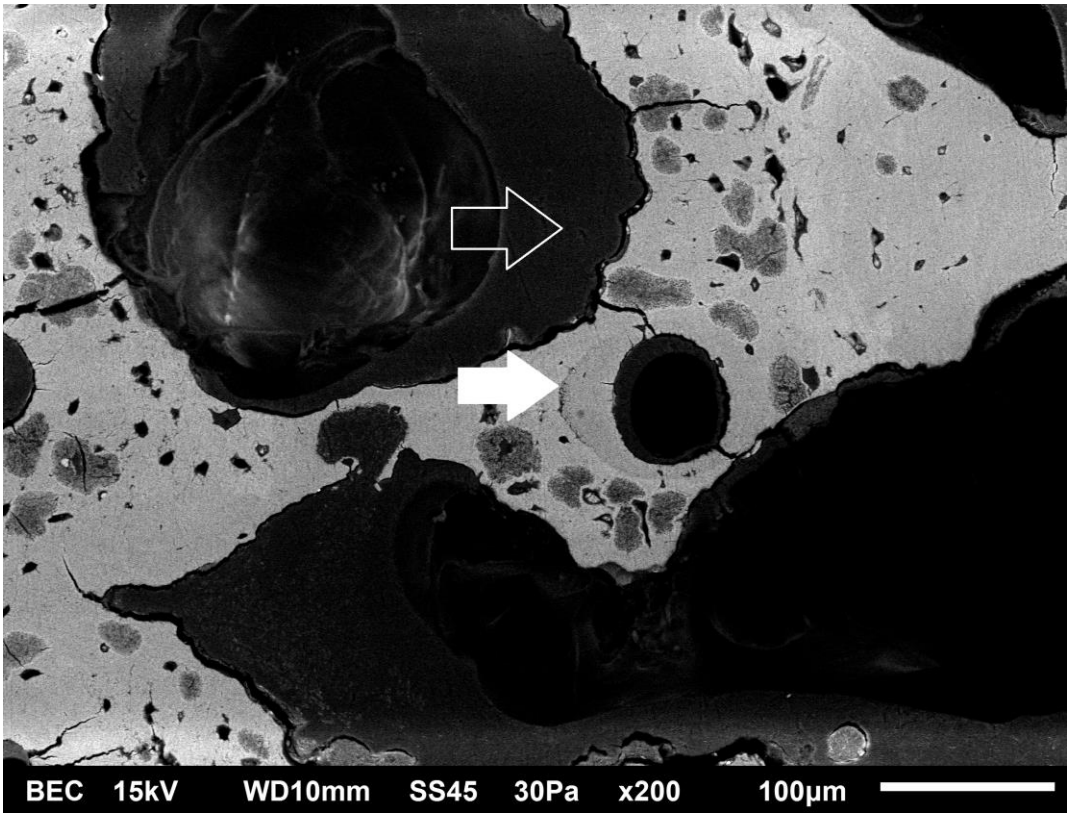


Figure 3.5. SEM section x200, juvenile 647. Solid arrow points to buried osteoid with surrounding cloudy zone of slightly lower mineralised bone. Open arrow points to Howship's lacunae. Diagenetic change can be seen as the very dark spots.

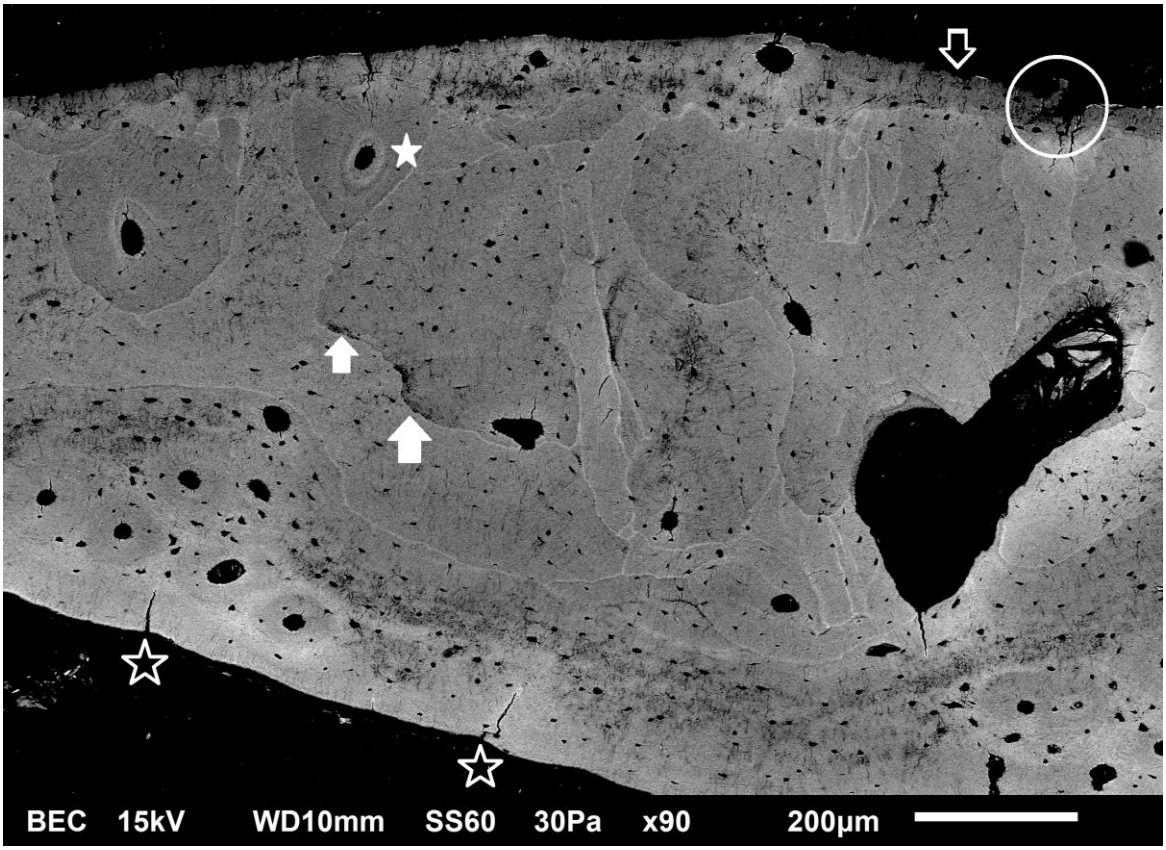


Figure 3.6. SEM section x90, juvenile 473. Solid in arrow image points to buried osteoid having undergone some mineralisation, resulting in a cloudy zone. Open arrow points to a poorly mineralised border. Many of the osteons appear to be less mineralised, an example is highlighted by the solid star. The circle appears to contain newer bone which is separating from the older bone. The open star points to cracks in the bone caused by the embedding process.

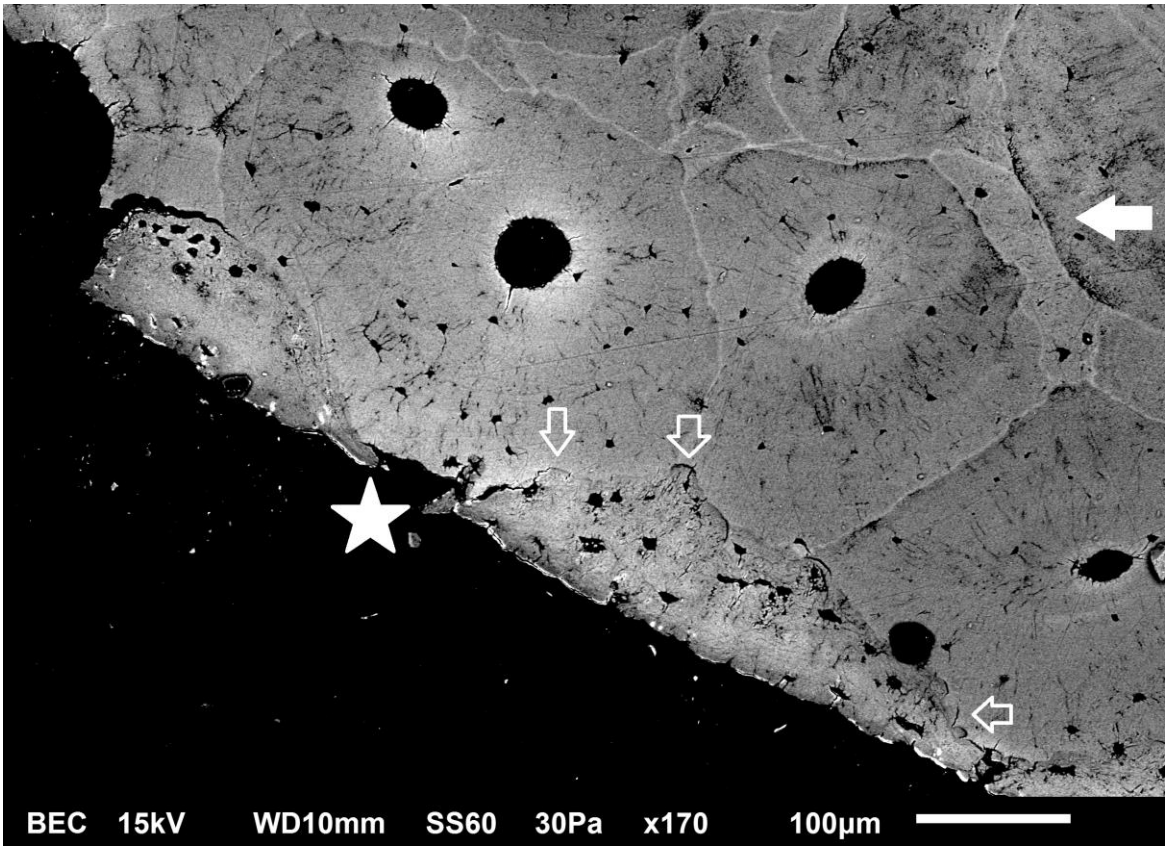


Figure 3.7. SEM section x170, juvenile 473. Solid arrow points to buried osteoid, possible seam, with some mineralization resulting in cloudy zones. Surrounding area is also less mineralised. Star highlights a possible area of poorly attached new bone. Open arrow points to older Howship's lacunae which have been covered by a new layer of bone.

3.7. Conclusion

Vitamin D deficiency is characterised by poorly mineralised bone. Poor mineralization causes the classic feature of bending as well as a host of others which can be observed macroscopically, radiographically and microscopically. The majority of features described in clinical cases can be observed in paleopathological cases. However, un-mineralised tissues are unlikely to survive in an archaeological setting. It is unfortunate considering the large role of un-mineralised tissue on the development of this disease. The chapter defined rickets, discussed the biological consequences of vitamin D deficiency, its causes, and explored both the clinical and paleopathological presentation of rickets (including clinical, radiographic and microscopic assessments) with the perspective on how the disease has been identified in previous literature.

Chapter 4: Background – The Co-Occurrence of Scurvy and Rickets

4.1. Introduction

The chapter will briefly explore current understanding of disease co-occurrence in paleopathology. An in-depth discussion will follow on reported cases of rickets and scurvy co-occurrence in the medical literature and documented disease expression from a clinical, radiographic and microscopic perspective. Hypotheses and ideas proposed regarding how the medical appearance of infantile scurvy and rickets co-occurrence may translate to an archaeological setting are also addressed.

4.2. Frameworks and Past Examinations of Co-Occurrence in Paleopathology

The co-occurrence of multiple diseases in a single individual is an interesting topic for the field of paleopathology and has not yet been systematically explored though cases have been suggested. As such, a theoretical framework to study disease co-occurrence and interaction has yet to be defined for an archaeological context.

In medical anthropology, Singer (2009) developed the syndemic approach from the critical biocultural approach to address the interaction between multiple diseases, the consequences of interaction, and how co-occurrence affects and is affected by social, political, cultural, biological and environmental factors. The intertwined factors produce a synergistic or 'syndemic' relationship. The experience of disease is studied at both the individual and population levels (Singer 2009; Singer & Clair 2003; Singer et al. 2011). The approach is useful to consider as it foregrounds the idea that multiple disease processes can act on a single individual or population within a broad context of factors and variables which shape disease expression and vulnerability to disease. The approach is also used to understand the boundaries and overlap of the diseases from a biological, social, and environmental perspective (Singer 2009). The holistic approach is important for the study of co-occurrence of disease in paleopathology. In an archaeological context, detailed social and environmental information is often limited. Therefore, exploration of the larger ideas promoted by the syndemics approach may be difficult. However, identification of disease co-occurrence cases should provide clues to the biological, sociocultural, and environmental conditions as diseases overlap in particular situations favourable to the development of those diseases. Developing a greater understanding of the variable expression and paleopathological appearance of disease co-occurrence is critical for the identification of such cases in paleopathology.

Recently, paleopathological reports have started to specifically mention and suggest cases of disease co-occurrence. Unfortunately many diseases do not affect the skeleton meaning many cases of co-morbidities will be missed by only looking at skeletal evidence. DNA is an exception and a useful means of exploring the presence of multiple diseases. Bianucci

et al. (2012) worked to confirm textual evidence suggesting Eleonora of Toledo (1522-1562) was affected by tuberculosis (TB) and died of malaria. Their analysis confirmed the presence of TB and found *Leishmania infantum* which commonly co-occurred with malaria prior to eradication (Bianucci et al. 2012). Two other recent studies using paleomolecular investigations (DNA) found co-infections of TB and leprosy, and TB and hepatitis B (Donoghue et al. 2005; Matheson et al. 2009). Additionally, technological advances in ancient DNA research are developing better opportunities to find co-morbidities of infectious diseases in ancient remains (e.g., Devault et al. 2014).

Recent studies on scurvy and rickets have found possible examples of co-occurrence but a detailed analysis was not completed. Further discussion of these cases is available in Section 4.5.

4.3. Medical Research on the Co-Occurrence of Scurvy and Rickets

Medical research has been produced on the topic of scurvy and rickets co-occurrence and a handful of cases have been reported (see Table 4.1). The first consideration for co-occurrence of scurvy and rickets in children within the medical literature was proposed by Francis Glisson (1650). Case studies were later reported by Cheadle (1878), Barlow (1894), and Owen (1899). Radiographic considerations and descriptions were produced by Bromer and Harvey (1948), while microscopic work was completed by Follis et al. (1940).

4.4. Clinical Presentation

Scurvy and rickets intersect when a culture or society has risk factors for both conditions. Vitamin C deficiencies are typically due to an inadequate diet, insufficient gut absorbency, and poor access to fresh fruits and vegetables (see Chapter 2 for details). The development of rickets is dependent upon sunlight exposure and vitamin D intake, as well as a balance of other nutritional elements (see Chapter 3 for details). Prevalence of co-occurrence cases is influenced by risk factors and available treatments. Initial mention of the disease co-occurrence appears in the 1600s but many case studies of scurvy and rickets co-occurrence were only published in the late 1800s and early 1900s. The number of reported cases declined significantly after the early 1900s as disease understanding increased, fortified foods were developed, and easy, efficient treatments became readily available (Bromer & Harvey 1948; Still 1935; Holick & Chen 2008). Rickets and scurvy are now considered rare conditions which develop only in peculiar situations. Recent cases of scurvy and rickets have been documented in refugee and ethnic minority populations (Lewis et al. 2006) and developmentally disabled individuals (Noble et al. 2007).

Context is critical to the development of scurvy and rickets co-occurrence. Co-occurrence of scurvy and rickets can occur at any age and development is dependent upon multiple factors including body stores of ascorbic acid, breast milk quality and quantity, sunlight exposure, and weaning age and foods. Early cases reported by Barlow (1894) and Cheadle (1878) typically occur between six and 18 months of age but full range of reported cases varies from four months to three years, possibly up to five years (see Table 4.1). Infants and young children are also more vulnerable to metabolite deficiencies as they are undergoing growth and use up their stores rapidly (Lewis 2007).

Diet is one of the most important factors for developing a co-occurrence of vitamin C and D (Cheadle 1878; Chick & Dalyell 1921). The use of alternative food to breast milk and weaning foods poor in the vitamins such as animal milk, cereals or formula lacking adequate vitamin C and D increases the risk of developing both scurvy and rickets in young children (Chick & Dalyell 1921; Fouron & Chichoine 1962; Demers et al. 1965). Whole grains and certain cereals are high in phytates which inhibit calcium intake (Harrison & Mellanby 1939; Lawson et al. 1999; Pettifor 2004), a risk factor for rickets, and are poor in vitamins unless supplemented. However, yeast, a component of leavened breads, can breakdown phytates (Nävert B et al. 1985; Türk et al. 1996). Infants and children also have vulnerable immune systems, increasing their risk of acquiring infectious and diarrhoeal diseases which can inhibit the absorption of important nutrients, causing deficiencies (Lewis 2007:100). After weaning, a diet of food rich in vitamin C and vitamin D is essential, though vitamin D is also produced from UVB exposure.

Socioeconomic status is another risk factor as it may limit access to vitamin C and D by restricting the types of products consumed and encouraging particular behaviours. Groups from varied socioeconomic backgrounds have been impacted by scurvy and rickets across time and space. In some cases, a lack of wealth prohibits access to vitamin D and vitamin C. For example, in Europe, the poor were often affected by scurvy until the introduction of the potato, a vegetable which can be efficiently stored and high in vitamin C, between the 16th and 17th centuries (Still 1935; Love & Pavek 2008; Geber & Murphy 2012). During the same period and the industrial revolution, the urban poor have lived in small, cramped, polluted quarters which increase the risk of vitamin D deficiency and had little money to purchase calcium rich foods (Cheadle 1878). The majority of reported co-occurrence cases by Barlow (1894) were from individuals of poor social status but a number of cases occurred in children from wealthy backgrounds. Early versions of baby formula were deficient in vitamin C. However the product was expensive and promoted by physicians to wealthier families who had the means to purchase the novel product for their children (Barlow 1894; Fomon 2001; Lomax 1986). Additionally, the children of the wealthy were confined to the indoors during the medieval to 17th century, significantly increasing the risk of developing rickets (Gibbs 1994; Mays 2003; Guiffra et al. 2013). Cheadle (1878) also found that the middle class were at increased risk as they refused to consume potatoes as the act was perceived to be below their social rank but the families

could not afford much milk which Cheadle believed to have antiscorbutic properties. Animal milk does contain some vitamin C but amounts are very low (Saxholt et al. 2009; USDA. 2014) and a child's diet would need to be supplemented with other foods rich in vitamin C to avoid scurvy. Food high in vitamin C may have been available to members of the upper class but too costly or for other reasons unavailable to other social groups. Swaddling infants was commonplace in the medieval and renaissance periods which would have limited accessibility of vitamin D (Senoir 1983:377; Shin et al. 2009; Guiffra et al. 2013). Avoidance of certain activities, foods for cultural practices, beliefs or personal agency can expose children to risk factors for co-occurrence (Pettifor 2004).

Environmental factors could also affect disease onset. Rickets is most common in the winter months as the solar zenith angle allows fewer UVB rays to penetrate the atmosphere and days are shorter, resulting in decreased vitamin D access (Pettifor 2003). Scurvy is typically believed to occur more commonly in the winter and spring due to poor availability of fresh food but reports have been variable and large numbers of cases developed early symptoms of scurvy during the summer months though no explanation has been proposed (Dogramaci 1946; Fouron & Chichoine 1962). Pollution similarly reduced UVB penetration (Gibbs 1994). Disasters such as unusual weather patterns and harvest failures will also limit access to the essential vitamins.

TABLE 4.1. Diagnosed cases of scurvy and rickets co-occurrence from the medical literature.

Number of Co-occurrence Cases	Age of onset	Sample	Means of Diagnosis	Source
3	16 months to 3 years	Cases from own clinical practice	*Scurvy and Rickets*: Clinical observation and patient history	Cheadle 1878
19 minimum	Earliest reported: 4 months, Observed by Barlow: 4 months to 3 years Typical: around 15 months	Cases from clinical practice and literature	*Scurvy and Rickets*: Clinical observation, patient history, autopsy	Barlow 1894
1	12 years old	Case from own clinical practice	*Scurvy and Rickets*: Clinical observation and patient history	Owen 1899
2 minimum	Approximately 1 or 2 years old	Case from own clinical practice	*Scurvy and Rickets*: Clinical observation, patient history and diet.	Chick & Dalyell 1921
9/19 autopsies	2 months to 2 years (sample of 532 children)	Children hospitalised for a variety of conditions	*Scurvy and Rickets:* Histology	Park et al. 1935
26/57 autopsies of children (45.6%), authors suspect more present but unconfirmed.	3 to 19 months	Series of children with scurvy, then diagnosed rickets	*Rickets*: Clinical observations, radiography, histology, and serum calcium, phosphorus *Scurvy*: Histology (confirmed)	Follis et al. 1940
15/186 infants (8%)	Infants (unspecified)	A series of children with scurvy	*Rickets*: Clinical observations (confirmed diagnosis with craniotabes)	McIntosh 1945, cited by Bromer & Harvey 1948:1
36/93 infants (38.7%)	Infants (unspecified)	A series of children with scurvy	*Rickets*: Autopsy observations	Evans 1945, cited by Bromer & Harvey 1948:1
1	3 months to 2 years	Case from own clinical practice	*Scurvy and Rickets*: Clinical observation, patient history, radiography, and biochemical tests	Bromer 1946

TABLE 4.1. (Continued)

Number of Co-occurrence Cases	Age of onset	Sample	Means of Diagnosis	Source
5/38 children (13%)	under 2 ½ years of age	A series of children with scurvy	*Rickets*: Biochemical test *Co-occurrence*: Radiography	Fouron & Chichoine 1962
6/69 cases of scurvy	Not specified, between 4 months and 5 years of age	Juvenile scurvy cases reported to the Canadian Pediatric Society 1961-1963	*Scurvy:* Clinical, patient history, radiography and effective treatment *Rickets*: Details not provided	Demers et al. 1965
1	11 months	Case from own clinical practice	*Scurvy and Rickets*: Clinical observation, radiography, patient history, confirmed by biochemical testing	Lewis et al. 2006

4.4.1. Physical Findings

The presentation of co-occurrence is highly variable, and co-occurrence is not associated with any pathognomonic features. Appearance of scorbutic and rachitic signs and symptoms are dependent on the order and length of disease development prior to the development of the secondary illness (Follis et al. 1940). Cases of co-occurrence are identified as they present with features of both rickets and scurvy. The works by Glisson (1650), Cheadle (1878), Barlow (1883), Follis et al. (1940) and Fouron and Chichoine (1962) highlight the aetiology, signs and symptoms of rickets and scurvy co-occurrence from a medical (patient and autopsies) perspective.

The majority of medical cases report that scurvy typically supervenes over rickets (Cheadle 1878; Barlow 1883; Bromer & Harvey 1948). In such cases, clinical descriptions detail that features of rickets are either mild or unobservable, but most of the features associated with scurvy are clearly present (Cheadle 1878; Bromer & Harvey 1948). The features are typically bilateral (Owen 1899). See Table 4.2 for a list of reported scorbutic symptoms and signs observed in cases of co-occurrence. If scurvy becomes resolved but the individual remains affected by rickets, clear rachitic features can develop with the resumption of both growth and bone forming processes previously inhibited by scurvy (Barlow 1883; Fouron & Chichoine 1962). Such a phenomenon is called rickets liberation ("liberation du rachitisme") (Fouron & Chichoine 1962). Barlow (1883) discusses at least 22 cases of co-occurrence observed clinically. Features of rickets were clearly observable in only three cases, seven had marked features and nine only developed a slight expression. Barlow (1883) performed three autopsies as part of his report and all three individuals showed 'marked' to 'clear' evidence of rickets but the exact cases are not clearly specified. In another three cases no evidence of rickets could be observed. Clearly, significant variation in degrees of rickets feature expression should be expected. Barlow (1883) also admits that in 19 of the cases where some evidence of rickets was observed, the only rickets feature recorded was beading of the ribs, a feature which can develop in scurvy. Similarly Cheadle (1878) makes a case for the presence of rickets on the presence of beading in the ribs. Unfortunately, radiographic, microscopic and biochemical testing was not available at the time for further analysis. Other cases diagnosed by Barlow (1883) showed such features as enlarged epiphyses and metaphyses as well as retarded dental eruption. See Table 4.3 for a list of rachitic symptoms and signs observed in cases of co-occurrence.

The mild expression or absence of rachitic features is explained by the interplay of scurvy and rickets inhibiting one another's mechanism within the skeletal system (Follis et al. 1940; Bromer & Harvey 1948; Fouron & Chichoine 1962). Rickets causes a defect in the mineralization process but osteoid is normal. Scurvy produces pathological osteoid and reduces osteoblastic activity but maintains a normal mineralisation process. The majority of classic rachitic features require some osteoid for growth or remodelling to develop

(e.g., softening and bending of the limbs, flaring of the metaphyses) which is inhibited by the presence of scurvy. When rickets occurs first, the features may have time to develop prior appearance of scurvy but this is not assured. However, rickets, if dominant, could mask classic scurvy feature of new bone formation due to poor mineralisation, and reduce the clinical symptoms of pain and tenderness caused by scurvy (Follis et al. 1940; Fouron & Chichoine 1962). Bony features of either disease are not clearly visible unless the one of the conditions occurred first and had some time to develop prior to the appearance of the secondary disease (Shore 2008). Classic scurvy etiology also affects the vascular system causing hemorrhages, swelling, and inflammation. The development of these features would not be disrupted by a co-occurrence with rickets.

TABLE 4.2. Clinical features typical of scurvy present in cases of reported co-occurrence. Dash (-) = No comment.

Features of Scurvy	Conditions	Source	Observe in Paleopathology
Skin: pale, shallow tint	Common	Barlow 1883; Owen 1899	No
Cachexia (weakness, weight loss, muscle atrophy, flabby muscles)	Often fatal if extreme, but moderate symptoms are common	Barlow 1883; Cheadle 1878	Indirectly, with time may result in osteopenia
Irritable, fretful, dislike handling	-	Cheadle 1878; Owen 1899; Bromer 1946	No
Fever	Linked to amount of swelling; Common	Cheadle 1878; Barlow 1883	No
Purpura (subcutaneous hematoma)	-	Barlow 1883; Bromer 1946	No
Subperiosteal hemorrhages	Extensive involvement, typically tibia, femur, fibula; Common	Barlow 1883; Bromer 1946	Yes
Swelling of the limb bones and joints	Most bilateral and/or multiple bones affected, typically lower limbs but wrists and ankles in very young; Occasional	Cheadle 1878; Barlow 1883; Owen 1899; Lewis et al. 2006	Yes
Immobility of legs	-	Owen 1899	No
Swelling and bleeding of the gums, loosening of teeth	Range from slight to significant, only develop ecchymoses when teeth are erupting/erupted; Occasional	Cheadle 1878; Barlow 1883; Owen 1899; Bromer 1946	Yes
Fractures	From separation of epiphysis from the metaphysis, or weakened pathological bone	Barlow 1883; Lewis et al. 2006	Yes
Rib beading (scorbutic rosary)	Could be confused with rachitic rosary	Owen 1899; Lewis et al. 2006	Yes
Swollen organs	Spleen and liver	Owen 1899	No
Urine is alkaline and may contain blood	-	Owen 1899	No

TABLE 4.3. Clinical features typical of rickets present in cases of reported co-occurrence. Dash (-) = No comment.

Features of Rickets	Conditions	Source	Observe in Paleopathology
Wide open fontanelles	-	Cheadle 1878; Bromer 1946	Possible if large and remains open beyond normal fusion schedule
Craniotabes	-	Bromer 1946	Yes
Frontal bossing	-	Lewis et al. 2006	Yes
Swelling (enlarged) limb bones and their metaphyses (joints)	Typically wrist and ankle, enlarged tibia.	Cheadle 1878; Owen 1899; Lewis et al. 2006	Yes
Beading of the ribs	Occasional	Cheadle 1878; Barlow 1883; Owen 1899; Bromer 1946; McIntosh 1945, cited by Bromer & Harvey 1948; Lewis et al. 2006	Yes
Decayed and broken teeth (poorly formed?)	-	Cheadle 1878	Yes

4.4.2. Radiographic Presentation

Few studies have been produced on the co-occurrence of scurvy and rickets using radiography (e.g., Bromer & Harvey 1948; Fouron & Chichoine 1962). As with the clinical presentation, the case history (order and amount of development) significantly impacts the radiographic presentation of co-occurrence. In cases of co-occurrence, classic features associated with either severe rickets (e.g., bending) or severe scurvy (e.g., line of Fraenkel, scurvy line, corner signs, elevated periosteum) are often absent and 'masked' by the processes of the other disease (Follis et al. 1940; Evans 1945; Fouron and Chichoine 1962). Many non-diagnostic radiographic features are shared between the diseases which makes diagnosis challenging if one of the diseases is not well developed (Bromer & Harvey 1948). See Table 4.4 for a list of radiographic features observed in cases of co-occurrence.

In some cases, one of the two diseases will dominate over the other and mask the features of the non-dominant disease. Bromer and Harvey (1948), and Valentini et al. (2011) discuss cases of dominant scurvy and found that radiographs did not provide any evidence of concomitant rickets unless rickets was well developed. Wimberger (1923, cited by Bromer & Harvey 1948) also came to a similar conclusion, finding evidence of both diseases only in cases where scurvy developed on healing cases of severe or moderate rickets. This is logical as deposition of osteoid from bone growth and/or remodelling must occur for the development of rachitic features (Shore 2008). Fouron and Chichoine (1962) present four cases where rickets was the dominant disease and one of liberated rickets. Many cases of scurvy were missed when first observed by clinicians but biochemical tests revealed the presence of low vitamin C. From a radiographic

perspective, the researchers found the features of rickets to predominate while those of scurvy were obscured with exception of a dense line of Frankel and one example of a Wimberger's ring. However, advanced features of neither disease were present, for example bending of rickets and periosteum elevation of scurvy (Fouron & Chichoine 1962). The identification of scurvy may be challenging in cases of dominant and severe rickets as the features of scurvy require proper mineralization to appear on radiographs (Follis et al. 1940; Bromer & Harvey 1948).

In summary, radiographs appear to be most helpful in identifying co-occurrence in cases where rickets is dominant and scurvy can be suggested as some scorbutic features were still occasionally observed. Bromer and Harvey (1948) propose that radiographic methods can produce a high percentage of accuracy in identifying co-occurrence in cases which meet these criteria. Ultimately, the radiographic appearance of disease will depend upon the individual's case history.

TABLE 4.4. Common radiographic features observed in cases of co-occurrence. Dash (-) = No comment.

Features of Co-occurrence	Conditions & Prevalence	Disease of Origin	Source	Observe in Paleopathology
Rarefaction and thinning of the cortex or diaphysis and epiphysis	Scurvy: ground-glass[1]; Rickets: moth-eaten appearance, observed rarefaction in metaphysis 3/5 and in the epiphysis 5/5 times in cases of dominant scurvy	Both	Evans 1945; Bromer 1946; Bromer & Harvey 1948; Fouron & Chichoine 1962; Lewis et al. 2006	Yes
Coarsening of trabeculae	-	Rickets	Evans 1945; Fouron & Chichoine 1962	Yes
Physis line at metaphysis is thin, not dense	In cases of dominant rickets	Rickets	Bromer & Harvey 1948; Follis et al. 1940	Yes
Physis line at metaphysis is thick	4/5 cases of dominant scurvy	Scurvy	Fouron & Chichoine 1962	Yes
Spreading or cupping at diaphyseal ends (preparatory calcification zone)	Ends appear frayed, hazy and poorly defined	Both (cupping), Rickets (fraying)	Bromer 1946; Bromer & Harvey 1948	Yes
Extended space between epiphysis and diaphysis (zone of preparatory calcification)	-	Rickets	Bromer & Harvey 1948	No

TABLE 4.4.
(Continued)

Features of Co-occurrence	Conditions & Prevalence	Disease of Origin	Source	Observe in Paleopathology
Lateral spurs	Extend outward at right angle to end of shaft, observed in 5/5 cases of dominant scurvy	Both	Bromer & Harvey 1948; Fouron & Chichoine 1962	Yes
Rib rosary	-	Both	Bromer & Harvey 1948; Lewis et al. 2006	Yes but should evaluate macroscopically
Periosteal reaction	Due to healing rickets or healing scurvy, as well as many other conditions. Slight in width; Laminated and origin at mid-shaft if rachitic; sharp, uniform and origin at metaphysis if scorbutic (see both types in same individual)	Both	Evans 1945; Bromer & Harvey 1948; Lewis et al. 2006	Yes but should evaluate macroscopically
Wimberger's ring	Only observed in 1/5 cases of dominant scurvy (Fouron & Chichoine) but in all cases by Bromer & Harvey 1948	Scurvy	Bromer & Harvey 1948; Fouron & Chichoine 1962	Yes
Frayed, cupped and flared metaphyses	-	Rickets	Evans 1945; Lewis et al. 2006	Yes
Metaphyseal displacement	-	Scurvy	Evans 1945	No
Pelkan's spurs[2]	Less defined, further from epiphyseal line	Scurvy	Evans 1945; Bromer 1946	Yes

[1] Ground-glass appearance of bone is absent in cases of co-occurrence (Evans 1945)
[2] Pelkan's spurs (submetaphyseal notch) can be masked by rickets (Evans 1945)

4.4.3. Microscopic Presentation

Few studies in the medical literature have studied co-occurrence of scurvy and rickets from a microscopic perspective. However, the logic governing the appearance of cases follows from macroscopic and radiographic changes. Follis et al. (1945) is one of these studies. Microscopic evidence for co-occurrence was observed and proved useful for diagnosis but many cases are believed to have been missed due to 'masking' of rickets by severe scurvy. The study autopsied 487 children, aged three to 19 months of age for evidence of scurvy and rickets. Histology supported a diagnosis of scurvy in 57 (11.7%) autopsies and of these cases, histological signs of rickets were observed in 26 individuals (45.6%). However, of 39 individuals, the study could only confirm with certainty 5 cases

of scurvy and rickets co-occurrence using multiple techniques (e.g., autopsies, radiography, histology, biochemical, patient history) (Follis et al. 1940). Further evidence supporting the hypothesis of scurvy masking rickets is the presence of rickets in 238 of 487 individuals autopsied while scurvy was only observed in 57 individuals. Clearly, rickets was a very common disease in their sample.

Rickets can be observed satisfactorily using microscopy at an early stage in disease formation while scurvy is only observable if it is extreme and long lasting (Follis et al. 1940). Dominant rickets disrupts the bone mineralization process and inhibits, partially or completely, the formation of a scorbutic lattice. Bromer and Harvey (1948) note that some of their cases demonstrated a dominance of features associated with rickets (e.g., thick osteoid bands and frayed zones of preparatory calcification). In such cases, only features of rickets should be observed. However, Follis et al. (1940) found that histological signs of scurvy could still be observed in presence of severe rickets. It is unclear what signs were observed and if they would be preserved in the archaeological record. When scurvy dominates and occurs first, the formation of rachitic features will be inhibited as osteoblastic activity is inhibited (Follis et al. 1940). However, Follis et al. (1940) found that osteoid was still being extensively produced in cases of co-occurrence, suggesting that osteoblast activity is not completely inhibited. Also, Follis et al. (1940) suggest confusion can arise when identifying features as both rickets and scurvy cause the formation of fibrous tissue between trabeculae, but this feature is unlikely to preserve archaeologically. Many microscopic features, as mentioned in Chapter 2 and 3 are soft tissue changes which will not be preserved in an archaeological setting. Based on Follis et al. (1940)'s findings, rachitic features should be observed in paleopathological cases of dominant rickets and may be observed in paleopathological cases of dominant scurvy as long as osteoblast activity is not completely inhibited. If scurvy was dominant, scorbutic features may be observed if the metaphyseal end is studied.

In summary, on a microscopic level, co-occurrence of scurvy and rickets inhibits the appearance of the less developed and more recent condition, effectively masking it (Follis et al. 1940). In cases reported by Follis et al. (1940), scurvy was the dominant disease and rickets was being masked while Bromer and Harvey (1948) identified cases of dominant rickets. Histology is a useful technique to support clinical and radiographic findings but alone it may not be useful to determine co-occurrence in paleopathology as features of either disease may be unobservable. Table 4.5 summarises the histological findings of scurvy dominance while Table 4.6 summarises the histological findings of rickets dominance.

TABLE 4.5. Described histological features observed at the metaphyseal end in cases of co-occurrence where scurvy is dominant.

Features of Co-occurrence	Conditions	Disease of Origin	Source	Observe in Paleopathology
Brittle lattice framework with fractures	Scorbutic appearance to lattice	Scurvy	Follis et al. 1940	Yes, could see the fractures
Fractures in the framework	Caused by hemorrhaging	Scurvy	Follis et al. 1940	Yes
Extravasation of fibrin and blood	Caused by hemorrhaging	Scurvy	Follis et al. 1940	No
Proliferation serum, and osteoblasts in spaces between trabeculae	Caused by fractures in framework, most characteristic sign of scurvy	Scurvy	Follis et al. 1940	No
Formation of connective tissues in empty spaces between trabeculae	Caused by proliferation of osteoblasts	Scurvy and Rickets	Follis et al. 1940	No
Abnormally thick osteoid bands along trabeculae	Can still be well developed in co-occurrence cases	Rickets	Follis et al. 1940; Bromer 1946	No, unless the seams become buried

TABLE 4.6. Histological features observed at the metaphyseal end in cases of co-occurrence where rickets is dominant. Dash (-) = No comment.

Features of Co-occurrence	Conditions	Disease of Origin	Source	Observe in Paleopathology
Little to no formation of brittle scorbutic lattice	Failure of mineralisation	Scurvy	Follis et al. 1940	Yes
Formation of fibrous tissues in empty spaces between trabeculae	Caused by proliferation of osteoblasts	Rickets	Follis et al. 1940	No
A wider zone of proliferation	-	Rickets	Bromer & Harvey 1948	Uncertain

4.5. Co-Occurrence of Scurvy and Rickets in Paleopathology

Cases of scurvy and rickets co-occurrence have been mentioned and sometimes discussed in a number of recent articles and posters (Lewis 2010; Devriendt et al. 2010; Brickley 2012; Redfern 2012; Geber & Murphy 2012; Klaus 2014). Table 4.7 outlines these findings. Brickley (2012) suggests cases of co-occurrence were likely present amongst the juveniles from the 19th century St. Martin's collection of Birmingham, but that initial investigations did not consider the issue of scurvy and rickets co-occurrence.

TABLE 4.7. Cases of co-occurrence reported to date in the paleopathological literature.

Number of Cases recorded	Age Category	Sample	Means of Diagnosis	Source
7/248 (2.82%)*	0-6.5y	Poundbury Camp, Romano-British, 3rd-5th century AD.	Macroscopy. Scurvy features (e.g., new bone in orbits) in skull and rickets in post-cranium (e.g., bowing, flared sternal ends, extensive porosity in long bones)	Lewis 2010
11/47 (23.40%)*	0-4y	Saint Amé, 950-1797 AD. France	Macroscopy. Scurvy features in skull and rachitic in post-cranium. Radiography produced but not discussed.	Devriendt et al. 2010
1/51 (1.96%)	0-3y	80 Romano-British and Late Iron Age juveniles, 1st century BC to 4th century AD.	Macroscopy to identify presence of disease. No details on criteria for co-occurrence identification.	Redfern et al. 2012
14/964 (1.45%)	Juvenile	Mass burial, 1845-1852 AD, great famine of Ireland	Macroscopy to identify presence of disease. No details on criteria for co-occurrence identification.	Geber & Murphy 2012
1/5 scurvy cases	16-21 months	900-1750 AD, Lambayeque, Peru, juveniles	Macroscopy. Scurvy: porosity and periosteal reaction. Rickets: bending, cupping and porosity	Klaus 2014

* Potentially more cases but too little preserved to diagnose with certainty.

The expected macroscopic, radiographic or histological features in cases of scurvy and rickets co-occurrence have not been systematically evaluated in an archaeological context. However, Lewis (2010), Devriendt et al. (2010), and Brickley (2012) provide some insight into their observed cases of co-occurrence, as listed below.

First, preservation (completeness and taphonomic change) has a significant impact on the ability to diagnose co-occurrence. Classic paleopathological features of scurvy are largely located in the cranium with some features on post-cranial elements. Classic rickets features have the reverse order; most features develop in the post-cranium with some in the cranium. Therefore if there is poor preservation of either portion of the body, a diagnosis of either disease can be very challenging (Lewis 2010; Devriendt et al. 2010).

Second, important scurvy features necessitate proper functioning of the mineralization process to develop, including ossified subperiosteal reactions, ossified haematomas, or the dense white line of Fraenkel on radiographs (Lewis 2010). A co-occurrence with rickets may inhibit the development of these features. Extensive porosity should still be observed as this feature is associated with increased vascularisation (Brickley 2012).

Third, features of extreme rickets, such as bending, are unobservable (Devriendt et al. 2010). The finding is consistent with the medical literature which found that severe

features of either disease were typically absent and features of scurvy dominate in expression. However, subtle features of rickets including rachitic porosity at the ends of the bones, growth plate porosity, thickening, and flaring of the metaphyses are present (Devriendt et al. 2010).

Fourth, the exact sequence of disease impacts the appearance of the features of uncomplicated scurvy and rickets (Brickley 2012). In most cases reported by clinicians, rickets occurs first followed by scurvy (Cheadle 1878; Barlow 1883; Owen 1899). As discussed previously, rickets typically inhibits scorbutic features from developing and vice-versa. Scurvy is a mortal disease while rickets is not. Individuals with long standing scurvy are suggested to unlikely to have enough time to develop extensive features of rickets (Brickley 2012). Table 4.8 outlines the expected features of co-occurrence cases in paleopathology. Further information on radiography and histology can be reviewed in sections 4.2.2. and 4.2.3.

TABLE 4.8. Expected features of co-occurrence in cases of paleopathology for macroscopic, radiographic, and microscopic techniques

Dominant Disease	Macroscopy	Radiography	Microscopy/Histology
Scurvy	<u>Scurvy</u> Classic scorbutic features, new bone development only with ingestion of trace amounts of vitamin C [1]*	<u>Scurvy</u> Classic scorbutic feature.	<u>Scurvy</u> Some features suggestive of scurvy may develop, ingestion of trace amounts of vitamin C [1]* allows new bone development but histology alone cannot be used to diagnose scurvy [2]
	<u>Rickets</u> Hidden but subtle features (e.g., porosity) could develop if growth resumes	<u>Rickets</u> Hidden	<u>Rickets</u> Unobservable but could develop if growth resumes*
Rickets	<u>Scurvy</u> Scorbutic features associated with porosity only (vascularity), new bone development inhibited*	<u>Scurvy</u> Classic scorbutic features inhibited	<u>Scurvy</u> Hidden
	<u>Rickets</u> Classic rachitic signs (subtle to extreme)	<u>Rickets</u> Classic rachitic signs (subtle to extreme)	<u>Rickets</u> Classic rachitic signs (subtle to extreme)*

[1] Mays (2014) states 2-5% of daily amount required in guinea pig studies and in humans some bone deposition observed in acute and chronic scurvy.
[1] de Boer et al. (2013)
* Brickley (2012).

The current study will attempt to identify cases of co-occurrence in a paleopathological context. The ability to suggest an individual was affected by co-occurrence will require the presence of features of both diseases. Thus, some cases of co-occurrence will likely be missed. Nonetheless, the majority of identifiable cases of scurvy and rickets co-occurrence will likely be of scurvy supervening over rickets with rickets having had some time to develop prior to the onset of scurvy. The scenario will allow features of macroscopic scurvy to appear and possible subtle rickets. The majority of scurvy features should be present as vascular changes are not affected by rickets and only trace amounts are required for some new bone to develop. Mays (2014) states 2-5% of daily amount required in guinea pig studies and in humans some bone deposition was observed in acute and chronic scurvy. Similarly, most features of rickets should be present but moderate to slight bending, flaring, fraying of the growth plate is expected rather than clear changes. Radiographically, rickets should be more prominent and microscopy will only be used to verify rachitic status as diaphyseal bone is sampled and scorbutic changes in archaeological bone are suggestive but not strongly diagnostic (Brickley & Ives 2008). Tables 4.2 to 4.6 can be consulted to know whether or not a particular feature should be observable in an archaeological context.

4.6. Conclusion

Cases of co-occurrence can be identified in paleopathology but consideration for subtle variation and expectation of significant variability in appearance between identified cases is critical. As clearly reported, the appearance of bone in individuals affected by co-occurrence is significantly altered by the order of appearance of disease and the amount each of these develops prior to a secondary condition occurring. Identification of co-occurrence cases should be pursued using multiple techniques to increase the likelihood of observing features consistent with multiple conditions. However, due to the high variability between cases, paleopathological investigations will fail to identify all cases.

Chapter 5: Materials and Methods

5.1. Introduction

The project's central question asks if it is possible to identify potential cases of scurvy and rickets co-occurrence in paleopathology, and how may the co-occurrence of these diseases affect the features currently associated with rickets and scurvy in paleopathological literature. To achieve this goal, a skeletal collection was analysed using multiple techniques, including macroscopy, radiography, and microscopy.

5.2. Materials

The Saint-Amé skeletal collection from Douai, France (Fig. 5.1) was determined as appropriate to answer the research questions based on the criteria of containing previously identified possible cases of scurvy and rickets (Devriendt et al. 2010). The cases were diagnosed using paleopathological features, macroscopic and radiographic, observed on the archaeological remains (Devriendt et al. 2010). The current study re-examines the juveniles under 5 years of age at death (n=48) from the 16th to 18th century portion of the Saint-Amé collection. The particular age group was selected because medical reports found that scurvy and rickets co-occurrence was most common with infants and young children, see Section 4.3., Chapter 4. Permission was granted by the Communauté d'Agglomération du Douaisis, Direction de l'Archéologie Préventive (CAD-DAP) to examine the remains.

Figure 5.1. The location of Douai on a map of France.

5.2.1. Saint-Amé

Saint-Amé is the name of a collegiate church founded in Douai during the Carolingian period, c. 950 A.D., by Arnoul the 1st, count of Flanders (Direction 2006). Throughout its use, the collegiate was a place of worship and burial. Not long after its initial construction a fire broke out damaging part of the building, c. 1000 A.D. The church was then reconstructed in Romanesque style. Another series of major renovation occurred between 1190 A.D. and the 16th century to style the building gothic and greatly expanding its size (Direction 2006). The latest rendition is illustrated in Figures 5.2 and 5.3. The church continued to be utilised throughout the early modern period, c. 1500 A.D. to 1798 A.D. In 1798, the municipality of Douai ordered its demolition, along with the collegiate church of Saint-Jacques (Direction 2006). The land upon which the collegiate once stood became the Place Saint-Amé, an important market square for the city of Douai to this day.

Figure 5.2. Original 1709 model of the city of Douai. The church of Saint-Amé is circled in black. Image from http://www.ville-douai.fr/index.php/Douai%20par%20ses%20plans?idpage=13986&idmetacontenu=3752, Colour brightness and contrast were adjusted, consulted January 2014.

Figure 5.3. Close up of the Collegiate of Saint-Amé from the original 1709 model. Image by Serge Ottaviani, October 9 2013, used under CC BY-SA / Colour brightness and contrast were adjusted. http://fr.wikipedia.org/wiki/Fichier:Coll%C3%A9giale_Saint-Am%C3%A9_-_douai.JPG, consulted February 2014.

5.2.2. The Skeletal Collection

An archaeological project to excavate the collegiate church of Saint-Amé was initiated in 1984 when restoration work on the plaza uncovered parts of the collegiate church's wall and a few burials (Direction 2006). Planning and financing issues delayed excavation until June 2004 and was completed in November 2005 (Direction 2006). Excavation was conducted by the Communauté d'Agglomération du Douaisis (CAD-DAP). As part of the project, a large number of human remains were excavated from burials on the grounds of the now demolished church. The Saint-Amé skeletal collection consists of roughly 1,000 individuals, ranging from young infants to older adults, dating between c. 950 A.D. and 1789 A.D.

The collection is affected by a recovery bias disproportionately favouring the burials of later centuries due to intensification in the number of burials performed on the land in and around the collegiate church over time. Burial intensification resulted in significant damage to earlier burials including burial re-cutting, damage to the skeletons, and loss or commingling of skeletons. The numerous construction events, including the successive church expansions, the recovery of the church's foundation stones to pave the streets of Douai in 1847, and the construction of a defensive trench through the plaza in 1939, also disturbed various burials (Direction 2006). Nonetheless, the skeletons recovered for the early modern period were best preserved making this portion of the collection ideal for further study.

5.2.3. Previous Scurvy and Rickets Research on the Infants and Children of the Collection

In 2010, W. Devriendt, B. Bertrand, and S. Vatteoni, anthropologists with the CAD-DAP, published results of initial paleopathological observations on scurvy and rickets made on 47 juveniles from the site. The juveniles consisted of nearly all the very young individuals for the early modern period, ranging from perinatal to 4 years of age at death, with the exception of a very partial skeleton. Results found that each individual had at least one bone with abnormal porosity. Proliferative porous lesions and hypertrophic new bone were observed on many cranial bones and many sternal ribs ends were enlarged (Devriendt et al. 2010). Scurvy was suspected but a diagnosis was only given if a combination of skeletal indicators were present at numerous locations throughout the body, including the cranium (sphenoid, mandible, maxilla and temporal bone), post-cranium (scapula, pelvis, ribs) and long bones. Based on the criteria, 25 individuals were diagnosed with scurvy, or 53% of the sample. Features associated with rickets (e.g., enlarged metaphyses and porous growth plates) were also noted in 11 individuals with probable scurvy (Devriendt et al. 2010). Extensive details on the features of rickets observed were not discussed as the focus of the poster was on scurvy.

5.3. Methods

5.3.1. Age at Death Estimation Analysis

Dental development scoring (Gustafson & Koch 1974) and diaphyseal limb bone length formulas (Fazekas & Kósa 1978; Maresh 1970) were used to estimate age at death. Additional details on the techniques are available in the following sections. The applicability of these techniques is dependent upon the completeness of the remains. The skeletons from Saint-Amé (n=48) were affected by completeness and recovery biases, summarised in Appendix II. However, both aging methods could still be applied to over half the individuals studied, as summarised in Table 5.1., allowing more reliable age estimates to be obtained. Only one individual could not be assessed using either technique.

TABLE 5.1. The number of individuals assessed using the dental development and/or diaphyseal limb bone length age techniques in the Saint-Amé sample.

Both Techniques	Dental Development Only	Limb Bone Length Only	Neither	Total
28	5	14	1	48

All the individuals were assessed using each of the techniques, regardless of size or estimated age, and were only exempt if the material, i.e., long bones or teeth, were absent or broken. Diaphyseal long bone length aging techniques have unique formulas whether an individual is pre- or postnatal. When an individual scored greater than 40 weeks in utero using Fazekas and Kósa (1978)'s prenatal estimates, the prenatal result was discounted if at least two other techniques indicated the individual was over three months of age. When an individual scored under 0.125 years from Maresh's (1970) postnatal estimates, the postnatal result was rejected if at least two other techniques indicated the individual was less than three months of age.

The use of multiple formulas produced multiple age at death estimates for each individual, see Appendix I for complete results. The results for each method were then compared to produce an age at death estimate based on overlap between the results. When conflicts results arose, priority was given to dental age estimate results as dental development is less variable than osseous development and is also largely regulated by genetics while bone development has greater susceptibility to external factors such as nutritional status and disease (Garn et al. 1959; Lewis & Garn 1960). After, each individual was classified into a likely age range category, including fetal/birth, 0-6 months, 6 months to 1 year, 1-2 years, 2-3y, 3-4y, 4-5y, and unknown. Discrete age categories are used in the literature to classify individuals (e.g., Ortner et al. 1999) but there is no standard to length of time covered per category. In the current research, categories were developed to reflect meaningful age categories to time periods which scurvy and rickets develop (see Chapter 2 and 3 respectively) and historically relevant to

important milestones such as weaning (see Chapter 7). For example, individuals were classified into the fetal/birth category if they were less than three months of age. The cut-off was selected as there may be some variability of a few weeks around 40 weeks in utero a child may be born. Additionally, in a typical situation, a child should not be able to develop scurvy and rickets under 3 months of age. If the remains show signs of deficiency, the deficiency can be linked to the mother and prenatal environment as long as age estimation is accurate. Point estimates are available for each technique in Appendix I. Aging results can be consulted in Chapter 6, Section 6.1.

5.3.1.1. Dental Aging

Dental development was scored using Gustafson and Koch (1974). The scoring system is considered rigorous as it was developed on data from multiples modern clinical and dental development studies including, radiographic and histological data (Gustafson & Koch 1974). The technique scores specific dental developmental stages, including tooth mineralization, eruption, crown, and root completion (Gustafson & Koch 1974).

Thirty three individuals from the skeletal collection had teeth which could be analysed. Teeth were scored macroscopically but additional radiographs (40ms-90kV) were taken of all available mandible and maxillae to score un-erupted dentition using a Faxitron digital radiograph system (Series 43855) (See Appendix I, Section A for details). Unfortunately, sometimes the 1st molar and occasionally other teeth could not be scored radiographically as they were obscured by bone (e.g., opposite half of the fused maxilla in older children) on the radiograph. Similarly, permanent dentition in very early calcification stages was not always visible on the radiographs or identifiable macroscopically. In both cases, the poorly visible teeth were discounted from use in estimating age to avoid under or over estimating an individual's age. See Section 5.3.1. for additional details on the analysis of age from obtained results.

5.3.1.2. Diaphyseal Long Bone Length Aging

The maximum diaphyseal length for each long bone element (e.g., humerus, radius, ulna, femur, tibia, and fibula) was previously measured by the CAD-DAP. The data was made available for the current study, permitting greater focus on recording features of scurvy and rickets. A few random bones were measured by the current researcher and agreed with the previous measurements by the CAD-DAP. Forty two individuals had preserved long bones which could be measured. Fourteen of these individuals relied exclusively on long bone length method for aging as no teeth were preserved. See section 5.3.1. for further details on age analysis. All diaphyseal length measurements, rounded to the closest half millimetre, were applied to both techniques employed.

Two long bone length techniques were employed to estimate the age at death of the remains from Saint-Amé. The first technique was developed by Fazekas and Kósa (1978) and only considers individuals of prenatal age, with scores given as weeks in utero. The technique is considered standard practice to assess age at death for foetal samples (Buikstra & Ubelaker 1994). The second technique was developed by Maresh (1970), using tables located in Scheuer and Black (2000), and only considers individual of postnatal age, with scores given in years. The technique is well known and very commonly employed (Scheuer & Black 2000:10). Separate datasets correlating limb bone length and age are provided for each sex. As sex is unknown in the current archaeological sample, each individual was scored using both tables. The results were then combined to determine a minimum age range based on overlap. Complete results of the techniques can be found in Appendix I, Section B. See Section 5.3.1. for additional details on the analysis of age from obtained results.

5.3.2. Inventory: Preservation Assessment

Preservation, including completeness and taphonomic changes, can limit a researcher's ability to score features, distort radiographs, and assess disease in archaeological remains. To address the issue, completeness and taphonomic changes were scored for each individual, see Appendix II for complete results.

Completeness was assessed by producing an inventory for each skeleton following a similar inventory procedures outlined by Buikstra and Ubelaker (1994:6-8). Changes were made to accommodate current needs, including rapidity in recording data as it was not central to the thesis, and increase sensitivity by separating the 25-75% completeness score into two scores. Completeness was assessed as follows. If present, a score in percentages was given to represent the amount observed. The scores were 0-25%, 25-50%, 50-75%, and 75-100%. Sometimes, completeness scores were given in relation to a whole bone, for example the entire occipital would be scored. Bones scored in this manner include the skull bones, bones of the pectoral and pelvic girdles. The limbs bones were divided into seven areas with each area receiving a unique completeness score. The areas included the proximal epiphysis, proximal metaphysis, proximal 3rd of the shaft, middle 3rd, distal 3rd of the shaft, distal metaphysis, and distal epiphysis. Other bones, including the ribs, vertebrae, sternum and bones of the hand and feed were instead counted to estimate completeness as percent scores were impractical to use. Due to their fragmentary nature, rib counts were made based on the number of proximal ends for each side. The number of sternal rib ends was also counted as they are an important site for macroscopic pathology. Vertebrae presence was assessed based on the number of complete bodies and arches, with separate counts for each section. Bones of the hands, feet, and sternum are not areas of the skeleton which exhibit marked changes in cases of rickets and scurvy. Therefore only a quick count of carpals, metacarpals, tarsals, metatarsals, and sternebrae (unfused pieces of the sternum) was produced. Perinatal and

early childhood skeletal recording forms from Schaefer, Black and Scheuer (2009) were also coloured in when the bone was present as a visual reminder of skeleton's completeness.

Taphonomy, specifically erosion and abrasion from roots, fungi, and exposure to the elements, was assessed following the scoring system devised in McKinley (2004: Figure 6) and published in the British Guidelines to the Standards for Recording Human Remains. McKinley's system is based on Behrensmeyer's (1978, cited by Buikstra & Ubelaker 1994) system devised to study weathering on exposed bone. McKinley (2004) adapted Behrensmeyer's work to reflect taphonomic changes occurring in buried contexts, specifically those commonly encountered at archaeological sites in England. Therefore, McKinley's (2004) system was selected.

Results of the preservation assessment were used to understand the preservation of co-occurrence cases. Overall completeness of the skeleton is summarised as good, moderate or poor. A score of 'good' indicates two thirds or more of the skeleton was recovered. When half the skeleton was recovered it is considered 'moderate' or 'poor' if less than half was recovered. Taphonomic scores are summarised by providing the range and mean of the scores across the whole skeleton. The numbers reflect the grade scores devised by McKinley (2004). The system begins with grade 0 indicating no modification; grades 1 to 3 includes some modification but the bone's profile is retained, and grade 4 indicates the whole bone is affected. Grades 5 and 5+ include heavy erosion and destruction of the surface and profile of the bone (MacKinley 2004). Summary results are available in Chapter 6, Section 6.2., and all original preservation scores are available in Appendix II.

5.3.3. Paleopathology: Methods and Diagnosis

A range of techniques were used to identify cases of scurvy, rickets and co-occurrence in the sample from Saint-Amé. Co-occurrence of disease can disturb the typical appearance of signs associated with either condition. Therefore, multiple techniques were used to provide a greater understanding, perspective and a more accurate diagnosis of disease. The selected techniques include macroscopy, radiography, and microscopy. Macroscopy is standard in paleopathology and features associated with rickets and scurvy are well reported in the paleopathological literature, while radiography is standard for diagnosis of both conditions in the clinical literature with support from biochemical tests. Microscopy was included as it could assist in identifying additional cases of rickets and the technique used to diagnose rickets and osteomalacia. Macroscopic and radiographic assessments were completed by the author at the CAD-DAP while microscopic assessments, specifically scanning electron microscopy (SEM) was carried out at Canadian Center for Electron Microscopy (CCEM), part of McMaster University.

Great care was taken when diagnosing an individual as scorbutic or rachitic. The presence of multiple features was required to suggest a likely diagnosis. To facilitate and standardize the process, every feature observed and scored during the project was assigned a rank (probable, possible, or non-diagnostic) on the basis of their diagnostic utility in paleopathology. If a feature was highly unique to the disease, almost always occurred when the disease was present, and present when disease was severe in expression, the feature was assigned the rank of 'probable' as it is indicative of a probable case of disease. If the feature was common, occurred when the disease was mild and severe in expression, but could also occur in the presence of other conditions, the feature was assigned the rank of 'possible' as it is indicative of a possible case of disease. A feature was given the 'non-diagnostic' rank if it was common and its cause was non-specific in paleopathology. The assigned rank for the features recorded can be found in Tables 5.2 to 5.5 and 5.8.

Subsequently, the scorbutic and rachitic status of each individual was evaluated based on the number of features present and their diagnostic categories. Separate evaluations were produced for each technique and disease. The evaluation resulted in an overall status score given as 'probable', 'possible' or 'unlikely' that the disease had affected the individual during life. If two or more probable features were present, or over three possible features, the individual was considered as a probable case of scurvy or rickets. When no probable features were present, but three or less possible features and some non-diagnostic features were observed, the individual was considered as a possible case of scurvy or rickets. If only non-diagnostic features were observed, the individual was considered as unlikely to have been affected by scurvy or rickets. Some of the individuals recorded were rather incomplete. Therefore, when too few sites could be scored, a diagnosis of 'too little information' was assigned. Nonetheless, each technique was still applied and features were scored according to the methods set out above. In case of doubt, the individual was placed in the lesser category to avoid over representation of disease. Greater weight was given to the macroscopic findings compared to radiographic and SEM results when reaching a final conclusion on the disease status of an individual because of experience level working with particular techniques and significant diagenetic change observed on the majority of SEM samples. The system was developed as features of rickets were found to be very subtle in expression in this collection and preservation of the collection was poor overall, see Appendix II. The methodology is therefore not directly transferable to other sites where disease expression may be different. Results can be found in Chapter 6, Tables 6.2 and 6.5.

5.3.3.1. Macroscopic Assessment and Diagnosis

Gross morphological changes associated with scurvy and rickets were assessed on each skeleton. The suite of features selected for analysis was based on previously published paleopathological literature, with consideration of medical literature (see Chapter 2 and

Chapter 3), and are compiled in Table 5.2 and 5.3 respectively. The features associated with the diseases were scored as present, absent, or unobservable, see Section 5.3.3. for details. The only exception was the use of a scoring system to assess rachitic changes on the distal growth plates of limb bones. The growth plates were scored on a scale of 1 to 4 as described by Ortner and Mays (1998). Mays et al. (2006) added an additional level to the scale between scores 1 (normal) and 2 (slight). The new level represents an earlier or slighter expression of abnormal growth plate, described as 'velvety', than initially reported by Ortner and Mays (1998). Individuals were scored as 2 when they exhibited a velvety surface in this study.

Porosity is a major feature of scurvy and was defined as abnormal when it occurred in localised areas previously reported in the literature, the pores penetrated the cortex of the bone, and the pores were greater in size than 1mm in diameter as defined by Ortner et al. (1997; 1999). In contrast, normal porosity is infrequent and irregular in size (Ortner et al. 2001). Occasionally, superficial hypertrophic bone occurred with the porosity (Ortner et al. 1999; Ortner et al. 2001). Patches of porous new bone were considered abnormal if they occurred in areas previously reported in paleopathological and clinical literature. Additional details on the scored features can be found in Chapter 2, Section 2.5. for scurvy and Chapter 3, Section 3.4. for rickets.

TABLE 5.2. Macroscopic features assessed for scurvy with assigned diagnostic category of each feature and conditions for diagnosis. [1] Porosity, [2] Bone hyperostosis, - = no comment.

Feature observed	Diagnostic Category	Condition(s)	Source
Sphenoid; Greater Wing[1, 2]	Probable	-	Ortner & Ericksen 1997; Ortner et al. 1999; Brickley & Ives 2006; Mays 2008.
Maxilla; Posterior[1]	Probable	-	Ortner & Ericksen 1997; Ortner et al. 1999; Ortner et al. 2001; Ortner 2003; Mays 2008.
Scapula; Supraspinous[1]	Probable	-	Barlow 1883; Ortner et al. 2001; Brickley & Ives 2006.
Scapula, Infraspinous[1]	Probable	-	Barlow 1883; Ortner et al. 2001; Brickley & Ives 2006.
Mandible; Medial coronoid[1]	Possible	-	Ortner et al. 1999; Ortner et al. 2001; Ortner 2003; Brickley & Ives 2006; Mays 2008.
Sphenoid; Lesser wing[1]	Possible	-	Mays 2008.
Sphenoid; Foramen rotundum[1]	Possible	-	Geber & Murphy 2012.
Orbit; Internal zygomatic[1]	Possible	-	Ortner et al. 1999; Ortner et al. 2001; Ortner 2003.
Maxilla; Infraorbital foramen[1,2]	Possible	-	Ortner et al. 1999; Ortner et al. 2001; Ortner 2003; Brickley & Ives 2006; Mays 2008.

TABLE 5.2. (Continued)

Feature observed	Diagnostic Category	Condition(s)	Source
Rib; Flaring	Possible	-	Ortner 2003; Brickley & Ives 2008.
Rib; Fracture(s) on the metaphyseal growth plate	Possible	-	Ortner 2003; Brickley & Ives 2008.
Rib; Porosity	Non-Diagnostic	-	Lovász et al. 2013.
Ilium[1]	Non-diagnostic	-	Mays 2008; Ortner 2003; Brickley & Ives 2008; Devriendt et al. 2010.
Maxilla; Palate[1]	Non-diagnostic	-	Ortner & Ericksen 1997; Ortner et al. 1999; Ortner et al. 2001; Ortner 2003; Brickley & Ives 2006.
Maxilla; Alveolar process[1]	Non-diagnostic	Only pathological when it extends significantly from the teeth sockets.	Ortner et al. 1999; Ortner 2003; Brickley & Ives 2006.
Mandible; Alveolar process[1]	Non-diagnostic	Only pathological when it extends significantly from the teeth sockets.	Ortner et al. 1999; Ortner 2003.
Cranial vault[1]	Non-diagnostic	-	Ortner & Ericksen 1997; Ortner et al. 1999; Ortner et al. 2001; Brickley & Ives 2006; Mays 2008.
Cranial vault[2]	Non-diagnostic	Consider if hyperostosis is located on the parietals and/or frontal.	Ortner & Ericksen 1997; Ortner et al. 1999; Ortner et al. 2001.
Endocranium[1,2]	Non-diagnostic	-	Ortner & Ericksen 1997; Ortner 2003; Brickley & Ives 2006; Mays 2008.
Orbital roof[1, 2]	Non-diagnostic	Consider as a possible feature if clear.	Ortner & Ericksen 1997; Ortner et al. 1999; Ortner et al. 2001; Brickley & Ives 2006; Mays 2008.
New bone formation; Arms	Non-diagnostic	-	Ortner 2003; Brickley & Ives 2008.
New bone formation; Legs	Non-diagnostic	-	Mays 2008; Ortner 20003; Brickley & Ives 2008.
Metaphyseal Porosity; Arms	Non-diagnostic	-	Ortner et al. 2001; Ortner 2003.
Metaphyseal Porosity; Legs	Non-diagnostic	-	Ortner et al. 2001; Ortner 2003.

TABLE 5.3. Macroscopic features assessed for rickets with assigned diagnostic category of each feature and conditions for diagnosis. - = no comment.

Feature observed	Diagnostic Category	Condition(s)	Source
Deformed arm and leg long bones	Probable	Only if it affects multiple bones, the distribution is consistent with rickets, clear bend, and radiography can rule out trauma.	Ortner & Mays 1998; Mays et al. 2006.
Abnormal growth plate	Probable	Study the distal growth plate only; Only a probable feature if it scored above a velvety appearance.	Ortner & Mays 1998; Mays et al. 2006.
Flattening of the femoral metaphysis & Coxa vara	Probable	Only probable if both features are present together. If alone, consider as a possible.	Mays et al. 2006.
Thickened long bones	Probable	If clear, supported by x-rays and affecting multiple bones bilaterally, consider as probable.	Ortner & Mays 1998; Mays et al. 2006.
Sternal rib end; Flaring	Probable	Only if cupped.	Ortner & Mays 1998; Mays et al. 2006.
Sternal rib end; porosity	Possible	-	Ortner & Mays 1998; Mays et al. 2006.
Rib deformity (sharp angle)	Possible	Only if it affects multiple bones, the distribution is consistent with rickets, clear bend, and radiography can rule out trauma.	Ortner & Mays 1998; Mays et al. 2006.
Deformed mandibular ramus	Possible	-	Ortner & Mays 1998; Mays et al. 2006.
Ilium concavity	Possible	-	Ortner & Mays 1998; Mays et al. 2006.
Long bone metaphyseal flaring; Arm and legs	Possible	Consider possible if feature is clear.	Mays et al. 2006.
Long bone concave curvature porosity	Possible	Consider as probable if curvature in bone is present along with porosity.	Mays et al. 2006.
Cranial vault porosity	Non-diagnostic	Ectocranial & endocranial	Ortner & Mays 1998; Mays et al. 2006.
Frontal bone bossing	Non-diagnostic	-	Brickley & Ives 2008
Orbital roof porosity	Non-diagnostic	-	Ortner & Mays 1998; Mays et al. 2006.
Irregular & porous metaphyseal cortex of long bones	Non-diagnostic	-	Ortner & Mays 1998; Mays et al. 2006.

5.3.3.2. Radiographic Assessment and Diagnosis

Radiographs were taken using the facilities housed at the CAD-DAP in Douai, France. Radiographic images of all the long bones (humerii, radii, ulnae, femora, tibiae, and fibulae) and their epiphyses, ribs (particularly those with sternal ends) and ilium were used to assess features associated with scurvy and rickets. The mandibles and maxillae were also radiographed to examine un-erupted teeth for better age estimation. Devriendt and colleges (2010) previously assessed the Saint-Amé collection using this technique on a Faxitron (Series 43855) digital radiograph system at 110kV. To reduce redundancies, the same images were used for the current project to score radiographic features of scurvy and rickets. However, not all bones of interest for the current project were radiographed. Therefore, an additional series of radiographs were taken using the Faxitron (Series 43855) digital radiograph system at 40ms-90kV.

Radiographic features of scurvy and rickets were assessed based on previous medical (e.g., Shore 2008) and paleopathological (Mays et al. 2006; Brickley & Ives 2008) literature. A summary of features assessed for scurvy and rickets can be found in Table 5.4. and 5.5. respectively. Similar to the macroscopic assessment, all recorded radiographic features for both diseases were scored as present, absent, or unobservable, see Section 5.3.3. for details. Osteopenia was scored when overall bone loss was observed, with particular focus on poor trabecular bone content and a moth eaten appearance. Cortical thinning was scored with a focus on the appearance of a thin cortex and cortical tunneling. When scoring all radiographic features, preservation and taphonomy were taken into account by comparing what was observed in the radiographic images to gross morphology photographs and notes. For example, osteopenia would not be scored as present if the bone was hollowed or broken in the area where osteopenia was suspected. Additional details on features scored can also be found in Chapter 2, Section 2.6. for scurvy and Chapter 3, Section 3.5. for rickets.

TABLE 5.4. Radiographic features assessed for scurvy with assigned diagnostic category of each feature and conditions for diagnosis. - = no comment.

Feature Observed	Diagnostic Category	Condition	Source*
Pelkan Spur	Probable	-	Shore 2008; Stark 2014.
Wimberger's ring	Probable	-	Brickley & Ives 2008; Stark 2014.
White line of Fraenkel	Probable	Only consider probable when occurring with a scurvy line. If alone, consider it non-diagnostic.	Grewar 1965; Brickley & Ives 2008; Stark 2014.
Scurvy line	Probable	Only consider probable when occurring with a White line of Fraenkel. If alone, consider it non-diagnostic.	Brickley & Ives 2008; Stark 2014.
Corner sign	Possible	-	Grewar 1965; Brickley & Ives 2008; Stark 2014.
Metaphyseal fracture(s)	Possible	-	Shore 2008.
Generalised osteopenia	Non-diagnostic	Evaluated in the vertebrae (if radiographed), ribs, pelvis, and long bones.	Brickley & Ives 2008; Shore 2008.
Long bones; Cortical thinning	Non-diagnostic	-	Brickley & Ives 2008; Shore 2008; Stark 2014.

* Additional medical sources were used, refer to Chapter 2, Section 2.6.

TABLE 5.5. Radiographic features assessed for rickets with assigned diagnostic category of each feature and conditions for diagnosis. [1]Active, [2] Healing, - = no comment.

Feature Observed	Diagnostic Category	Condition	Source*
Trabecular coarsening[1]	Possible	When preservation is good.	Brickley & Ives 2008.
Rib; Loss of integrity at the sternal end	Possible	When preservation is good.	Brickley & Ives 2008.
Fraying of the growth plates[1]	Possible	When preservation is good.	Brickley & Ives 2008.
Thick cortex with periosteal apposition[2]	Possible	-	Brickley & Ives 2008.
Cortical thinning[1]	Possible	When preservation is good.	Brickley & Ives 2008.
Trabecular coarsening in distal metaphysis[2]	Possible	When preservation is good.	Brickley & Ives 2008.
Osteopenia in growth plate & metaphyses[1]	Non-diagnostic	-	Brickley & Ives 2008.
Osteopenia in epiphyses[1]	Non-diagnostic	-	Brickley & Ives 2008.

* Additional medical sources were used, refer to Chapter 3, Section 3.5.

5.3.3.3. Microscopic Assessment

Microscopic techniques were pursued to provide additional evidence to assist in the identification of potential rickets cases. Two techniques were chosen, scanning electron microscopy (SEM) and histological stains (Villanueva and Goldner's trichrome). The techniques are destructive. Permission was obtained from the CAD-DAP to collect rib samples from 27 individuals.

Individuals were selected for sampling based on a quick, in-field diagnosis focused on gross macroscopic and radiographic features of rickets. The rachitic features were listed in columns and check marks were given if they were scored as present in the individual. Once tallied, the individuals were classified into one of the following categories: suspected probable rickets, suspected possible rickets, or unlikely cases of rickets. If an individual had clearly bent limbs, a feature commonly associated with rickets, or multiple possible features associated with rickets, the individual was classified into the suspected probable category. When multiple features associated with rickets, but non-specific, were observed the individual was classified as a suspected possible case. Individuals displaying few non-specific features, or no features associated with rickets, were classified into the unlikely category. From the classification, a total of 27 individuals were sampled with individuals from each category as summarised in Table 5.6.

TABLE 5.6. Results of rickets in-field diagnosis of individuals sampled for microscopic analysis. Numbers correspond to the individual number as assigned by the CAD-DAP. - = not sampled.

Diagnostic Level	Rib	Femur
Suspected probable case of rickets	221, 270, 307	91, 190, S678
Suspected possible case of rickets	6, 7, 95, 110, 125, 153, 208, 218, 232, 473, 514, 528, 634, 835	-
Unlikely case of rickets	37, 124, 272, 273, 647, 219, 414	-

Microscopy is destructive and concerns about the collection's completeness were considered during sampling selection. Ribs were therefore preferentially sampled. Compelling arguments determined the selection of this bone for sampling. First, previous clinical, experimental, and paleopathological research has used this bone for histological and SEM analyses (e.g., Oppenheimer & Snodgrass 1980; Reid & Boyde 1987; Brickley et al. 2007). Second, ribs are fast growing bones with elevated turnover rates due to high trabecular bone content (Epker & Frost 1965). As such, ribs should rapidly develop signs of rickets if the disease was present or healing. Lastly, ribs are readily available and are not used in most skeletal assessments. Sampling this bone is overall less destructive to the precious archaeological material and ribs are frequently already broken or damaged. Rib position within the chest (or number) was not taking into consideration when sampling. Individuals with ribs affected by a lot of taphonomic change were removed from the sampling pool as the bone's condition was felt to unlikely produce adequate microscopic

specimens, though amount of internal fungal or bacterial destruction may not correlate with the external appearance of bone (Bell 1990). Exceptions were made in cases of suspected probable rickets, replacing the rib with a femoral shaft fragment.

Sampling was performed using a small electric hand saw. Rib and femoral samples, roughly 1.5cm in length, were taken from the shaft area, preferentially located as close as possible to the sternal end of the ribs or distal metaphysis of the femora. See Table 5.7 for a summary of samples taken and location. If the sternal end was present, a sample was taken at least 1.5cm away from the growth plate (physis). In cases where the sternal end was not present, the closest shaft fragment to the sternal end available was sampled. Femoral fragments were preferentially cut from the distal most part of the shaft available but as the three femora in question were broken, sampling was forced to extend into the mid-shaft area. Selection of bone locations for sampling was consistent between the ribs and femora as both targeted previously formed sections of bone rather than the growth plate. Previous histological research in medical and animal experimental literature has focused on the growth plate area of the rib or the metaphyses of long bones for signs of scurvy, rickets, and co-occurrence (e.g., Sherman & Pappenheimer 1921; Maxwell & Turnbull 1932; Ranström & Sydow 1949). In this study the sternal ends were not sampled as they are important locations for diagnosing (and re-analysis) of these diseases using macroscopic techniques and few sternal ends were preserved in the collection. Additionally, sampling was preferentially conducted on already damaged rib samples, i.e. incomplete or broken ribs, to reduce the damage done to the collection.

TABLE 5.7. Summary table of the samples taken for microscopic analysis. Some ribs could be sided, R = right, L = left.

Individual	Bone	Location
6	Rib	Shaft
7	Rib (L)[1]	Shaft
37	Rib	Shaft
91	Femur (R)	Distal Shaft
95	Rib (R)	Metaphysis end
110	Rib	Shaft
124	Rib	Shaft
125	Rib	Shaft
153	Rib	Shaft
190	Femur (R)	Mid shaft
208	Rib (L)	Shaft
218	Rib (R)	Metaphysis end
219	Rib (L)	Shaft
221	Rib	Shaft
232	Rib (R)	Shaft
270	Rib (L)	Shaft
272	Rib	Shaft
273	Rib	Shaft
307	Rib (R)	Metaphysis end
414	Rib	Shaft
473	Rib (L)	Shaft
514	Rib (L)	Shaft
528	Rib (R)	Metaphysis end
634	Rib (R)	Metaphysis end
647	Rib	Shaft
678	Femur (R)	Distal shaft
835	Rib (R)	Shaft (close to metaphysis)

[1]When known, the side of the bone is indicated

Ribs, especially archaeological ones, are friable and susceptible to pressure. Samples were therefore embedded into resin blocks using a methyl methacrylate (MM) resin with styrene (resin stabilizer) and an accelerator (reactive agent). The protocol is as follows. The bones were dried for 72-hours using ethyl alcohol (EtOH) in a vacuum desiccator. Alcohol was changed every 24-hours. Methyl methacrylate was then introduced to the samples in a 1:1 solution EtOH and MM for 24-hours in the vacuum desiccator. This was replaced with a MM only solution for another 24 hours with the same set up. The resin mixture was then prepared. First, the MM was 'washed' using a separating funnel to remove any stabiliser. It is accomplished by rinsing the MM with two washes of sodium hydroxide (5% concentration) mixture and then three washes with distilled water. Additional washes with distilled water could be performed if the pH of the MM is basic (higher than seven), as measured after the last distilled water wash. The MM was then drained through calcium chloride to purify the MM from any residual water. The final solution of 95% MM, 5% styrene, and 0.2% accelerator were mixed together, then distributed to each samples. The samples were then capped and placed in a water bath at

37°C. The resin embedded samples were kept in the water bath, capped, until the resin was hard. The process results in clear blocks of embedded bone which can be cut to create blocks for SEM and histological thin sections.

5.3.3.3.1. Scanning Electron Microscopy Assessment

Scanning electron microscopy was completed at the Canadian Center for Electron Microscopy (CCEM). Blocks 3mm in height were cut from each embedded bone sample using a diamond blade Buehler Isomet 1000 Precision Saw. Both a 5" diamond blade with 2.95" flanges and 4" diamond blade with 2.5" flanges were used depending on the size of cut. To be eligible for SEM use, a parallel and polished surface is required. The SEM blocks were ground down in a figure "8" motion to remove any evidence of saw markings or imperfections using 600 grit pads (Buehler Carbimet Paper Discs). Once ground down, the surface of the block was polished on a textmet pad (Buehler), again in a figure "8" motion, using a combination of diamond polish (Buehler Metadi diamond polishing compound for metallography, 3 microns, No 40-6142) and Buehler Metadi Fluid (extender for diamond abrasives, No. 40-6032). The blocks were sonicated between each step, including the last one, to clean the SEM surface. The end result was a parallel plane and clear surface which could be studied under the SEM.

As neither bone nor the resin is a conductor, the blocks were carbon coated and mounted onto stubs at the CCEM. The bones were studied using a JEOL scanning electron microscope (JEOL JSM-6610LV tungsten filament equipped SEM) on backscatter imaging at 15kV, with a working distance of 15-17mm. Some settings were variable depending on the amount of charging. PA varied between 30-50 while spot size varied between 40-45. Magnification was fluid as the goal was to isolate particular features. Photographs of the whole section of bone were made for reference with additional close ups of interesting features.

There are no pathognomic features of rickets which can be observed using SEM. However a number of suggestive and non-specific features may be present. The features recorded for this study are summarized in Table 5.8. An explanation of each feature can be found in Chapter 3, Section 3.6. The features recorded in this study reflect the slight expression of rickets observed in the individuals from Saint-Amé. Additional indicative features of severe rickets may be observed using SEM but these were not observed or recorded in the sample. Similar to the macroscopic and radiographic assessments, all recorded SEM features for rickets were scored as present, absent, or unobservable.

TABLE 5.8. SEM features assessed for rickets with assigned diagnostic category of each feature and conditions for diagnosis. - = no comment.

Feature Observed	Diagnostic Category	Condition	Source
Buried osteoid remnants	Probable	Only when buried, includes buried osteoid seams	Frost 1962.
Separation of new bone from old bone	Probable	-	Bonucci et al. 1969; Brickley & Ives 2008.
Poorly mineralised osteons	Possible	-	Engfeldt et al. 1956; Jaffe 1972; Pitt 2002; Brickley & Ives 2008.
Poorly mineralised osteocytes (bearded)	Possible	-	Steendijk et al. 1965; Steendijk & Boyde 1973.
Howship's lacunae	Non-diagnostic	Typically diagnostic when using histomorphometry but the technique is unavailable.	Raubenheimer 1997; Marie et al. 1982.

Care was taken when diagnosing an individual with rickets. Each feature was awarded a label (probable, possible, or non-diagnostic) based on its diagnostic value for rickets in a paleopathological context; see Section 5.3.3. for details. The categories assigned for each SEM feature can be found in Table 5.8. Subsequently all sampled individuals underwent an evaluation to assess their rachitic status based on the features observed. The individuals were classified as probable, possible, or unlikely cases of rickets following the method outlined in Section 5.3.3. Typically, the presence of multiple features was required to suggest the presence of disease. However, thick osteoid seams are considered as highly suggestive of rickets and osteomalacia (Mankin 1974a) and buried osteoid seams are an unusual phenomenon linked to previous disruption in the bone forming process (Frost 1962; Teitelbaum et al. 1976; Priemel et al. 2010). When buried osteoid, including seams, was observed the cases were classified as probable rickets. Howship's lacunae is key feature in the medical literature but is considered non-diagnostic in the current project as the feature can be produced by causes other than rickets, notably normal growth. When a significant amount of Howship's lacunae was observed, the condition was considered abnormal but it is difficult to be certain from visual inspection alone. Results can be found in Chapter 6, Table 6.7. Quantitative techniques, such as histomorphometrics, may provide additional certainty but could not be pursued in the current study.

5.3.3.3.2. Histological Stains Assessment

Histological stains are used in medicine and biology to highlight or differentiate structures in a tissue thin section. Villanueva and Goldner's trichrome stains were selected as they are used with bone and highlight osteoid, the structure of interest to diagnose rickets. Thin sections were cut serially from the resin embedded bone samples at

a thickness of 0.7mm using the same 5" and 4" diamond blade Buehler saw as for the SEM blocks. Once cut, the sections were stained.

Pre-prepared Villanueva stain solution from Polysciences, Inc. (Villanueva Osteochrome Bone Stain, Cat #16280) was used. The solution was poured into a glass vial wrapped in aluminium foil to block all light. Up to six sections were placed in each vial. The vials were then sealed with a sheet of Parafilm and foil. The sections were left to soak in the stain for forty-eight hours. They were then washed serially three times in EtOH, about thirty seconds each time, and left to dry.

Goldner's trichrome is composed of four stains (Weigert's hematoxylin; fuchsin ponceau; molybdic orange G; and light green) and two cleaning solutions prepared as follows. Weigert's hematoxylin was created by mixing a 1:1 solution of pre-prepared Weigert's solution A with Weigert's solution B from Polysciences, Inc (Weiger's Hematoxylin solution A, cat#25373A, & B, cat#25373B). The solution was then left to settle before being filtered to remove any excess particles. Only half a recipe of fuchsin ponceau was required, so ingredient amounts were adjusted in their original ratios. Powder form acid fuchsin (0.13g) [Sigma-Aldrich Life Science, Product #F8129-25G] and powder form ponceau (0.52g) [Ponceau Xylidine, by Sigma-Aldrich Life Science, Product #P2395-25G] were mixed with 260ml of distilled water. Once the powders were dissolved, acetic acid (0.52g) was added to the solution. Molybdic orange G was prepared by mixing orange G powder (5g) [Sigma-Aldrich Life Science, Product #O3756-25G] with 10% solution of phosphomolybdic acid, and distilled water (500ml). Light green was produced by mixing distilled water (250ml) with light green powder (0.25g) [Harleco, Light Green SF-Yellowish, cat# 254-12]. The cleaning solutions were prepared as follows. The first is a 1% aqueous acetic acid solution and the second is a 50% EtOH solution.

Goldner's trichrome thin sections were serially stained, free floating, in a specific order. First the sections were placed into a filtered Weigert's hematoxylin solution for twelve minutes, then fuchsin ponceau for twenty minutes. The sections were soaked in molybdic orange G solution for six minutes. Only the top portion of molybdic orange G solution was used to avoid precipitate. Finally, the sections were placed in light green for eighteen minutes. Between each solution, the samples were washed three times, for one minute each, in 1% acetic acid solution followed by three washes, one minute each, in distilled water. The final wash, performed after the samples soaked in light green, was slightly different. First, the samples were rinsed in 50% EtOH, then rinsed three times in 100% EtOH solution, for one minute each rinse, and then left to dry.

Once the sections were stained, they were ground down, in a figure 8 pattern, on either side, using a 320 grit abrasive disc (Buehler Carbimet Paper Discs) to remove any overt saw marks. The sections were then mounted onto a glass slide using a UV glue and ground down to an appropriate thickness, 0.10mm or less for Villanueva and 0.05mm or

less for Goldner's trichrome. Grinding was performed on a Buehler Minimet (1000 Grinder-Polisher), starting with a 400 grit abrasive disc (Buehler Carbimet Paper Discs), moving up to a 600 grit abrasive disc (Buehler Carbimet Paper Discs) for finer adjustments. Before starting the grinding process, the sections were measured on all sides using digital calipers (Mitutoyo, Absolute Digimatic, Model No NTD12-6"C, serial number 001559, Code No. 573-221-10^{CE}) to determine the appropriate course of action. Measurements were taken after every run until the appropriate thickness was reached. The speed, set at 50, and force, set at 0, were kept constant.

The Villanueva thin sections appeared to stain well following the protocol as outlined above. However, once the thin sections were mounted on slides and the grinding process began, the stain was slowly removed as the bone thinned. The stain clearly did not fully penetrate the bone. Attempts were made to test the possibility of re-staining mounted bone thin sections by developing a methodology on mounted and ground (to 0.19mm or less) anatomy bone sections. Five methods were tried, see Table 5.9 for details. Results can be found in Chapter 6, Section 6.3.3.2.

TABLE 5.9. Villanueva thin section re-stain tests.

Test	Notes
1.	A few drops of stain were placed directly onto the thin section which was then wrapped in Glad® ClingWrap to seal the stain on the section. Left for 24 hours.
2.	A few drops of stain were placed directly onto the thin section which was then wrapped in Parafilm to seal the stain on the section. Left for 24 hours.
3.	A cotton ball wet with stain was placed directly onto the thin section. Left for 24 hours.
4.	A few drops of stain were dripped onto a section then placed in a sealed plastic container. To keep the section from drying out, 0.4-0.5mm of double distilled water was added to the bottom of the container. Suction cups were placed under the slide to hold it up above the water. Left for 24 hours.
5.	A section was placed in a glass container, sealed with parafilm, and covered with aluminium foil. Left sample to bathe in the stain for 24 hours. (See Figure 6.1B, Chapter 6, Section 6.2.c.2.).
6.	A section covered with a few drop of stain was placed in a sealed plastic container covered with aluminium foil. Left for 90 minutes. (See Figure 6.1A, Chapter 6, Section 6.2.c.2.).

Once stained, the sections were studied using bright field observation under a digital microscope (Keyence digital microscope, multiscan) using a VH-Z20R (RZx20-x200) lens, for evidence of osteoid. Reference photos (.tiff) were taken at varying magnifications.

The goal of using stained thin sections was to potentially observe preserved osteoid. Villanueva stains osteoid a transparent green to jade green or red colour, while Goldner's stains it red. Figure 5.4 is an example of the expected appearance of osteoid using the Goldner's stain protocol and as tested on an adult anatomy rib sample. The presence of a significant amount (large width) of osteoid around the edges of trabeculae is the major

indicator of a case of potential rickets or osteomalacia (Mankin 1974a; Pitt 2002). Both stains also colour a few other structures, e.g., Goldner's stains mineralised bone green, marrow cells red, cell nuclei black, and cartilage orange.

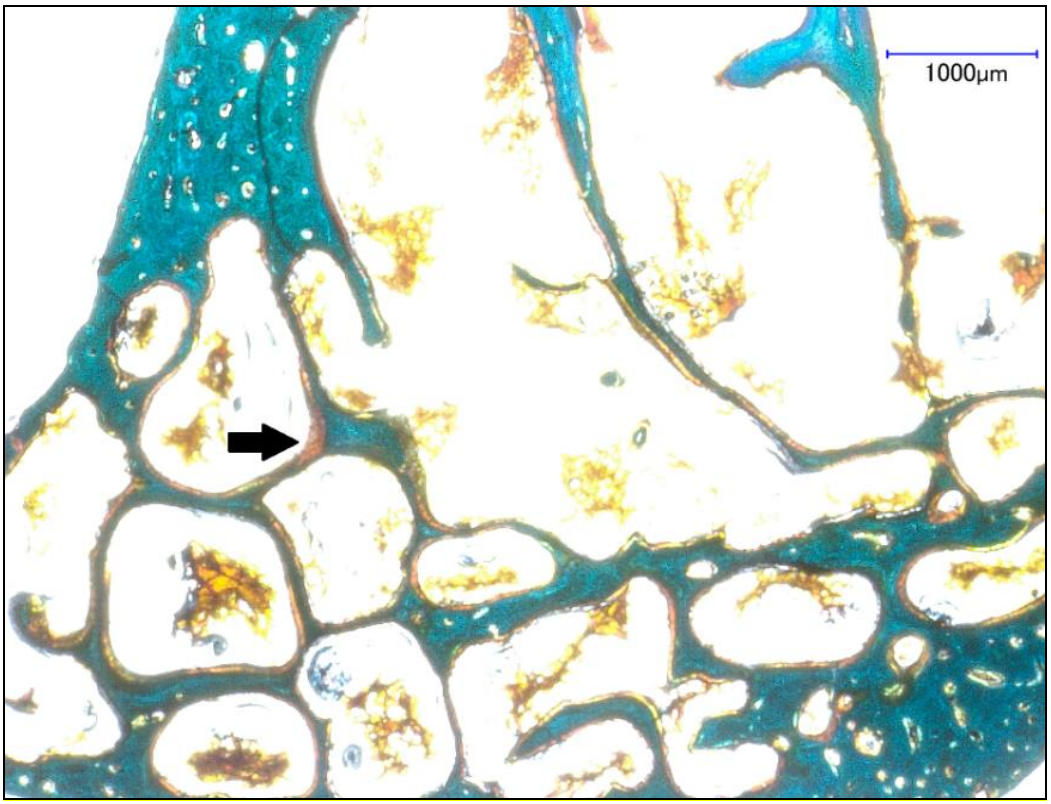


Figure 5.4. Adult anatomy control rib (A2B8) stained using Goldner's Trichrome, X50 VH-Z20R (RZx20-x200) lens, sharpened image, backlight on high. Solid arrow points to red unmineralised osteoid.

Chapter 6: Results

6.1 Results of Age at Death Estimation

The age at death estimation results are summarised in Table 6.1. Individuals are classified into a yearly age category, see Chapter 5, Section 5.3.1. for details. Complete point estimate results of each age at death estimation technique are available in Appendix I.

TABLE 6.1. The assigned age at death category of the juveniles from Saint-Amé. mo = month; y = year, - = no individual

Individual	Age category
6	Fetal/Birth
7	Fetal/Birth
74	Fetal/Birth
95	Fetal/Birth
175	Fetal/Birth
191	Fetal/Birth
208	Fetal/Birth
218	Fetal/Birth
219	Fetal/Birth
220	Fetal/Birth
221	Fetal/Birth
272	Fetal/Birth
273	Fetal/Birth
414	Fetal/Birth
528	Fetal/Birth
647	Fetal/Birth
699	Fetal/Birth
37	0-6 months
66	0-6 months
91	0-6 months
100	0-6 months
124	0-6 months
125	0-6 months
153	0-6 months
231	0-6 months
264	0-6 months
514	0-6 months
110	6 months-1 year
232	6 months-1 year
270	6 months-1 year
367	6 months-1 year
657	6 months-1 year
678	6 months-1 year
784	6 months-1 year
56	1-2 years
267 [265]	1-2 years
307	1-2 years

TABLE 6.1. (Continued)

Individual	Age category
332	1-2 years
385	1-2 years
190	2-3 years
296	2-3 years
111	3-4 years
450	3-4 years
473	3-4 years
634	3-4 years
18	4-5 years
835	4-5 years
361	Unknown (infant/child)

The sample is composed of a large number of fetuses/neonates (n=17, 35%) and infants (n= 24, 50%). Infants are defined as a juvenile between birth and 3 years of age (Buikstra & Ubekaler 1994:9). Well over half (n=34, 70.83%) the collection were under 1 year of age at death while five individuals (10.42%) fall between 1 and 2 years of age at death. After 1 year, the number of individuals per age category decreases except for the 3 to 4 year age category. Few children (n=6, 12.5%) were analysed. Children are defined as juveniles between 3 to 12 years of age (Buikstra & Ubekaler 1994:9). One individual was classified as unknown as all their teeth were missing and no limb bones were preserved.

6.2 Preservation Results

Preservation, including the completeness and amount of taphonomic change undergone, impacts the researcher's ability to suggest the presence of disease. A comprehensive inventory, scoring completeness and taphonomic change, specifically erosion and abrasion, was recorded for each individual assessed as part of the project. Scores were given following the method outlined in Chapter 5, Section 5.3.2. Complete results are provided in Appendix II. A summary of the results for individuals considered to be affected by scurvy and rickets co-occurrence can be found below in Table 6.2.

The completeness of suggested cases of co-occurrence varies from 'good' to 'poor' but the majority (n=9, 75%) are scored as 'moderate' or better. Three individuals could be classified as 'poor', five as 'moderate', and four as 'good'. The erosion and abrasion of bones ranged from 0 to a maximum of 5 but the mean score is under 3. Therefore, the juveniles overall displayed a range from good to moderate taphonomic change. Individual 264 is the only individuals with a score above 3 for a bone. Of its bones, only has two bones, the basilaris and vertebrae, have a score over 3. Neither of these bones were critical sites for scoring pathology in the current research. Each type of bone also exhibited a range of taphonomic scores (see Table II-19 in Appendix II). The upper limbs, an important site for scorbutic and rachitic signs in infants under 1 year, had the greatest number of individuals affected by taphonomy. The humerus had the most 3 scores with

four juveniles affect but the ilium, radius, and ulna each have three juveniles affected. The humerus also had the most 2 scores, with 6 juveniles but the radius, ulna, femur and tibia all had 5 juveniles affected. Completeness, erosion and abrasion do not have a strong relationship. For example, 208 and 647 are given a 'good' for completeness but scored on average 2 for erosion and abrasion, while 221 scored 'poor' in completeness but a 1.36 for average erosion and abrasion.

TABLE 6.2. Summary of skeletal preservation for the suggested cases of co-occurrence.

Individual	Completeness[1]	Taphonomy[2]	Notes
56	Poor	Range: 1 to 3 Mean: 1.8	Most long bones are present but the ends are broken and damaged; only a few rib fragments preserved and the skull is mostly missing.
66	Poor	Range: 1 to 3 Mean: 2.3	Most of the skull and upper axial bones are missing; only limbs of the left side preserved, except the humerus.
95	Good	Range: 0 to 2 Mean: 0.8	Nearly all the bones present and good condition, though skull is fragmentary.
110	Moderate	Range: 0 to 2 Mean: 1.15	The skull was mostly present and lower limbs complete, though little of the upper limbs was preserved (fragmentary).
208	Good	Range: 1 to 3 Mean: 2	Mostly all preserved, except the tibiae and fibula which were fragmentary.
221	Poor	Range: 1 to 4 Mean: 1.36	Only half the skull was preserved; lower limbs were missing, except for the right femur; upper limbs and axial elements were present.
264	Moderate	Range: 1 to 5 Mean: 1.36	Most of the skull and all upper body preserved; missing lower portion of the post cranium.
367	Moderate	Range: 0 to 2 Mean: 0.9	Half the skull preserved and most limbs though they were very fragmentary; few ribs.
514	Moderate	Range: 1 to 3 Mean: 1.87	Near full skull preserved and most ribs. Upper limbs were all present but no lower post-cranium.
634	Moderate	Range: 0 to 3 Mean: 2.5	Near full skull preserved and most ribs. Upper limbs were all present but no lower post-cranium.
647	Good	Range: 1 to 3 Mean: 2	Half the skull was missing and half of the upper limb bones. All the lower limb bones and most ribs are preserved.
657	Good	Range: 0 to 3 Mean: 1.62	Near full skull preserved. Though over half the upper limbs are present, most were damaged; as were the lower limbs, though they were all preserved. Few ribs were recovered.

[1]See method outlined in Chapter 5, Section 5.3.2. for a definition.

[2] See method outlined in Chapter 5, Section 5.3.2. for a definition. A score of 0 is no modification; 1 is affected by a few patches; 2 has moderate amount of change; 3 nearly the whole surface is affected with some erosion; 4 the whole surface is affected with erosion; and 5 displays heavy erosion (McKinley 2004). The mean of these scores is provided to approximate overall skeletal taphonomy.

6.3. Paleopathology Results

6.3.1. Scurvy Diagnosis Results

Following the methodology outlined in Chapter 5, Sections 5.3.3, 5.3.3.1. and 5.3.3.2., two verdicts concerning an individual's probability of being affected by scurvy were reached for data collected from macroscopic and radiographic techniques. Taken together, the two verdicts suggest a comprehensive (final) diagnosis on the scurvy status of the individual. A summary of the diagnosis results for scurvy can be found in Table 6.3. Complete raw data used to determine the diagnosis of scurvy can be found in Appendix III.

Without consideration for co-occurrence, eight individuals (16.67% of individuals recorded) are suggested as probable cases of scurvy. Seventeen other individuals (35.42%) are considered as possible cases. Eighteen individuals (37.5%) are classified as unlikely cases. Five remaining individuals (10.42%) provided too little information to suggest a diagnosis. Two individuals (66 and 208) classified as possible scurvy cases have pathological features not associated with scurvy or rickets. Extra caution was taken when diagnosing these individuals.

The prevalence of macroscopic features associated with scurvy per diagnosis category are compiled in Table 6.4. All recorded macroscopic features used to identify scurvy were observed in at least one individual. In all individuals, the most common observed scurvy feature was new bone formation on the legs (81.82% prevalence). Other features observed in over half of the individuals recorded include porosity at the palate, infraorbital, and posterior regions of the maxilla; porosity on the medial coronoid of the mandible, internal surface of the zygomatic and cranial vault (ectocranium). Additionally, flaring and porosity on the ribs, new bone formation on the arms, and metaphyseal porosity on the legs were also present in over half of all individuals recorded. The least common feature is porosity in the infraspinous region of the scapula (17.86% prevalence).

In probable cases only, nearly all features were observed with exception of porosity on the lesser wing of the sphenoid and fractures on the sternal growth plate of the rib. The features classified as probable were always observed (100% prevalence) except for porosity on the infraspinous area of the scapula which was uncommon (25% prevalence) in probable cases. All possible and non-diagnostic features are present in at least two thirds of probable cases, except for porosity of the ilium and cranial vault bone hypertrophy, endocranial changes, porosity of the orbital roof and cranial vault. The last three features are still very common as they were observed in half of all probable cases.

TABLE 6.3. Diagnosis of scurvy for the Saint-Amé juveniles, including macroscopic and radiographic results. TLI = Too Little Information.

Individual	Macroscopy	Radiography	Final
6	Probable	Probable	Probable
7	Probable	Possible	Possible
18	TLI	TLI	TLI
37	Unlikely	Unlikely	Unlikely
56	Possible	Probable	Possible [1]
66	Possible [2]	Probable [2]	Possible [1,2]
74	TLI	Probable	Possible
91	TLI	TLI	TLI
95	Probable	Possible	Probable [1]
100	TLI	Unlikely	Unlikely
110	Possible	Possible	Possible [1]
111	Unlikely	Unlikely	Unlikely
124	Probable	Probable	Probable
125	Probable	Probable	Probable
153	Possible	Unlikely	Unlikely
175	Unlikely	Unlikely	Unlikely
190	TLI	Unlikely	Unlikely
191	TLI	TLI	TLI
208	Possible [2]	Probable [2]	Possible [1,2]
218	Unlikely	Possible	Unlikely
219	Probable	Unlikely	Possible
220	TLI	Possible	Possible
221	Probable	Unlikely	Probable [1]
231	Unlikely	Unlikely	Unlikely
232	Unlikely	Unlikely	Unlikely
264	Possible	Probable	Probable [1]
267 [265]	TLI	Unlikely	Unlikely
270	Unlikely	Unlikely	Unlikely
272	Unlikely	Probable	Possible
273	Possible	Unlikely	Possible
296	TLI	Unlikely	Unlikely
307	TLI	Unlikely	Unlikely
332	TLI	Unlikely	Unlikely
361	TLI	TLI	TLI
367	Possible	Possible	Possible [1]
385	TLI	Probable	Possible
414	TLI	Probable	Possible
450	Unlikely	Unlikely	Unlikely
473	Unlikely	Possible	Unlikely
514	Probable	Unlikely	Possible [1]
528	Probable	Probable	Probable
634	Unlikely	Probable	Possible [1]
647	Possible	Probable	Probable [1]
657	Possible	Possible	Possible [1]
678	TLI	Unlikely	Unlikely
699	TLI	TLI	TLI
784	Possible	TLI	Possible
835	Unlikely	Unlikely	Unlikely

[1] Individual is also considered as either a probable or possible cases of co-occurrence with rickets.
[2] Individual possesses other pathological features not associated with either scurvy or rickets

TABLE 6.4. Summary results of observed prevalence for macroscopic scorbutic features. [1] Porosity, [2] Bone hyperostosis. [3] Counts do not include individuals affected by co-occurrence (classified as probable or possible). N/A = not available, feature could not be scored as bone was damaged or missing in all individuals.

Feature Observed	Probable Cases[3]	Possible Cases[3]	Unlikely Cases[3]	Too Little Information Cases[3]	Co-occurrence Cases[3]	Total Observed[3]
Sphenoid; Greater Wing [1,2]	3/3 (100%)	2/5 (40%)	2/9 (22.22%)*	N/A	2/7 (28.57%)	9/24 (37.5%)
Maxilla; Posterior[1]	3/3 (100%)	4/5 (80%)	1/4 (25%)	N/A	6/6 (100%)	14/18 (77.78%)
Scapula; Supraspinous[1]	4/4 (100%)	4/6 (66.67%)	0/11 (0%)	N/A	6/11 (54.55%)	14/32 (43.75%)
Scapula; Infraspinous[1]	1/4 (25%)	1/5 (20%)	0/8 (0%)	N/A	3/11 (27.27%)	5/28 (17.86%)
Mandible; Medial coronoid[1]	3/4 (75%)	3/3 (100%)	4/11 (36.36%)	N/A	7/10 (70%)	17/28 (60.71%)
Sphenoid; Lesser wing[1]	0/2 (0%)	1/2 (50%)	1/5 (20%)	N/A	5/7 (71.43%)	7/16 (43.75%)
Sphenoid; Foramen rotundum[1]	2/3 (66.67%)	0/3 (0%)	0/4 (0%)	N/A	4/7 (57.14%)	6/17 (35.29%)
Orbit; Internal zygomatic[1]	4/4 (100%)	2/4 (50%)	2/9 (22.22%)	N/A	5/8 (62.5%)	13/25 (52%)
Maxilla; Infraorbital foramen[1,2]	4/4 (100%)	3/3 (100%)	2/8 (25%)	N/A	4/6 (66.67%)	13/21 (61.91%)
Rib; Flaring	4/4 (100%)	6/7 (85.71%)	7/12 (58.33%)	0/1 (0%)	10/12 (83.33%)	27/36 (75%)
Rib; Fracture(s) on the sternal growth plate	0/2 (0%)	0/1 (0%)	1/4 (25%)	N/A	6/7 (85.71%)	7/14 (50%)
Rib; Porosity	4/4 (100%)	6/7 (85.71%)	2/12 (16.67%)	0/1 (0%)	8/12 (66.67%)	22/36 (61.11%)
Ilium[1]	1/4 (25%)	2/4 (50%)	2/12 (16.67%)	0/1 (0%)	3/8 (37.5%)	8/29 (27.59%)
Maxilla; Palate[1]	4/4 (100%)	0/1 (0%)	3/7 (42.86%)	0/1 (0%)	3/5 (60%)	10/18 (55.56%)
Maxilla; Alveolar process[1]	3/4 (75%)	3/5 (60%)	7/9 (77.78%)	N/A	5/6 (83.33%)	18/24 (75%)
Mandible; Alveolar process[1]	3/4 (75%)	4/5 (80%)	2/11 (18.18%)	N/A	6/11 (54.55%)	15/31 (48.39%)
Cranial vault[1]	2/4 (50%)	4/5 (80%)	7/13 (53.85%)	0/1 (0%)	10/12 (83.33%)	23/35 (65.71%)
Cranial vault[2]	1/4 (25%)	2/5 (40%)	3/13 (23.08%)	0/1 (0%)	7/12 (58.33)	13/35 (37.14%)
Endocranium[1,2]	2/4 (50%)	1/5 (20%)	1/12 (8.33%)	1/2 (50%)	3/12 (25%)	8/35 (22.86%)
Orbital roof[1,2]	2/4 (50%)	1/3 (33.33%)	0/10 (0%)	N/A	6/11 (54.55%)	9/28 (32.14%)
New bone formation; Arms	2/3 (66.67%)	5/7 (71.43%)	9/16 (56.25%)	1/1 (100%)	8/12 (66.66%)	27/39 (69.23%)
New bone formation; Legs	4/4 (100%)	5/6 (83.33%)	8/11 (72.73%)	2/3 (66.67%)	8/9 (88.89%)	27/33 (81.82%)

Table 6.4. (Continued)

Feature Observed	Probable Cases[3]	Possible Cases[3]	Unlikely Cases[3]	Too Little Information Cases[3]	Co-occurrence Cases[3]	Total Observed[3]
Metaphyseal Porosity; Arms	2/3 (66.67%)	4/7 (57.14%)	5/16 (31.25%)	1/1 (100%)	6/12 (50%)	18/39 (46.15)
Metaphyseal Porosity; Legs	3/4 (75%)	5/6 (83.33%)	7/11 (63.64%)	3/3 (100%)	5/9 (55.56%)	23/33 (69.70%)
Total Individuals diagnosed	4	9	18	5	12	48

The results are given as the number of individuals observed with a feature out of the number of individuals where the feature could be observed.
* Individuals 111 and 175 have very slight porosity of the sphenoid and few other features classified as probable or possible features. Therefore, a higher diagnosis category could not be assigned.

TABLE 6.5. Summary results of observed prevalence for radiographic scorbutic features. N/A = not available, feature could not be scored as bone was damaged or missing in all individuals. [1] Counts do not include individuals affected by co-occurrence (classified as probable or possible).

Feature Observed	Probable Cases[1]	Possible Cases[1]	Unlikely Cases[1]	Too Little Information Cases[1]	Co-occurrence Cases	Total Observed
Pelkan Spur	3/4 (75%)	4/9 (44.44%)	6/18 (33.33%)	0/2 (0%)	5/12 (41.67%)	18/45 (40%)
Wimberger's ring	0/1 (0%)	N/A	0/6 (0%)	0/1 (0%)	0/3 (0%)	0/11 (0%)
White line of Fraenkel	4/4 (100%)	7/9 (77.78%)	14/18 (77.78%)	0/2 (0%)	10/12 (83.33%)	35/45 (77.78%)
Scurvy line	4/4 (100%)	6/9 (66.67%)	1/18 (5.56%)	0/2 (0%)	10/12 (83.33%)	21/45 (46.67%)
Corner sign	1/4 (25%)	0/9 (0%)	0/18 (0%)	0/2 (0%)	1/12 (8.33%)	2/45 (4.44%)
Metaphyseal fracture(s)	2/4 (50%)	0/9 (0%)	2/18 (11.11%)	0/2 (0%)	4/12 (33.33%)	8/45 (17.78%)
Generalised osteopenia	4/4 (100%)	5/9 (55.56%)	14/18 (77.78%)	2/2 (100%)	11/12 (91.67%)	36/45 (80%)
Long bones; Cortical thinning	4/4 (100%)	4/9 (44.44%)	14/18 (77.78%)	2/2 (100%)	11/12 (91.67%)	35/45 (77.78%)
Total Individuals diagnosed	4	9	18	5	12	48

The results are given as the number of individuals observed with a feature out of the number of individuals where the feature could be observed.

Two features were recorded in all possible cases of scurvy, including porosity around the infraorbital foramen and medial coronoid of the mandible. Regarding probable features observed in possible cases, porosity on the supraspinous area and on the posterior of the maxilla are the most frequent and present in two thirds of the cases. Porosity on the greater wing of the sphenoid is common at 40% prevalence while porosity at the infraspinous area of the scapula was uncommon, similar to probable cases. Possible features are either common (50% prevalence) or nearly always observed in possible cases of scurvy, with the exception of porosity at the foramen rotundum. Porosity on the lesser wings of the sphenoid was absent in probable cases but was observed in half of the possible cases. Most non-diagnostic features are common, occurring in prevalences ranging from 50% to 86% in possible cases. The only exceptions are the porosity of the endocranium and porosity of the orbital roof which were recorded in only 20% to 33% of possible cases. Unobserved features in possible cases include porosity at the foramen rotundum (sphenoid), rib fractures, and porosity at the maxillary palate.

In unlikely cases, no feature was always observed. In fact, the majority of probable (4/4) and possible (4/6) features were uncommonly observed (25% or less prevalence) in unlikely cases of scurvy. Rib flaring (58% prevalence) was the most common possible or probable feature in unlikely cases, followed by porosity at the medial coronoid process of the mandible (36% prevalence). Two individuals displayed very slight porosity on the greater wing of the sphenoid. Although the feature was classified as probable and Ortner et al. (2001) consider the feature to be "virtually pathognomonic" in paleopathology, few other features were recorded in these individuals. Therefore, although there is good anatomical reasoning for the presence of porosity at this location (e.g. Ortner & Ericksen 1997), the individuals could not be classified into a higher diagnosis category following the methodology outlined in Chapter 5, Section 5.3.3. The most frequent features were non-diagnostic in unlikely cases, including porosity of the alveolar process of the maxilla (77.8% prevalence) and new bone formation of the legs (73% prevalence). Common non-diagnostic features with prevalences around 50% include new bone formation on the arm limb bones, porosity on the cranial vault and on the palate of the maxilla. Remaining non-diagnostic features scored a prevalence of 25% or under with exception of metaphyseal porosity on the arm and leg limb bones. Porosity on the scapula and orbital roof were not observed in any unlikely case.

In cases of co-occurrence, all features were observed at least once. Porosity at the posterior of the maxilla was the only feature recorded in all co-occurrence cases, similar to probable cases. Fractures at the sternal growth plate were very common (86% prevalence) in cases of co-occurrence while this feature was rarely recorded in other cases. Probable features were either commonly observed (e.g., porosity at the supraspinous region with around 50% prevalence) or uncommon with prevalences under 30% (e.g., porosity at the greater wing of the sphenoid) in co-occurrence cases. Possible features were very common in co-occurrence cases with prevalences over 55%. Flaring of

the rib is the most prevalent possible feature, at 83%, in co-occurrence cases. A majority of non-diagnostic features were also recorded with prevalences over 50% in cases of co-occurrence. In particular, porosity of alveolar process of the maxilla and cranial vault, as well as new bone formation on the arms and legs were observed with prevalences over 80%. Overall, the prevalences of features in cases of co-occurrence closely resemble those of probable and possible cases.

The prevalence of observed radiographic features associated with scurvy per diagnosis category are compiled in Table 6.5. In all cases, generalised osteopenia is the most prevalent feature (80%) prevalence. It is quickly followed by cortical thinning and the white line of Fraenkel with prevalences of 78%. The high prevalences are expected as both osteopenia and cortical thinning are non-diagnostic and occur in both scurvy and rickets. High prevalence of a white line of Fraenkel was also expected as a white line at the growth plate is also a normal feature, only thickened in scurvy. In contrast Wimberger's ring was not observed in any radiographs. Overall, both possible features, corner signs and metaphyseal fractures were also poorly observed.

In probable cases, the majority of features scored were always observed. The features include the white line of Fraenkel, scurvy line, and both non-diagnostic features. Pelkan spurs and metaphyseal fractures were also very common with prevalences of 75% and 50% respectively. Only corner signs were uncommon, with 25% prevalence, in probable cases.

The white line of Fraenkel and scurvy lines were the most common features recorded for possible cases with 66% and greater prevalence. Common features include Pelkan spurs, and both non-diagnostic features with prevalences between 44% and 57% in possible cases. Corner signs and fractures, both possible signs, were not observed in possible cases.

In unlikely cases, the white line of Fraenkel and both non-diagnostic features have very high prevalences, around 78%. Other probable features are poorly observed with under 35% prevalence, including Pelkan spurs and scurvy line. The possible features are also rare, with under 15% prevalence, in unlikely cases; similar to possible cases.

Feature prevalences in co-occurrence cases are similar, though slightly reduced, from those of probable cases. The most common features are non-diagnostic with over 90% frequency. The white line of Fraenkel and scurvy line are also nearly always observed with prevalences of over 80% in co-occurrence cases. Other features are much less common. Pelkan spurs and metaphyseal fractures are observed with 42 and 33% prevalence respectively, and corner signs are rare.

6.3.2. Rickets Diagnosis Results

Following the methodology outlined in Chapter 5, Sections 5.3.3., 5.3.3.3.1. and 5.3.3.2., three verdicts concerning an individual's probability of being affected by rickets were given using data collected from macroscopic, radiographic and microscopic techniques. Histological staining methods were excluded from the process, see Section 6.3.3.2. and 6.3.3.3 for details. Further SEM results are provided in Section 6.3.3.1. Taken together, the verdicts suggest a comprehensive (final) diagnosis concerning rickets for the individual, summarised in Table 6.6. Complete raw data used to determine the diagnosis can be found in Appendix IV.

Without consideration for co-occurrence, eight of 48 individuals (16.67%) were found to present as probable cases of rickets, including 175, 190, 264, 267, 307, 473, 634, and 835. Sixteen (33.33% of total) individuals were classified as possible cases. Fourteen other individuals (29.17%) were considered unlikely cases. Of the 48 individuals studied, 10 (20.83%) provided too little information to produce a conclusive diagnosis. Extra caution was taken when diagnosing individuals 66 and 208, see Section 6.3.1 for details.

The prevalence of observed macroscopic features associated with rickets per diagnosis category are compiled in Table 6.7. Overall, the expression of rickets was very subtle. In total, the most common feature was distal abnormal growth plate (score 2), closely followed by sternal rib end flaring, irregular, porous metaphyseal cortex, and cranial vault porosity. High prevalences are unsurprising as many of these features are shared with scurvy. Nearly half of all features recorded (8/17 features) were uncommonly observed with prevalences between 30% and 45% in total. Rib deformities were rarely observed (3% prevalence) while iliac concavity deformation was not recorded in any case.

Leg bone deformities and frontal bossing were present in all probable cases. Other very commonly observed features of probable cases, with over 75% prevalence, include limb bone thickening, leg bone flaring, arm bone deformities, and irregular, porous metaphyseal cortex. All diagnostic features were present in over half of the probable cases with the exception of costochondral rib flaring which was only recorded in a quarter of cases. Only three possible features were recorded in probable cases, two of which were recorded in less than half of the probable cases. Many features were unobserved in probable cases including porosity at the sternal rib ends, rib deformities, deformed mandibular ramii, cranial vault porosity and orbital roof porosity.

TABLE 6.6. Diagnosis of rickets for the Saint-Amé juveniles, including macroscopic, radiographic and scanning electron microscopy (SEM) results. TLI = Too Little Information, CA = Cannot Assess, N/A = Samples not available for study.

Juvenile	Macroscopy	Radiography	SEM	Final	Juvenile	Macroscopy	Radiography	SEM	Final
6	Unlikely	Unlikely	Unlikely	Unlikely	232	Possible	Probable	Unlikely	Possible
7	Unlikely	Unlikely	CA	Unlikely	264	Probable	Probable	N/A	Probable[1]
18	TLI	TLI	N/A	TLI	267 [265]	Probable	Possible	Probable*	Probable
37	Unlikely	TLI	Unlikely	Unlikely	270	Probable	Possible	Unlikely	Possible
56	Possible	Possible	N/A	Possible[1]	272	Unlikely	Unlikely	Unlikely	Unlikely
66	Probable [2]	Possible [2]	N/A	Possible [1,2]	273	Possible	Unlikely	Unlikely	Unlikely
74	TLI	TLI	N/A	TLI	296	Unlikely	Unlikely	N/A	Unlikely
91	TLI	Possible	CA	Possible	307	Probable	Possible	Possible	Probable
95	Possible	Unlikely	CA	Possible[1]	332	Unlikely	Unlikely	N/A	Unlikely
100	TLI	TLI	N/A	TLI	361	TLI	TLI	N/A	TLI
110	Probable	Unlikely	CA	Possible[1]	367	Probable	Unlikely	N/A	Possible[1]
111	TLI	TLI	N/A	TLI	385	TLI	TLI	N/A	TLI
124	Unlikely	Possible	CA	Unlikely	414	Unlikely	Unlikely	Unlikely	Unlikely
125	Possible	Unlikely	Unlikely	Unlikely	450	Unlikely	Unlikely	N/A	Unlikely
153	TLI	TLI	CA	TLI	473	Probable	Possible	Probable	Probable
175	Probable	Possible	N/A	Probable	514	Possible	Unlikely	Unlikely	Possible[1]
190	Probable	Probable	CA	Probable	528	Possible	Unlikely	Unlikely	Unlikely
191	Probable	Unlikely	N/A	Possible	634	Probable	Possible	Probable	Probable[1]
208	Probable [2]	Possible [2]	Unlikely	Possible [1,2]	647	Unlikely	Possible	Probable	Possible[1]
218	TLI	Unlikely	CA	Unlikely	657	Probable	Possible	N/A	Possible[1]
219	TLI	TLI	CA	TLI	678	Probable	Unlikely	Unlikely	Possible
220	Unlikely	Unlikely	N/A	Unlikely	699	TLI	TLI	N/A	TLI
221	Probable	Possible	CA	Possible[1]	784	TLI	TLI	N/A	TLI
231	Possible	Unlikely	N/A	Possible	835	Probable	Possible	Probable	Probable

[1] Individual is also considered as either a probable or possible cases of co-occurrence with scurvy.

[2] Individual possesses other pathological features not associated with either scurvy or rickets

* Personal communication, January 2014 Megan Brickley. Dr. Brickley observed remnant space of buried osteoid on a thin section of individual 267 [265] in France.

TABLE 6.7. Summary results of observed prevalence for macroscopic rachitic features. N/A = not available, feature could not be scored as bone was damaged or missing in all individuals. [1] Counts do not include individuals affected by co-occurrence (classified as probable or possible).

Feature observed	Probable Cases[1]	Possible Cases[1]	Unlikely Cases[1]	Too Little Information	Co-occurrence Cases	Total Observed
Deformed arm long bones	4/5 (80%)	1/5 (20%)	4/12 (33.33%)	0/5 (0%)	2/12 (16.67%)	11/39 (28.21%)
Deformed leg long bones	4/4 (100%)	3/5 (60%)	1/11 (9.09%)	0/5 (0%)	4/9 (44.44%)	12/34 (35.29%)
Abnormal growth plate; Score 2	3/6 (50%)	6/6 (100%)	8/13 (61.54%)	5/6 (83.33%)	11/12 (91.67%)	33/43 (76.75%)
Abnormal growth plate; Score 3 or 4	3/6 (50%)	1/6 (16.67%)	2/13 (15.38%)	1/7 (14.29%)	7/12 (58.33%)	14/44 (31.82%)
Flattening of the femoral metaphysis & Coxa vara	2/4 (50%)	1/3 (33.33%)	1/8 (12.5%)	0/2 (0%)	4/8 (50%)	8/25 (32%)
Thickened long bones	5/6 (83.33%)	6/6 (100%)	7/14 (50%)	1/8 (12.5%)	8/12 (66.67%)	27/46 (58.70%)
Sternal rib end; Flaring	1/4 (25%)	1/3 (33.33%)	11/12 (91.67%)	4/5 (80%)	10/12 (83.33%)	27/36 (75%)
Sternal rib end; Porosity	0/4 (0%)	0/3 (0%)	10/12 (83.33%)	1/5 (20%)	8/12 (66.67%)	19/36 (52.78%)
Rib deformity (sharp angle)	0/4 (0%)	0/3 (0%)	1/11 (9.09%)	0/5 (0%)	0/10 (0%)	1/33 (3.03%)
Deformed mandibular ramus	0/4 (0%)	0/2 (0%)	5/9 (55.56%)	3/4 (75%)	2/10 (20%)	10/29 (34.48%)
Ilium concavity	0/5 (0%)	0/3 (0%)	0/10 (0%)	0/3 (0%)	0/8 (0%)	0/29 (0%)
Long bone metaphyseal flaring; Arm	2/5 (40%)	4/5 (75%)	4/12 (33.33%)	1/5 (20%)	10/12 (83.33%)	21/39 (53.85%)
Long bone metaphyseal flaring; Leg	3/4 (75%)	3/5 (50%)	4/10 (40%)	0/5 (0%)	3/9 (33.33%)	13/33 (39.39%)
Long bone concave curvature porosity	2/6 (33.33%)	2/3 (66.67%)	1/2 (50%)	N/A	2/5 (40%)	7/16 (43.75%)
Cranial vault porosity	0/3 (0%)	3/4 (75%)	6/11 (54.54%)	5/6 (83.33%)	10/12 (83.33%)	24/36 (66.67%)
Frontal bone bossing	1/1(100%)	0/1 (0%)	0/2 (0%)	0/2 (0%)	0/8 (0%)	1/14 (7.143%)
Orbital roof porosity	0/2 (0%)	0/3 (0%)	3/10 (30%)	0/2 (0%)	6/11 (54.55%)	9/28 (32.14%)

Table 6.7. (Continued)

Feature Observed	Probable Cases[3]	Possible Cases[3]	Unlikely Cases[3]	Too Little Information Cases[3]	Co-occurrence Cases[3]	Total Observed[3]
Irregular & porous metaphyseal cortex of long bones	5/6 (83.33%)	5/6 (83.33%)	9/14 (64.29%)	6/8 (75%)	9/12 (75%)	34/46 (73.91%)
Total Individuals	6	6	14	10	12	48

The results are given as the number of individuals observed with a feature out of the number of individuals where the feature could be observed.

TABLE 6.8. Summary results of observed prevalence for radiographic rachitic features. N/A = not available, feature could not be scored as bone was damaged or missing in all individuals. [1] Counts do not include individuals affected by co-occurrence (classified as probable or possible).

Feature observed	Probable Cases[1]	Possible Cases[1]	Unlikely Cases[1]	Too Little Information Cases[1]	Co-occurrence Cases	Total Observed
Trabecular coarsening	5/6 (83.33%)	3/6 (50%)	4/14 (28.57%)	2/7 (28.57%)	7/12 (58.33%)	21/45 (46.67%)
Rib; Loss of integrity at the sternal end	2/3 (66.67%)	2/4 (50%)	4/11 (36.36%)	1/4 (25%)	5/10 (50%)	14/32 (43.75%)
Fraying of the growth plates	1/6 (16.67%)	1/6 (16.67%)	2/14 (14.29%)	0/7 (0%)	0/12 (0%)	4/45 (8.89%)
Thick cortex with periosteal apposition	5/6 (83.33%)	4/6 (66.67%)	5/14 (35.71%)	2/7 (28.57%)	7/12 (58.33%)	23/45 (51.11%)
Cortical thinning	3/6 (50%)	5/6 (83.33%)	4/14 (28.57%)	2/7 (28.57%)	10/12 (83.33%)	24/45 (53.33%)
Trabecular coarsening in distal metaphysis	5/6 (83.33%)	4/6 (66.67%)	3/14 (21.43%)	2/7 (28.57%)	7/12 (58.33%)	21/45 (46.67%)
Osteopenia in growth plates & metaphyses	6/6 (100%)	4/6 (66.67%)	10/14 (71.43%)	5/7 (71.43%)	10/12 (83.33%)	35/45 (77.78%)
Osteopenia in epiphyses	2/3 (66.67%)	N/A	3/3 (100%)	1/2 (50%)	3/4 (75%)	9/12 (75%)
Total Individuals diagnosed	6	6	14	10	12	48

The results are given as the number of individuals observed with a feature out of the number of individuals where the feature could be observed.

Two probable features distal abnormal growth plate (score 2) and thickened long bones, were recorded in all possible cases. These features are closely followed in prevalence by irregular and porous metaphyseal cortex of long bones with a prevalence over 80%. Probable features were either commonly observed (60% prevalence and higher) or uncommon (prevalence of 35% or less). Similarly, few possible features were observed, and those which were have high prevalences. Non-diagnostic features were very common with one exception. Unobserved features include porosity at the sternal rib ends, rib deformities, deformed mandibular ramii, ilium concavity, cranial vault porosity, and frontal bossing. Many of these features were also unobserved in probable cases.

In unlikely cases, no feature was always observed but sternal rib end flaring and porosity have the highest prevalences with 92% and 83% respectively. The majority of probable features were uncommonly observed, with prevalences under 50%, of which three have prevalences under 20%. Abnormal growth plate (score 2) was still common with a prevalence of 62%. Possible features also have mixed results though the prevalences are higher overall. Deformed mandibular ramus, long bone metaphyseal flaring (both arm and leg) have prevalences between 40% and 56%. The only case of rib deformity noted in the study belongs to an individual classified as an unlikely case. Frontal bossing was unobserved in unlikely cases.

Cases of co-occurrence produced a similar overall feature prevalence patterns to possible cases. No feature was always observed in co-occurrence cases but probable and non-diagnostic features are the most frequently observed. Specifically, abnormal growth plate (score 2), sternal rib end flaring, arm bone metaphyseal flaring, cranial vault porosity and irregular, porous metaphyseal cortex were observed with prevalences of 75% and greater in co-occurrence cases. The majority of probable features have prevalences greater than 50%, with exception of arm and leg bone deformities, though the leg bone deformities remain somewhat common. The majority of possible features have prevalences under 50%. All non-diagnostic features are very common with prevalences over 50% except for frontal bossing. Rib deformities and frontal bossing were unobserved in co-occurrence cases.

The prevalence of observed radiographic features associated with rickets per diagnosis category is compiled in Table 6.8. In total, the most common feature is osteopenia in the growth plates and metaphyses (78%), and closely followed by osteopenia in the epiphyses (75%). Both features are considered non-diagnostic and shared with scurvy, therefore high prevalences are expected. The least observed feature is fraying of the growth plates with 9% prevalence overall (in all cases overall).

In probable cases, osteopenia of the growth plates and metaphyses is the only feature recorded in all cases. Trabecular coarsening and a thickened cortex are also very common, with over 80% prevalence in probable cases. All other features were very

common, present in over half of documented probable cases. The only exception is fraying of the growth plates with a prevalence of 16%. No feature was unobserved.

The prevalence of observed features is similar to probable cases but lower in possible cases. No feature was always observed but cortical thinning followed closely with a prevalence of over 80%. All other features were observed in over half of the possible cases except for fraying of the growth plates (17% prevalence) and osteopenia of the epiphyses which could not be recorded in any possible case due to lack of preservation.

In unlikely cases, osteopenia of the epiphyses was recorded in all individuals, closely followed in frequency by osteopenia of the growth plates and metaphyses with a prevalence of 71%. All other features were uncommonly observed with prevalences under 37%. No feature was unobserved but fraying of the growth plates, as in probable and possible cases, had the lowest frequency at 14%.

Cases of co-occurrence have similar feature frequency to possible cases. No feature was always observed but cortical thinning and osteopenia of the growth plates and metaphyses followed closely with a prevalence of over 80%. All other features have a prevalence of 50% or greater except for fraying at the growth plates which was unobserved in any co-occurrence case.

6.3.3. Microscopy Results

Microscopic data was evaluated in two parts, scanning electron microscopy (SEM) and histological stains. The results of these techniques can be found in the following sections. In addition to these results, a thin section for light microscopy of individual 267 consulted by Dr. Megan Brickley at the CAD-DAP was found to have remnants of buried osteoid seams, suggesting a possible case of rickets (pers. communication, January 2014).

Histomorphometry was considered to quantitatively evaluate cases of rickets. The technique would have allowed a quantitative comparison of bone quantity between the archaeological samples and expected values for normal as well as rachitic juveniles. However, required standard values for neonates and juvenile ribs could not be found in the literature. Values concerning total area, total cortical bone values for ribs were available but none were found for trabecular bone area (e.g., Streeter 2005, Takahashi & Frost 1965, Takahashi & Frost 1966). The absence of this value is problematic for calculating total bone volume. The values were found in older literature when rib biopsies were commonplace but today modern medicine typically relies on hip biopsies (e.g., Glorieux et al. 2000) when sampling bone for rickets or osteomalacia. Archaeological research has produced some values for the cortical portion of the bone but the trabecular portion has not yet been investigated (Streeter 2005).

6.3.3.1. Scanning Electron Microscopy Results

A summary of rachitic cases diagnoses utilizing SEM data can be found in Table 6.6, Section 6.3.2. The majority of samples (n=12; 44.4%) did not yield any features associated with rickets and were classified as unlikely. Six samples were classified as probable (n=5) and possible (n=1) cases. Unfortunately, a third (n=10; 37.04%) of the 27 individuals sampled were too poorly preserved at a microscopic level to assess rachitic features.

The prevalence of observed SEM features associated with rickets are compiled in Table 6.8. Complete SEM feature data is available in Appendix IV. In total, the most common feature was abundant Howship's lacunae, a non-diagnostic feature. The least common feature was poorly mineralised osteocytes, recorded in less than a tenth of cases.

No SEM feature was observed in all probable cases of rickets. However, buried osteoid remnants, separation from newly formed bone and poorly mineralised osteons were the most common with 67% prevalence. Poorly mineralised osteocytes were only observed in a third of probable cases and abundant Howship's lacunae were unobserved in any probable case.

Only two features were observed in possible cases, Howship's lacunae (100% prevalence) and separation from newly formed bone with only 25% prevalence. In unlikely cases, only three features were observed, Howship's lacunae (60% prevalence), poorly mineralised osteons and osteocytes. The both possible features had prevalences under 35%.

Prevalence of features in cases of co-occurrence most resemble unlikely cases. No feature was always observed in co-occurrence cases and no feature had very high prevalence rates. Only Howship's lacunae was observed with a prevalence slightly over 50%. Closely following is poorly mineralised osteons and buried osteoid remnants with between 30% and 45% prevalence. Only probable and co-occurrence cases displayed buried osteoid remnants.

TABLE 6.9. Summary results of observed prevalence for SEM rachitic features. [1] *Counts do not include individuals affected by co-occurrence (classified as probable or possible).*

Feature observed	Probable Cases[1]	Possible Cases[1]	Unlikely Cases[1]	Too Little Information Cases[1]	Co-occurrence Cases	Total Observed
Buried osteoid remnants (including seams)	2/3 (66.67%)	0/3 (0%)	0/10 (0%)	0/1 (0%)	2/6 (33.33%)	4/23 (17.39%)
Separation of new bone from old bone	2/3 (66.67%)	1/4 (25%)	0/9 (0%)	0/1 (0%)	0/7 (0%)	3/24 (12.5%)
Poorly mineralised osteons	2/3 (66.67%)	0/3 (0%)	3/9 (33.3%)	1/1 (100%)	3/7 (42.86%)	9/23 (39.13%)
Poorly mineralised osteocytes (bearded)	1/3 (33.33%)	0/3 (0%)	1/9 (11.1%)	0/1 (0%)	0/7 (0%)	2/23 (8.70%)
Howship's lacunae; abundant	0/3 (0%)	4/4 (100%)	6/10 (60%)	0/1 (0%)	4/7 (57.14%)	14/25 (56%)
Total Individuals diagnosed	4	4	10	2	7	27

The results are given as the number of individuals observed with a feature out of the number of individuals where the feature could be observed.

6.3.3.2. Villanueva Osteoid Bone Stain Results

The Villanueva thin sections appeared to stain well following the protocol as outlined in Chapter 5, Section 5.3.3.3.2. However, once the thin sections were mounted on slides and the grinding process began, the stain was slowly be removed as the bone thinned. Attempts were made re-staining mounted bone thin sections, see Chapter 5, Section 5.3.3.3.2.

Each re-staining attempt resulted in the stain reacting poorly with the UV glue. The stain would harden into a jelly-like substance and often cover the thin section (See Figure 6.1.A). Limiting the amount of stain used helped reduce the effect but not halt it. The reaction also caused the section to lift away from the slide (See Figure 6.1.B). When pieces of cotton ball were used, these clung to the section and could not be easily removed. Villanueva stain also requires moisture which was challenging to retain while limiting amount of stain used for poor reaction with UV glue. As a result, the thin section would harden, crack, flake off, and disintegrate. Lastly, the bone did not uptake the stain, either under-or over-staining, with uneven distribution throughout the section. In conclusion, the attempts to re-stain thin sections of bone failed.

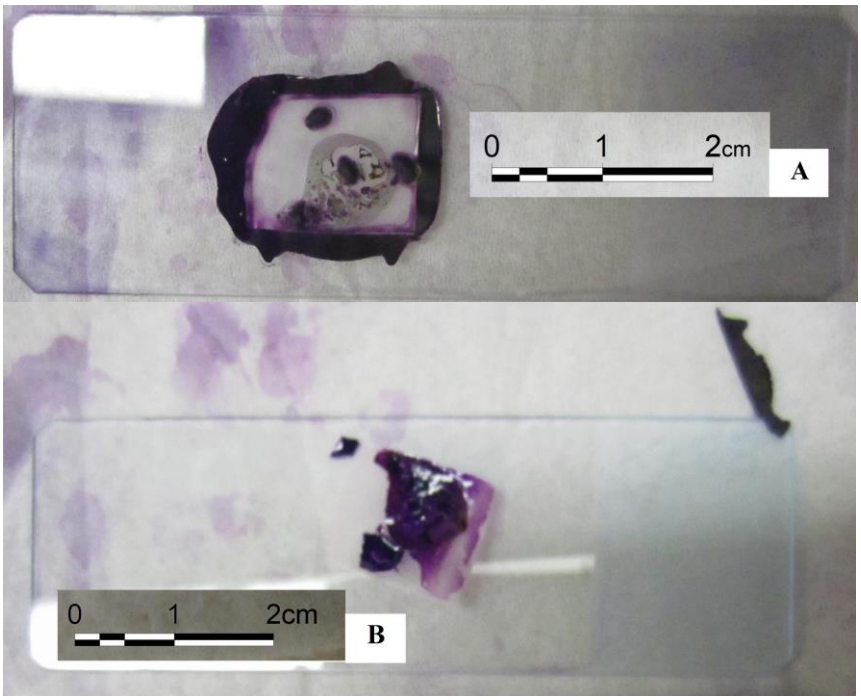


Figure 6.1. Villanueva re-stain tests. (A) Section placed in a sealed plastic container covered with aluminium foil; Left for 90 minutes. (B) Section placed in a glass container, sealed with parafilm, and covered with aluminium foil. Left for 24 hours.

Villanueva staining results were therefore not used to confirm or refute a diagnosis of rickets. Future attempts at developing a re-staining protocol for histological thin sections should be attempted as it would mitigate issues with poor stain uptake and reduce waste produced by creating a large number of thin sections.

6.3.3.3. Goldner's Trichrome Bone Stain Results

Thin sections stained with Goldner's Trichrome were colourful. The stain normally mineralised bone green. In the archaeological thin sections. The mineralised bone portion nearly always stained a colour within the range of green and blue, with variable brightness. These results demonstrating that the stain adequately penetrated the bone samples. However, the stain produced no results confirming a diagnosis of rickets. Results can be consulted in Table 6.10.

Osteoid is stained by Goldner's Trichrome. In the archaeological samples, no thin section demonstrated any red stained areas with the exception of 835, as can be seen in Figure 6.2. However, the red stain is located deep in the cortex of the bone and this is an area where unmineralised osteoid is unexpected (Frost 1962:636). It is expected that buried osteoid takes a shape of a band (Frost 1962) but in this case the bone is stained in spots. Poorly mineralised bone can stain diffusely but in this case, there is no clear association between the stain and bone element (e.g., Haversian system, osteocyte) (Frost 1962). Lastly, the colour is a very dark red-wine while the colour should approximates an orange-red, as in Figure 5.4., Chapter 5.

Some unexpected colours were also observed on the archaeological thin sections, including a bright yellow (see Figure 6.3.), and a brown to near clear colour (see Figure 6.2). The bright yellow was sometimes found along the outside borders of the rib cortex. These borders correspond to an area of poor mineral density under the SEM (see Figure 3.6.). The brown, near clear, colour did not correspond with anything consistently but appears to likely be inclusions within the bone such as soil, which is expected in archaeological remains.

TABLE 6.10. Results of Goldner's Trichrome stain. Notes describe the amount the bone stained a particular colour. A distinction is made when bright yellow appears along the outside border of the rib cortex. When not specified, yellow was observed within the bone cortex or along some edges of trabecular bone.

Juvenile	Osteoid (red)	Normal Green	Bright Yellow	Brown or near clear colour
6	No	Yes, nearly all	Yes, few	Yes, little
7	No	Yes, nearly all	No	Yes, little
37	No	Yes, nearly all	Yes, few	Yes, little
95	No	Yes, all	No	No
91	No	Yes, nearly all	Yes, few	No
110	No	Yes, nearly all	Yes, small border	No
124	No	Yes, all	No	No
125	No	Yes, nearly all but large portion blue	Yes, small border	No
153	No	Yes, nearly all	Yes, borders and in bone	No
190	No	Yes, all	No	No
208	No	Yes, nearly all	Yes, some	No
218	No	Yes, nearly all	Yes, some	Yes, some
219	No	Yes, nearly all	No	No
221	No	Yes, nearly all	Yes, few (borders and in bone)	No
232	No	Yes, nearly all	Yes, few	No
270	No	Yes, nearly all	Yes, few	No
272	No	Yes, nearly all	Yes, some	Yes, some
273	No	Yes, nearly all	Yes, borders	No
307	No	Yes, nearly all	Yes, some (slight border)	No
414	No	Yes, nearly all	Yes, some	No
473	No	Yes, nearly all	Yes (borders and in bone)	Yes, few
514	No	Yes, some, and about ½ of bone is blue	Yes, nearly ½ of the cortex and thick borders	No
528	No	Yes, nearly all	Yes (borders and in bone)	Yes, some
634	No, possible blue-red spots, unclear	Yes, ½ of the bone	Yes, some	Yes, lots
647	No	Yes, nearly all	Yes, borders and in bone	No
678	No	Yes, nearly all	Yes, some	No
835	No but wine colour patch	Yes, nearly all	Yes (borders and in bone)	Yes, some

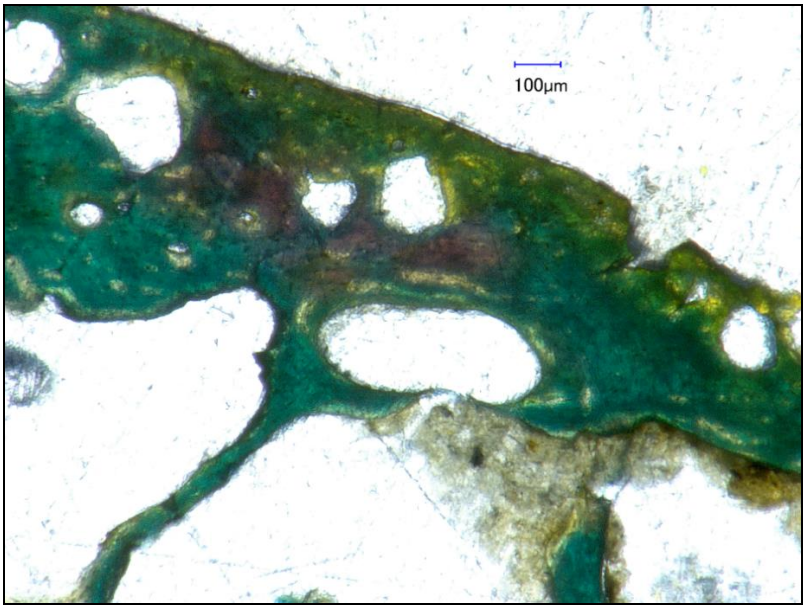


Figure 6.2. Goldner's stain: wine colour. Juvenile 835 x200 VH-Z20R (RZx20-x200) lens, sharpening mode. Picture of the wine-coloured patch observed in the upper middle portion of the graphic. On the bottom right of the picture, there is the "brown or near clear" colour observed.

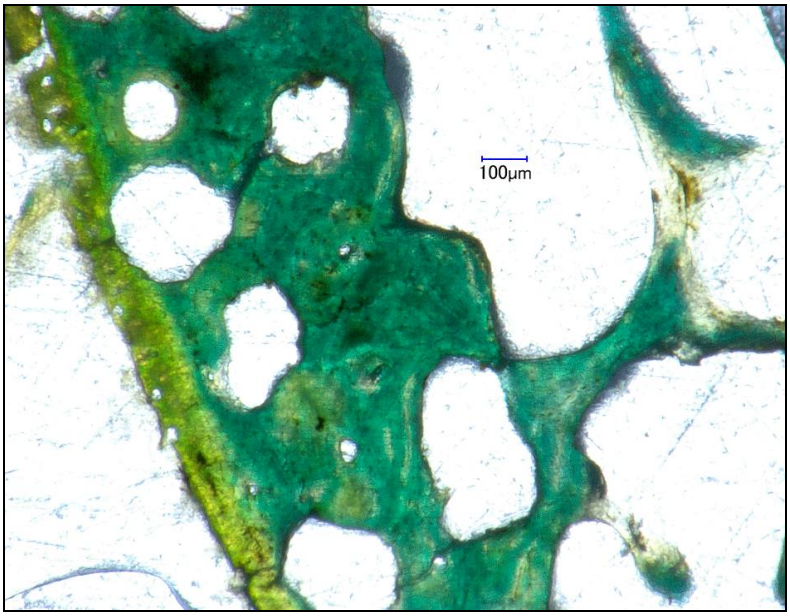


Figure 6.3. Goldner's stain: yellow border. Juvenile 835 x200 VH-Z20R (RZx20-x200) lens, sharpening mode. The image demonstrates a bright yellow border along the left edge and yellow spots of demineralisation within the bone, around the osteons. The graphic also shows areas of a blue-gray colour along the edges of the bone which is the resin.

TABLE 6.11. Final diagnosis of scurvy and rickets co-occurrence for all the Saint-Amé juveniles. TLI = Too Little Information.

Juvenile	Scurvy Final Diagnosis	Rickets Final Diagnosis	Co-Occurrence?	Juvenile	Scurvy Final Diagnosis	Rickets Final Diagnosis	Co-Occurrence?
6	Probable	Unlikely	Unlikely	232	Unlikely	Possible	Unlikely
7	Possible	Unlikely	Unlikely	264	Probable	Probable	Probable
18	TLI	TLI	TLI	267 [265]	Unlikely	Probable	Unlikely
37	Unlikely	Unlikely	Unlikely	270	Unlikely	Possible	Unlikely
56	Possible	Possible	Possible	272	Possible	Unlikely	Unlikely
66	Possible [1]	Possible [1]	Possible [1]	273	Possible	Unlikely	Unlikely
74	Possible	TLI	Unlikely	296	Unlikely	Unlikely	Unlikely
91	TLI	Possible	Unlikely	307	Unlikely	Probable (Healed?)	Unlikely
95	Probable	Possible	Possible	332	Unlikely	Unlikely	Unlikely
100	Unlikely	TLI	Unlikely	361	TLI	TLI	TLI
110	Possible	Possible	Possible	367	Possible	Possible	Possible
111	Unlikely	TLI	Unlikely	385	Possible	TLI	Unlikely
124	Probable	Unlikely	Unlikely	414	Possible	Unlikely	Unlikely
125	Probable	Unlikely	Unlikely	450	Unlikely	Unlikely	Unlikely
153	Unlikely	TLI	Unlikely	473	Unlikely	Probable	Unlikely
175	Unlikely	Probable	Unlikely	514	Possible	Possible	Possible
190	Unlikely	Probable (healed)	Unlikely	528	Probable	Unlikely	Unlikely
191	TLI	Possible	Unlikely	634	Possible	Probable	Possible
208	Possible [1]	Possible [1]	Possible [1]	647	Probable	Possible	Possible
218	Unlikely	Unlikely	Unlikely	657	Possible	Possible	Possible
219	Possible	TLI	Unlikely	678	Unlikely	Possible	Unlikely
220	Possible	Unlikely	Unlikely	699	TLI	TLI	TLI
S221	Probable	Possible	Probable	784	Possible	TLI	Unlikely
231	Unlikely	Possible	Unlikely	835	Unlikely	Probable	Unlikely

[1] Individual possesses other pathological features not associated with either scurvy or rickets

6.4. Co-Occurrence Results

Co-occurrence of scurvy and rickets was determined in cases where individuals were diagnosed as both probable and/or possible for scurvy and rickets. Results are summarized in Table 6.11. The results suggest 12 cases of probable or possible co-occurrence amongst the juveniles five years of age and under from Saint-Amé. Of these 12 individuals, two are probable cases (221, 264) and ten are possible cases (56, 66, 95, 110, 208, 367, 514, 634, 647, and 657). Amongst the possible cases, one juvenile (264) was given a diagnosis of probable rickets and possible scurvy while three juveniles (95, 221, and 647) were given diagnoses of probable scurvy and possible rickets. The remaining seven individuals were given diagnoses of possible for both diseases. Feature prevalence was recorded for each technique (see Tables 6.4., 6.5., 6.7., 6.8., 6.9.) and commented upon in these sections of the chapter.

Two of the individuals (66, 208) listed amongst the twelve potential cases of co-occurrence were identified as having pathological features not associated with scurvy or rickets. Regardless, these individuals remained included in all assessments conducted, including diagnosis and feature prevalence, for cases of co-occurrence. Although a third pathological process was likely occurring in these juveniles with the potential to affect the appearance of scurvy and rickets, the juveniles remained included in the assessments as enough features of scurvy and rickets were observed to warrant a diagnosis of possible scurvy and rickets. Additionally, the cause of death is unknown for all individuals included in this study. It is likely that a number succumbed to disease, or other pathological processes which may have had the potential to affect the appearance and/or disease course of individuals observed in this study.

6.5. Conclusion

The results suggest 12 cases of probable or possible co-occurrence amongst the juveniles under five years of age from Saint-Amé. The study also identified four probable cases of scurvy, nine possible cases of scurvy. Additional rickets cases were also found, including six cases of probable rickets and six cases of possible rickets. Three cases could not be assigned a diagnosis due to a lack of completeness.

Chapter 7: Discussion

7.1. Introduction

This chapter will discuss the major findings of suggested cases of scurvy and rickets co-occurrence from Saint-Amé using clinical and paleopathological perspectives. The discussion will focus on answering the thesis' two central questions to provide insights on identifying cases of co-occurrence in future paleopathological research. The questions are first, is it possible to diagnose cases of co-occurrence? And second, how might co-occurrence affect the appearance of scurvy and rickets features? Lastly, the discussion will contextualise the results within the cultural and historical framework of 16th to 18th century France.

7.2. Is it Possible to Identify Cases of Scurvy and Rickets Co-Occurrence in Paleopathology?

The ability to identify cases of scurvy and rickets co-occurrence in paleopathology is a central question of the thesis. The results of research conducted on remains from Saint-Amé indicate that it is possible and twelve cases of co-occurrence were suggested.

The use multiple techniques in the current study was critical for co-occurrence identification as it expanded the number of features potentially observed and likelihood of recording features associated with either disease. The ability to suggest a diagnosis of scurvy and rickets is particularly challenging in paleopathology as the diseases have no pathognomonic features and many of their features are non-diagnostic. The observation of numerous features in locations across the skeleton is therefore required to suggest the presence of disease, otherwise confidence in suggesting diagnosis is significantly reduced. The process proved successful in the current study. For example, juvenile 647 from Saint-Amé did not present with clear macroscopic or rachitic features but the SEM (scanning electron microscopy) sample had remnants of buried osteoid. The observation of buried osteoid provides some evidence for suggesting the individual was affected by a previous episode of disruption in the mineralization process, such as rickets (Teitelbaum et al. 1976; Priemel et al. 2010). Without the use of microscopy, the evidence of mineralization disruption would have been missed. The use of multiple techniques is also especially important as co-occurrence may mask the typical expression of rachitic and scorbutic features.

Each technique utilized in the current study provided various challenges to identifying disease. Macroscopically, infant and young child bone is challenging to study as the bones were undergoing significant growth and development during life which naturally produces features such as porosity, new bone formation, and imperfect porous metaphyses. Differentiating between normal growth and pathology is difficult.

Definitions of pathology have been produced to reduce the confusion, for example, Ortner and Ericksen (1997) describe porosity as large and penetrating the bone, features which differ from natural porosity. New bone resulting from natural growth appears as layers and is arranged differently from the patches observed in scurvy. Metaphyseal porosity is also defined by Ortner et al. (2001) but it fails to account for difference in bone size. Developing a new method based on distance from major bone features for metaphyseal porosity may alleviate the issue. Infantile bone shape also rapidly changes during growth and the metaphyseal area is naturally enlarged. The use of a comparative sample containing individuals of comparable age and no known disease or pathology is also very helpful to identify abnormal features. Unfortunately, such a sample was unavailable for the study. Therefore, bones from different individuals were compared between each other to reduce false positives when recording features such as bending and deformities. For example, the right femur of all individuals recorded were compared between each other.

Radiographic assessment in paleopathology is also challenging as taphonomic processes are acting on and changing the appearance of the bones. Complications observed in the Saint-Amé sample include soil infiltration and poor bone preservation. Trabecular bone is particularly vulnerable to post-mortem damage and confusion can arise between pathological bone loss and degradation of the bone. The issue was reduced by simultaneously consulting macroscopic photographs of the bones when scoring radiographic features.

Diagenesis is an important issue affecting the microscopic samples. Half of the samples (14/27 samples) had sufficient diagenetic change resulting in inability to score many of the SEM features associated with rickets. Another seven samples could be considered to have moderate diagenetic change as some areas of the section were preserved but a significant portion was diagenetically altered (see Figure 7.1). Only six samples were considered good and the entire surface could be scored for rickets. The number of samples affected by diagenesis is unfortunate as it significantly reduced the utility of this technique in assessing rickets in the Saint-Amé collection. However, the technique is still useful and recommended to researchers as it did provide evidence supporting a diagnosis of probable or possible rickets in six cases. Selecting bone with little diagenetic change for microscopic analysis is difficult. The macroscopic appearance of bone is not an indicator of diagenetic change as damage is internal and caused by microorganisms (Bell 1990) (see Figure 7.1.). Cracking in the cortex and trabeculae was observed on the SEM samples and is an unfortunate side effect of the embedding process and cutting methods employed. Inexperienced researchers could potentially confuse cracking with remnants of osteoid seams (see Figure 7.1) and can refer to Bell's (1990) descriptions and images. The method for embedding the samples was also imperfect. Some rib samples broke apart during the embedding process and little remained. Many air bubbles also became trapped in the embedding medium which had the potential to obstruct the view of the sample,

though rarely completely inhibited an even surface to be cut for SEM analysis. Nonetheless, SEM is a very useful tool.

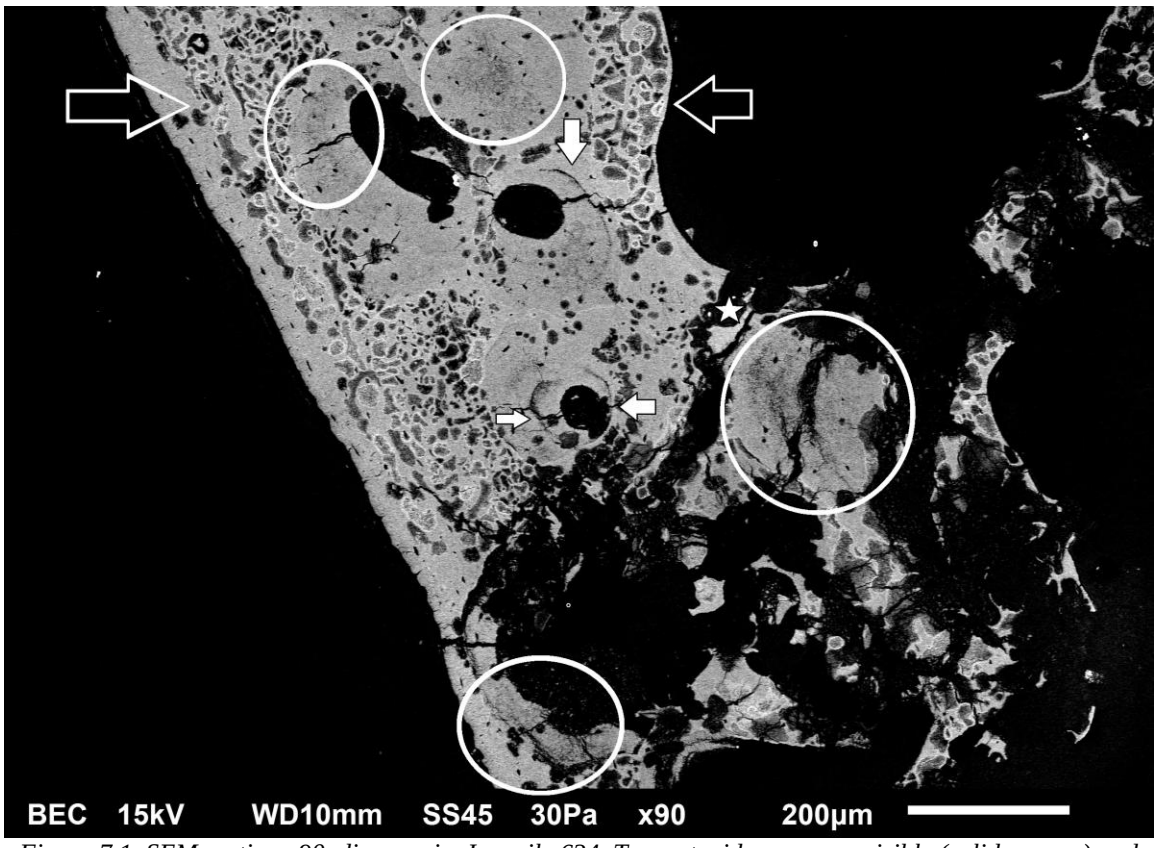


Figure 7.1. SEM section x90, diagenesis. Juvenile 634. Two osteoid seams are visible (solid arrows) and areas of slightly mineralised or 'cloudy' (circles). Diagnostic change (open arrow) and cracking (start) are visible.

Preservation, including completeness of the skeleton and amount of taphonomic change, is a key issue which affects all of the techniques by significantly impacting the visibility of paleopathological evidence required for identifying disease and suggesting co-occurrence. Completeness and taphonomic change of each individual was scored following the methodology outlined in Chapter 5, Section 5.3.2., and summary results for suggested co-occurrence cases can be consulted in Chapter 6, Section 6.2. A minimum amount of bone is required as certain areas of the skeleton have been privileged over others as locations of key features for disease identification in the paleopathological literature. Although there is some overlap between the key areas for scurvy and rickets, there are also important differences. A largely complete skeleton is therefore preferable when attempting to identify co-occurrence of scurvy and rickets. In paleopathology, macroscopic features of scurvy focus significantly on lesions of the cranium with few post-cranial ones (e.g., Ortner et al. 2001; Brickley & Ives 2006). Clinically, post-cranial

lesions are well documented (e.g., Barlow 1883; Fain 2005) but the features producing porosity and new bone, are challenging to identify in paleopathology as discussed previously. These features are also common to many diseases and cannot by themselves identify scurvy in a young juvenile. In contrast, macroscopic features of rickets rely heavily on the post-cranial skeleton for positive diagnosis (e.g., Mays et al. 2006). Radiographic features of both diseases rely on changes observed in long bone diaphysis, metaphyses, and epiphyses (Stark 2014; Mays et al. 2006). Thus, the skull and some post-cranium are critical for scurvy identification while the post-cranium, particularly the long bones, are critical for rickets identification.

The infants and children from Saint-Amé varied from good to poorly complete, in keeping with most archaeological collections. Those suggested to be affected by co-occurrence varied in completeness but the majority were moderate or more complete. Suggesting co-occurrence was however not impossible in very incomplete skeletons but was dependent on which skeletal elements were present and the expression of pathological features. In three cases, a variety of bones fragments, including skull, ribs, and limb bones were recovered, permitting a variety of locations to be scored. A typical skeleton suggested to be affected with co-occurrence would appear as described. The skull bones were highly fragmentary but the lesions of interest (new bone and porosity) could still be scored. At least a few sternal ends of ribs, an important location for features of both diseases, were recovered but the ends are often separated from the rest of the rib. At least half of the limb bones were usually preserved with minimal damage. Depending on the age of the child, upper limbs or lower limbs may be more useful to identify rickets.

Taphonomic damage can also obscure features which may otherwise be present, inhibiting disease identification if the taphonomic changes are severe. Porosity observed in scurvy and rickets is a non-diagnostic feature which can be confused with taphonomic damage on the skeleton. Damage present in the metaphyses will also significantly limit the ability to score rickets features such as changes on the growth plates which are easily damaged post-mortem. Good completeness with a surface clear of taphonomic change is ideal, but disease diagnosis may be feasible with up to moderate taphonomic damage. In keeping with this notion, taphonomic score results for the juveniles of Saint-Amé were found to vary between minimal to moderate taphonomic change.

Age determination significantly impacts the understanding of the underlying disease process, likelihood an individual was affected by a disease, and expected pathological features. Age determination using archaeological remains is imperfect. Many factors impact skeletal growth and the remains represent juveniles who did not survive. Caution is particularly important in this sample as scurvy and rickets can cause delayed or decreased growth, and stunting (Alvarez & Navia 1989; Ellender & Gazelakis 1996; Sakamoto & Takano 2002; Pai & Shaw 2011). Vitamin D deficiency can also delay dental development (Mankin 1974a). Other factors which can delay or stunt growth in

both limb bones and dentition include normal random variability, sex of the individual, nutrition, health status, disease, socioeconomic status, environment, and genetics amongst others (Miles 1963; Liversidge et al. 1998). Recently, at the histological level, White and Booth (2014) did not observe any diagenetic changes in bones of still born neonate pigs buried and left exposed on the ground surface for one year. The authors ascribe the absence of change to a paucity of internal gut bacterial colonies which form during the first weeks of life. The study was conducted over only one year so exogenous bacterial sources may not have had time to exert much effect on the pig bones. The high amount of diagenetic change observed microscopically on the rib samples from Saint-Amé would, if White and Booth (2014) are correct, indicate the juveniles may be older than foetal or neonatal. The idea supports the notion that the age estimation results (see Table 6.1.) are underestimating the true chronological age of the juveniles and both scurvy and rickets were likely present and impacting growth. The assigned age should therefore be interpreted as a minimum age, or biological age, instead of a chronological age. Confidence in the assigned ages is also affected by amount of material preserved, increasing proportionately to the amount of material available, and errors built into the formulas. A summary of the applicability of various aging techniques can be found in Chapter 5, Table 5.1 and full data in Appendix I. Further investigation into aging may provide confirmation of underaging. For example, enamel is known to be laid down following a known "regular circadian rhythm" of roughly 24 hours (e.g., Antoine et al. 2009). Dental cross sections can be made to count the cross-striations of the enamel microstructure to provide an age at death estimation. Although the technique is highly accurate, it is destructive, highly specialised, and time consuming.

In summary, it is possible to identify cases of co-occurrence and the current methodology is recommended for future research in co-occurrence. Ultimately, identification will rely on the presence of numerous features across the skeleton. Cases present in poorly preserved collections will likely, but not always, be missed as not enough data will be available to suggest the presence of disease with confidence. Nonetheless, the use of multiple techniques can help mitigate the problems associated with preservation and variability, discussed in the next section. The current study relied heavily on the presence of macroscopic and radiographic data with support from some SEM data. SEM is an expensive technique and accessibility can be limited. Nonetheless, it was useful but not required to suggest the presence of co-occurrence. Understanding and considering the limitations and factors which impede the process of diagnosis is important for future research on the co-occurrence of scurvy and rickets co-occurrence.

7.3. Disease Sequence and Impact on Presence or Absence of Features

The presence or absence of a scorbutic or rachitic feature is determined by disease order, specifically which disease occurs first and how much it develops prior to the second disease occurring. Knowing which disease occurred first is near impossible when

studying archaeological skeletons as their clinical history is rarely available. Though, a guess could be made based on observed features and their severity. Greater certainty can be given to assessing disease dominance as the features of this disease appear more prominently and better developed. In the current study, scurvy was likely the dominant disease as both its macroscopic and radiographic features were well developed. The result is consistent with clinical literature which found that scurvy is typically, but not always, dominate over rickets (Bromer & Harvey 1948; Follis et al 1940]. Although the sequence of deficiency remains unclear, the majority of cases are likely scurvy supervening onto rickets. This sequence is the most commonly documented disease order clinically (Cheadle 1878; Barlow 1883; Bromer & Harvey 1948). In such cases, the features of scurvy are clear but those of rickets vary from mild to unobservable. Scurvy supervening upon rickets is likely the most visible disease order sequence in the archaeological record as there is an increased chance for features of both diseases to develop and due to paleopathology's limitations in diagnosing disease, as discussed in the Section 7.2.

The juveniles suggested to be affected by co-occurrence from Saint-Amé have a variety of disease expression for both scurvy and rickets. Some, have features considered by Ortner et al. (2001; 1998) as a well-developed form of scurvy (e.g., new bone formation and porosity compared to porosity only) and severe rickets (e.g., long bone bending and flaring), but the expression is usually mild. Nonetheless, the prevalence of features observed in co-occurrence cases more closely resemble those of probable scurvy and possible. The presence of clear features of rickets necessitates that the disease had enough time to develop before scurvy affected the individual. Rickets was scored as probable for only two juveniles (264, 634) suspected of co-occurrence while a score of probable scurvy was given to a quarter of co-occurrence cases (95, 221, 647). The remaining seven co-occurrence suspected individuals were classified as possible for both diseases.

An even stronger case for scurvy supervening upon rickets can be made for four specific individuals (66, 95, 634, and 514) from Saint-Amé. Each of these juveniles has a moderate expression of rachitic features with no evidence of bending in the limb bones but flaring was present. Individual 634 also had evidence of remnants of osteoid seams and poorly mineralised osteons. The scorbutic features in these cases are clear with the majority of sites displaying porosity, the presence of new bone, and both the Fraenkel white line and scurvy line on radiographs. These results are consistent with the notion that the features of scurvy are most likely to be explicit while those of rickets are infrequently explicitly expressed. However, as the features of both diseases are present, the possibility of the reverse disease sequence cannot be excluded.

Juvenile 647 is a particularly interesting case from Saint-Amé whereby rickets could have occurred first but did not develop much prior to the occurrence of scurvy. The features of scurvy are clearly present with porosity on the majority of sites, presence of new bone formation, and both a Fraenkel white line and scurvy line. As for evidence of rickets, the

bones do not display clear features (e.g., bending or flaring of the long bones) except for the presence of a remnant of buried osteoid observed in the SEM. The remnant of osteoid (see Figure 3.1) was buried within the bone, indicating a previous episode of disruption in bone forming process, possibly rickets. The evidence suggests rickets occurred first followed by scurvy developing secondarily (Teitelbaum et al. 1976; Priemel et al. 2010). The extent and overlap of the two diseases is unclear but, if rickets did occur the disease may have been resolved at the onset of scurvy as scurvy reduces bone formation and osteoid deposition. The individual aged quite young, within the 'fetal/birth' category. The presence of deficiencies indicate congenital disease, likely shared with the mother, or the techniques are under-aging the infant.

Disease sequence is not the only factor to determine the presence or absence of features. To complicate matters, clinical research clearly demonstrates that either rickets or scurvy disease process can interfere with the competing disease and inhibit the development of its features. Rickets compromises the mineralization of new bone which inhibits the development of certain scorbutic features. Examples include subperiosteal new bone formation, the dense white line of Fraenkel and scorbutic lattice (at least partially) (Park et al. 1935; Bromer & Harvey 1948). Porosity, a primary paleopathological feature of scurvy, would be unaffected by rickets as it is the result of increased vascularization in response to the circulatory system, specifically inflammation and haemorrhaging. Severe or longstanding scurvy, in turn, impairs endochondral growth and growth in width of bones due to a reduction in osteoblastic activity and osteoid production (Hess 1916; Park et al. 1935; Fain 2005). These changes would limit the development, or mask, key rickets features which require bone growth, such as flaring of the metaphyses, and remodelling, such as bone deformities, to develop. However, Follis et al. (1940) found that osteoid development was not affected in cases of co-occurrence which could indicate that some rickets features, such as thickened osteoid bands, may still occur in the presence of scurvy. Similarly, mineralisation may still occur but is significantly delayed in rickets unless disease is extreme. For example, Marie et al. (1982) observed some, though very little, bone mineralization in clinical cases of severe childhood calcium deficiency osteomalacia. The effects of the underlying processes and interactions can be observed at all levels and all techniques, including macroscopy, radiology and microscopy. The interaction ultimately inhibits the development of severe features of either disease which are uncommonly reported in cases of co-occurrence (Fouron & Chichoine 1962; Evans 1945).

Receiving trace amounts of vitamin C and/or vitamin D can reverse the deficiency process, slightly, allowing the development of otherwise masked features such as new bone formation (scurvy) and bone ossification (rickets). For example, Mays (2014) notes that receiving trace amounts (2-5%) of vitamin C allowed osteoblasts to resume some function in animal experiments. This data suggests that total absence of osteoid development and mineralization are unlikely except in very exceptional conditions such

as severe cases or experimental tests where absolutely no trace amount of the vitamins can be ingested. Nonetheless, deficiency was still occurring and the juveniles developed features associated with scurvy and/or rickets. The relationship between the two diseases won't change by receiving trace amounts of vitamin but some features otherwise masked may develop. It is unlikely that many of the cases from Saint-Amé could be considered as extreme cases of scurvy or rickets as, for the most part, the expression of paleopathological features are overall slight to mild. Cycles of disease and healing are possible which can lead to the appearance of new bone formation and remnants of buried osteoid seams within the bone.

Scurvy can increase the risk of death, as noted by Follis (1942) and Grewar (1965). If death occurs before typical paleopathological features have a chance to develop, it will be near impossible to identify the individual as being affected by disease in an archaeological setting. Time of development is therefore important to recognise and a limiting factor in identifying cases of disease and co-occurrence. Clinicians can circumvent this issue by using soft tissue, such as serum level tests of vitamin C and D. In summary, the presence or absence of features is determined by the order of disease occurrence, disease interaction, and the amount of time a disease can develop before the secondary disease occurs. Many cases of co-occurrence will likely be missed in archaeological collections as a certain amount of bony evidences of both diseases is required to suggest the presence of disease. The identification and description of more cases of co-occurrence will allow for a greater understanding of disease sequence, disease interaction and their consequences on the presence or absence of features.

7.4. Expected and Observed Features in Cases of Co-occurrence in Paleopathology

Clinically, a number of case studies have been reported and some research has produced insights into the possible observed features in paleopathological cases of co-occurrence. As discussed in the previous section, the appearance can be quite variable. Archaeologically, the materials available and constraints are quite different to the clinical setting. Original paleopathological research is therefore important to fill in any gaps of knowledge.

Recently, a few paleopathological cases of scurvy and rickets co-occurrence have been reported (Lewis 2010; Devriendt et al. 2010; Redfern 2012; Geber & Murphy 2012; Klaus 2014) but a detailed report on observed features has not been published as the aims of the research was not related to co-occurrence. The results of the current study can begin to fill the gap on the understanding of feature expression in cases of co-occurrence with the extensive recorded datasets on macroscopic, radiographic and microscopic features. The wide range of features scored provides a starting point but the data is not exhaustive due to the potential variability observed and small sample size.

Overall, a good consistency of prevalence percentages was observed between features recorded in cases of co-occurrence to those of singular disease, with the occasional exception. These results suggest that most macroscopic and radiographic features of either singular disease could still be observed in most cases of co-occurrence. Fractures on the sternal growth plate, a macroscopic scurvy feature, were nearly only observed (83% prevalence) in cases of co-occurrence while rarely or unobserved in cases of singular scurvy. Fractures can also develop in cases of rickets such as the compression fracture observed by Ellis (2010) on a metaphyseal growth plate. Data on metaphyseal growth plate fractures was not collected in the current study but should be further investigated. Scurvy and rickets, when longstanding and/or severe, cause a net loss of bone. Weakened bone is easier to fracture and therefore fractures are not unexpected in co-occurrence cases. However, fractures are typically non-diagnostic as they can occur due to a wide variety of causes including a number of diseases which result in pathological bone and force. Although the high prevalence of fractures in co-occurrence cases is interesting, the feature is not exclusive to or identifying of co-occurrence cases. The presence of features common to either disease remains the best means of distinguish cases of co-occurrence from cases of singular scurvy or rickets. The conclusion is in keeping with the clinical reports of co-occurrence (e.g., Cheadle 1878; Barlow1883; Follis et al 1940; Bromer & Harvey 1948; Fouron & Chichoine 1962; Lewis et al. 2006). For example, in the current study orbital roof porosity prevalence was high in cases of co-occurrence (55% prevalence) but was not recorded in cases of singular rickets. The difference in expression is due to the presence of scurvy in co-occurrence cases. Overall, a range of expression from clear to very mild scurvy and rickets was observed. The findings are consistent with the majority of cases being identified as possible disease rather than probable. Some differences between the different diagnosis levels (e.g., probable or possible) were observed but this is consistent with the diagnosis methodology which classified individuals on the likelihood of being affected by disease based on the types of features observed, with severe features being more diagnostic. A detailed explanation of the methodology is available in Chapter 5, Section 5.3.3. Some of the features, for example bending of the limbs, were observed in very young children, including those estimated to be fetal or neonatal age at death. These results seem unlikely but the exact age at death of the children is unknown and underaging is possible, as discussed in Section 7.2.

In the following sections, I will expand on the results and discuss the trends observed of features recorded in cases of scurvy and rickets co-occurrence. Discussion will concern the possible presence or absence of features which have been reported in the clinical literature and could also be observed in archaeological bone. Some features reported clinically using radiographic images will be discussed within the macroscopic section as these features are typically scored macroscopically in paleopathology, for example, changes at the growth plate and fraying. Full details of the results can be consulted in Chapter 6. When discussing cases of unique disease, data will include results of cases

diagnosed as probable and possible cases of singular disease. Slight variation in observed prevalences will be disregarded due to small sample size.

7.4.1. Macroscopy

The majority of features observed in clinical studies and autopsies of co-occurrence are scorbutic in origin. Expected macroscopic appearance for cases of co-occurrence, based on the clinical literature, includes new bone formation, particularly on the lower limbs, enlarged sternal rib ends, possible but not obligatory observation of fractures in the metaphyses of bones. Porosity, caused by swelling and bleeding onto bone should throughout the body, including the limbs (subperiosteal), mouth (alveolar process of maxilla, mandible and the palate), joints (especially the wrists) (Cheadle 1878; Barlow 1883; Barlow 1894; Owen 1899; Bromer 1946; Lewis et al. 2006). Expected rachitic features include frontal bone bossing, swelling of limbs, joints (metaphyses) and ribs (as in scurvy). Prevalence results for all scurvy and rickets features can be consulted in Chapter 6, Table 6.4. and 6.7. respectively. Dental pathology was not recorded in the current study. Future work may provide additional information on this subject.

Results of scorbutic feature prevalences, as observed in cases of co-occurrence, are consistent with the expected expression from clinical research. All the features scored to identify scurvy were observed in cases of co-occurrence. All but four of 24 features (83% of features) scored have prevalences around 50% or above. Clearly, the features of scurvy were present in a majority of cases. In comparison, seven of 17 features (41% of features) scored for rickets have prevalences of 50% or greater. The expression of rickets was very subtle in the cases from Saint-Amé.

Features associated with vascular changes of scurvy should be present in most cases of co-occurrence as the vascular system is not affected by rickets. Bony hypertrophy can be affected by rickets due to disruption of the mineralisation process (Follis et al. 1940; Pitt 2002). Fouron and Chichoine (1962) failed to find evidence of subperiosteal haemorrhages on radiographic assessments of five co-occurrence cases, aged five months to a year old. In the current study, the prevalence results of new bone development on the cortex of the limb bones were unexpectedly very high in the children from Saint-Amé. Prevalences for arms and legs are 83% and 89% respectively for co-occurrence cases. In comparison, metaphyseal porosity for the same limbs are 50% and 57% respectively. The results contradict those of Ortner et al. (1999; 2001) which found that porosity was usually more common than bone hypertrophy. The authors reasoned the lower occurrence of new bone was because hematomas and new bone formation represent a more severe bleeding reaction and required the periosteum to lift rather than localised bleeding and inflammation which result in porosity alone. One potential explanation for the current study's results is that the children had access to trace amounts of vitamin D or the mineralization was not completed disrupted by rickets. Either example would support the

development of new bone. The prevalence observed in co-occurrence cases is comparable to results for cases of singular disease (probable and possible cases), suggesting scurvy was the dominant process. Another explanation is that high prevalence may not be linked to scurvy. For example, subperiosteal new bone formation is a normal feature of growth in neonates and infants one to four months in age (Kwon 2002). Thus, some difficulty in scoring new bone formation may have occurred as the majority of the children studied were under a year old and the limb bone is very reactive at that age due to rapid growth, both in length and width. If new bone formation was linked to scurvy, following Ortner et al.'s (1999; 2001) reasoning, the high prevalence of reactive new bone could indicate the children were affected by severe or longstanding scurvy prior to death. The higher prevalence of reactive bone on the lower limbs is consistent with clinical cases which typically observed subcutaneous haemorrhages and swelling on the legs (Evans 1945; Bromer & Harvey 1948; Lewis et al. 2006). Barlow (1894) appears to suggest that scorbutic changes occur first in the legs and only later, with continued development, the upper limbs become affected. Subperiosteal haemorrhaging is caused by micro trauma and the legs of a young child may be a vulnerable location. Young children are quite active, moving around, including crawling and walking which would expose their limbs to trauma. Exact risk is variable on socio-cultural and economic context regarding acceptable behaviours. More studies on the appearance of new bone in cases of co-occurrence are required to understand its prevalence in co-occurrence cases.

The expression of porosity in cases of co-occurrence is expected to highly resemble the expression observed in cases of singular scurvy. Across the skeleton, the prevalence of individual locations known to be affected by porosity ranged from 25% to 100% prevalence and was roughly consistent with prevalences noted in cases of singular disease. Locations which scored highly (70% prevalence or greater) are those which have been reported clinically, including the gums (posterior and alveolar process of the maxilla, coronoid process of the mandible) and cranium (cranial vault, and sphenoid lesser wing). Metaphyseal porosity occurred in a lower, but still very common, prevalence (50-56%) which is consistent with clinical evidence of co-occurrence as swelling of the limbs and joints was inconsistently observed (Cheadle 1878; Barlow 1883; Owen 1899; Bromer 1946). Rickets can also cause porosity in paleopathology. Noted locations in this study include the cranial vault, orbital roof, metaphysis, and sternal end of the ribs. Devriendt et al. (2010) point out that differentiating the cause of the porosity, whether scorbutic or rachitic, is extremely difficult if not impossible.

Swelling and bleeding in the mouth are typical features of scurvy (e.g., Jaffe 1972; Popovich et al. 2009; Besbes 2010). As expected, all features associated with the maxilla and mandible were present in over half of individuals scored with co-occurrence. In the current cases, only the juveniles under six months of age scored positive for alveolar porosity, with three cases aged as fetal at death. The results are possibly contradictory to clinical observations which found that reactions of the gums occurred around erupted or

oncoming teeth which can erupt as early as four months (Barlow 1883; Hess 1920; Grewar 1965; Gustafson & Koch 1974). However, the mouth is quite porous naturally at that age and when teeth are developing and erupting. Brickley and Ives (2006) also suggest that suckling and movement of muscles would promote irritation of the blood vessels and subsequent bleeding not related to dental eruption.

Bleeding in the orbits has been observed clinically and one known cause is scurvy. The prevalence of porosity was 55% (roof) and 63% (zygomatic) in co-occurrence cases. In paleopathology, orbital and cranial porosity, with periosteal reactions, are also suggested to be associated with anemia (Ortner 2003). The significant amount of haemorrhaging which occurs in scurvy can promote the development of anemia if significant blood loss occurs. Children are particularly susceptible to developing anemia if their diets are inadequate and suffer from severe diarrhea (Haschke & Javaid 1991), both factors which also promote vitamin C and D deficiency. Scurvy and anemia are documented to have co-occurred together (Bromer & Harvey 1948; Grewar 1965; Jaffe 1972). Therefore, it is possible that some of the children from Saint-Amé were also affected by anemia. Anemia can also be caused by infections (e.g., Tolentino & Friedman 2007; Walter et al. 1997).

Unexpectedly, the prevalence of porosity at the greater wing of the sphenoid was quite low, only 29% in cases of co-occurrence. Porosity at this location is considered by Ortner et al. (1998; 1999; 2001) as the main identifying feature of scurvy in juveniles. In singular cases the prevalence is much higher, 40% in possible and 100% in probable cases. The scapula has a similar vascular organization as the sphenoid (Ortner & Ericksen 1997; Gray 1959). Porosity of the supraspinous region of the scapula in cases of co-occurrence was 54% and the infraspinous region was 27%. In cases of singular disease, the prevalence is similar for the infraspinous region but increase significantly (80-100%) for the supraspinous region. Interaction with rickets should not affect the expression of these features. The results therefore suggest that that although these regions of the skeleton are prone to porosity in cases of scurvy, they are not a required feature to identify scurvy in all cases.

Two other features which are expected in archaeological cases of co-occurrence are flaring and fractures as both features occur in scurvy and rickets. Flaring (83% prevalence) and porosity (66.7% prevalence) of the sternal ends of the ribs scored highly in cases of co-occurrence and their prevalence is consistent with clinical cases of co-occurrence (e.g., Cheadle 1878; Follis et al. 1940). The very high prevalence of flaring may be a result of the feature developing in both conditions. Singular cases of scurvy all had flaring of the sternal ends of the ribs but the feature was moderately observed in cases of singular rickets (25-50% prevalence). Park et al. (1935) suggests that the disease causing the flaring of the sternal end (either scurvy or rickets) can be distinguished from one another but Bromer and Harvey (1948) disagree. Archaeologically, it is unlikely a

differentiation could be made as soft tissue is missing and a cross section of the sternal end of the rib would be required.

Fractures are a feature of severe and/or longstanding scurvy. Over time bone becomes brittle and net bone loss occurs, increasing the individual's risk of developing fractures (Park et al. 1935). In rickets, fractures are uncommon but have been associated with bone loss (insufficiency fractures) (Shore & Chesney 2013). Ellis (2010) found compression fractures on the proximal end of two tibias of individuals suggested to be affected by rickets. In the current study, fractures located on the growth plate of the sternal ends of the ribs have been observed in most cases of co-occurrence (86% prevalence). The only other individual recorded with the feature was an unlikely case of scurvy. No sternal end rib fractures were observed in cases of singular disease though few individuals could be scored for this feature in those categories. The presence of a fracture will not identify co-occurrence cases but may provide support. Further investigations with a large sample size is required to confirm the utility of the feature. Additionally, metaphyseal growth plates of the long bones was not systematically recorded in this project. Collecting data on possible fractures at these location would be very interesting to compare with the results observed at the sternal ends. The presence of a fracture(s) suggest that the scurvy and rickets may have been longstanding and the bone was weakened. Evidence of new bone formation would support the notion that scurvy was well developed, as discussed above, but the lack of clear bending in the bones would suggest rickets was not very well developed. Growth plate fractures have not yet been reported in the paleopathological literature on scurvy. However the ends of the shaft of limb bones or sternal ribs are often damaged in archaeological collections. The presence of fractures in those locations likely increases the area's susceptibility to breakage and recovery of the broken pieces is unlikely. Therefore, the feature is unlikely to survive archaeologically and may explain the few noted instances in the current study. Further investigations should be conducted to review the utility of this feature for co-occurrence identification.

Bending deformities are unexpected in cases of co-occurrence as it is a feature of advanced rickets (e.g., ilium deformities) (Doran 1912; Ortner & Mays 1998; Haduch et al. 2009) and scurvy typically masks this feature. The feature has also not been explicitly observed in clinical cases of co-occurrence with exception of one individual noted by Fouron and Chichoine (1962) who developed severe features of rickets during treatment. The study results are mostly consistent with the clinical observations. Low bending prevalences were observed including deformities of the mandible (20%), limb bones (arm) (16.6%), ribs (0%), and ilium (0%) in co-occurrence cases. Leg bone deformities are the only exception with a prevalence of 44.5%. Few individuals displayed very clear bending noticeable without comparison to other individuals. Individuals with bending of the limb bones also scored positive for most other features scored when assessing rickets. Individuals without bending also display most other features but the expression is subtle. For example, half of the individuals without bending did not have abnormal growth

plates' scores of 3 or 4, a severe and/or longstanding feature of rickets. These results are consistent with the idea that bending is a severe form of rickets. Although the presence of bending is not consistent with clinical observation on co-occurrence, the prevalence of both arm and leg bone bending are much lower in co-occurrence cases than in singular disease. Very slight deformities are also much easier to notice in paleopathology than clinically as paleopathologists are working directly with the bone. Though, modern imaging techniques and future technology are producing very high quality images. Mays and Ortner (1998) suggest that bending is due to weight bearing and that upper limbs would be affected in infancy from activities such as crawling but once a child was walking the lower limbs would be affected. In the current studies, the individuals are usually consistent with this idea. The children with arm bending are between 0 to 6 months of age and those with leg bending are between 6 months to 1 year of age. Children start learning to walk around 1 year of age. Two individuals are exceptions to this trend. One has arm deformities and the other has leg bending but the individuals were aged to the fetal/birth category. These individuals may either be underaged or the bending was caused by a different process than rickets. Completeness of the remains was a bias for assessing bending as complete bones are required but often ribs and many limb bones were missing or damaged. Nonetheless, the absence of overt bending indicates that rickets had some time to develop prior to the onset of scurvy but that scurvy developed before the bending became overt. These results are consistent with clinical suggestions that features of severe disease are typically absent and that scurvy was the dominant disease process, masking rickets features.

Similar to bending, increased porosity on the distal growth plates of the long bones is an important rickets feature. Clinically, the feature is studied in a radiographic context as fraying but the bone surface can be directly scored in paleopathology. When scurvy is dominant, fraying may be masked due to scurvy's inhibition of osteoblastic activity (Follis et al. 1940; Fouron & Chichoine 1962). Fraying is expected when rickets dominates or is well developed (Bromer & Harvey 1948). Fouron and Chichoine (1962) observed fraying in all their cases of co-occurrence. Macroscopically, the overwhelming majority of growth plates scored as a "two" (92% prevalence) in co-occurrence cases. A prevalence of 58.3% was observed in growth plate scored as "three" but and none scored a "four". The results are consistent with the literature and suggest that rickets did develop somewhat before the onset on scurvy. However, development was generally not extensive as most scores fall under the "two" category. The growth plate changes could not be seen radiographically in cases of co-occurrence but a few individuals were scored with singular disease. The results lend credence to the suggestion that the pathological changes of the growth plate are slight and scurvy may have intervened to limit the amount of development. Mays et al. (2006) describe a velvety surface texture which should be considered as a step between the normal and slight roughening (second step) as determined by Ortner and Mays (1998). However, differentiating neonatal growth plates, undergoing massive amounts of rapid growth, and a pathological surface is difficult.

Flaring of the limb metaphyses is the last major macroscopic feature of active rickets. Similar to growth plate changes, flaring was observed in cases of co-occurrence and should be expected in paleopathological cases (Bromer & Harvey 1848). Scurvy can also result in enlarged metaphyses as cartilage matrix accumulates at the growth plate and fractures displace the material (Barlow 1883). Flaring of the arm limb bone metaphyses was typical in the current cases of co-occurrence (83% prevalence) while flaring of the leg limb bones was much less common at 33% prevalence. As mentioned previously, Ortner and Mays (1998) and Mays et al. (2006) have suggested that changes in the arm bones are linked to crawling and leg changes to walking and weight bearing. Unfortunately, the results for arm flaring are not consistent with the age parameters set out by Ortner and Mays (1998) or Mays et al. (2006) but two of the three cases which had leg bone flaring, were between six months of age and one year.

7.4.2. Radiography

Fouron and Chichoine (1962) found that rickets was the dominant disease process in radiographs when studying individuals affected by co-occurrence. Clinically, scurvy was dominant in the same individuals. It is logical as rickets halts the process of mineralization and the features of scurvy require mineralization to appear on radiographs. Specifically, Follis et al. (1940) suggests the white line of Fraenkel, scorbutic rarefaction (e.g., scurvy line), and metaphyseal clefts (Pelkan's spurs) would be unable to develop. However, it is not always the case. Bromer and Harvey (1948), and Follis et al. (1940) found that evidence of rickets was unobservable unless rickets was very well developed. The notion is in keeping with the majority of noted cases of co-occurrence being of scurvy supervening onto rickets. In addition, one of the five co-occurrence cases observed by Fouron and Chichoine (1962) only developed features of rickets after the start of treatment, suggesting that the disease process had been previously masked and was 'liberated' by the treatment of scurvy. A complete list of features observed by clinician is provided in Chapter 4, Table 4.

In archaeological bone, the majority of radiographic features observed in clinical cases of co-occurrence are expected. Features caused by scurvy include rarefaction of the cortex and trabeculae, Pelkan spurs, as well as a dense line of Fraenkel (Bromer & Harvey 1948; Fouron & Chichoine 1962). Expected features caused by rickets include rarefaction, trabecular coarsening, and thickening of cortices from periosteal apposition (Evans 1945; Bromer & Harvey 1948; Fouron & Chichoine 1962; Lewis et al. 2006). Specific result prevalences for the current study are available in Chapter 6, Table 6.5. and 6.8. for scurvy and rickets respectively.

The presence or absence of a dense white line of Fraenkel may indicate whether or not rickets was the dominant disease. Clinically, conflicting ideas on the development of a

dense metaphysis line in cases of co-occurrence are given. Follis et al. (1940) suggest that the dense band at the metaphysis would be thin if rickets occurred first, was severe and longstanding. The feature could develop if rickets was mild (Fouron & Chichoine 1962). The argument is logical as the band is formed by a build-up of mineralised cartilage. However, Fouron and Chichoine (1962) observed a dense metaphyseal plate in their cases of co-occurrence, whereby scurvy was the dominant disease process macroscopically. Bromer and Harvey (1948) found the feature to be uncommon. Similarly to Fouron and Chichoine (1962), the majority (83%) of juveniles from Saint-Amé displayed a white line of Fraenkel line. Other explanations could be given for the appearance of the feature than mild rickets. In most non-experimental or controlled circumstances, children will consume or obtain trace amounts of vitamin C and D, resulting in some mineralization and bone formation. For example, even in the cases of extreme calcium deficiency documented by Marie et al. (1982), some mineralization was observed. Calcium homeostasis is regulated by vitamin D and a deficiency of calcium results in rickets. Two individuals (208 & 657) did not have a thick white line and clinically this is associated with cases of dominant rickets (Follis et al. 1940). Both individuals had many features of rickets, including bending, growth plate scores of 3, rib changes, and flaring for individuals 657. These results suggest that in these two cases, rickets may have been more developed.

The Wimberger's ring is also formed from deposition of calcified matrix within the epiphyses and is expected to not develop due to rickets. The feature was not observed in any of the cases of co-occurrence (n=3). These results are consistent with expectation though the results are biased as epiphyses were rarely recovered for the juveniles of Saint-Amé. If recovery of epiphyses was good, a Wimberger's may be present as Bromer and Harvey (1948) observed the feature is some of their cases. Reasoning for the feature's presence will follow that of the white line of Fraenkel.

Building from the common observation of the Fraenkel white line, fractures typically occur in the scorbutic lattice in longstanding scurvy. Fractures develop because the scorbutic lattice is very brittle, in conjunction with significant bone loss weakens the metaphysis (Park et al. 1935). As the white line of Fraenkel was present, fractures were expected. Pelkan spurs and metaphyseal fractures (42% and 33% prevalence respectively) were common features in the cases of co-occurrence but are, as expected, less prevalent than in cases of singular disease. If rickets dominated, it could, inhibit the development of the scorbutic lattice and scorbutic fractures (Follis et al. 1940). Rickets can produce fractures but they are due to insufficiency of bone, indicating the disease was severe and/or longstanding. Corner signs remain rare, reflecting the range of fracture expression. Paleopathologically, the spurs formed in scurvy can appear similar to the flaring of limbs in rickets. Bromer and Harvey (1948) agree the features can be confused and suggest care should be taken. Some confusion when scoring the feature could artificially inflate the score value.

Loss of bone, resulting in osteopenia and cortical thinning are features of both scurvy and rickets. Bone loss is unlikely to be greater due to co-occurrence as the loss is from continued normal bone remodelling processes. Fouron and Chichoine (1962), Evans (1945) and Bromer and Harvey (1948) observed that the loss of bone was of a rickets type (moth-eaten) rather than of a scurvy type (ground-glass) in cases of co-occurrence. Unfortunately, a particular type could not be determined in the radiographs of the juveniles. As expected, most of the juveniles affected by co-occurrence were affected by osteopenia (92% generalised, 75% in epiphyses, and 83% in metaphyses and growth plate). The scurvy line, together with the white line of Fraenkel, is highly suggestive of scurvy. The feature was observed in 83% of co-occurrence cases, the majority in conjunction with the white line of Fraenkel. These results further support the dominance of scurvy in most co-occurrence cases from Saint-Amé. Trabecular coarsening was observed in fewer individuals with 58% prevalence. The result differs from Fouron and Chichoine (1940) whereby all cases had trabecular coarsening. Although trabecular coarsening was less common in the cases from Saint-Amé, it is not necessarily indicative of which disease process was dominant as the feature is produced by bone loss.

7.4.3. Scanning Electron Microscopy

Follis et al. (1940) have investigated, and Bromer and Harvey (1948) have summarised histological findings used to identify cases of scurvy and rickets co-occurrence. The authors do not employ scanning electron microscopy (SEM) or backscatter imaging. Insight into possible expected appearance of bone can still be drawn from comments made of histological thin sections.

Changes to the microscopic structure of bone occur early in the disease courses of rickets (Follis et al. 1940) and microscopic features are some of the first to develop in scurvy but a lapse of time is required for definitive features to appear (Park et al. 1935; Follis et al. 1940). Disease order also factors into the development of pathological features. Follis et al. (1940) suggest when rickets dominates it would limit or halt (if severe) the development of the scorbutic lattice (ossified cartilage). In turn, the failure of the scorbutic lattice to develop inhibits other scurvy features including characteristic fractures in the metaphysis. Important microscopic features can be reviewed in Chapter 2, Section 2.7. The major microscopic feature of rickets is increased bands of osteoid matrix. If scurvy occurs first, Follis et al. (1940) suggest it can inhibit the development of thickened osteoid bands due to a reduction in osteoblast activity. Though, Follis et al. (1940) found that in some cases of co-occurrence, even with severe scurvy, well developed osteoid was present. Therefore, we should expect that the features of rickets could be present but in many cases they may be masked. Osteopenia is found in both conditions as the result of continued resorption and is observable in cases of co-occurrence. Histomorphometric analysis is required to confirm or deny the presence of bone loss.

Transposing the clinical information for diagnosis use in paleopathology is particularly challenging for scurvy as the majority of changes appear in soft tissue which does not preserved archaeologically. Cases of scurvy will be missed in paleopathology if they are in the early stage of feature development or being masked by rickets. As the scorbutic lattice is composed of ossified cartilage, the feature could potentially be seen in paleopathology. Further investigation on the scorbutic lattice's utility would be interesting. The scorbutic lattice may assist in identifying co-occurrence in some cases (Follis et al. 1940). Non-diagnostic scurvy features of bone loss and micro-fractures could also be observed but this evidence will not significantly increase confidence for a diagnosis of scurvy or co-occurrence. Microscopy is a destructive technique and scorbutic changes are significant in the metaphyses of long bones or sternal ends of the ribs. Destruction of these elements will limit future research on the individual sampled. The use of microscopy to diagnose scurvy in paleopathology is currently not recommended as it will unlikely provide significant information. Further research may change this view. In contrast, the results of the current study demonstrate that microscopic investigation to identify rickets is successful and that microscopic features of rickets can still be observed in co-occurrence cases. An example is that of individual 647. The juvenile did not present with clear macroscopic or radiographic features of rickets but buried osteoid was observed using SEM, suggesting that a previous episode of disruption to the mineralisation process occurred, possibly rickets.

The results of the current project can be reviewed in Chapter 6, Table 6.9. Some of the results were particularly interesting. Singular rickets, specifically those diagnosed as probable cases, two thirds of the individuals exhibited remnant buried osteoid, considered by Frost (1962) to be rare in nondiseased individuals of all ages. The same feature was observed in only a third of co-occurrence cases. To be preserved archaeologically, osteoid need to become buried within the cortex or trabecular bone, requiring some new bone (well or poorly mineralised) to form beyond the osteoid. Scurvy could interfere with the development of this feature and potentially explains the low prevalence in cases of co-occurrence. However, the explanation does not explain why the feature was unobserved in possible cases. Small sample sizes may be the issue; for example only three individuals studied using SEM were classified as possible cases. The current study used rib shaft fragments slightly down the shaft from the sternal end. The children classified as co-occurrence ranged in age. Cases where the feature was unobserved may be experiencing active disease while those with those with the feature previously experienced an episode of disease or are affected by recurrent disease. The diagnosis methodology also classified buried osteoid as a probable feature which increased the chance the individual was classified in the probable category rather than the possible one.

Howship's lacunae and areas of poorly mineralised bone scored around 50% prevalence. The prevalence values of features are, when recorded, lower in cases of co-occurrence

than in cases of singular disease. Interference from scurvy, recent development of rickets, and/or small sample sizes may account for the discrepancies. The presence of numerous Howship's lacunae and poorly mineralised bone are indicative of rickets rather than scurvy although scurvy does result in net bone loss over time. Howship's lacunae also occur naturally in areas undergoing remodelling (Ortner 2003:13) and the feature is a concern when working with infant and young child bone. Remodelling can remove features in locations undergoing active remodelling. Areas of poorly mineralised bone were observed around osteons but not osteocytes. Poorly mineralised bone occurs over time in cases of rickets. Some poorly mineralised bone would be expected if the disease was active as microscopic signs develop quickly at onset (Follis et al. 1940). However, the prevalence of the two features is somewhat consistent between the different diagnosis categories. An explanation is that most samples were significantly affected by internal diagenetic change which affected large sections of the bone surface studied and likely masked the presence of 'bearded' osteocytes.

7.4.4. Conclusions on Observed Features

The results of the current study begin to fill the gap in the understanding of co-occurrence expression of features with the extensive datasets on macroscopic, radiographic and microscopic features. Features of both scurvy and rickets should be expected in cases of paleopathological co-occurrence. The expression of disease observed at Saint-Amé is mostly in keeping with the one described in medical studies but additional features of rickets were observed macroscopically. Macroscopic and radiographic features of scurvy were clear in their presentation. Macroscopically the features of rickets were present but underdeveloped and were not necessarily extensive. Radiographically, most individuals had some features to suggest the presence of rickets. Although clinical authors suggest the features of rickets should be dominant in radiography, scurvy appeared to dominate in the co-occurrence cases from Saint-Amé. Additional evidence from SEM helped identify at least one additional case of co-occurrence (individual 647) but preservation was a major issue limiting the overall utility of the technique for this collection. Our understanding of the co-occurrence is limited by few published clinical notes and the small sample size of the current study. Overall, the results suggest that in most cases, scurvy was the likely dominant disease, with scurvy supervening upon rickets. The features of scurvy were clear while those of rickets, although present, were not extreme in expression and varied. The presence of rachitic features suggests rickets has some time to develop before scurvy occurred.

Unfortunately, no particular feature of scurvy or rickets can be used to identify cases of co-occurrence. Fractures on the growth plates of the sternal end of the ribs were only observed in cases of co-occurrence and one unlikely case. However, fractures can result from a variety of processes. The presence or absence of the white line of Fraenkel could provide some clues as to the dominant disease in paleopathology. If the feature is present,

rickets was not as well developed as the scurvy. The reverse is true if the feature is absent. Overall, the prevalence of recorded features was similar to those in cases of singular disease. The sample size of co-occurrence cases studied in the current project was small (n=12). As such, the study's conclusions should be considered as preliminary evidence and are by no means comprehensive on possible disease expression. It is clear from the clinical research that co-occurrence cases have some general patterns but the interplay between rickets and scurvy can result in a variable appearance and not all features occur in every individual (e.g., Cheadle 1878; Barlow 1883; Owen 1899; Bromer 1946; Lewis et al. 2006). It is interesting to note that although the diseases result in changes to the bone formation process, the theoretical interplay does not always match what is observed. For example, bending was not observed in clinical cases of scurvy and rickets co-occurrence, but was present in many juveniles in the current study. It is possible that the bending may have been too subtle to observe on clinical radiographs and some of the early studies predate the invention of radiography (e.g., Cheadle 1878; Barlow 1883). Variable severity and duration of disease must also be considered. The relationship of scurvy and rickets in cases of co-occurrence is not yet completely understood. The unavailability of large scale clinical assessments of scurvy and rickets co-occurrence hinders our ability to identify solid general trends regarding expected features. Exceptions and oddities are noted in the various case studies currently published. In this regard, it is important to score as many features as possible in the hope of better understanding disease expression in co-occurrence and observing potential trends. Further research on cases of scurvy and rickets co-occurrence in the archaeological record will help define the full range of paleopathological disease expression and the likelihood of observing particular features. If possible, it would be interesting to devise a means of scoring the expression of the features of scurvy and rickets as slight or clear to enable better discussion of disease expression and development.

Preservation was an issue which limited the ability to suggest that co-occurrence was present. Being affected by co-occurrence also result in bone changes that contribute to poor bone preservation. However, the use of multiple techniques helped to mitigate the issue. Better preservation may enable the identification of a greater number of cases, particularly when subtle features and little development of the disease condition are present. Future research into techniques used to identify of scurvy and rickets may provide better means of suggesting or confirming a diagnosis of co-occurrence when features are subtle, one of the diseases is masked by the other, or preservation is poor. For example, Koon (2012) is investigating the development of a technique to analyse bone collagen peptides which are modified when scorbutic. It is hoped the technique will identify subclinical cases of scurvy in archaeological bone.

The juveniles were also likely to have been affected by other conditions during life as they all died at a very young age. Vitamin D has a role in the immune system and deficiency is correlated with higher susceptibility to certain infectious diseases including

tuberculosis (Walker & Modlin 2009; Baeke et al. 2010). Additional diseases could impact the appearance and development of scurvy and rickets, susceptibility of acquiring scurvy and rickets, or, in some cases, produce similar features. Differential diagnoses are therefore important to briefly consider. Possible differential diagnoses include anemia, congenital syphilis, and leukemia. Anemia was previously discussed in the macroscopy section. Congenital syphilis and leukemia are diseases which could be confused with scurvy as the diseases develop similar features (Still 1935; Ortner 2003; Burgener et al. 2008; Waldron 2009). Congenital syphilis has also been documented to co-occur with rickets (Bromer & Harvey 1948). However, both syphilis and leukemia result in lytic lesions and many classic congenital syphilis features (e.g., saddle shaped nose and dental deformities) (Ortner 2003; Waldron 2009) were not observed in the juveniles from Saint-Amé. Both diseases are very deadly and without modern treatments the children would have died very rapidly, limiting the extent of scurvy and rickets bony features development.

Secondary hyperparathyroidism can develop in a child as a response to longstanding rickets (Anspach & Clifton 1939; Jaffe 1940; Soffer & Cohn 1943). The disease develops rickets-like features. For example, increased amounts of Howships's lacunae are linked to increased bone resorption observed in secondary hyperparathyroidism (Mankin 1974b). As the condition persists, some feature become extreme such as extensive bone loss, spontaneous fractures, and the bones can distort in multiple directions (Jaffe 1940; Soffer & Cohn 1943; Ortner 2003). Linear radiolucencies can develop in the in the cortical bone (Mays et al. 2007) and this feature was potentially observed in ulnae and radii of individual 264 from Saint-Amé. Macroscopically, the cortex of the individual's bones is distorted and very porous. Radiographically there is little trabecular bone and coarsening is observed. The features are consistent with rickets and secondary hyperparathyroidism. Unfortunately, juvenile 264 was not sampled for histological assessment to assess presence of secondary hyperparathyroidism. Mild cases of secondary hyperparathyroidism are likely to be difficult to differentiate from rickets.

Tuberculosis (TB) was identified with DNA from an adult male buried at Saint-Amé between the 16th and 18th centuries (Müller et al. 2014). As such, the disease could have affected the juveniles considered in this study. Skeletal features are rare in infants and children (Teo & Peh 2004; Lewis 2011). Unfortunately, the spine, a major location for TB changes, was not scored for evidence of pathological changes in the project. New bone formation along the ribs was observed but it was located on the pleural aspect and was not lytic as observed in TB (Lewis 2011). Only individual 125 has some fine porous new bone on the visceral aspect (shaft and neck) of four ribs but a few radiographed vertebrae do not show any pathological changes. No other features recorded were consistent with TB but an extensive review was not carried out. Therefore, TB cannot be ruled out as a possible infectious disease which affected the juveniles of Saint-Amé.

Many other rare conditions can also cause similar non-specific features as those observed in the juveniles of Saint-Amé but these diseases do not fit the lesion pattern observed and other skeletal features were not observed to support other diagnoses. For example, porosity and new bone formations are produced by many inflammatory and infectious conditions (e.g., infantile cortical hyperostosis also known as Caffey's disease) and neuroblastoma can cause radiolucencies similar to the scurvy line. Consultation of medical works (e.g., Burgener et al. 2008) can provide further diagnoses and Brickley and Ives (2008) provide a differential diagnosis for long bone bending deformities. The subtle changes observed in the skeletons were mild and similar to normal growth and taphonomic changes but precaution was taken in the methodology to account for these variables.

In conclusion, the majority of cases appear to be of dominant scurvy and, likely, scurvy supervening upon rickets. Rickets appeared, overall, to be slight in expression in the juveniles from Saint-Amé. Dominant scurvy and/or scurvy superimposed onto rickets may be the cause as scurvy halts or significantly reduces bone development and the development of rachitic features. Scurvy could also have supported the continued development of certain features such as osteopenia and Howship's lacunae. The results do not provide a clear pattern or list of unique co-occurrence features. Instead it suggests paleopathologist should expect a range from clear to very mild rickets with clear features of scurvy. Certain features were found to rarely occur in cases of co-occurrence, including Pelkan spurs, fraying of the growth plates on radiographs, areas of skipped mineralization, and poorly mineralised osteocytes. Variable presentation is expected and larger datasets will identify trends in feature expression.

7.5. Cultural and Social Context for Rickets and Scurvy in 16th to 18th Century French Flanders

Scurvy and rickets are rare diseases under normal and healthy circumstances. The diseases only develop in the presence of particular biocultural and social contexts. A variety of factors contribute to create favourable situations for vitamin C and D deficiencies. The factors influence the ability of individuals to access food rich in vitamin C and D, exposure to UVB rays for vitamin D production, and susceptibility to diseases affecting the digestive system's ability to absorb the vitamins.

7.5.1. Socioeconomic Standing

A critical variable which affects food access, living conditions and choices available to families of the early modern period is their socioeconomic standing. The parishioners of Saint-Amé are the locals living within the vicinity of the church. Recovery of imported and rare goods from archaeological excavations of houses in the vicinity of Saint-Amé suggests that some individuals were wealthy and of upper class (Demolon et al. 2012).

Nonetheless, little information is known about the socioeconomic standing of the familes of juveniles buried at Saint-Amé. Juveniles were recovered both in the church and in the general churchyard cemetery (Pers. Communication, Benoît Bertrand, July 2013). Extensive parish and city records confirmed that wealthier and/or well-respected members of the parish were buried within the church while those of lower social standing were buried in the churchyard (Demolon et al. 2012). Therefore the social standing of the individuals studied is likely mixed, including upper and lower social classes.

Twelve juveniles were found buried in the church signalling these individuals may be from high status families. Individuals include 18, 208, 265[267], 473, 514, 634, 647, 657, 678, 699, 784, and 835 (Pers. Communication, Benoît Bertrand, Nov 2014). Of these individuals, 647, 835, 657, and 784 are buried near an important chapel. As per the burial records, only individuals of high status (assessed by notes on employment) were buried in this location (Pers. Communication, Benoît Bertrand, Nov 2014). The remaining 36 individuals studied were recovered outside of the church but around the apse, the semi-circular domed recess at the head of the church (Pers. Communication, Benoît Bertrand, Nov 2014). Of the 27 individuals recorded as under 6 months of age, 23 were located in this area of the church. A draft of the site report suggests that the association between burial location and status may not necessarily extend to them. The infants were all very young, the location, and clustering recalls the late medieval practice of répit. The practice was a means of reassuring the family of dead un-baptised neonates. A dead neonate would be brought to a church or sanctuary for inspection of signs of life and, if observed, would be baptised posthumously (Gordon & Marshall 2000; Delattre 2008; Tzortzis & Séguy 2008). The custom is known to have been practiced in northern and western France, including Flanders (Delattre 2008), and archaeological evidence of similar burials have been found in Haut Savoie and Seine-et-Marne (e.g., Bizot & Serralongue 1988; Delattre 2008; Tzortzis & Séguy 2008). Neonates who underwent répis and baptised posthumous would be buried at the chevet in the sanctuary or church. Those who could not be baptised were not permitted to be buried on consecrated grounds.

Disease frequencies were different between those buried within the church and outside, see Table 7.1. for details. The number of individuals affected by disease is slightly higher within the church (10/12 individuals; 83% prevalence) compared to the juveniles buried outside the church (27/36 individuals; 75% prevalence). In individuals of higher status, rickets was more prevalent (9/12 cases, 75% prevalence) compared to scurvy (6/12 cases; 50% prevalence). The majority of scurvy cases were cases of co-occurrence rather than singular disease. Outside the church, a greater number of individuals were affected by scurvy (19/36 cases; 53% prevalence) than rickets (15/36 cases; 41.67%). However, the difference in number of cases between scurvy and rickets is slight and it is unclear if the difference is significant. The high prevalence of rickets in individuals of higher social standing may suggest the wealthy had particular social or cultural practices which inhibited exposure to UV light and foods rich in vitamin D. Age is an important factor. A

greater number of juveniles over the age of 1 year at death are buried within the church (5/12 individuals; 42%) than outside (8/35 individuals; 23%). Cases of singular scurvy and co-occurrence typically affect children under the age of 1 year at death while singular cases of rickets typically affect juveniles older than 6 months at death.

TABLE 7.1. Comparison between burial location and final diagnosis given to the juveniles studied. TLI = Too little information.

Burial Location	Scurvy[1]	Rickets[1]	Co-occurrence	Unlikely or TLI	Total individuals
Inside the church	1	4	5	2	12
Outside the church	12	8	7	9	36
Total	13	12	12	14	48

[1]Individuals classified as probable and possible scurvy or rickets are included in this count, excluding all co-occurrence cases.

7.5.2. Foetuses and Neonates, Mothers and Food Access

Roughly a third of juveniles (n=17) were aged with bioarchaeological techniques to be around fetal or recently neonatal. The current study found that 72% of suggested scurvy cases were juveniles less than six months of age (n=18), with a few above in age. Prior to birth, vitamin C and D are transmitted to the infant via the placenta. After birth, vitamin C is obtained from breast milk but infantile stores of vitamin C, build prior to birth, can last up to 5 months post-partum (Ingalls et al. 1938; Woodruff 1956). Therefore, any evidence of vitamin C deficiency in the offspring under 6 months of age likely indicates that the mother was deficient (e.g. Hess 1920; Jackson & Park 1935; Hirsch et al. 1976; Bhat & Srinivasan 1989; Shore 2008). Breast milk contains some vitamin D but quantities are inadequate. The infant typically produces the remaining required vitamin D from exposure to UV rays (Lammi-Keefe 1995; Pettifor 2003; Pettifor 2004). Evidence of prenatal vitamin D deficiency prenatally deficiency would suggest the mother was deficient but postnatal cases are likely caused by diet and cultural practices. Upon quick inspection of a few adult individuals from Saint-Amé by Dr. Blondiaux, some appeared to have features consistent with vitamin D deficiency (Pers. communication, Joel Blondiaux, July 2013) though it is unclear if the deficiency was residual from childhood or recent. This evidence may support the notion that the greater population was at risk for metabolic conditions and some of the mothers of the children studied could have been affected.

The city of Douai is situated on rich fertile land and, in normal circumstances, could have produced all required food for the city (Demolon et al. 2012). Foods consumed by the citizens appear to have been largely local in origin (Demolon et al. 2012). An archaeozoological study completed on four urban houses inhabited during the same time period as the juveniles studied from Saint-Amé found evidence for consumption of a wide variety of meats including fish from both salt water and fresh water sources (Demolon et al. 2012). The consumption of fish, particularly salt water fish, and its oils would have protected the individual from vitamin D deficiency but the amount of oil varies per

species. Only members of the elite could have purchase fresh salt water fish but lower classes did consume preserved, salted fish (Le Hamon 2008). Preserved fish known to have been sold in Douai include herring and cod (Le Hamon 2008). The above observations appear to indicate that the citizens of Douai, in normal circumstances, should have been protected from metabolic disease. However, evidence of metabolic disease is reported by Demolon et al. (2012) and by the current study. Certain situations could have reduced the availability of vitamin C and D rich food during the early modern period (circa 1500 A.D. to 1800 A.D.) in Flanders, including the extreme weather phenomenon of the Little Ice Age, multiple major famines and a few wars. These events affected the region sporadically over hundreds of years but during one of these events, childhood under-nutrition, malnutrition, and mortality would likely have increased. For very young juveniles, including the majority of the juveniles studied as for the current project, these events could have had devastating consequences on their health.

Between 1590 A.D. and 1750 A.D. an important weather phenomenon, called the Little Ice Age affected northern Europe. The Little Ice Age caused extreme weather fluctuations, including decades of severe cold weather, storms and some periods of warming (Bradley & Jones 1993; Fagan 2002; Nesje & Dahl 2003). The coldest year was 1740 with the 1690s also recording some very cold winters (Appleby 1980). On average, the seasons were much colder than today and winters may have been longer. Supporting evidence for the climate shift includes textual records, tree rings, corals, ice cores, and glacier shifts (Fagan 2002; Jones & Mann 2004). Winters and early spring were difficult times in the past as access to fresh fruits and vegetables were limited, increasing the risk of developing scurvy. However, trace amounts of vitamin C can help mitigate against severe expression of the disease (Mays 2014). Consumption of fruits and vegetables was discouraged during the medieval period but the modern period saw a rise in consumption of these foods (Le Hamon 2008). Foods available varied by social class and are reviewed by Le Hamon (2008). Important vegetables available to most people living in Douai include peas, beans, lentils, cabbage, leeks, turnips, pickles, and spinach amongst others. Common fruits include apples, pears and plums (Le Hamon 2008). Clear evidence regarding the cultivation of the potato in Flanders starts in 1735 A.D. but only became a widespread crop after the French Revolution (Le Hamon 2008). The crop was promoted as a substitute for cereals which made up the vast proportion of the diet of French citizens of the period, increasing with lower socioeconomic status (Le Hamon 2008). The potato as an important crop as it is one of few crops which can be easily stored over winter, contains a notable amount of vitamin C, and is easy to feed children. In the site report, it is suggested that the crop had not yet become a common household food item in Douai (Pers. Communication, Benoît Bertrand, July 2013). However, it is known the vegetable was first imported to Europe in 1567 (sent to Antwerp) and gained widespread popularity by mid-19th century (Vos 1992; Hawkes & Francisco-Ortega 1993).

The poor weather led to a number of harvest yield failures. Douai's main economy during the early modern period was grains, wheat and cereal production, though the sector begins to declines in the 17th century (Le Hamon 2008). Grains and wheat make up the majority of a Frenchman's diet and calorie intake of the early modern period, both in the city and countryside. Wheat was of such importance that production is well documented and the rise and fall of wheat prices were used to set the price of all other foods (Le Hamon 2008). Harvest failures would have limited access to vitamin C rich foods and increased the cost of living. The rise of living costs could have affected a family's wealth and possibly socioeconomic standing. The cost of food was already very high. Historical studies found that, in a normal year, around 72% of a wage worker's income would be used to pay for the family's food (Le Hamon 2008). The exact percentage is variable by sex, age, and profession but the lowest percentage calculated was 66% for a craftsman (weaver) (Le Hamon 2008). Clearly, a failure of wheat and grain harvests would have been devastating. Harvest yield failures are most devastating the following year as this is when the produce would have been consumed (Appleby 1980; Le Hamon 2008; Demolon et al. 2012). Harvest failures would result in major price increase for basic food items, including wheat. Douai, specifically, is known to have been affected by at least eight famines, occurring in 1625-1626, 1640-1644, 1648-1652, 1661-1662, 1692-1694, 1710-1712, 1741-1742, and 1763. Supporting evidence includes high numbers of deaths were recorded for both Saint-Amé and Saint-Jacques parishes in Douai in years subsequent to harvest failures (Demolon et al. 2012). Other recorded famines in France include 1590s and 1630-1631 (Briggs 1977; Appleby 1980) but it is unclear if the events affected the Northern region. Earlier recorded poor harvests includes 1565 (very cold winter 1564-1565), 1583 to 1586, 1590s (Limm 1989; Parker 2004). A particularly high death toll was recorded for 1586 in the Netherlands as a blockage against Baltic grain was imposed by the Dutch until September 1588 (Parker 2004).

Past famines investigated for paleopathological evidence of scurvy include Geber and Murphy's (2012) mid-19th century poor Irish workhouse study and Pētersone-Gordina et al.'s (2013) 17-18th century wealthy German community study. The socioeconomic standing of the individuals from the studies are opposite with very low status in Ireland and high status in Germany. The context of Saint-Amé is likely to have been similar to the German community but the results of the respective studies are not the same. The results from Saint-Amé have greater similarities to the poor Irish study. The overall prevalence of scurvy was very high in both the Irish community (52% overall) and Saint-Amé (52% overall) while only a few cases were reported in the German community. This indicates that the children of Saint-Amé were affected by the disease and their diets were quite deficient. Similar prevalence rates of scurvy in the current study and Geber and Murphy's (2012) could signal that at Saint-Amé, at least some vitamin C was being ingested from food or transferred from the mother to the child through the placenta or breast milk. One slight difference is that the lowest prevalences were found in the neonates (28%) and infants (32%), defined as less than 12 months of age in the Irish

study (Geber & Murphy 2012) while at Saint-Amé it is these age groups which have the greatest number of cases. A sample bias may be distorting the results of the current study as many neonates and young infants but few older children were studied, see Table 6.1. Under aging of the children of Saint-Amé is a possibility as both vitamin C and vitamin D deficiencies reduce growth and could account for the low age (less than one year of age) of most suggested scurvy and rickets cases. For example, as a consequence of repeated affliction of scurvy and rickets, Chick (1976) observed significant growth retardation in her patients. The juveniles from Saint-Amé could have been affected by a similar issue. Similar to the Irish study, the German study reports that only one infant under six months of age is considered a possible case of scurvy while the rest are over one year of age at death. Geber and Murphy (2012) explain the low prevalence in neonates and infants as a result of the osteological paradox. Most of the individuals recovered from excavation are likely to have died amidst the worst part of the long-term famine (Geber & Murphy 2012). In contrast, the German community would have felt the effects of famine and war on an inconsistent basis, similar to Douai. Historical sources about Douai indicate famines lasted at most for three years though effects could have been felt over longer periods if recovery was slow. The type of lesions can also provide clues. In all cases of scurvy at Saint-Amé, evidence of active (porosity) and possible healing (new bone formation) lesions of scurvy are present. The combination of active and possible healing lesions suggests that the community members still had access to some trace amounts of vitamin C (Mays 2014). Similarly, the diets were very limited and inadequate in the Irish population but individuals still consumed some vegetables and milk, providing them with trace amounts of vitamin C which would have allowed the development of new bone (Geber & Murphy 2012). Le Hamon's (2008) review of common foods available to citizens of Douai suggests a variety of foods were available to the citizens of Douai, which, even during a famine, access to some of these foods was likely possible. However, emphasis is placed on the calorie importance of bread and wheat to the diet of the average citizen and high cost of meat. Potatoes and rice are substitutes mentioned for wheat during shortages (Le Hamon 2008).

War can also lead to famine or reduced access to vitamin C and D rich foods through its many indirect effects such as reduction in trade, increased taxation; movement of soldiers and retinue which can cause property damages and has high food requirements (Limm 1989; Parker 2004; Le Hamon 2008), spread of disease, and breakdown of social systems, among many others. These effects limit access to fresh foods, increase the risk of scurvy, and facilitate the spread of infectious diseases which can inhibit proper body functioning. Cases of scurvy and rickets are documented to occur in children who survive war. For example, Chick and Dalyell (1921) studied many Viennese infants and children whom survived World War I and the aftermath. The researchers describe nine observed cases of rickets. Eight of the nine cases were also noted to have had developed scurvy, either previously, currently or intermittently. Clinically, cases of vitamin C and D deficiencies are unexpected in such a young age group as those observed in the Saint-Amé sample, see

Chapter 2, Chapter 3, and the first few paragraphs of the current section for details. However, the extent the citizens of Douai were affected by the war remains unclear. Le Hamon (2008:14) seems to suggest the war did have a significant impact on the city. The territory of and neighbouring the French Flanders were engaged in multiple wars (e.g., Dutch revolution, War of Devolution, Thirty Year's War, Nine Years' War) during the early modern period. Douai is known to have been directly affected in some of the conflicts. In 1578, Douai and Arras were seized by revolutionaries and were quickly retaken by Spanish militia (Parker 1990; Tracy 2008). In 1667, Douai was sieged by the French (Ostwald 2007). Lastly, as part of the Spanish Succession War, Douai was besieged for seven weeks (52 days) in the campaign of 1710 and was later reclaimed by the French during a 25 days siege in 1712 (Ostwald 2007). Details regarding the extent the city and its citizens were affected on a personal level by the ongoing wars are not forthcoming. Nonetheless, the region appears to have been unstable. Evidence of louse borne typhus (Spanish strain) and *B. quintana* (trench fever) was recently identified from a mass burial from Douai of soldiers dating between 1710 A.D. and 1712 A.D. (Nguyen-Hieu et al. 2010).

7.5.3. Infants and Breast Milk

Vitamin C and D stores protect the juvenile for a number of months postnatal, after which it must receive vitamin C and D from other sources, see Chapter 2, Chapter 3, and Chapter 7, Section 7.5.2. for details. Typically, neonates and infants are breast fed and this was the main food source for infants of all social classes from Saint-Amé. Human breast milk contains all required vitamin C, protecting the child from scurvy, but only some vitamin D (Picciano 2001).

The length of breastfeeding is critical as it provides additional protection and nutrients to infants. From the 16th century to mid-18th century, most French and English infants were weaned between one and two years, to a maximum of three years (Flandrin 1976; Fildes 1982; Senoir 1983; Lebrun 1986). Douai had multiple orphanages from which the children buried at Saint-Amé may have come. Senoir (1983) notes that orphanages in Paris often struggled to find wet nurses for all their children due to lack of money. Infants who could not be sent out to nurse remained in the orphanage, shared few nurses with many other infants and often died (66-90% mortality). The length of placement with a nurse varied, but Senoir (1983) mentions it was typically for five years and the children were weaned by 1 year (Senoir 1983). Infants could have been at risk for vitamin C deficiency if weaning foods poor in vitamin C were used. Isotope studies to assess age of weaning have been considered but not completed on this collection. Extended breast feeding, over six months, requires additional supplementation as the child's nutritional need increase with grow (Dewey et al. 1984; Institute of Medicine 2000). For example, at first the milk content is enough, providing 93% energy intake but by seventh months, the percentage falls to 50% (Dewey et al. 1984). Vitamin C content of milk is known to

decrease with extended lactation (Karra et al. 1986) but whether or not it decreases to levels considered inadequate without additional supplementation is not confirmed. Values for milk produced at 10 days post-partum was measured as 6mg while mature milk fell to 4mg as measured by Saxholt et al. (2009). Vitamin D content of mature milk was also measured as 0.04µg (Saxholt et al. 2009).

The high prevalence of scurvy amongst the youngest individuals aged foetal to neonatal from Saint-Amé might suggest that they were not breast fed or breast milk was inadequate in vitamin C content. Alternative foods to breast milk were available to mothers and would have been important in many circumstances, such as where breast feeding was difficult, milk production stopped (Brickley & Ives 2008), or a wet nurse could not be found. The foods could were also used for weaning. The foods include animal milk (cow, goat), pap (called *bouillie*) made of boiled animal milk or water, flour and sometimes sweetened with sugar, or panado (*panade)* consisting of bread and milk (Drake 1930; Senoir 1983). Pap could be given as a supplementary food between breast feeding sessions or used for dry nursing (Drake 1930) while both panado and pap were also used as weaning foods. Animal milk has inadequate content of vitamin C to meet a child's needs which increases the child's risk of developing scurvy, while wheat contains neither vitamin. Boiling the milk can also destroy some of the vitamin C content, if boiled for long periods of time.

At the time, breast feeding was also not usually performed by mothers of urban upper and middle classes, ranging from aristocracy to merchants employed had the means to employ wet nurses (Senoir 1983; Fildes 1995). Families of lower social standing likely nursed their own children due to lack of funds to pay for a nurse. In fact, wet nurses were usually of lower social standing, cared for multiple children at once, and were felt by some authors be malnourished and unhygienic but this was challenged by contemporaries as urban environments were also very unhygienic (Senoir 1983). Parisian records note that upper class families could pay for a nurse to live in-house while the majority of middle and some of the lower classes had to send their infants away to nurses in the countryside, sometimes at a great distance from the family home (Senoir 1983). In Douai's orphanages, wet nurses were employed from the surrounding countryside (Le Hamon 2008). Sending a child away significantly increased their risk of death. In Douai, the mortality rate of orphan children sent to nurse fluctuated between 40-50% (Le Hamon 2008). Exact reasons for the documented high mortality are debated. In England, nursed children were usually buried in the local cemetery where they nursed, not necessarily returned to their parents (Fildes 1988). The customs practiced in Douai is unclear but it is possible the infants were returned if the distance was not great. The use of wet nurses was regulated in France with multiple employment bureaus operating in Paris by 1715 but control of the service was challenging as the nurses often lived far away and the parents did not necessarily meet the nurse or visit the home (Fildes 1995). Parents relied on reports, providing good supplies and salary to the wet nurse (Senoir 1983). At Saint-Amé,

prevalence of scurvy is roughly the same based on socioeconomic standing (50% individuals in church, 53% individuals outside the church) but the number of singular cases of scurvy are different. Scurvy as a singular disease was uncommon in individuals of high socioeconomic status while quite prevalent in individuals of lower socioeconomic standing, see Table 7.1. for details. The risk of acquiring scurvy increases if the breast milk was deficient in vitamin C but the source of milk, the wet nurse, would have fed both the upper class and lower class children. It is possible that the nurse's own child may have received a lesser amount of milk or given alternative foods. Additionally, length of breast milk production can impact its nutritional value, see above paragraph for details. Vitamin D could be acquired from other environmental sources and its levels would not be so critical. Although rare, in some cases elderly women and pregnant women were able to foster infants and children for wages. Breastfeeding was not an option to these caretakers, therefore animal milk and other foods substitutes were provided which are vitamin C and D deficient (Senoir 1983:372). Partial feeding or dry nursing were other feeding options sometimes used with children sent away to nurse. Age is an important factor when considering the distribution of scurvy in the sample. A greater number of infants are affected by singular scurvy and co-occurrence under the age of one than above one year. These results reflect a poor postnatal diet, supporting the suggestion the children studied were fed alternative diets to breastfeeding as discussed above.

Another customary practice throughout France in the 18th century to not breastfeed a newborn for the first few days and withhold the colostrum (first milk post-natal) as it was believed to be impure (Wilkes 1935; Fildes 1980; Senoir 1983). The custom may have stressed a child's body but the vitamin stores should protect the infant unless they were inadequate. However, colostrum contains IgA (intestinal antibody) which are not produced independently in the gut of the child for six weeks postpartum and therefore the gut is at greater susceptibility to intestinal disease (Lilius & Marnila 2001). The practice may account for the unusually prevalence of childhood diarrhoea recorded in Upper Brittany, France towards the end of the 18th century. However, in other locations, including England, diarrhoeal disease and infant mortality was on a decline (Appleby 1980). Children are often affected by diarrhoeal diseases but the episodes are short. Diarrhoeal disease can significantly increase the risk of developing deficiencies if the episodes are recurrent and long lasting as diarrhea inhibits proper gut absorption of vitamin C, calcium, iron, dietary vitamin D, and other essential food matter.

7.5.4. Infants, Children and Vitamin D Sources

Giuffra et al. (2013) reported cases of rickets in juveniles under the age of two years which corresponds to the accepted weaning time during the Renaissance in Italy. Paleopathological investigations revealed the infants were all affected by active disease, except for one. The authors suggest other factors were involved outside of diet, including clothing, architecture, seasonal shifts in habits, deficiencies in the mothers and repeated

pregnancies with little time between for full recovery for the mother's vitamin stores. Similar factors are likely at play with the juveniles of Saint-Amé. After weaning, around two years of age, the number of scurvy cases decreases significantly at Saint-Amé while a few cases of rickets remained. These results indicate that after weaning, vitamin C deficiency was not a significant issue and some vitamin C could be obtained. Any issues acquiring vitamin C would have been similar to that of adults, which can be reviewed for the location and time period in section 7.5.2. Vitamin D still remained an issue but the sample size in age categories above two years is very small thus results may be biased. Calcium deficiencies can result in the development of rickets (Mankin 1974a; Marie et al. 1982; Pitt 2002) however, as previously discussed in Section 7.5.2., alternative foods include milk which is high in calcium. Particular practices of the early modern period would have increased the risk of vitamin D deficiency.

Clothing has a significant impact on ability for UV rays to reach the skin. Western Europe, through to the end of the 18th century, practiced swaddling, the act of wrapping an infant very tightly in cloth (Senoir 1983). The practice limits skin exposure and could have put the infants at risk of vitamin D deficiency. Details of clothing worn by older children who were weaned were not found but genre paintings of the period from Dutch masters depict everyday life of peasants and the middle class. The evidence is biased towards children of higher socioeconomic status whose parents could commission paintings and the children may have worn special clothes rather than every day wear Romanticism and ideologies may also be reflected in the paintings but they can provide some ideas about possible clothing worn by children. The paintings show children dressed in big dresses with long sleeves, coats, pants, stockings and caps of cloth on girls and infants. Nearly their entire bodies are covered. The ideas would certainly fit the 12 juveniles, which were buried within the church of Saint-Amé, see Section 7.5.1. A very high prevalence, around 75%, of rickets in children of high socioeconomic status was found and could be explained by these cultural practices.

The extreme cold of certain winters would have likely kept the children indoors for longer periods of time, reducing their vitamin D levels. Details on social expectation of upper class children and those fostered by wet nurses in the country is not discussed in detail. However, if the child was sickly from disease, such as diarrhoeal disease or scurvy, they would likely have been kept inside. If the length of illness was extensive, vitamin D stores of the infant or child could be drained. This hypothesis was suggested by Ortner and Mays (1998) for the skeleton of juveniles from Wharram Percy. In that collection, the juveniles displayed severe active vitamin D deficiency with significant bending, abnormal porosity, active changes at the growth plates, and no evidence of healing, characterised as porous new bone forming in the concavities of abnormal curvatures and remnants of vitamin D deficiency in adult skeletons. Saint-Amé is similar to Wharram Percy in that many of the juveniles displayed features associated with active vitamin D deficiency but the disease was not severe, some evidence of healing rickets was observed, and there may

have been evidence of vitamin D deficiency in the adult skeletons (Pers., Joel Blondiaux, July 2013). Records of juveniles sent out to be nursed suffered high mortality and, if the children survived, they often returned sickly and with deformities (rickets?) (Senoir 1983; Le Hamon 2008). In this scenario, the infant would have contracted vitamin D deficiency before its second year which is in line with the current findings.

Once returned home, urban living could have restricted children to the indoors and/or reduced light access due to crowding, though little information could be found on the topic. Flanders was an early region to develop industrialisation, particularly in the textile trade (wool and cloth) (Clark 1957; Peters 1985). Being kept indoors to work in trade would impact vitamin D levels for both mother and child. Urban living and introduction to novel environments puts children, particularly to those which nursed in the countryside, in contact with a host of new bacteria and infectious diseases. The diseases could cause intestinal problems, reducing the ability to absorb the vitamins, as well as leave a child bedridden, reducing UV light exposure. Other urban 19th century studies with paleopathological cases of rickets include Ellis (2010) and Mays et al. (2006). High rates of vitamin D deficiency were observed (over 34%) by Ellis, the current study at 46% prevalence and Mays et al. (2006) at roughly 13%. Mays et al (2006) observed features of both active and healing rickets, suggesting recurrent disease episodes. The results would be comparable to the findings in the current study whereby the features are expressed but not severe and individuals are affected into older age categories such as 3 to 5 years of age, similar to the Mays et al. (2006) findings.

7.6. Conclusions

Cases of co-occurrence can be identified in the paleopathological record. Twelve cases were found at the site of Saint-Amé. Based on the observed features, it would appear the individuals were most consistent with scurvy supervening upon rickets but the reverse cannot be excluded. The use of multiple techniques was critical to the identification of co-occurrence and is recommended for future research. Features of scurvy tend to be clear, although the features are non-diagnostic. Rickets can be well developed but this is uncommon. Very subtle bending, flaring, porosity and growth plate changes should be observed. Further comprehensive clinical and paleopathological research will be needed to strengthen or refute the observed results. Many other diseases potentially affected the juveniles but rickets and scurvy are most likely. Additional contextual evidence on child rearing practices and environment, both physical and social, provide evidence suggesting access to vitamin C and D was reduced between 16th and 18th century French Flanders and could account for the observed scurvy and rickets co-occurrence cases. Factors included the Little Ice age, famines, wars, infant feeding practices, and practices limiting UV ray exposure, including swaddling and clothing. Significant limitations did reduce the probability of identification, including preservation (at least half the skeleton is required with little taphonomic change), disease sequence and interaction. As a result, cases of

clear scurvy and rickets are those which will likely be identified archaeologically, missing those whereby one disease is masked. Nonetheless, cases of co-occurrence can be identified archaeologically and contribute a more nuanced understanding of disease in the past.

Chapter 8: Conclusion

The co-occurrence of disease has long been observed in the medical field and remains a serious problem today. Disease weakens the body inhibiting the optimal function of its various systems, including its ability to ward off diseases and acquire essential nutrients, increasing the risk of developing a second disease. The presence of multiple diseases significantly weakens our bodies and increases the risk of death, particularly in the past.

Paleopathology has long focused on singular disease identification and definition within an archaeological point of view. Some paleopathological research has begun to investigate co-occurrence but not yet in a systematic way. DNA research is the only exception (e.g., Devault et al. 2014). The current project is unique as it considers the possible co-occurrence of disease rather than a single pathology in skeletal paleopathology and produced a systematic investigation.

The current research attempted to investigate the clinically well documented co-occurrence of two metabolic diseases, scurvy and rickets. The project had two main questions. First, is it possible to identify cases of co-occurrence, specifically scurvy and rickets, using skeletal data? Second, if it is possible to identify cases, how might the disease present itself and are any of the typical paleopathological scorbutic and rachitic features used to identify the diseases different in expression? For example, are any of the features present, absent or less severe in expression than we might otherwise expect. To answer the questions, a collection of juvenile remains with previously reported cases of scurvy and rickets was identified as a collection with potential cases of disease co-occurrence. The juveniles (n=48, foetal to five years of age) from Saint-Amé, Douai, France, dating from the early modern period (circa 1500 A.D. to circa 1800 A.D.) were selected for further study. Multiple techniques were applied to the collection to increase the chance of identifying co-occurrence. The techniques included macroscopy, radiography, and microscopy (histology and scanning electron microscopy). Each of the techniques used in this study proved effective in producing evidence to help identify potential cases of co-occurrence. The only exception was the use of Villanueva and Goldner's Trichrome bone histological stains. The stains are used clinically to observe cellular and osteoid changes which occur when an individual is vitamin D deficient. The results were inconclusive as no osteoid was found to be preserved in the bones samples taken from Saint-Amé. Nonetheless, the use of multiple techniques is vital to enable the documentation of as much evidence as possible to support a potential diagnosis. The current methodology is recommended for future research in scurvy and rickets co-occurrence

The results of the study were able to suggest the presence of at least 12 potential cases of scurvy and rickets co-occurrence. The ability to diagnose co-occurrence is limited by reliance on observing skeletal features of both diseases. In a clinical setting, co-

occurrence can sometimes be diagnosed in the absence of clear skeletal features due to resources such as serum tests or declaration of the secondary disease during treatment. Unfortunately, in archaeological skeletons, it is unlikely we will be able to identify cases of co-occurrence where the secondary disease is masked as there will not be enough evidence to suggest the presence of the secondary disease with any confidence. Therefore, many cases of co-occurrence are likely to be missed in paleopathology. The development of new technologies which rely on other materials than bone, such as collagen (e.g., Koon 2012) may provide additional independent evidence, much like the serum tests in clinical settings. DNA evidence is another such technique which has previously identified co-occurrence of infectious disease where skeletal evidence was missing (e.g., Bianucci et al. 2012).

The appearance and confidence in scurvy and rickets diagnosis was variable in the current study but overall, the results of the different techniques are consistent with the medical literature. Most cases also appear to have been of dominant scurvy, likely scurvy supervening upon rickets but the reverse cannot be excluded. On a macroscopic level of analysis, features of both scurvy and rickets should be expected, however the features of rickets may be subtle in expression. Fractures on the sternal rib end growth plate occurred in very high prevalence in co-occurrence cases. Further research on the utility of this feature and its relation to co-occurrence is important. In radiography, a mixture of scurvy and rickets features should be expected though the features of rickets have little diagnostic power. In the current research, the white line of Fraenkel was useful in suggesting a possible dominant disease process. When formed, it suggested that scurvy was dominant and vice-versa if it failed to form. Although osteopenia and cortical thinning are common features to both diseases, the expression of these features should be comparable to a case of singular disease. Microscopic investigations with the SEM were successful in providing additional evidence to support a diagnosis of rickets when the samples were preserved and not destroyed by diagenetic processes. Additional biocultural and socioeconomic information was able to support the presence of scurvy and rickets in Douai during the early modern period. Further information (e.g., isotope data) on weaning practices amongst the children affected by co-occurrence would be interesting. Based on the evidence of co-occurrence in some infants, it is unlikely that they would have been breast fed for long periods after birth which is contradictory to the norms of society at the time. The presence of co-occurrence could also support the notion that during some periods, Douai and its citizens were significantly affected by terrible circumstances which limited access to vitamin C rich foods and vitamin D. Circumstances including climatic fluctuations, famines, and war.

The use of both macroscopy and radiography are highly recommended for future research on scurvy and rickets as the results from these techniques provided the foundation for suggesting disease presence and co-occurrence. Histology is recommended for situations, such as the study of co-occurrence, where histological information can provide critical

additional information and features of high diagnostic strength to support a diagnosis of rickets. Future research on co-occurrence of disease is strongly encouraged. Each disease combination will have its own unique expression therefore creating standards is challenging but will provide interesting new and realistic ideas about the past.

Numerous limitations affected the ability to correctly identify features of scurvy and rickets. First, the research was conducted on very young juveniles. Differentiating pathology from normal bone was challenging as young juvenile bones undergo massive amounts of growth and development. Second, scurvy and rickets co-occurrence is documented in the clinical literature but there are not many reports published on the phenomenon and few large scale research studies. The limited number of clinical resources on the potential appearance of disease whether clinical, radiographic or histological means the clinical patterns are not extensively documented and many exceptions were noted. The limited resources reduces certainty of the interaction boundaries between the two diseases. This issue will not be relevant or encountered by all types of disease co-occurrence and is specific to scurvy and rickets as both diseases are highly treatable. Third, scurvy and rickets are diseases with mostly non-diagnostic skeletal features which require significant amount of evidence throughout the skeleton to suggest diagnosis. Greater innovations in metabolic disease scoring to quantify or qualify slight change and extreme change will help provide a greater nuanced view of the features and should help identify potential cases of co-occurrence. Also, developing a qualitative or quantitative way of measuring metaphyseal porosity, a scurvy feature, would be of great help as the current suggestion for what is pathological is non-specific to bone size and age. Fourth, preservation of bones, including completeness of the skeleton and amount of taphonomic change, as well as diagenesis of bone was a limiting factor to the current study. If preservation was excellent, very subtle cases of co-occurrence may be recorded. Developing methods to assess subtleties and variations would be very valuable for co-occurrence research. Collections with good to moderate preservation are therefore recommended for co-occurrence study as there is greater material and increased chance of identifying features associated with multiple diseases.

In summary, the investigation of disease co-occurrence is interesting, challenging, and promotes a realistic understanding of disease. The study demonstrates that the suggestion of disease co-occurrence is possible in paleopathology; twelve cases of scurvy and rickets co-occurrence were identified from the sample of juveniles from Saint-Amé. The expression of disease, overall, was consistent with clinical documentation; dominant scurvy with subtle rickets was observed. Further research on disease co-occurrence of any type is highly encouraged to further our understanding of disease and to create and promote a realistic understanding of disease in the past.

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Appendices
Appendix I: Age at Death Estimation

This appendix contains the results of age at death for each technique described in Chapter 5. Section A contains all the results for dental age estimation. Section B contains the results for long bone length estimates.

A - Dental Age Estimation

*TABLE I-1. Gustafson & Koch (1974). Results for deciduous dentition, part 1: maxillary. P = prenatal, m = months, * = assessed by radiography, - = could not assess.*

Juve-nile	Final range for whole dentition	1st Incisor	2nd Incisor	Canine	1st Molar	2nd Molar
S6	**5m P to 2m**	5m P to 2m	-	-	-	-
S7	**6m P to 2m**	5m P to 2m	-	6m P to 9m	-	-
S18	**3y to 6.25y**	-	-	-	-	-
S37	**2m-4m, under 5m**	2m to 4m *	2.5 to 4.5m *	After 6m P to 9m	After 5m P to 5.5m	After 6m P to 10m
S56	**1y, under 1.5y**	6m to 11m	6m to 1y	9m to 1y	-	-
S66	**4m to 5.5m**	-	-	-	-	-
S74	**-**	-	-	-	-	-
S91	**-**	-	-	-	-	-
S95	**5m P to 2m**	5m P to 2m	5m P to 2y	-	5m P to 6m	-
S100	**-**	-	-	-	-	-
S110	**4m to 7m**	5m P to 4m	-	-	5m P to 5.5m	6m P to 10m
S111	**3.5-5y, likely 4y**	After 2y	After 2y	After 3.5y	After 2.5y *	-
S124	**3-4m, under 5m**	2-4m	-	6m P to 9m	5m P to 5.5m	6m P to 10m
S125	**4m-5m**	4m to 6m	2-4.5m to 6m	-	6m P to 5m	-
S153	**2m, under 5m**	2-4m	5m P to 2m	-	6m P to 5m	-
S175	**6m P to 2m**	5m P to 2m	-	-	-	6m P to 10m
S190	**-**	-	-	-	-	-
S191	**-**	-	-	-	-	-
S208	**5.5 m P to Birth**	4m P to 2m	-	6m P to 9m	-	-
S218	**-**	-	-	-	-	-
S219	**-**	-	-	-	-	-
S220	**-**	-	-	-	-	-
S221	**-**	-	-	-	-	-
S231	**2-4m, under 5m**	-	2-4.5m	6m P to 9m *	-	6m P to 10m
S232	**5 to 7m**	4m to 6-11m	5m to 6m-1.5y	6m P to 9m	6m P to 6-7m	6m P to 10m
S264	**2-4m, under 5m**	2m to 4m	2m to 5m	-	-	-

S265	**-**	-	-	-	-	-
S270	**4 to 7.5m**	>4m to 11m	4-6m	-	6m P to 6-7m	-
S272	**6m P to 2m**	4m P to 2m	4m P to 2m	-	5.5m P to 5.5m	-
S273	**6m P to 2.5m**	-	5m P to 2.5m	-	-	-
S296	**-**	-	-	-	-	-
S307	**-**	-	-	-	-	-
S332	**7m to 1.5y**	-	-	-	-	-
S361	**-**	-	-	-	-	-
S367	**5.5-10m, under 1y**	-	-	-	5.5-7m to 1y	6m P to 10m
S385	**-**	-	-	-	-	-
S414	**-**	-	-	-	-	-
S450	**2.5 to 2.5y**	-	>1.5y	>2.5-3.5y	>2.5y	2.5y to 3y
S473	**2y to 3y**	>1.5	>1.5	>3-3.5y	>2y	>2.5y
S514	**2 to 4m**	5m P to 4m	5m P to 5m	6m P to 9m	5m P to 5m	5m P to 10m
S528	**6m P to 2m**	5m P to 2m	-	-	-	-
S634	**3.5y to 4.5 y**	>1.5y *	>1.5y	>2.75	>2	>2.75
S647	**6m P to 2m**	5m P to 2m	-	6m P to 9m	6m P to 5m	-
S657	**5 to 7m**	-	-	6m P to 9m*	>6m P to 5-7m *	>6m P to 10m *
S678	**4m to 7m, likely 5-7m**	4m to 6-11m	-	6m P to 9m	5-7m, <1y *	6m P to 10m
S699	**6m P-2m, under 4m**	5m P to 2m	5m P to 2-4m	-	5m P to 5.5m	6m P to 10m
S784	**4m-7m, likely 5-7m**	>4m to 11m	5m P to 7m	5m P to 7m	5m P to 7m	6m P to 10m
S835	**4y to 5y**	>2y	>2y	>3.5y	>1.5-2.5y	>3y *

*TABLE I-2. Gustafson & Koch (1974). Results for deciduous dentition, part 2: mandibular. P = prenatal, m = months, * = assessed by radiography, ** = assessed by both macroscopy and radiography, - = could not assess.*

Juvenile	1st Incisor	2nd Incisor	Canine	1st Molar	2nd Molar
S6	-	-	-	-	-
S7	5m P to 2m	5m P to 2m	6m P to 7m	-	-
S18	-	-	-	-	-
S37	2m to 4m	2m to 5m	-	5m P to 5m	6m P to 10m
S56	10m to 1.5y	7.5m to 1.5y	1-2y	1-1.5y	1y to 2y
S66	>4m	5m to 7m	6m P to 6.5m	5m P to 5.5m	6m P to 10m
S74	-	-	-	-	-
S91	-	-	-	-	-
S95	-	-	-	-	-
S100	-	-	-	-	-
S110	-	5m to 7m	6m P to 7m	5m P to 5.5m	6m to 10m
S111	>1.5y	>1.5y	>2.5y	>2.5y *	-
S124	2-4m	2-4.5m	6m P to 6.5m	5m P to 5.5m **	6m P to 10m **
S125	2-4m	2-4m to 7m	-	5m P to 5m	-
S153	5m P to 2-4m	5m P to 3m	-	6m P to 5m	6m P to 10m
S175	-	5m P to 3m	-	5m P to 5m	-
S190	-	-	-	-	-

S191	-	-	-	-	-
S208	-	-	-	5.5m P to 5.5m	-
S218	-	-	-	-	-
S219	-	-	-	-	-
S220	-	-	-	-	-
S221	-	-	-	-	-
S231	5m P to 4m	>5m P to 2.5-3m	6m P to 7m **	5m P to 5.5m	6m P to 10m
S232	4m to 10m	5m to 7m	6m P to 7-9m	5m P to 5-7m	6m P to 10m
S264	2m to 4m	3m to 5m	6m P to 7m	5m P to 5.5m	5m P to 10m
S265 (S267)	-	-	-	-	-
S270	4m to 10m	5m to 7.5m	-	5m P to 5-7m	6m P to 10m
S272	-	4m P to 2.5m	6m P to 7m	5m P to 5m	-
S273	-	4m P to 2.5m	6m P to 9m	5m P to 5m	6m P to 10m
S296	-	-	-	-	-
S307	-	-	-	-	-
S332	-	5m to 7m-1.5y	-	-	-
S361	-	-	-	-	-
S367	-	-	-	-	6m P to 10m
S385	-	-	-	-	-
S414	-	-	-	-	-
S450	>1.5y	-	2y to 3.5y	>2.5y	>3y *
S473	>1.5y	>1.5y	2.5y to 3.5y	>1.75y	2.5y to 3.5y *
S514	5m P to 2-4m	5m P to 5m	6m P to 7m	5m P to 5m	5m P to 10m
S528	-	-	6m P to 7m	5m P to 5m	-
S634	>1.5	>1.5y	1.75-3.5y	>1.75y	>2.75y
S647	-	-	-	-	-
S657	4m to 10m	5m to 7m	>6m P to 7-9m, <1y	>6m P to 5-7m, <1y	6m P to 10m
S678	-	-	-	-	-
S699	-	-	-	5.5m P to 5m	6m P to 10m
S784	4m to 10m	5m P to 7m	6m P to 7m	5m P to 7m	6m P to 10m
S835	>2y	>2y	-	>2.5y*	>3y*

*TABLE I-3. Gustafson & Koch (1974). Results for permanent dentition, part 1: maxillary. P = prenatal, m = months, * = assessed by radiography, - = could not assess.*

Juvenile	1st Incisor	2nd Incisor	Canine	1st Premolar	2nd Premolar	1st Molar	2nd Molar
S6	-	-	-	-	-	-	-
S7	-	-	-	-	-	-	-
S18	-	-	-	-	-	4.5y to 5y	-
S37	-	-	-	-	-	-	-
S56	-	-	-	-	-	1m to 2.5y	-
S66	-	-	-	-	-	-	-
S74	-	-	-	-	-	-	-
S91	-	-	-	-	-	-	-
S95	-	-	-	-	-	-	-
S100	-	-	-	-	-	-	-
S110	-	-	-	-	-	-	-
S111	4m to 4-5y *	1y to 4y *	5m to 5y *	-	-	2.5y to 4.5y *	-

S124	-	-	-	-	-	-	-
S125	-	-	-	-	-	-	-
S153	-	-	-	-	-	-	-
S175	-	-	-	-	-	-	-
S190	-	-	-	-	-	-	-
S191	-	-	-	-	-	-	-
S208	-	-	-	-	-	-	-
S218	-	-	-	-	-	-	-
S219	-	-	-	-	-	-	-
S220	-	-	-	-	-	-	-
S221	-	-	-	-	-	-	-
S231	-	-	-	-	-	-	-
S232	-	-	-	-	-	-	-
S264	-	-	-	-	-	-	-
S265 (S267)	-	-	-	-	-	-	-
S270	-	-	-	-	-	-	-
S272	-	-	-	-	-	-	-
S273	-	-	-	-	-	-	-
S296	-	-	-	-	-	-	-
S307	-	-	-	-	-	-	-
S332	-	-	-	-	-	-	-
S361	-	-	-	-	-	-	-
S367	-	-	-	-	-	-	-
S385	-	-	-	-	-	-	-
S414	-	-	-	-	-	-	-
S450	4m to 4-5y *	1y to 4y *	5m to 5.5-7y *	2y to 5y *	-	-	-
S473	4m to 4y *	-	5m to 5.5y *	1y to 5y *	-	-	-
S514	-	-	-	-	-	Birth to <2.5y	-
S528	-	-	-	-	-	-	-
S634	4m to 4-5.25y *	1y to 4-5.5y *	5m to 5.5-7y *	-	-	Birth to 2.5-4.5y	-
S647	-	-	-	-	-	-	-
S657	-	-	-	-	-	Birth to <2.5y	-
S678	-	-	-	-	-	-	-
S699	-	-	-	-	-	-	-
S784	-	-	-	-	-	-	-
S835	>4m to 4-5.25y *	>1y to 4-5.5y	>5m to 5.5-7y	>2.5y to 5-7.5y	-	Birth to 2.5-4.5y	-

*TABLE I-4. Gustafson & Koch (1974). Results for permanent dentition, part 2: mandibular.. P = prenatal, m = months, * = assessed by radiography, - = could not assess.*

Juvenile	1st Incisor	2nd Incisor	Canine	1st Premolar	2nd Premolar	1st Molar	2nd Molar
S6	-	-	-	-	-	-	-
S7	-	-	-	-	-	-	-
S18	-	-	-	-	-	-	3y to 6.25-8y
S37	-	-	-	-	-	-	-
S56	4m to 3.5y *	4m to 4y *	5m to 4.5y *	?? *	-	1m to 2.5y	-
S66	-	-	-	-	-	-	-
S74	-	-	-	-	-	-	-
S91	-	-	-	-	-	-	-
S95	-	-	-	-	-	-	-
S100	-	-	-	-	-	-	-
S110	-	-	-	-	-	-	-
S111	4m to 3.5-5y *	4m to 4-5y **	5m to 4.5-7y **	2y to 4.5y *	-	2.5y to 4y *	-
S124	-	-	-	-	-	-	-
S125	-	-	-	-	-	-	-
S153	-	-	-	-	-	-	-
S175	-	-	-	-	-	-	-
S190	-	-	-	-	-	-	-
S191	-	-	-	-	-	-	-
S208	-	-	-	-	-	-	-
S218	-	-	-	-	-	-	-
S219	-	-	-	-	-	-	-
S220	-	-	-	-	-	-	-
S221	-	-	-	-	-	-	-
S231	-	-	-	-	-	-	-
S232	-	-	-	-	-	-	-
S264	-	-	-	-	-	-	-
S265 (S267)	-	-	-	-	-	-	-
S270	-	-	-	-	-	-	-
S272	-	-	-	-	-	-	-
S273	-	-	-	-	-	-	-
S296	-	-	-	-	-	-	-
S307	-	-	-	-	-	-	-
S332	-	-	-	-	-	-	-
S361	-	-	-	-	-	-	-
S367	-	-	-	-	-	-	-
S385	-	-	-	-	-	-	-
S414	-	-	-	-	-	-	-
S450	4m to 3.5-5y *	5m to 4-5y *	5m to 4.5-7y **	2y to 4.5y **	-	2.5-6y to 5-7y **	-
S473	5m to 4.5y *	5m to 4y *	4m to 3.5y *	-	-	2.5 to 4y	-
S514	-	-	-	-	-	Birth to <2.5y	-
S528	-	-	-	-	-	-	-

S634	4m to 3.5-5y *	1y to 4-5y *	5m to 4.5-7y *	>2y *	-	2.5-4y *	-
S647	-	-	-	-	-	-	-
S657	-	-	-	-	-	-	-
S678	-	-	-	-	-	-	-
S699	-	-	-	-	-	-	-
S784	-	-	-	-	-	-	-
S835	>4m to 4-5y *	>1y to 4-5y *	>5m to 4.5-7y *	>2y to 4.5-7y *	-	>Birth to 2.5-4y *	-

B - Long Bone Length Age Estimation

TABLE I-5. Kazekas & Kósa (1978). Age estimate results, given in weeks in utero. - = could not assess. Result under 40 weeks was considered perinata. B = observed length value is greater than the longest chart value, therefore the juvenile's age estimate is postnatal.

Juvenile	Humerus	Radius	Ulna	Femur	Tibia	Fibula	Range
S6	-	-	-	38 to 40	38 to 40	38 to 40	38 to 40
S7	36 to 38	38	38	36 to 38	36 to 38	36 to 38	36 to 38
S18	-	-	-	-	-	-	-
S37	-	-	-	-	-	-	-
S56	B	-	-	-	B	-	B
S66	-	B	B	B	B	B	B
S74	-	-	-	-	36 to 38	-	36 to 38
S91	-	-	-	-	B	B	B
S95	B	B	B	B	B	B	B
S100	-	B	B	-	-	-	B
S110	-	-	B	B	B	B	B
S111	B	-	-	-	-	-	B
S124	B	B	B	-	-	-	B
S125	B	B	-	B	B	B	B
S153	B	-	38 to 40	-	-	-	38 to B
S175	B	B	-	-	-	-	B
S190	-	-	-	B	B	B	B
S191	-	-	-	B	-	-	B
S208	36	36 to 38	36 to 38	32 to 34	-	-	32 to 38
S218	B	-	B	-	-	-	B
S219	38 to 40	-	-	-	-	-	38 to 40
S220	B	-	-	-	B	B	B
S221	36 to 38	36 to 38	36 to 38	36	-	-	36 to 38
S231	-	B	-	-	-	-	B
S232	B	B	B	B	B	B	B
S264	B	B	B	-	-	-	B
S265 (S267)	-	B	B	-	-	-	B

S270	B	B	-	B	B	B	B
S272	36 to 38	38	38 to 40	-	-	-	36 to 40
S273	B	-	B	B	B	B	B
S296	-	-	-	B	B	B	B
S307	B	B	B	-	-	-	B
S332	-	-	-	-	-	-	-
S361	-	-	-	-	-	-	-
S367	-	B	B	-	-	-	B
S385	-	-	-	B	B	B	B
S414	-	B	38 to 40	-	-	-	38 to B
S450	-	-	-	-	B	-	B
S473	-	-	-	B	B	B	B
S514	B	B	B	-	-	-	B
S528	B	B	B	B	B	40	40 to B
S634	B	B	B	-	-	-	B
S647	34 to 36	34	32 to 34	34 to 36	34 to 36	34	32 to 36
S657	-	-	-	-	B	B	B
S678	B	-	B	B	-	-	B
S699	-	-	-	-	-	-	-
S784	-	-	-	-	-	-	-
S835	B	-	-	-	B	-	B

TABLE I-6. Maresh (1970) as found in Scheuer & Black (2000). Age estimate results, given in weeks in utero. - = could not assess. Result under 40 weeks was considered perinatal, and under lowest chart value. P = perinatal, y = year, m = months. Male and female scores were the same, except if noted.

Juvenile	Humerus	Radius	Ulna	Femur	Tibia	Fibula	Range
S6	-	-	-	P	P	P	P
S7	P	P	P	P	P	P	P
S18	-	-	-	-	-	-	-
S37	-	-	-	-	-	-	-
S56	1y	-	-	-	1y	-	1y
S66	-	1.5m (male) 3m (female)	3m	1.5m	3m	3m	1.5m to 3m
S74	-	-	-	-	P	-	P
S91	-	-	-	-	3m	3m	3m
S95	1.5m	1.5m	1.5m	P	1.5m	1.5m	P to 1.5m
S100	-	3m	3m	-	-	-	3m
S110	-	-	1.5m	1.5m	1.5m	1.5m	1.5m
S111	3y to 3.5y	-	-	-	-	-	3y to 3.5y
S124	1.5m	1.5m	1.5m	-	-	-	1.5m

S125	1.5m	1.5m	-	1.5m	1.5m	1.5m	1.5m
S153	P	-	P	-	-	-	P
S175	P	P	-	-	-	-	P
S190	-	-	-	1.5y	1.5y	1.5y	1.5y
S191	-	-	-	P	-	-	P
S208	P	P	P	P	-	-	P
S218	P	-	P	-	-	-	P
S219	P	-	-	-	-	-	P
S220	P	-	-	-	1.5m	1.5m	P to 1.5m
S221	P	P	P	P	-	-	P
S231	-	P (male) 1.5m (female)	-	-	-	-	P to 1.5m
S232	3m	1.5m (male) 3m (female)	1.5m (male) 3m (female)	1.5m (male) 3m (female)	3m	3m	1.5m to 3m
S264	1.5m	1.5m	1.5m	-	-	-	1.5m
S265 (S267)	-	1y	1y	-	-	-	1y
S270	3m	3m	-	3m	3m	3m	3m
S272	P	P	P	-	-	-	P
S273	1.5m	-	1.5m	1.5m	1.5m	1.5m	1.5m
S296	-	-	-	2y	2y	2y	2y
S307	1.5y	1.5y	1y (male) 1.5y (female)	-	-	-	1y to 1.5y
S332	-	-	-	-	-	-	-
S361	-	-	-	-	-	-	-
S367	-	3 to 6m (male) 6m (female)	3 to 6m (male) 6m (female)	-	-	-	3m to 6m
S385	-	-	-	6m to 1y	1y	1y	6m to 1y
S414	-	P	P	-	-	-	P
S450	-	-	-	-	1.5y	-	1.5y
S473	-	-	-	2 to 2.5y (male) 2.5y (female)	2.5y (male) 2y (female)	2.5y	2y to 2.5y
S514	P	P	P	-	-	-	P
S528	1.5m	P (male) 1.5m (female)	P	P	1.5m	P (male) 1.5m (female)	40 to B
S634	2 to 2.5y (male)	1.5y (male)	1.5y (male) 1.5y to 2y	-	-	-	1.5y to 2.5y

	2.5y (female)	2y (female)	(female)				
S647	P	P	P	P	P	P	P
S657	-	-	-	-	3m	3m (male) 3m to 6m (female)	3m to 6m
S678	3m	-	3m (male) 3m to 6m (female)	1.5 to 3m (male) 3m (female)	-	-	1.5m to 6m
S699	-	-	-	-	-	-	-
S784	-	-	-	-	-	-	-
S835	2y	-	-	-	2y	-	2y

Appendix II: Completeness and Taphonomy

This appendix contains all the preservation scores (completeness and taphonomy) for the individuals suggested as being affected by scurvy and rickets co-occurrence. Section A contains the scores for completeness. Tables 18 to 20 contain the cranial scores, Tables 21 to 22 contain axial skeletal scores, and Tables 23 to 34 contains scores for limb bones, and Table 35 has the hand and leg bones. Section B contains the taphonomy scores. Data was collected following the protocol outlined in Chapter 5, Section 5.3.b.1. Consistent data was collected for all 48 individuals recorded but remaining data was not included here as it was not found to be pertinent.

A - Completeness

TABLE II-1. Completeness scores for cranial bones, Part 1. - = not preserved, Frags = only fragments remain, NR = not recorded.

Juvenile	Occipital	Pars basilaris	Pars lateralis	Parietal	Temporal	Zygomatic	Frontal	Orbits	Nasal
6	50-75%	-	R: - L: -	R:25-50% L: -	R:0-25% L: -	R:75-100% L: -	R: - L:75-100%	R: - L:75-100%	-
7	-	75-100%	R:75-100% L:75-100%	-	R:75-100% L:25-50%	R:75-100% L:75-100%	R:0-25% L:25-50%	-	-
18	75-100%	25-50%	R:75-100% L:75-100%	R:50-75% L:0-25%	R:25-50% L:0-25%	-	-	-	-
37	50-75%	75-100%	R:75-100% L:75-100%	R:50-75% L:50-75%	R:75-100% L:75-100%	-	R:75-100% L:25-50%	R:75-100% L:0-25%	R:75-100% L:75-100%
56	50-75%	Frags	R:25-50% L:25-50%	R:50-75% L: -	R:25-50% L:25-50%	R:75-100% L: -	R:0- 25% L:0-25%	-	-
66	50-75%	-	-	R:50-75% L: -	R:25-50% L:25-50%	-	R:0-25% L: -	R:25-50% L: -	-
74	-	-	-	-	-	-	-	-	-
91	-	-	-	-	-	-	-	-	-
95	25-50%	75-100%	R:75-100% L:75-100%	R:25-50% L:25-50%	R:50-75% L:50-75%	R:75-100% L:0-25%	R:50-75% L:0-25%	R:25-50% L: -	R:75-100% L: -
100	-	-	-	-	-	-	-	-	-
110	25-50%	75-100%	R:25-50% L:50-75%	-	R:0-25% L:-	-	R:0-25% L:75-100%	R:0-25% L:25-50%	R: - L:75-100%
111	75-100%	75-100%	R:25-50% L:0-25%	R:25-50% L:25-50%	R:75-100% L:50-75%	R: - L:75-100%	R:50-75% L:0-25%	R:25-50% L:50-75%	R:50-75% L: -

124	75-100%	75-100%	R:75-100% L:75-100%	-	R:75-100% L:25-50%	R:75-100% L:75-100%	-	-	-
125	25-50%	75-100%	R:25-50% L:50-75%	R:50-75% L:0-25%	R:25-50% L:75-100%	R:75-100% L:75-100%	R:50-75% L:0-25%	R:25-50% L:25-50%	-
153	50-75%	75-100%	R:75-100% L:75-100%	R:25-50% L:25-50%	R:50-75% L:0-25%	R:75-100% L:75-100%	R:75-100% L:0-25%	R:75-100% L:25-50%	-
175	50-75%	50-75%	R:50-75% L:25-50%	-	R:50-75% L:0-25%	R:0-25% L:75-100%	R: - L:0-25%	R: - L:0-25%	-
190	-	-	-	-	-	-	-	-	-
191	-	-	-	-	-	-	-	-	-
208	0-25%	75-100%	R:75-100% L:75-100%	R:25-50% L:0-25%	R:50-75% L:75-100%	R:75-100% L:75-100%	R:75-100% L:50-75%	R:50-75% L:50-75%	R:75-100% L: -
218	0-25%	75-100%	R:75-100% L:75-100%	-	R:50-75% L:50-75%	R:75-100% L:75-100%	R:0-25% L: -	R:50-75% L: -	-
219	25-50%	75-100%	R:75-100% L:25-50%	R:25-50% L:25-50%	R:0-25% L:50-75%	-	-	-	-
220	-	-	-	-	-	-	-	-	-
221	-	75-100%	R:75-100% L:75-100%	-	R:25-50% L:25-50%	R:75-100% L: -	R:25-50% L:25-50%	R:50-75% L:25-50%	-
231	-	75-100%	R:50-75% L:0-25%	R:25-50% L:25-50%	R:25-50% L:25-50%	R:75-100% L: -	R:50-75% L:50-75%	R:50-75% L:50-75%	R:50-75% L: -
232	0-25%	75-100%	R:75-100% L:50-75%	R:25-50% L:25-50%	R:25-50% L:25-50%	R:75-100% L:75-100%	R:50-75% L:50-75%	R:0-25% L:50-75%	-
264	25-50%	50-75%	R:50-75% L:25-50%	R:75-100% L:50-75%	R:50-75% L:25-50%	R: - L:0-25%	R:75-100% L:75-100%	R:50-75% L:50-75%	-
267 (265)	0-25%	-	R: Present L:Present	R:0-25% L: -	R:0-25% L: -	-	-	-	-
270	-	-	R:75-100% L:50-75%	-	R:25-50% L:0-25%	R:75-100% L: -	R:0-25% L: -	R:0-25% L: -	R:75-100% L: -
272	0-25%	75-100%	R:25-50% L:50-75%	R:0-25% L:0-25%	R:25-50% L:75-100%	R:75-100% L:75-100%	R:50-75% L:25-50%	R:50-75% L:50-75%	-
273	-	75-100%	R:50-75% L:75-100%	-	R:25-50% L:0-25%	R:75-100% L:75-100%	R:0-25% L:25-50%	R:0-25% L:25-50%	-
296	-	-	-	-	-	-	-	-	-
307	-	-	-	-	-	-	-	-	-
332	0-25%	-	R:75-100%	0-25%	R:25-50%	-	R:0-25%	R:50-75%	-

361	-	-	L: -	-	L: -	-	L:0-25%	L:50-75%	-
367	50-75%	75-100%	R:25-50% L:50-75%	R:0-25% L:0-25%	R:25-50% L:0-25%	-	R:25-50% L:50-75%	R: - L:0-25%	R:75-100% L:75-100%
385	-	-	-	-	-	-	-	-	-
414	-	-	-	-	-	-	-	-	-
450	25-50%	50-75%	R:25-50% L:25-50%	-	R:50-75% L: -	-	-	-	-
473	75-100%	-	R:75-100% L:75-100%	R:75-100% L:75-100%	R:75-100% L:0-25%	R:75-100% L:75-100%	R:75-100% L:75-100%	R:0-25% L:0-25%	R: - L:75-100%
514	-	75-100%	R:75-100% L:75-100%	-	-	R:75-100% L:75-100%	R:50-75% L:25-50%	R:25-50% L:25-50%	-
528	25-50%	-	R:75-100% L: -	R:25-50% L:25-50%	R:75-100% L:50-75%	R:75-100% L: -	R:25-50% L:25-50%	R:50-75% L:50-75%	R:75-100% L: -
634	-	NR	R:75-100% L:75-100%	R:75-100% L:75-100%	R:75-100% L:75-100%	R:75-100% L:75-100%	R:75-100% L:75-100%	R:75-100% L:75-100%	R:75-100% L:75-100%
647	0-25%	-	-	R: - L:25-50%	R:75-100% L: -	R:75-100% L: -	R:75-100% L:25-50%	R:0-25% L:25-50%	-
657	50-75%	75-100%	R:50-75% L:75-100%	R:50-75% L:25-50%	R:25-50% L:25-50%	-	R:0-25% L:50-75%	R:0-25% L:0-25%	-
678	-	-	-	-	-	-	-	-	-
699	0-25%	-	R: - L:25-50%	-	R:50-75% L:0-25%	-	-	-	-
784	75-100%	75-100%	R:75-100% L:75-100%	R:50-75% L:50-75%	R:50-75% L:75-100%	R: - L:75-100%	0-25%	-	-
835	75-100%	-	-	R:75-100% L:75-100%	R:75-100% L:75-100%	R: - L:75-100%	R:75-100% L:75-100%	R:25-50% L:0-25%	-

TABLE II-2. Completeness scores, in percentages, for cranial bones, Part 2. R = right, L = left, - = not preserved, Frags = only fragments remain.

	Sphenoid								
Juvenile	Greater Wing	Foramen rotundum	Lesser wing	Body	Lacrimal	Palatine	Maxilla	Mandible	Ethmoid
6	-	-	-	-	-	-	R: - L:75-100%	R: - L:75-100%	-
7	R:75-100% L:75-100%	R:75-100% L:75-100%	R:75-100% L:75-100%	75-100%	-	R:0-25% L:50-75%	R:50-75% L:25-50%	R:75-100% L:25-50%	-

163

18	-	-	-	-	-	-	R:75-100% L: -	-	-
37	R:0-25% L:0-25%	R: Present L:-	R:- L:50-75%	50-75%	-	R:50-75% L:-	R:50-75% L:25-50%	R:75-100% L:25-50%	-
56	R:50-75% L:50-75%	R:75-100% L:75-100%	-	Yes	-	R:50-75% L:75-100%	-	R:25-50% L:50-75%	0- 25%
66	-	-	-	-	-	-	-	R:75-100% L: -	-
74	-	-	-	-	-	-	-	-	-
91	-	-	-	-	-	-	-	-	-
95	R:75-100% L:25-50%	R:75-100% L:75-100%	R:75-100% L:75-100%	75-100%	-	-	R: - L:0-25%	R:75-100% L: -	-
100	-	-	-	-	-	-	-	-	-
110	R:75-100% L: -	R:75-100% L: -	-	75-100%	-	R:25-50% L: -	R: - L:0-25%	R:75-100% L:25-50%	-
111	R:50-75% L:50-75%	-	-	75-100%	-	-	R:50-75% L:75-100%	R:75-100% L:75-100%	-
124	R:75-100% L:75-100%	R:75-100% L:75-100%	R:50-75% L: -	75-100%	-	R:75-100% L:50-75%	R:75-100% L:25-50%	R:50-75% L:75-100%	-
125	R:75-100% L:75-100%	R:75-100% L:75-100%	R:75-100% L:75-100%	75-100%	-	-	R:25-50% L:75-100%	R:0-25% L:50-75%	-
153	R:75-100% L:75-100%	R:75-100% L:75-100%	R:75-100% L:75-100%	75-100%	-	R:50-75% L:50-75%	R:50-75% L:50-75%	R:75-100% L:75-100%	-
175	R:0-25% L:0-25%	R:75-100% L: -	R:75-100% L: -	0-25%	-	-	-	R: - L:50-75%	-
190	-	-	-	-	-	-	-	-	-
191	-	-	-	-	-	-	-	-	-
208	R:75-100% L:75-100%	R:75-100% L:75-100%	R:0-25% L:75-100%	-	-	R:75-100% L:75-100%	R:75-100% L:75-100%	R:75-100% L:75-100%	-
218	R:0-25% L: -	R:75-100% L: -	-	75-100%	-	R:50-75% L:75-100%	R:75-100% L:0-25%	R:75-100% L:75-100%	-
219	R:0-25% L:50-75%	R: - L:75-100%	-	-	-	-	R: - L:0-25%	R:25-50% L:25-50%	-
220	-	-	-	-	-	-	-	-	-
221	R:75-100% L:75-100%	-	R:75-100% L:25-50%	75-100%	-	-	R:0-25% L:0-25%	R:0-25% L:25-50%	-

164

231	R:0-25% L:0-25%	-	R:75-100% L:75-100%	75-100%	-	-	R:25-50% L:0-25%	R:0-25% L:75-100%	-
232	R:0-25% L:0-25%	-	R:75-100% L:75-100%	75-100%	-	-	R:25-50% L:0-25%	R:0-25% L:75-100%	-
264	-	-	-	-	-	-	R:0-25% L:50-75%	R:50-75% L:25-50%	-
267 (265)	R: - L:75-100%	R: - L: Present	-	-	-	-	-	-	-
270	R:0-25% L:0-25%	-	R:75-100% L:75-100%	50-75%	-	-	-	R:50-75% L:75-100%	-
272	R:50-75% L:75-100%	R: - L:75-100%	R: - L:50-75%	50-75%	-	-	-	R:0-25% L:25-50%	-
273	R:25-50% L:25-50%	-	-	75-100%	-	R: - L:25-50%	-	R:75-100% L:75-100%	-
296	-	-	-	-	-	-	-	-	-
307	-	-	-	-	-	-	-	-	-
332	-	-	R:50-75% L:50-75%	75-100%	-	-	R:25-50% L: -	-	-
361	-	-	-	-	-	-	-	-	-
367	-	R: - L:75-100%	R: - L:75-100%	75-100%	-	-	R: - L:0-25%	R:0-25% L:0-25%	-
385	-	-	-	-	-	-	-	-	-
414	-	-	-	-	-	-	-	-	-
450	-	-	-	-	-	-	R:25-50% L:0-25%	R:75-100% L:50-75%	-
473	R: - L:50-75%	-	-	-	-	-	R:25-50% L:50-75%	R:75-100% L:75-100%	-
514	R:75-100% L:75-100%	R:75-100% L:75-100%	R:75-100% L:75-100%	75-100%	-	R:75-100% L:75-100%	R:75-100% L:75-100%	R:0-25% L:75-100%	-
528	R:75-100% L:75-100%	R:75-100% L:75-100%	R:75-100% L:75-100%	75-100%	-	R:75-100% L:75-100%	R:75-100% L:75-100%	R:75-100% L: -	-
634	R:75-100% L:75-100%	R:75-100% L:75-100%	R:75-100% L:25-50%	75-100%	R:75-100% L: -	R:75-100% L:75-100%	R:75-100% L:75-100%	R:75-100% L:75-100%	50-75%
647	-	-	R:75-100% L: -	-	-	-	R:75-100% L: -	R:75-100% L: -	-
657	R:50-75%	-	R:50-75%	-	-	-	-	R:75-100%	-

165

	L:25-50%		L: -					L:75-100%	
678	-	-	-	-	-	-	R:75-100% L: -	-	-
699	-	-	R:0-25% L: -	-	-	-	-	-	-
784	R:0-25% L:75-100%	-	-	75-100%	R:75-100% L: -	R:75-100% L: -	R: - L:75-100%	R:75-100% L:75-100%	-
835	R: - L:75-100%	-	-	-	-	-	R:0-25% L: -	R:50-75% L:75-100%	-

TABLE II-3. Completeness and taphonomy scores for cranial bones, Part 3, and the vertebral column. - = not preserved, NR = not recorded, B =bodies, A = arches, +_ = number of extra fragments.

Juve-nile	Vomer	Cranial Bits?	Cranial taphonomy Score	Cervical	Thoracic	Lumbar	Sacral	Taphonomy Score	Unknown vertebral bits
6	-	Yes	Grade 1	-	-	-	-	Grade 2	14B, 12+2A
7	75-100%	-	-	1 (dens)	-	-	-	Grade 0	25B, 48+12A,
18	-	Yes	Grade 4	1 (dens)	-	-	-	-	-
37	75-100%	Yes	Grade 1	-	-	-	-	Grade 0	15B, 30A
56	Present	Yes	Grade 2	-	-	-	-	-	3A
66	-	Yes	Grade 2	-	-	-	-	Grade 1	7B, 6A
74	-	-	-	-	-	-	-	-	-
91	-	-	-	-	-	-	-	-	-
95	50-75%	Yes	Grade 1	-	-	-	-	Grade 0	29B, 51+7A
100	-	-	-	-	-	5B, 8+3A	4B, 3+2A	Grade 1	4B, 4+10A
110	Present	Yes	Grade 1	1 (dens)	-	-	4B	Grade 0	24+2B, 32+29A
111	Present	-	Grade 0	5B, 12+9A	6B, 6+8A	-	-	Grade 3-4	-
124	-	Yes	Grade 1	1 (dens)	-	-	-	-	25B, 40+8A
125	75-100%	Yes	Grade 1	6B, 10+1A	4B, 12+5A	5B, 10A	6B, 1A	Grade 0-1	-
153	Present	Yes	Grade 3	-	-	-	-	Grade 1	12+1B, 35+4A
175	0-25%	Yes	Grade 2	-	-	-	-	Grade 1	20B, 13+19A
190	-	-	-	-	-	3B, 4A	1+1B, 1A	Grade 0-1	-
191	-	-	-	-	-	-	-	-	-
208	-	Yes	Grade 1	-	-	-	-	Grade 0	17+2B, 49+5A
218	-	Yes	Grade 0	-	-	-	-	Grade 0	15B, 29A
219	-	Yes	Grade 1	-	-	-	-	Grade 1	22+1B, 39+12A
220	-	-	-	-	-	-	-	Grade 2-3	9+1B, 15+5A

221	-	No	Grade 1	-	-	-	-	Grade 1, some 2	20B, 37+6A
231	-	-	-	-	-	-	-	Grade 0-1	10B, 7+13A
232	-	-	-	-	-	-	-	Grade 2	22+1B, 41+9A
264	-	Yes	Grade 2, basilaris grade 5	-	-	-	-	Grade 4	17+2B, 29+15A
267 (265)	-	Yes	Grade 4	1 (dens)	-	-	-	Grade 4	1+5B, 8A, +9
270	Present	Yes	Grade 0	3B,13A	11B, 19+6A	4+1B, 12+5A	2B	Grade 1-3	-
272	-	Yes	Grade 2	-	-	-	-	Grade 0	17B, 32+5A
273	-	-	Grade 1	-	-	-	-	Grade 0	22+3B, 20+25A
296	-	-	-	-	-	3+1B, 3+11A	4+1B, 4+4A	Grade 1	-
307	-	-	-	-	-	-	-	Grade 1	10+2B, 10+28A
332	-	-	Grade 1	-	-	-	-	Grade 0	11B, 1+6A
361	-	Yes	-	-	-	+1A	2B	Grade 2-3	+2B, +10A
367	-	NR	Grade 1	-	-	-	+1A	Grade 1	15+9B, 12+25A
385	-	-	-	-	-	1A	-	-	-
414	-	Yes	-	-	-	-	-	Grade 1	12B, 12+10A
450	-	Yes	Grade 2	-	-	-	3B, 2+3A	Grade 3-4	10+5B, 19+3A
473	-	Yes	Grade 4	4+2B, 7+2A	+4B, 12A	4+2A	4B, 3+3A	Grade 3-4	-
514	75-100%	Yes	NR	1 (dens)	-	-	-	Grade 1 to 2	20+6B, 37+4A
528	75-100%	Yes	Grade 2	-	-	-	-	Grade 0	22B, 40+11A
634	75-100%	NR	Grade 1	6B, 3 +10A	12B, 5 +14A	5B, 5A	1+2B, 1+7A	Grade 0	-
647	-	Yes	Grade 2	-	-	-	-	-	- B, 5A
657	-	Yes	Grade 3	-	-	-	-	Grade 0	6 B, 7+17A
678	-	-	Grade 1	-	-	-	-	Grade 0	3B, 5A
699	-	-	Grade 2-3	-	-	-	-	Grade 3	3B, 5+4A
784	75-100%	Yes	Grade 4	1 (dens)	-	-	-	Grade 1 & 3	9+5B, 15+2A
835	-	-	Grade 4	6B, 5+5A	7B, 12A	5+2A	5B, 4A	Grade 0-1	-

TABLE II-4.Completeness counts for ribs and sternebrae (sternum), and taphonomy scores for ribs. R = right, L = left, U = unsided, SF = shaft fragment, ~ = minimum, - = not preserved.

Juvenile	Total Number of Ribs	Number of Sternal rib ends	Taphonomy score	Sternebrae
6	6+4SF	1	Grade 2	-
7	R: 13 +16SF, L: 12 +16SF	R: 4, L: 9	Grade 1	2
18	2	-	Grade 4	-

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37	18+27SF	8	Grade 3	1
56	R: 1+3 SF, L: 3+3SF	R: 1 L:3	Grade 2	-
66	6 (unknown side)	6 (unknown side)	Grade 3	1
74	-	-	-	-
91	-	-	-	-
95	R: 10 L: 11	R: 10 L: 11	Grade 1	-
100	R: 5, L: -	R: 1, L: -	Grade 1	-
110	R: 5+11SF, L: 4+40SF, 5 unsided +19SF	R: 4 L:1 U:5	Grade 1	4
111	R: ~7+34SF, L: ~4+11SF	-	Grade 2	-
124	R: 11+4 SF, L: 7+16SF	R: 5, L: 7	Grade 2-3	1
125	9	7	Grade 1	-
153	R: 10, L: 8	R: 9, L: 1	Grade 4	5
175	R: 3+13 SF, L: 10	R: 0, L: 5	Grade 2	1
190	-	-	-	-
191	1	1	Grade 1	-
208	R: 11, L: 11	R: 5 L:7	Grade 1	2
218	R: 6, L: 7	R: 6, L: 7	Grade 0	-
219	R: 6, L: 10	R: 2, L: 7	Grade 2-3	2
220	R: 6, L: 2+11SF	R: 1, L: 1	Grade 1	-
221	R: 7, L: 6	R: 5 L:1	Grade 2	-
231	R: 4, L: ~2+21SF	R: 2, L: 0	-	2
232	R: 8+12SF, L: 10+12SF	R: 4, L: 5	Grade 1	2
264	R: 10, L: 10	R: 7 L:10	Grade 1	-
267 (265)	-	-	-	-
270	R: 12+11SF, L: 10+12SF	R: 4, L: 7	-	3
272	R: 5+20SF, L: 9+8SF	R: 1, L: 3	Grade 2	-
273	13+44SF	7	Grade 1	3
296	-	-	-	-
307	R: 6+23SF, L: 9+15SF	R: 6, L: 4	-	1
332	3	3	Grade 1	-
361	R: - , L: 4+33SF	R: - , L: 3	Grade 1	-
367	R: 9 +23SF, L: 9 +17SF	R: 1 L:1	Grade 1	-
385	-	-	-	-
414	R: 1, L: 1	R: 0, L: 1	Grade 2	-
450	R: 3+31SF, L: 8+23SF	R: 0, L: 0	Grade 4	-

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473	R: 12, L: 12	R: 2, L: 1	Grade 1	-
514	R: - , L:11	-	Grade 1	1
528	R: 12+6 SF, L: 12+7SF	R: 11, L: 10	Grade 1	3
634	R: 12, L: 12 +2SF	R: 11 L: 9	Grade 3	3
647	U: 4 +2SF	U: 4	Grade 1	-
657	R: 4 +11SF, L: 6 +11SF	R: - , L: 3	Grade 2	-
678	-	-	-	1
699	-	-	-	-
784	R: 9+3SF, L: 5+3SF	R: 0, L: 1	Grade 4-5	-
835	R: 12, L: -	R: 2, L: -	Grade 2-3	-

TABLE II-5. Completeness scores for axial bones and taphonomy scores for the scapulae and ilium. R = right, L = Left, T = taphonomy grade, - = not preserved.

Juvenile	Scapula	Clavicle	Patella	Ilium	Ischium	Pubis
6	R: - L:75-100% (T:1)	-	-	R: 75-100% (T:2) L:50-75% (T:2)	R: 50-75% L:25-50%	R: 0-25% L:25-50%
7	R:75-100% (T:1) L:25-50% (T:1)	R:75-100% L:75-100%	-	R:75-100% (T:1) L:75-100% (T:1)	R:75-100% L:75-100%	R: - L:75-100% (T:0)
18	-	-	-	-	-	-
37	R:25-50% (T:3) L:25-50% (T:2)	R:50-75% L:25-50%	-	R: - L:0-25% (T:2)	-	-
56	R:0-25% (T:1) L:50-75% (T:1)	-	-	R: - L:50-75% (T:2)	-	-
66	-	-	-	R: - L:0-25% (T:3)	R: - L:0-25%	-
74	-	-	R: - L:75-100%	-	-	-
91	-	-	-	-	-	-
95	R:75-100% (T:0) L:0-25% (T:1)	R:75-100% L:75-100%	-	R:75-100% (T:0) L:75-100% (T:0)	R:75-100% L:75-100%	R:75-100% L:75-100%
100						
110	R:50-75% (T:1) L:0-25% (T:1)	R:75-100% L:25-50%	-	R:75-100% (T:1) L:75-100% (T:1)	-	-
111	-	-	-	R:75-100% (T:1) L:25-50% (T:1)	R:75-100% L:75-100%	R:75-100% L: -
124	R:0-25% (T:1)	R:75-100%	-	R:75-100% (T:1)	R:75-100%	-

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	L:50-75% (T:1)	L:0-25%		L:0-25% (T:1)	L: -	
125	R: -	R:75-100%	-	R:75-100% (T:2)	R:75-100%	R:75-100%
	L:50-75% (T:2)	L: -		L:75-100% (T:2)	L:75-100%	L:75-100%
153	R:75-100% (T:4) L:75-100% (T:3)	-	-	-	-	-
175	R:75-100% (T:2)	R:75-100%	-	R:0-25% (T:1)	R:75-100%	R:75-100%
	L: -	L:50-75%		L: -	L:75-100%	L:75-100%
190	-	-	-	R:75-100% (T:0) L:50-75% (T:2)	R:50-75% L:0-25%	-
191	-	-	-	-	-	-
208	R:75-100% (T:1)	R:75-100%	-	R:50-75% (T:1)	R:75-100%	R:75-100%
	L:50-75% (T:1)	L:75-100%		L:75-100% (T:0)	L:75-100%	L: -
218	-	R:75-100% L:25-50%	-	-	-	-
219	R: -	R:50-75%	-	R:75-100% (T:0)	-	-
	L:75-100% (T:0)	L:0-25%		L: -		
220	R:75-100% (T:0)	R:0-25%	-	-	-	-
	L: -	L: -				
221	R:50-75% (T:1) L:25-50% (T:1)	-	-	R:75-100% (T:3) L:25-50% (T:3)	-	-
231	R: -	R:25-50%	-	-	-	-
	L:0-25% (T:0)	L: -				
232	R: -	R:50-75%	-	R:75-100% (T:2)	R:75-100%	R:75-100%
	L:0-25% (T:1)	L:75-100%		L:75-100% (T:2)	L:75-100%	L: -
264	R:75-100% (T:1) L:25-50% (T:1)	R:75-100% L:75-100%	-	-	-	-
267 (265)	-	-	-	R:25-50% (T:3) L:0-25% (T:4)	-	R:75-100% L:0-25%
270	R:50-75% (T:3)	-	-	R:0-25% (T:4)	R: -	-
	L:75-100% (T:1)			L:50-75% (T:3)	L:50-75%)	
272	R: -	R:75-100%	-	-	-	-
	L:0-25% (T:1)	L:75-100%				
273	R:25-50% (T:2)	-	-	R:75-100% (T:2)	-	R: -
	L:75-100% (T:2)			L:75-100% (T:1)		L:25-50%
296	-	-	-	R:75-100% (T:1)	R:75-100%	R:75-100%
				L:75-100% (T:1)	L:75-100%	L:25-50%

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307	R:50-75% (T:1) L:25-50% (T:1)	R:50-75% L:50-75%	-	-	-	-
332	-	-	-	R:75-100% (T:0) L:50-75% (T:1)	R: - L:50-75%	R: - L:50-75%
361	-	R: - L:0-25%	-	R:0-25% (T:3) L:50-75% (T:3)	R:75-100% L:75-100%	R:75-100% (T:0) L: -
367	R:0-25% (T:0) L:0-25% (T:0)	R:50-75% L:50-75%	-	R:50-75% (T:1) L:25-50% (T:1)	-	-
385	-	-	-	-	-	-
414	-	-	-	R:75-100% (T:3) L:-	-	-
450	R:25-50% (T:2) L:25-50% (T:3)	R: - L:75-100%	-	R:75-100% (T:3) L:25-50% (T:3)	R: - L:75-100%	-
473	R:75-100% (T:2) L:75-100% (T:2)	R:75-100% L:75-100%	-	-	-	-
514	R:75-100% (T:2) L:75-100% (T:2)	R:75-100% L:75-100%	-	-	-	-
528	R:75-100% (T:1) L:75-100% (T:1)	R:75-100% L:75-100%	-	R:75-100% (T:1) L:75-100% (T:1)	R:75-100% L:75-100%	R:75-100% L:75-100%
634	R:75-100% (T:3) L:75-100% (T:3)	R:75-100% L:75-100%	-	R:75-100% (T:3) L: - (T:-)	-	-
647	R:75-100% (T:2) L:50-75% (T:2)	R: - L:75-100%	-	-	-	-
657	R:75-100% (T:1) L: -	R:75-100% L: -	-	R:0-25% (T:1) L:75-100% (T:1)	-	-
678	-	-	-	-	R: - L:75-100%	R:75-100% L:75-100%
699	-	R: - L:0-25	-	-	-	-
784	R:25-50% (T:5) L:75-100% (T:3)	R:75-100% L:50-75%	-	-	-	-
835	R:75-100% (T:4) L:75-100% (T:4)	R:75-100% L:75-100%	-	R:75-100% (T:4) L:75-100% (T:4)	R:75-100% L:75-100%	R:0-25% L:25-50%

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TABLE II-6. Right humerus completeness and taphonomy scores. Prox = proximal, Dis = distal, - = not preserved.

Juvenile	Prox. epiphysis	Prox. metaphysis	Prox. 1/3 of the shaft	Medial 1/3 of the shaft	Dis. 1/3 of the shaft	Dis. metaphysic	Dis. epiphysis	Taphonomy Score
6	-	-	-	-	-	-	-	-
7	-	50-75%	75-100%	75-100%	75-100%	75-100%	-	Grade 1
18	-	-	-	-	-	-	-	-
37	-	75-100%	50-75%	25-50%	-	-	-	Grade 2
56	-	75-100%	50-75%	75-100%	25-50%	-	-	Grade 3
66	-	-	-	-	-	-	-	-
74	-	-	-	-	-	-	-	-
91	-	-	-	-	-	-	-	-
95	-	75-100%	75-100%	75-100%	75-100%	75-100%	-	Grade 1
100	-	-	-	-	-	-	-	-
110	-	75-100%	75-100%	-	75-100%	75-100%	-	Grade 2
111	75-100%	0-25%	75-100%	75-100%	75-100%	75-100%	-	Grade 1
124	-	50-75%	75-100%	75-100%	75-100%	75-100%	-	Grade 1
125	-	-	-	-	-	-	-	-
153	-	25-50%	75-100%	75-100%	75-100%	75-100%	-	Grade 4
175	-	-	-	0-25%	-	-	-	Grade 2
190	-	-	-	-	-	-	-	-
191	-	-	-	-	-	-	-	-
208	-	75-100%	75-100%	75-100%	75-100%	75-100%	-	Grade 1
218	-	75-100%	75-100%	75-100%	75-100%	75-100%	-	Grade 1
219	-	50-75%	75-100%	75-100%	75-100%	75-100%	-	Grade 0
220	-	75-100%	75-100%	75-100%	75-100%	75-100%	-	Grade 1
221	-	50-75%	75-100%	75-100%	75-100%	75-100%	-	Grade 3
231	-	-	50-75%	75-100%	75-100%	25-50%	-	Grade 0
232	-	25-50%	75-100%	75-100%	75-100%	25-50%	-	Grade 1
264	-	-	75-100%	75-100%	75-100%	-	-	Grade 1
267 (265)	-	-	-	-	-	-	-	-
270	-	-	-	25-50%	75-100%	0-25%	-	Grade 3
272	-	75-100%	75-100%	75-100%	75-100%	75-100%	-	Grade 1
273	-	-	-	-	-	-	-	-
296	-	-	-	-	-	-	-	-
307	-	75-100%	75-100%	75-100%	75-100%	75-100%	-	Grade 2
332	-	-	-	-	-	-	-	-

361	-	-	-	-	-	-	-	-
367	-	-	75-100%	50-75%	-	-	-	Grade 2
385	-	-	-	-	-	-	-	-
414	-	-	-	-	25-50%	75-100%	-	Grade 3
450	-	0-25%	50-75%	-	-	-	-	Grade 2
473	75-100%	-	75-100%	75-100%	75-100%	25-50%	-	Grade 4
514	-	0-25%	25-50%	25-50%	25-50%	-	-	Grade 3
528	-	75-100%	75-100%	75-100%	75-100%	-	-	Grade 1
634	75-100%	75-100%	75-100%	75-100%	75-100%	25-50%	0-25%	Grade 3
647	-	75-100%	75-100%	75-100%	75-100%	75-100%	-	Grade 2
657	-	0-25%	75-100%	75-100%	75-100%	-	-	Grade 1
678	-	75-100%	75-100%	75-100%	75-100%	75-100%	-	Grade 2
699	-	-	-	-	-	-	-	-
784	-	50-75%	75-100%	75-100%	50-75%	-	-	Grade 4
835	75-100%	50-75%	75-100%	75-100%	75-100%	75-100%	-	Grade 3

TABLE II-7. Left humerus completeness and taphonomy scores. Prox = proximal, Dis = distal, - = not preserved.

Juvenile	Prox. epiphysis	Prox. metaphysis	Prox. 1/3 of the shaft	Medial 1/3 of the shaft	Dis. 1/3 of the shaft	Dis. metaphysic	Dis. epiphysis	Taphonomy Score
6	-	-	-	-	-	-	-	-
7	-	75-100%	75-100%	75-100%	75-100%	0-25%	-	Grade 1
18	-	-	-	-	-	-	-	-
37	-	-	-	-	-	-	-	-
56	-	75-100%	75-100%	75-100%	75-100%	75-100%	-	Grade 2
66	-	-	-	-	0-25%	-	-	-
74	-	-	-	-	-	-	-	-
91	-	-	-	-	-	-	-	-
95	-	75-100%	75-100%	75-100%	75-100%	75-100%	-	Grade 2
100	-	-	-	-	-	-	-	-
110	-	0-25%	-	-	25-50%	-	-	Grade 1
111	75-100%	-	0-25%	-	-	-	-	Grade 1
124	-	75-100%	75-100%	75-100%	75-100%	75-100%	-	Grade 1
125	-	75-100%	75-100%	75-100%	75-100%	75-100%	-	Grade 2
153	-	75-100%	75-100%	-	-	-	-	Grade 4
175	-	75-100%	75-100%	75-100%	75-100%	75-100%	-	Grade 2
190	-	-	-	-	-	-	-	-

191	-	-	-	-	-	-	-	-
208	-	75-100%	75-100%	75-100%	75-100%	75-100%	-	Grade 1
218	-	25-50%	75-100%	75-100%	75-100%	75-100%	-	Grade 2
219	-	-	-	-	-	-	-	-
220	-	-	-	-	-	-	-	-
221	-	0-25%	75-100%	75-100%	75-100%	75-100%	-	Grade 3
231	-	0-25%	50-75%	75-100%	75-100%	25-50%	-	Grade 0
232	-	-	25-50%	75-100%	75-100%	0-25%	-	Grade 1
264	-	0-25%	75-100%	75-100%	75-100%	0-25%	-	Grade 1
267 (265)	-	-	-	-	-	-	-	-
270	-	0-25%	75-100%	75-100%	75-100%	50-75%	-	Grade 1
272	-	-	75-100%	75-100%	75-100%	75-100%	-	Grade 1
273	-	-	-	-	-	-	-	-
296	-	-	-	-	-	-	-	-
307	75-100%	75-100%	75-100%	75-100%	0-25%	-	-	Grade 3
332	-	-	-	-	-	-	-	-
361	-	-	-	-	-	-	-	-
367	-	-	50-75%	75-100%	75-100%	75-100%	-	Grade 1
385	-	-	-	-	-	-	-	-
414	-	-	-	-	-	-	-	-
450	-	-	50-75%	75-100%	75-100%	-	-	Grade 2
473	75-100%	0-25%	75-100%	75-100%	75-100%	-	-	Grade 4
514	-	25-50%	75-100%	75-100%	75-100%	-	-	Grade 2
528	-	75-100%	75-100%	75-100%	75-100%	75-100%	-	Grade 1
634	-	25-50%	75-100%	75-100%	75-100%	50-75%	-	Grade 3
647	-	-	-	-	-	-	-	-
657	-	-	75-100%	75-100%	75-100%	-	-	Grade 1
678	-	-	-	-	-	-	-	-
699	-	-	-	-	-	-	-	-
784	-	-	-	50-75%	-	-	-	Grade 5++
835	-	50-75%	75-100%	75-100%	75-100%	75-100%	-	Grade 3

TABLE II-8. Right radius completeness and taphonomy scores. Prox = proximal, Dis = distal, - = not preserved.

Juvenile	Prox. epiphysis	Prox. metaphysis	Prox. 1/3 of the shaft	Medial 1/3 of the shaft	Dis. 1/3 of the shaft	Dis. metaphysic	Dis. epiphysis	Taphonomy Score
6	-	-	-	-	-	-	-	-
7	-	75-100%	75-100%	75-100%	75-100%	-	-	Grade 1
18	-	-	-	-	-	-	-	-
37	-	-	-	-	-	-	-	-
56	-	-	-	-	-	-	-	-
66	-	-	-	-	-	-	-	-
74	-	-	-	-	-	-	-	-
91	-	-	-	-	-	-	-	-
95	-	75-100%	75-100%	75-100%	75-100%	75-100%	-	Grade 1
100	-	75-100%	75-100%	75-100%	75-100%	75-100%	-	Grade 3
110	-	75-100%	75-100%	75-100%	0-25%	-	-	Grade 2
111	-	-	-	-	-	-	-	-
124	-	75-100%	75-100%	75-100%	75-100%	75-100%	-	Grade 1
125	-	75-100%	75-100%	75-100%	75-100%	0-25%	-	Grade 2
153	-	-	-	-	-	-	-	-
175	-	75-100%	50-75%	25-50%	-	-	-	Grade 2
190	-	-	-	-	-	-	-	-
191	-	-	-	-	-	-	-	-
208	-	75-100%	75-100%	75-100%	75-100%	-	-	Grade 1
218	-	75-100%	75-100%	75-100%	25-50%	-	-	Grade 1
219	-	-	-	-	25-50%	75-100%	-	Grade 0
220	-	-	-	-	-	-	-	-
221	-	75-100%	75-100%	75-100%	75-100%	75-100%	-	Grade 2
231	-	-	75-100%	75-100%	75-100%	75-100%	-	Grade 0
232	-	0-25%	75-100%	75-100%	75-100%	75-100%	75-100%	Grade 1
264	-	25-50%	75-100%	75-100%	75-100%	-	-	Grade 1
267 (265)	-	-	-	-	-	-	-	-
270	-	-	-	25-50%	75-100%	0-25%	-	Grade 1
272	-	75-100%	75-100%	75-100%	75-100%	50-75%	-	Grade 1
273	-	-	-	-	-	-	-	-
296	-	-	-	-	-	-	-	-
307	-	-	-	25-50%	75-100%	75-100%	-	Grade 2
332	-	-	-	-	50-75%	75-100%	-	Grade 1

361	-	-	-	-	-	-	-	-
367	-	25-50%	75-100%	25-50%	-	-	-	Grade 1
385	-	-	-	-	-	-	-	-
414	-	75-100%	75-100%	75-100%	75-100%	75-100%	-	Grade 3
450	-	-	75-100%	25-50%	-	-	-	Grade 2
473	-	-	-	-	-	-	-	-
514	-	-	75-100%	75-100%	75-100%	0-25%	-	Grade 2
528	-	75-100%	75-100%	75-100%	75-100%	75-100%	-	Grade 1
634	-	75-100%	75-100%	75-100%	75-100%	25-50%	75-100%	Grade 3
647	-	75-100%	75-100%	75-100%	75-100%	-	-	Grade 2
657	-	-	-	-	-	-	-	-
678	-	-	25-50%	75-100%	75-100%	75-100%	-	Grade 2
699	-	-	-	-	-	-	-	-
784	-	-	-	-	-	-	-	-
835	-	50-75%	75-100%	75-100%	75-100%	-	-	Grade 4

TABLE II-9. Left radius completeness and taphonomy scores. Prox = proximal, Dis = distal, - = not preserved.

Juvenile	Prox. epiphysis	Prox. metaphysis	Prox. 1/3 of the shaft	Medial 1/3 of the shaft	Dis. 1/3 of the shaft	Dis. metaphysic	Dis. epiphysis	Taphonomy Score
6	-	-	-	-	-	-	-	-
7	-	75-100%	75-100%	75-100%	75-100%	75-100%	-	Grade 1
18	-	-	-	-	-	-	-	-
37	-	-	-	-	-	-	-	-
56	-	75-100%	75-100%	75-100%	75-100%	75-100%	-	Grade 1
66	-	50-75%	50-75%	75-100%	75-100%	75-100%	-	Grade 3
74	-	-	-	-	-	-	-	-
91	-	-	-	-	-	-	-	-
95	-	75-100%	75-100%	75-100%	75-100%	75-100%	-	Grade 1
100	-	-	-	-	-	-	-	-
110	75-100%	0-25%	-	-	-	-	-	Grade 1
111	-	-	-	-	-	-	-	-
124	-	75-100%	75-100%	75-100%	75-100%	75-100%	-	Grade 1
125	-	-	-	75-100%	75-100%	50-75%	-	Grade 2
153	-	-	-	-	-	-	-	-
175	-	0-25%	75-100%	75-100%	75-100%	75-100%	-	Grade 2
190	-	-	-	-	-	-	-	-

176

191	-	-	-	-	-	-	-	-
208	-	75-100%	75-100%	75-100%	75-100%	50-75%	-	Grade 1
218	-	75-100%	75-100%	50-75%	-	-	-	Grade 1
219	-	-	-	-	-	-	-	-
220	-	-	-	-	-	-	-	-
221	-	75-100%	75-100%	75-100%	75-100%	75-100%	-	Grade 3
231	-	-	75-100%	75-100%	75-100%	-	-	Grade 0
232	-	75-100%	50-75%	75-100%	75-100%	75-100%	75-100%	Grade 1
264	-	0-25%	75-100%	75-100%	75-100%	0-25%	-	Grade 1
267 (265)	-	-	50-75%	75-100%	25-50%	50-75%	-	Grade 2
270	-	25-50%	50-75%	75-100%	75-100%	50-75%	-	Grade 2
272	-	75-100%	75-100%	75-100%	75-100%	75-100%	-	Grade 1
273	-	-	-	-	-	-	-	-
296	-	-	-	-	-	-	-	-
307	-	75-100%	75-100%	75-100%	75-100%	75-100%	-	Grade 2
332	-	-	-	-	-	-	-	-
361	-	-	-	-	-	-	-	-
367	-	0-25%	75-100%	75-100%	75-100%	-	-	Grade 2
385	-	-	-	-	-	-	-	-
414	-	-	75-100%	75-100%	75-100%	25-50%	-	Grade 3
450	-	-	50-75%	75-100%	75-100%	-	-	Grade 2
473	-	-	-	0-25%	75-100%	-	-	Grade 4
514	-	25-50%	75-100%	75-100%	75-100%	-	-	Grade 1
528	-	75-100%	75-100%	75-100%	75-100%	75-100%	-	Grade 1
634	-	75-100%	75-100%	75-100%	75-100%	75-100%	75-100%	Grade 3
647	-	-	-	-	-	-	-	-
657	-	-	-	-	-	-	-	-
678	-	-	-	-	-	-	-	-
699	-	-	-	-	-	-	-	-
784	-	-	-	-	-	-	-	-
835	-	-	75-100%	75-100%	50-75%	-	-	Grade 4

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TABLE II-10. Right ulna completeness and taphonomy scores. Prox = proximal, Dis = distal, - = not preserved.

Juvenile	Prox. epiphysis	Prox. metaphysis	Prox. 1/3 of the shaft	Medial 1/3 of the shaft	Dis. 1/3 of the shaft	Dis. metaphysic	Dis. epiphysis	Taphonomy Score
6	-	-	-	-	-	-	-	-
7	-	75-100%	75-100%	75-100%	75-100%	-	-	Grade 1
18	-	-	-	-	-	-	-	-
37	-	-	-	-	-	-	-	-
56	-	-	-	-	-	-	-	-
66	-	-	-	-	-	-	-	-
74	-	-	-	-	-	-	-	-
91	-	-	-	-	-	-	-	-
95	-	75-100%	75-100%	75-100%	75-100%	75-100%	-	Grade 3
100	-	75-100%	75-100%	75-100%	75-100%	-	-	-
110	-	25-50%	75-100%	75-100%	75-100%	50-75%	-	Grade 2
111	-	75-100%	25-50%	-	-	-	-	Grade 1
124	-	25-50%	50-75%	75-100%	75-100%	75-100%	-	Grade 1
125	-	-	-	-	-	-	-	-
153	-	75-100%	75-100%	75-100%	75-100%	75-100%	-	Grade 4
175	-	-	-	-	-	-	-	-
190	-	-	-	-	-	-	-	-
191	-	-	-	50-75%	75-100%	25-50%	-	Grade 2
208	-	75-100%	75-100%	50-75%	50-75%	-	-	Grade 1
218	-	75-100%	75-100%	75-100%	25-50%	-	-	Grade 1
219	-	75-100%	75-100%	25-50%	-	-	-	Grade 0
220	-	-	-	-	-	-	-	-
221	-	75-100%	75-100%	75-100%	75-100%	25-50%	-	Grade 2
231	-	75-100%	75-100%	0-25%	-	-	-	Grade 0
232	-	75-100%	75-100%	75-100%	75-100%	75-100%	-	Grade 1
264	-	0-25%	75-100%	75-100%	75-100%	75-100%	-	Grade 1
267 (265)	-	-	-	-	-	-	-	-
270	-	0-25%	0-25%	75-100%	-	-	-	Grade 1
272	-	75-100%	75-100%	75-100%	75-100%	-	-	Grade 1
273	-	-	-	-	-	-	-	-
296	-	-	-	-	-	-	-	-
307	-	75-100%	75-100%	75-100%	75-100%	75-100%	-	Grade 2
332	-	-	-	-	-	-	-	-

361	-	-	-	-	-	-	-	-
367	-	-	75-100%	75-100%	-	-	-	Grade 0
385	-	-	-	-	-	-	-	-
414	-	75-100%	75-100%	75-100%	75-100%	75-100%	-	Grade 3
450	-	-	25-50%	75-100%	75-100%	-	-	Grade 2
473	-	-	-	-	-	-	-	-
514	-	-	50-75%	75-100%	75-100%	-	-	Grade 2
528	-	75-100%	75-100%	75-100%	75-100%	75-100%	-	Grade 1
634	-	75-100%	75-100%	75-100%	75-100%	75-100%	-	Grade 3
647	-	-	-	-	-	-	-	-
657	-	-	50-75%	75-100%	-	-	-	Grade 3
678	-	75-100%	75-100%	75-100%	75-100%	75-100%	-	Grade 2
699	-	-	-	-	-	-	-	-
784	-	-	-	-	-	-	-	-
835	-	75-100%	75-100%	75-100%	75-100%	-	-	Grade 4

TABLE II-11. Left ulna completeness and taphonomy scores. Prox = proximal, Dis = distal, - = not preserved.

Juvenile	Prox. epiphysis	Prox. metaphysis	Prox. 1/3 of the shaft	Medial 1/3 of the shaft	Dis. 1/3 of the shaft	Dis. metaphysic	Dis. epiphysis	Taphonomy Score
6	-	-	-	-	-	-	-	-
7	-	75-100%	75-100%	75-100%	75-100%	75-100%	-	Grade 1
18	-	-	-	-	-	-	-	-
37	-	-	-	-	-	-	-	-
56	-	75-100%	75-100%	75-100%	75-100%	75-100%	-	Grade 2
66	-	50-75%	50-75%	75-100%	75-100%	75-100%	-	Grade 3
74	-	-	-	-	-	-	-	-
91	-	-	-	-	-	-	-	-
95	-	75-100%	75-100%	75-100%	75-100%	75-100%	-	Grade 1
100	-	-	25-50%	-	75-100%	75-100%	-	Grade 1
110	-	-	0-25%	50-75%	-	-	-	Grade 1
111	-	-	-	-	-	-	-	-
124	-	75-100%	75-100%	75-100%	75-100%	75-100%	-	Grade 1
125	-	75-100%	75-100%	75-100%	0-25%	-	-	Grade 2
153	-	-	-	-	-	-	-	-
175	-	75-100%	50-75%	50-75%	0-25%	-	-	Grade 2
190	-	-	-	-	-	-	-	-

179

191	-	-	-	-	-	-	-	-
208	-	75-100%	75-100%	75-100%	75-100%	75-100%	-	Grade 1
218	-	75-100%	75-100%	75-100%	75-100%	75-100%	-	Grade 1
219	-	-	-	-	-	-	-	-
220	-	-	-	-	-	-	-	-
221	-	75-100%	75-100%	75-100%	75-100%	75-100%	-	Grade 2
231	-	-	-	-	-	-	-	-
232	-	75-100%	75-100%	75-100%	0-25%	-	-	Grade 1
264	-	0-25%	75-100%	75-100%	75-100%	0-25%	-	Grade 1
267 (265)	-	50-75%	75-100%	75-100%	75-100%	75-100%	-	Grade 2
270	-	75-100%	75-100%	75-100%	25-50%	-	-	Grade 1
272	-	75-100%	75-100%	75-100%	75-100%	75-100%	-	Grade 1
273	-	-	-	-	-	-	-	-
296	-	-	-	-	-	-	-	-
307	-	-	75-100%	75-100%	75-100%	75-100%	-	Grade 2
332	-	-	-	-	-	-	-	-
361	-	-	-	-	-	-	-	-
367	-	75-100%	25-50%	75-100%	75-100%	-	-	Grade 1
385	-	-	-	-	-	-	-	-
414	-	-	50-75%	75-100%	50-75%	-	-	Grade 3
450	-	-	25-50%	75-100%	75-100%	-	-	Grade 2
473	-	-	-	-	-	-	-	-
514	-	75-100%	75-100%	75-100%	75-100%	-	-	Grade 2
528	-	75-100%	75-100%	75-100%	75-100%	75-100%	-	Grade 1
634	-	75-100%	75-100%	75-100%	75-100%	0-25%	-	Grade 3
647	-	75-100%	75-100%	75-100%	75-100%	75-100%	-	Grade 2
657	-	25-50%	0-25%	-	-	-	-	Grade 1
678	-	-	-	-	-	-	-	-
699	-	-	-	-	-	-	-	-
784	-	-	-	-	-	-	-	-
835	-	50-75%	75-100%	75-100%	50-75%	-	-	Grade 4

180

TABLE II-12. Right femur completeness and taphonomy scores. Prox = proximal, Dis = distal, - = not preserved.

Juvenile	Prox. epiphysis	Prox. metaphysis	Prox. 1/3 of the shaft	Medial 1/3 of the shaft	Dis. 1/3 of the shaft	Dis. metaphysic	Dis. epiphysis	Taphonomy Score
6	-	50-75%	75-100%	75-100%	75-100%	75-100%	-	Grade 3
7	-	75-100%	75-100%	75-100%	75-100%	75-100%	-	Grade 1
18	-	-	-	-	-	-	-	-
37	-	-	-	-	-	-	-	-
56	-	-	0-25%	25-50%	0-25%	-	-	Grade 1
66	-	-	-	-	-	-	-	-
74	-	-	-	-	-	-	-	-
91	-	25-50%	75-100%	0-25%	-	-	-	Grade 2
95	-	75-100%	75-100%	75-100%	75-100%	75-100%	-	Grade 1
100	-	-	-	-	75-100%	75-100%	-	-
110	-	-	-	-	-	-	-	-
111	-	-	-	-	-	-	-	-
124	-	50-75%	75-100%	75-100%	25-50%	-	-	Grade 1
125	-	75-100%	75-100%	75-100%	75-100%	50-75%	-	Grade 2
153	-	-	-	-	-	-	-	-
175	-	-	-	-	-	-	-	-
190	-	75-100%	75-100%	75-100%	50-75%	25-50%	-	Grade 1
191	-	0-25%	75-100%	75-100%	75-100%	25-50%	-	Grade 2
208	-	50-75%	75-100%	75-100%	75-100%	50-75%	-	Grade 2
218	-	-	-	-	-	-	-	-
219	-	-	-	-	-	-	-	-
220	-	-	-	-	-	-	-	-
221	-	50-75%	75-100%	75-100%	75-100%	75-100%	-	Grade 2
231	-	-	-	-	-	-	-	-
232	-	0-25%	75-100%	75-100%	75-100%	0-25%	-	Grade 1
264	-	-	-	-	-	-	-	-
267 (265)	-	75-100%	75-100%	75-100%	75-100%	25-50%	-	Grade 3
270	-	-	50-75%	75-100%	75-100%	-	-	Grade 4
272	-	-	-	-	-	-	-	-
273	-	0-25%	75-100%	75-100%	25-50%	-	-	Grade 1
296	75-100%	75-100%	75-100%	75-100%	75-100%	75-100%	75-100%	Grade 1
307	-	-	-	-	-	-	-	-
332	-	0-25%	75-100%	75-100%	75-100%	-	-	Grade 1

361	-	-	0-25%	50-75%	25-50%	-	-	Grade 0
367	-	25-50%	75-100%	75-100%	50-75%	-	-	Grade 1
385	-	-	-	-	-	-	-	-
414	-	75-100%	75-100%	75-100%	-	-	-	Grade 3
450	75-100%	-	-	-	-	-	-	Grade 1
473	-	-	-	-	-	-	-	-
514	-	-	-	-	-	-	-	-
528	-	75-100%	75-100%	75-100%	75-100%	75-100%	-	Grade 1
634	-	75-100%	75-100%	75-100%	75-100%	75-100%	-	Grade 2
647	-	75-100%	75-100%	75-100%	75-100%	75-100%	-	Grade 2
657	-	-	50-75%	75-100%	50-75%	-	75-100%	Grade 2
678	75-100%	75-100%	75-100%	75-100%	25-50%	-	-	Grade 1
699	-	-	-	-	-	-	-	-
784	-	-	-	-	-	-	-	-
835	75-100%	-	75-100%	75-100%	75-100%	-	75-100%	Grade 4

TABLE II-13. Left femur completeness and taphonomy scores. Prox = proximal, Dis = distal, - = not preserved.

Juvenile	Prox. epiphysis	Prox. metaphysis	Prox. 1/3 of the shaft	Medial 1/3 of the shaft	Dis. 1/3 of the shaft	Dis. metaphysic	Dis. epiphysis	Taphonomy Score
6	-	75-100%	75-100%	75-100%	75-100%	75-100%	-	Grade 3
7	-	75-100%	75-100%	75-100%	75-100%	-	-	Grade 1
18	-	-	-	-	-	-	-	-
37	-	-	-	-	-	-	-	-
56	-	75-100%	75-100%	75-100%	0-25%	-	-	Grade 3
66	-	50-75%	50-75%	75-100%	75-100%	75-100%	-	Grade 3
74	-	-	-	-	-	-	-	-
91	-	-	-	-	-	-	-	-
95	-	75-100%	75-100%	75-100%	75-100%	75-100%	-	Grade 1
100	-	-	-	-	-	-	-	-
110	-	-	-	-	-	-	-	-
111	-	-	-	-	-	-	-	-
124	-	-	-	-	-	-	-	-
125	75-100%	75-100%	75-100%	75-100%	75-100%	75-100%	-	Grade 2
153	-	-	-	-	-	-	-	-
175	-	-	-	-	-	-	-	-
190	-	75-100%	75-100%	75-100%	50-75%	25-50%	-	Grade 1

191	-	-	0-25%	75-100%	0-25%	-	-	Grade 2
208	-	75-100%	75-100%	75-100%	75-100%	75-100%	-	Grade 1
218	-	-	-	-	-	-	-	-
219	-	-	-	-	-	-	-	-
220	-	-	-	-	-	-	-	-
221	-	-	-	-	-	-	-	-
231	-	-	-	-	-	-	-	-
232	-	0-25%	75-100%	75-100%	75-100%	-	-	Grade 2
264	-	-	-	-	-	-	-	-
267 (265)	-	25-50%	50-75%	75-100%	25-50%	-	-	Grade 1
270	-	-	75-100%	75-100%	25-50%	-	-	Grade 1
272	-	-	-	-	-	-	-	-
273	-	50-75%	75-100%	75-100%	50-75%	50-75%	-	Grade 1
296	75-100%	75-100%	75-100%	75-100%	75-100%	75-100%	75-100%	Grade 1
307	-	-	-	-	-	-	-	-
332	-	75-100%	75-100%	75-100%	50-75%	-	-	Grade 1
361	75-100%	25-50%	75-100%	75-100%	50-75%	-	-	Grade 2
367	-	25-50%	75-100%	75-100%	50-75%	-	-	Grade 1
385	-	50-75%	75-100%	75-100%	50-75%	-	75-100%	Grade 1
414	-	75-100%	75-100%	-	-	-	-	Grade 3
450	-	-	-	-	-	-	-	-
473	-	-	-	-	-	-	-	-
514	-	-	-	-	-	-	-	-
528	-	50-75%	75-100%	75-100%	75-100%	75-100%	-	Grade 1
634	-	-	-	-	-	-	-	-
647	-	75-100%	75-100%	75-100%	75-100%	0-25%	-	Grade 2
657	-	25-50%	75-100%	75-100%	25-50%	-	-	Grade 2
678	-	75-100%	25-50%	75-100%	75-100%	75-100%	-	Grade 2
699	-	-	-	-	-	-	-	-
784	-	-	25-50%	25-50%	25-50%	-	-	Grade 5
835	75-100%	-	75-100%	75-100%	75-100%	-	75-100%	Grade 4

183

TABLE II-14. Right tibia completeness and taphonomy scores. Prox = proximal, Dis = distal, - = not preserved.

Juvenile	Prox. epiphysis	Prox. metaphysis	Prox. 1/3 of the shaft	Medial 1/3 of the shaft	Dis. 1/3 of the shaft	Dis. metaphysic	Dis. epiphysis	Taphonomy Score
6	-	75-100%	75-100%	75-100%	75-100%	75-100%	-	Grade 3
7	-	75-100%	75-100%	75-100%	75-100%	75-100%	-	Grade 1
18	-	-	-	-	-	-	-	-
37	-	-	-	-	-	-	-	-
56	-	-	0-25%	0-25%	0-25%	-	-	Grade 2
66	-	-	-	-	-	-	-	-
74	-	75-100%	75-100%	75-100%	75-100%	75-100%	-	Grade 2
91	-	50-75%	50-75%	75-100%	75-100%	75-100%	-	Grade 2
95	-	75-100%	75-100%	75-100%	75-100%	75-100%	-	Grade 1
100	-	-	-	-	-	-	-	-
110	-	-	-	-	-	-	-	-
111	-	-	-	-	-	-	-	-
124	-	-	-	-	-	-	-	-
125	-	50-75%	50-75%	75-100%	75-100%	0-25%	-	Grade 2
153	-	-	-	-	-	-	-	-
175	-	-	-	-	-	-	-	-
190	75-100%	75-100%	75-100%	75-100%	75-100%	50-75%	-	Grade 0
191	-	-	-	-	-	-	-	-
208	-	50-75%	75-100%	75-100%	25-50%	-	-	Grade 1
218	-	-	-	-	-	-	-	-
219	-	-	-	-	-	-	-	-
220	-	25-50%	25-50%	75-100%	75-100%	75-100%	-	Grade 1
221	-	-	-	-	-	-	-	-
231	-	-	-	-	-	-	-	-
232	-	0-25%	75-100%	75-100%	75-100%	0-25%	-	Grade 2
264	-	-	-	-	-	-	-	-
267 (265)	-	-	-	-	-	-	-	-
270	-	-	75-100%	75-100%	75-100%	0-25%	-	Grade 2
272	-	-	-	-	-	-	-	-
273	-	-	-	-	75-100%	75-100%	-	Grade 1
296	-	75-100%	75-100%	75-100%	75-100%	75-100%	-	Grade 1
307	-	-	-	-	-	-	-	-
332	-	75-100%	75-100%	75-100%	50-75%	-	-	Grade 1

184

361	-	-	-	-	-	-	-	-
367	-	-	50-75%	75-100%	50-75%	-	-	Grade 1
385	-	-	-	25-50%	-	-	-	Grade 1
414	-	-	-	-	-	-	-	-
450	-	-	50-75%	75-100%	50-75%	-	-	Grade 2
473	-	-	-	-	-	-	-	-
514	-	-	-	-	-	-	-	-
528	-	75-100%	75-100%	75-100%	25-50%	-	-	Grade 1
634	-	-	-	-	-	-	-	-
647	-	75-100%	75-100%	75-100%	75-100%	0-25%	-	Grade 2
657	-	0-25%	75-100%	75-100%	75-100%	75-100%	-	Grade 2
678	-	-	-	-	-	-	-	-
699	-	-	-	-	-	-	-	-
784	-	0-25%	75-100%	75-100%	25-50%	-	-	Grage 5
835	-	-	75-100%	75-100%	75-100%	75-100%	-	Grade 4

TABLE II-15. Left tibia completeness and taphonomy scores. Prox = proximal, Dis = distal, - = not preserved.

Juvenile	Prox. epiphysis	Prox. metaphysis	Prox. 1/3 of the shaft	Medial 1/3 of the shaft	Dis. 1/3 of the shaft	Dis. metaphysic	Dis. epiphysis	Taphonomy Score
6	-	75-100%	75-100%	75-100%	75-100%	75-100%	-	Grade 3
7	-	75-100%	75-100%	75-100%	75-100%	75-100%	-	Grade 1
18	-	-	-	-	-	-	-	-
37	-	-	-	-	-	-	-	-
56	-	0-25%	0-25%	75-100%	75-100%	75-100%	-	Grade 2
66	-	75-100%	75-100%	75-100%	75-100%	75-100%	-	Grade 2
74	-	75-100%	75-100%	75-100%	75-100%	75-100%	-	Grade 2
91	-	25-50%	50-75%	75-100%	50-75%	25-50%	-	Grade 2
95	-	75-100%	75-100%	75-100%	75-100%	75-100%	-	Grade 1
100	-	-	-	-	-	-	-	-
110	-	-	-	-	-	-	-	-
111	-	-	-	-	-	-	-	-
124	-	-	-	-	-	-	-	-
125	-	75-100%	75-100%	75-100%	75-100%	75-100%	-	Grade 2
153	-	-	-	-	-	-	-	-
175	-	-	-	-	-	-	-	-
190	50-75%	25-50%	75-100%	75-100%	-	-	-	Grade 0

185

191	-	-	-	-	-	-	-	-
208	-	50-75%	25-50%	75-100%	25-50%	-	-	Grade 2
218	-	-	-	-	-	-	-	-
219	-	-	-	-	-	-	-	-
220	-	75-100%	75-100%	75-100%	75-100%	75-100%	-	Grade 1
221	-	-	-	-	-	-	-	-
231	-	-	-	-	-	-	-	-
232	-	0-25%	75-100%	75-100%	0-25%	25-50%	-	Grade 2
264	-	-	-	-	-	-	-	-
267 (265)	-	-	-	-	-	-	-	-
270	-	-	75-100%	75-100%	75-100%	-	-	Grade 3
272	-	-	-	-	-	-	-	-
273	-	75-100%	75-100%	75-100%	75-100%	50-75%	-	Grade 1
296	75-100%	75-100%	75-100%	75-100%	75-100%	75-100%	75-100%	Grade 1
307	-	-	-	-	-	-	-	-
332	-	0-25%	0-25%	75-100%	0-25%	-	-	Grade 1
361	-	-	50-75%	50-75%	0-25%	-	-	Grade 1
367	-	0-25%	75-100%	75-100%	50-75%	-	-	Grade 1
385	-	75-100%	75-100%	75-100%	25-50%	-	75-100%	Grade 1
414	-	-	-	-	-	-	-	-
450	-	-	-	50-75%	50-75%	-	-	Grade 2
473	-	-	-	-	-	-	-	-
514	-	-	-	-	-	-	-	-
528	-	75-100%	75-100%	75-100%	75-100%	75-100%	-	Grade 1
634	-	-	-	-	-	-	-	-
647	-	75-100%	75-100%	75-100%	75-100%	75-100%	75-100%	Grade 3
657	-	75-100%	75-100%	75-100%	75-100%	75-100%	-	Grade 2
678	-	-	-	-	-	-	-	-
699	-	-	-	-	-	-	-	-
784	-	-	25-50%	0-25%	0-25%	-	-	Grage 5
835	-	75-100%	75-100%	75-100%	75-100%	0-25%	75-100%	Grade 4

TABLE II-16. Right fibula completeness and taphonomy scores. Prox = proximal, Dis = distal, - = not preserved.

Juvenile	Prox. epiphysis	Prox. metaphysis	Prox. 1/3 of the shaft	Medial 1/3 of the shaft	Dis. 1/3 of the shaft	Dis. metaphysic	Dis. epiphysis	Taphonomy Score
6	-	75-100%	75-100%	75-100%	75-100%	75-100%	-	Grade 3
7	-	-	75-100%	75-100%	75-100%	75-100%	-	Grade 1
18	-	-	-	-	-	-	-	-
37	-	-	-	-	-	-	-	-
56	-	-	-	0-25%	-	-	-	Grade 1
66	-	-	-	-	-	-	-	-
74	-	75-100%	75-100%	75-100%	75-100%	75-100%	-	Grade 2
91	-	50-75%	50-75%	75-100%	75-100%	75-100%	-	Grade 2
95	-	75-100%	75-100%	75-100%	75-100%	75-100%	-	Grade 1
100	-	-	-	-	-	-	-	-
110	-	-	-	-	-	-	-	-
111	-	-	-	-	-	-	-	-
124	-	-	-	-	-	-	-	-
125	-	75-100%	75-100%	75-100%	75-100%	75-100%	-	Grade 2
153	-	-	-	-	-	-	-	-
175	-	-	-	-	-	-	-	-
190	-	-	50-75%	75-100%	75-100%	75-100%	-	Grade 0
191	-	-	-	-	-	-	-	-
208	-	-	-	-	-	-	-	-
218	-	-	-	-	-	-	-	-
219	-	-	-	-	-	-	-	-
220	-	75-100%	75-100%	75-100%	75-100%	75-100%	-	Grade 1
221	-	-	-	-	-	-	-	-
231	-	-	-	-	-	-	-	-
232	-	-	75-100%	75-100%	75-100%	-	-	Grade 2
264	-	-	-	-	-	-	-	-
267 (265)	-	-	-	-	-	-	-	-
270	-	-	75-100%	75-100%	75-100%	-	-	Grade 2
272	-	-	-	-	-	-	-	-
273	-	-	-	-	75-100%	75-100%	-	Grade 1
296	-	75-100%	75-100%	75-100%	75-100%	75-100%	-	Grade 1
307	-	-	-	-	-	-	-	-
332	-	-	-	75-100%	-	-	-	Grade 1

361	-	-	-	-	-	-	-	-
367	-	-	-	25-50%	25-50%	-	-	Grade 1
385	-	-	-	-	-	-	-	-
414	-	-	-	-	-	-	-	-
450	-	-	50-75%	75-100%	50-75%	-	-	Grade 3
473	-	-	-	-	-	-	-	-
514	-	-	-	-	-	-	-	-
528	-	0-25%	75-100%	75-100%	75-100%	75-100%	-	Grade 1
634	-	-	-	-	-	-	-	-
647	-	75-100%	75-100%	75-100%	75-100%	-	-	Grade 2
657	-	-	50-75%	75-100%	75-100%	-	-	Grade 2
678	-	-	-	-	-	-	-	-
699	-	-	-	-	-	-	-	-
784	-	-	-	-	-	-	-	-
835	-	-	50-75%	75-100%	75-100%	75-100%	-	Grade 4

TABLE II-17. Left fibula completeness and taphonomy scores. Prox = proximal, Dis = distal, - = not preserved.

Juvenile	Prox. epiphysis	Prox. metaphysis	Prox. 1/3 of the shaft	Medial 1/3 of the shaft	Dis. 1/3 of the shaft	Dis. metaphysic	Dis. epiphysis	Taphonomy Score
6	-	-	-	-	75-100%	75-100%	-	Grade 3
7	-	-	75-100%	75-100%	75-100%	75-100%	-	Grade 1
18	-	-	-	-	-	-	-	-
37	-	-	-	-	-	-	-	-
56	-	-	0-25%	50-75%	75-100%	75-100%	-	Grade 3
66	-	25-50%	25-50%	75-100%	75-100%	75-100%	-	Grade 1
74	-	-	75-100%	75-100%	75-100%	75-100%	-	Grade 2
91	-	50-75%	75-100%	75-100%	50-75%	75-100%	-	Grade 2
95	-	75-100%	75-100%	75-100%	75-100%	75-100%	-	Grade 1
100	-	-	-	-	-	-	-	-
110	-	-	-	-	-	-	-	-
111	-	-	-	-	-	-	-	-
124	-	-	-	-	-	-	-	-
125	-	25-50%	75-100%	75-100%	75-100%	75-100%	-	Grade 2
153	-	-	-	-	-	-	-	-
175	-	-	-	-	-	-	-	-
190	-	-	75-100%	75-100%	75-100%	75-100%	-	Grade 0

188

191	-	-	-	-	-	-	-	-
208	-	-	-	0-25%	-	-	-	Grade 3
218	-	-	-	-	-	-	-	-
219	-	-	-	-	-	-	-	-
220	-	-	-	-	25-50%	-	-	Grade 0
221	-	-	-	-	-	-	-	-
231	-	-	-	-	-	-	-	-
232	-	-	-	25-50%	-	-	-	Grade 0
264	-	-	-	-	-	-	-	-
267 (265)	-	-	-	-	-	-	-	-
270	-	-	75-100%	75-100%	75-100%	75-100%	-	Grade 2
272	-	-	-	-	-	-	-	-
273	-	75-100%	75-100%	75-100%	75-100%	75-100%	-	Grade 1
296	-	75-100%	75-100%	75-100%	75-100%	75-100%	-	Grade 1
307	-	-	-	-	-	-	-	-
332	-	-	50-75%	75-100%	75-100%	50-75%	-	Grade 1
361	-	-	-	-	-	-	-	-
367	-	-	25-50%	75-100%	25-50%	-	-	Grade 1
385	-	75-100%	75-100%	75-100%	75-100%	75-100%	-	Grade 1
414	-	-	-	-	-	-	-	-
450	-	-	25-50%	75-100%	50-75%	-	-	Grade 2
473	-	-	-	-	-	-	-	-
514	-	-	-	-	-	-	-	-
528	-	75-100%	75-100%	75-100%	75-100%	75-100%	-	Grade 1
634	-	-	-	-	-	-	-	-
647	-	75-100%	75-100%	75-100%	75-100%	75-100%	75-100%	Grade 2
657	-	50-75%	75-100%	75-100%	75-100%	50-75%	-	Grade 2
678	-	-	-	-	-	-	-	-
699	-	-	-	-	-	-	-	-
784	-	-	-	-	-	-	-	-
835	-	-	25-50%	75-100%	25-50%	-	-	Grade 4

TABLE II-18. Hands and feet counts, given as the number of bones and fragment present. - = not preserved.

	Hands		Feet	
Juvenile	Carpals	Metacarpals & phalanges	Tarsals	Metatarsals & phalanges
56	-	-	-	-
66	-	9	-	-
95	-	22	3	19
110	-	22 +9fragments	-	7 +3fragments
208	-	15 +8fragments	-	-
221	-	3	-	-
264	-	11 +2fragments	-	-
367	-	12 + 1fragment	-	5 + 6fragments
514	4	17 +3fragments	-	-
634	2	33 +9 epiphyses	-	-
647	-	-	-	-
657	-	-	-	3

B - Taphonomy

TABLE II-19. Taphonomic scores of the juveniles analysed from Saint-Amé. R = right, L = left. NR = not recorded.

Juve-				Scapula		Ilium		Humerus		Radius		Ulna		Femur		Tibia		Fibula	
nile	Skull	Vertebra	Ribs	R	L	R	L	R	L	R	L	R	L	R	L	R	L	R	L
6	1	2, few 3-4	2	-	1	2	2	-	-	-	-	-	-	3	3	3	3	3	3
7	NR	0	1	1	1	1	1	1	1	1	1	1	1	1	1	1	1	1	1
18	4	-	4	-	-	-	-	-	-	-	-	-	-	-	-	-	-	-	-
37	1	0	3	3	2	-	2	2	-	-	-	-	-	-	-	-	-	-	-
56	2	-	2	1	1	-	2	3	2	-	1	-	2	1	3	2	2	1	3
66	2	1	3	-	-	-	3	-	-	-	3	-	3	-	3	-	2	-	1
74	-	-	-	-	-	-	-	-	-	-	-	-	-	-	-	2	2	2	2
91	-	-	-	-	-	-	-	-	-	-	-	-	-	2	-	2	2	2	2
95	1	0	1	0	1	0	0	1	2	1	1	1	1	1	1	1	1	1	1
100	-	1	1	-	-	1	1	-	-	3	-	3	1	-	-	-	-	-	-
110	1	0	1	1	1	1	1	2	1	2	1	2	1	-	-	-	-	-	-
111	0	3	2	1	1	-	-	1	1	-	-	1	-	-	-	-	-	-	-
124	1	-	2-3	1	1	1	1	1	1	1	1	1	1	1	-	-	-	-	-
125	1	0-1	1	-	2	2	2	-	2	2	2	-	2	2	2	2	2	2	2

190

153	3	1	4	4	3	-	-	4	4	-	-	4	-	-	-	-	-	-	-
175	2	1	2	2	-	1	-	2	2	2	2	-	2	-	-	-	-	-	-
190	-	0-1	-	-	-	0	2	-	-	-	-	-	-	2	1	0	0	0	0
191	-	-	2	-	-	-	-	-	-	-	-	2	-	2	2	-	-	-	-
208	1	0	1	1	1	1	0	1	1	1	1	1	1	2	1	1	2	-	3
218	0	0	0	-	-	-	-	1	2	1	1	1	1	-	-	-	-	-	-
219	1	1	2-3	-	0	0	-	-	0	0	-	0	-	-	-	-	-	-	-
220	-	2	1	0	-	-	-	1	-	-	-	-	-	-	-	1	1	1	0
221	1	1 some 2	2	1	1	3	3	3	3	2	3	2	2	2	-	-	-	-	-
231	NR	0	NR	-	0	-	-	0	0	0	0	0	-	-	-	-	-	-	-
232	NR	2	1	-	1	2	2	1	1	1	1	1	1	1	2	2	2	2	0
264	2, basilaris 5	4	1	1	1	-	-	1	1	1	1	1	1	-	-	-	-	-	-
267 (265)	4	4	-	-	-	3	4	-	-	-	2	-	2	3	1	-	-	-	-
270	0	1-3	NR	3	1	4	3	3	1	1	2	1	1	4	3	2	3	2	2
272	2	0	2	-	1	-	-	1	1	1	1	1	1	-	-	-	-	-	-
273	1	0	1	2	2	2	1	1	1	1	1	1	1	1	1	1	1	1	1
296	-	1	-	-	-	1	1	-	-	-	-	-	-	1	1	1	1	1	1
307	-	1	NR	1	1	-	-	2	3	2	2	2	2	-	-	-	-	-	-
332	1	0	1	-	-	0	1	-	-	1	-	-	-	1	1	1	1	1	1
361	-	2-3	1	-	-	3	3	-	-	-	-	-	-	0	2	-	1	-	-
367	1	1	1	0	0	1	1	2	1	1	2	0	1	1	1	1	1	1	1
385	-	-	-	-	-	-	-	-	-	-	-	-	-	1	-	1	1	-	1
414	-	1	2	-	-	3	3	3	-	3	3	3	3	3	3	-	-	-	-
450	2	4	4	2	3	3	3	2	2	2	2	2	2	1	-	2	2	3	2
473	4	3-4	1	2	2	-	-	4	4	-	4	-	-	-	-	-	-	-	-
514	NR	1 to 2	1	2	2	-	-	3	2	2	1	2	2	-	-	-	-	-	-
528	2	0	1	1	1	1	1	1	1	1	1	1	1	1	1	1	1	1	1
634	1	0	3	3	3	3	-	3	3	3	3	3	3	2	-	-	-	-	-
647	2	-	1	2	2	-	-	2	-	2	-	-	2	2	2	2	3	2	2
657	3	0	2	1	-	1	1	1	1	-	-	3	1	2	2	2	2	2	2
678	1	0	1	-	-	1	-	2	-	2	-	2	-	1	2	-	-	-	-
699	2-3	3	-	-	-	-	-	-	-	-	-	-	-	-	-	-	-	-	-
784	4	1-3	4-5	5	3	-	-	4	5	-	-	-	-	-	5	5	5	-	-
835	4	0-1	2-3	4	4	4	4	3	3	4	4	4	4	4	4	4	4	4	4

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Appendix III: Paleopathology Scores for Evidence of Scurvy

This appendix contains the scores used to assess scurvy in all 48 individuals from Saint-Amé. Tables 37 to 40 contain the macroscopy scores *but* scores regarding ribs (flaring, porosity and fractures) can be found in Appendix IV and Table 41 contains the radiography scores. Data was collected following the protocol outlined in Chapter 5, Section 5.3.3.

A – Macroscopic scores

TABLE III-1. Cranial scores for the presence or absence of scurvy, Part 1. Y = yes, N = no, - = cannot assess, NB = new bone, NR = not recorded.

	Cranial vault			Sphenoid			Orbit	
Juve-nile	Porosity	New bone	Endo-cranium	Greater wing	Lesser wing	Rotundum	Roof	Zygomatic internal
6	N	N	N	-	-	-	Y	Y
7	Y	N	N	Y NB; N porosity	Y	N	N	Y
18	-	N	Y plaques	-	-	-	-	-
37	Y	Y	N	N	NR	NR	N	-
56	Y	Y	N	Y	-	N	-	N
66	Y	Y	N	-	-	-	Y	-
74	-	-	-	-	-	-	-	-
91	-	-	-	-	-	-	-	-
95	Y	Y	N	Y	N	Y	N	Y
100	-	-	-	-	-	-	-	-
110	Y	Y	Y	N	-	Y	Y	Y
111	Y	N	N	Y	N	N	N	N
124	Y	N	Y	Y	NR	Y	Y	Y
125	Y	Y	Y some	Y	N	N	N	Y healed
153	Y	N	Y some	N	N	N	N	N
175	N	N	N one piece with grooves and 3-4 fragments with plaques; Y occipital	Y	NR	NR	-	N
190	-	-	-	-	-	-	-	-
191	-	-	-	-	-	-	-	-
208	Y	N	N	N (fine NB)	Y	N	N	Y
218	N	N	N	N	-	N	N	Y
219	Y	Y	N	N	-	N	-	-
220	-	-	-	-	-	-	-	-
221	Y (all fine NB)	Y	N	N	Y	Y	Y	Y
231	Y	Y	N	-	N	-	N	Y orbit only
232	Y	N	N	N	-	N	N	N
264	Y	Y	Y	-	-	-	Y	-

267 (265)	N	N	N	N	-		-	-
270	N	N	N	-	N	-	N	N
272	N	N	N one meningeal impression	N	N	N	N	N
273	Y	N	Y lots of grooves/ plaque	N	-	-	Y	Y
296	-	-	-	-	-	-	-	-
307	-	-	-	-	-	-	-	-
332	Y	N	N	-	Y	-	N	-
361	-	-	-	-	-	-	-	-
367	N	N	Y	-	N	N	N	-
385	-	-	-	-	-	-	-	-
414	-	-	-	-	-	-	-	-
450	N	N	N	-	-	-	-	-
473	N	N	N	N	-	-	N	N
514	Y	Y	N	N	Y slight	Y	Y slight	Y
528	N	N	N	Y slight	N	Y	N	Y
634	N	N	N	N	NR	-	N	N
647	Y	N	N	-	Y	-	N	N
657	Y	N	N	-	Y	-	Y slight	-
678	Y	Y	-	-	-	-	-	-
699	N	-	N	-	-	-	-	-
784	Y	Y	N	Y slight	-	-	-	N
835	-	-	-	N	-	-	N	N

TABLE III-2. Cranial scores for the presence or absence of scurvy, Part 2. Y = yes, N = no, - = cannot assess, NB = new bone. NR = not recorded.

	Maxilla					Mandible	
Juve-nile	Infraorbital foramen	Palate	Alveolar process	Posterior	Palatine palate	Coronoid process	Alveolar process
6	Y	Y	N	Y	-	N	Y
7	Y	N	Y	Y	N	Y	Y
18	-	N	-	-	-	-	-
37	N	-	-	N	N	N	N
56	-	-	-	-	-	Y	N
66	-	-	-	-	-	N	Y
74	-	-	-	-	-	-	-
91	-	-	-	-	-	-	-
95	Y	-	Y	Y	-	Y	Y
100	-	-	-	-	-	-	-
110	-	-	-	NR	N	Y	N
111	N	N	N	N	N	N	N
124	Y	Y	Y	NR	Y	Y	Y slight
125	Y	Y	Y	Y slight	-	Y	N
153	Y	Y	Y	N	N	Y slight	Y
175	-	-	-	NR	-	N	N
190	-	-	-	-	-	-	-

191	-	-	-	-	-	-	-
208	Y	Y	Y	Y	N	Y	Y
218	Y light expression	Y	Y	NR	Y	Y	N
219	-	-	Y	Y	-	-	Y
220	-	-	-	-	-	-	-
221	-	-	-	Y	-	Y	N (incisive only)
231	-	Y	Y	-	-	N	Y
232	N	N	Y slight	NR	N	N	N
264	N	Y	Y	-	-	-	Y
267 (265)	-	-	-	-	-	-	-
270	-	-	Y	NR	-	Y	N
272	-	-	N	N	-	-	N
273	Y	-	Y	Y	-	Y	Y
296	-	-	-	-	-	-	-
307	-	-	-	-	-	-	-
332	-	-	-	-	-	-	-
361	-	-	-	-	-	-	-
367	-	-	-	NR	-	-	-
385	-	-	-	-	-	-	-
414	-	-	-	-	-	-	-
450	N	N	Y	-	-	Y slight	N
473	N	-	N	NR	-	N	N
514	Y	N	Y	Y	N	Y slight	Y
528	Y	Y	Y	Y slight	Y	Y slight	Y
634	N	N	N	Y some	N	N	N
647	Y	Y	Y	Y	-	N	Y
657	-	-	-	-	-	Y	N
678	N	N	Y	Y slight	-	-	-
699	-	-	-	-	-	-	-
784	Y	-	N	N	Y	Y	Y
835	-	-	-	-	-	N	N

TABLE III-3. Axial scores for the presence or absence of scurvy. Y = yes, N = no, - = cannot assess, NB = new bone.

	Scapula			Ilium (internal/external surface)
Juvenile	Supraspinous fossa	Infraspinous fossa	Ventral aspect	
6	Y	N	N	N
7	Y	N	Y	N
18	-	-	-	-
37	Y	N	N	N
56	N	N	N	N
66	-	-	-	-
74	-	-	-	-
91	-	-	-	-
95	Y	Y	N	N
100	-	-	-	N
110	Y	N	N	N
111	N	-	-	N

124	Y	Y	N	Y slight
125	Y	N	N	N
153	N	N	N	-
175	N	N	N	-
190	-	-	-	N
191	-	-	-	-
208	N	N	N	N
218	-	-	-	-
219	Y	Y	N	Y
220	Y	N	Y	-
221	Y	N	Y	Y (internal)
231	N	-	-	-
232	N	N	-	N
264	N	Y	Y	-
267 (265)	-	-	-	Y
270	N	N	N	-
272	Y	-	-	-
273	N	N	N	Y
296	-	-	-	N
307	N	N	N	-
332	-	-	-	N
361	-	-	-	N
367	N	N	N	Y
385	-	-	-	-
414	-	-	-	N
450	N	-	-	N
473	N	N	N	N
514	Y	N	N	-
528	Y slight	N	N	N
634	N	N	N	N
647	Y slight	N	N	-
657	Y slight	Y	N	Y slight
678	-	-	-	Y slight
699	-	-	-	-
784	N	N	N	-
835	N	N	N	N

TABLE III-4. Limb bone scores for the presence or absence of scurvy. Y = yes, N = no, - = cannot assess, L_ = left _ , R_ = right, H = humerus, R = radius, U = ulna, F = femur, T = tibia, Fi = fibula.

	New Bone		Metaphyseal Porosity		Fractures in metaphysis		
Juvenile	Arm (humerus, radius, ulna)	Leg (femur, tibia, fibula)	Arm	Leg	Y/N	Location	Cupping of weakened metaphyses
6	-	Y (LF)	-	Y (F,T) hip + ankle	N	-	N
7	Y (R,U)	Y (RT,RFi)	Y (RR) wrist	Y (F,RFi,LT) knee + ankle	Y	LF	Y (LU)
18	-	-	-	-	-	-	-
37	Y (RH)	-	N	-	N	-	N
56	Y (LR)	Y (F,LT, RFI)	N	Y (LT)	N	-	Y (LT,LFi)
66	Y (LU,LR)	Y (LF, LT, LFi)	N	Y (LF,LT) hip + knee	N	-	N
74	-	N	-	Y (T) knee + ankle	N	-	Y (F)
91	-	N	-	Y (T,Fi) knee + ankle	Y	RT	N
95	Y (H, R)	Y (LF)	Y (RU) wrist	Y (F,T) hip + ankle	N	-	N
100	Y (U,RR)	Y (LF)	Y (U,RR) wrist	Y (LF) hip	N	-	N
110	N	Y (T)	N	Y (LF [enlarged], T) knee, ankle	Y	R, LF, T	Y (RU)
111	N	-	N	-	N	-	N
124	Y (LR)	Y (RF)	Y (U,RR) wrist	N	N	-	N
125	N	Y (T)	Y (R) wrist	Y (F,T,Rfi) hip, knee, wrist	Y	RF, RT	Y Slight (F)
153	N	-	N	-	N	-	N
175	Y (H, U, R)	-	N	-	N	-	N
190	-	Y (RFi)	-	Y (F,T) knee, ankle	N	-	N
191	Y	Y (ostemyelitis plaques)	Y (osteomyelitis/ destruction)	Y (osteomyelitis/ destruction)	N	-	N
208	Y (All)	Y (all)	N	N	N	-	Y (LU)
218	Y (U, LR)	-	N	-	N	-	Y (LU)
219	Y (RU)	-	Y (LH) shoulder	-	Y (remodelled)	-	N

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220	N	Y (T,RFi)	N	Y (T,Rfi) knee, ankle	N	-	N
221	Y (U,R)	N	Y (H) shoulder	N	N	-	N
231	Y (All present)	-	Y (H) elbow and shoulder	-	N	-	N
232	Y (H, LU,R)	Y (T,RFi)	Y (LH, LU, R) shoulder, elbow enlarged, distal radii	Y (RF, T) knee, ankle	Y	R	N
264	Y (H,U,R)	-	Y (H,U,R) shoulder, elbow, wrist	-	Y	LH	Y (RU)
267 (265)	N	Y (F)	Y (LR) wrist	Y (Femora) hip strut/slit spacing is large	N	-	Y (LU, RR)
270	Y (H,U,R)	Y (T)	N	Y (RF)	Y	LU, LR	N
272	N	-	N	-	N	-	N
273	Y (F,RR)	Y (F,T,LFi)	N	Y (F,T,Rfi) knee, ankle	Y	-	N
296	-	Y (F,T,Fi)	-	N (F,T,Fi) healing slit/strut	N		N
307	Y (U,R)	-	N	-	N		Y (R)
332	N	N	N	Y (F,T)	Y 2 depressions	-	N
361	-	Y (F, RT)	-	Y (F) hip	N		N
367	Y (H,LU,R)	Y (F,T,FI)	Y (LU,R) elbow, wrist	Y (LT) knee	N		Y (RFi)
385	-	Y (LT)	-	N	N		N
414	Y (U,R)	Y (F,LT)	Y (U) elbow	Y (F) hip	N		N
450	N	N	N	N	Y	RT	N
473	N (osteomyelitis /periostitis)	Y (T)	N	Y (Fi) knee, ankle	N	-	Y (LR, F, T, Fi)
514	Y (R)	-	Y (LH, U, R) elbow, wrist	-	N	-	Y (RR)
528	Y (U,R)	Y (T,Fi)	N	Y (F,T,Rfi) knee, ankle	N	-	N

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634	Y (H)	-	Y (U,R) wrist	-	N	-	N
647	Y (RH, LR)	Y (T,Fi)	N	N	N	-	N
657	N	Y (H,RT,Fi)	N	N	N		N
678	Y (RH,RU,RR)	Y (F)	N	N	N	-	N
699	-	-	-	-	-	-	-
784	Y (LH)	-	Y (LH)	-	N	-	N
835	N	N	Y (RH) shoulder	N	Y	LT	Y (RF)

B – Radiographic Scores

TABLE III-5. Radiographic scores for the presence or absence of scurvy. Prox = proximal, Dis = distal, - = cannot assess. Y = yes, N = no, - = cannot assess, L_ = left _ , R_ = right, H = humerus, R = radius, U = ulna, F = femur, T = tibia, Fi = fibula

								Epiphyses	
Juve-nile	Generalised (shaft) osteopenia	Cortical thinning	White line of Fraenkel	Scurvy line	Pelkan spur	Corner sign	Metaphyseal fractures	Wimberger's ring	Osteopenia
6	Y (RFi)	Y + tunneling (RT,RFi)	Y (F,Lfi)	Y	N	N	N	-	-
7	Y (Fi)	Y (RT, tunnelling Fi)	N (except: LU)	Y slight shadow on LU, LR, T,Fi)	N	N	N	-	-
18	-	-	-	-	-	-	-	-	-
37	N	Y (RH)	N	N	N	N	N	-	-
56	Y (LH,LU,LF, LT,Lfi)	Y (H,LU,LR,F ,T,Fi)	Y (RH,LF)	Y (RH,LF)	Y (RH)	N	N	-	-
66	Y (FI)	Y (LU,LR,LF, LT,LFi)	Y (LU,LR,LF, LT,Lfi)	Y (LR,LF,LT)	Y (LF)	N	N	-	Y (LF,LT)
74	Y (All observable long bone)	Y (Fi)	Y (All observable long bone)	Y (All observable long bone)	N	N	N	-	-

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91	Y (Fi)	Y (FI)	N	N	N	N	N	-	-
95	Y (Fi)	Y (LFi)	Y (LU)	Y	N	N	N	N	Y (LF)
100	Y (RU, RR)	N	Y (RU,RR,LF)	N	N	N	N	-	-
110	Y (RFi)	Y (RFi)	Y (RH, RU, RR, RT, RFi)	Y (RH, RT)	N	N	N	N	N
111	N	Y	Y	N	N	N	N	N	Y (RF)
124	Y (LU)	Y (H, RR)	Y (all observable bones)	Y (RH, RU, RR)	Y (RU)	N	N	-	-
125	Y (LH, LR, F,T, Rfi)	Y (RFi)	Y (R)	Y (RR, RF,RT, Rfi)	Y (RR)	N	Y (LR)	N	Y (LT)
153	Y (RU)	Y (all observable long bones)	N	N	N	N	N	-	-
175	Y (all observable long bones)	Y (LH)	Y (LH)	N	N	N	N	-	-
190	Y (Lfi)	Y (LF, RT, Lfi)	Y (RF, T, R)	N	N	N	N	N	Y (RF)
191	Y (all observable long bones)	Y (all observable long bones)	N	N	N	N	N	-	-
208	Y (H, R, F,T)	Y (H, LR, LF, RT)	Y (U, LR, LF)	Y (LH, LU, LR, F)	N	N	Y (LF)	-	-
218	N	N	Y slight (LU)	N	Y (possible RH)	N	Y (RU)	-	-
219	Y	N	Y (LH slight)	N	Y (RR)	N	N	-	-
220	Y (RH, T)	Y (RH,RT)	Y (all observable bones)	Y (RH)	N	N	N	-	-
221	Y (all	Y (all	N	Y (LU,LR)	N	N	N	-	-

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	observable long bones)	observable long bones)							
231	Y (H,R)	Y (LH,R)	Y (H,RU)	N	N	N	N	-	-
232	Y (RU,R, Rfi)	Y (RU, RR, RFi)	Y (U,RR,F,LT)	N	Y (R,RF)	N	N	-	-
264	Y (U,R)	Y (U,R)	Y (H,RU,R)	N	Y (RU,R)	N	N	-	-
267 (265)	Y (LU)	N	Y (LU)	N	N	N	N	-	-
270	Y (LH, LU, LR,F,T,FI)	Y (RR,F,T,Fi)	Y (LH,R,LT, RFI)	N	N	N	Y (LF)	-	-
272	N	N	Y (H,LU,RR)	Y (RH,RR)	Y (LU)	N	N	-	-
273	N	N	Y (LH,LU,RF, LFi)	N	N	N	N	-	-
296	Y (Fi)	Y (all observable long bones)	N	N	Y (RFI)	N	N	N	Y (all observable)
307	Y (all observable long bones)	Y	Y (RH, RU,LR)	N	Y (RH,LU,RR)	N	N	-	-
332	Y (F,T,LFi)	Y (F,T,LFi)	Y (LFi)	N	N	N	N	-	-
361	-	-	-	-	-	-	-	N	N
367	Y (U,LR)	Y (U,R, LF, RT)	Y (LH,LT)	Y (RR)	Y (LH slight, LT)	N	Y (RR)	-	-
385	Y (all observable long bones)	Y (all observable long bones)	Y (LT,LFi)	Y (LFi)	Y (LFi)	N	N	-	-
414	N	N	Y (RU,R, F)	Y (RU,R)	Y (RU, R)	N	N	-	-
450	Y (LT)	N	N	N	N	N	N	N	Y (LT)
473	Y (Fi)	Y (H,RR,Fi)	Y (LH,F,T,Fi)	Y (LFi)	Y (LR)	N	N	N	Y (F)

514	N	N	Y (RR)	N	N	N	N	-	-
528	Y (LU,LR, F,T,Fi)	Y (Fi)	Y (LU,LR, F,T, Fi)	Y (LU, F,T, Fi)	Y (LU)	Y (LU)	Y (LH)	-	-
634	Y (all observable long bones)	Y (all observable long bones)	Y (LH,RU,R)	Y (LH,RU,LR)	Y (LR)	N	N	N	Y (RH)
647	Y (RH,RU, LR,Fi)	Y (RH,RU, LR, F, T, Fi)	Y (RH,RU, LR, F, RT)	Y (RH,RU, LR, F, RT)	N	Y (RU)	Y (LR)	-	-
657	Y (RU, T, Fi)	Y (RU, T, Fi) + Tunneling	N	Y (T)	N	N	Y (T)	-	-
678	N	Y (RH,RR,F)	Y (All observable long bone)	N	N	N	N	-	-
699	-	-	-	-	-	-	-	-	-
784	N	N	N	N	N	N	N	-	-
835	Y (U,R)	Y (H,U,R,F,T)	Y (H,U,T,RFi)	N	Y (LH)	N	N	N	N

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Appendix IV: Paleopathology Scores for Evidence of Rickets

This appendix contains the scores used to assess scurvy in all 48 individuals from Saint-Amé. Tables 42 and 43 contain the macroscopy scores but cranial vault porosity, orbital roof porosity, and porosity of the metaphyses (arm and leg limb bones) can be found in Appendix III. Table 44 contain radiography scores and Table 45 contains the SEM scores. Data was collected following the protocol outlined in Chapter 5, Section 5.3.3.

A – Macroscopic Scores

TABLE IV-1. Cranial and axial element scores for the presence or absence of rickets. Y = yes, N = no, - = cannot assess, NB = new bone.

			Rib				
Juve-nile	Frontal bone bossing	Deformed Mandibular ramus	Porosity	Flare	Sharp Angle Deformity	Fracture at growth plate	Ilium concavity
6	N	N	Y	Y	N	-	N
7	-	N	Y	Y Mix	N	-	N
18	-	-	-	-	-	-	-
37	N	-	N	Y slight	N	N	-
56	-	N	Y some	N	-	N	N
66	-	N	Y	Y	N	Y Possible	-
74	-	-	-	-	-	-	-
91	-	-	-	-	-	-	-
95	N	Y medial/ posterior	Y	Y	N	Y	N
100	-	-	Y	Y	N	-	N
110	N	-	N, only 1	Y some	-	Y	N
111	N	Y medial	-	-	N	-	-
124	-	N	Y	Y	N	N	N
125	-	Y twisted medially	Y	Y	N	N	N
153	N	N	N	Y slight	N	N	-
175	-	N	N slight	N	N	Y	-
190	-	-	-	-	-	-	N
191	-	-	-	-	-	-	-
208	N	N	Y	Y some	N	Y	N
218	-	Y slight medial	Y	Y	Y	-	-
219	-	Y slight	N	Y most	N	-	N
220	-	-	Y slight	N	N	-	-
221	-	N	Y	Y slight	N	-	N
231	-	N	N	N	N	-	-
232	N	-	N	Y some	N	-	N
264	N	-	Y	Y	N	-	-
267 (265)	-	-	-	-	-	-	N
270	-	N	N	N some	N	N	N
272	-	Y	Y slight	Y	N	N	-
273	-	Y	Y	Y	N	-	N

296	-	-	-	-	-	-	N
307	-	N	N	Y some slight	N	-	N
332	-	-	N	Y	-	-	N
361	-	-	N	N	-	-	N
367	-	N	Y slight	Y	N	Y	N
385	-	-	-	-	-	-	-
414	-	-	Y	Y	-	-	N
450	-	N	-	-	N	-	N
473	Y	N	N	N	N	-	N
514	N	Y slight	Y	Y	N	-	-
528	-	Y	Y	Y	N	-	N
634	N	N slight	N	Y slight	N	Y	N
647	N	N	N	Y	N	-	-
657	N	N	N	N	N	-	N
678	-	-	-	-	-	-	N
699	-	-	-	-	-	-	-
784	-	Y	N	Y	N	-	-
835	-	N slight	N	N	N	-	N

TABLE IV-2. *Limb bone scores for the presence or absence of rickets. Y = yes, N = no, - = cannot assess, L_ = left, R_ = right, H = humerus, R = radius, U = ulna, F = femur, T = tibia, Fi = fibula, NR = not recorded.*

	Deformities		Abnormal distal growth plate				Long bone metaphyseal flaring		
Juve-nile	Arm (humerus, radius, ulna)	Leg (femur, tibia, fibula)	Score 2	Score 3-4	Coxa Vara & Flattening of the femoral metaphysis	Thickened long bones	Arm (humerus, radius, ulna)	Leg (femur, tibia, fibula)	Long bone concave curvature porosity
6	-	N	Y (LF,T,Fi)	Y (RF)	N	N	-	N	-
7	Y (RH)	N	Y (all obsevable long bones)	N	N	N	N	N	-
18	-	-	-	-	-	-	-	-	-
37	N	-	N	N	-	N	N	-	-
56	N	N	Y (LFi)	N	N	Y (LU, F, LT, LFi)	Y (RH)	N	-
66	N	N	Y (LFi)	Y (LU,LR,LF, LT)	Y (Coxa Valga) and flattened head (LF)	Y (LU,LR,LF, LT, +L.Fi)	Y (LR) wrist	Y (LF) hip	Unobservable - involucrum - osteomyelitis
74	-	N	Y (all obsevable long bones)	N	-	N	-	N	-
91	-	Y (LT)	Y (all obsevable long bones)	N	-	Y (T, Lfi) (Note: RT, Lfib are puffy)	-	Y (T,Rfi) Knee, ankle	NR
95	N	N	Y (H,F,T,Fi)	Y (U,R)	Y, flat and coxa (RF)	Y (RR, LT)	Y (RH)	N	NR
100	N	N	Y (U,RR)	N	N	N	Y (LU,RR)	N	-
110	N	Y slight (T)	Y (RH,R,RF, RT,Fi)	Y (LF,LT)	N	Y (RT, Fi)	Y (RU)	N	N
111	N	-	N	N	-	N	N	-	-
124	N	N	N	N	N	N	N	N	-

125	N	N	Y (LH,LU, R,T,Fi)	Y (F)	N	Y (LF)	Y (LH) elbow	-	-
153	N	-	-	Y (RH)	-	N	N	-	-
175	Y (RR)	-	Y (LR)	N	-	Y (R)	Y (R)	-	N
190	-	Y (T, LFi)	N	N	N	Y (LT,LFi)	-	Y (healed Lfi, maybe RF,LT) Knee, ankle	Y (Lfi)
191	N	Y (RF clear)	Y (RU)	Y (LF)	-	Y (RU,RF)	N	N	Y (RF)
208	N	Y(RF, LF min)	Y (RH)	Y (F)	N	N	N	N	N
218	Y slight (RH)	-	N	N	-	Y (RH)	N	-	N
219	N	-	Y (LH, RR)	N	-	N	N	-	-
220	N	N	N (possible velvety look)	N	-	Y (RFi)	N	N	-
221	Y (H)	N	Y (U,LR, RF)	N	-	N	Y (LH,LR) Shoulder, elbow, wrist	N	Y (LH)
231	N	-	Y (RR)	N	-	Y (RU, R)	Y (R)	-	-
232	N	N	Y (LH,RU, RR,T)	N	N	Y (RFi, F)	Y (R) Wrist	N	-
264	Y (RR, LR)	-	Y (LU, RR)	Y (RU,LR)	-	Y (slight RH, RU, LU)	Y (H,LU,RR) Elbow Wrist	-	Y (RR)
267	Y (LU,LR)	Y (RF)	N	Y (LU,LR)	Y (F); LF coxa valga	Y (LU,F)	Y (LU) Wrist	Y (F) Hip	Y (RF)
270	N	Y (RT, Rfi)	Y (RR, RT, Fi)	N	N	Y (RH, RR, RF, Fi)	Y (RR) Wrist	Y (F,T) Hip, Knee, Ankle	N
272	N	-	Y (LR)	N	-	Y (maybe RR)	Y (RH, thickened	-	-

273	Y (LU slight)	N	Y (T,Fi)	N	N	Y (LU, LT)	RR) Elbow, Wrist Y (R clear) Wrist	Y (Fi) Ankle	Y (LU)
296	-	N	N	N	Y (F) + flat head (RF)	Y (LF)	-	Y (F,RT,Fi) Knee, ankle	-
307	Y (H, R)	-	Y (RH)	Y (U,R)	-	N	N	-	N
332	N	N	Y (LFi)	N	-	N	N	Y (LF) Knee	-
361	-	N	Y (F)	N	N	Y (F)	-	N	-
367	N	Y (Fi clear)	N	N	Y	Y (LH, LU, R, RF,LT, LFi)	Y (LH,LU) Elbow, Wrist	Y (RF,LFi) hip, ankle	NR
385	-	N	Y (LFi)	N	-	N	-	N	-
414	Y (RR)	N	Y (RU, R)	N	N	N	N	N	NR
450	N	N	-	-	-	Y (LT)	N	N	-
473	N	Y (T)	Y (H,fi)	Y (F,RT)	N	Y (H,LR)	N	Y (F,T,LFi) Knee, ankle	N
514	N	-	Y (all obsevable long bones)	N	-	Y (RR, RU)	Y (LR) Wrist	-	-
528	N	Y (Fi)	Y (RH, U, RR)	N	N	N	Y (LU) Wrist	Y (LF) Knee	NR
634	N	-	Y (RU,RR)	Y (LU,LR)	-	N	Y (RH,U) elbow, wrist	-	-
647	N	N	Y (RU, RF, T)	N	Y	N	N	N	-
657	N	Y (RF slight)	Y (LT,Lfi)	Y (RH)	N	Y (U, RF, T, Rfi)	Y (RH,LH) Shoulder, elbow	Y (T) ankle	N
678	Y (RH)	N	Y (RH,RU,RR,	N	Y (LF)	Y (all bones)	Y (RU,RR) Wrist	Y (LF) Knee	Y

			LF)						
699	-	-	-	-	-	-	-	-	-
784	N	N	-	-	-	N	N	N	-
835	Y (H)	Y (RF)	N	N	Y, flattened (RF)	Y (RF)	N	N	N

B – Radiographic Scores

TABLE IV-3. Radiographic scores for the presence or absence of rickets. Y = yes, N = no, - = cannot assess, L_ = left _ , R_ = right, H = humerus, R = radius, U = ulna, F = femur, T = tibia, Fi = fibula.

	Trabecular coarsening		Fraying at the growth plate	Cortical thinning	Thick cortex with periosteal apposition	Rib, loss integrity at the sternal end	Osteopenia	
Juve-nile	Overall	Distal metaphysis					Growth plate and metaphysis long bone	Epiphyses
6	N	N	N	Y (Rfi), no tunneling	Y	Y	Y (F,T,Fi)	-
7	N	N	Y (LH)	Y (RT, Fi, possible tunneling of Fi)	Y	N	Y (U,R,Fi)	-
18	-	-	-	-	-	-	-	-
37	N	N	Y slight (RH)	Y (RH)	Y	N	Y slight (RH)	-
56	Y (H,LU)	Y (LH)	N	Y (H,LU,LR,F,T,Fi)	N	-	Y (H,LU,LF,LT,Lfi)	-
66	Y (LU slight ,LR,Lfi)	Y (LR,Lfi)	N	Y (LU,LR,LF,LT,LFi)	Y	Y	Y (LU,LR,LF,LT,Lfi)	Y (LF,LT)
74	N (possible in Lfi but damaged)	N	N	Y (Fi)	N	-	N	-
91	N , heavily damaged	Y (RT)	N	Y (FI) + tunneling	Y	-	Y (RF, T, LFi)	-
95	N	N	N	N	Y	N	N	Y (LF) slight
100	N	N	N	N	Y	-	Y (RU)	-
110	N	N	N	Y (RFi)	Y	N	Y (RH, RT, Fi)	N
111	Y	Y	N	N	N	-	Y slight	Y slight in one

124	Y (all observable bones)	Y (H,RU, R)	N	N, possible	N	-	Y (all observable bones)	-
125	N	N	N	N	N	N	Y slight (R)	Y (LT)
153	N	N	N	Y (all observable long bones)	N	Y	Y (RH, RU)	-
175	N	N	N	Y (LH)	Y	Y	Y (LH)	-
190	Y (All observable bones)	Y (All observable bones)	N	N	Y	-	Y slight (All observable bones)	Y (RF)
191	Y, (all observable bones)	Y (All observable bones)	N	Y (all observable long bones)	Y	N	Y (all observable bones)	-
208	Y (H, LU, LR)	Y(H,LU,L R)	N	Y (H, LR, LF, RT)	Y	N	Y (LU, LR, F)	-
218	N	N	N	N	N	N	N	-
219	N	N	N	N	N	N	N	-
220	N	N	N	Y (RH,RT)	N	Y	Y (all observable bones)	-
221	N	N	N	Y (all observable long bones)	N	Y	Y (all observable bones except femur)	-
231	N	N	N	Y (LH,R) but damaged	N	N	N	-
232	Y (F slight; Rti slight, R, H, Rfi)	Y slight (RR, RH)	Y (RF)	Y slight (RU, RR, RFi)	N	Y	Y (Rfi, Ti, F, RH, RU, RR)	-
264	Y (RH)	Y (RH)	N	Y (U,R)	Y	Y	Y (H, U, R)	-
267 (265)	Y (F)	Y (F)	Y (RU)	N	Y	-	Y (RU,F)	-
270	Y slight (H,LR,F,T, RFI)	Y slight (H,F,RT)	N	Y (RR,F,T,FI)	Y	Y	Y (LH,LU,LR,F, T, RFI)	-
272	N	N	N	N	N	Y	N	-
273	N	N	N	N	N	N	N	-
296	Y (all observable	Y (F,T, LFI)	N	N	N	-	Y (RFI)	Y (2/4 affected)

	long bones)							
307	Y (H,LU,LR)	Y LH, LR	N	Y (H, R)	N	N	Y (H,LU, R)	-
332	N	N (slight distal RF)	N	N	N	N	Y (LF,RT)	-
361	-	-	-	-	-	N	-	N
367	N	N	N	Y (LR,LU)	N	Y	Y (LH,LU,R)	-
385	Y (LF,LT)	Y (LF)	N	N	N	-	Y (all observable long bones)	-
414	N	N	N	N	Y	N	Y (U,R,F)	-
450	Y (U)	N	N	N	N	-	N	Y (LT)
473	Y slight (H,LR,F,RT)	Y slight (H,LR,F,T, RFi)	N	N	Y	Y	Y (F,T,Fi)	Y (F)
514	N	N	N growth plate damaged	N	N	N	N	-
528	Y (LH) slight	Y (LH) slight	N	N	Y	Y	Y (LU,LR,Fi)	-
634	Y (all observable long bones)	Y (all observable long bones)	N	Y (LR)	Y	Y some	Y H but other bones, unlikely	Y (RH)
647	Y RH, LR	Y RH	N	Y (RH,RU, LR, F, T ,Fi), Maybe tunneling of Lfi	N	N	Y (RH,RU,LR,Fi)	-
657	Y (H,RU, RF,T,FI)	Y (H,RU,T,FI)	N	Y (RU, T, Fi) + Tunneling in Fi,	Y	-	Y (H,RU, LF, T,Lfi)	-
678	N	N	N	N	Y	-	N	-
699	-	-	-	-	-	-	-	-
784	N	N	N	N	Y	N	Y (all observable long bones)	-
835	Y slight (H,U,F, Rfi)	Y (H,RU,T,Fi)	N	Y (H,U,R,)	Y	-	Y (all observable long bones)	N

C- Scanning Electron Microscopy Scores

TABLE IV-4. SEM section scores for the presence or absence of rickets. Y = yes, N = no, - = cannot assess.

Juve-nile	Sample #	Preservation	Buired osteoid remnant (including seams)	Area of skipped mineralisation	Poorly mineralised			Howship's lacunae
					Osteons	Osteocyte	Borders	
6	2	Poor	N	N	N	N	Y some	Y
7	3	V. Poor	N	N	N	N	Y	N little
37	16	OK	N	N	N	N	Y some	Y
91	11+12	V. Poor	-	N	-	-	N	Y
95	29	V. Poor	-	N	Y	N	-	Y
110	15	Poor, broken apart, ok in some areas while others are poor	N	N	N	N	Y some, others are very white	Y
124	14	V, V. Poor	N	-	-	-	-	N
125	10	V. Poor	N	N	N	N	N	Y
153	23	V. Poor	-	-	-	-	-	-
190	20	V. Poor	-	-	-	-	-	-
208	21	Good	N	N	N	N	Y slight some	N
218	6	V. Poor	N	N	Y	N	N	N
219	26	V. Poor	N	N	Y	N	N borders are taphonomic	N
221	4	V. Poor	N	N	N	N	N	N
232	8	Poor	N	N	N	N	Y	Y
270	1	V. Poor	N	Y	N	N	Y some	Y, some
272	19	Poor	N	N	N	N	Y	N
273	27	V. Poor	N	N	Y, a few	N	Y	Y
307	22	V. Poor	N	Y	N	N	Y	N
414	28	Good	N	N	Y	Y	N	Y
473	5	Good	Y	Y	Y	Y	Y	N

514	13	Good (mostly)	N	N	Y, 1 spot at least	N	Y	Y
528	7	Good (mostly)	N	N	N	N	Y	Y
634	24	Poor/good in different spots	Y	N	Y	N	N	N
647	25	V. Poor	Y	N	N	N	Y	Y
678	17+18	Poor	N	N	N	N	Y	Y
835	9	Good	Y	N	Y	N	Y	N