AN INVESTIGATION OF THE PREVALENCE OF RICKETS AMONG SUBADULTS FROM THE ROMAN NECROPOLIS OF ISOLA SACRA, ITALY

AN INVESTIGATION OF THE PREVALENCE OF RICKETS AMONG SUBADULTS FROM THE ROMAN NECROPOLIS OF ISOLA SACRA (1st TO 3RD CENTURIES AD), ITALY

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ABSTRACT

The earliest written reference to rickets comes from Soranus of Ephesus, a Roman medical writer of the early second century AD who describes a high number of children in Rome suffering from rachitic deformities. The current study utilizes two independent forms of evidence, skeletal and historic, to study the prevalence of rickets among the human subadult skeletons of the 1st to 3rd century Imperial Roman necropolis of Isola Sacra. The necropolis of Isola Sacra represents the remains of individuals who inhabited Imperial Rome's key maritime port, *Portus Romae* on the Mediterranean coast 23 kilometers west of Rome. One hundred and eighty-two subadult skeletons were examined morphologically and radiographically to search for diagnostic indicators of rickets.

Fifteen percent (27/182) of Isola Sacra subadults, birth to 15 years, show rachitic traits with a wide range of morphological presentation. Most individuals suffered from hyperplastic rickets, less likely due to malnutrition. All age categories and burial types show rachitic traits. No association was found between age and the appearance of rickets. No association was found between burial type and the appearance of rickets.

Roman cultural practices, social values and socioeconomic status of the populace using the necropolis may have predisposed the subadult population of Isola Sacra to rickets. Low maternal vitamin D is likely a strong contributing factor to rickets prevalence in the Isola Sacra sample and was the result of socio-cultural factors influencing maternal vitamin D intake.

For my Baba, Cassie Mischuk.

The great matriarch of our family who taught me generosity, independence, to always be yourself, say what you mean, mean what you say and not to take crap from anyone.

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Chapter 1

INTRODUCTION

This thesis is an investigation of the prevalence of rickets, among a sample of children from the classical Roman period. For this purpose, the human subadult skeletons of the 1st to 3rd century Imperial Roman necropolis of Isola Sacra were examined for physical evidence of rickets. The necropolis of Isola Sacra represents the remains of individuals who inhabited Imperial Rome's key maritime port, *Portus Romae* on the Mediterranean coast 23 kilometers west of Rome, after the beginning of the 2nd century A.D. One hundred and eighty-two subadult skeletons were examined morphologically and radiographically to search for diagnostic indicators of rickets.

The earliest written reference to rickets comes from Soranus of Ephesus, a Roman medical writer of the early second century A.D. Soranus describes the symptoms of rickets, indicating a high number of children suffering from deformed spinal columns and legs (Temkin 1956). He wrote "if no one supervises the movement of infants the limbs become distorted in most cases" (Colón & Colón 1999:57).

> When the infant attempts to sit and to stand, one should help it in its movements. For if it is too eager to sit up too early and for too long a period, it usually becomes hunchbacked. If, moreover, it is too prone to stand up and desirous of walking, the legs become distorted in the region of the thighs (Colón & Colón 1999:57).

Later in the second century, the Roman physician Galen also described a number of rachitic skeletal deformities in infants and young children, specifically knock-knees, bowlegs, funnel-shaped chests and the pigeon breasts (Steinbock 1993). Galen states that it is not good to coerce children to walk "lest their legs become bent" (Colón & Colón 1999:59). There is little, if any, physical evidence of rickets from Roman times despite the descriptions of 'epidemic' rates by classical authors. The classical writings are suggestive but inconclusive.

The physical evidence of rickets in the Isola Sacra skeletal sample was integrated with an extensive survey of the classical literature regarding Roman childcare practices concerning the health and well being of Roman children. It is the goal of this thesis to go beyond a biomedical approach to disease, to search for biocultural explanations for the prevalence of rickets in past populations. It is essential to transcend a biomedical methodology of cause and effect which treats mainly the symptoms of a disease, to determine the underlying biocultural factors that lead to disease. Childcare practices are intimately linked to the cultural norms and values in a given society. The ultimate causes of disease are social, cultural and socioeconomic in nature and it is these factors that have significantly influenced the prevalence of rickets in both past and present populations.

In order to demonstrate that biocultural factors were directly contributing to the presence of rickets, a reliable and precise estimate of the prevalence of the disease in the Roman population of Isola Sacra was needed. This involved choosing a method of identifying cases of rickets that could be standardized for

inter and intra observer variation. Skeletal abnormalities typically attributed to rickets were assessed individually in order to arrive at a differential diagnosis of the disease in archaeological human skeletal samples. Lesions associated with rickets were identified through a search of the literature. Careful attention to anatomical detail, combined with knowledge of pathological processes that affect bone tissue, and knowledge of the range of morphological variation present in subadult skeletal remains were utilized to determine rachitic individuals. Rachitic morphological traits exhibiting consistency, with no overlap with other diseases or problems associated with age were emphasized and deemed primary traits. Those traits with less than optimal characteristics were given secondary consideration.

Rickets is a disease of infancy and childhood and is fundamentally a failure of osteoid mineralization. Environmental rickets occurs as a result of insufficient vitamin D deposition due to a lack of exposure of the skin to sufficient sunlight and/or to dietary inadequacy (Aufderheide & Rodríguez-Martin 1998). During infancy and childhood, rapid bone growth and turnover occurs creating the potential for considerable buildup of unmineralized osteoid (Ortner & Mays 1998). As a result, in individuals with rickets the bones lack mechanical strength, and are easily deformed when subject to weight bearing or muscular tension (Ortner & Mays 1998).

Rickets is most commonly the result of vitamin D deficiency, but also can occur through vitamin D malabsorption, errors in metabolism, receptor and/or

collagen defects, as well as calcium and/or phosphate deficiency. Other possible causes of rickets were researched to establish cause, skeletal expression, and age at onset, in order to determine ways in which they could be distinguished from vitamin D deficiency rickets. The frequency with which these diseases occur in modern populations was gathered in order to determine their probability and prevalence. Other potential causes of the lesions were considered, particularly, scurvy, and iron deficiency anemia. As all can be associated with malnutrition, it is possible that more than one of these diseases can occur in the same individual, or group of people (Ortner and Ericksen 1997; Aufderheide & Rodríguez-Martin 1998). It is therefore necessary to delineate and define the pathological features associated with each disease, in order to differentiate between these three conditions, and other pathological bone entities present.

Chapter 2 reviews the pathological basis of rickets including the sources and metabolism of vitamin D, the contribution and metabolism of cutaneous versus dietary sources of vitamin D, the role of the liver and kidneys, the calcium and phosphate transport systems, the consequences of vitamin D depletion and the complications of rickets. Genetic forms of rickets are researched to establish cause, skeletal expression and frequency with which they appear in modern populations to differentiate them from deficiency rickets. Chapter 3 is a brief history of rickets from antiquity to modern day. Cultural, socioeconomic and environmental trends through the ages are highlighted. Chapter 4 illustrates the

skeletal manifestations of rickets and osteomalacia. Modern methods of diagnosis of rickets are explored in order to assist in the diagnosis of the Isola Sacra individuals. Chapter 4 introduces other deficiency diseases such as scurvy and iron deficiency anemia which can have similar skeletal manifestations. Criteria to establish a diagnosis of rickets in an archaeological population are established as well as criteria to distinguish between rickets and other deficiency diseases. Chapter 5 provides background information about the inhabitants of *Portus Romae* and the burial types and customs found in the Isola Sacra necropolis. Chapter 6 presents the results of the examination of rickets in the Isola Sacra subadult population on a per case basis with photographs and descriptions of each trait. Associations between age, burial type and rickets are ascertained. Chapter 7 is a discussion of the rachitic trends and biocultural causes of rickets in the Isola Sacra subadult population.

Chapter 2

PATHOLOGICAL BASIS OF RICKETS

Rickets is a disease of infancy and childhood and is fundamentally a failure of osteoid mineralization. This may result from insufficient vitamin D deposition due to a lack of exposure of the skin to sunlight necessary for the conversion of a vitamin D precursor to vitamin D, dietary inadequacy, failure to absorb vitamin D during digestion (chronic intestinal disorders), failure by the kidneys to convert vitamin D precursors to vitamin D, or a failure of the kidneys to retain phosphate (chronic renal tubular failure) (Aufderheide & Rodríguez-Martin 1998; Ortner & Mays 1998). The skeletal changes observed in rickets are direct effects of the metabolic disturbance, deformities secondary to the susceptibility and flexibility of the poorly mineralized bone, and retardation of growth (Ortner 2003). During infancy and childhood, rapid bone growth and turnover occurs creating the potential for considerable buildup of unmineralized osteoid (Aufderheide & Rodríguez-Martin 1998; Ortner & Mays 1998). As a result, in individuals with rickets the bones lack mechanical strength, and are easily deformed when subject to weight bearing or muscular tension (Ortner & Mays 1998). Consequently, rickets is most commonly seen during periods of rapid growth, usually between the ages of 6 months and 3 years of age and during puberty (Stuart-Macadam 1989). Rickets is rare before 4 months of age provided that the mother receives enough vitamin D during pregnancy. Vitamin

D passes from the mother to the fetus through the placenta and is stored in the liver of the infant (Ortner 2003). Often, substantial healing of the gross bone deformities occurs some 5-10 years later in life as the rate of growth of the body structures and its vitamin D needs diminish proportionately (Malgosa *et al.* 1996).

An uncommon form of rickets has been described in various parts of Africa where vitamin D stores are adequate but dietary calcium intake is limited (Chesney 2001). Calcium deficiency as the result of poverty and thus access to calcium containing foods such as milk was first described in South Africa (Chesney 2001). Recently, calcium deficiency in infants has been tentatively linked to rickets in Nigerian children who breast-feed for shorter duration (Bishop 1999). Studies have documented calcium deficient rachitic children are older than the usual age of presentation of vitamin D deficiency rachitic children (Pettifor 2002).

2.1.1 *Sources of Vitamin D*

The most important source of vitamin D is exposure to sunlight, not dietary intake (Fraser 1983). Dietary intake is thought to contribute minimally to vitamin D stores. It has been observed that human exposure to ultraviolet radiation is followed by enhanced intestinal absorption of calcium and healing of vitamin D-deficiency states (Haddad & Hahn 1973). Haddad & Hahn (1973) carried out a study to determine the separate contributions of cutaneous production versus dietary ingestion of vitamin D in humans through the measurement of different types of circulating vitamin D in the blood. They found that 84% of circulating vitamin D was derived from non-ingested sources and state that unless the diet is

unusually rich in fish products, ingested sources are not important. Therefore, circulating vitamin D levels appear to reflect endogenous vitamin D production by the skin (Haddad & Hahn 1973; Holick & Adams 1998). Concentrations of circulating vitamin D in plasma vary with season. The highest values occur in summer and the lowest in winter, following the seasonally changing intensity of solar ultraviolet-B radiation in temperate geographical regions independent of the supply of vitamin D in the diet (Fraser 1983). In order to maintain satisfactory circulating vitamin D plasma levels without any input from skin radiation, vitamin D would have to be supplied by mouth in amounts of 12.5 µg or more per day (Fraser 1983).

Both sources of vitamin D, however, have equal biologic potency. If one source is lacking, the body can compensate by using the other (Parfitt 1998; Fitzpatrick *et al.* 2000). Although dietary intake of vitamin D is a relatively unreliable way of supplying the vitamin, when sufficient sunlight for vitamin D synthesis is not available, a dietary source is essential for maintaining adequate vitamin D levels (Fraser 1983). Few foods naturally contain vitamin D. These are egg yolks, fish liver oil, fatty fishes, shrimp, & chicken liver. The best dietary source is fish oil, as fish, unlike mammals, birds, reptiles and amphibians can synthesize their own vitamin D without the use of ultraviolet light (Stuart-Macadam 1989; Holick 2001).

2.1.2 *Metabolism of Vitamin D*

Vitamin D is more accurately classified as a prohormone than a vitamin (Steinbock 1993). Its main function is to preserve calcium and phosphorus homeostasis by increasing the efficiency of intestinal calcium & phosphorous absorption in order to maintain signal transduction, metabolic activities, and neuromuscular function and to promote skeletal mineralization (Steinbock 1993; Aufderheide & Rodríguez-Martin 1998; Holick 2001).

The two types of vitamin D are ergocalciferol (D_2) and cholecalciferol (D_3) (Holick & Adams 1998). Cholecalciferol is produced by the action of ultraviolet light on a precursor molecule, 7-dehydrocholesteral, a substance synthesized by the body and deposited in the skin and is considered a hormone (Saffran 1995; Holick & Adams 1998). Ergocalciferol is produced by ultraviolet irradiation of ergosterol (Mankin 1974a). Ergosterol (or provitamin D_2) is a sterol occurring mainly in yeast and some fish oils which upon irradiation becomes ergocalciferol (Dorland 2000). While ergocalciferol is widely used in therapeutics it is rarely found in nature (Passmore & Eastwood 1986). In adult skin about 50% of the entire cutaneous stores of provitamin D_3 are found in the epidermis with the other 50% residing in the dermis (Holick & Adams 1998). Most of the cutaneous provitamin D_3 synthesis in adults occurs in the actively growing levels of the epidermis, in close proximity to the dermal capillary bed; as a result, changes in temperatures in the surface of the skin do not significantly alter the rate of conversion of provitamin D_3 in humans (Holick & Adams 1998).

The precursors, ergosterol and 7-dehydrocholesteral, have no anti-rachitic properties, but radiant energy breaks their ring structure and bonds to convert their sterols to their active forms (see **table 2.1** below) (Mankin 1974a; Holick & Adams 1998).

TABLE 2.1 - Cholecalciferol and Related Metabolites of Vitamin D (Dorland 2000).

Systematic Name	Vitamin	Abbreviation
7-dehydrocholesteral	Provitamin D_3	
Cholecalciferol	Vitamin D_3	D_3
25-hydroxycholecalciferol	25-hydroxyvitamin D_3	$25(OH)D_3$
1,25-dihydroxycholecalciferol	1,25-dihydroxyvitamin D_3	$1,25(OH)_2D_3$
24,25-dihydroxycholecalciferol	24,25-dihydroxyvitamin D_3	$24,25(OH)_2D_3$

After formation, the provitamin D_3 undergoes temperature-dependent isomerization to vitamin D_3 (cholecalciferol) which at body temperature takes 10 hours to reach completion (Maxwell 1994; Holick & Adams 1998). The vitamin D is then transported in the blood bound to vitamin D-binding protein (Maxwell 1994).

Endogenous synthesis of cholecalciferol from 7-dehydrocholesteral is related to the amount of biologically effective ultraviolet radiation, the duration of exposure of ultraviolet radiation, and the degree of skin pigmentation (Maxwell 1994). With increased pigment concentrations in the skin, the needed exposure time increases from 30 minutes to between 1 and 3 hours, depending on the degree of pigmentation (Maxwell 1994). Prolonged exposure to sunlight does not necessarily increase vitamin D synthesis because once vitamin D is formed in the dermis and epidermis it can either be turned to vitamin D_3 or during exposure to sunlight, absorb a photon of UV-B radiation and isomerize to

biologically inert isomers lumisterol and tachysterol (Holick & Adams 1998). Vitamin D deficiency does not appear to alter provitamin D concentrations in the skin. Adams and coworkers (1982) found when vitamin D deficient patients were exposed to ultraviolet radiation their circulating concentrations of vitamin D and 25(OH)D increased almost identically to that seen for the vitamin D sufficient subjects.

2.1.3 *Metabolism of Cutaneous versus Dietary sources of Vitamin D*

There is a physiological difference in the vitamin D molecules obtained orally compared with those from the skin. Dietary vitamin D is absorbed in the small intestine when fat digestion and absorption are normal and is carried in chylomicrons (lipoproteins that transport dietary cholesterol and triglycerides from the small intestine to tissues after meals) to the liver (Fraser 1983; Passmore & Eastwood 1986). In contrast, cutaneous vitamin D diffuses slowly into the blood so the hepatic uptake and subsequent conversion to 25(OH)D occurs at a slower rate than a comparable amount of dietary vitamin D (Fraser 1983). Consequently, much of the vitamin D from the skin remains outside the liver, protected from the hepatic inactivating metabolism (Fraser 1983). This more gradual supply of vitamin D to the liver allows for a more continuous and prolonged production of 25(OH)D (Fraser 1983). The plasma concentration of this metabolite is then maintained, although the exposure of the skin to sunlight occurs only intermittently (Fraser 1983). The amount of 25(OH)D produced by the liver is not directly proportional to the input of vitamin D. When reserves are depleted, 25(OH)D is made with greater efficiency than when there is an

abundance of vitamin D (Fraser 1983). Fraser (1983) states that it is possible that more vitamin D is shunted down the degradative pathway when it is presented to the liver with chylomicrons lipid than when it arrives from the skin bound to the circulating vitamin D-specific binding protein. Fraser (1983) asserts that the hepatic inactivation of vitamin D acts as a protective mechanism, preventing the accumulation of a potentially toxic molecule as large doses can induce vitamin D toxicity. Extensive irradiation with ultraviolet light does not cause vitamin D toxicity (*ibid.*). Possibly because of this toxicity, hepatic metabolism appears to regard small amounts of vitamin D absorbed from the intestine as a foreign substance and directs it into destructive rather than conservative pathways (*ibid.*). Aging significantly decreases the capacity of the skin to produce D_3 (Rao 1999). However, aging does not significantly affect the intestinal absorption of vitamin D (Holick & Adams 1998:131).

2.1.4 *The Role of the Liver and Kidneys*

In the liver, vitamin D of cutaneous or dietary origin (25-hydroxyvitamin D) is converted in the microsomes to 25(OH)D, the circulating form of the vitamin which is carried on a special transport globulin (Passmore & Eastwood 1986). The use of the term vitamin D without a subscript refers to both vitamin D_2 and vitamin D_3. A lesser known function of the liver is the metabolic inactivation of the vitamin D molecule and excretion of the products in bile (Fraser 1983). The active form of vitamin D is $1,25(OH)_2D$ (1,25-dihydroxycholecalciferol), which is formed only in the kidney by action of a specific mitochondrial hydroxylase on 25(OH)D (Passmore & Eastwood 1986; Holick & Adams 1998). The dihydroxy

metabolite is about 10 times more active than vitamin D_3 and acts more quickly (Passmore & Eastwood 1986). The efficiency of utilization of vitamin D is determined by the liver, not the kidneys, but the liver is not the storage site for vitamin D (Fraser 1983). The metabolite $1,25(OH)_2D$ is the primary hormone acting in the intestine and possibly the bone cells (Stuart-Macadam 1989). It is this hormone that is of critical importance in calcium/phosphate transport and absorption by the intestine (Stuart-Macadam 1989; Saffran 1995).

The three effects of $1,25(OH)_2D$ are: (1)promotion of calcium absorption in the upper small intestine by inducing the synthesis of a specific calcium-binding protein in the epithelial cell. There, it appears to pass to the nucleus of the cell and stimulate production of a specific messenger RNA (Passmore & Eastwood 1986). (2) It acts on bone to mobilize calcium into the circulation but requires the presence of parathyroid hormone (parathormone) (Passmore & Eastwood 1986). (3) $1,25(OH)_2D$ facilitates phosphate absorption by stimulating a separate phosphate transport mechanism in intestinal epithelial cells which is independent of the calcium transport system (Passmore & Eastwood 1986).

The other two hormones responsible for maintaining normal calcium and phosphorous levels in the body are parathormone and calcitonin (Stuart-Macadam 1989). Parathormone is a major regulator of bone metabolism. It promotes the release of calcium from bone to extracellular fluid by activating osteoclasts and inhibiting osteoblasts, indirectly promotes intestinal absorption of calcium and renal tubular reabsorption of calcium and increases renal excretion of phosphates (Dorland 2000). Secretion of parathormone increases when the

level of calcium in the extracellular fluid is low (Dorland 2000). Calcitonin (thyrocalcitonin) is responsible for lowering plasma calcium and phosphate levels, inhibiting bone resorption and acts as an antagonist to parathormone (Dorland 2000).

2.1.5 *The Calcium Transport System*

Calcium absorption occurs in the distal duodenum or proximal jejunum (Mankin 1990). Calcium requires a transport system in order to cross a cell barrier which consists of three parts. Parathyroid hormone first binds to a receptor on the cell membrane and through the action of adenyl cyclase and cyclic adenosine monophosphate, allows calcium to enter the cell and mitochondria to discharge calcium which results in flooding the cell cytosol with calcium ions (Mankin 1990). Next, 1,25-dihydroxyvitamin D acts to enhance the message to synthesize one of several calcium binding proteins (CBP or calbindin or cholecalcin) which transport the calcium across the cell barrier to the extracellular space (Mankin 1990). The concentration of calcium ions is maintained within the narrow limits required to avoid neurologic or cardiac complications or precipitation of calcium salts by a delicate balance between tubular reabsorption of calcium and the deposit or release of the ion from the hydroxyapatite crystal of the bone (Mankin 1990). Lastly, if the cytosol concentration of phosphate rises above a critical level, transport is turned off (Mankin 1990).

2.1.6 *The Phosphorus Transport System*

The management of phosphorus in the body occurs lower in the jejunum (Mankin 1990). Phosphorus kinetics are less complicated than those for calcium as phosphorus makes its way into the extracellular space much more rapidly as absorption from the gastrointestinal tract is not limited (Mankin 1990). The ionized phosphate serves as a buffer system for the blood and is an obligate partner to calcium in either synthesis or breakdown of hydroxyapatite salt of the bone (Mankin 1990). The primary control mechanism for phosphorus is renal. A finely tuned tubular reabsorption mechanism for phosphate is primarily under the control of parathyroid hormone which causes phosphate excretion in the urine (Mankin 1990). Therefore, rickets and its adult form osteomalacia, cause an increase in parathyroid hormone release, a response to lowered calcium, which causes a simultaneous decline in the amount of tubular reabsorption for phosphate (Mankin 1990).

2.1.7 *Consequences of Vitamin D Depletion*

In the absence of sufficient amounts of vitamin D, the following events take place. Lacking the precursors of the biologically active metabolites of vitamin D, insufficient 1,25 dihydroxycholecalciferol is produced. This in turn reduces absorption and transport of calcium and phosphates, ultimately triggering a negative feedback that results in the increased production of parathormone (Stuart-Macadam 1989). Parathormone increases serum levels of calcium and phosphorus by activating osteoclasts to destroy bone, which in turn, serves to release bone minerals. The formation of bone is also increased in a

fruitless attempt to substitute quantity for quality (Stuart-Macadam 1989). Less mineralized bone per unit exists. The trabeculae and cortex have less bone that is surrounded by a layer of unmineralized bone known as an osteoid seam (Mankin 1990). With insufficient vitamin D, the osteoid produced by the osteoblasts and the cartilage produced by the chondroblasts (cartilage forming cells) are not properly mineralized (Stuart-Macadam 1989). The end result is an excess of unmineralized cartilage at the growing ends of bones as well as bones that are smaller, lighter and more susceptible to deformation under pressure (Stuart-Macadam 1989).

2.1.8 *Atrophic versus Hypertrophic Rickets and the Paradox of Rickets*

Depending on the nutritional status of the child, two types of rickets may occur. If general undernourishment is present, then an atrophic or porotic form of rickets will develop, resulting in bones with thin porous cortices, wide marrow spaces, accompanied by relatively few, and thin, spongy trabeculae (Stuart-Macadam 1989; Ortner 2003). These bones are very fragile and especially subject to fracture (Ortner 2003).

The better-nourished child develops the "hypertrophic" or "hyperplastic" type of rickets. In this type, the bone cortices are porous but thick, the result of excessive deposition of osteoid with the marrow spaces narrowed, due to an abundance of spongy osteoid trabeculae (Stuart-Macadam 1989; Ortner 2003). Fractures are less common in this form but the bones can become distorted in shape.

Children with rickets exhibit less evidence of severe malnutrition than acutely malnourished children. This may be due in part to the fact that only actively growing children can show evidence of rickets (Chesney 2001). This is known as the "paradox of rickets", and as the disease becomes more severe, growth slows and changes in the epiphyseal plate become milder and may eventually disappear (Mankin 1974a). Therefore, the most acute forms of rickets (florid rickets), showing the most severe deformities, are found in children that are relatively well nourished (Loomis 1970).

2.1.9 *Complications of Rickets*

Rickets was first reported as a cause of death in 1634, in the London Bills of Mortality (Clarke 1962; Steinbock 1993). The relative risk of death for children with rickets, compared to those not affected by rickets is 1.7 times higher (Bishop 1999). Chali and coworkers (1998) found over a one year period of total deaths in Addis Ababa, about 20% had severe rickets with the cause of death directly or indirectly related to rickets.

Rickets may be accompanied by tetany, gastrointestinal upsets, diarrhea, increased susceptibility to respiratory infections, a delay in general development, and shorter than average stature (Agrawal *et al.* 1969; Stuart-Macadam 1989). Tetany is caused by a reduction in plasma ionic calcium increasing the excitability of the nerves and muscles causing muscles to twitch and spasm (Passmore & Eastwood 1986). A strong relationship exists between rickets, acute respiratory infection (ARI) and enteritis (inflammation of the small intestine). A study conducted in Turkey found the prevalence of ARI and enteritis to be

approximately 1.5 times higher in children with rickets when compared to children without rickets (Beser & Cakmakci 1994). A study conducted in Kuwait found that 43.6% of children with rickets had ARI and 28% of children with rickets had enteritis (Lubani *et al.* 1989). Fighting rickets is an important endeavor in developing countries where deaths of those under five years of age are largely due to ARI and enteritis (Beser & Cakmakci 1994). Prior to World War II infant mortality from ARI was high among infants who were not breastfed (Cunningham 1995). Infection of the respiratory and gastrointestinal tracts was the presenting clinical finding in nearly half of the cases of rickets from 1972 to 1984 in Manitoba (Haworth & Dilling 1986).

Classical descriptions of rickets emphasized an association with pneumonia. In North America and Europe this association has not been prominent (Ferguson 1918; Chesney 2001), but it is commonly observed in young children in developing countries (Chali *et al.* 1998; Bishop 1999; Chesney 2001). It is often overlooked that the mouth is a portal of entry for the respiratory system as well. It has not been established whether vitamin D deficiency is the result of immunologic abnormalities and pneumonia, or if hypocalcemia and myopathy result in poor clearing of bronchopulmonary secretions (Chesney 2001).

Rickets is associated with an increase in the frequency of infections and impaired neutrophil phagocytosis (white blood cells that search the body for foreign matter and engulf them), and tuberculosis (Maxwell 1994). Vitamin D is known to have effects on immunological function in animals and may be the factor responsible for reduced immunocompetence and an independent risk

factor for tuberculosis (Maxwell 1994). Cunningham (1995) asserts that our knowledge regarding resistance to infections is incomplete and it is difficult to appreciate the complex interrelationships among immunologic defense factors and different organ systems.

2.2 Deficiency Rickets versus Genetic Forms

While the most common cause of rickets and osteomalacia is vitamin D deficiency, these diseases can also occur through vitamin D malabsorption, metabolism, receptor defects as well as collagen and phosphate defects (**table 2.2** below). These illnesses were researched to establish cause, skeletal expression, and age at onset, in order to distinguish them from vitamin D deficiency rickets.

TABLE 2.2 – Other forms of rickets in children

I.	**Vitamin D Malabsorption** a. Celiac disease (Hepatic disease or steatorrhea) b. Short gut syndrome* c. Pancreatic insufficiency
II.	**Vitamin D Metabolism Defects** a. Hepatic rickets – failure of 25 hydroxylation b. Vitamin D dependency type I 1 α (Hydroxylation abnormalities) c. Renal osteodystrophy d. Anticonvulsant induced osteomalacia*
III.	**Vitamin D Receptor Defect** vitamin dependency type II (hereditary vitamin D-deficiency rickets)
IV.	**Phosphate Deficiency Rickets** a. X-linked hypophosphatemic rickets b. Hypophosphatemic rickets (ADHR) (autosomal dominant) c. Hypophosphatemia (autosomal recessive) d. Fanconi syndrome e. Oncogenous rickets (tumor induced rickets) f. Antacid-induced rickets*
V.	**Collagen Defects** a. Metaphyseal Dysplasia (Schmid variety) b. Osteogenesis imperfecta

(*Not relevant to this discussion).

In ancient remains, determining if rickets is a disease of deficiency versus of a genetic origin presents a difficulty. Molecular ways of determining genetic origins of rickets are possible, but highly improbable (Barta 2002, *pers. comm.*). Nuclear genes would be required. The locations of the genes causing genetic types of rickets may be multiple and not well identified. In ancient skeletal remains, nuclear DNA is only preserved under very special circumstances (Höss

et al. 1996; Krings *et al.* 1997; Stone 2000). Due to the highly fragmented nature of ancient DNA, yielding low copy numbers, the preservation and extraction of nuclear DNA in the Roman samples of Isola Sacra is highly unlikely.

Determining the cause of rickets in the Isola Sacra sample will then depend on the frequency with which genetic forms of rickets are found in modern populations. Relying on the highest birth frequencies for genetic forms of rickets in modern populations, the probability of finding individuals from an archaeological sample with skeletal changes as a result of genetically induced rickets is assessed. It may then be possible to discount various forms of genetic rickets because of their low incidence rate.

Determining the spatial relationship of rachitic individuals will assist in determining a genetic origin of rickets. Relatedness of rachitic individuals will be ascertained by discovering their spatial distribution in the necropolis. Sample number and subsequent burial location will be mapped. Using the spatial relationship and the frequency with which genetic rickets appear in modern populations vitamin D deficiency rickets may be confirmed or rejected as the nosological entity causing the rachitic skeletal changes within the population of study.

2.3 Frequencies of genetic forms of rickets

Table 2.3 (below) refers to the frequency with which genetic forms of rickets affect modern populations. With the exception of celiac disease, the remainder of these diseases are relatively rare with many having unique features which allow for differentiation. As a result, in determining the type of rickets

found in the Isola Sacra sample, only celiac disease need be taken into consideration.

Table 2.3 – Frequencies of genetic forms of rickets*

Type of Genetic Rickets	Frequency
Celiac disease (Hepatic disease or steatorrhea)	1/250 births (Halsted 1996).
Pancreatitis or Pancreatic insufficiency	Primarily caused by alcoholism, mainly occurs in old age (Dorland 2000).
Hepatic rickets – failure of 25 hydroxylation	Rare† (Dorland 2000).
Vitamin D dependency type I (VDDR1)	Rare, most commonly found in the French Canadian population (Thomas & Demay 2000).
Renal osteodystrophy	Frequency not known, causes dwarfism (Cheng & Holman 1980).
Vitamin D-dependency type II (VDDRII)	Rare† ((Thomas & Demay 2000).
X-linked hypophosphatemic rickets	Rare – 1/20,000 births, associated tendon ossifications, increase in bone density, extreme elongation of the skull (Formicola 1995).
Autosomal dominant Hypophosphatemic rickets	Rare – 1/20,000 births (Dorland 2000).
Hypophosphatemia (autosomal recessive)	Rare - 1/100,000 births (Dorland 2000).
Fanconi syndrome	Extremely rare† (Dorland 2000).
Oncogenous rickets (tumor induced rickets)	Rare†, does not affect infant, only late childhood on (Dorland 2000).
Metaphyseal Dysplasia (Schmid variety)	Overall incidence of all skeletal dysplasias is 1/4000 - 5000 births (Olsen 1995).
Osteogenesis imperfecta	1/30,000 to 1/60,000 births Aufderheide & Rodríguez-Martin 1998).

*Based on frequencies in North America. † Specific frequency for these diseases could not be located.

In ***celiac disease*** (hepatic disease or steatorrhea), fat-soluble vitamin D is eliminated with the fatty diarrheic in the stools caused by an intestinal mucosa toxicity of gluten (Sylvester 1999; Ivarsson *et al.* 2002; Ortner 2003). Celiac disease, also known as gluten intolerance, is a genetic disorder that affects 1 in 250 Americans (Halsted 1996). Symptoms of celiac disease can range from the classic features, such as diarrhea, weight loss, bone pain, osteoporosis and malnutrition, to latent symptoms such as isolated nutrient deficiencies but no gastrointestinal symptoms (Halsted 1996). Those affected suffer damage to the villi (shortening and villous flattening) in the lamina propria and crypt regions of their intestines when they eat specific food-grain antigens (toxic amino acid sequences) that are found in wheat, rye, barley and possibly oats (Halsted 1996).

2.4 Relatedness and the spatial distribution of the Isola Sacra sample

In order to assess the spatial relationship of rachitic individuals to assist in the determination of genetic origins of rickets it is necessary to research the burial practices of the inhabitants of *Portus Romae* as well as the excavation practices utilized.

At Isola Sacra, tombs were to be used for the burial of family members, their freedmen, freedwomen and their descendants (Meiggs 1960). Tombs were sometimes divided between families after they had been built and the expenses of a tomb could also be shared, making provisions for their dependants in the same tomb (Meiggs 1960; Toynbee 1971). Evidence at Isola Sacra also confirms many examples of individuals securing niches or graves within the tombs of others (Meiggs 1960; Toynbee 1971). It was written that no provision was made

for slaves in the family tombs but it should be noted that slaves were in fact buried within tombs with the family (Prowse 2001). The majority of slaves were marked in the Isola Sacra cemetery by amphora necks projecting from the ground but these types of burials were not confined to slaves (Meiggs 1960). No new tombs seem to have been built after the middle of the third century which is thought to be a consequence of the leaner economic times (*ibid.*). It is speculated that it is at this time many of the older Isola Sacra tombs were reused for late burials as it is probable that families for which the tombs were built had died out (Meiggs 1960).

Isola Sacra was first excavated between 1925 and 1940 by G. Calza. Between 1973 and 1982, the Archaeological Superintendency of Ostia, the University of Rome 'La Sapienza', and the University Institute of Oriental studies of Naples focused on the restoration of the monumental tombs and the recovery of the human skeletal remains excavated by Calza that had been arbitrarily dumped back into the tombs once his excavations were completed (Baldassarre 1984, 1990; Prowse 2001; Rossi *et al.* 1998; Sperduti 1995). From this excavation approximately 1,000 individuals were recovered, forming half of the Isola Sacra skeletal collection (Prowse 2001). The majority of individuals in this sample are from the later excavations found in between the tombs (*ibid.*). While the 'zone' information from the excavations of each burial is recorded, the archaeological details about these zones are not known.

The lack of knowledge regarding the burial zones, the practice of burying those not genetically related (freedmen/women, friends, slaves) as well the

subsequent recycling of tombs complicates the determination of relatedness of individuals in the Isola Sacra necropolis. Many of the tombs have inscriptive evidence to indicate the tomb's owners but only DNA analysis can establish relatedness. In addition, due to the careless excavation techniques of Calza, *in situ* burials have been completely lost. It is concluded that the relatedness of rachitic individuals cannot be ascertained by their spatial distribution in the necropolis and an estimation of the prevalence of genetic forms of rickets in the Isola Sacra sample must rely solely on inferences based on the frequencies with which they appear in modern populations.

Chapter 3

A BRIEF HISTORY OF RICKETS

3.1 RICKETS IN ANTIQUITY

Convincing paleopathological cases of rickets are rare in the prehistoric and early historic periods (Stuart-Macadam 1988, 1989; Larsen 1997). Those cases that have been detected are healed lesions in adults or subadults, rather than active cases in infants or young children (Ortner and Mays 1998). Rickets was not known to occur in prehistoric peoples in North America (Stuart-Macadam 1988, 1989). Paleolithic skeletal remains from Egypt reveal rickets to be virtually nonexistent. The lack of rachitic deformity in Egyptian remains has been attributed to the absence of the practice of swaddling and leaving young children naked, thus allowing their skin to be exposed to sunlight (Fildes 1986a; Larsen 1997). Few cases have been found in Mesoamerica. Neolithic northern European specimens, in Norway, Sweden, Denmark and the British Isles show rickets, while more plentiful evidence comes from Roman times in Hungary (Steinbock 1993; Roberts & Manchester 1995; Colón & Colón 1999). The Chinese seem to have been acquainted with rickets as skeletal deformation was mentioned in Chinese writings from 2000 years ago (Beck 1997). Skeletal remains dating from the twelfth century A.D. onward from the Waterford city, Ireland, excavations display mild rachitic deformities (Power & O'Sullivan 1987). The apparent rarity of skeletal evidence of rickets in prehistoric and early historic

periods is attributed to the belief that children in antiquity likely spent much of their time outdoors, even during the winter months (Roberts & Manchester 1995).

3.2 RICKETS IN ANCIENT ROME

Most of the surviving written sources referring to the presence of rickets come from ancient medical texts, such as Cornelius Celsus (1st c. A.D.) *DeMedicina*, Soranus' (1st c. A.D.) *Gynaecology*, and Galen's (2nd c. A.D.) *De sanitate tuenda*.

Soranus of Ephesus, who lived during the reigns of Trajan and Hadrian (98-138 A.D.), was the first to give a detailed description of rickets early in the 2nd century A.D., observing the disease to be particularly prevalent in Rome (Foote 1927; Temkin 1956; Simonetti 1960; Colón & Colón 1999). Soranus describes the symptoms of rickets, indicating a high number of children suffering from deformed spinal columns and legs (Steinbock 1993). He wrote "if no one supervises the movement of infants the limbs become distorted in most cases" (Colón & Colón 1999:57).

Later, Galen's (130-200 A.D.) medical writings describe a number of skeletal deformities in infants and young children, specifically knock-knees, bowlegs, funnel-shaped chests and pigeon breasts, seen in rickets (Steinbock 1993). Galen also comments on the habits of women, that they "stay indoors, neither engaging in strenuous labour nor exposing themselves to direct sunlight", having detrimental consequences on the vitamin D status of mother and thus infant (Garnsey 1991).

3.3 RICKETS FROM THE MIDDLE AGES TO THE RENAISSANCE

Rickets is thought to be a disease of increased urbanization and industrialization. There appears to be a gradual increase in the skeletal evidence of rickets in Medieval times (350-1450 A.D.), at least in cities across northern and central Europe (Steinbock 1976; Larsen 1997). The prevalence of rickets during the medieval and post-medieval periods is, however, difficult to assess, as rickets could often not be differentiated from other diseases affecting the skeleton of the child (Stuart-Macadam 1988, 1989). Shorter (1982) states that Viking skeletons from the 15th century in Greenland showed many rickety changes. These changes consist of twisted spines and narrow pelvises and it was thought that the women possessing them would be incapable of giving birth (Shorter 1982).

With the passing of feudalism came industrialism and people began to congregate in cities where weaving, metalworking and other industries developed (Foote 1927). In the last half of the 15th century ideal conditions existed for the development of rickets in Europe. The people lived in crowded, walled, cities in a northern climate with little sunlight and a poor diet (Park 1923). Evidence of rickets is found in the cities of Cologne, Nuremberg and Bruges between 1450 and 1500 (Foote 1927). The 15th and 16th century marked the rise of religious art in Central Europe, especially in Germany and the Netherlands. It is reasonable to suppose that the presence of rickets would be depicted in the art of the time. Painters of the period depict infants with square heads, potbellies

and grooved chests (Foote 1927). Foote (1927) found clinical signs of rickets in at least fifteen paintings, dating from 1440 to 1500.

Rickets was not just a disease of the workers or proletariat, nobility also suffered. King Vaclav II of Poland and Bohemia suffered from rickets (Dudziński 1989). A portrait painted in 1568 of the two year old King Sigismund Vasa III of Poland revealed he also suffered from rickets (Dudziński 1989). Charles I of England suffered from severe rickets and his apparent shame of his crooked legs spawned a fashion trend of boots for men and long coats for women to hide their deformities (Clarke 1962; Fildes 1986b). The second daughter of King Charles I, Princess Elizabeth who died in 1650, also suffered from rickets to an advanced degree (Clarke 1962; Cone 1979a).

3.4 RICKETS IN THE 17TH AND 18TH CENTURIES

In 17th and 18th century England, rickets was the principle disease of infancy and the clinical manifestations were well known (Fildes 1986b). Rickets was seen to accompany industrialization and urbanization, occurring with great frequency in the smoky manufacturing towns of Northern Europe and North America. In the 17th and 18th century, rickets was also quite prevalent in the New England colonies, as it had been in the European countries from where the settlers had come (Weick 1967). The majority of medical writers were English and all described rickets as a new disease, first noticed around 1620 (Fildes 1986b). Daniel Whistler, 1645, was the first to describe the disease in any language,

although he had been accused of plagiarizing his contemporary, Francis Glisson, 1650 (Fildes 1986b).

After 1650 and the discovery of rickets by Glisson, early English obstetricians of the time were describing the problems of delivering infants through the rickety pelves of women (Hess 1929; Dunn 1998). It is interesting that in the middle of the 17th century forceps were introduced into practice and male midwives were becoming more common (Dunn 1998).

Accounts of rickets compiled by 17th and 18th century medical writers state that those children affected by rickets came from wealthy families, as it was, "the children of the aristocracy and higher gentry who suffered most frequently and most severely" (Fildes 1986b: 125; Rosen von Rosenstein 1984). Glisson stated that, "this disease doth more frequently invade the cradles of the rich than afflict poor men's children" (cited in Dunn 1998:F155). Fildes (1986b: 125) states, "some of the poorest infants (foundlings and some parish children) also contracted rickets, but those from the poor and middle ranks of society remained unaffected". Glisson prescribed swaddling to straighten the bones (Colón & Colón 1999). Physicians at the time blamed the prevalence of rickets in wealthy children to the "ill orderings" of wet nurses, transmitted by the milk of a diseased wet nurse, (Fildes 1986a, 1986b). The occurrence of rickets was also linked to weaning from the breast and supplemental feeding. Glisson found signs of rickets to appear between the ages of 6 months to 2.5 years (Fildes 1986b; Dunn 1998). The onset of rickets may have been a consequence of the type of foods given during the weaning process, and to the total or partial loss of

breast milk (Fildes 1986a). The age at which rickets appeared may be related to the fact that this is the average age at which children first crawl, stand and walk. It is at this time that bowing of the legs and other skeletal deformities would become apparent. Another possible factor is the mention of rickets increased in the medical literature; physicians may have been more aware of it and able to put a name to an existing disease (Fildes 1986a). The high incidence of rickets in wealthy children was likely due to cultural factors linked to socioeconomic class but may also have been the result of under reporting, as the poorer classes could not consult a physician (Fildes 1986b).

The recommended weaning diet of the upper classes consisted of mainly meat and bread, resulting in a lack of vitamins D, C, A, and calcium (Fildes 1986a). Children in poorer families probably had a more balanced diet; however, they may have suffered from lack of quantity at times (Fildes 1986a). Fildes (1986a) states that the diet of most of the population was different in that it was better balanced than that of the wealthy. Diet may have been more of a factor for the lower class children, but for the upper classes, it was likely an issue of social and cultural practices. Wealthy women and children were more likely to stay indoors; not having to take on the burden of physical labour, such as would the poorer classes. Fildes (1986a: 127; Gibbs 1994) notes richer children were, "cosseted and kept well wrapped up in nurseries", whereas poorer children would have been outside from an early age, thus their diet could have been deficient but exposure to sunshine would have counteracted the effects. Cultural and socio-economic factors relating to the amount of access children

had to sunlight, rather than on diet and weaning foods, are more likely to explain the near epidemic incidence of rickets during this period.

Wealthy women employing wet nurses would not have benefited from the contraceptive effects of breastfeeding. Child bearing habits of the poor and wealthy differed. Wealthy families put their children out to nurse in order to have as many children as possible to counteract high infant mortality, to ensure heirs to inherit land or property (Fildes 1986b). McLaren (1979) states based on parish records, that it was not uncommon for wealthy women to have up to twenty or thirty children, whereas the average English family at the time had four or five children (Fildes 1986b). With an increased number of pregnancies and a diet which contained little vitamin D or calcium, the cultural practices of the wealthier women may have been predisposing infants to rickets (Fildes 1986a & b). There is a significant relationship between the number of children under five in a household and the prevalence of rickets. The more children under five years of age, the more the demand on maternal stores of vitamin D and calcium, as closely spaced pregnancies leave little time for the replenishment of maternal stores (Ferguson 1918; Akpede *et al.* 1999). Under spacing of pregnancies predisposes infants to rickets in cultures where mothers already have subnormal levels of vitamin D. The literary evidence suggests that many children seem to have recovered from rickets after three years of age, but its long-term effects on females became obvious when they attempted to have children (Fildes 1986b).

3.5 RICKETS AFTER THE EIGHTEENTH CENTURY

During the affluent 18th century the occurrence of rickets seemed to decrease, but towards the end of the 18th century rickets was again on the rise, especially in the industrialized cities of England, Scotland and Germany (Owen 1889; Fildes 1986b; Wilton 1995; Larsen 1997). During this time, families moved from the countryside to factory towns to seek work. With this change of residence and political economy, came changes in family size and infant care. Most wealthy women were more likely to breast feed their own children, and the practice of wet-nursing declined (Fildes 1986b). Poor, nutritionally deficient children were expected to work the daylight hours in the shelter of factories (Roberts & Manchester 1995). There is an early introduction of weaning foods in populations in which mothers experience chronic poor nutrition and health and where they must meet heavy labour and reproductive demands (Gray 1996). As poorer women were now expected to work long hours in factories, they were no longer able to fully breastfeed and used nutritionally poor weaning foods as a substitute (Fildes 1986a & b; Dunn 1998). Without the contraceptive advantage of breastfeeding, poorer women had larger families who were brought up in small, enclosed environments lacking sunlight (Dunn 1998). The poor did not have access to dietary sources of vitamin D and living in industrial cities clouded with smoke, their exposure to sunlight was insufficient to prevent rickets. As these changes occurred, a new picture of rickets emerged. It became a disease of the urban poor, no longer a disease of the rich and affluent (Fildes 1986b). In British industrial cities during this period working-class children may have gone for

weeks or even months without sunlight exposure (Beck 1997). The classic Charles Dickens's character Tiny Tim from *A Christmas Carol* is thought to be understood as a rachitic child (Beck 1997).

From about the mid-19th century visual judgments regarding the prevalence of rickets can be made through the examination of photographs. Many children had severe rickets and in some city communities milder manifestations were almost universal (Gibbs 1994). Photographs of groups of children from this period give further evidence as to the extent of the problem (Gibbs 1994). During this time rickets was thought to be the product of poor diet, unhygienic conditions and bad air (Gibbs 1994). William Macewan, working in Glasgow, referred to children in some parts of the city as being "shut out from the light partly by the height of the houses (and) partly from the fact that even the sun's rays which do manage to struggle through the canopy of smoke which envelops them, are so diluted that they are of comparatively little value" (Gibbs 1994).

Children were kept in factories, schools and cities where the sun was blotted out by soot and smoke (Shorter 1982). Additionally, children behind window glass are not protected from rickets as window glass filters out ultra-violet radiations (Park 1923). A study conducted in 1889 determined a relationship between city dwelling and rickets in the British Isles; the higher the population density, the more rickets (Stuart-Macadam 1988, 1989). In contrast, rickets was rare in rural districts and virtually absent in villages and village towns (Stuart-

Macadam 1988, 1989). Rickets during the Victorian era was largely the result of socio-economic conditions, which limited children's access to sunlight.

For the year 1910, 1282 cases of caesarean section in Great Britain were examined for causation. In 1058 of these cases the indication for the operation was pelvic deformity (Hess 1929). Hess (1929) found that in Glasgow and Manchester where rickets was especially prevalent, compared with cities of similar size with less rickets, the number of Caesarean sections was greatly increased.

Infantile deficiency rickets was very common in urban America at the turn of the last century (1900) (Welch *et al.* 2000; Fomon 2001). In the United States, poverty, tenements and a general neglect of the immigrant population added to the prevalence of rickets (Weick 1967). Ferguson (1918) found that social and economic factors played a role in the causation of rickets, as families of lower class were more susceptible. In the late 19th century rickets was frequently observed in African Americans and immigrant families (Weick 1967). At the turn of the century it was said that no nationality was exempt from rickets that lived in the city (Weick 1967). In the United States, records show that between 1910 and 1961, 13,807 deaths were attributed to rickets (Weick 1967).

With the introduction of pollution controls and the use of cod-liver oil the severity of rickets began to decline in Europe and North America (Wilton 1995; Beck 1997; Fomon 2001). In remaining parts of the world, rickets was relatively rare, only occurring among those in special circumstances, primarily the result of cultural practices (Stuart-Macadam 1988, 1989).

3.6 CONTEMPORARY RICKETS

Most cases of rickets today are the result of cultural factors, affecting people of all socioeconomic backgrounds or of the severely malnourished (Pugliese *et al.* 1998; Oken & Lightdale 2001). Rickets is now more commonly found in the tropics and subtropics of Third World countries, despite adequate sunlight (Stuart-Macadam 1988, 1989; El Hag & Karrar 1995; Thacher *et al.* 2000; Chesney 2001; Harris *et al.* 2001).

Immigrants from sunny countries who move to higher latitudes with less sunshine do not accumulate stores of vitamin D, because of limited exposure to sunlight (Sanders 1995; Pal & Shaw 2001; Tomashek *et al.* 2001). Custom may not encourage taking babies outdoors, and when taken out they are always fully covered or clothed (Binet & Kooh 1996). Colour preference is another cultural factor that may predispose children to rickets. Some mothers of African and Asian ancestry deliberately keep their children out of the sun because they do want to him to become "a black man" (Passmore & Eastwood 1986).

Cultural dietary factors (e.g. veganism, Sikhism, East Asian consumption of unleavened breads i.e. chapattis) resulting in a high phytate or high fiber diet will increase vitamin D and calcium requirements (Sanders 1995; Dunnigan & Henderson 1997; Kaper *et al.* 2000; McCaffree 2001). Dairy products fortified with vitamin D may not be a traditional food source for some groups, and lactose intolerance may also be a factor (Haworth 1995; Moffatt 1995; Binet & Kooh 1996).

Purdah requires that a woman be veiled during pregnancy, parturition and lactation, exactly the time at which vitamin D and calcium need is intensified (Chesney 2001). Inadequate maternal sun exposure in women, who practice purdah results in a greater risk for osteomalacia and in relation to perinatal vitamin D deficiency, may have adverse consequences for infants of these women (Chesney 2001; Grover & Morley 2001; Mason & Diamond 2001; McCaffree 2001; Nozza & Rodda 2001).

As maternal vitamin D status is the major factor regulating the vitamin D content of human milk, "prolonged" breastfeeding without adequate vitamin D supplementation or exposure to sunlight may predispose infants to rickets (Kreiter *et al.* 2000; Oken & Lightdale 2001). Babies that are breast fed are less likely to develop rickets providing the fitness level of the mother is high (Fildes 1986a; Fildes 1995; Stuart-Macadam 1988, 1989). Reduced duration of breastfeeding, coupled with nutritionally poor weaning foods may also predispose infants to rickets. Other factors contributing to contemporary rickets are fear of skin cancer and the increased sedentary indoor lifestyle of children (Edidin *et al.* 1980; Saffran 1995; Rao 1999; Holick 2001; Tomashek *et al.* 2001).

Chapter 4

SKELETAL MANIFESTATIONS OF RICKETS

Rickets can be traced back to antiquity through the examination of the skeletal evidence. Skeletal involvement of rickets varies from individual to individual, ranging from mechanical deformities, to porosity of the skull and epiphyseal plates, as well as flaring of the bone ends (Ortner & Mays 1998). Rickets produces characteristic skeletal deformities in the skull, long bones, ribs, vertebrae and pelvis. Unmineralized osteoid does not survive in the post-burial environment any more than muscle or soft tissues and as a result, the pores or other defects where unmineralized osteoid was present are what make the condition apparent (Ortner and Mays 1998).

4.1.1 Skull - Often the earliest changes seen in rickets appear in the skull (Stuart-Macadam 1989). Cranial manifestations of rickets include the delayed closure of the fontanels and thin, softened areas of the cranial vault and facial bones. In active infantile rickets, the posterior portions of the parietal bones and occipital squama are especially affected (Aufderheide & Rodríguez-Martin 1998; Ortner 2003). Permanent posterior flattening or lateral and asymmetrical deformity of the skull may result when the area is subjected to pressure when the infant is resting (Ortner 2003). Thickening of the cranium is seen, with disappearance of the outer table and, often, the inner table. This resembles the porous appearance of diploë, due to external (and sometimes internal) subperiosteal bone deposition (**figure 4.1**) (Ortner 2003). The thickening is almost entirely on the

outer surface and is not evenly distributed being more pronounced at the eminences of the frontal and parietal bones (Steinbock 1976). In more advanced rickets the 'square head' is a familiar clinical hallmark due to the bone deposition on the four frontal and parietal bosses (**figure 4.2**) (Steinbock 1976). Frontal bossing or the visual and palpable prominence of the centers of ossification for the frontal bones is a normal feature of the growing fetal and infant skull and can be differentiated from rachitic frontal bossing through the presence of porotic bone deposition (Scheuer & Black 2000). During the healing process, part of the excessive osteoid on the frontal and parietal bosses is absorbed but most ossifies and remains as permanent evidence of rickets (Steinbock 1976).

Figure 4.1 – Rachitic calvarium showing external & internal deposition of subperiosteal cancellous bone (Ortner 2003: 395).

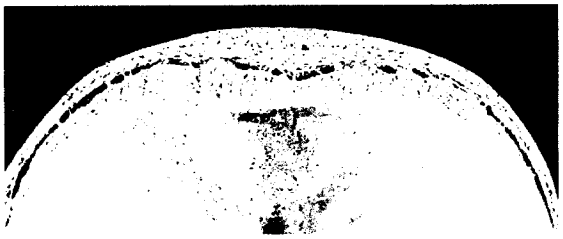
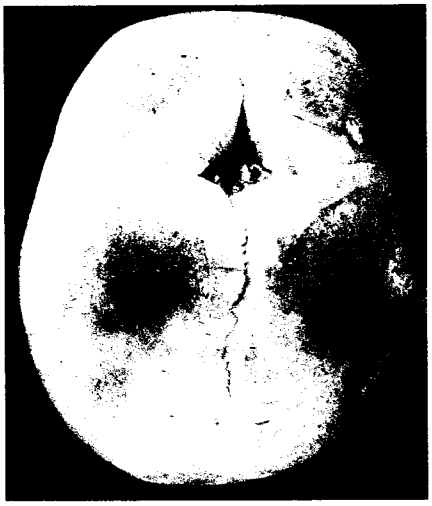


Figure 4.2 (below)– Squared head of a rachitic child showing fine porotic bone deposition of frontal and parietal bones (Ortner 2003:394).).



The external surface of the cranial vault and roof of the orbits may show porosity (**figure 4.3 & 4.4** below) and the frontal bones may also show deposits of woven bone (Ortner & Mays 1998). Fine subperiosteal bone deposition may be seen on the glabella and facial bones (**figure 4.5** below) (Ortner 2003). The mandibular ramus may show abnormal medial-

posterior bending due to muscle action during chewing (**figure 4.6** below) (Ortner & Mays 1998).

Figure 4.3 – Orbital roof porosity SCR 103 (photo C. Wood).

Figure 4.4 – Outer table, parietal bone, showing porous periosteal bone (Ortner 2003:395).

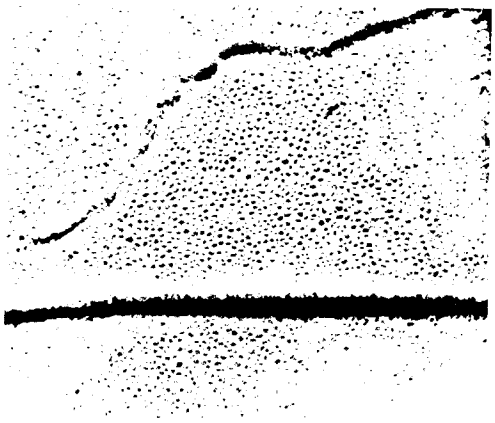


Figure 4.5 – Fine subperiosteal bone deposition on the glabella & facial bones (Ortner 2003:397).

Figure 4.6 – Mandibular ramus showing abnormal medial posterior bending (Ortner & Mays 1998:49).

Rickets also causes a delay in tooth eruption as well as enamel hypoplasia in the deciduous teeth (Hess 1929; Passmore & Eastwood 1986; Aufderheide & Rodríguez-Martin 1998). Dentine and bone are analogous structures of similar origin and in the absence of vitamin D; dentine and enamel will not develop

normally. Defects in the enamel and dentine may increase caries formation. There is a much greater prevalence of dental caries in northern countries than in tropical countries which may in part be due to less exposure to sunlight and the consequent reduced synthesis of vitamin D in the skin (Passmore & Eastwood 1986).

4.1.2 Long bones - The most obvious skeletal manifestation of rickets is the characteristic bowing of the femur, tibia and fibula. The weight bearing bones become bent when the child begins to walk and the arm bones may also become deformed when a child begins to crawl (Roberts & Manchester 1995). These bending deformities occur only in children who have good muscle tone and are able to walk. A child who has good muscle tone but is not ambulant will show mild or no deformity, whereas a child who has poor muscle tone and is not ambulant, will not show bowing deformities (Stuart-Macadam 1989). In addition, long bones may exhibit flaring, fraying and cupping of the metaphyseal ends (Aufderheide & Rodríguez-Martin 1998). This is the result of excessive unmineralized cartilage causing an increase in the length and width of the growth plates of the bones (**figure 4.7** below) (Roberts & Manchester 1995; Ortner 2003).

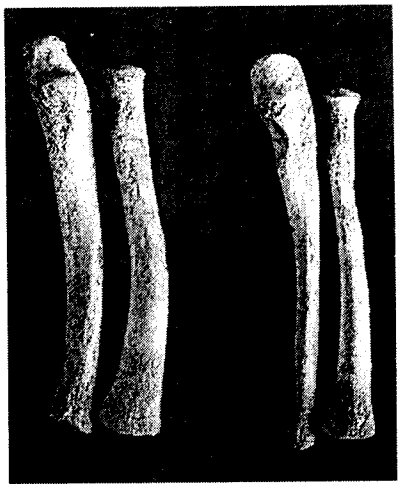


Figure 4.7 – Rachitic radius and ulna showing abnormal curvature and thickening, (normal radius & ulna on right for comparison (Ortner & Mays 1998:49).

The growth plates of the long bones can show abnormality in the form of porosity and roughening of the epiphyseal plates (Ortner & Mays 1998). These changes range from fine-grained-roughness, to pitting, to extreme roughness and porosity (Ortner & Mays 1998). Changes in the shafts of the bone develop later than the changes at the ends. The diaphyses may show a loss of density in addition to thinning of the cortex (Stuart-Macadam 1989). Fractures and pseudofractures can occur, which appear radiographically as symmetrical, transverse, ribbon-like zones of decreased density (Stuart-Macadam 1989). The cortex of the metaphyses may also appear porous and an irregular "strut/slit" anatomy may be present, particularly towards the distal end of the affected bone (Ortner & Mays 1998).

Jain and coworkers (1984) described 20 cases of advanced rickets showing changes in the short bones of the hand and feet. These changes are characterized by general osteoporosis with marked cupping in the metacarpals and metatarsals (*ibid.*).

4.1.3 Ribs - The ribs may show flattening of their curves and flaring towards their sternal ends (**figure 4.8** below).

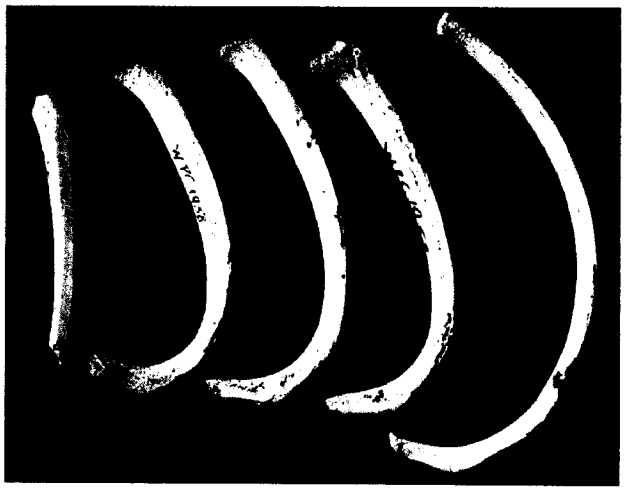


Figure 4.8 – Inferior view of ribs, together with a normal rib (on the extreme right) for comparison. Ribs show thickening, particularly towards the sternal ends and flaring at the sternal ends (Ortner & Mays 1998:52).

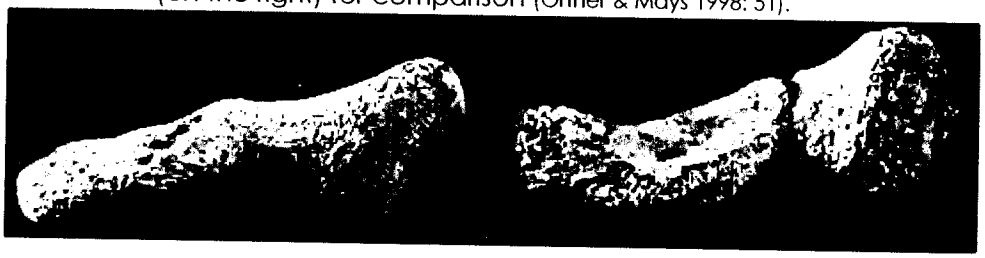
There is often asymmetrical flattening of one or the other hemithorax and the sternum may protrude or become acutely angled (Mankin 1974a). The costochondral areas of the ribs become nodular prominences, referred to as a 'rachitic rosary', and push the sternum out to produce a pigeon breast deformity (Aufderheide & Rodríguez-Martin 1998). Alternatively, the arched costochondral junction pushes the sternum dorsally, creating a funnel shaped breast (Aufderheide & Rodríguez-Martin 1998). Softened, unmineralized bone bends to the traction of the diaphragm at its points of attachment; this inward pull causes a circular depression in the lower rib cage (Harrison's groove) (Aufderheide & Rodríguez-Martin 1998). The cortices of the costochondral ends of the ribs show irregularity and porosity, specifically pitting (Ortner & Mays 1998).

4.1.4 Vertebrae – In severe cases, the vertebrae may become porous and decrease in height due to compression (**figure 4.9)** often combined with a scalloped appearance of the end plate (Ortner 2003). A prominent lordosis pushes the abdominal content forward producing a pot-bellied child (Aufderheide & Rodríguez-Martin 1998).

Figure 4.9 – Porous vertebrae, decreased in height due to compression (Ortner 2003: 400).

4.1.5 Pelvis – The pelvis is more affected by altered growth than by mechanical deformation (Ortner 2003). During active disease the rachitic pelvis appears smaller and plumper than normal (**figure 4.10** below), but does not show the deformity of the adult postrachitic pelvis (Ortner 2003).

Figure 4.10 – Inferior view of the left ilium showing abnormal lateral curvature with a normal ilium (on the right) for comparison (Ortner & Mays 1998: 51).



4.2 SKELETAL MANIFESTATIONS OF OSTEOMALACIA

Osteomalacia is the adult form of rickets most often associated with females during adolescence or young adulthood (Steinbock 1993; Aufderheide & Rodríguez-Martin 1998; Ortner 2003). Osteomalacia develops lack of sunshine, from poor diet, and the strain of multiple pregnancies during adolescence. Osteomalacia is especially prevalent among women of childbearing age (20-40 years old), as numerous closely spaced pregnancies, and the demand of prolonged lactation, represent a heavy drain on the bone mineral and vitamin D of the mother (Passmore & Eastwood 1986; Ortner 2003).

The first changes seen in osteomalacia appear radiographically as a diffuse diminished density of the bone, not undistinguishable from osteoporosis (Ortner 2003). In dry bone, the skeletal elements are very lightweight and have the consistency of cardboard (*ibid.*). Osteomalacia is characterized by severe mechanical deformities, rather than by changes in the growth plates (*ibid*). Skeletal changes associated with osteomalacia are most often observed in bones that contain the most cancellous bone (*ibid*). These are the vertebrae, pelvis, ribs, sternum and occasionally the lower limbs. The skull is rarely affected (Ortner 2003). The replacement of structured osseous tissue with decalcified osteoid results in soft and fragile bones, with the end product being scoliosis, kyphosis, bowing of the long bones and fractures and deformities of the pelvis (Zimmerman & Kelley 1982). Distortion of the pelvis may complicate pregnancy and the infant can be born with rickets if its mother had osteomalacia in a severe form (Zimmerman & Kelley 1982).

The adult postrachitic pelvis is characterized by the anteroposterior narrowing of the pelvic canal, primarily a consequence of growth deficiency of the iliac portion of the pelvic ring (**figure 4.11** below) (Hess 1929; Larsen 1997; Ortner 2003). In addition, the sacrum may protrude into the pelvic canal and the acetabula have a more forward facing position that normal (Hess 1929; Ortner 2003).

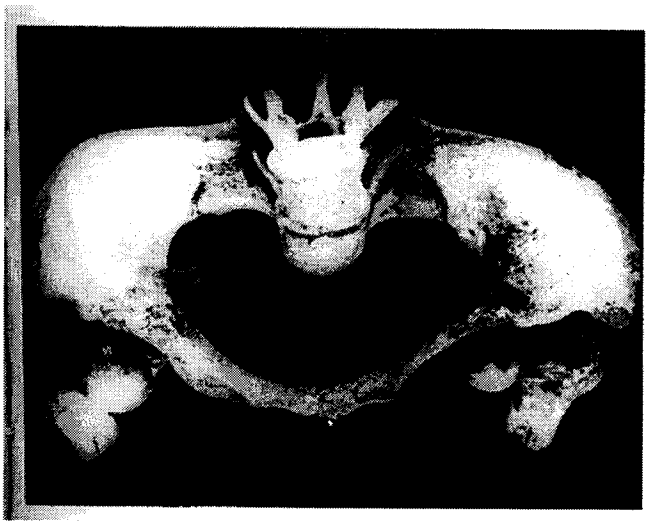


Figure 4.11 – Rachitic flat pelvis. (32-year-old woman, 1860) (Ortner & Putschar 1981: 279).

The rachitic pelvis would produce difficulty during child birth. It is estimated that prior to the 1920's, before the mechanisms of rickets were discovered, approximately one in four women was likely to have pelvic bones sufficiently malformed to cause a delay in labour (Shorter 1982). Flattening of the pelvis is well documented in clinical populations and modern anatomical samples in comparison with lower and middle class groups (Larsen 1997). Women who were children during the First World War have flattened pelvic inlets due to poor nutrition during these years (Larsen 1997).

4.3 MEDICAL DIAGNOSIS OF RICKETS

Medical diagnosis of rickets is determined through clinical, biochemical, radiological and histological means (Freaney *et al.* 1986). In many cases, physical findings may not be obvious to the clinician during the early stages of vitamin D deficiency rickets. Skeletal findings are the most striking clinical manifestations and may be evident within several months after the onset of vitamin D deficiency (Joiner *et al.* 2000).

Respiratory infections and pulmonary atelectasis (incomplete expansion of the lungs) that are related to severe chest deformities are frequently associated with rickets (Joiner *et al.* 2000). Iqbal and colleagues (2001) found that family screening is effective in finding undiagnosed vitamin D deficient subjects. They found that 67% of family members of patients diagnosed with vitamin D deficiency also have similar problems.

Suspected rickets cases often present with deformities, aches and pains, failure to thrive including delayed milestones, recurrent diarrhea and respiratory tract infections with signs of widening of the wrist, ankle and knees and bowing of the legs (Ahmed *et al.* 1975). Thacher and coworkers (2000) determined whether or not clinical features of rickets can identify children with active rickets. They found that wrist and costochondral enlargement were the most independently predictive signs of active rickets. Seventy-five percent of children with active rickets had wrist enlargement and seventy-seven percent had costochondral enlargement (Thacher *et al.* 2000). Due to the fact that these clinical signs resolve more rapidly than leg deformities, they are more reliable

than other clinical features of rickets (Thacher *et al.* 2000). Clinical features of rickets such as genu valgum (knock knee) and genu varum (bow leg) may remain long after rickets has healed biochemically and radiologically (Thacher *et al.* 2000). The limitations in using wrist and costochondral enlargement are poor reproducibility between investigators due to the subtlety of the traits, and a lack of an objective definition of rib beading and wrist enlargement, limitations that the physical anthropologist also faces in diagnosing rickets (Thacher *et al.* 2000). The more pronounced the wrist or costochondral enlargement the greater likelihood they signify active rickets.

A radiograph of the growing ends of the long bones is a necessary investigation in a suspected or established case of rickets (Opie *et al.* 1975; Pettifor *et al.* 1983). Radiographic findings include fraying and cupping of the metaphyses of the long bones, rib flaring, decreased mineralization of the bone matrix (generalized osteopenia) and multiple fractures in various stages of healing (Joiner *et al.* 2000). Radiographs may not be helpful in early diagnosis of rickets because 50% of calcium has to be lost before the radiological changes become visible (Ahmed *et al.* 1975).

In past clinical practice the authorative statement regarding the presence of metabolic bone disease was dependent upon examination of bone using histomorphological methods (Freaney *et al.* 1986). Some or all of the trabeculae and the cortex are not only lacking bone, but what bone there is, is surrounded by a layer of unmineralized bone known as an osteoid seam (Mankin 1995). Osteoid seams can be revealed through histologic sectioning but *cannot*

be detected in dry bone samples. Osteoid seams appear in cortical bone as rings and in cancellous bones as crescents tapering at each end (Parfitt 1998). The width and number (total count) of the osteoid seams provide a good index of the severity of the process (Mankin 1974a). Ahmed and colleagues (1975) found wide osteoid seams present in 97.3 percent of rachitic individuals and state that osteoid seams may be the only evidence of rickets in an older child. Those suffering from rickets and osteomalacia, regardless of the cause, show similar histologic features of bone and as a result the correct diagnosis cannot be made on the basis of histomorphometry alone (Mankin 1995). These osteoid seams are also seen in hyperparathyroidism, fibrous dysplasia and bone forming tumors, and while are highly suggestive of rickets and osteomalacia, are not pathognomonic (Mankin 1974a, 1995).

A review of medical approaches to the diagnosis of rickets show great variation in which traits were expressed. This is encouraging for skeletal diagnosis of rickets in archaeological human remains. The physical anthropologist may still be able to diagnose rickets as all skeletal elements may not be required. It also shows the researcher that variation in the expression of traits is expected, reducing frustration and the chance of misdiagnosis. Medical approaches to diagnosis reflect what was found in the preliminary diagnosis of the Isola Sacra individuals, variation in the expression of rachitic traits. Biochemical tests are often thought to be the gold standard of medical diagnosis but in many instances no correlation between clinical and biochemical data was found. The diagnosis of rickets can be established or excluded with reasonable certainty by

radiological and clinical examination, tools that are readily available to the physical anthropologist.

4.4 OTHER DEFICIENCY DISEASES: SCURVY & IRON DEFICIENCY ANEMIA

Skeletal abnormalities typically attributed to rickets were assessed individually in order to arrive at a differential diagnosis of this disease in archaeological human skeletal samples. Lesions associated with rickets were identified through a search of the literature. Other potential causes of the lesions were considered, particularly scurvy and iron deficiency anemia. As all are associated with malnutrition, it is possible for more than one of these to occur in the same individual, or group of people (Ortner & Ericksen 1997; Aufderheide & Rodríguez-Martin 1998). It is therefore necessary to delineate and define the pathological features associated with each disease, in order to differentiate between these conditions, and other pathological bone entities present. Morphological traits exhibiting consistency, with no overlap or problems associated with age will be utilized in diagnosis and deemed primary traits. Those traits with less than optimal characteristics will be given secondary consideration.

4.4.1 Skeletal Manifestations of Scurvy

Scurvy was not clearly recognized by Greek, Roman or Medieval physicians (Passmore & Eastwood 1986). Scurvy is a deficiency disease resulting from a lack of vitamin C (ascorbic acid) in the diet. It occurs in the absence of fresh fruit and vegetables but still can be avoided when these are not consumed if the diet is rich in cooked meat (Steinbock 1976; Steinbock 1993). Many writers

found that the disease could be cured by a variety of fresh fruits and vegetables, but medical learning was so constricted by Galen's classical pathology of "humours" that the concept of a deficiency disease was not realized until long after (Passmore & Eastwood 1986). Children who experience rapid and demanding growth show the signs of scurvy much more rapidly than adults (Stuart-Macadam 1989). Lack of ascorbic acid is responsible for the characteristic features of the disease, but such a diet is likely to lack other nutrients such as iron, folate, vitamin A and sometimes protein (Passmore & Eastwood 1986). As a result, ascorbic acid may relieve the predominant signs of the disease but it may not cure the patient. Although the diet may appear to be rich in ascorbic acid, it may in fact be counterproductive if the vitamin C has been destroyed by cooking (Passmore & Eastwood 1986). Ascorbic acid passes from the mother through the placenta to the fetus so vitamin C deficiency does not exist at birth. If no vitamin C intake occurs after birth it takes several months before the deficiency manifests itself (Ortner 2003). The infantile form of scurvy becomes apparent during the latter half of the first year, seldom before 4 months of age, and presents some skeletal manifestations similar to rickets. Unrelieved scurvy usually results in severe infection, most commonly pneumonia and eventually death (Steinbock 1976; Aufderheide & Rodríguez-Martin 1998; Ortner 2003).

Vitamin C is essential for the body to combat infection, absorb iron, and for the normal formation of the body tissues, especially osteoid and collagen (Zimmerman & Kelley 1982; Stuart-Macadam 1989; Roberts & Manchester 1995;

Aufderheide & Rodríguez-Martin 1998). Collagen is the main protein component of connective tissue, including skin, cartilage and bone (Stuart-Macadam 1989). The defect of collagen synthesis is manifested by an inability to produce effective osteoid (Aufderheide & Rodríguez-Martin 1998). As a result, the vascular extension into the cartilage zone, to resorb the cartilaginous cells and replace them with osteoid, does not occur. The presence of calcified cartilage creates an expansion of the zone of calcification, creating a radio-dense structure known as a "white line of Fraenkel" (Aufderheide & Rodríguez-Martin 1998; Riepe *et al.* 2001). A thin diaphyseal core is the result of continuing osteoclastic activity, with osteoblastic activity, impaired by defective osteoid formation during bone remodeling (Aufderheide & Rodríguez-Martin 1998; Ortner 2003).

Vitamin C deficiency predisposes the body to bleeding into the skin and beneath the periosteum (Zimmerman & Kelley 1982). Scurvy is a paleopathological rarity, likely due to non-recognition or misdiagnosis (Ortner 2003). Skeletal evidence of scurvy follows the identification of the bony reaction to hemorrhaging, periodontal disease, antemortem tooth loss and hemorrhaging into the joints (Roberts & Manchester 1995). An important characteristic of scorbutic hemorrhages is that they are commonly symmetrical (Stuart-Macadam 1989; Aufderheide & Rodríguez-Martin 1998). During the healing process, amounts of new bone are deposited beneath the periosteum producing a distinctive irregular thickening in the diaphyses (Steinbock 1976). The periosteum of infants is much more easily separated from the cortex; the resulting fragility is

such that the subperiosteal hemorrhage involves a proportionately greater area and volume (Aufderheide & Rodríguez-Martin 1998).

A number of anatomical sites within the skeleton are associated with scorbutic lesions and specifically, abnormal porosity and hypertrophic bone formation on the subadult skull. Ortner and Ericksen (1997) explain these changes as a reaction of bone tissue to: (a) chronic inflammation associated with bleeding, resulting from a combination of abnormal blood vessels formed when an individual has scurvy, and minor mechanical trauma from muscle contraction; and (b) the unusual anatomical relations of the branches of the maxillary artery. Although these lesions mimic those seen in anemia and infection, their distinctive anatomical location and association with chewing may differentiate them in most cases.

Skull – Abnormal porosity and hypertrophic bone formation are common on the orbital roof (**figure 4.12** below), cranial vault (**figure 4.13** below), lateral portion of the zygomatic in the orbit, internal surface of the zygomatic bone (**figure 4.14** below), posterior surface of the maxilla (**figure 4.14** below), the palate (**figure 4.15**), and infraorbital foramen (**figure 4.16**) (Ortner *et al.* 2001).

Figure 4.12 – Porous lesions of the orbit in scurvy (Ortner *et al.* 1999:326).

Figure 4.13 – Skull vault of infant with porous enlargement of the cranial bosses (Ortner & Ericksen 1997:214).

Figure 4.14– Porous lesions of the right posterior maxilla and internal zygomatic bone (Ortner *et al.* 1999:324).

Figure 4.15 – Abnormal porosity of the maxilla, palatine process and the palatine bone (Ortner *et al.* 1999:329).

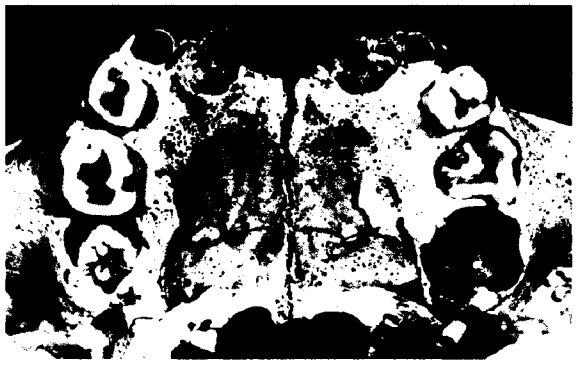


Figure 4.16 – Porous and hypertrophic lesions of the infraorbital foramen (Ortner *et al.* 1999:328).

The coronoid process of the mandible and medial surface can also be affected (**figure 4.17** below). Porosity of the orbital roof is often mistaken for the changes of anemia. Ortner and Ericksen (1997) report features on the external surface of the greater wing of the sphenoid pathognomonic of scurvy (**figure 4.18** below). These lesions are bilateral, and are characterized by porosity and hypertrophic bone formation in some cases. Teeth may be lost antemortem due to the

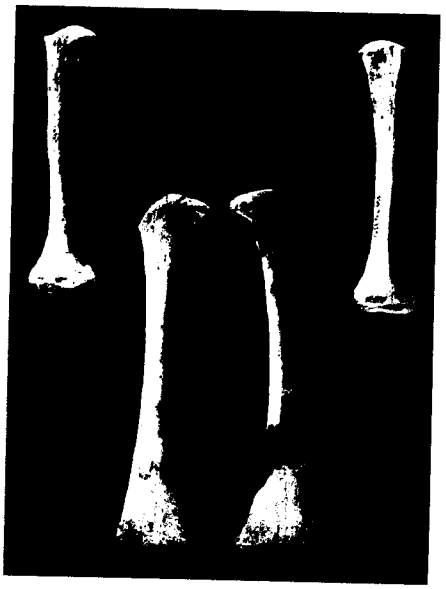
weakened collagenous attachment of the gingival to the periosteum. Antemortem remodeling of tooth sockets may provide evidence of this.

Figure 4.17 (left) – Abnormal porosity of the left, medial coronoid process in the mandible of a child 4 years of age (Ortner *et al.* 1999:329).

Figure 4.18 (below) – Abnormal porosity of the greater wing of the sphenoid (Ortner *et al.* 1999:323).

Long Bones – Cortical thinning and deposition of reactive periosteal bone can occur up to 1 centimeter thick (Ortner 2003). Enlarged zones of porosity may occur in the metaphyseal ends of the long bones (**figure 4.19** below) (Ortner *et al.* 2001).

Figure 4.19 – Porosity in metaphyseal ends of humerii & femur (Ortner *et al.* 2001:348).



Metaphyseal infraction (incomplete fractures) and fractures of the long bones can occur (Stuart-Macadam 1989). The metaphysis of the femur can cave in beneath the head, leading to a depressed angle of the femoral neck (Ortner 2003). Scorbutic infants may also show widening of the metaphyses, as a result of hemorrhaging in the wrists, ankles and knees (see **figure 4.19**). The hemorrhage into the bone inferiorly in infants is usually targeted at the metaphyses, between the osseous end of the bone shaft and the zone of calcified cartilage (Steinbock 1993; Aufderheide & Rodríguez-Martin 1998). This may result in the complete separation of the epiphysis, and the growth plate itself may be affected. Radiologically, a "ground glass" appearance in the metaphyses results from the resorption and poor formation of trabeculae (Steinbock 1976; Zimmerman & Kelley 1982). Maat (1986) states that the lower part of the lower extremity is affected most frequently as the result of stress by body weight. For the same reason, injuries to the bones of the legs are always symmetrical while unilateral changes are almost exclusively seen in the upper part of the body (Maat 1986).

Ribs – Bone adjacent to the osteochondral junction can be transversely fractured, resulting in the inward dislocation of the sternum and the rib cartilages (Ortner 2003). This produces a distortion in the costochondral junction of the ribs, creating a knobby appearance that bears some similarity to the rachitic rosary expression of vitamin D deficiency (Zimmerman & Kelley 1982). The scorbutic beading of the ribs is more angular and less knobby than in rickets, while subperiosteal hemorrhages often appear on the rib shaft (Steinbock 1976).

Flat Bones – Subperiosteal hemorrhages can occur on the pelvic bones but are more commonly seen on the supraspinous and infraspinous fossa of the scapulae (Ortner 2003).

4.4.2 Skeletal Manifestations of Iron-Deficiency Anemia

Anemia is defined as a reduction in the concentration of hemoglobin and/or red blood cells below normal levels. Iron is necessary for the development of hemoglobin in the newly formed red blood cells in bone marrow. In those who suffer from anemia, red blood cells become small and pale and experience a shortened lifespan of up to half the normal life of 120 days (Roberts & Manchester 1995). Storage of iron takes place in the liver and the spleen when old blood cells are broken down. Ninety percent of the iron in old red blood cells is needed in order to form new cells (Roberts & Manchester 1995). As iron deficiency develops, the stores of iron are depleted, while the body attempts to absorb increased amounts of iron. In addition to hemoglobin formation in red blood cells, and the transfer of oxygen to the body cells, iron is required for transmission of nerve impulses, for collagen (protein) synthesis and contributes to immune system strength (Roberts & Manchester 1995).

Bone changes attributed to anemia are most common in childhood and vary in extent and severity (Stuart-Macadam 1992; Roberts & Manchester 1995). Specific criteria for identification of anemia in archaeological groups have been identified. Many anemias are reflected by changes in skeletal mass as a result of

widening of the marrow cavity and as a consequence of decreased bone mass relative to bone volume (Garn 1992).

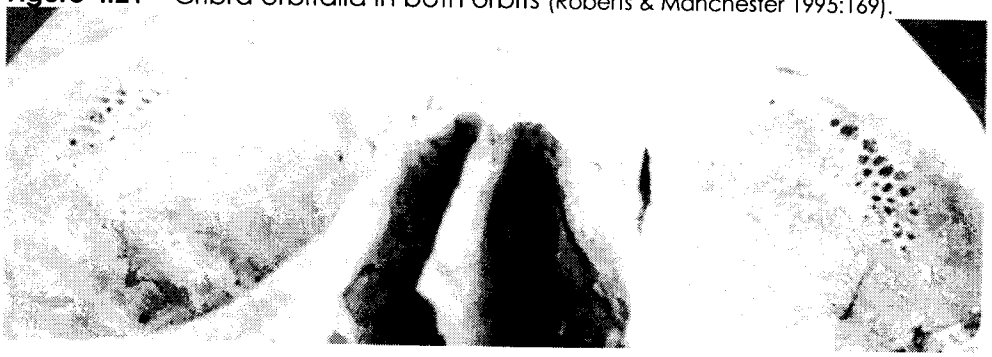
Porotic hyperostosis and cribra orbitalia are useful in determining the possibility of nutritional stress (Steinbock 1976; Steinbock 1993; Roberts & Manchester 1995; Aufderheide & Rodríguez-Martin 1998; Ortner 2003). These bony manifestations are not diagnostic of a specific type of anemia, but serve as general indicators of iron deficiency anemia, but see Hershkovitz (1997) for an alternative view. Iron deficiency anemia may result from blood loss, parasitic infection, dietary deficiencies and/or the inadequate absorption of iron (Steinbock 1976; Roberts & Manchester 1995; Ortner 2003).

Skull - Porotic hyperostosis is characterized by symmetrically distributed cranial lesions involving the outer table of the frontal and parietal bones and rarely, the occipital. Radiographically, bone changes include "hair on end" trabeculation, outer table thinning, texture changes, diploic thickening, orbital roof thickening and orbital rim changes (Stuart-Macadam 1992). In a well-developed lesion, the affected areas of the skull are thickened by expansion of the diploic layer, and the outer table overlying the lesions has been resorbed completely (**figure 4.20** below) (Steinbock 1993).

Figure 4.20 - Cranial vault lesions in anemia (Roberts & Manchester 1995:168).

This reveals the trabeculae of the expanded cancellous bone, while coarsening of these trabeculae is evident. These changes are the result of the body's attempt to produce more red blood cells in the marrow to compensate for the lack of iron. In less advanced cases, the outer table is incompletely resorbed which appears as multiple pinhead-sized perforations (Steinbock 1993). Cribra orbitalia is a similar, but smaller lesion, specifically located in the orbital roof (**figure 4.21** below). Orbital and vault lesions of porotic hyperostosis can occur concurrently or independently. Orbital lesions may appear alone, but vault lesions rarely occur without involvement of the orbits (Stuart-Macadam 1992). Cribra orbitalia is considered more common in a population, which suggests vault lesions are indicative of a more severe form of anemia, and is therefore regarded as a more sensitive indicator of the underlying stimulus of anemia (Stuart-Macadam 1989; Steinbock 1993).

Figure 4.21 – Cribra orbitalia in both orbits (Roberts & Manchester 1995:169).



Postcranial skeleton - Postcranial changes related to iron-deficiency anemias are less well known. It had been suggested that a lack of involvement of the postcranial skeleton was a diagnostic factor in differentiating between iron-deficiency anemia and genetic anemia (Stuart- Macadam 1989). It is now apparent that postcranial changes do occur, but with less frequency and severity, than those seen in genetic anemias (Stuart-Macadam 1989). Other postcranial changes attributed to anemias in general are mild to severe osteoporosis of the pelvic bones and lumbar vertebrae (Stuart-Macadam 1989). Widening of the hand bones due to an expansion of the medullary space and thinning of the cortices was found in children (Stuart-Macadam 1989).

Iron deficiency anemia is the most common form but two genetic anemias, thalassemia and sickle cell anemia, produce similar lesions in the skull, and both produce changes in other areas of the skeleton as well (Roberts & Manchester 1995). Thalassemia is a genetically determined disorder caused by a problem in hemoglobin synthesis where pale cells with too low hemoglobin content are produced, but are rapidly destroyed (Steinbock 1976). A high frequency is seen

in populations of Mediterranean origin and also south-east Asia (Stuart-Macadam 1989; Aufderheide & Rodríguez-Martin 1998). Thalassemia shows bone abnormalities in the paranasal sinuses, mastoids and facial bones, in addition to the vault and orbital lesions. The metacarpals and metatarsals enlarge, with thinning of their cortices and a generalized osteoporosis of the spine develops.

Sickle cell anemia results in the distortion of the red blood cells. The skeleton shows the effects of changes in the skull, vertebrae, pelvis, hand and foot bones. These occur due to marrow over activity and enlargement as the body tries to produce more red blood cells (Steinbock 1976). Necrosis of the bone occurs due to blockage of the blood vessels by these abnormally shaped cells (Steinbock 1976).

Skeletal changes attributed to anemias are common to all types, making the diagnosis of a specific type of anemia difficult on this basis alone (but see Hershkovitz *et al.* 1997). Geographic location or ethnicity may narrow down the list of possible anemias (Roberts & Manchester 1995; Aufderheide & Rodríguez-Martin 1998). For example, there is no unequivocal evidence of sickle cell anemia or thalassemia in the pre-Columbian New World, so iron deficiency anemia is usually used to explain the presence of porotic hyperostosis and cribra orbitalia in these areas (Aufderheide & Rodríguez-Martin 1993). In some cases of porotic hyperostosis, the anemia is a genetic type, found mostly in geographic areas where malaria was common and balanced polymorphism evolved as an adaptive mechanism. Stuart-Macadam (1992) states that it is more likely that most cases of porotic hyperostosis reflect an acquired anemia, such as iron-

deficiency anemia, and cites three lines of evidence to support this. First, according to calculations based on the highest gene frequencies for genetic anemia in modern populations, the probability of finding individuals from archaeological collections with skeletal changes as a result of genetic anemia are quite low (Stuart-Macadam 1992). Secondly, high levels of porotic hyperostosis exist in groups from northern Europe and North America, where genetic anemias are not known to have existed in the past (Stuart-Macadam 1992). Lastly, severe bone changes as a result of genetic anemias, particularly as they affect the postcranial skeleton, have rarely been found in archaeological collections.

4.4.3 DIFFERENTIATING RICKETS, SCURVY & ANEMIA

There is often a lack of similarity between clinical knowledge of a disease, and what is actually observed in skeletal remains. Stages of different diseases may have a similar skeletal morphology and thus, present a difficulty of differentiation. The morphological amalgamation of different pathological processes is not readily apparent, but something observers should be aware of. These problems further illustrate that careful attention to anatomical detail, combined with extensive knowledge of pathological processes that affect bone tissue are necessary to establish differential diagnoses.

There is no definitive protocol for the morphological assessment of rickets in skeletonized individuals. Overlap between rachitic traits and those of other diseases makes them ineffective for the purpose of identification (see **Table 4.1** below). Single traits should never be looked at in isolation. Instead, knowledge of a combination of traits, and possibly, the individual frequency with which they appear in rachitic individuals should be employed. Unfortunately, the paleopathological literature does not provide references to the frequency of rachitic traits found in archaeological samples.

TABLE 4.1 - Summary of Pathological Features associated with rickets, scurvy & anemias.

Location	Rickets	Scurvy	Anemia
Skull	- Cranial vault/orbital roof porosity - Glabellar/facial bone porosity - Delayed closure of the fontanelles - Posterior flattening or lateral & asymmetrical deformity of skull - Squared head (frontal & parietal bossing - Thickening of the cranium w/ reduction of outer and/or inner table - Deformed mandibular ramus - Delayed tooth eruption & hypoplasia	- Greater wing of sphenoid hyperostosis - Maxilla, posterior porosity - Zygomatic bone internal porosity - Cranial vault/orbital roof porosity - Cranial vault/orbital roof hyperostosis -Squared head (frontal & parietal bossing) - Slight thickening of the cranium - Lateral margin porosity of the orbit - Infraorbital foramen porosity & hyperostosis - Palate porosity - Coronoid process porosity - Antemortem tooth loss	- Porotic hyperostosis - Cribra orbitalia - Porosity in paranasal sinuses, mastoids & facial bones
Long bones	- Deformed arm bone - Flaring & cupping of metaphyses - Deformed leg bones - Metaphyseal infraction & fractures of long bones - Cortex porosity of the metaphyseal ends of long bones - Growth plate abnormality of long bones - Thickening of long bones	- Cortex porosity of the metaphyseal ends of long bones - Cortical thinning and deposition of reactive periosteal bone - Metaphyseal infraction & fractures of long bones - Widening of the metaphyses in the wrists, ankles & knees	- Expansion of medullary space & thinning of cortices in hand bones
Ribs	- Rachitic rosary - Pigeon or funnel breast - Harrison's grooves - Flared costochondral end of the ribs - Flattening of rib curvature - Cortex of costochondral ends of ribs irregular & porous	- Scorbutic beading of the ribs - Thickening of ribs toward costochondral junction - Transverse fracture of ribs adjacent to the costochondral junction	N/A
Scapulae	N/A	- Supraspinous fossa porosity & hyperostosis - Infraspinous fossa porosity & hyperostosis	N/A
Vertebrae	- Vertebrae compressed and porous	N/A	- Mild to severe osteoporosis of the lumbar vertebrae
Pelvis	- Abnormal curvature and plumpness of ilium	- Pelvic bone hyperostosis	- Mild to severe osteoporosis of the pelvis

Individually each trait is capable of substantially increasing one's chance of correctly diagnosing rickets. The correctness of the diagnosis of rickets will increase with the number of traits found in an individual, when using traits in

combination. In arriving at a final trait list, numerous traits were deemed less diagnostic due to associations with scurvy, anemia and/or general malnutrition. Therefore, morphological traits exhibiting consistency with no overlap or problems associated with age were emphasized (primary traits) while the less optimal characteristics were given secondary consideration. Each trait was evaluated individually – see below.

Many of the diagnostic traits of rickets, scurvy and anemia, refer to porosity as an indicator of disease. Porous bone or porosity are defined after Ortner and Ericksen (1997), as a localized abnormal bone condition in which fine holes, visible without magnification, but less than 1 mm in diameter, penetrate a lamellar bone surface. Porous bone is the result of chronic inflammation and needs to be distinguished from porotic hyperostosis, a porous condition of bone resulting from hyperplasia of hematopoietic marrow (Capasso *et al.* 1995; Ortner *et al.* 2001). Porosity occurs in the subadult skeleton as a normal feature of growth and remodeling, so caution must be used when assessing pathological conditions based on porosity alone. It would be ideal in the future to establish a range of normal and abnormal expressions of porosity for subadult bones tissues. Helpful in making this distinction is the extent of the porous lesion relative to the long axis of the bone (Ortner *et al.* 2001). Ortner and colleagues (2001) state that cortical bone porosity rarely extends beyond 5-10 mm from the growing end of the metaphysis in normal growing subadult bone. The extent of this normal porous zone varies within, and between bones, and increases with the age of the individual (Ortner *et al.* 2001). Another problem faced when using porosity as

a disease indicator are issues of preservation, as taphonomic processes may also create porosity.

Bone is limited in the number of ways in which it can react to disturbances in metabolism. Metabolic bone disturbances generally result in the reduction of bone mass by inadequate osteoid production, inadequate osteoid mineralization or excessive resorption of normal bone tissue (Steinbock 1976). It is a well-documented fact that in the past, scurvy and rickets often occurred in the same individuals (Park 1923; Ortner 2003). Thomas Barlow was the first to clarify the true nature of infantile scurvy in 1883 and attempt to differentiate it from rickets (Barlow 1883; Evans 1983; Rajakumar 2001). The presence of one disease does not seem to have an inhibiting effect on the expression of the other, and the question of which disease is dominant over the other is unclear (Ortner 2003). Follis and colleagues (1940) state it is possible that an antagonism exists between vitamin C and vitamin D in that a deficiency in one vitamin may prevent a deficiency in the other from showing itself in characteristic lesions, signs and symptoms. Ortner & Putschar (1981) argue that vitamin D deficiency inhibits vitamin C deficiency from expressing itself). In contrast, Park and colleagues (1935) state that as vitamin C deficiency inhibits osteoid formation, expression of rickets would be inhibited by scurvy. If the deficiency of vitamin C is extreme and advanced, the signs can be recognized histologically even in the presence of extreme rickets (Follis *et al.* 1940). The most characteristic sign lies in the connective tissue formations carpeting the spaces between the trabeculae (Follis *et al.* 1940). From a histologic point of view vitamin D deficiency definitely

inhibits vitamin C deficiency from expressing itself and may in some cases mask vitamin C deficiency altogether (Follis *et al.* 1940). Rickets may mask all radiographic evidence of scurvy, specifically, the bright band at the end of the shaft, the characteristic clefts and zones of rarefaction in and beneath the lattice of the calcified cartilaginous matrix framework and the shadows cast by the periosteum elevated as the result of hemorrhage (Follis *et al.* 1940). Rickets also interferes with the clinical signs of scurvy as well, such as enlargement of the costochondral junctions, epiphyseal separation and rickets may give rise to pain through fracture (Follis *et al.* 1940). Deficiency in vitamin C should interfere with development of signs of rickets. Osteoblastic activity is inhibited by vitamin C deficiency and the development of the osteoid covering of the bones is diminished or stopped. Follis and colleagues (1940) found that osteoid formation can be well developed even in the presence of scurvy.

Rickets is similar to scurvy in that its effects are most noticeable in areas of rapid bone growth. Scurvy and rickets share several features: cranial vault/orbital porosity, squaring of the skull due to woven bone deposition on the parietal and frontal bones, thickening of the cranium, porosity of the metaphyses of the long bones, widened metaphyses of long bones (especially those of the lower limb), metaphyseal infraction/fracture of long bones, and prominent "knobby" costochondral rib junctions, similar to the "rachitic rosary" (**table 4.1** above). While these features are produced by different mechanisms, to which often enough would be sufficient to differentiate between the two, in cases of archaeological remains taphonomic processes often complicate this matter.

Cranial/vault porosity and thickening of the cranium are found in rickets, scurvy and anemia. Various disease processes often preferentially affect groups of bones, specific bones and specific locations of bones. Predilection or a lack there of is an important aspect in itself. It is this concept that allows for the separation of rachitic and scorbutically induced porosity of the skull. When diagnosing rickets it is important that the trait appear bilaterally. This aids in diagnosis in order to differentiate between localized infection and taphonomic processes. In rickets, porosity is found at the glabella and on the facial bones. Porosity in the facial bones should be bilateral; if not, localized infection may be the cause. In scurvy, the presence of other areas of cranial porosity, such as the greater wing of the sphenoid, maxilla, palate, zygomatic bone, and infraorbital foramen make it possible to differentiate between the two diseases. To differentiate between rickets and anemia, porous bone, the result of chronic inflammation, can be distinguished from porotic hyperostosis, resulting from hyperplasia of hematopoietic marrow. Bone marrow reactions in porotic hyperostosis produce labyrinth-like lesions of the skull and expansion of the diploë (Ortner 2003).

In rickets, deposition of subperiosteal cancellous bone does not show labyrinth-like lesions. Conversely, the atrophic form of rickets also shows wide spaces accompanied by relatively few, and thin, spongy bone trabeculae. Radiographs may further aid in diagnosis. As cranial porosity is indicative of all three metabolic diseases, caution should be used. It is clear that skeletal

manifestations of this trait may often be indistinguishable. It would be wise to look for other evidence of rachitic activity in other areas of the skeleton.

Posterior flattening of the cranial vault, or lateral asymmetry, may result when the area is subjected to pressure when the infant is resting. Some general degree of flattening is seen in infants due to the open fontanelles and subsequent pliability of the infant cranium. In children with rickets, the cranium is softened, which would produce more extreme degrees of flattening. Identification of this trait as rachitic should depend on the degree of flattening. In archaeological remains it is often rare that the skull is in one piece. Pettifor and colleagues (1984) found that craniotabes (abnormal softness of the bone in the area of the occipital and parietals along the lambdoidal suture) is a common finding in 3 month old infants and is of no help in diagnosing rickets in young infants.

The rachitic squared head is a product of thickening of the outer eminences of the frontal and parietal bones and is thought to be a clinical hallmark of rickets. However, porous enlargements of the cranial bosses are also found in scurvy (Ortner & Ericksen 1997). In comparing the rachitic squared head found in **figure 4.2** (pg. 39) to the scorbutic squared head found in **figure 4.13** (pg. 54) it was concluded this trait was too similar to be deemed a primary trait.

The mandibular ramus may show abnormal medial-posterior bending due to muscle action during chewing (Ortner & Mays 1998). Observation of this trait demonstrated that correct diagnosis is dependant upon knowledge of the range of variation in infant mandibular form.

Dick (1916) described as "almost pathognomonic of rickets" the forms of enamel hypoplasia symmetrically involving upper and lower permanent incisors, canines and first molars. This trait was observed in 20% of rachitic London schoolchildren. An association of rickets with interglobular dentine (IGD) was discovered by Mellanby (1929) through experimentation with puppies deprived of sunlight. Interglobular dentine within the crown of the tooth is often found with linear pitting and staining of the enamel surface (Ivanhoe 1994). Mellanby (1934) reported a higher prevalence of severe grades of enamel hypoplasia and IGD in deciduous and permanent teeth, extracted from children of the lower socioeconomic classes, where rickets was rampant. This data was compared to teeth of children from a higher socioeconomic class where rickets was much less common and subsequently, enamel hypoplasia was found to be much less common (Mellanby 1934). The presence of dental enamel hypoplasia is a good indicator of *general* nutritional stress (Johnston & Zimmer 1989). Enamel hypoplasia is, however, a common occurrence and cannot be used as a diagnostic characteristic of rickets in isolation.

Porosity of the metaphyses of the long bones is quite similar in both scurvy and rickets. Unless thickening of the long bones accompanies porosity, a diagnosis of rickets cannot be certain. Observation of thickening is best carried out when one has a normal bone for comparison. In diagnosing porosity of the metaphyses, cortical bone porosity should extend 10 mm from the growing end of the metaphysis in order to be considered abnormal (Ortner *et al.* 2001). The age of the individual should be taken into consideration as the extent of normal

zone of porosity varies within, and between bones, dependant upon age (Ortner *et al.* 2001). Observation of this trait suggests caution, as taphonomic processes may create porosity.

The most obvious and the trait considered most pathognomonic of rickets are the bending deformities seen in the femur, tibia and fibula, ulna, radius and humerus. Observation of this trait reveals that it is comparatively easy to diagnose, provided one is familiar with the normal variation of curvature in these bones in the subadult skeleton. It is essential to confirm that taphonomic processes that produce warping of the bones are not responsible for these deformities.

Metaphyseal infraction and fracture are common in both scurvy and rickets, and appear relatively similar in radiographs. It is suggested that infraction and fracture be used as a secondary trait only.

In rickets, metaphyseal widening, cupping and flaring is the result of extreme abnormality in the metaphyseal-epiphyseal junction due to multiple instead of single ossification centers and irregular cartilage proliferation (Aufderheide & Rodríguez-Martin 1998). In scurvy, metaphyseal widening, cupping and flaring is the result of ossification of subperiosteal hemorrhages whose form is often recognizable (Stuart-Macadam 1989). When examining these traits radiographically, difficulty was met in trying to discern between metaphyseal widening as a result of ossification of hemorrhages, and the result of multiple ossification sites and irregular cartilage proliferation. It is suggested

that metaphyseal widening be used in combination with other traits of rickets, but not in isolation.

Growth plate abnormalities in long bones consist of porosity and roughening of the epiphyseal plates. Some difficulty was met when trying to determine a normal range of variation in porosity of growth plates in infants. Further observation of this trait is required.

The rachitic rosary characteristic of rickets is produced by an arching cartilaginous mass above which the edges of the osseous portion of the rib projects discretely (Aufderheide & Rodríguez-Martin 1998). For the most part, cartilage does not survive the burial environment and this trait is diagnosed skeletally by observing flaring of the bony rib ends. The enlargement of the costochondral junction in scurvy is caused by subperiosteal hemorrhage, whose ossification thickens the bony rib, expanding the rib ends, ending abruptly at its cartilaginous junction (Stuart-Macadam 1989). The scorbutic beading of the ribs is more angular and less knobby than in rickets.

The rachitic pigeon breast deformity is the result of the pushing out of the sternum due to the costochondral areas of the ribs becoming nodular prominences and appears to be unique to rickets. This trait is shown in the reduced curvature of the ribs, a unique feature of rickets. In scurvy, ribs adjacent to the costochondral junction can be transversely fractured resulting in the inward dislocation of the sternum. In rickets, the arched costochondral junction pushes the sternum dorsally, creating a funnel-shaped breast. These skeletal

manifestations are similar, but may be differentiated by the macroscopic or radiographic presence of fracture.

Harrison's groove, a circular depression in the lower rib cage, is a trait unique to rickets. In rickets, the cortex of the costochondral ends of the ribs show flaring and porosity, specifically pitting. Cortical bone porosity should extend 10 mm from the growing end of the subadult rib in order to be considered abnormal. The irregularity and porosity of rachitic ribs needs to be differentiated from the scorbutic subperiosteal hemorrhages appearing on the rib shaft.

In severe rickets, the vertebrae may become porous resulting in a decrease in height due to compression. This is often accompanied by a scalloped appearance of the end plate. In anemia, mild to severe osteoporosis of the lumbar vertebrae may be present. Postcranial changes in general anemia occur less frequently than those seen in genetic anemias (Stuart-Macadam 1989). Compression of the vertebrae is likely the result of rickets, while porosity may be due to anemia or taphonomic processes. Observation of this trait suggests caution in diagnosis.

During active disease, the rachitic pelvis exhibits abnormal lateral curvature and appears plumper than normal. These traits are unique to rickets but a normal pelvic bone should be used for comparison.

Age related traits, when diagnosing rickets in archaeological populations, are also problematic. Age related traits, such as the delayed closure of the fontanels, delayed tooth eruption and stunting, are of limited use as there are no

records to compare with actual age. Therefore, these traits were removed from the trait list.

The primary features of rickets are as follows: glabellar/facial porosity, deformed mandibular ramus, deformed leg and arm bones, thickening of long bones, flared costochondral ends of the ribs, cortex of costochondral ends of ribs porous and irregular, flattening of rib curvature, and abnormal curvature, plumpness of the ilium and vertebral compression and porosity. Secondary features of rickets include: cranial vault/orbital roof porosity, squared head, thickening of the cranium, dental deformation and enamel hypoplasia, growth plate abnormalities of the long bones, metaphyseal infraction & fractures of long bones, flaring, cupping and cortex porosity of the metaphyseal ends of the long bones. This study found that that for the most part rickets is better diagnosed by traits that result in bone deformities, for example shape or girth, rather than by the presence of porosity.

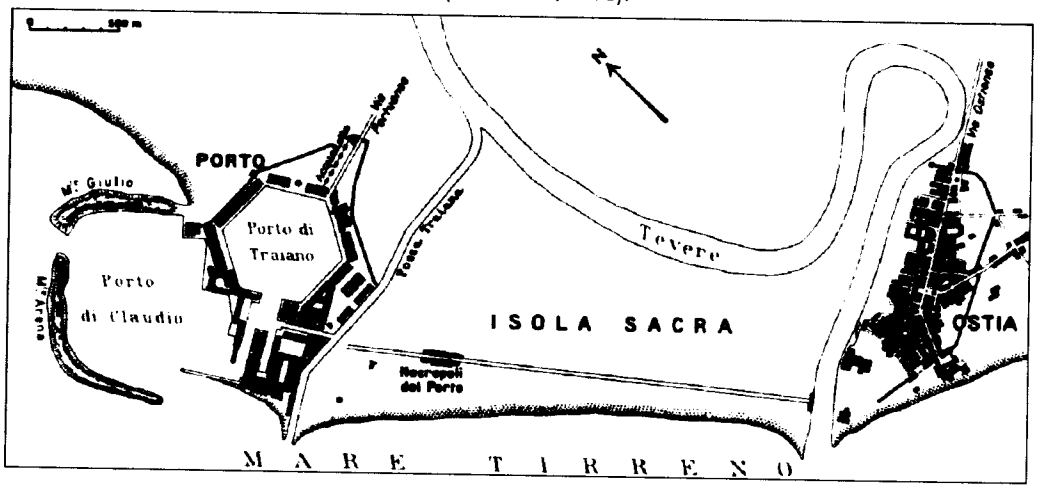
Chapter 5

MATERIALS & METHODS

5.1 *The Necropolis of Isola Sacra*

The examination of infantile rickets among the children of the classical Roman Period was accomplished through the study of human skeletons of Isola Sacra, an Imperial Roman necropolis of the 2nd-3rd century A.D. Isola Sacra is located approximately 23 kilometers west of Rome between the urban centres of *Ostia Antica* and Fiumicino (Pavolini 1996). It is situated on an artificial island that was created during the dredging of a canal, the *Fossa Traiana*, in 103 A.D that connected the Tiber River with the coast (**figure 5.1**) (Mannucci & Verduchi 1996).

Figure 5.1 – Map showing the location of *Portus Romae* ('Porto' on map), *Ostia Antica*, and the necropolis of Isola Sacra (Rossi *et al.*, 1998).



Stretching along 1.5 km of ancient road connecting *Ostia Antica* and *Portus Romae*, the necropolis contains inhumations and cremations of the population from the nearby city of *Portus Romae* (Calza & Becatti 1974). The necropolis was used by the inhabitants of *Portus Romae* from the 1st to 3rd centuries A.D. (**figure 5.2**, below). The cemetery eventually fell into disuse and was gradually covered over by encroaching sand (Sperduti 1995). The area around *Portus Romae* is now completely surrounded by land, and Fiumicino airport now occupies the area to the NE of Trajan's harbour.

The necropolis of Isola Sacra is comprised of a wide variety of burial structures, ranging from simple interments in sand to monumental multiple tombs reflecting the complex and diverse funerary practices of the Roman period. Seventy-five chamber tombs have been uncovered at Isola Sacra (**figure 5.3, 5.4 & 5.5**, below) (Prowse 2001).

Figure 5.2 – Layout of the Isola Sacra necropolis (Baldassarre 1996: appendix).

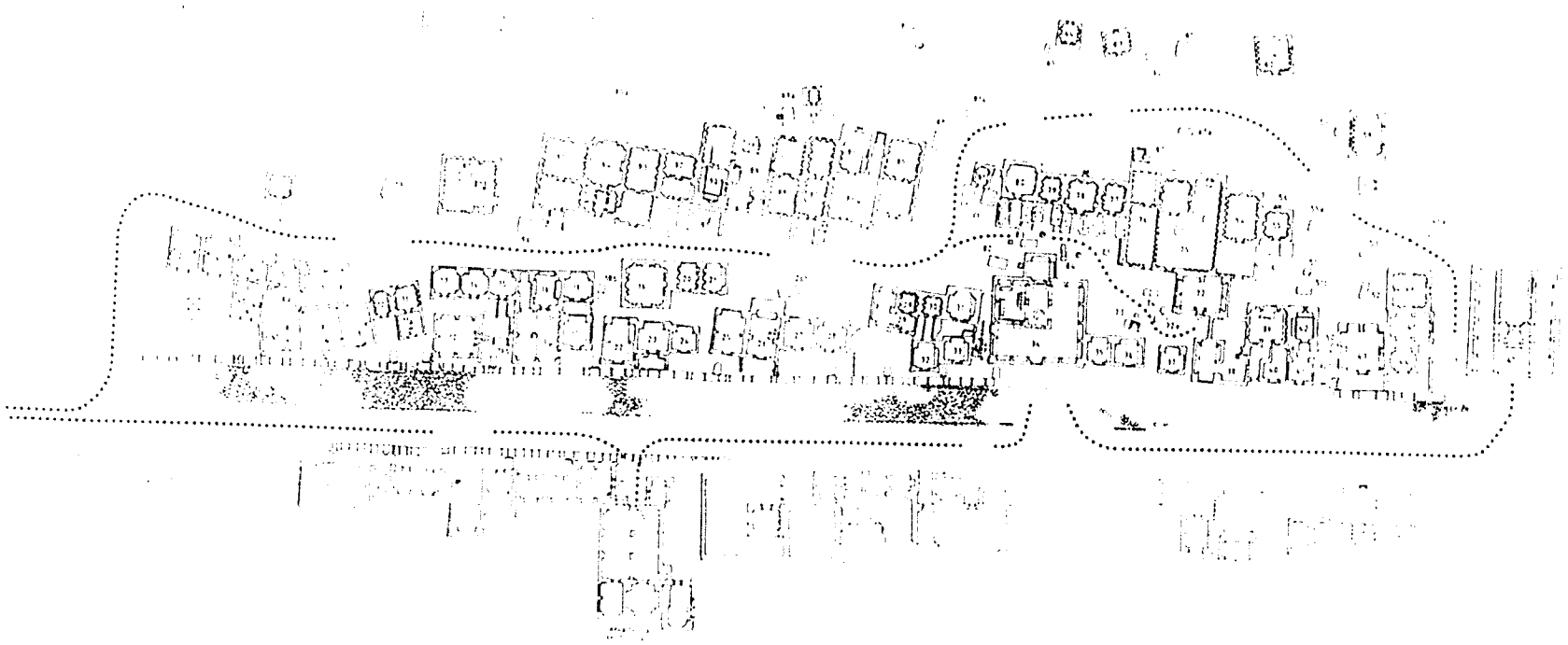


Figure 5.3 – Chamber tombs at Isola Sacra (photo S.R. Saunders).

Figure 5.4 – Chamber tombs at Isola Sacra (photo S.R. Saunders).

Figure 5.5 – Sarcophagus within chamber tomb (photo J. Barta).

Single internments of different kinds are interdispersed among structures of different dimensions, which contained both single and multiple burials (Angelucci *et al.* 1990). Soil and sand burials are interments in the ground without any evidence of a protective structure while inhumation burials are associated with coffins made of wood or terracotta bricks (Angelucci *et al.* 1990). 'Cappuccina' burials are covered by a series of large terracotta tiles (*tegulae*) stacked to form a roof over the burial structure (Angelucci *et al.* 1990). Many of the infants and children had been buried in large storage vessels called amphorae (**figure 5.6**) (Prowse 2001).

Figure 5.6 – Amphorae burials (Angelucci *et al.* 1990:75).

Columbaria burials are rectangular barrel-vaulted tomb chambers with the remains stored in urns placed in niches in the walls (Meiggs 1960; Toynbee 1971). Columbaria differed in size and capacity, with many of them containing more than one hundred urns, facilitating long term use (Meiggs 1960). The majority of descriptions found in archaeological reports focus on the architectural features of the monumental tombs, so there is comparatively little information on the secondary burial structures found at the site (Prowse 2001).

The skeletal collection from Isola Sacra includes the remains of approximately 2000 individuals of both sexes and from all age groups (Sperduti 1995). Many of these are commingled remains from Calza's early excavations, but there are over 800 skeletons that have been individually catalogued and analyzed (Prowse 2001). This sample is housed at the Pigorini National Museum of Prehistory and Ethnography, Rome Italy, and represents one of the most significant skeletal collections from Mediterranean Europe, for the Classical Period.

G. Calza first excavated Isola Sacra between 1925 and 1940 and uncovered some of the large monumental tombs (Prowse 2001). The next excavations took place between 1973 and 1982, and were carried out by the Archaeological Superintendency of Ostia, the University of Rome 'La Sapienza', and the University Institute of Oriental Studies of Naples (*ibid*). The main focus of their efforts were to restore the monumental tombs and recover the human skeletal remains excavated by Calza that had been haphazardly dumped back

into the tombs once his excavations were completed (Baldassarre 1984, 1990; Rossi *et al.* 1998). During this time approximately 1,000 individuals were recovered (Baldassarre *et al.* 1985; Angelucci *et al.* 1990).

The most recent excavations at Isola Sacra occurred in 1988 and 1989 (Prowse 2001). Focusing on areas in between the monumental tombs, the excavation yielded 600 additional single and multiple burials to which the majority of the subadult sample used here belong (Baldassarre 1990). In 1992 all skeletal material recovered from Isola Sacra was entrusted to members of the Anthropology Section of the L. Pigorini museum, whose commission it was to identify, catalogue, and study the skeletal sample.

According to Baldassarre (1984), the oldest tombs (1st century A.D.) are located closest to the road, while the next phase of the cemetery corresponds to the construction of tombs located further back. Toward the end of the cemetery's use some of the existing structures were in fact reused (*ibid.*).

5.2 Portus Romae during the Roman Imperial Period

The early Roman Empire was characterized by a period of relative political and economic stability beginning under the rule of Augustus (63 B.C. – A.D. 14). During this time the Romans enjoyed increased urbanization, wider distribution of trade goods, and expansion of agricultural activities (Alston 1998). Taxation of imported goods was low and control of trade was left largely in the hands of private traders and entrepreneurs, greatly encouraging trade throughout the Mediterranean region (Prowse 2001). The grain supply however was imported from the provinces under the close supervision of Roman officials and soldiers (Alston 1998).

It was during the 1st century A.D. that the original port at *Ostia Antica* (ca. 3 km to the South) was no longer able to accommodate heavy ship traffic due to progressive silting of the waterway (Prowse 2001). Claudius then ordered the construction of a new port, built between 42 and 64 A.D., and Trajan ultimately completed an additional inner harbor in 112 A.D. This port complex was known as *Portus Ostiensis* or *Portus Augusti* until the 4th century A.D. (Mannucci & Verduchi 1996).

The construction of *Portus Romae* did not automatically result in the decline of *Ostia Antica*. *Ostia Antica* increased its economic development which changed it from a market town to an affluent residential city (Mannucci & Verduchi, 1996). All main administrative activities continued to occur at *Ostia Antica*, while *Portus Romae* was considered an extension of the first port with

many of its earliest inhabitants coming from *Ostia Antica* and from Rome (Mannucci & Verduchi, 1996). By the end of 2nd c. A.D., *Portus Romae* was handling all commercial traffic coming in to Rome (Prowse 2001).

Commercial activity at *Portus Romae* started to decline by the end of the 4th century A.D., but it remained a harbor for the Roman fleet during the Vandal invasion in the 5th century and the Gothic war of the 6th century A.D. (Prowse 2001). With the decline of the Roman Empire after the 5th century A.D., *Portus Romae* too had collapsed, and by the 9th century it was completely abandoned (Prowse 2001).

Trajan's port (*Portus Romae*) was directly linked to Rome through a series of docks and quays along the Tiber River. Trade activities at *Portus Romae* were also supported by a series of smaller ports along the coast through interconnecting rivers and roads (Rickman 1996). *Portus Romae* held an exclusive commercial role because it was the trading entry point to the Imperial city of Rome (Prowse 2001). The social status of the inhabitants was considered middle-class, their occupation cited as administrators, traders, and merchants (Meiggs 1960; Mannucci & Verduchi, 1996). The distribution of burials between the tombs appears to be fairly homogeneous, and there is no evidence for systematic expansion of the cemetery that implies a special location for 'poorer' burials (Angelucci *et al.* 1990). The majority of inhumations did not have any associated grave goods, with the exception of some coins, lamps, and 'female' ornaments (Sperduti 1995).

The great wealth of the city of Rome was the result of the political and economic stability of the Mediterranean region at the beginning of the Roman Imperial period. Due to its exclusive role, the lives of the inhabitants of *Portus Romae* were directly linked with the prosperity and political domination of the Roman Empire.

The main living quarters of the inhabitants of *Portus Romae* were found to the East and South of the hexagonal basin (Prowse 2001). Much of the port complex is now buried under the modern Fiumicino Airport. Luckily *Ostia Antica* is extremely well preserved, offering some information about the organization of Rome's port towns.

5.3 *Estimation of Age of the Isola Sacra Subadult Skeletal Sample*

Age at death was estimated by A. Sperduti (1995) for her PhD dissertation on the palaeodemography of the Isola Sacra sample (University of Rome, 'La Sapienza'). Aging methods used for the subadult skeletons were based on the development and emergence of the deciduous and permanent dentition, development of the temporal and occipital bones, development and fusion of the epiphyses, and maximum long bone diaphyseal length (see Sperduti, 1995).

5.4 Diagnosis of Rachitic traits in an archaeological population

Observation of rachitic traits took place during two weeks of analysis, during which time 282 subadult skeletons were examined morphologically for diagnostic indicators of rickets. Age estimates of the Isola Sacra subadults range from birth/fetal (0 – 0.25) to 15 years of age. Bone preservation is generally good, but of these individuals, 98 had to be excluded due to their fragmentary state. In addition, 2 individuals lacked any data for age and were also excluded. The final number of individuals for analysis is 182. A full discussion of the diagnosis of rachitic individuals in an archaeological context is found in Chapter 4, pages 63-74.

Precision is a measure of an observer's ability to reproduce her/his results and is both a reflection of the researcher's capabilities as well as a commentary on the nature of the features being examined. Low precision (high intraobserver error) indicates that a feature cannot be assessed reliably. Intraobserver tests stress the importance of familiarizing oneself with the range of variation present in diagnostic criteria. Morphological traits show wide ranges of expression; in order to remain consistent, calculation of intraobserver error can be found in **table 5.1** (below). The first observation took place in Rome, Italy in May of 2002 on the skeletons themselves. In addition, skeletal lesions were diagnosed by another observer (S.R. Saunders) during the Rome observation. The second observation took place in August of 2003 and was carried out by the author using photographs of the skeletal elements and by reviewing the data collected in

Rome. The discrepancy between the first and second observation is likely due to an increased familiarity with rachitic traits as well as confirmed knowledge of primary traits.

TABLE 5.1 – Calculation of Intraobserver error

First Observation		
Positive	Negative	Uncertain
24	132	27
Second Observation		
Positive	Negative	Uncertain
27	132	24
Difference		
3	0	3

Rickets, scurvy and iron deficiency anemia can occur in any combination with any, or all other metabolic diseases. Rickets and scurvy can express somewhat similar skeletal symptoms. A number of anatomical sites are associated with scorbutic lesions, specifically abnormal porosity and hypertrophic bone formation on the subadult skull. Although these lesions mimic those seen in rickets and infection, their distinctive anatomical location and association with chewing may allow for differentiation in most cases. Iron-deficiency anemia shares with rickets porosity of the orbital roof, frontal and parietal bones. Iron deficiency anemia can sometimes be distinguished from rickets by the location and form of porosity.

To differentiate between genetic causes of rickets and those of a deficiency origin the skeletal expression and age at onset of the disease was determined. The frequencies with which these genetic diseases occur in modern

populations determined the probability with which they may appear in archaeological skeletal samples.

The morphological traits exhibiting consistency with no overlap with scurvy, anemia and/or general malnutrition were considered primary while the less optimal characteristics were given secondary consideration. Traits associated with age (delayed closure of the fontanels and dental development) were removed as they are of no use without accompanying documentation. Skull fragmentation is commonplace among the Isola Sacra individuals. Therefore, posterior flattening and lateral asymmetry could not be readily assessed in the majority of cases and were removed from the trait list. Diagnosis of rickets in the Isola Sacra subadults employed the list of primary traits found in **table 5.2** (below), with secondary traits used only in combination with primary traits.

TABLE 5.2 - Primary & Secondary Pathological Features associated with Rickets.

Primary features of Rickets	Secondary features of rickets
1. Glabellar/facial bone porosity 2. Deformed mandibular ramus 3. Deformed arm bone 4. Deformed leg bones 5. Thickening of long bones 6. Flared costochondral rib ends 7. Flattening of rib curvature 8. Costochondral rib ends irregular and porous 9. Abnormal curvature and plumpness of the ilium 10. Vertebrae compressed and porous	1. Cranial vault porosity/orbital roof porosity 2. Thickening of the cranium 3. Squared head (frontal/parietal bossing) 4. Enamel hypoplasia/ dental deformities 5. Growth plate abnormality of long bones 6. Metaphyseal infraction and fractures of long bones 7. Flaring and cupping of metaphyses 8. Cortex porosity of the metaphyses of long bones

Chapter 6

RESULTS

A summary of all Isola Sacra subadults showing rachitic characteristics is found in **Table 6.1** (page 89). **Table 6.2** (page 90) shows the percentage of Isola Sacra individuals suffering from rickets. Individuals identified as rachitic are presented on a by case basis with accompanying photographs and descriptions of each trait present. The completeness of the skeleton is stated. The age and burial information for each individual can be found in **Appendix B**. These cases are grouped as either rachitic individuals or secondary rickets. These individuals exhibit a variety of features associated with the inadequate mineralization of bone. **Table 6.3** (page 93) summarizes the pathological features associated with the Isola Sacra individuals. **Table 6.4** (page 95) shows the percentage of rachitic traits found in the Isola Sacra individuals. Fifteen percent (27/182) of Isola Sacra subadults show rachitic traits. Seven percent of individuals are deemed secondary rickets as they are more subtle in their manifestation or a limited number of skeletal elements were available for observation. **Figure 6.67** (page 119) shows the age distribution of rachitic, secondary rachitic and nonrachitic individuals. **Figure 6.68** (page 119) shows the distribution by burial type of rachitic, secondary rachitic and nonrachitic individuals. No overlap with scurvy or anemia was detected. However, three possible cases of genetic thalassemia were tentatively identified by other workers (Macchiarelli 2002, *pers. comm.*). It is

suspected that an additional 13% suffered from rickets but due to a lack of skeletal elements, they could not be properly diagnosed.

Statistical analysis consists of the chi-square test to explore for the independence of two variables. The purpose of this test is to determine if the observed frequency of events, in this case the presence of rickets, depart significantly from frequency proposed by a null hypothesis. It is the goal of the statistical analysis to determine if there is an association between age category and the presence of rickets and burial type and the presence of rickets. **Figure 6.69** (page 122) shows the age distribution of rachitic and secondary rachitic individuals. **Figure 6.70** (page 124) shows the distribution of burial type of rachitic and secondary rachitic individuals.

TABLE 6.1 – PERCENTAGE OF ISOLA SACRA SUBADULTS SHOWING RICKETS

Rickets	Secondary rickets	Total Rickets
14/182	13/182	27/182
= 8%	**= 7%**	**= 15%**

TABLE 6.2 – SUMMARY OF ISOLA SACRA SUBADULTS SHOWING RACHITIC CHARACTERISTICS (n=27)

#	SCR	Burial Type/Zone	Age category	Completeness	Diagnosis	Notes
1.	**1**	Amphora 14B	1-2	Complete skeleton, no sacrum or sternum	Rickets	Slight curvature of fibulae (broken), tibiae flaring at prox. ends, x-rays confirm
2.	**10**	Cassa 21	2-3	Complete skeleton minus epiphyses	Subtle rickets	Coronoid processes of mandible laterally bent, head medially bent, ribs porous at sternal ends
3.	**14**	Inhumation 23	2-3	Cranial frags, some ribs, frag Ilia, r. femur, diaphysis of l. femur, tibiae & humerii, prox. radii & ulnae	Subtle rickets	Abnormal curvature of the femora, genu valgum, orbital porosity,
4.	**47**	Amphora 23A	0-0.5	Complete skeleton, but no epiphyses of femora, tibiae & fibulae, sacrum or sternum	Rickets	Cranial vault & orbital roof porosity, deformed mandibular ramus, abnormal curvature of humerii & thickening, thickening & flaring of distal radii
5.	**62**	Amphora 14A	0-0.5	Complete skeleton, except epiphyseal ends of all long bones, sacrum & sternum	Rickets	Deformed mandibular ramus, fracture of 5th & 6th rib, bending of tibiae (no x-rays available), discoloration of incisor, deformed l. & r. m^2, vertebral porosity
6.	**72**	Inhumation 12A	9-10	Almost complete, sternum & epiphyses of all long bones missing	Rickets	Thickening of cranial vault, flared rib ends, LEH in canines, CTP's in pm
7.	**94**	Amphora 12A	4-5	Complete skeleton minus sacrum, sternum, epiphyses of arm bones & midface	Subtle rickets	Active pitting in orbits & over glabella, some pitting approx. 2cm. over postero-lateral area of parietals. Some medial curvature of prox. ulnae
8.	**100**	Sand 21	14-15+	Complete skeleton	Rickets Osteomalacia?	Microporosity along sagittal suture line, external periosteal overgrowth (thickening) of cranium, healing orbital roof porosity, radii & ulnae thickened, rib ends irregular & porous, rib depressions, microporosity of epiphyseal plates of tibiae & femora, depression & porosity on medial/frontal l. humeral head, depression distal ends of tibiae, plumping of ilia, compression & porosity of vertebrae, discoloration of canine, LEH lower left M_1, PM_1, & upper M^1, C, I^2
9.	**102**	Amphora 22	1-2	Complete skeleton, minus sacrum, distal ulnae	Subtle rickets	Slight flaring of prox. tibia & distal femur. Orbital porosity
10.	**110**	Pit 22	1-2	Complete skeleton but no epiphyses & sternum	Rickets	Slight porosity of glabella & orbital roof, bending of ulnae, thickening of radii, prox. & distal ends of humerii porotic, distal & prox. flaring of tibiae, flaring of femora distally, ends of ribs

11.	**139**	Amphora 10	1-2	Complete skeleton	Rickets	irregular & porous, abnormal epiphyses, vertebrae porous
12.	**140**	Amphora 14A	0.5-1	Complete skeleton except lower sternum & sacrum	Rickets	Flared distal end of femora (no x-rays available), internal & external cranial vault porosity, porosity of vertebrae
13.	**175**	Amphora 10	8-9	Complete except missing some cranial bones frag., frontal missing	Rickets	Orbital roof porosity, deformed mandibular ramus, porosity of prox. ends of humerii, bending of ulnae & flaring of distal ends, discoloration of upper & lower incisors, upper canine & l & r m^1
14.	**176**	Inhumation 19	8-9	Complete skeleton minus the epiphyses of the right humerus	Subtle rickets	Bowing of tibiae, r. femur flaring distal ends & slight bowing (left has been cut), (no x-rays available), porosity of all epiphyses, porosity of vertebrae
15.	**244**	Pit 13	5-6	Complete skeleton, no mid-face, r. fibula, distal portion of left humerus only	Rickets	Pitting over glabellar region & palate. Proximal ulna appears bent. Woven bone/micro pitting on anterolateral surface of proximal shaft of r. tibia. Microfossae in cortex of femur. Thoracic vert. porous & anterior lesions in bodies.
16.	**276**	Amphora 15	1-2	Complete skeleton minus sternum	Rickets	Squared head, r. humerus deformed & bony spur, curvature of l. fibula & thickening, curvature of femora, curvature of tibiae & flaring, fibula, flaring of ribs, rib depressions & abnormal curvature, epiphyses porous, upper & lower m^2's severe defects, LEH lingual of m_1, l & r m^2 pit hypoplasia on buccal & lingual side, r. m_1 lingual & buccal pit hypoplasia, CTP's in molars, vertebral porosity
17.	**511**	Amphora 1	0.5-1	Missing area around orbits, left radius & ulna, scapulae, sacrum, sternum, left fibula. mid portion of r. humerus only, prox. r. ulna only	Rickets	Squaring of head, orbital roof porosity, slight medial in bending of mandibular ramus, radii flared, humerii turned medially, porosity at rib ends in costal groove, vertebrae porous
18.	**523**	Cappuccina 16	5-6	Complete skeleton minus some facial bones, left humerus, sternum, sacrum, all epiphyses. Left femur cut for sampling	Subtle rickets	Tibiae bent anteriorly, porosity distal ends of tibiae, l. tibia show large area of porotic woven bone on disto-lateral shaft, femora show flaring of distal metaphyses w/ flattening of the ends (surfaces have very little relief), femoral shaft cortices show extreme porosity, all but 3 sternal rib ends flared & porous
19.	**530**	Colombario G forma 3 5	9-10	Skull, r. femur, distal ends of left femur, r. tibia, diaphysis of left tibia, prox. & distal fibulae, frag humeri, radii & ulnae	Subtle rickets	R. femur bent posteriorly & twisted, compression of vertebral bodies, porous diaphyseal ends, orbital porosity, LEH @ CEJ incisors & molars
						Subtle bending of tibiae & femora. Compression of vertebral bodies

20.	**542**	Inhumation	1-2	Complete skeleton minus long bones epiphyses, some portions of frontal, temporal and parietal bones.	Subtle rickets	Slight flaring of proximal & distal tibiae, orbital porosity
21.	**612**	tomb 47b forma D	2-3	Tibiae & left humerus only	Subtle rickets	Some curvature of humerus & tibiae. Flaring of proximal tibiae.
22.	**636**	tomb 38	1-2	l. clavicle, r. radius	Rickets	Radius shows strong lateral curvature, thickening of prox end & midshaft, flaring of distal end
23.	**644**	tomb 45 Forma 2	1-2	Complete mandible, left femoral diaphysis	Subtle rickets	Inward curvature of the mandibular condyles
24.	**660**	tomb 47a forma C	7-8	Elements present are right side of mandible & maxilla, humeri minus humeral heads, right radius, distal right ulna, distal left ulna, three-fourths of right femur, distal portion of left femur, left tibia & fibula, proximal right tibia & distal right fibula.	Rickets	Slight medial bending of r. radius & flaring of distal end, twisting of humerii, r. femur abnormal curvature, l. tibia slight bending, LEH on l[1]
25.	**683**	tomb 76	13-14	Left mandible and maxilla, clavicles, scapulae, humeri, radii, proximal left ulna, ilia, right femur, distal left femur and a couple vertebrae.	Subtle rickets	Lateral curvature of rt. radius (l. broken) Rib frags. and vertebrae porous
26.	**773**	tomb 45	0.5-1	Mandible minus r. ramus, clavicles, r. scapulae, r. humerus, distal pt of l. humerus, l. ilium, r. radius, prox. pt of r. ulna, midshaft of r. femur, prox pt of r. fibula	Subtle rickets	Posterior/medial bending of l. mandibular ramus (r. missing)
27.	**780**	tomb 43 forma 4	1-2	Occipital, scapulae, humeri, ulnae, l. radius, r. ilia, femora, tibiae, fibulae & fragmentary vertebrae.	Subtle rickets	Bending & flaring of fibulae

TABLE 6.3 - PATHOLOGICAL FEATURES ASSOCIATED WITH THE ISOLA SACRA SUBADULTS

	SCR 1	SCR 10	SCR 14	SCR 47	SCR 62	SCR 72	SCR 94	SCR 100	SCR 102	SCR 110	SCR 139	SCR 140	SCR 175	SCR 176
Estimated age (years)	1-2	2-3	2-3	0-0.5	0-0.5	9-10	4-5	15+	1-2	1-2	1-2	0.5-1	8-9	8-9
Glabellar /facial porosity	A	A	-	A	A	A	P	A	A	P	A	A	-	P
Deformed mandibular ramus	A	P	-	P	P	A	A	A	A	A	A	P	A	A
Deformed arm bones	A	A	A	P	A	A	P	P	A	P	A	P	A	P
Deformed leg bones	P	A	P	A	P	A	A	A	A	A	A	A	P	A
Thickening of long bones	A	A	A	P	A	A	A	P	A	P	A	A	A	A
Flared rib ends	A	A	-	A	A	P	A	A	A	A	A	A	A	A
Flattening of rib curvature	A	A	-	A	A	A	A	A	A	A	A	A	A	A
Rib porosity/irregularity	A	P	-	A	P	A	A	P	A	P	A	A	A	A
Abnormal plumpness & curvature of ilium	A	A	-	A	A	A	A	P	A	A	A	A	A	A
Vertebral porosity & compression	A	A	A	A	P	A	A	P	A	P	P	A	P	P
Cranial vault/orbital roof porosity	A	A	P	P	A	A	P	P	P	P	P	P	-	A
Thickening of cranium	A	A	-	A	A	P	A	P	A	A	A	A	-	A
Squared head	A	A	-	A	A	A	A	A	A	A	A	A	-	A
Tooth deformation /EH	A	A	A	A	P	P	A	P	A	A	A	P	A	A
Plate abnormality of long bones	A	A	A	A	-	-	A	P	A	P	A	P	A	P
Fracture of long bones	-	-	-	-	-	-	-	-	-	-	-	-	-	A
Flaring/cupping of metaphyses	A	A	A	P	A	-	A	A	P	P	P	P	A	A
Cortex porosity of long bones	A	A	A	A	-	-	-	P	A	-	-	A	A	P

P = present A = absent - = not observable

Cont...TABLE 6.3 - PATHOLOGICAL FEATURES ASSOCIATED WITH THE ISOLA SACRA SUBADULTS

	SCR 244	SCR 276	SCR 511	SCR 523	SCR 530	SCR 542	SCR 612	SCR 636	SCR 644	SCR 660	SCR 683	SCR 773	SCR 780
Estimated age (years)	5-6	1-2	0.5-1	5-6	9-10	1-2	2-3	1-2	1-2	7-8	13-14	0.5-1	1-2
Glabellar /facial porosity	A	A	-	-	A	A	-	-	-	-	-	-	-
Deformed mandibular ramus	A	P	A	A	A	A	-	-	P	-	A	P	P
Deformed arm bones	P	P	-	A	-	A	P	P	-	P	P	-	A
Deformed leg bones	P	A	P	P	P	A	P	-	-	P	A	-	P
Thickening of long bones	P	A	A	A	A	A	-	P	-	A	A	-	A
Flared rib ends	P	A	P	A	-	A	-	-	-	-	-	-	-
Flattening of rib curvature	P	A	-	A	-	A	-	-	-	-	-	-	-
Rib porosity/irregularity	P	P	P	A	-	A	-	-	-	-	P	-	-
Abnormal plumpness & curvature of ilium	A	A	A	A	-	A	-	-	-	-	A	A	A
Vertebral porosity & compression	P	P	A	P	P	A	-	-	-	-	P	-	P
Cranial vault/orbital roof porosity	A	P	-	P	A	P	-	-	-	-	-	-	-
Squared head	P	P	-	-	A	-	-	-	-	-	-	-	-
Thickening of cranium	-	A	-	A	A	A	-	-	-	-	-	-	-
Tooth deformation /EH	P	A	A	P	-	A	-	-	-	P	-	-	-
Plate abnormality of long bones	A	A	P	P	A	A	-	-	-	A	A	A	A
Fracture of long bones	-	-	-	A	-	-	-	-	-	-	-	-	-
Flaring/cupping of metaphyses	A	P	P	A	-	P	P	P	-	P	A	A	P
Cortex porosity of long bones	A	-	P	A	-	A	-	-	-	A	A	A	A

TABLE 6.4 – PERCENTAGE OF RACHITIC TRAITS FOUND IN ISOLA SACRA SUBADULTS

Rachitic trait	# of individuals	Percentage
Deformed arm bones	12	44%
Vertebral porosity & compression	12	44%
Flaring/cupping of metaphyses	12	44%
Deformed leg bones	11	41%
Cranial vault/orbital roof porosity	11	41%
Deformed mandibular ramus	8	30%
Rib porosity/irregularity	8	30%
Tooth deformation /EH	7	26%
Plate abnormality of long bones	7	26%
Thickening of long bones	5	18.5%
Glabellar /facial porosity	3	11%
Flared rib ends	3	11%
Cortex porosity of long bones	3	11%
Squared head	2	7%
Thickening of cranium	2	7%
Flattening of rib curvature	1	4%
Abnormal plumpness/curvature of ilium	1	4%
Fracture of long bones	0	0%

6.1 – Rachitic individuals

SCR 1 – Dental age 1.5-2 yrs, Skeletal Age 1.5 yrs

The skeleton is complete with exception of the sacrum and sternum. The fibulae show slight curvature (**figure 6.1**). The tibiae are flared at their proximal ends, (**figure 6.2**) evident on x-ray as well. The right fibula below has been broken and has not adhered properly.

Figure 6.1 – SCR 1 Slight curvature of the right fibula (photo C. Wood).

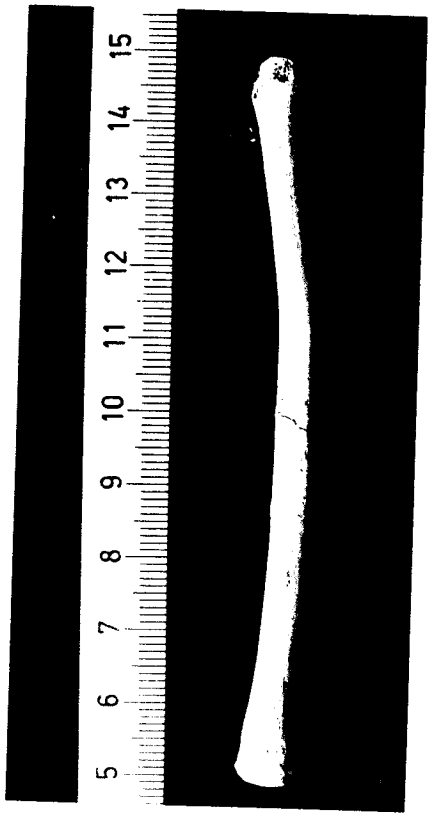
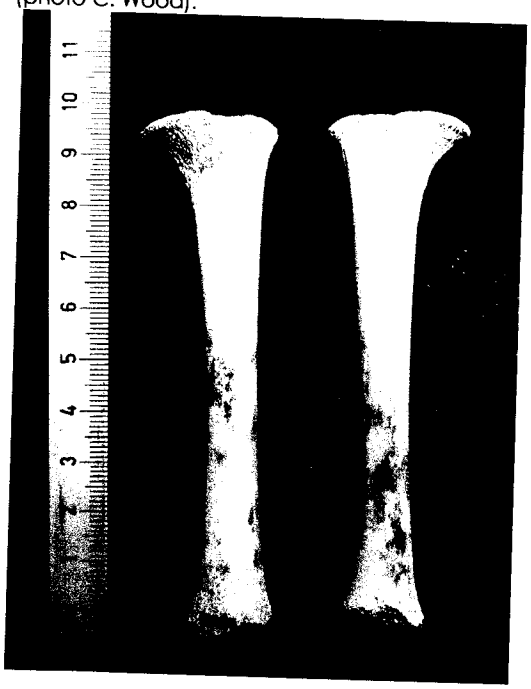


Figure 6.2- SCR 1 Proximal flaring of tibiae (photo C. Wood).



SCR 47 – Dental Age 0.5, Skeletal Age 0-0.5

The skeleton is complete with exception of the sternum, sacrum and epiphyseal ends of the femora, tibiae and fibulae. The cranial vault and orbital roof are porous (**figure 6.3**). The mandibular ramus shows abnormal medial/posterior

bending. The humerii show abnormal curvature and thickening (**figure 6.4**). The radii appear thickened and are flared at their distal ends (**figure 6.5**).

Figure 6.3 – SCR 47 Orbital roof porosity (photo C. Wood).

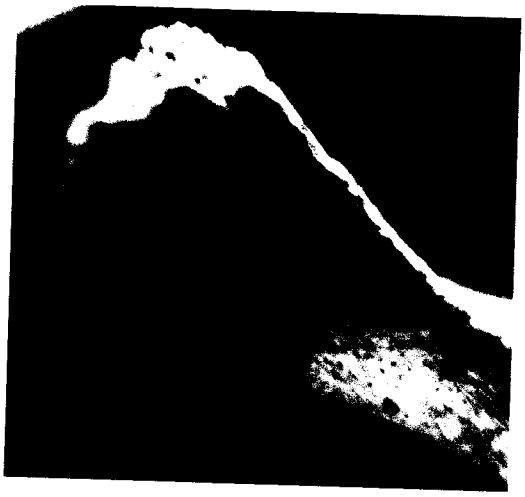
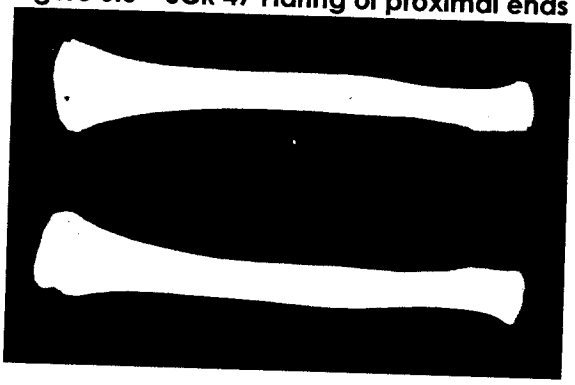


Figure 6.4 – SCR 47 Abnormal curvature of humerii (photo C. Wood).

Figure 6.5 – SCR 47 Flaring of proximal ends & thickened of radii (photo C. Wood).



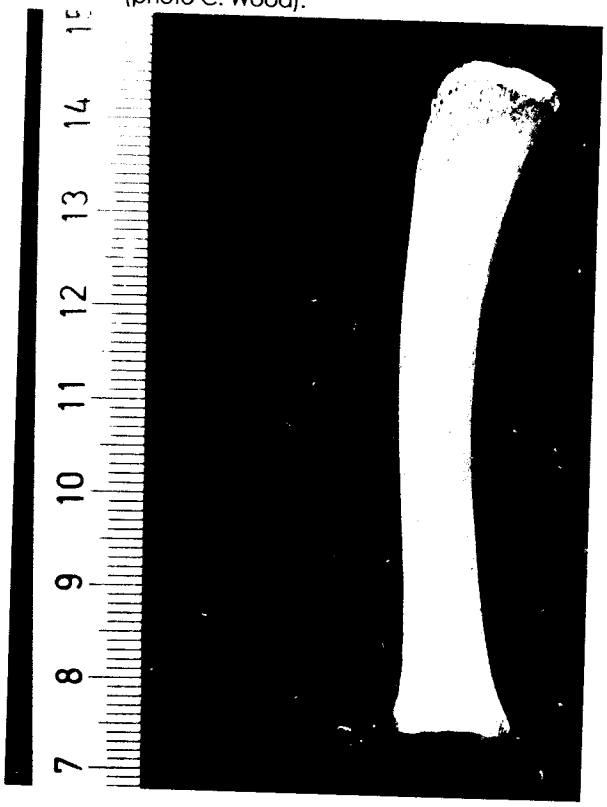
SCR 62 – Dental Age 0.5, Skeletal Age 0.25-0.5

The skeleton is complete with the exception of the epiphyseal ends of all the long bones, the sacrum and sternum. All skeletal elements are quite porous; especially the vertebrae (**figure 6.6**). The mandibular ramus shows abnormal

medial/posterior bending. There appears to be two postmortem fractures on possibly the 5th and 6th ribs. Bending of tibiae (no x-rays available) is apparent (**figure 6.7**). The left and right deciduous upper second molars are deformed.

Figure 6.6 – SCR 62 Porosity of vertebrae (photo C. Wood).

Figure 6.7 – SCR 62 Bending of tibia (photo C. Wood).



SCR 72 – Dental Age 9 yrs, Skeletal Age 11 yrs

The skeleton is relatively complete but the sternum and the epiphyses of all the long bones are missing. The cranial vault is thickened. The ends of the ribs are flared. Linear enamel hypoplasia is apparent on the canines. Cusp tip pits are present in the premolars. No pictures are available for SCR 72.

SCR 100 – Dental Age 15 yrs, Skeletal Age 15-16 yrs

The skeleton is complete. There is microporosity along the sagittal suture line. External periosteal overgrowth (thickening) is evident all over the cranium with exception of the temporal bones and along the sagittal suture line (**figure 6.8**). Healing of orbital roof porosity is apparent (**figure 6.9**). The rib ends are irregular and porous. Rib depressions are apparent (**figure 6.10**). The radii and ulnae are thickened (**figure 6.11 & 6.12**). No abnormal bending of the leg bones is apparent on x-ray. Microporosity of the epiphyseal plates of the tibiae and femora are present (**figure 6.13**). There is a depression on medial/frontal side of the left humeral head. The humeral head is also quite porous. Macroporosity and depression is apparent on the distal ends of the tibiae (**figure 6.14**). The ilia are plumped (**figure 6.15**). The vertebrae are compressed and porous. Linear enamel hypoplasia is evident in the lower left and right molars, and premolars, the upper left and right first molars, canines, and incisors (**figure 6.16**). There is discoloration of the right canine (left missing) (**figure 6.17**).

Figure 6.8 – SCR 100 Thickening of cranium (photo C. Wood).

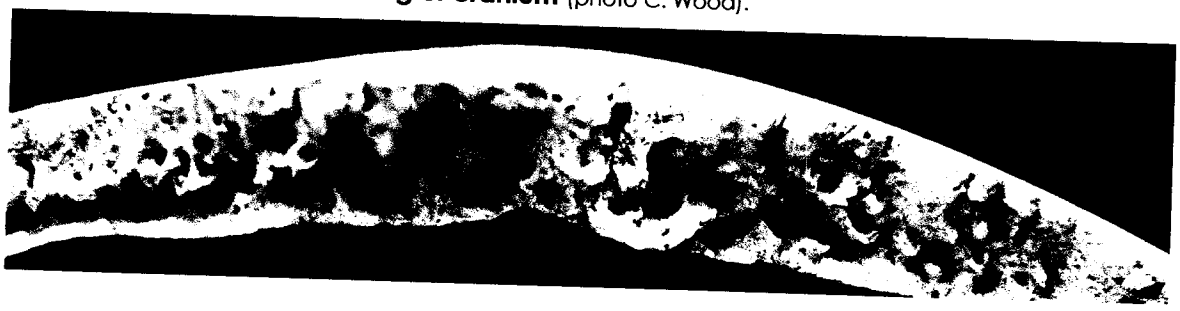


Figure 6.9 – SCR 100 Healing of orbital porosity (photo C. Wood).

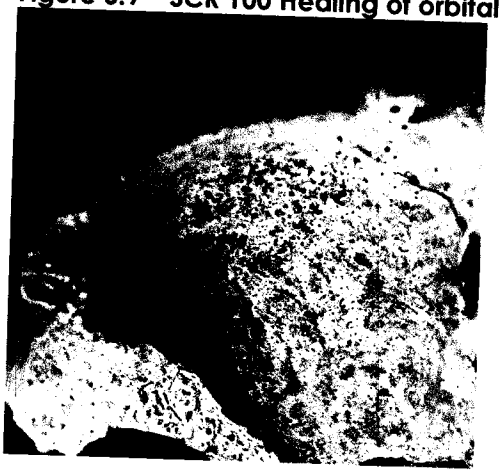


Figure 6.10 – SCR 100 Rib depression (photo C. Wood).

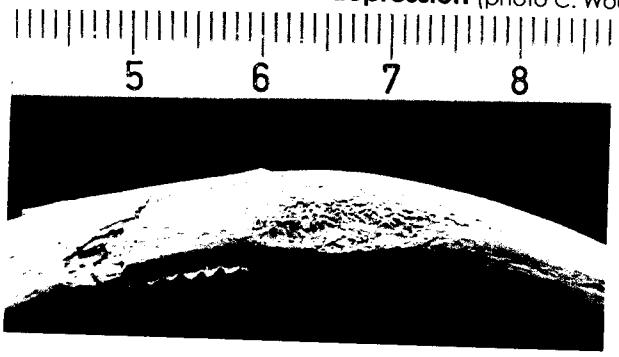


Figure 6.11 – SCR 100 Thickened radii (photo C. Wood).

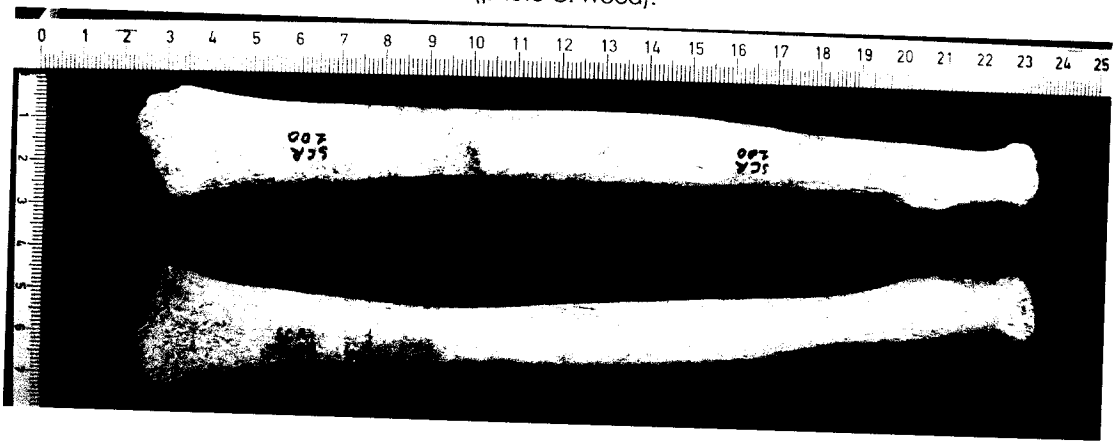


Figure 6.12 – SCR 100 Thickened ulnae (photo C. Wood).

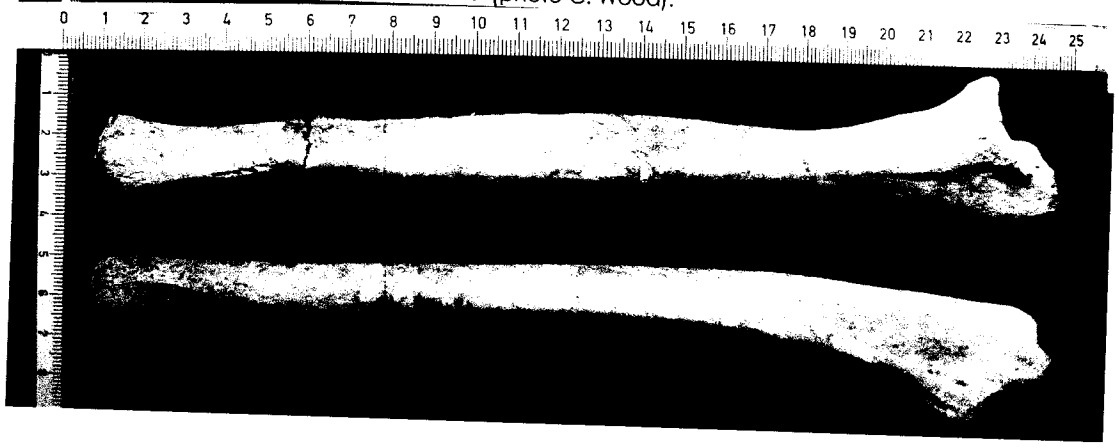


Figure 6.13 – SCR 100 Porosity of trochanter (photo C. Wood).

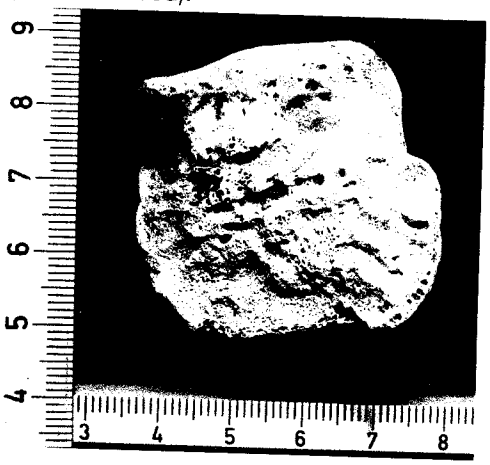


Figure 6.14 – SCR 100 Depression of tibial epiphysis (photo C. Wood).

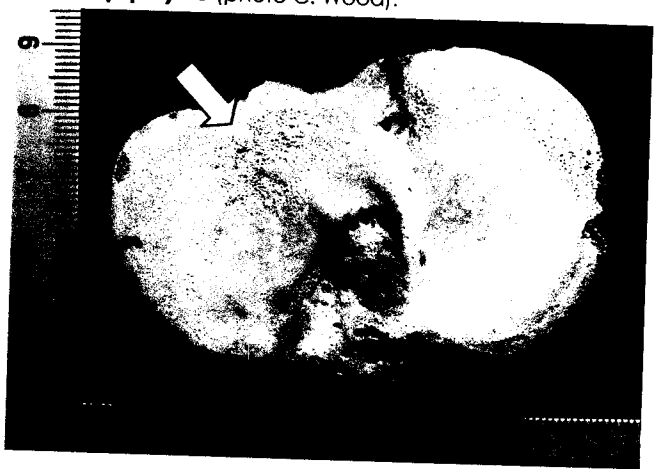


Figure 6.15 – SCR 100 Plumping of ilium (photo C. Wood).

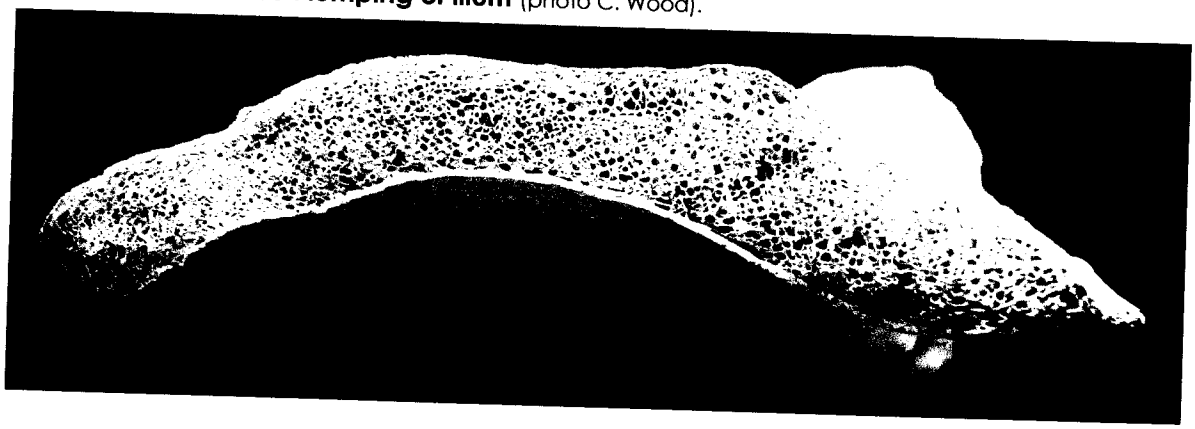
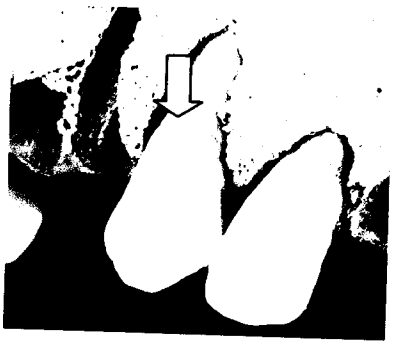


Figure 6.16 – SCR 100 LEH of incisors (photo C. Wood).

Figure 6.17 – SCR 100 Discoloration of canine (photo C. Wood).



SCR 110 – Dental Age 2 yrs, Skeletal Age 1.5 yrs

The skeleton is complete with exception of the epiphyses and the sternum. The glabella (**figure 6.18**) and orbital roofs are slightly porous and irregular. The ends of the ribs are irregular and porous (**figure 6.19**). The ulnae show abnormal bending (**figure 6.20**). The radii appear thickened. The proximal and distal ends (**figure 6.21**) of the humerii are porotic. The distal and proximal ends of the tibiae are flared (**figure 6.22**). All epiphyses are abnormally shaped and porous (**figure 6.23**). The femora are flared at the distal end but even more so at the proximal end (**figure 6.24, a & b**). The vertebrae are porous and have holes on their anterior side.

Figure 6.18 – SCR 110 Glabellar porosity (photo C. Wood).

Figure 6.19 – SCR 110 Rib ends porous (photo C. Wood).

Figure 6.20 – SCR 110 Curvature of ulnae (photo C. Wood).

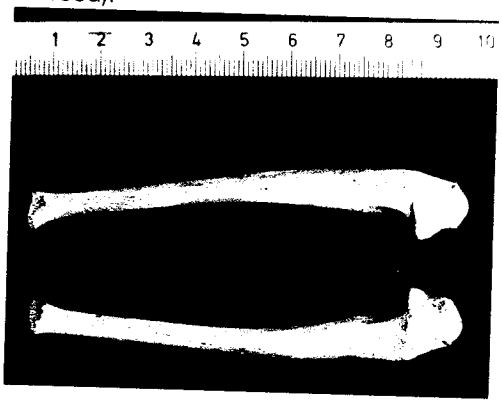


Figure 6.21 – SCR 110 Porosity of the distal humerus (photo C. Wood).

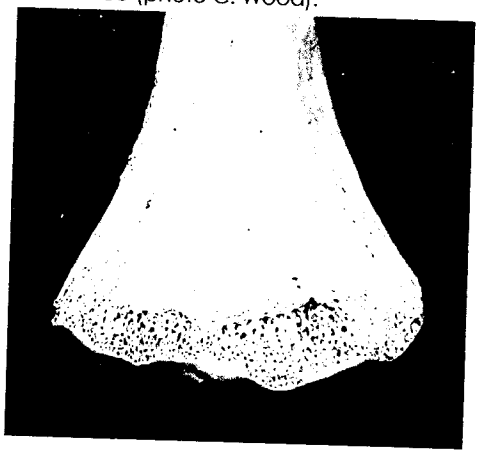


Figure 6.22 – SCR 110 Proximal & distal flaring of tibiae (photo C. Wood).

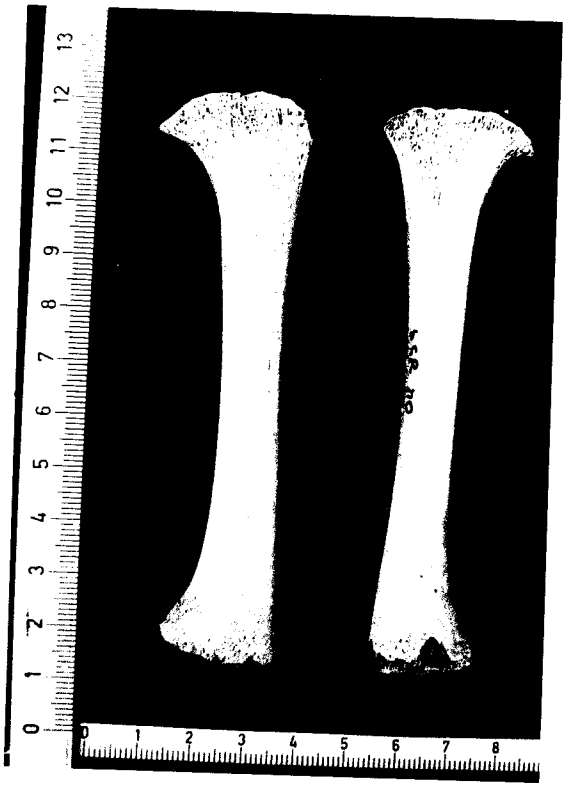


Figure 6.23 – SCR 110 Abnormal epiphyses of humerii (photo C. Wood).

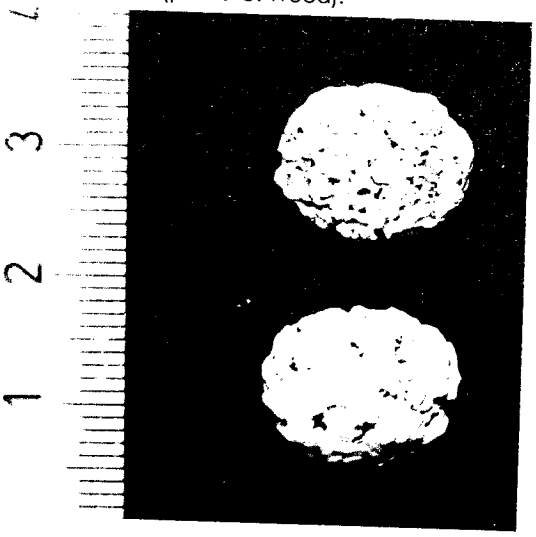
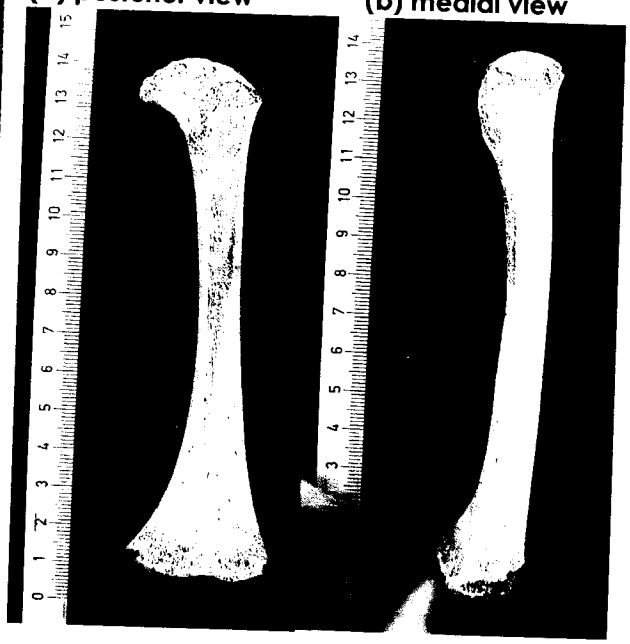


Figure 6.24 – SCR 110 Distal flaring of femur (photo C. Wood).

(a) posterior view **(b) medial view**



SCR 139 – Dental Age 1.5 yrs – Skeletal 1 year

The skeleton is complete with exception of the sternum and sacrum. The cranial bones are porous (**figures 6.25** & **6.26**). The distal ends of the femora are flared (**figure 6.27**). The vertebrae are very porous (**figure 6.28**).

Figure 6.25 – SCR 139 External porosity of the cranium (photo C. Wood).

Figure 6.26 – SCR 139 Porosity of the temporal bone (photo C. Wood).

Figure 6.27 - SCR 139 Flaring of distal ends of femora (photo C. Wood).

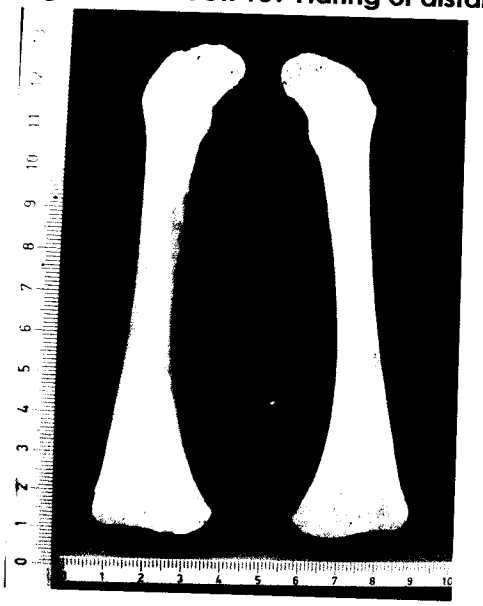
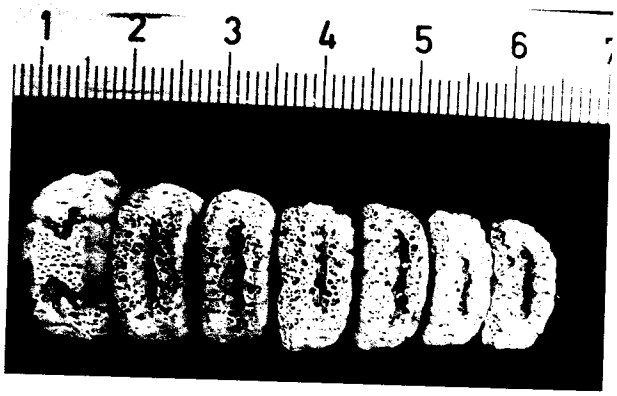


Figure 6.28 – SCR 139 Porosity of the vertebrae (photo C. Wood).



SCR 140 – Dental Age 0.75-1 yr, Skeletal Age 0.5

The skeleton is complete with the exception of the lower portion of the sternum and the sacrum. The orbital roofs are porous and irregular (**figure 6.29**). The mandibular condyles show abnormal medial/posterior bending (**figure 6.30**). The proximal ends of the humerii are porous (**figure 6.31**). There is bending of the ulnae and flaring of their distal ends (**figure 6.32**). All deciduous upper incisors, canines and first molars are discolored (**figure 6.33**).

Figure 6.29 – SCR 140 Orbital roof porosity (photo C. Wood).

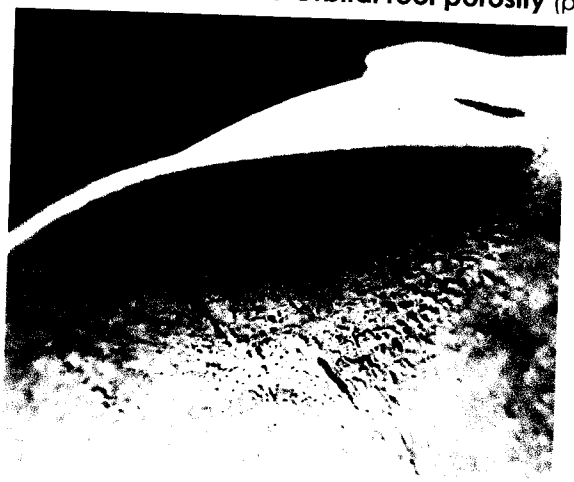


Figure 6.30 – SCR 140 In bending of mandibular condyles (photo C. Wood).

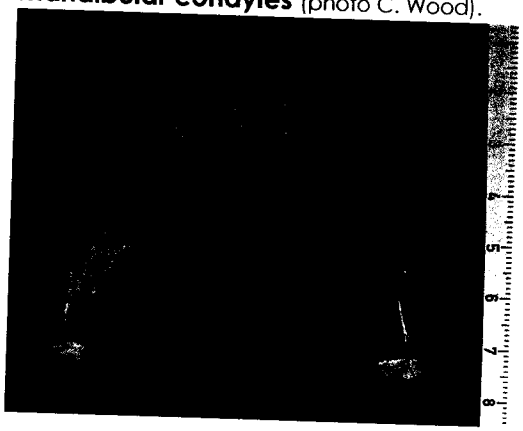


Figure 6.31 – SCR 140 Porosity of proximal humerii (photo C. Wood).

Figure 6.32 – SCR 140 Bending of ulnae & flaring of distal ends (photo C. Wood).

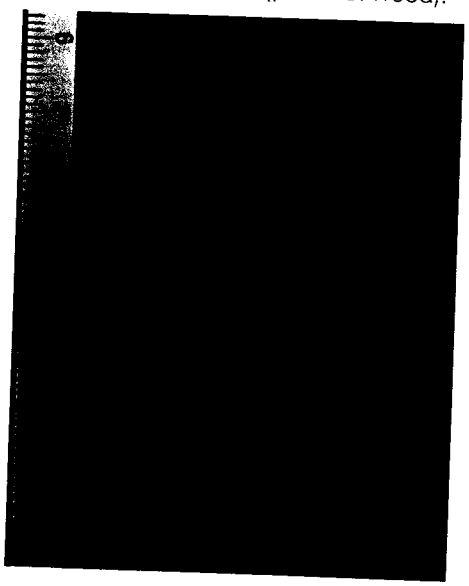
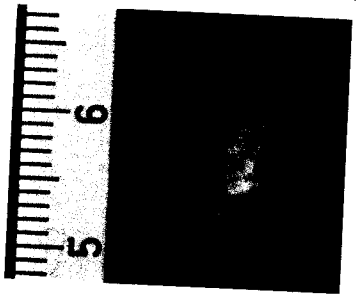


Figure 6.33 – SCR 140 Discoloration of incisor (photo C. Wood).



SCR 175 – Dental Age 8-9yrs, Skeletal Age 7 yrs

The skeleton is three-quarters present. Those elements missing are the sternum, sacrum and upper portion of the cranium. The right femur is slightly bowed and flared at its distal end (**figure 6.34**). The left femur has been cut for sampling. The tibiae are bowed and thickened (**figure 6.35**). The distal ends of the fibulae are flared and bent (**figure 6.36**). No x-rays are available. All epiphyses are porous and irregular (**figure 6.37**). All vertebrae are porous (**figure 6.38**).

Figure 6.34 – SCR 175 Slight bending & flaring of distal femur (photo C. Wood).

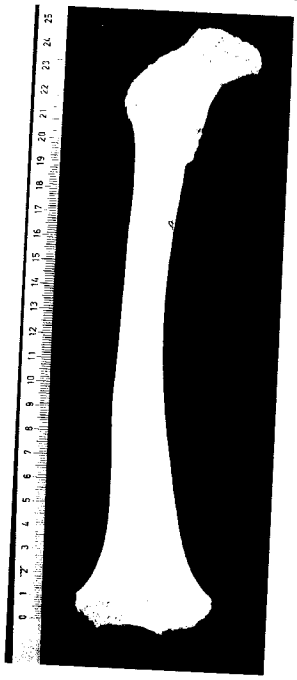


Figure 6.35 – SCR 175 Bending of tibia (photo C. Wood).

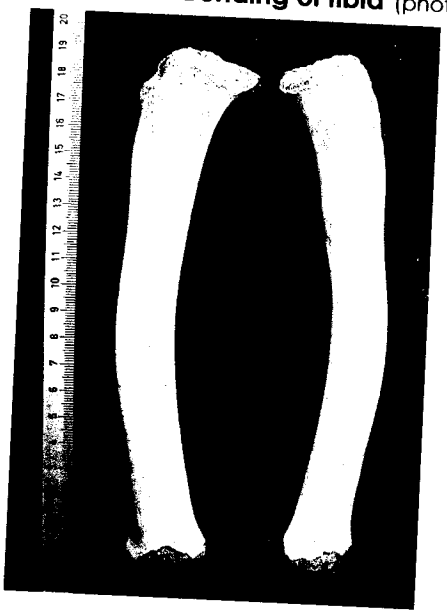


Figure 6.36 - SCR 175 Flaring & bending of distal ends of fibulae (photo C. Wood).

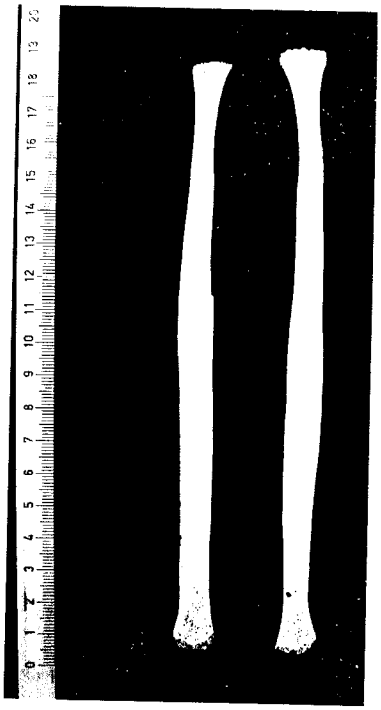


Figure 6.37 – SCR 175 Porosity of epiphysis (photo C. Wood).

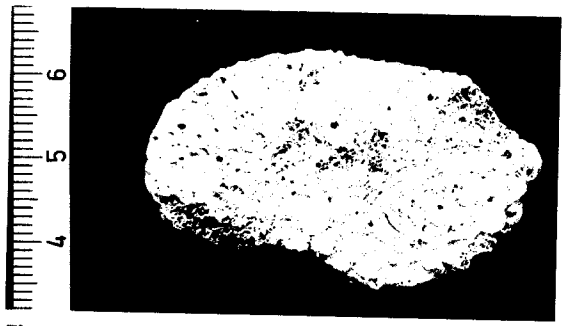
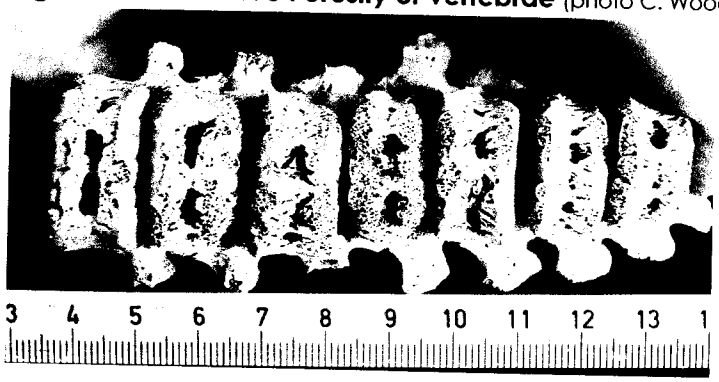


Figure 6.38 – SCR 175 Porosity of vertebrae (photo C. Wood).



SCR 244 – Dental Age 5-6 yrs, Skeletal Age 4-5 yrs

The skeleton is complete minus the mid-face. A squared appearance of the head is apparent (**figure 6.39**). The right humerus is deformed and a bony spur is present (**figure 6.40**). The proximal portion of the left humerus is missing and bending deformities cannot be assessed. The left fibula is curved and thickened (**figure 6.41**). The right fibula is missing. The femora (**figures 6.42**) and tibiae (**figure 6.43**) are abnormally curved. Rib depressions are evident (**figure 6.44**). The sternal ends of the ribs are flared (**figure 6.45**). The ribs are abnormally curved (**figure 6.46**). All epiphyses are very porous and irregular (**figure 6.47 & 6.48**). The upper and lower deciduous second molars have enamel defects (**figure 6.49**).

Linear enamel hypoplasia is apparent on the lingual portions of the deciduous first molars. Pit hypoplasia is apparent on the buccal and lingual sides of the deciduous second molars. The right deciduous first molar has lingual & buccal pit hypoplasia. Cusp tip pits are apparent in the molars. All vertebrae are porous.

Figure 6.39 – SCR 244 Squaring of the head/frontal bossing (photo C. Wood).

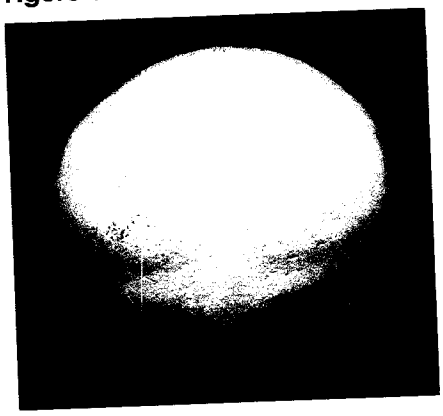


Figure 6.40 – SCR 244 Bending of right humerii and bony spur (photo C. Wood).

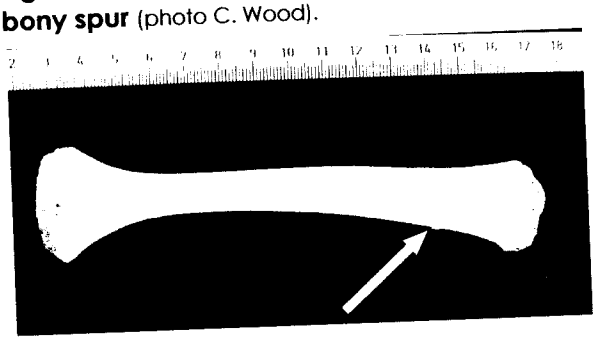


Figure 6.41 – SCR 244 Bending & thickening of left fibula (right missing) (photo C. Wood).

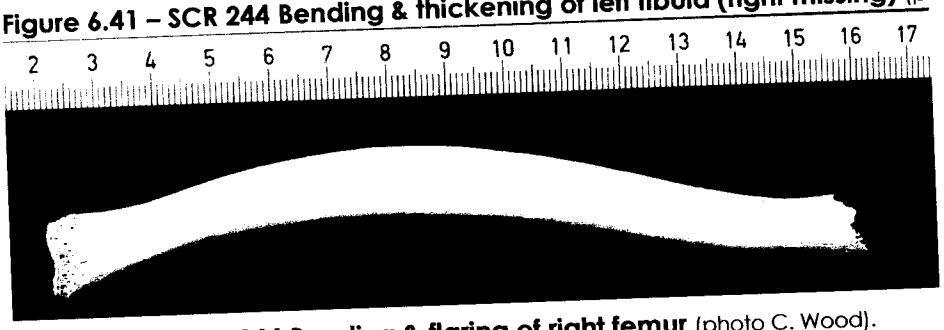


Figure 6.42 – SCR 244 Bending & flaring of right femur (photo C. Wood).

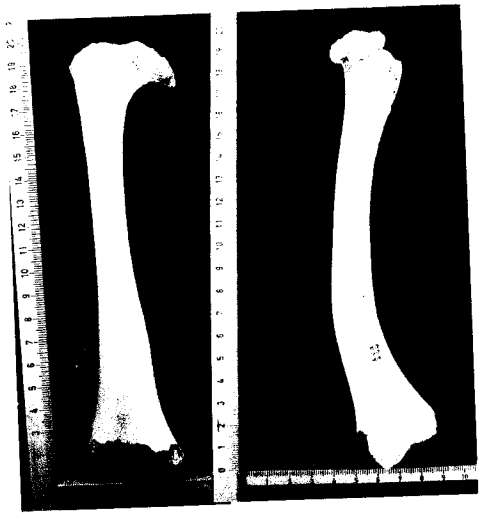


Figure 6.43 – SCR 244 Bending & flaring of tibiae (photo C. Wood).

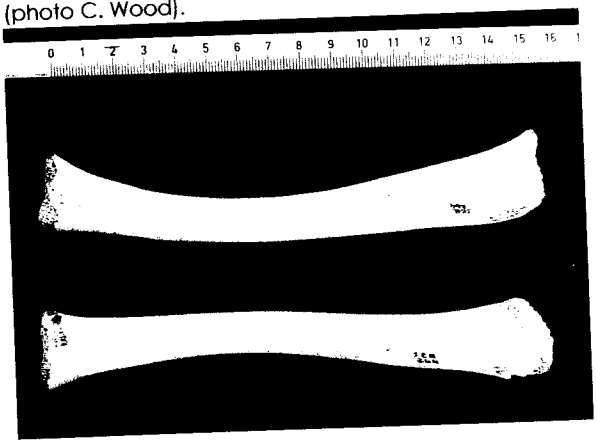


Figure 6.44 – SCR 244 Rib depression (photo C. Wood).

Figure 6.45 – SCR 244 Flaring of rib (photo C. Wood).

Figure 6.46 – SCR 244 Abnormal curvature of ribs (photo C. Wood).

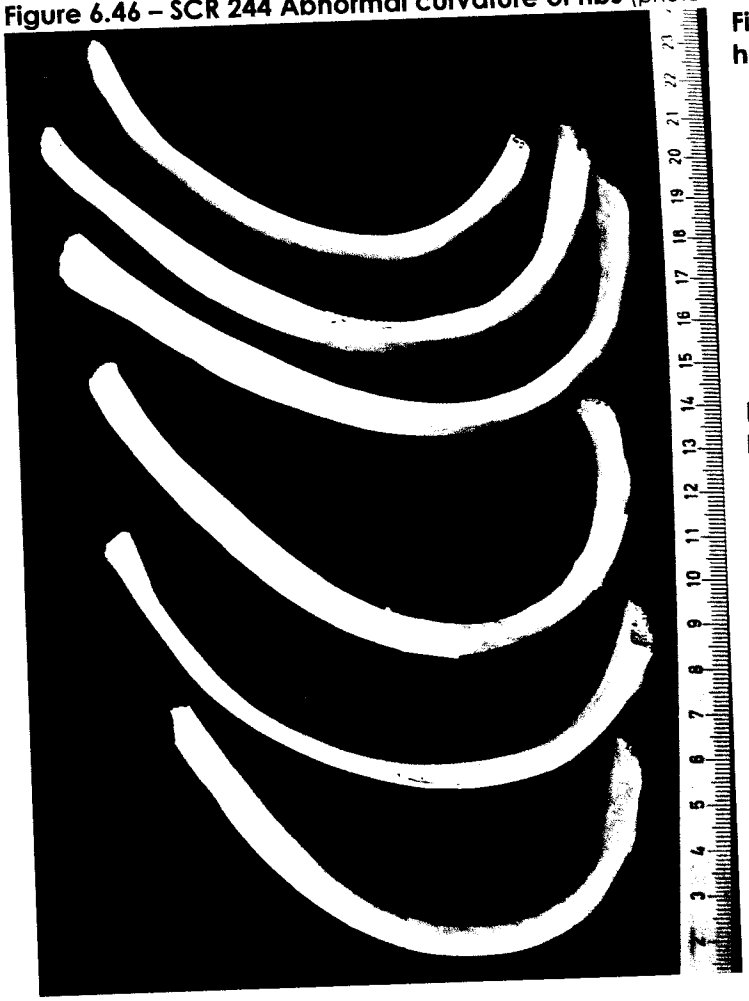


Figure 6.47 - SCR 244 Porosity of head of femur (photo C. Wood).

Figure 6.48 – SCR 244 Porosity of humeral head (photo C. Wood).

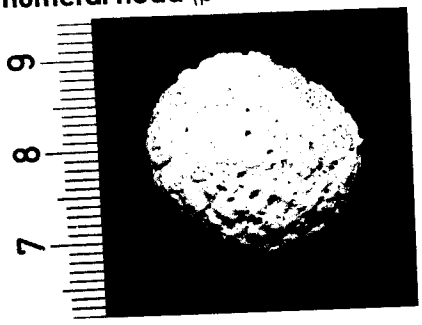
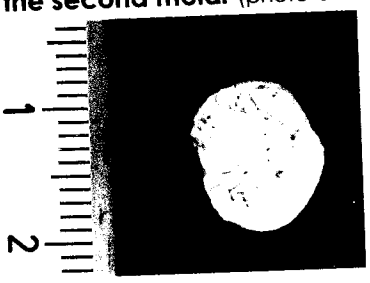


Figure 6.49 – SCR 244 Defects of the second molar (photo C. Wood).



SCR 276 – Dental Age 1.5-2 yrs, Skeletal Age 1.5 yrs

The skeleton is complete minus the sternum. A squared appearance of the head is apparent (**figure 6.50**). There is orbital roof porosity (**figure 6.51**). The mandibular ramus shows slight medial/posterior bending (**figure 6.52**). The right humerii are turned medially (**figure 6.53**). The radii show flaring (**figure 6.54**). Porosity is apparent at rib ends in costal grooves with some porosity at the rib ends. The vertebrae are porous.

Figure 6.50 – SCR 276 Squaring of the head/frontal bossing (photo C. Wood).

Figure 6.51 – SCR 276 Orbital porosity (photo C. Wood).

Figure 6.52 – SCR 276 Slight in bending of mandibular condyles (photo C. Wood).

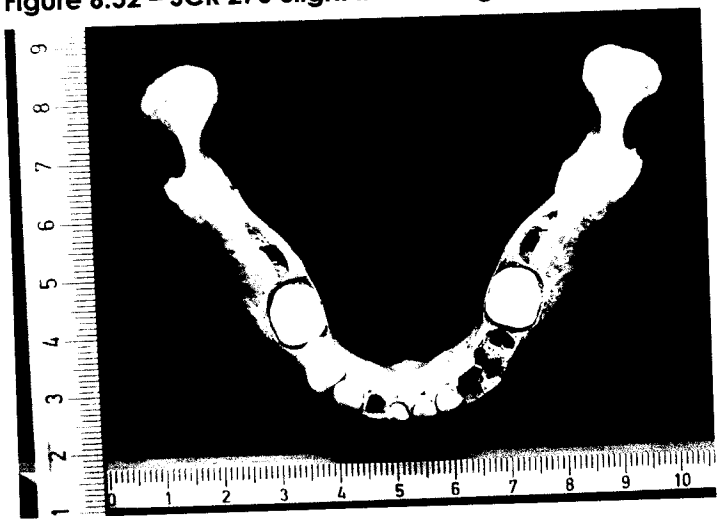


Figure 6.53 – SCR 276 Humerii turned medially (photo C. Wood).

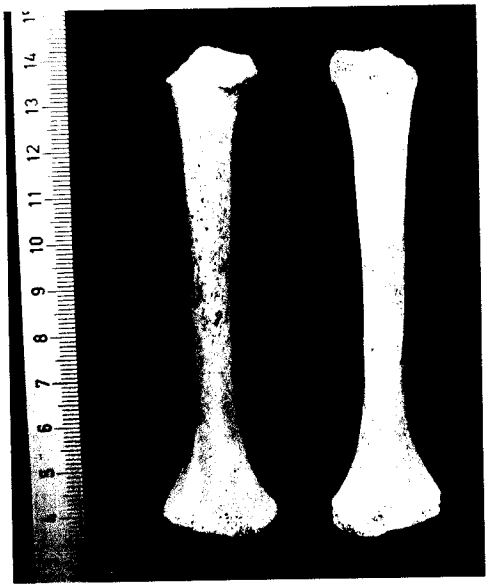
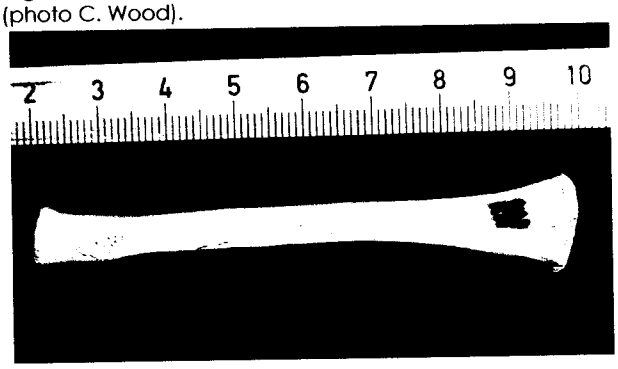


Figure 6.54 – SCR 276 Flaring of distal end of radius (photo C. Wood).



SCR 511 – Dental Age 0.5-0.75, Skeletal Age 0.5

The skeleton is almost complete. The tibiae are abnormally bent anteriorly and show extensive porosity at their distal ends (**figure 6.55**). The left tibia shows a large area of porotic woven bone on its disto-lateral shaft. The femora show flaring of their distal metaphyses and flattening their distal of ends (surfaces have very little relief). The femoral shaft cortices show extreme porosity (**figure 6.56**). All but three of the sternal rib ends are flared and porous.

Figure 6.55 – SCR 511 Bending of tibia, distal porosity (photo C. Wood).

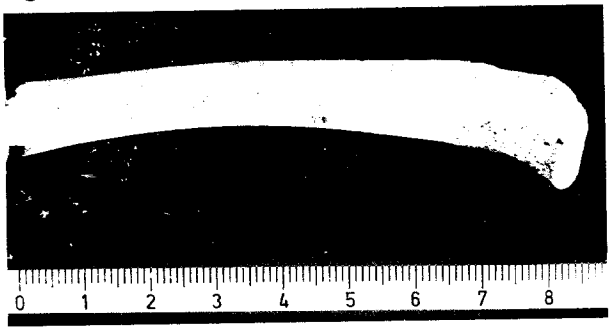
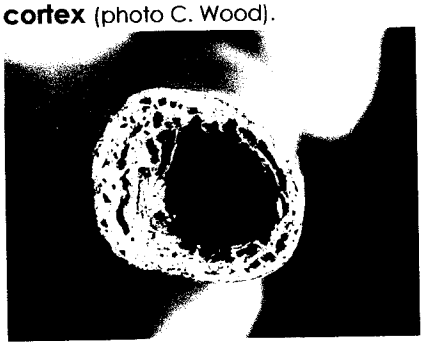


Figure 6.56 – SCR 511 Porosity of femoral cortex (photo C. Wood).



SCR 636 - Skeletal Age 1.5 yrs

SCR 636 consists of a left clavicle and right radius. The radius shows strong lateral curvature, thickening of its proximal end and midshaft and flaring and cupping at its distal end (**figure 6.57**).

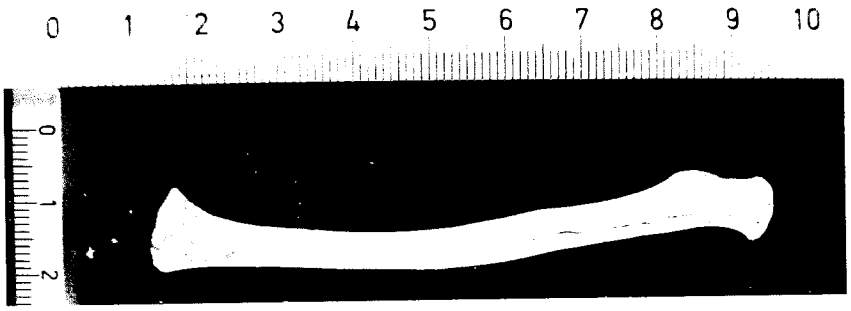


Figure 6.57 – SCR 636 Right radius showing curvature, thickening, flaring & cupping at its distal end (photo C. Wood).

SCR 660 – Dental Age 7 yrs, Skeletal Age 7-8 yrs

The elements present are as follows: right side of mandible and maxilla, humeri missing humeral heads, right radius, distal right ulna, distal left ulna, three-fourths of the right femur, distal portion of left femur, left tibia and fibula, proximal right tibia and distal right fibula. The humerii are twisted but the picture below does not illustrate that well (**figure 6.58**). There is slight medial bending and flaring of the right radius (**figure 6.59**). The right femur (**figure 6.60**) and left tibia (**figure 6.61**) show abnormal curvature. The other long bones are missing. Linear enamel hypoplasia is present on the upper incisors.

Figure 6.58 – SCR 660 Twisting of humerii (photo C. Wood).

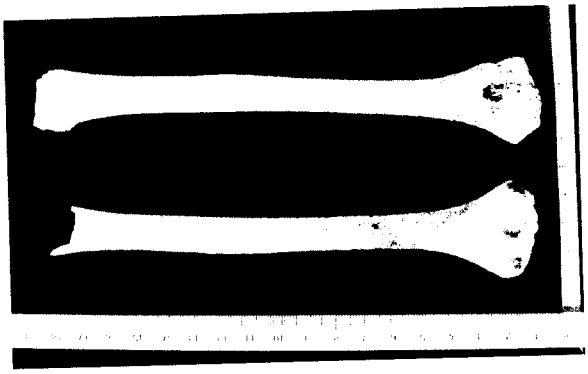


Figure 6.59 – SCR 660 Medial bending & distal flaring of right radius (photo C. Wood).

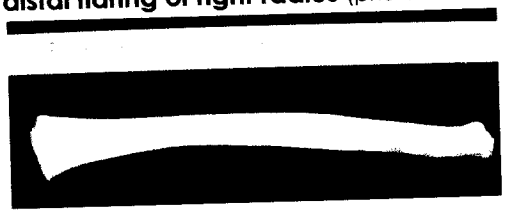


Figure 6.60 – SCR 660 Abnormal curvature of right femur (photo C. Wood).

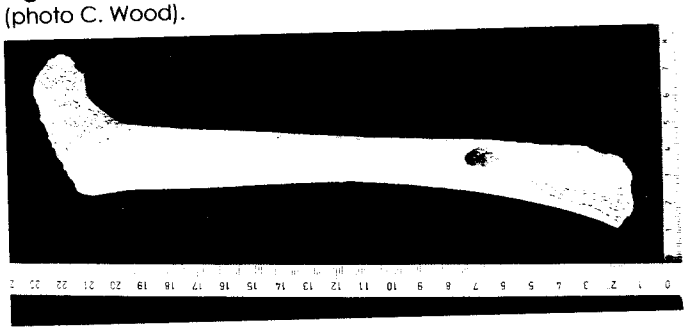
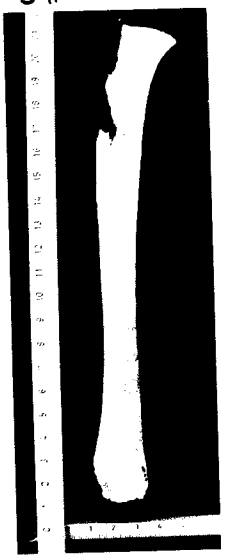


Figure 6.61 – SCR 660 Left tibiae shows slight bending (photo C. Wood).



6.2 – Secondary rachitic individuals

SCR 10 – Dental Age 1.5-2 yrs, Skeletal Age 1.5 yrs

The skeleton is complete with the exception of the epiphyses of all the long bones. Coronoid processes of the mandible are curved laterally (**figure 6.62**). The heads of the rami are curved medially. Ribs are porous at their sternal ends.

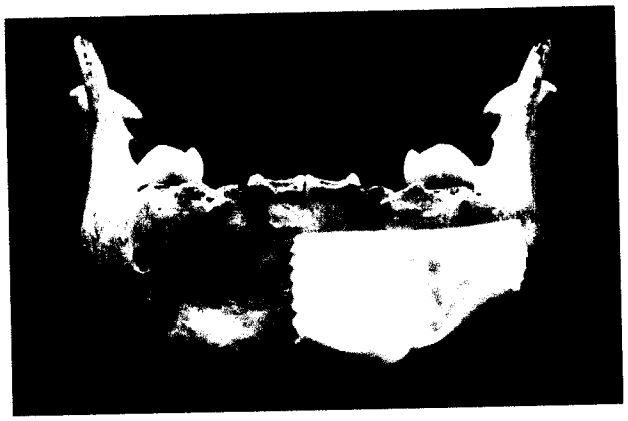


Figure 6.62 – SCR 10 Lateral curvature of coronoid processes (photo C. Wood).

SCR 14 – Dental Age 2-3 yrs, Skeletal Age 2-3 yrs

SCR 14 consists of the occipital, parietals, body of the mandible and fragments of the frontal bone, maxilla, ribs and ilia. Also present are the proximal ends of the ulnae and radii, the mid-shafts of the humeri, left femur and tibia. The femora are abnormally curved indicating genu valgum. The ends of all long bones cannot be observed because they are missing. Orbital porosity is apparent. No pictures are available.

SCR 94 – Dental Age 4-5, Skeletal Age 4-5 yrs

The skeleton is complete with the exception of the epiphyses of the arm bones, the mid-face and the sternum and sacrum. There is active pitting in the orbits, glabella and approximately 2 cm over the parietals. Some medial curvature of the proximal ulnae is apparent. No pictures are available.

SCR 102 – Dental Age 1.5 yrs, Skeletal Age 1 yr

The skeleton is complete with exception of the distal ulnae and sacrum. The proximal ends of the tibiae and distal ends of the femora are slightly flared. Orbital porosity is present. No pictures are available.

SCR 176 – Dental Age 7-8 yrs, Skeletal Age 10-11 yrs

The skeleton is complete with the exception of the epiphyses of the right humerus. There is healed pitting over the glabellar region and palate. The proximal ulnae appear bent (**figure 6.63**). Woven bone and micro-pitting is apparent on the anterolateral surface of the proximal shaft of the right tibia. There are many microfossae in the cortex of the femora. The thoracic vertebrae show severe porosity and anterior lesions in their bodies (**figure 6.64**).

Figure 6.63 – SCR 176 Slight bending of proximal ulnae (photo C. Wood).

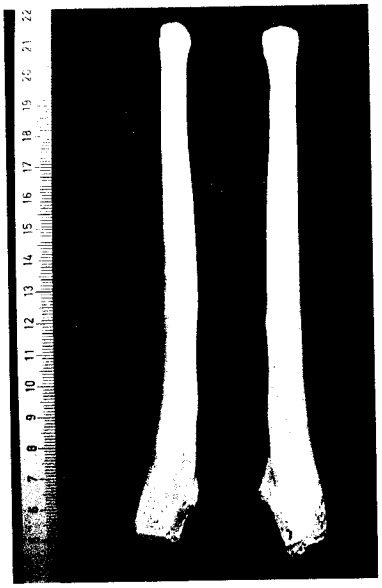
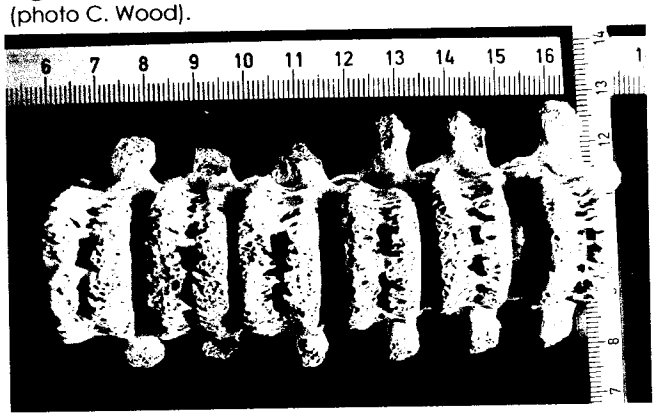


Figure 6.64 – SCR 176 Porosity of the thoracic vertebrae (photo C. Wood).



SCR 523 – Dental Age 5-6 yrs, Skeletal Age 5-6 yrs

The skeleton is complete minus some facial bones, the right humerus, sternum, sacrum, and all epiphyses. The left femur has been cut for sampling. The right femur is bent posteriorly and twisted. The diaphyseal ends of the long bones are porous. Orbital porosity is present. Linear enamel hypoplasia of the incisors and molars is apparent at the cemento-enamel junction. No pictures are available.

SCR 530 – Dental Age 10-11 yrs, Skeletal Age 8-9 yrs

SCR 530 consists of the skull minus the right temporal and facial bones, the right femur, distal ends of left femur, right tibia, diaphysis of left tibia, distal ends of the fibulae, portions of the humeri, radii and ulnae fragments and some vertebrae. There is subtle bending of the tibiae and femora. The vertebral bodies are compressed. No pictures are available.

SCR 542 – Dental Age 1-1.5 yrs, Skeletal Age 1-1.5 yrs

The skeleton is complete with the exception of the epiphyses of the long bones and some portions of the frontal, temporal and parietal bones. Slight flaring of the proximal and distal tibiae is apparent. There is porosity of the orbital roofs. No pictures are available.

SCR 612 – Skeletal Age 2-2.5 yrs

SCR 612 consists of the mandible and the left humerus. Some curvature of the left humerus and tibiae is apparent. The proximal ends of the tibiae are flared. No pictures are available.

SCR 644 – Dental Age 1-1.5 yrs

SCR 644 consists of a mandible and the diaphysis of the left femur. There is inward curvature of the mandibular condyles. No pictures are available.

SCR 683 – Dental Age 12-15 yrs, Skeletal Age 12-14 yrs

SCR 683 consists of the left mandible and maxilla, clavicles, scapulae, humeri, radii, proximal left ulna, ilia, right femur, distal left femur and some vertebrae. Lateral curvature of the right radius is apparent. The left radius is broken. The rib fragments show porosity. The vertebrae are porous. No pictures are available.

SCR 773 – Dental Age 0.75, Skeletal Age 0.5-1 yr

SCR 773 consists of the mandible minus the right ramus, clavicles, right scapulae, right humerus, and distal part of left humerus, left ilium, right radius, proximal portion of right ulna, midshaft of right femur, and the proximal portion of the right fibula. Medial bending of the head and neck of the mandible is apparent (**figure 6.65**). The right side is missing.

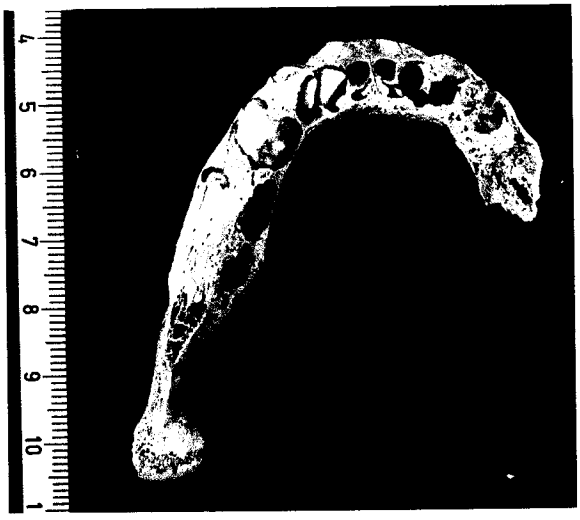


Figure 6.65 – SCR 773 Medial bending of the head & neck of the mandible (photo C. Wood).

SCR 780 – Dental Age 1-1.5 yrs, Skeletal Age 1-1.5 yr

SCR 780 consists of the occipital bone, scapulae, humeri, ulnae, left radius, right ilia, femora, tibiae, fibulae and some fragmentary vertebrae. Bending and flaring of the fibulae is apparent (**figure 6.66**).

Figure 6.66 – SCR 780 Bending of fibulae and flaring of ends (photo C. Wood).

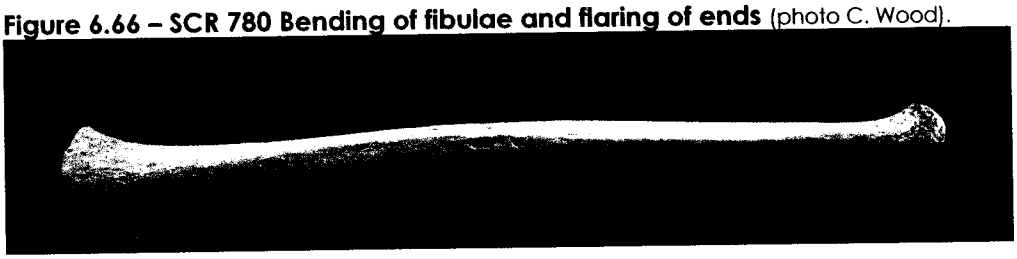


FIGURE 6.67 –

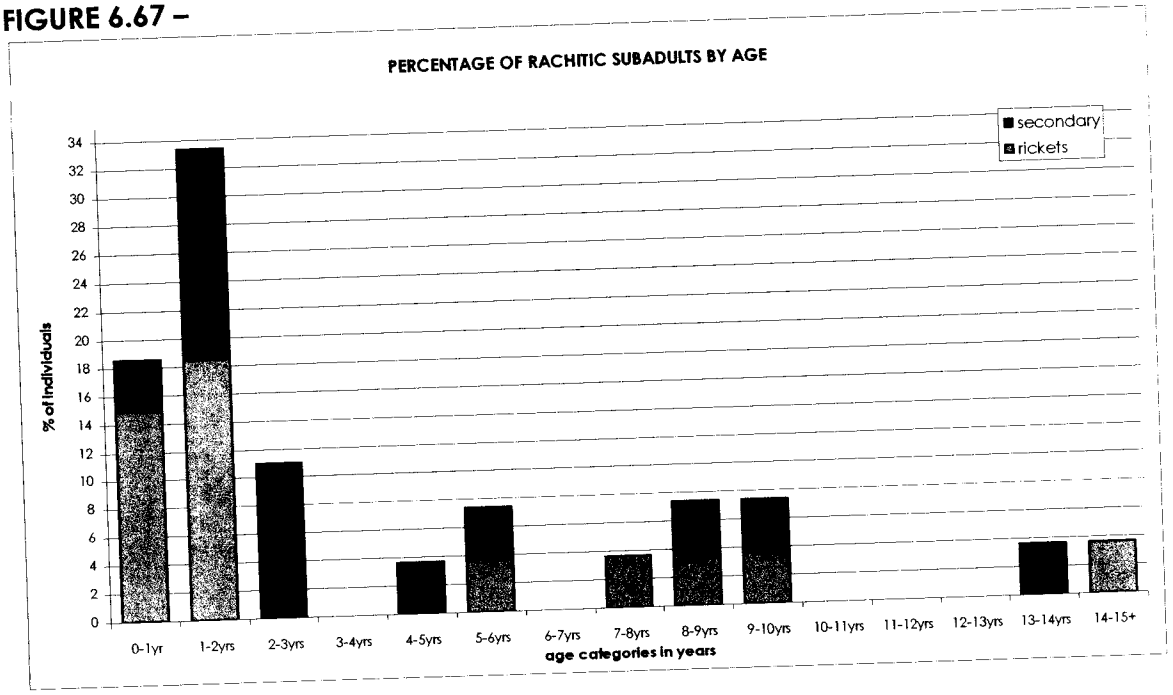
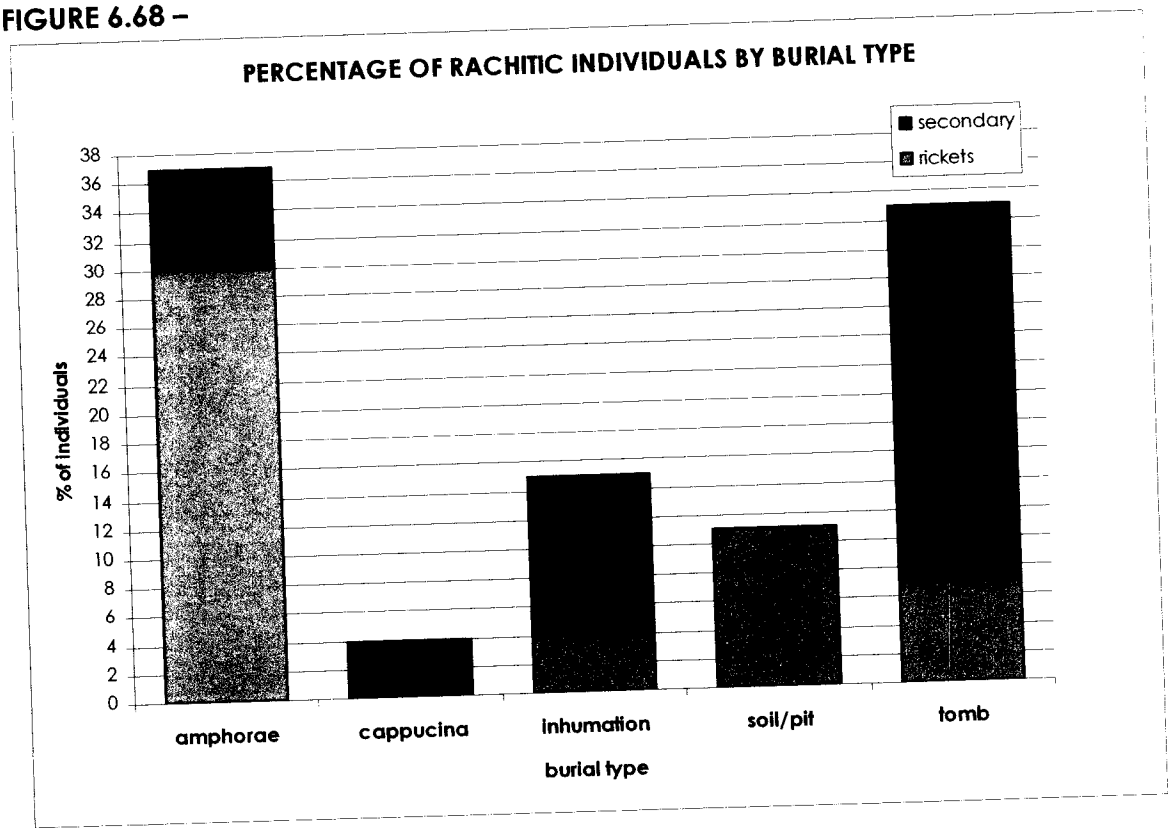


FIGURE 6.68 –



6.3 STATISTICAL ANALYSIS

6.3.1 – Relationship between age and rachitic and nonrachitic subadults

TABLE 6.5 – Age distribution categories of rachitic & nonrachitic subadults

Age categories	Non-rachitic	Rickets	Age categories	Non-rachitic	Rickets	Totals
0-1yr	22 (12.1%)	5 (2.8%)	1 - 2.99 yrs	65 (35.7%)	17 (9.3%)	82 (45%)
1-2yrs	26 (14.3%)	9 (4.9%)				
2-3yrs	17 (9.3%)	3 (1.7%)				
3-4yrs	12 (6.6%)	0 (0%)	3 - 5.99yrs	40 (22%)	3 (1.7%)	43 (23.7%)
4-5yrs	16 (8.8%)	1 (0.55%)				
5-6yrs	12 (6.6%)	2 (1.1%)				
6-7yrs	14 (7.7%)	0 (0%)	6 - 8.99yrs	28 (15.4%)	3 (1.7%)	31 (17.1%)
7-8yrs	10 (5.5%)	1 (0.55%)				
8-9yrs	4 (2.2%)	2 (1.1%)				
9-10yrs	4 (2.2%)	2 (1.1%)	9 - 11.99yrs	7 (3.8%)	2 (1.1%)	9 (4.9%)
10-11yrs	2 (1.1%)	0 (0%)				
11-12yrs	1 (0.5%)	0 (0%)				
12-13yrs	1 (0.5%)	0 (0%)	12 - 15yrs	15 (8.2%)	2 (1.1%)	17 (9.3%)
13-14yrs	5 (2.8%)	1 (.55%)				
14-15+yrs	9 (4.9%)	1 (0.55%)				
TOTAL	155 (85.1%)	27 (14.9%)	**TOTAL**	155 (85.1%)	27 (14.9%)	182 (100%)

Chi-square: age association

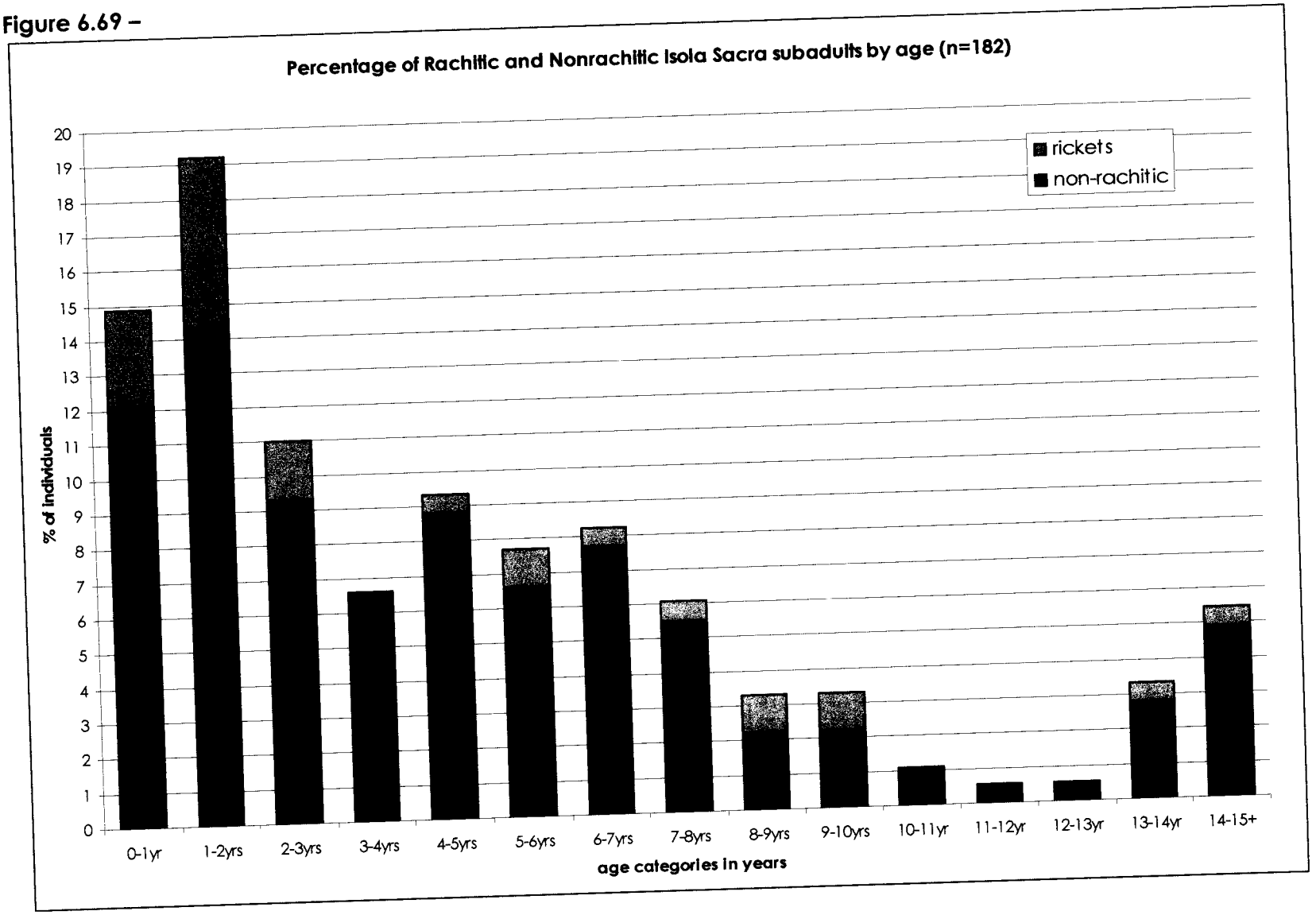
To test for the independence of the two variables, diagnosed rickets and age, the chi-square test was utilized. The null hypothesis states that there is no association between age at death and the appearance of rickets and that there is no statistically significant difference between rickets in each age group (H_o: the two variables are independent). If the chi-square value is equal to or

greater than the critical value, then we reject the null hypothesis. The standard value of p is 0.05.

Chi square = 5.53
4 degrees of freedom (5 categories – 1)
p value = 0.05
Critical value = 9.488

The chi-square 5.53 is less than the critical value 9.488 so the null hypothesis cannot be rejected. Therefore, there is no association between age and the appearance of rickets. The distribution of the percent of rachitic and nonrachitic subadults by age is found in **figure 6.69** below.

Figure 6.69 –


Percentage of Rachitic and Nonrachitic Isola Sacra subadults by age (n=182)
rickets
non-rachitic
% of individuals
0
1
2
3
4
5
6
7
8
9
10
11
12
13
14
15
16
17
18
19
20
0-1yr
1-2yrs
2-3yrs
3-4yrs
4-5yrs
5-6yrs
6-7yrs
7-8yrs
8-9yrs
9-10yrs
10-11yr
11-12yr
12-13yr
13-14yr
14-15+
age categories in years

6.3.2 - Relationship between Burial type and Rachitic and Nonrachitic subadults

TABLE 6.6 – Rachitic & nonrachitic subadults by burial type

Burial type	Non-rachitic	Rickets	Totals
amphorae	32 (17.6%)	10 (5.5%)	42 (23.1%)
cappucina	9 (5%)	1 (0.5%)	10 (5.5%)
inhumation	15 (8.2%)	4 (2.2%)	19 (10.4%)
soil/pit	12 (6.6%)	3 (1.6%)	15 (8.2%)
tomb	75 (41.2%)	9 (5%)	84 (46.2%)
unknown*	12 (6.6%)	0 (0%)	12 (6.6%)
Totals	143 (78.6%)	27 (14.8%)	170 (100%)

*unknown category not included

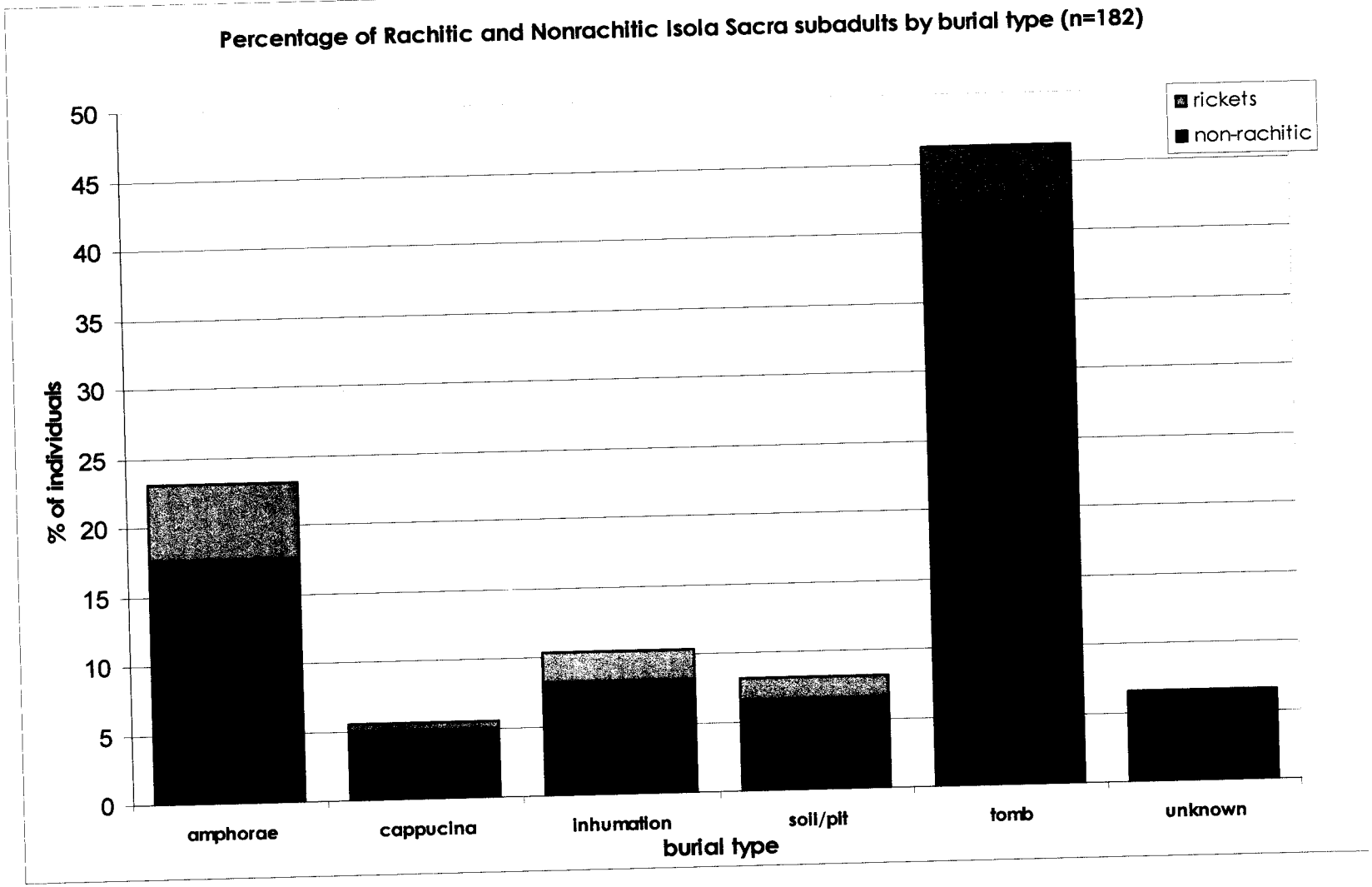
Chi-square: Burial type

To test for the independence of the two variables, diagnosed rickets and burial type, the chi-square test was utilized. The null hypothesis states that there is no association between burial type and the appearance of rickets and that there is no statistically significant difference between rickets in each burial group (HO: the two variables are independent). If the chi-square value is equal to or greater than the critical value, then we reject the null hypothesis. The standard value of p is 0.05.

Chi square = 4.48
4 degrees of freedom (5 categories – 1)
p value = 0.05
Critical value = 9.488

The chi-square 4.48 is less than the critical value 9.488 so the null hypothesis cannot be rejected. Therefore, there is no association between burial type and the appearance of rickets. The distribution of the percent of rachitic and nonrachitic subadults by burial type is found in **figure 6.70** below.

FIGURE 6.70 –


Percentage of Rachitic and Nonrachitic Isola Sacra subadults by burial type (n=182)
rickets
non-rachitic
% of individuals
50
45
40
35
30
25
20
15
10
5
0
amphorae
cappucina
inhumation
soil/pit
tomb
unknown
burial type

6.3.3 Summary

Fifteen percent (27/182) of Isola Sacra subadults show rachitic traits. Eight percent of these cases are considered severe while seven percent are milder in form. The Isola Sacra subadults suffered from hyperplastic rickets, less likely due to acute malnutrition although in the more subtle rickets cases, hypertrophic versus atrophic forms were difficult to determine. This would indicate that the majority of the Isola Sacra subadults identified as rachitic were fairly well nourished.

Rickets was better diagnosed by traits that result in bone deformities rather than by the presence of porosity. There was a great range in the morphological presentation of traits and the combination of traits varied widely. The most common rachitic traits are deformed arm (44%), and leg bones (41%), vertebral compression and porosity (44%), flaring and cupping of the metaphyses (44%) and cranial vault/orbital roof porosity (41%).

Six individuals showed active rickets prior to the onset of walking shown by deformities in the upper limbs as a result of crawling. Two individuals showed deformities of the upper and lower limbs indicating active rickets both before and after walking commenced. Seven individuals showed deformities of the lower limbs indicating active rickets only after walking began. Eight individuals, ranging in age from birth to 3 years, showed deformities of the mandibular ramus indicating that these individuals had started to be weaned on solid food requiring chewing or were chewing on hard non-food objects to alleviate the discomfort of teething.

All age categories (0-3, 3-6, 6-9, 9-12, 12-15+) and burial types showed rachitic traits. The highest number of rachitic cases was found between birth to age three, but no statistically significant association was found between age and the appearance of rickets. The highest number of rachitic cases was found in the tomb and amphorae burials but differences in burial type were not statistically significantly different.

Chapter 7

DISCUSSION

Not all children who suffered from rickets, or died of rickets or other causes when they were suffering from rickets, will be represented in a mortality sample. Rickets may not leave permanent and characteristic bone changes in every case of the disease. Children dying quickly, before telltale skeletal evidence of rickets manifests itself, would not be identified. This would especially be an issue in Roman times as it has been estimated that 25-40% of all children born in Roman antiquity died within their first year, with 33 -50% dying before age 10, while others have placed the mortality rate of infants to be 50% before age two (Jackson 1988; Bradley 1998). Rickets may have been a common disease but infants and children regularly survived, with complete healing and remodeling of skeletal lesions, suggesting the prevalence of rickets in the Isola Sacra sample is likely higher than found here. In the future, examination of healed cases of rickets in adults and older children in the Isola Sacra collection may give a better idea of the prevalence of this disease. Further proof of rickets could be gleaned from histological methods.

All skeletal features of rickets were seen in the Isola Sacra individuals with the exception of metaphyseal infraction and fracture of the long bones, likely due to the fact that radiographs were only available for a limited number of individuals. Rachitic traits involving the ribs were often difficult to observe due to

poor preservation. The squared appearance of the head was also difficult to determine due to the fragmentary nature of the cranial bones. There was a great range in the morphological presentation of traits and the combination of traits varied widely. The most common rachitic traits are deformed arm (44%), and leg bones (41%), vertebral compression and porosity (44%), flaring and cupping of the metaphyses (44%) and cranial vault/orbital roof porosity (41%).

The most probable explanation for the skeletal changes found in the Isola Sacra individuals is the hypertrophic or hyperplastic form of rickets. In most cases, the bone cortices are porous but thick, and are distorted in shape. This would indicate that the majority of the Isola Sacra subadults identified as rachitic were fairly well nourished. In the more subtle rickets cases, hypertrophic versus atrophic forms were difficult to determine.

Rickets active prior to the onset of walking can produce deformities in the upper limbs as a result of weight bearing as the infant crawls. Rickets active prior to walking is apparent in 6 individuals (SCR 47, 110, 140, 276, 612 & 636) from the ages of 6 months to 3 years. Active rickets before and after walking commenced produced deformities in both the upper and lower limbs of SCR 612 and 660. If rickets occurs after walking begins only the lower extremities will show the telltale bending deformities. This is apparent in SCR 1, 14, 175, 244, 523, 530 & 780 who range in age from 1 to 10 years of age.

Eight individuals (SCR 10, 47, 62, 140, 276, 644, 773 & 780), ranging in age from birth to 3 years, showed deformities of the mandibular ramus likely due to the result of muscle action during chewing. This deformity may indicate that

these individuals had started to be weaned on solid food requiring chewing or were chewing on hard non-food objects to alleviate the discomfort of teething (Ortner & Mays 1998).

All age categories (0-3, 3-6, 6-9, 9-12, 12-15+) and burial types showed rachitic traits. No statistically significant association was found between age and the appearance of rickets. Five of the rachitic individuals were under 1 year of age, suggesting low maternal stores of vitamin D contributed to rickets in these individuals. Two older subadult individuals, 14-15+ years in age (SCR 100) and 13-14 years of age (SCR 683) are diagnosed as having suffered from rickets. It was thought that these individuals may be female and possibly the product of vitamin D deficiency due to pregnancy. However, while SCR 683 is female, SCR 100 is possibly male (Sperduti 1995).

No statistically significant association was found between burial type and the appearance of rickets. The highest number of rachitic cases was found in the tomb and amphorae burials but this is likely because the greatest numbers of subadults are found in these categories.

Biocultural factors contributing to Rickets in the Isola Sacra subadult sample

The most important source of vitamin D is exposure to sunlight as 84% of circulating vitamin D is derived from non-ingested sources (Haddad & Hahn 1973; Holick & Adams 1998). Diet alone is an insufficient explanation for rickets based on the physiological and pathological basis of the disease and the great extent of information about predisposing behavioral factors.

7.2.1 *Roman Childcare Practices*

In many contemporary societies much of the knowledge concerning childcare practices is learned from other women and passed from one generation to the next. Much of the evidence used by modern scholars to reconstruct elements of ancient Roman life, such as Roman childcare practices, is based on surviving ancient literary texts. These writings are written from the point of view of upper class doctors, advising their male, upper class clients on what kind of regimen they should put their children and women through (Garnsey 1999). The majority of literary evidence is also likely biased towards descriptions of the Roman elite, as the life ways of the urban and rural poor were not usually the focus of the ancient authors (White 1976). There is no way of telling to what extent the literary prescriptions were put to use and how far down the social hierarchy the suggestions reached. It can be argued that any reconstruction of Roman life based on medical writers is prescriptive rather than descriptive and ideological rather than informative (Garnsey 1999). What is true is that these medical writers are commenting on the issues that were of

importance during their time as physicians, reflecting existing problems to which they wished to offer the best solution or if they could not, lay appropriate blame.

The most significant surviving text is the *Gynaecology* written by the Greek physician Soranus of Ephesus (A.D. 98-117). *Gynaecology* was one of the first pediatric guides and was considered the single most authority on infant feeding in the pre-industrial world (Fildes 1986a). Soranus's *Gynaecology* was in fact still authorative as late as the19th century (Garnsey 1991).

Many parents in antiquity unknowingly followed child-rearing practices that undermined the health and survival prospects of their children. The perilous risks of being an infant are demonstrated in the number of protective deities for children and childbirth (Harlow & Laurence 2002). There is Diespater for the birth, Mena and Lucina for women menstruating and childbirth, Opis and Levana for acceptance of the child; Vaticanus for the first cry, Cunina to guard the cradle, Rumina for breastfeeding, as well as many more (Harlow & Laurence 2002).

In ancient Rome, predisposing behavior appears to have begun immediately after the child was born. According to Soranus, Roman children were swaddled from birth, including complete covering of the head (Temkin 1956). The infant was wrapped mummy-like in a long linen bandage and kept this way usually for the first 6 months of life (Shorter 1982). Soranus stated that swaddling was practiced to give the child firmness and an undistorted figure (Temkin 1956). The practice of swaddling predisposed children to rickets, when coupled with confinement indoors and a poor diet. In fact, babies swaddled

and kept indoors would have a tendency to develop rickets even before dietary factors became an issue (Garnsey 1999).

7.2.2 *Infant feeding practices in Ancient Rome*

Soranus thought feeding on demand should not be practiced and the infant should not always be given the breast when it cried (Temkin 1956). Soranus recommended that Roman infants not be fed for two days after birth, unless the infant appeared to have an appetite, in which case it was to be fed boiled honey or goat's milk (Temkin 1956). Soranus also recommended a purge made of butter/oil of sweet almonds combined with sugar/honey/syrup (Fildes 1995). After two days, milk from a wet nurse was given to the infant, but milk from a woman who had given birth 2-3 months earlier was recommended, as it was believed to be of better quality than that of the recently delivered mother. Colostrum was thought to be unhealthy for the infant (Temkin 1956). Galen, however, makes no mention of this (Colón & Colón 1999; Garnsey 1999).

Fildes (1986a) believes wet nurses were a means of spacing children closer together. During this time Romans were aware that lactation inhibited pregnancy (Garnsey 1999). Wet nurses were common among the wealthy Roman classes and in the empire, at large (Garnsey 1999). In Roman period Egypt, wet-nursing contracts from ancient times have been discovered. The contracts range from periods of 6 months to 3 years, with 2 years being the average (Fildes 1986a; Garnsey 1999). Soranus argued that wet nursing was an efficient means of rearing children and it is likely that wealthy parents were

influenced by his writings, to adhere to the *status quo* (Temkin 1956; Garnsey 1999). The poor, who were not constrained by trends in child rearing, may have been at an advantage, as a baby fed by its own mother has a greater resistance to disease, despite the mother's nutritionally deficient diet (Garnsey 1999).

The distrust of colostrum has a long history in Europe and still exists in present-day third world countries (Fildes 1986a; Fildes 1995). Colostrum is three times richer in protein than mature human milk, and in the first six weeks of life provides antibodies and proteins, which guard the newborn against infections, particularly in the gastrointestinal tract (Fildes 1986a: Cunningham 1995; Stuart-Macadam 1995). The introduction of cow's milk or any other food interferes with the immunological protection provided by breast milk by altering the pH of the stomach and intestines, which creates a medium for the growth of bacteria (Venkataraman *et al.* 1985; Stuart-Macadam 1995). Breastfeeding provides a system whereby the mother's body manufactures antibodies to microorganisms that the infant is exposed to and transmits these antibodies to the infant via the breast milk (Cunningham 1995). The body's entry point for microbial or allergenic invaders is the mucous membrane of the aeroalimentary system, known as the respiratory and gastrointestinal tracts (Cunningham 1995). It is this secretory system that is the primary defender against invasion. The secretory immune system is a system of antibody producing cells that lines the mucous membranes of the gastrointestinal and respiratory tracts (Cunningham 1995). These cells produce secretory immunoglobulin A – (S-IgA). Newborn infants have antibodies

circulating in their bloodstream that are acquired transplacentally from their mother but their secretory immune system is not functional and requires additional time and exposure to pathogens for its development (Cunningham 1995). Colostrum and human milk contain abundant quantities of S-IgA and in fact, the mammary glands contain cells that produce S-IgA (Cunningham 1995). The sources of these cells are the enteromammary and bronchomammary axes (Cunningham 1995). Gastrointestinal and respiratory tract pathogens encountered by the mother stimulate the development of cells that line her mucosal membrane, which are cells that produce S-IgA (Cunningham 1995). The S-IgA then acts as a barrier to invasion of the blood and organs systems and is considered the first line of defense (Cunningham 1995). Simultaneously some of the S-IgA producing cells circulate to the mammary gland in order to produce S-IgA for the infant (Cunningham 1995).

The association between rickets and the denial of colostrum is related to a compromised immune system, specifically a strong correlation with acute respiratory infections, especially pneumonia. It has not been established if vitamin D deficiency could be the result of immunologic abnormalities and pneumonia, or if hypocalcemia results in poor clearing of the bronchopulmonary secretions (Chesney 2001). Vitamin D is known to have effects on immunological function in animals and may be a factor responsible for reduced immunocompetence (Maxwell 1994). New research is showing that additional biological roles of vitamin D in many organs and tissues are related to cellular growth and differentiation which suggests that clinically dissimilar diseases may

share an aberration in vitamin D (Maxwell 1994). As previously mentioned, it is the actions of $1,25(OH)_2D$ that alter gene transcription directly in combination with the vitamin D receptor (VDR) (Holick & Adams 1998). Holick and Adams (1998) believe that $1,25(OH)_2D$ has immunoregulatory effects. The expression of the VDR in mitogen or antigen activated human lymphocytes and peripheral blood monocytes as well as the ability of stimulated macrophage-like cells to make $1,25(OH)_2D$ suggests the production and interaction of active vitamin D metabolites with cells in the lymphocyte and monocyte/macrophage lineage may modulate the immune response in animals and humans (Holick & Adams 1998). A variety of tissues and cells that are not the usual target tissues for vitamin D have nuclear receptors for $1,25(OH)_2D_3$ (Holick & Adams 1998). The brain, parathyroid gland, gonad, thymus, pancreas and skin have high-affinity, low-capacity nuclear receptors for $1,25(OH)_2D$ as do many circulating mononuclear cell populations such as monocytes and activated B and T lymphocytes (Holick & Adams 1998). $1,25(OH)_2D$ may help regulate hormone secretion, induce maturation of a variety of tissues including the skin, alter immune function, and affect myocardial activity, although its physiological importance is not known (Holick & Adams 1998).

Colostrum aids in the evacuation of meconium in the neonate's intestine (Fildes 1995). It could be hypothesized that infants denied colostrum, resulting in the improper evacuation of meconium may somehow impair the absorption of vitamin D in their intestines. However, it is possible that if a child did not receive colostrum, he or she would be more likely to die before rickets is seen in the

skeleton as the paradox of rickets is that the child has to be growing sufficiently to develop the disease.

7.2.3 *Health Status of the Mother*

Adequate growth and development of the newborn infant depend upon the quantity and quality of available fetal stores and ingested milk, the efficiency of gastrointestinal absorption and energy expenditure (Boersma *et al.* 1991). Low maternal vitamin D intake likely played a central role in the prevalence of rickets throughout history. The source of rickets for newborn children has been difficult to determine. It has been presumed that the diet is a source of vitamin D for very young children. In many species of mammals including humans, newborns are cared for away from sunlight and as a result, early postnatal vitamin D can come from neither dietary nor solar sources. The only other possible source is a reserve laid down before birth (Fraser 1983). At birth in human infants, the circulating 25(OH)D concentration is strongly correlated with that in the maternal blood (Fraser 1983; Park *et al.* 1987). Therefore, fetal requirements of vitamin D are obtained directly from the maternal pool (Fraser 1983). Premature infants appear to have only a limited ability to synthesize 25(OH)D, with this activity increasing at the time corresponding to full-term gestation (Fraser 1983; Bates & Prentice 1994). As there is low fetal 25(OH)D activity, the 25(OH)D is being supplied from maternal metabolism. Vitamin D that crosses the placenta will accumulate unused in fetal hepatic tissues and will be available to meet postnatal needs (Fraser 1983). Therefore, infants of vitamin D deficient mothers are at increased

risk for rickets because vitamin D stores of the newborn are entirely dependent upon the mother (Bachrach *et al.* 1979; Fraser 1983; Tomashek *et al.* 2001). The fetus is protected from any disturbance in calcium homeostasis, while any vitamin D the mother ingests, goes to the fetus first (McCaffree 2001).

Not surprisingly, it has been found that osteomalacia of the mother results in rickets of the newborn. Neonates of mothers with osteomalacia are born with softened cranial bones, rarefied bone structure, widened distal metaphyses and cupping of the radius and ulna and occasional fracture of arm bones (Park *et al.* 1987; Mankin 1990). Five (SCR 47, 62, 140, 511 & 773) of the Isola Sacra infants aged birth to 1 year showed signs of rickets. These infants showed thickening of the cranial vault, deformed mandibular rami, widened distal metaphyses, thickened and cupped radii and ulnae, flaring and fracture of ribs, enamel defects and general porosity of all skeletal elements.

The most important factor influencing the vitamin content of human milk is the nutritional status of the mother. Vitamin D content of breast milk varies with maternal vitamin D intake, which is dependent upon the mother's sun exposure and nutrition (Rothberg *et al.* 1982; Tomashek *et al.* 2001). When maternal vitamin intake via foods is low, human milk vitamin levels are low and respond to supplementation (Olafsdottir *et al.* 2001). However, when the maternal intakes are high, milk vitamin levels approach a plateau and are less responsive to supplementation (Olafsdottir *et al.* 2001). The vitamin D content of breast milk is low, even with adequate maternal vitamin D intake (Tomashek *et al.* 2001). Maternal calcium intakes likely have little influence on breast milk calcium levels

(Bates & Prentice 1994). Provided that the vitamin D supply is adequate, then the calcium and phosphorus requirements of term infants can be provided by breast milk for at least 26 weeks postpartum (Bates & Prentice 1994).

7.2.4 *Weaning and Weanling diet*

Modern health experts acknowledge that infants being weaned are particularly vulnerable to disease. They identify two periods of particular concern. The first period begins at around 3 months of age and is associated with the initial introduction of supplementary foods, which may be nutritionally suspect and unhygienically prepared (Fildes 1986a; Garnsey 1999). The risks to the infant at this time are increased morbidity and the predicament of "weanling diarrhea" (Garnsey 1999; Moffat 2001). The second period of high vulnerability to disease occurs when the supply of breast milk falls behind need, and the child is increasingly dependent upon inadequate weaning foods (Garnsey 1999). This period is estimated to begin at 9 months of age and is characterized by an increase in growth velocity, thought to continue for approximately a year (Garnsey 1999). It is often characterized by nutritional deficiency, in addition to a high susceptibility to infection and malnutrition.

Soranus recommended weaning begin between 6 and 7 months of age and finish between 2 and 3 years of age. Prowse (2001) found support for this scenario as isotopic evidence from the Isola Sacra subadults indicated that they were likely breastfeeding for a short period of time after birth, followed by a longer period during which breast milk was gradually removed from the diet. This

extended weaning period began just after 3 months and lasted until 2 years of age (*ibid.*).

The most common weaning foods were cereals and crumbs of bread, softened with milk or wine (Garnsey 1991; Colón & Colón 1999). When the infant got a little older Soranus recommended a soup made of spelt, a very moist porridge, and an egg that could be sipped (Fildes 1995). Evidence of this practice is seen in isotopic values for the Isola Sacra site in Rome (Prowse 2001) and the Dakhleh oasis in Roman Egypt (Dupras *et al.* 2001). Foods that contain vitamin D, with the exception of egg yolks, are not typically found in infant or toddler diets (Tomashek *et al.* 2001).

When reviewing the genetic causes of rickets (chapter 2, pgs. 20-23) only celiac disease was considered a concern for the Isola Sacra population as its incidence is relatively high (1 in 250 Americans) (Halsted 1996). Celiac disease, also known as gluten intolerance, damages the villi in the intestines when specific food-grain antigens found in wheat, rye and barley are consumed (Halsted 1996). Gluten is a structural protein in all wheat, rye and barley (Greco 1995). The high incidence may in fact be the consequence of modern diets consisting of highly refined and gluten rich foods (*ibid.*). Over the last 200 years active genetic selection changed wheat dramatically; from few grains and little gluten, to large harvests and highly enriched gluten content (50% of the protein content) (Greco 1995).

The main types of grains consumed in the Italian diet during the pre-Roman and early periods were spelt, emmer wheat and barley (Moritz 1958).

Barley was considered an inferior quality of cereal, suitable for slaves, animals and the poor and never actually attained popularity in the diet of ancient Romans (*ibid.*). Emmer wheat was the main cultivated wheat in early Roman Italy and mainly for the production of bread (*ibid.*). Spelt, the porridge commonly consumed by infants is in fact poorer in gluten content than other types of wheat (Greco 1995). While celiac disease appears to be a modern phenomenon, it is possible that the Romans could in fact represent the first cases of celiac disease as a consequence of the high content of grains and thus gluten, in their diet.

Cereals are low in calcium and almost totally deficient in B_2 riboflavin, vitamins A, C, and D, with the exception of thiamin and vitamin E (Passmore & Eastwood 1986; Sippel 1987; Garnsey 1999). Phytate acid found in cereals, especially in bran and the germ, impedes the absorption of vital minerals such as calcium and iron (Mellanby 1949; Garnsey 1999). The main anti-calcifying effect of phytate occurs because it competes in the intestine with its structural analogue, inorganic phosphate, for calcium (Mellanby 1949). As a result, phytate limits the amount of calcium available for absorption under the influence of vitamin D. While vitamin D in sufficient quantity prevents phytate from producing rickets, it cannot suppress the anti-calcifying effect of phytate (Mellanby 1949). Even very high vitamin D intake (1000 i.u. daily) does not increase the amount of calcium absorbed from the intestine and still results in bones that are less calcified and more osteoporotic than when phytate is absent from the diet or replaced by inorganic phosphate (Mellanby 1949). A diet high in

phytate requires not only an adequate amount of vitamin D but also a high calcium intake (Mellanby 1949). Although uncommon, low dietary calcium intakes may be a factor in the pathogenesis of rickets by either exacerbating vitamin D deficiency or by directly precipitating hypocalcemia and cannot be ruled out as a factor in the Isola Sacra subadult sample.

In ancient Rome, the quality of cereal and flour used was class specific; the lower the class, the lower the quality of cereal available (Garnsey 1999). The highest quality of flour for bread making was produced by at least two grindings and siftings using either a hand mill or an animal-driven mill (**figure 7.1** below) (White 1988).

Figure 7.1 - Millstones in a Bakery in *Ostia Antica* (photo S.R. Saunders).

It was the siftings, not grindings of the grain that would produce different qualities of flour (Moritz 1958). The ways in which cereals are processed make a considerable nutritional difference. Flour inefficiently sieved would have high phytate content, the higher the phytate content the more deprived of vital

minerals the body would likely be (Garnsey 1999). Low extraction (over-sieved) flours have lost much of their mineral content but what remains may be better absorbed because of a loss of phytate during the extraction process (Garnsey 1999). While the social status of the inhabitants of *Portus Romae* were considered middle-class (Meiggs 1960; Mannucci & Verduchi, 1996), it is difficult to ascertain the quality of flour they would have eaten.

7.2.5 *Socioeconomic Aspects of Food Allocation in Roman Society*

Food behaviours reflect the social hierarchy and social relationships. The status of an individual in the household, and in society in general, will be crucial in food allocation. Patterns of infant and child feeding, based on cultural beliefs affect the nutritional status, health and growth of children. Patterns of infant feeding have underlying cultural beliefs concerning the nature of the child, the nature of food, how, when, and what kinds of foods children should eat (Dettwyler 1987). The dietary regime of a middle-class population from *Portus Romae* would be expected to fall somewhere between that of the urban elite of Imperial Rome and of the rural peasantry in the surrounding countryside (Prowse 2001).

The Roman dinner table was more about public display than family values. Although women and children were not completely excluded, dinner at the Roman home was for the man of the house, and his male friends above all (Bradley 1998). The division of food in the Roman family was based on needs, status and power (Garnsey 1999). The functional and physiological explanation

of food distribution is the equivalent of "needs" (Garnsey 1999). Garnsey (1999:101) states: "if the overall aim is to secure the survival and well-being of the family, then the larger share will go to its most productive members, the workers".

Power, or control over resources, with a focus on material and power relations is another aspect of Roman food distribution systems within the family. Cultural bias is evident in the history of the Roman family especially in regard to children. Children were expected to eat more "frugal foods", and older children were favored (Garnsey 1991; Bradley 1998). Prowse (2001) found that subadults in the Roman site of Isola Sacra were consuming a predominantly herbivorous diet and were eating at a lower trophic level than others in the sample. Beser and Cakmakci (1994) found that the prevalence of rickets is found to be three times higher in children who do not eat fish when compared to those who do. In patriarchal societies, such as Greco- Roman society, women would have been given less of a share of the family food resources than men (Garnsey 1999). Status, power and control do not act in the interests of women in Roman society. Greater emphasis would have been put on the needs of men and older male children. In societies where males are preferred, "women may themselves be the instruments of their subordination" as they may have not fed children of both sexes equally (Garnsey 1999:112).

Roman girls were married young, often by the age of 15 and at the latest 18 (Jackson 1988; Harlow & Laurence 2002). The Roman concept of marriage was an institution for the procreation of children and large families were the norm (Jackson 1988; Harlow & Laurence 2002). An increase in skeletal density is

critical during pregnancy and lactation. Increased serum $1,25(OH)_2D_3$ concentrations of pregnancy promote augmented intestinal calcium absorption from the maternal gut, because the skeleton loses bone mass during lactation (Chesney 2001). If a woman becomes pregnant during late puberty, when bone mass is increasing most rapidly, she is at risk of even greater skeletal loses during pregnancy (Chesney 2001). If pregnancy occurs before major modeling has ceased in mid-puberty, the skeleton loses bone mineral with lactation at a period prior to the rapid gain in bone mineral density (Mora *et al.* 1999). Two older subadult individuals, 14-15+ years in age (SCR 100) and 13-14 years of age (SCR 683) suffered from rickets. It was thought that these individuals may be the product of vitamin D deficiency due to pregnancy. One of these individuals is female SCR 683 however the other SCR 100 is possibly male (Sperduti 1995).

Medical writers of the time reflect power, status and need in their suggestions of a dietary regime appropriate for girls and women. Women were to have a "simple diet" and it was recommended to "regulate and moderate their intake of food and not let them touch meat at all, or other foods that are very nourishing" (Oribasius, Liber Incutus, in Garnsey 1999:101). Roman doctors were seemingly concerned with limiting the food consumption of females whether rich or poor or young or mature. This was put forth as knowledge regarding the "needs" of women. It was justified as women did not, or were not expected to work; they had reduced food-energy requirements and should be fed less than active men (Garnsey 1999). However, medical writers of the time were aware that pregnant women required more nourishment and should eat

and exercise more (Garnsey 1999). The idea that women required less food than men must be received in the context of male interest and a male-dominated patriarchal society. It is not known how closely the advice of male doctors was followed.

Another relevant factor regarding the physiological needs of women was the concept that good health was the result of the harmony of the four bodily fluids, or "humours" (Garnsey 1999). Food and drink were two of the factors that affected the humours. Women were perceived to have a cold and wet constitution, which needed to be weighted towards the warm and dry. Women were advised to avoid foods that would make them colder and wetter such as: "eels, sheat-fish, sturgeon, turbot and in general river fish, fatty fish and meats that come from newborn animals" (Garnsey 1999:105).

The Romans consumed a wide variety of marine fish, freshwater fish, and seafood, although this aspect of the Roman diet is not widely discussed by modern scholars (André, 1981; Brothwell 1988, 1998; Frayn, 1993). Again, the best dietary source of vitamin D is fish oil, fatty fishes and shrimp (Stuart-Macadam 1989; Holick 2001). Fish were considered expensive food items by some ancient authors (e.g., Juvenal and Pliny), suggesting that the regular consumption of fish may have been restricted to the elite (Frayn, 1993). Fish consumption may have been higher along the coastal regions, so that the proportion of fish and seafood in the diets of the people of *Portus Romae* was likely higher than the 'typical' consumption levels for inland populations (Prowse 2001). However, Prowse (2001) found a slight isotopic ($\delta^{13}C$ and $\delta^{15}N$) difference between males and

females suggesting that females were probably consuming more terrestrial resources, while males were consuming a greater portion of marine foods. This may in fact have had implications for the vitamin D status of women.

Other good sources of vitamin D are egg yolks and chicken liver. Chickens were found on every farm and used for the production of eggs, but goose, rather than chicken was the preferred poultry meat (Dosi & Schnell, 1986). Methods for the preservation of eggs were known during the Roman period and eggs were widely consumed, often at the beginning of a meal (Brothwell 1988, 1998; Dosi & Schnell, 1992).

Besides status, Roman women and children were prey to a parallel aspect of social hierarchy, power. Power in this case, can be taken to be male control over resources. In Roman society women did not work, in the same sense that men did, or control the product of their work, therefore limiting their access to power. In upper class society, where women were confined to the home, they were handed the power to indulge their alleged weaknesses in the matter of food and drink and, as a result, culturally prescribed limitations were thought to be needed (Garnsey 1999). In a rural context, however, women participated in agriculture and may have been allotted a greater portion of the food resources (Garnsey 1999). While diet and energy demands of work may have discriminated against the poor and laboring classes of women, by working outside, rural women would have access to the most important source of vitamin D, sunlight.

Garnsey (1999) states that it is improbable that the advice of men who were practicing doctors was completely ignored, even if there are few ways of measuring its impact. One could predict that malnutrition and morbidity would have been more widespread and serious among women, especially those of childbearing age. No figures are available for the maternal death rate. It has been estimated by Jackson (1988) that the rates would be similar to England between the 16th and 18th centuries, 25/1000 maternal deaths. Joshel (1986) found that inscriptional evidence from Roman tombs suggests a higher death rate for women in their reproductive years. Unhealed childhood rickets would have had serious implications for the reproductive health of females later in life. Bone deformities of the pelvis in women leads to obstructed labour and increased perinatal morbidity and mortality (Hess 1929; Bishop 1999).

As Garnsey (1999) states, access to food is a reflection of the social and economic distinction between different classes in any society. Available foods may be identified through local production or trade, but access to these foods was determined by an individual's status in that society.

Food shortages and famine are widely recorded in the ancient literature and were influenced by climatic fluctuations causing crop failures, political instability affecting distribution and transport of supplies, or warfare (Garnsey 1999). The literary evidence suggests that there were status-based differences in the diet of the Roman population, with non-elites having limited access to expensive food items such as fish and meat and possibly consuming greater quantities of 'inferior' grains (Prowse 2001). Garnsey (1999) re-examined the

question of malnutrition in antiquity, and suggests that malnutrition and disease were probably more common among women and children in the ancient Mediterranean, due to socio-cultural factors.

7.2.6 *The Habits of Roman Women: lifestyle, clothing, and beauty ideals*

Galen comments on the habits of Roman women writing that they "stay indoors, neither engaging in strenuous labour nor exposing themselves to direct sunlight" (Garnsey 1999:48). The Romans believed that it was "a finer thing for the woman to stay indoors", than to spend time in the open (Lefkowitz & Fant 1982:146). Husbands preferred their wives to stay indoors and to "send out those servants whose work is outside" rather than have the wives go out themselves (Lefkowitz & Fant 1982:146). These sentiments likely reflect the wishes of men as women must have gone out to shop, gone to the baths, visited friends, worshiped at temples and to attended public spectacles (Baldsdon 1974). It is thought that Roman women in fact enjoyed greater freedom than their Greek counterparts (Dixon 2001). The cloistering of Roman women was likely primarily confined to elites who could rely on a multitude of servants to do their bidding. It is unlikely that the middle-class women of *Portus Romae* were confined indoors.

Women's clothing was to be simple and nontransparent (Lefkowitz & 1982). In public, women wore a stola, which was essentially a tunic that extended to the ground over the feet and a palla, which is a very large, rectangular shawl used to cover the head, its lower edge extending as far as the knees (Baldsdon 1974). In early Republican Rome when a woman went outdoors

no more of her body would be exposed than a nun in full habit (Baldsdon 1974). Women were to cover their heads when outdoors, as failure to do so might result in punishment and in one case were grounds for divorce (Lefkowitz & Fant 1982). This husband, Gaius Sulpicius Gallus, explains his reasoning as the law prescribes; you for my eyes alone, "if you invite the look of someone else, you must be suspected of wrongdoing" (Lefkowitz & Fant 1982:176). The palla survived without change until the third century A.D. and in fact the style of women's clothing in general changed very little for three hundred years, with the exception of the colour and texture of the material (Baldsdon 1974). The stola and palla bear close resemblance to the burca of Muslim women. In contemporary society, Muslim women are at the greatest risk for osteomalacia and consequently have an increased incidence of rickets in their children. Lack of the skin's exposure to sunlight through culturally prescribed dress may have had serious implications for early infancy and maternal stores of vitamin D passed during pregnancy to the placenta (Garnsey 1999; Mughal *et al.* 1999).

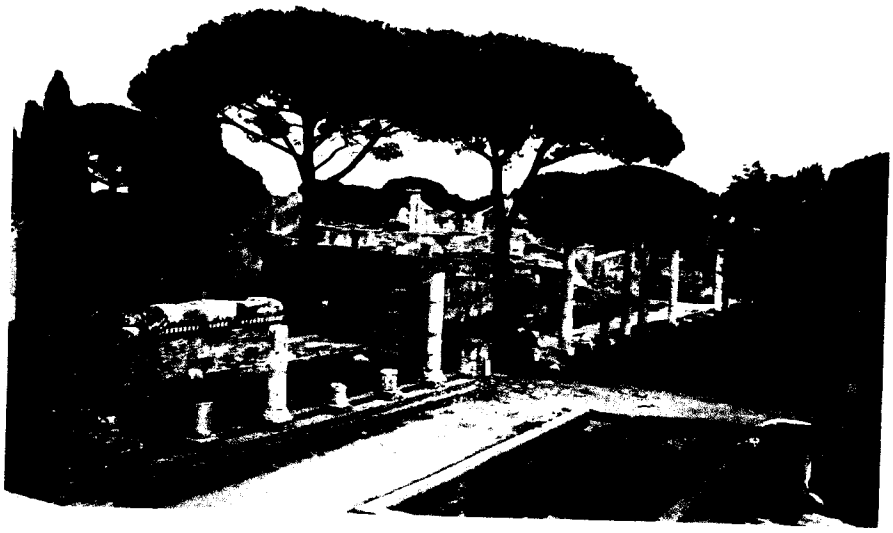
Women whitened their faces using white-lead (cerussa) and chalk or Melian-white (melinum) (Baldsdon 1974; Wyke 1994). This suggests there may have been beauty issues connected with the avoidance of sun. Many cultures avoid sun as lighter skin is thought to be more attractive. As well, lighter skin tone is sometimes a symbol of higher status whereas those who had to work in sunshine would be much darker. The outdoor work of the poor and laboring classes of women would have given them access to a constant supply of vitamin D. Those of upper status who wished to convey a life of leisure may have

avoided the sun and consequently limited their access to vitamin D. Additionally, the white lead and chalk on the face may have impeded absorption of ultraviolet rays. Again, sun avoidance due to beauty and status issues may have been detrimental for early infancy and maternal stores of vitamin D.

7.2.7 *Issues of the Environment: the consequences of urbanization.*

Environmental conditions provide opportunity and constraint to human action and health (Thomas 1998). Children still suffer from rickets in a Mediterranean environment in modern times (Garnsey 1991). Rickets was thought to be rare before the Roman period brought the growth of cities (Grmek 1983, in Garnsey 1991). However, cities are not necessarily a defining characteristic of the Roman period. There are other earlier cities, like Alexandria, that make no mention of rickets. Perhaps it is apartment/tenement dwelling that is a defining characteristic of the Roman period. The lack of sunlight in urban environments due to the closely spaced, overhanging houses of Rome may have allowed very little sunlight to penetrate into the street. It has been suggested that even when mothers went out with their children, the Roman style architecture blocked out much of the available sunlight (Sear 1992). While the dwellings of *Portus Romae* have not yet been excavated, a street in *Ostia Antica* illustrates the narrowness of Roman streets (**figure 7.2** below).

Figure 7.2 – Street in *Ostia Antica* (photo S.R. Saunders).



The residential population density of *Portus Romae* is likely less than Rome, but the living situation was not necessarily much more spacious. There seems to have been a preference for living in close quarters perhaps a consequence of costs (Mancioli 1989). Most people lived in apartment buildings with few or small windows, working on the main floor and living on the upper floors (Mancioli 1989). Windows and balconies opened onto the street or an internal courtyard (*ibid.*). Windows were closed with blinds, skins or soportelloni, a solid wood window covering propped open at the bottom. (Mancioli 1989). Windows were placed such that sunlight could only come in at certain hours of the day, with lamps and candles used for light *(ibid.)*

The environment of infants and young children can be quite different from the adult situations. It is possible that children were not exposed to the sunlight easily, having been swaddled and living in small apartments or houses. Still, in a

climate like that of south central Italy, it seems much more probable that socio-cultural and socioeconomic factors were of primary importance, more than socially constructed environmental factors. It is social factors that dictate disadvantageous Roman childcare practices, and undermine maternal stores of vitamin D which in turn may have influenced the prevalence of rickets in the Isola Sacra subadult population.

Chapter 8

CONCLUSIONS

This study utilized two independent forms of evidence, skeletal and historic, to study the prevalence of rickets in an archaeological sample of subadults. Fifteen percent (27/182) of Isola Sacra subadults show rachitic traits. It is suspected that an additional 13% or 23 individuals did in fact suffer from rickets but due to a lack of skeletal elements could not be properly diagnosed. The Isola Sacra subadults suffered from hyperplastic rickets, which is less likely due to acute malnutrition. Observation of traits in individuals showed great variation in what traits were expressed in any one individual. To some extent this may be dependant upon age, yet, this can be used to demonstrate the importance of using numerous traits for disease diagnosis. This study found that rickets is better diagnosed by traits that result in bone deformities, for example shape or girth, rather than by the presence of porosity. There was a great range in the morphological presentation of traits and the combination of traits varied widely. The most common rachitic traits are deformed arm (44%), and leg bones (41%), vertebral compression and porosity (44%), flaring and cupping of the metaphyses (44%) and cranial vault/orbital roof porosity (41%).

Active rickets was found prior to walking shown by deformities in the upper limbs as a result of crawling. Deformities of the upper and lower limbs indicated active rickets both before and after walking commenced. Active

rickets was apparent only after walking began indicated by deformities of the lower limbs. Deformities of the mandibular ramus in subadults aged birth to 3 years indicated that these individuals had started to be weaned on solid food or were chewing on hard non-food objects to alleviate the discomfort of teething.

All age categories (0-3, 3-6, 6-9, 9-12, 12-15+) and burial types showed rachitic traits. No statistically significant association was found between age and the appearance of rickets, nor was any association found between burial type and the appearance of rickets. The highest number of rachitic cases is found in the tomb and amphorae burials but differences in burial type were not statistically significantly.

While the residential population density of *Portus Romae* was less than Rome, spacious living was not cost effective. In a climate like that of south central Italy, it seems much more probable that socio-cultural and socioeconomic factors were of primary importance, more than socially constructed environmental factors.

Roman childcare practices unknowingly predisposed children to rickets and undermined their health and survival prospects. Such practices include: the swaddling of infants from birth, denial of colostrum, wet nursing, and the use of nutritionally poor weaning foods. The denial of colostrum may result in the improper evacuation of the meconium and may somehow impair the absorption of vitamin D in the intestines. Vitamin D is known to have effects on immunological function in animals and may be an additional factor responsible for reduced immunocompetence.

Although the vitamin D content of breast milk is already low, in comparison to nutritionally poor weaning foods such as cereal, breast milk would still provide the largest ingested source of vitamin D. For the children of Isola Sacra weaning began just after 3 months (Prowse 2001) resulting in a reduction of the best source of vitamin D relatively soon in life.

Cereals were a major food source for the ancient Romans and a porridge made of spelt was the main weaning food for children. While spelt is relatively low in gluten, the high gluten content of other grains in the Roman diet may have resulted in gluten intolerance or celiac disease which produced osteoporosis. The high phytate found in cereals can impede the absorption of calcium which results in bones that are less calcified and osteoporotic. Therefore although rare, low dietary calcium intakes cannot be ruled out as a factor causing rickets in the Isola Sacra subadult sample.

Patterns of infant and child feeding, based on cultural beliefs affect the nutritional status, health and growth of children. Roman texts suggest children eat more "frugal foods", reflecting their designated lower status (Garnsey 1991; Bradley 1998). Isotopic data indicates that subadults in the Roman site of Isola Sacra were in fact eating at a lower trophic level than adults in the sample (Prowse 2001).

As vitamin D stores of infants are entirely dependent upon the mother, low maternal vitamin D is likely a strong contributing factor of rickets in the Isola Sacra sample. Five of the rachitic individuals were under 1 year of age, suggesting maternal stores of vitamin D contributed to rickets in these individuals.

A number of socio-cultural factors influenced maternal nutrition and sun exposure. Marrying young, on average age 15 and beginning to have children during puberty, when bone mass is increasing most rapidly, increases the loss of vitamin D and calcium (Jackson 1988; Chesney 2001). The high demands of multiple, closely spaced pregnancies would have had dire consequences for the vitamin D status of some Roman women.

The culturally prescribed dress of Roman women, which bore close resemblance to the burca of Muslim women, limited sun exposure as did sun avoidance due to issues of beauty and status. Women whitened their faces, suggesting they avoided the sun as lighter skin is thought to be more attractive and a symbol of higher status. The outdoor work of the laboring classes of women would have made their skin much darker while those of upper status who wished to convey a life of leisure may have avoided the sun and consequently limited their access to vitamin D.

Women did not fare well in patriarchal Roman society when it came to their share of food resources. The socio-cultural beliefs about what women should eat limited their access to vitamin D rich foods as suggested by their slightly lower isotopic levels. Due to socio-cultural factors, malnutrition and morbidity was likely more widespread and serious among women of childbearing age. The maternal death rate has been estimated at 25 per 1000 births (Jackson 1988) and inscriptional evidence suggests a higher death rate for women in their reproductive years (Joshel 1986). Unhealed childhood rickets and osteomalacia, the adult form of rickets, would have had serious implications

for the reproductive health of females as deformities of the pelvis results in obstructed labour and consequently increased perinatal morbidity and mortality. In the future, the adults in the Isola Sacra skeletal collection could be examined for the presence of osteomalacia to determine if maternal stores of vitamin D played a central role in the prevalence of rickets in the Isola Sacra subadults. Distortion of the pelvis may complicate pregnancy and the discovery of pelvic bone deformities may explain the high maternal death rate of Roman women. Lastly, examination of healed cases of rickets in late adolescence and adulthood may give a better idea of the prevalence of this disease in the Isola Sacra collection.

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APPENDIX A –
TABLE OF ALL ISOLA SACRA SUBADULT INDIVIDUALS

TABLE 1 - TABLE OF ALL ISOLA SACRA SUBADULT INDIVIDUALS (n=282)

#	SCR	Dental Age	Skeletal Age	Completeness	Notes
1.	**1**	1.5-2	1.5	Complete skeleton, no sacrum or sternum	Slight curvature of fibulae (broken), tibiae flaring at prox. ends, x-rays confirm
2.	**2**	7-8	8-9	Complete skeleton	No observable pathology
3.	**3**	0.75-1	1	Complete skeleton	Porosity of superior portion of orbits, x-rays=no curvature
4.	**4**		3-4	Portions of diaphyses of leg bones only	No observable pathology
5.	**6**	4	3-4	Most of skull & mandible, ribs, humerii, radii, ulnae, prox. femora only, diaphyses of fibula, tali & calcanei	Orbital porosity, fracture of 5th left rib, vertebrae porous
6.	**7**	6	7	Complete skeleton (bag has extra set of parietals)	No observable pathology
7.	**8**	0.5-1	0.5	Frag. Cranial bones, some ribs, diaphyses of humerii, prox. ulnae, diaphyses of femora, diaphyses of r. tibia only. Cannot observe flaring no epiphyses present	All bones very porous, orbital vault porosity but probably damaged postmortem, discoloration of m_1 & dm_2, poor mineralization of i_2, i_1, m_1, m^1, m^2, m_2, no x-rays available
8.	**9**	10-11	14	Complete skeleton	Very straight clavicle, slight LEH of upper incisors,
9.	**10**	3	2.5-3	Complete skeleton minus epiphyses	Coronoid processes of mandible laterally bent, head medially bent, ribs porous at sternal ends
10.	**11**	0.5-1		Fragmentary cranium, maxilla & a few ribs fragments	Cannot be evaluated
11.	**12**	0.75	0.6	Medial portion of mandible, lower pt of temps, mostly complete humerii, diaphyses of radii, femora & tibia, r. ilia	No observable pathology
12.	**13**	2	1.5	Fragmentary, lower portion of temporals, mandible minus rami, not enough long bones to observe, portion of right ilia	Cannot be evaluated
13.	**14**	2-3	2-3	Occipital, parietals, lower portion of maxilla, mandible minus rami, some ribs, frag. ilia, r. femur, diaphysis of l. femur, diaphyses of tibiae & humerii, prox. radii & ulnae	Abnormal curvature of the femora, genu valgum, orbital porosity,
14.	**22**	0.25-0.5	0.25-0.5	Frag. Cranial bones, diaphysis of r. humerus, pt of l. radius, femora, tibia & fibula mostly complete	No observable pathology
15.	**23**	4	3-4	Complete skeleton	No observable pathology
16.	**25**	6-7		Complete skeleton	No observable pathology
17.	**26**	2-2.5		Cranial bones minus face, rib frag., frags of diaphyses of humerii, femora & r. tibia	Cannot be evaluated
18.	**29**	15	14-15	Complete skeleton, minus l. femur & r. tibia	No observable pathology
19.	**31**	5	4-5	Complete skeleton	Orbital porosity, LEH & discoloration l & r i^1 i^2, LEH c_1 l & r m_1
20.	**33**	1.5		Fragmentary – most bones broken	Ribs show what appears to be more porosity at sternal ends

21.	**40**	5-6		Fragmentary	Orbital porosity
22.	**41**	0.75-1		Fragmentary	Cannot be evaluated
23.	**42**	4	4	Almost complete, missing r. femur & tibia	Vertebral porosity
24.	**43**	4	4	Complete skeleton, minus prox humerii	No observable pathology
25.	**45**	1	0.5-1	Lower facial bones, some ribs, r. humerus, prox r. rad & ulna, prox l. ulna only, femora, pts of tib & fib only, very brittle & porous	No observable pathology
26.	**46**	0.5-0.75		Fragmentary	Cannot be evaluated
27.	**47**	0.5	0-0.5	Complete skeleton, but no epiphyses of femora, tibiae & fibulae, sacrum or sternum	Cranial vault & orbital roof porosity, deformed mandibular ramus, abnormal curvature of humerii & thickening, thickening & flaring of distal radii
28.	**48**	0.5		Fragmentary	Cannot be evaluated
29.	**49**	6	5	Complete skeleton, missing r. tibia & l. ulna	Extreme enamel hypoplasia
30.	**54**	1-1.5	0.5-1	Some cranial & long bones	No observable pathology
31.	**56**	1.5-2		Fragmentary	Cannot be evaluated
32.	**61**	2-3	2	Cranial bones, diaphyses of long bones	No observable pathology
33.	**62**	0.5	0.25-0.5	Complete skeleton, except epiphyseal ends of all long bones, sacrum & sternum	Deformed mandibular ramus, fracture of 5th & 6th rib, bending of tibiae (no x-rays available), discoloration of incisor, deformed l. & r. m^2, vertebral porosity
34.	**63**	1.5-2		Fragmentary cranium only, long bone fragments, rib & vert fragments	Cannot be evaluated
35.	**65**	1.5-2		Fragmentary	Cannot be evaluated
36.	**66**	2-3	3	Complete skeleton	No observable pathology
37.	**72**	9	11	Almost complete, sternum and epiphyses of all long bones missing	Thickening of cranial vault, flared rib ends, LEH in canines, CTP's in pm
38.	**74**	0.25	0-0.5	Few long bone fragments, 1 tooth crown, 1 temporal frag.	Cannot be evaluated
39.	**81**	3-4	2.5-3	All bones broken, ends damaged, fragmentary	Cannot be evaluated
40.	**91**	0-0.25	Birth	Only missing some cranial bones	No observable pathology
41.	**92**	birth	fetal	Complete skeleton, frag bones of the arms	No observable pathology
42.	**93**	3-4		Skeleton almost complete but fragmentary, ends of long bones broken	No observable pathology
43.	**94**	4-5	4-5	Complete skeleton minus sacrum, sternum, epiphyses of arm bones & midface	Active pitting in orbits, some pitting over glabella, some pitting approx. 2cm. over postero-lateral are of parietals. Some medial curvature of prox. ulnae
44.	**95**	4-5		Proximal & distal ends of all long bones missing	No observable pathology
45.	**96**	5-6		Too fragmentary	Cannot be evaluated
46.	**100**	15	15-16	Complete skeleton	Microporosity along sagittal suture line, external periosteal overgrowth (thickening) of cranium, healing orbital roof porosity, radii & ulnae thickened, rib ends irregular & porous, rib depressions, microporosity of epiphyseal plates of tibiae &

					femora, depression & porosity on medial/frontal l. humeral head, depression distal ends of tibiae, plumping of ilia, compression & porosity of vertebrae, discoloration of canine, LEH lower left M_1. PM_1, and upper M^1, C, I^2
47.	**101**	1.5-2	1.5-2	Almost complete skeleton, cranium fragmentary	Slight (unhealed) pitting in r. orbit, whorled trabeculated new bone formation on sinuses of occipital
48.	**102**	1.5	1	Complete skeleton, minus sacrum, distal ulnae	Slight flaring of prox. tibia & distal femur. Orbital porosity
49.	**103**	10-11	7-8	Complete skeleton, diaphyses of fibula only	Healed orbital roof porosity, light LEH, tibia appears distorted on x-rays but not
50.	**107**		3-4	Very frag cranium & a few long bone fragments	Cannot be evaluated
51.	**108**	1-1.5	1.5	Complete skeleton	Fracture of left clavicle, right missing
52.	**109**	0.75	0.25-0.5	Leg bones missing, no vertebrae	No observable pathology
53.	**110**	2	1.5	Complete skeleton but no epiphyses & sternum	Slight porosity of glabella & orbital roof, bending of ulnae, thickening of radii, prox. & distal ends of humerii porotic, distal & prox. flaring of tibiae, flaring of femora distally, rib ends of irregular & porous, abnormal epiphyses, vertebrae porous
54.	**114**		12-13	Long bones, pelvis vert. ribs, scapula, small cranial frags.	Only proximally & medially curved rt. Ulna, Surface depressions/lesions of distal r. femur & prox. r. tibia
55.	**115**	6-7	8-9	Complete skeleton	No observable pathology
56.	**119**	6-7		Fragmentary	Cannot be evaluated
57.	**131**		5-6	Cranial frags, r. ilium, foot bones & epiphyses, r & l femur, tibiae, fibulae, ulnae, r. humerus, some ribs	No observable pathology
58.	**136**	0.75-1	0.5-1	No leg bones or ilia, humerii present, only r. ulna	Thalassemia, porosity of cranial vault, orbital roof, ribs metaphyses & vert.
59.	**137**	1-1.5	1	Cranial bones, some long bones missing – good condition	Micro pitting across ant/sup orbital roof of both sides
60.	**139**	1.5	1	Complete skeleton	Flared distal end of femur (no xrays available), internal & external cranial vault porosity, porosity of vertebrae
61.	**140**	0.75-1	0.5	Complete skeleton minus the lower sternum & sacrum	Orbital roof porosity, deformed mandibular ramus, porosity of prox. ends of humerii, bending of ulnae & flaring of distal ends, discoloration of upper & lower incisors, upper canine & l & r m^1
62.	**141**	1.5-2	1.5	Long bones, ribs, ilia, vertebra, some cranial frags	No observable pathology
63.	**149**	15	15-17	Complete except for diaphyses of long bones only	No observable pathology
64.	**153**	2-3	3	Complete skeleton, distal femora missing	Prox. tibia appears flared
65.	**154**	1.5-2	1	Most long bones present, cranium fragmentary	No observable pathology
66.	**162**	4-5	3-4	Complete skeleton	No x-rays, porous epiphyseal ends

67.	**164**	4	4-5	Missing some portions of long bones	No observable pathology
68.	**165**	7-8		2 cranial frags, tibial, femoral & fibular frags, mandible & a few ribs	No observable pathology
69.	**167**	3	3	Complete but all bone ends/edges damages	No observable pathology
70.	**168**	4-5	4-5	Complete skeleton in good condition	Light pitting & light plague-like formation in orbits
71.	**175**	8-9	7	Complete except missing some cranial bones frag., frontal missing	Bowing of tibiae, r. femur flaring distal ends & slight bowing (left has been cut), (no x-rays available), porosity of all epiphyses, porosity of vertebrae
72.	**176**	7-8	10-11	Complete skeleton minus epiphyses of right humerus	Pitting over glabellar region & palate. Proximal ulna appears bent. Woven bone/micro pitting on anterolateral surface of proximal shaft of r. tibia. Microfossae in cortex of femur. Thoracic vert. porous & anterior lesions in bodies.
73.	**182**	6-7	5-6	Relatively complete but very brittle, white	No observable pathology
74.	**187**	2-3	2	Complete skeleton, very good condition	No observable pathology
75.	**200**	6-7	7-8	Complete skeleton in fair condition	Strong prox. flaring tibiae on medial side
76.	**202**	1.5-2		Fragmentary cranium, long bones, pelves, ribs, some vert	No observable pathology BUT long bones all broken
77.	**204**	1.5-2	1.5-2	Missing prox. & distal ends of most long bones	Orbital porosity
78.	**206**	1-1.5		Fragmentary	Cannot be evaluated
79.	**209**	7-8	11	Complete skeleton	No observable pathology
80.	**214**		0-0.25	Very fragmentary bits of long bone & cranial frags	Cannot be evaluated
81.	**222**	0.75-1	0.5-1	Complete skeleton	No observable pathology
82.	**229**		0.5-1	Fragmentary cranium, 1 piece of rib, distal r. humerus, l. femur, r. tibia	Femoral shaft is very porous Tibial shaft is bent anteriorly.
83.	**230**		0-1	Very fragmentary cranial, long bone & ribs	Cannot be evaluated
84.	**235**	4-5	4-5	Complete skeleton, good condition	No observable pathology
85.	**244**	5-6	4-5	Complete skeleton, no mid-face, r. fibula, distal portion of left humerus only	Squared head, r. humerus deformed & bony spur, curvature of l. fibula & thickening, curvature of femora, curvature of tibiae & flaring, fibula, flaring of ribs, rib depressions & abnormal curvature, epiphyses porous, upper & lower m^2's severe defects, LEH lingual of m_1 l & r m^2 pit hypoplasia on buccal & lingual side, r. m_1 lingual & buccal pit hypoplasia, CTP's in molars, vertebral porosity
86.	**248**	1.5	1	Complete skeleton, good condition	Healed pitting in both orbits
87.	**255**	1-1.5		Fragmentary, long bones, cranial pieces, ribs	No observable pathology
88.	**273**	2-2.5		Fragmentary, long bones too frag, ribs too frag.	No observable pathology
89.	**274**	2.5-3		Fragmentary facial bones, cranium, frag. long bones, vert & ribs	No bowing of long bones
90.	**275**	1-1.5		Fragmentary cranium, long bones & some ribs	Cannot be evaluated
91.	**276**	1.5-2	1.5	Complete skeleton minus sternum	Squaring of head, orbital roof porosity, slight medial

					in bending of mandibular ramus, radii flared, humerii turned medially, porosity at rib ends in costal groove, vertebrae porous
92.	**278**	2.5-3	2.5-3	Prox. ends of all long bones missing	No observable pathology
93.	**311**		0-0.25	Only a fragmentary radius & humerus	Cannot be evaluated
94.	**315**	7-8	9-10	Complete skeleton, very good condition	Micro pitting on shaft surfaces of tibiae. Very small amount of cribrotic areas in sup/ant, orbits: just pitting
95.	**345**	1.5		Long bone frags only, some teeth	Cannot be evaluated
96.	**357**	11-12	12	Complete skeleton some matrix still present	No observable pathology
97.	**359**	1-1.5	1-1.5	Almost complete, excellent condition	No observable pathology
98.	**364**		5-6	r. femur, r. ilium, r. pubis. L. clavicle, metatarsals	Cannot be evaluated
99.	**367**		0-0.5	Lateral occipital, rib piece, prox l. humerus & l. scapula	Cannot be evaluated
100.	**372**		Birth	r. tibia, l. femur, r. radius, r. ulna	No observable pathology
101.	**373**		4-5	Radius, rib frag., vert body	Cannot be evaluated
102.	**375**		4-5	Only portion of l. femur & l humerus	Cannot be evaluated.
103.	**378**		12-15	Very fragmentary	Cannot be evaluated
104.	**381**		9-10	r. ilium, l. radius, tibial shafts, l. fib	No observable pathology
105.	**384**		5-6	Cranial frags, l. femur	Cannot be evaluated
106.	**385**	8-9		Teeth only	Cannot be evaluated
107.	**388**		1-1.5	r. ilium, r. humerus, r. ulna, r. clavicle – all in good condition	No observable pathology
108.	**392**	7-8	8-9	Skull & mandible, ilia, humerii, l. radius, femora, r. tibia	No observable pathology
109.	**400**	1-1.5	0.5-1	Complete skeleton in good condition except for vertebral bodies	No observable pathology
110.	**402**	5-6	5-6	Complete skeleton	Cortices of long bones thin
111.	**441**	1.5-2	1.5	Complete skeleton	No observable pathology
112.	**460**	2-3	1.5-2	Teeth, r. humerus, r. ulna, l. radius, femora, r. fibula, diaphysis of l. tibia	Appearance of bending of the l. radius, right missing
113.	**473**	0.75-1		Fragmentary	Cannot be evaluated
114.	**475**		2-3	Fragmentary	Cannot be evaluated
115.	**478**	0-0.25		Fragmentary	Cannot be evaluated
116.	**483**	1.5	1-1.5	Fragmentary	Cannot be evaluated
117.	**491**		0.0.5	Cranial frags, l. clavicle, humeri, r. scapulae, few vert frags, 4 rib frags (no sheet)	No observable pathology
118.	**492**		6-7	1 cranial frag, 1 rib frag., some fragments of long bones: fem, fib, tib, humeral shaft	Cannot be evaluated
119.	**496**		2.5-3	r. humerus & radius, tibiae	No observable pathology
120.	**506**		3-4	Femoral frags.	Cannot be evaluated
121.	**511**	0.5-0.75	0.5	Missing area around orbits, left radius & ulna,	Tibiae bent anteriorly, porosity distal ends of tibiae, l.

				scapulae, sacrum, sternum, left fibula. mid portion of r. humerus only, prox. r. ulna only	tibia show large area of porotic woven bone on disto-lateral shaft, femora show flaring of distal metaphyses w/ flattening of the ends (surfaces have very little relief), femoral shaft cortices show extreme porosity, all but 3 sternal rib ends flared & porous.
122.	**512**	15-21	15-16	Complete skeleton – cranium missing	No observable pathology
123.	**513**	0.5-0.75	0.25-0.5	Long bones broken at ends, cranial bones quite fragmentary	No observable pathology
124.	**514**	4-4.5	2.5-3	Complete except for lower leg bones	Irregular epiphyses, orbital porosity
125.	**516**	0-0.25	Birth	Fragmentary	Cannot be evaluated
126.	**517**		14-16	Complete but no skull	No observable pathology
127.	**518**	2-3		Some cranial bones & mandible, fragmental long bones	No observable pathology
128.	**520**	1-1.5		Too fragmentary	Cannot be evaluated
129.	**522**	6-7		Complete skeleton	No observable pathology
130.	**523**	5-6	5-6	Complete skeleton minus some facial bones, l. humerus, sternum, sacrum, all epiphyses. Left femur cut for sampling	R. femur bent posteriorly & twisted compression of vertebral bodies, porous diaphyseal ends, orbital porosity, LEH @ CEJ incisors & molars
131.	**529**	2-3	2-3	Cranial fragments, some ribs, vertebrae, midshaft of left humerus, ulna & radius, sternum, left ilium frags., frags of left tibia, r. tibiae minus epiphyses, midshaft of tibiae & fibulae	Lots of micro-pitting (woven bone) on medial surfaces of tibiae. Cortical bone of long bones is thin but no other observable pathology
132.	**530**	10-11	8-9	Skull, r. femur, distal ends of left femur, r. tibia, diaphysis of left tibia, prox. & distal fibulae, frag humeri, radii & ulnae	Subtle bending of tibia & femora. Compression of vertebral bodies
133.	**531**	6-7	6-7	Complete skeleton	No observable pathology
134.	**532**	0.5-1	0.5-1	Complete skeleton minus some facial bones, epiphyses of long bones, l. tibia, sacrum & sternum	Cortices of long bones very porous, external surfaces micro pitting i.e. woven bone.
135.	**533**	1.5-2		Fragmentary cranium, mandible & a few long bone frags	No observable pathology
136.	**534**	15	15-17	Fragmentary	Cannot be evaluated
137.	**536**	4	5-6	Complete skeleton in good condition	No observable pathology
138.	**541**	3-4	2.5-3	Cranium & mandible, diaphyses of long bones only	Orbital porosity
139.	**542**	1-1.5	1-1.5	Complete skeleton minus long bones epiphyses, some portions of frontal, temporal and parietal bones.	Slight flaring of proximal & distal tibiae, orbital porosity
140.	**543**	0.5	0.25-0.5	Fragmentary arm & leg bones & some cranial frags.	Thalassemia, very porous, no x-rays
141.	**546**	5	5-6	Mandible, diaphyses of some long bones	No observable pathology
142.	**547**		6-7	Fragmentary	Cannot be evaluated
143.	**548**		2.5-3	Occipital , rib frags, l. radius, distal ends of femora,	No observable pathology

				diaphyses of l. tibia & fibula	
144.	**552**	4-5	4-5	Some cranial frags, observable portions of most long bones	No observable pathology
145.	**555**		Birth	Fragmentary	Cannot be evaluated
146.	**560**		6-7	Fragmentary	Cannot be evaluated
147.	**564**	1.5-2	1.5-2	Complete skeleton	Flaring of rib end (only one present)
148.	**580**	6-7	7-8	Complete skeleton	Thickening of l. fibula and bending
149.	**581**		6-7	Fragmentary	Cannot be evaluated
150.	**582**		birth	Fragmentary	Cannot be evaluated
151.	**583**	8-9		Fragmentary	Cannot be evaluated
152.	**584**		6-7	Fragmentary	Cannot be evaluated
153.	**586**		1-1.5	Only r. femur and r. ulna present	Downward turning of femoral neck
154.	**587**		2-4	Fragmentary, distal portion of humeri, diaphysis of r. femur	Cannot be evaluated
155.	**588**		6-7	r. humerus, ulna & radius, diaphyses of femora, tibiae	Right ulna bent, left missing
156.	**594**		1.5-2	r. humerus, ulnae, radii, r. femur, l. tibia, l. ilia	No observable pathology
157.	**595**	3	3-4	l. maxilla, l & r radius complete, r. ulna, prox. L. ulna, distal l. humerus, tibial shaft	No observable pathology
158.	**596**		5-6	Intact r. ulna, l. radius, distal humeri, l. fibula, l. tibia	L. tibia cut longitudinally – show a number of Harris lines
159.	**597**		6-7	Intact l. & r. humeri, r. radius, r. ulna, l. fibula & tibia	R. tibia cut longitudinally – show a number of Harris lines
160.	**598**		6-7	Distal end of r. humeral metaphysis, r. femur, intact tibiae, fibulae	R. tibia cut longitudinally – show a number of Harris lines
161.	**599**	3-4		Cranial fragments only	Thalassemia, thickening & porosity of cranial vault & orbital roof, no x-rays available
162.	**610**	7-8	6-8	Very fragmentary, l. humerus, r. ulna, r. radius, r. femur, r. tibia, 1 scapula, mandible	Curvature of the femoral shaft but too fragmentary to be evaluated properly
163.	**612**		2-2.5	Tibiae & left humerus only	Some curvature to humerus & tibiae. Flaring of proximal tibiae
164.	**613**		5-6	Rt. Radius, ulna & distal humerus	Cannot be evaluated
165.	**614**		0-0.25	l. humerus, r. radius only	Cannot be evaluated
166.	**615**		15-16	r. & l. pelvic frags, r. humerus, 2 vert, 2 epiphyses	Cannot be evaluated
167.	**619**		0.5-1	Fragmentary	Cannot be evaluated
168.	**620**		1-1.5	Fragmentary	Cannot be evaluated
169.	**621**		2.5-3	l. humerus, radii, r. ulna, r. femur prox. l. tibia & fibula, r. fibula	No observable pathology
170.	**622**		12-14	r. diaphysis of humerus, l. femur, pt of diaphyseal shaft of l. femur, r. tibia, fibulae	No observable pathology
171.	**623**		13-14	Fragmentary	Cannot be evaluated
172.	**624**	5-6	5	Fragmentary	Cannot be evaluated

173.	**625**		1-1.5	Distal pt of humerus, l. ilia, l. femur, distal pt of r. femur, distal pt of l. tibia	No observable pathology
174.	**626**		14-17	Fragmentary	Cannot be evaluated
175.	**627**		0-0.5	r. femur & l. tibia	Cannot be evaluated
176.	**628**		0.5-1	Fragmentary	Cannot be evaluated
177.	**629**		0.25-0.5	l. femur & r. fibula	Cannot be evaluated
178.	**630**		2-2.5	r. femur & l. tibia	No observable pathology
179.	**631**		2.5-3	l. ulna, radii	No observable pathology
180.	**632**		8	r. femur, fibula, l. humerus, r. radius, ulnar fragment & epiphysis	No observable pathology
181.	**633**			Humeri, l. radial frag., fibula frag, femoral epiphyses, sternal & scapulae frags.	No observable pathology
182.	**634**		9-10	Tibial shaft, distal femoral diaphysis, 1st metatarsal	No observable pathology
183.	**635**		0-0.5	r. humerus, prox. R. femur	No observable pathology
184.	**636**		1.5	l. clavicle, r. radius	Radius shows strong lateral curvature/also some thickening of proximal & middle shaft
185.	**637**		3	Radii., r. clavicle	No observable pathology
186.	**639**	4		r. humerus, frag of r. femur, tibia, r. mandible fragments includes condyle	No observable pathology
187.	**640**	4-5	5-6	R. mandible no condyle, r. humerus, prox. R. ulna, femoral & tibial fragments	No observable pathology
188.	**641**		8-9	Humeri, femora fragments	No observable pathology
189.	**642**		7-9	2 femoral shafts, 1 humeral shaft	No observable pathology
190.	**644**	1-1.5		Complete mandible, left femoral diaphysis	Inward curvature of the mandibular condyles
191.	**646**		13-14	l. humerus, l. radius, r. ulna, r & l. femur frag, r. scapula	No observable pathology
192.	**647**	0.75-1	0.5-1	Fragmentary	Cannot be evaluated
193.	**648**	10-11	8-10	2 fibula, 1 tibia, partial maxillae, some vert	No observable pathology
194.	**649**		1-1.5	Long bones, l. scapula, l. pelvis, l. frontal, some rib frags.	No observable pathology
195.	**650**		7-8	Tibia, fibula, calcanei, l. humerus	No observable pathology
196.	**651**		13-14	Fragmentary long bones, talus, calcaneous, scapula	No observable pathology
197.	**653**		0-0.5	r. tibia & fibula, r. femur, l. humerus	Very slight bending of tibia & fibula, apparent on x-rays
198.	**654**		Birth	R. femur, r & l. tibiae, l. & R. fibulae, need skull to evaluate rickets in fetal	Cannot be evaluated
199.	**655**		0.5-1	l. ulna, l. humerus	Cannot be evaluated
200.	**656**		4-5	R & l. ilium, l. ischium, r & l humerus, l. radius, distal femur, cranial fragments	No observable pathology
201.	**657**		6-7	Very fragmentary, humeri, fibulae	No observable pathology
202.	**658**	8-9		Partial mandible, no condyles	Cannot be evaluated

203.	**659**		3-4	Fragments of r. humerus, tibia, femur, l. ilium	No observable pathology
204.	**660**	7	7-8	Elements present are right side of mandible & maxilla, humeri minus humeral heads, right radius, distal right ulna, distal left ulna, three-fourths of right femur, distal portion of left femur, left tibia & fibula, proximal right tibia & distal right fibula.	Slight medial bending of r. radius & flaring of distal end, twisting of humerii, r. femur abnormal curvature, l. tibia slight bending, LEH on l^1
205.	**661**	1.5-2	2	Mandible & maxilla, humeri, r. ilia, r. femur, tibiae	Flaring apparent at prox. end on r. tibia, left broken off at distal end
206.	**671**		Birth	l. humerus, prox. r. ulna, tibial & femoral fragments, 1 rib fragment	Cannot be evaluated
207.	**672**	3-4	2.5-3	Humerii, tibae, l. radius	No observable pathology
208.	**675**		0.25-0.5	r. ulna & radius, humerii frags., 4 cranial fragments	No observable pathology
209.	**681**		15-16	Quite fragmentary, 1 femur, few vert, 1 calcaneous, few metatarsals	No observable pathology
210.	**683**	12-15	12-14	Left mandible and maxilla, clavicles, scapulae, humeri, radii, proximal left ulna, ilia, right femur, distal left femur and a couple vertebrae.	Lateral curvature of rt. radius (l. broken) Rib frags. and vertebrae porous
211.	**684**	7	6	r. femur, face & r. cranium	Some medial & lateral cribrotic pitting in superior orbits – some healing of this.
212.	**687**	7-8	7-8	Mandible, humeri, radii, ulnae & ilia. Ends damaged on all.	No observable pathology
213.	**696**		0-0.25	Fragmentary	Cannot be evaluated
214.	**700**		0.25-0.5	Rt. Femur & 3 cranial fragments	Cannot be evaluated
215.	**701**		5-6	1 femur shaft, 2 tibial shafts, 1 fibula, 2 humeri, 1 ulna, 1 scapula & clavicle	No observable pathology
216.	**731**		5-6	Fragmentary	Cannot be evaluated
217.	**733**		Birth	Fragmentary, need cranial bones to evaluate	Cannot be evaluated
218.	**734**		0-0.5	Fragmentary, need cranial bones to evaluate	Cannot be evaluated
219.	**735**		0-0.5	Fragmentary, need cranial bones to evaluate	Cannot be evaluated
220.	**736**		5-6	Long bones on right side of body only	No observable pathology
221.	**737**	5		Left side of cranium, some ribs, r. ilium	Cannot be evaluated
222.	**738**		4-5	Fragmentary	Cannot be evaluated
223.	**739**		Birth	Fragmentary, need cranial bones to evaluate	Cannot be evaluated
224.	**740**		12	Fragmentary	Cannot be evaluated
225.	**741**		5-6	r. ilium & femur, l. tibia, distal pt of r. fibula	No observable pathology
226.	**742**	15-16	15-16	Skull and clavicles	Thickening of cranium, no clear table
227.	**743**		15-16	Ilia, pt of sacrum, most portions of long bones	No observable pathology
228.	**744**	7-8	6-8	Fragmentary	Cannot be evaluated
229.	**745**		12-13	Fragmentary	Cannot be evaluated
230.	**746**		0-0.5	Fragmentary	Cannot be evaluated
231.	**747**		0.25-0.5	Fragmentary, need cranial bones to evaluate	Cannot be evaluated
232.	**748**		4-5	r. humeri, r. prox. radius, 1 rib, diaphysis of r. femur	No observable pathology

233.	**749**		6-7	r. ilium, manubrium tibia/fibula, l. ulna, sacral #1, axis, L1, r. calcaneous, 2 metatarsals. these items are in good condition	No observable pathology
234.	**750**	8-9		Fragmentary, Cranial pieces, demi-mand, diaphyseal pieces	Cannot be evaluated
235.	**751**		Birth	Fragmentary, need cranial bones to evaluate	Cannot be evaluated
236.	**752**		1-1.5	l. ilium, l. pelvis, humerii, 2 rib frags.	No observable pathology
237.	**753**		15-17	r. femur, l. humerus, max. & mand.	No observable pathology
238.	**754**		8-9	r. femur, r. tibia, prox. l. humerus, 5th lumbar,	Metaphyseal ends of tibia & possibly femur look depressed
239.	**755**	No age recorded	Large individual	2 femora, 1 tibial shaft, rt calcaneous, 1 first metatarsal	No observable pathology
240.	**756**		1-1.5	Parietals, occipital, r. femur shaft , maxillae	Cannot be evaluated
241.	**757**	1.5	1.5	Parietals, frontals, r. mand & max., r. femur, r. ischium, rib frag.	Very tiny pitting in r. orbit.
242.	**758**		0-0.25	l. prox. femur, l. radius, r. ulna, r. humerus & rib frag.	No observable pathology
243.	**759**		5-6	l. humerus &l. radius, 4 vert, r. ilium, rib frags, prox. tibia	No observable pathology
244.	**760**		0-0.25	r. humerus, 2 cranial frags	Cannot be evaluated
245.	**761**	5-6		Very fragmentary cranial & long bone frags	Cannot be evaluated
246.	**762**		Birth	Fragmentary, need cranial bones to evaluate	Cannot be evaluated
247.	**763**		0-0.5	Fragmentary, need cranial bones to evaluate	Cannot be evaluated
248.	**764**	12-15	14-15	Maxilla, scapulae, ribs, humeri, r. tibia & fibula	No observable pathology
249.	**765**		Perinatal	Fragmentary, need cranial bones to evaluate	Cannot be evaluated
250.	**766**		Birth	Fragmentary, need cranial bones to evaluate	Cannot be evaluated
251.	**767**	1.5-2	1.5-2	Some teeth, cranial fragments, diaphyses of femora, tibiae	No observable pathology
252.	**768**		Perinatal	Fragmentary, need cranial bones to evaluate	Cannot be evaluated
253.	**769**		2-3	Fragmentary	Cannot be evaluated
254.	**770**		5-6	r. femur, diaphyses of tibiae, fragments of fibulae	No observable pathology
255.	**772**	2-3		Fragmentary	Cannot be evaluated
256.	**773**	0.75	0.5-1	Mandible minus r. ramus, clavicles, r. scapulae, r. humerus, distal pt of l. humerus, l. ilium, r. radius, prox. pt of r. ulna, midshaft of r. femur, prox pt of r. fibula	Posterior/medial bending of l. mandibular ramus (r. missing)
257.	**774**		1.5	r. scapula, r. prox. humerus, l. ilium, l. femur, pts of tibiae	No observable pathology
258.	**775**		0-0.5	Fragmentary, need cranial bones to evaluate	Cannot be evaluated
259.	**776**		1-1.5	Fragmentary	Cannot be evaluated
260.	**777**		0.25-0.5	Femora & tibiae	No observable pathology
261.	**778**		0.5-1	Some cranial bones, l. humerus & shaft of r. humerus, missing distal end of r. ulna, shaft of r.	No observable pathology

				femur, r. tibia, shaft of fibulae	
262.	**779**		4-5	Fragmentary cracked & warped	Cannot be evaluated
263.	**780**	1-1.5	1-1.5	Occipital, scapulae, humeri, ulnae, l. radius, r. ilia, femora, tibiae, fibulae & fragmentary vertebrae.	Bending & flaring of fibulae
264.	**781**	4-5	4-5	Portions of most long bones present	No observable pathology
265.	**782**		Fetal	Fragmentary, need cranial bones to evaluate	Cannot be evaluated
266.	**783**	9-10	10-12	Portions of mandible and maxilla, left humerus, radius & ulna, head of l. femur pox. End of l. fibula	No observable pathology
267.	**785**	4-5	4-5	fragmentary	Cannot be evaluated
268.	**786**		10-15	Quite damaged, frag long bones, epiphyses, r. scapulae	Cannot be evaluated
269.	**788**		2-4	R & l humeral shafts, r & l fibula frags, 1 rib frag	No observable pathology
270.	**791**	4	4	Cranial frags, long bones, rib frags, pelvis mandible	No observable pathology
271.	**795**		0-1	r. scapula, r. femoral shaft	Cannot be evaluated
272.	**798**		Perinatal	Fragmentary, need cranial bones to evaluate	Cannot be evaluated
273.	**799**		0-0.25	1 rt. Radius shaft	Cannot be evaluated
274.	**800**		Fetal	Fragmentary, need cranial bones to evaluate	Cannot be evaluated
275.	**803**	6-9		Complete skeleton – long bone ends broken	No observable pathology
276.	**810**	13-14		Complete skeleton in good condition	Periostitis on lateral tibiae shafts Harris lines visible in tibia. Transverse fossa lesions on thoracic vert bodies.
277.	**811**	2-3		Humerii, femora, radius & ulna shafts, mandible, maxilla	Pitting –open in antero-medial part of l. orbit
278.	**812**	13-17		Complete skeleton in good condition	Slight pitting in antero-medial portions of orbits. Transverse slit-like fossae/lesions on T4-T8 anterior bodies of vert
279.	**815**	7-9		Complete skeleton but brittle & fragmentary, no bone ends preserved	No observable pathology
280.	**818**	9-10		Cranial frags, l. humerus, r. mandibular fragment	Cannot be evaluated
281.	**824**	16-19		Very fragmentary	Cannot be evaluated
282.	**839**	15-18		Complete skeleton, good condition, most epiphyses fused	No observable pathology

APPENDIX B –
ISOLA SACRA SUBADULT INDIVIDUALS USED IN ANALYSIS

TABLE 2 - ISOLA SACRA SUBADULT INDIVIDUALS INCLUDED IN ANALYSIS (n=182)

#	SCR	Zone	Burial Type	Age category	Completeness	Notes
1.	**1**	14B	amphora	1-2	Complete skeleton, no sacrum or sternum	Slight curvature of fibulae (broken), tibiae flaring at prox. ends, x-rays confirm
2.	**2**	24	inhumation	7-8	Complete skeleton	No observable pathology
3.	**3**	14B	amphora	0.5-1	Complete skeleton	Porosity of superior portion of orbits, x-rays=no curvature
4.	**4**	24	amphora	3-4	Portions of diaphyses of leg bones only	No observable pathology
5.	**6**	24	inhumation	3-4	Most of skull & mandible, ribs, humerii, radii, ulnae, prox. femora only, diaphyses of fibula, tali & calcanei	Orbital porosity, fracture of 5th left rib, vertebrae porous
6.	**7**	14B	cappuccina	6-7	Complete skeleton (bag has extra set of parietals)	No observable pathology
7.	**8**	24	amphora	0.5-1	Frag. Cranial bones, some ribs, diaphyses of humerii, prox. ulnae, diaphyses of femora, diaphyses of r. tibia only. Cannot observe flaring no epiphyses present	All bones very porous, orbital vault porosity but probably damaged postmortem, discoloration of m_1 & dm_2, poor mineralization of i_2, i_1, m_1, m^1, m^2, m_2, no x-rays available
8.	**9**	14H	amphora	10-11	Complete skeleton	Very straight clavicle, slight LEH of upper incisors,
9.	**10**	21	cassa	2-3	Complete skeleton minus epiphyses	Coronoid processes of mandible laterally bent, head medially bent, ribs porous at sternal ends
10.	**12**	24	cassa	0.5-1	Medial portion of mandible, lower pt of temps, mostly complete humerii, diaphyses of radii, femora & tibia, r. ilia	No observable pathology
11.	**14**	23	inhumation	2-3	Occipital, parietals, lower portion of maxilla, mandible minus rami, some ribs, frag. Ilia, r. femur, diaphysis of l. femur, diaphyses of tibiae & humerii, prox. radii & ulnae	Abnormal curvature of the femora, genu valgum, orbital porosity,
12.	**22**	25	amphora	0-0.5	Frag. Cranial bones, diaph. Of r. humerus, pt of l. radius, femora, tibia & fibula mostly complete	No observable pathology
13.	**23**	24	Inhumation	3-4	Complete skeleton	No observable pathology
14.	**25**	25	amphora	6-7	Complete skeleton	No observable pathology
15.	**29**	22	pit	14-15+	Complete skeleton, minus l. femur & r. tibia	No observable pathology
16.	**31**	24	inhumation	4-5	Complete skeleton	Orbital porosity, LEH & discoloration l & r i^1 i^2, LEH c_1, l & r m_1
17.	**33**	22	inhumation	1-2	Fragmentary – most bones broken	Ribs show what appears to be more porosity at sternal ends
18.	**40**	22	sarcophagus	5-6	Fragmentary	Orbital porosity
19.	**42**	25	inhumation	4-5	Almost complete, missing r. femur & tibia	Vertebral porosity
20.	**43**	25	amphora	4-5	Complete skeleton, minus prox humerii	No observable pathology
21.	**45**	2	amphora	0.5-1	Lower facial bones, some ribs, r. humerus,	No observable pathology

					prox r. rad & ulna, prox l. ulna only, femora, pts of tib & fib only, very brittle & porous	
22.	**47**	23A	amphora	0-0.5	Complete skeleton, but no epiphyses of femora, tibiae & fibulae, sacrum or sternum	Cranial vault & orbital roof porosity, deformed mandibular ramus, abnormal curvature of humerii & thickening, thickening & flaring of distal radii
23.	**49**	22	sarcophagus	5-6	Complete skeleton, missing r. tibia & l. ulna	Extreme enamel hypoplasia
24.	**54**	2	cappucina	1-2	Some cranial & long bones	No observable pathology
25.	**61**	19	sand	2-3	Cranial bones, diaphyses of long bones	No observable pathology
26.	**62**	14A	amphora	0-0.5	Complete skeleton, except epiphyseal ends of all long bones, sacrum & sternum	Deformed mandibular ramus, fracture of 5th & 6th rib, bending of tibiae (no x-rays available), discoloration of incisor, deformed l. & r. m^2, vertebral porosity
27.	**66**	14B	sarcophagus	2-3	Complete skeleton	No observable pathology
28.	**72**	12A	inhumation	9-10	Almost complete, sternum and epiphyses of all long bones missing	Thickening of cranial vault, flared ends of rib, LEH in canines, CTP's in pm
29.	**91**	22	amphora	0-0.5	Only missing some cranial bones	No observable pathology
30.	**92**	22	amphora	0-0.5	Complete skeleton, frag bones of the arms	No observable pathology
31.	**93**	26	inhumation	3-4	Skeleton almost complete but fragmentary, ends of long bones broken	No observable pathology
32.	**94**	12A	amphora	4-5	Complete skeleton minus sacrum, sternum, epiphyses of arm bones & midface	Active pitting in orbits, some pitting over glabella, some pitting approx. 2cm. over postero-lateral area of parietals. Some medial curvature of prox. ulnae
33.	**95**	14B	inhumation	4-5	Proximal & distal ends of all long bones missing	No observable pathology
34.	**100**	21	sand	14-15+	Complete skeleton	Microporosity along sagittal suture line, external periosteal overgrowth (thickening) of cranium, healing orbital roof porosity, radii & ulnae thickened, rib ends irregular & porous, rib depressions, microporosity of epiphyseal plates of tibiae & femora, depression & porosity on medial/frontal l. humeral head, depression distal ends of tibiae, plumping of ilia, compression & porosity of vertebrae, discoloration of canine, LEH lower left M_1, PM_1, and upper M^1, C, I^2
35.	**101**	21	sand	1-2	Almost complete skeleton, cranium fragmentary	Slight (unhealed) pitting in r. orbit, whorled trabeculated new bone formation on sinuses of occipital
36.	**102**	22	amphora	1-2	Complete skeleton, minus sacrum, distal ulnae	Slight flaring of prox. tibia & distal femur. Orbital porosity

37.	**103**	21	amphora	9-10	Complete skeleton, diaphyses of fibula only	Healed orbital roof porosity, light LEH
38.	**108**	14B	amphora	1-2	Complete skeleton	Fracture of left clavicle, right missing
39.	**109**	20	sand	0.5-1	Leg bones missing, no vertebrae	No observable pathology
40.	**110**	22	pit	1-2	Complete skeleton but no epiphyses & sternum	Slight porosity of glabella & orbital roof, bending of ulnae, thickening of radii, prox. & distal ends of humerii porotic, distal & prox. flaring of tibiae, flaring of femora distally, ends of ribs irregular & porous, abnormal epiphyses, vertebrae porous
41.	**114**	10	sand	12-13	Long bones, pelvis vert. ribs, scapula, small cranial frags.	Only proximally & medially curved rt. Ulna, Surface depressions/lesions of distal r. femur & prox. r. tibia
42.	**115**	20	sand	7-8	Complete skeleton	No observable pathology
43.	**131**	21	amphora	5-6	Cranial frags, r. ilium, foot bones & epiphyses, r & l femur, tibiae, fibulae, ulnae, r. humerus, some ribs	No observable pathology
44.	**136**	10	amphora	0.5-1	No leg bones or ilia, humerii present, only r. ulna	Thalassemia, porosity of cranial vault, orbital roof, ribs metaphyses & vert.
45.	**137**	16	cappucina	1-2	Cranial bones, some long bones missing – good condition	Micro pitting across ant/sup orbital roof of both sides
46.	**139**	10	amphora	1-2	Complete skeleton	Flared distal end of femur (no xrays available), internal & external cranial vault porosity, porosity of vertebrae
47.	**140**	14A	amphora	0.5-1	Complete skeleton minus lower sternum & sacrum	Orbital roof porosity, deformed mandibular ramus, porosity of prox. ends of humerii, bending of ulnae & flaring of distal ends, discoloration of upper & lower incisors, upper canine & l & r m^1
48.	**141**	20	amphora	1-2	Long bones, ribs, ilia, vertebra, some cranial frags	No observable pathology
49.	**149**	16	sand	14-15+	Complete except for diaphyses of long bones only	No observable pathology
50.	**153**	16	sand	2-3	Complete skeleton, distal femora missing	Prox. tibia appears flared
51.	**154**	18	amphora	1-2	Most long bones present, cranium fragmentary	No observable pathology
52.	**162**	10	amphora	4-5	Complete skeleton	No x-rays, porous epiphyseal ends
53.	**164**	13	amphora	4-5	Missing some portions of long bones	No observable pathology
54.	**165**	19	amphora	7-8	2 cranial frags, tibial, femoral & fibular frags, mandible & a few ribs	No observable pathology
55.	**167**	16	sand	3-4	Complete but all bone ends/edges damages	No observable pathology
56.	**168**	14	amphora	4-5	Complete skeleton in good condition	Light pitting & light plague-like formation in orbits
57.	**175**	10	amphora	8-9	Complete except missing some cranial	Bowing of tibiae, r. femur flaring distal ends &

					bones frag., frontal missing	slight bowing (left has been cut), (no x-rays available), porosity of all epiphyses, porosity of vertebrae
58.	**176**	19	inhumation	8-9	Complete skeleton minus epiphyses of right humerus	Pitting over glabellar region & palate. Proximal ulna appears bent. Woven bone/micro pitting on anterolateral surface of proximal shaft of r. tibia. Microfossae in cortex of femur. Thoracic vert. porous & anterior lesions in bodies.
59.	**182**	n/a	tomb 5E	6-7	Relatively complete but very brittle, white	No observable pathology
60.	**187**	16	amphora	2-3	Complete skeleton, very good condition	No observable pathology
61.	**200**	12A	amphora	6-7	Complete skeleton in fair condition	Strong prox. flaring tibiae on medial side
62.	**202**	15	amphora	1-2	Fragmentary cranium, long bones, pelves, ribs, some vert	No observable pathology BUT long bones all broken
63.	**204**	16	cappucina	1-2	Missing prox. & distal ends of most long bones	Orbital porosity
64.	**209**	16	sand	8-9	Complete skeleton	No observable pathology
65.	**222**	10	amphora	0.5-1	Complete skeleton	No observable pathology
66.	**229**	n/a	tomb 81 forma 2	0.5-1	Fragmentary cranium, 1 piece of rib, distal r. humerus, l. femur, r. tibia	Femoral shaft is very porous. Tibial shaft is bent anteriorly
67.	**235**	12	cappucina	4-5	Complete skeleton, good condition	No observable pathology
68.	**244**	13	pit	5-6	Complete skeleton, no mid-face, r. fibula, distal portion of left humerus only	Squared head, r. humerus deformed & bony spur, curvature of l. fibula & thickening, curvature of femora, curvature of tibiae & flaring, fibula, flaring of ribs, rib depressions & abnormal curvature, epiphyses porous, upper & lower m^2's severe defects, LEH lingual of m_1, l & r m^2 pit hypoplasia on buccal & lingual side, r. m_1 lingual & buccal pit hypoplasia, CTP's in molars, vertebral porosity
69.	**248**	12	amphora	1-2	Complete skeleton, good condition	Healed pitting in both orbits
70.	**255**	26	pit	1-2	Fragmentary, long bones, cranial pieces, ribs	No observable pathology
71.	**273**	10	sarcophagus	2-3	Fragmentary, long bones too frag, ribs too frag.	No observable pathology
72.	**274**	13	amphora	2-3	Fragmentary facial bones, cranium, frag. long bones, vert & ribs	No observable pathology
73.	**276**	15	amphora	1-2	Complete skeleton minus sternum	Squaring of head, orbital roof porosity, slight medial in bending of mandibular ramus, radii flared, humerii turned medially, porosity at rib ends in costal groove, vertebrae porous
74.	**278**	12A	inhumation	2-3	Prox. ends of all long bones missing	No observable pathology
75.	**315**	14B	cappucina	8-9	Complete skeleton, very good condition	Micro pitting on shaft surfaces of tibiae. Very small amount of cribrotic areas in sup/ant, orbits:

						just pitting
76.	**357**	?	colombario forma 1	11-12	Complete skeleton some matrix still present	No observable pathology
77.	**359**	10	amphora	1-2	Almost complete, excellent condition	No observable pathology
78.	**372**	n/a	tomb EE forma 2	0-0.5	r. tibia, l. femur, r. radius, r. ulna	No observable pathology
79.	**381**	5	found in rubble	9-10	r. ilium, l. radius, tibial shafts, l. fib	No observable pathology
80.	**388**	n/a	tomb 47b	1-2	r. ilium, r. humerus, r. ulna, r. clavicle – all in good condition	No observable pathology
81.	**392**	n/a	tomb 72	7-8	Skull & mandible, ilia, humerii, l. radius, femora, r. tibia	No observable pathology
82.	**400**	25	inhumation	1-2	Complete skeleton in good condition except for vertebral bodies	No observable pathology
83.	**402**	12A	inhumation	5-6	Complete skeleton	Cortices of long bones thin
84.	**441**	n/a	tomb 2E forma E	1-2	Complete skeleton	No observable pathology
85.	**460**	n/a	Tomb E5 forma 2	2-3	Teeth, r. humerus, r. ulna, l. radius, femora, r. fibula, diaphysis of l. tibia	Appearance of bending of the l. radius, right missing
86.	**491**	7C	amphora	0-0.5	Cranial frags, l. clavicle, humeri, r. scapulae, few vert frags, 4 rib frags (no sheet)	No observable pathology
87.	**496**	n/a	tomb 2E	2-3	r. humerus & radius, tibiae	No observable pathology
88.	**511**	1	amphora	0.5-1	Missing area around orbits, left radius & ulna, scapulae, sacrum, sternum, left fibula. mid portion of r. humerus only, prox. r. ulna only	Tibiae bent anteriorly, porosity distal ends of tibiae, l. tibia show large area of porotic woven bone on disto-lateral shaft, femora show flaring of distal metaphyses w/ flattening of the ends (surfaces have very little relief), femoral shaft cortices show extreme porosity, all but 3 sternal rib ends flared & porous.
89.	**512**	n/a	tomb 2E forma H	14-15+	Complete skeleton – cranium missing	No observable pathology
90.	**513**	26	amphora	0.5-1	Long bones broken at ends, cranial bones quite fragmentary	No observable pathology
91.	**514**	25	pit	3-4	Complete except for lower leg bones	Irregular epiphyses, orbital porosity
92.	**517**	5	colombario	14-15+	Complete but no skull	No observable pathology
93.	**518**	25	amphora	2-3	Some cranial bones & mandible, fragmental long bones	No observable pathology
94.	**522**	12A	sarcophagus	6-7	Complete skeleton	No observable pathology
95.	**523**	16	cappucina	5-6	Complete skeleton minus some facial bones, l. humerus, sternum, sacrum, all epiphyses. Left femur cut for sampling	R. femur bent posteriorly & twisted compression of vertebral bodies, porous diaphyseal ends, orbital porosity, LEH @ CEJ incisors & molars
96.	**529**	1	inhumation	2-3	Cranial fragments, some ribs, vertebrae,	Lots of micro-pitting (woven bone) on medial

					midshaft of left humerus, ulna & radius, sternum, left ilium frags., frags of left tibia, r. tibiae minus epiphyses, midshaft of tibiae & fibulae	surfaces of tibiae. Cortical bone of long bones is thin
97.	**530**	5	colombario G forma 3	9-10	Skull, r. femur, distal ends of left femur, r. tibia, diaphysis of left tibia, prox. & distal fibulae, frag humeri, radii & ulnae	Subtle bending of tibia & femora. Compression of vertebral bodies
98.	**531**	12A	amphora	6-7	Complete skeleton	No observable pathology
99.	**532**	25	inhumation	0.5-1	Complete skeleton minus some facial bones, epiphyses of long bones, l. tibia, sacrum & sternum	Cortices of long bones very porous, external surfaces micro pitting i.e. woven bone.
100.	**533**	12A	cassa bt tomb 57-58	1-2	Fragmentary cranium, mandible & a few long bone frags	No observable pathology
101.	**536**	10	inhumation	4-5	Complete skeleton in good condition	No observable pathology
102.	**541**	19	cappucina	3-4	Cranium & mandible, diaphyses of long bones only	Orbital porosity
103.	**542**	1	inhumation	1-2	Complete skeleton minus long bones epiphyses, some portions of frontal, temporal and parietal bones.	Slight flaring of proximal & distal tibiae, orbital porosity
104.	**543**	12	amphora	0-0.5	Fragmentary arm & leg bones & some cranial frags.	Thalassemia, very porous, no x-rays
105.	**546**	n/a	tomb – 5E forma 2	5-6	Mandible, diaphyses of some long bones	No observable pathology
106.	**548**	n/a	tomb – 43 forma 1	2-3	Occipital, rib frags, l. radius, distal ends of femora, diaphyses of l. tibia & fibula	No observable pathology
107.	**552**	n/a	tomb 5E forma 2	4-5	Some cranial frags, observable portions of most long bones	No observable pathology
108.	**564**	n/a	tomb 4E	1-2	Complete skeleton	Flaring of rib end (only one present
109.	**580**	?	Missing	6-7	Complete skeleton	Thickening of l. fibula and bending
110.	**586**	n/a	tomb 47a forma P	1-2	Only r. femur and r. ulna present	Downward turning of femoral neck
111.	**588**	n/a	tomb 47a forma P	6-7	r. humerus, ulna & radius, diaphyses of femora, tibiae	Right ulna bent, left missing
112.	**594**	n/a	tomb 47a	1-2	r. humerus, ulnae, radii, r. femur, l. tibia, l. ilia	No observable pathology
113.	**595**	n/a	tomb 47a	3-4	l. maxilla, l & r radius complete, r. ulna, prox. L. ulna, distal l. humerus, tibial shaft	No observable pathology
114.	**596**	n/a	tomb 47a	5-6	Intact r. ulna, l. radius, distal humeri, l. fibula, l. tibia	L. tibia cut longitudinally – show a number of Harris lines
115.	**597**	n/a	tomb 47a	6-7	Intact l. & r. humeri, r. radius, r. ulna, l. fibula & tibia	R. tibia cut longitudinally – show a number of Harris lines
116.	**598**	n/a	tomb 47a	6-7	Distal end of r. humeral metaphysis, r.	R. tibia cut longitudinally – show a number of

					femur, intact tibiae, fibulae	Harris lines
117.	**599**	n/a	tomb 47a	3-4	Cranial fragments only	Thalassemia, thickening & porosity of cranial vault & orbital roof, no x-rays available
118.	**610**	n/a	tomb 47a forma T	7-8	Very fragmentary, l. humerus, r. ulna, r. radius, r. femur, r. tibia, 1 scapula, mandible	Apparent curvature of the femoral shaft but too fragmentary
119.	**612**	n/a	tomb 47b forma D	2-3	Tibiae & left humerus only	Some curvature of humerus & tibiae. Flaring of proximal tibiae.
120.	**621**	n/a	tomb 47a	2-3	l. humerus, radii, r. ulna, r. femur prox. l. tibia & fibula, r. fibula	No observable pathology
121.	**622**	n/a	tomb 47a	13-14	r. diaphysis of humerus, l. femur, pt of diaphyseal shaft of l. femur, r. tibia, fibulae	No observable pathology
122.	**625**	n/a	tomb 47b forma E	1-2	Distal pt of humerus, l. ilia, l. femur, distal pt of r. femur, distal pt of l. tibia	No observable pathology
123.	**630**	n/a	tomb 38	2-3	r. femur & l. tibia	No observable pathology
124.	**631**	n/a	tomb 38	2-3	l. ulna, radii	No observable pathology
125.	**632**	n/a	tomb 38	8-9	r. femur, fibula, l. humerus, r. radius, ulnar fragment & epiphysis	No observable pathology
126.	**634**	n/a	tomb 45 forma 3	9-10	Tibial shaft, distal femoral diaphysis, 1st metatarsal	No observable pathology
127.	**635**	n/a	tomb 38	0-0.5	r. humerus, prox. R. femur	No observable pathology
128.	**636**	n/a	tomb 38	1-2	l. clavicle, r. radius	Radius shows strong lateral curvature, thickening of prox end & midshaft, flaring of distal end
129.	**637**	n/a	tomb 38	3-4	Radii., r. clavicle	No observable pathology
130.	**639**	n/a	tomb 47b forma H	4-5	r. humerus, frag of r. femur, tibia, r. mandible fragments includes condyle	No observable pathology
131.	**640**	n/a	tomb 47b forma H	4-5	R. mandible no condyle, r. humerus, prox. R. ulna, femoral & tibial fragments	No observable pathology
132.	**641**	n/a	tomb 46 forma 7/8	8-9	Humeri, femora fragments	No observable pathology
133.	**642**	n/a	tomb 46 forma 7/8	7-8	2 femoral shafts, 1 humeral shaft	No observable pathology
134.	**644**	n/a	tomb 45 forma 2	1-2	Complete mandible, left femoral diaphysis	Inward curvature of the mandibular condyles
135.	**646**	n/a	tomb 45 forma 1b	13-14	l. humerus, l. radius, r. ulna, r & l. femur frag, r. scapula	No observable pathology
136.	**648**	n/a	tomb 47a forma G	9-10	2 fibula, 1 tibia, partial maxillae, some vert	No observable pathology
137.	**649**	n/a	tomb 45	1-2	Long bones, l. scapula, l. pelvis, l. frontal, some rib frags.	No observable pathology
138.	**650**	n/a	tomb 47	7-8	Tibia, fibula, calcanei, l. humerus	No observable pathology
139.	**651**	n/a	tomb 47	13-14	Fragmentary long bones, talus,	No observable pathology

					calcaneous, scapula	
140.	**653**	n/a	tomb 47b forma A II	0-0.5	r. tibia & fibula, r. femur, l. humerus	Very slight bending of tibia & fibula, apparent on x-rays
141.	**656**	n/a	tomb 47a forma J	4-5	R & l. ilium, l. ischium, r & l humerus, l. radius, distal femur, cranial fragments	No observable pathology
142.	**657**	n/a	tomb 47	6-7	Very fragmentary, humeri, fibulae	No observable pathology
143.	**659**	n/a	tomb 47b	3-4	Fragments of r. humerus, tibia, femur, l. ilium	No observable pathology
144.	**660**	n/a	tomb 47a forma C	7-8	Present are right side of mandible & maxilla, humeri minus humeral heads, right radius, distal right ulna, distal left ulna, three-fourths of right femur, distal portion of left femur, left tibia & fibula, proximal right tibia & distal right fibula.	Slight medial bending of r. radius & flaring of distal end, twisting of humerii, r. femur abnormal curvature, l. tibia slight bending, LEH on l^1
145.	**661**	n/a	tomb 47a forma C	1-2	Mandible & maxilla, humeri, r. ilia, r. femur, tibiae	Flaring apparent at prox. end on r. tibia, left broken off at distal end
146.	**672**	n/a	tomb 76	3-4	Humerii, tibae, l. radius	No observable pathology
147.	**675**	n/a	tomb 44 forma 4	0-0.5	r. ulna & radius, humerii frags., 4 cranial fragments	No observable pathology
148.	**681**	?	missing	15-16	Quite fragmentary, 1 femur, few vert, 1 calcaneous, few metatarsals	No observable pathology
149.	**683**	n/a	tomb 76	13-14	Left mandible and maxilla, clavicles, scapulae, humeri, radii, proximal left ulna, ilia, right femur, distal left femur and a couple vertebrae.	Lateral curvature of rt. radius (l. broken) Rib frags. and vertebrae porous
150.	**684**	?	missing	6-7	r. femur, face & r. cranium	Some medial & lateral cribrotic pitting in superior orbits – some healing of this.
151.	**687**	n/a	tomb 38	7-8	Mandible, humeri, radii, ulnae & ilia. Ends damaged on all.	No observable pathology
152.	**701**	n/a	tomb 43 forma 5	5-6	1 femur shaft, 2 tibial shafts, 1 fibula, 2 humeri, 1 ulna, 1 scapula & clavicle	No observable pathology
153.	**736**	?	missing	5-6	Long bones on right side of body only	No observable pathology
154.	**741**	n/a	tomb 7 forma 9	5-6	r. ilium & femur, l. tibia, distal pt of r. fibula	No observable pathology
155.	**742**	24	Tomb 85 cappuccina	14-15+	Skull and clavicles	Thickening of cranium, no clear table
156.	**743**	n/a	tomb 47b	14-15+	Ilia, pt of sacrum, most portions of long bones	No observable pathology
157.	**748**	n/a	tomb 43 forma 12	4-5	r. humeri, r. prox. radius, 1 rib, diaphysis of r. femur	No observable pathology
158.	**749**	?	missing	6-7	r. ilium, manubrium tibia/fibula, l. ulna, sacral #1, axis, L1, r. calcaneous, 2	No observable pathology

					metatarsals. these items are in good condition	
159.	**752**	n/a	tomb 44	1-2	l. ilium, l. pelvis, humerii, 2 rib frags.	No observable pathology
160.	**753**	n/a	tomb 39	14-15+	r. femur, l. humerus, max. & mand.	No observable pathology
161.	**754**	?	Inhumation unknown	8-9	r. femur, r. tibia, prox. l. humerus, 5th lumbar,	Metaphyseal ends of tibia & possibly femur appear depressed
162.	**757**	n/a	tomb 72 forma 19	1-2	Parietals, frontals, r. mand & max., r. femur, r. ischium, rib frag.	Very tiny pitting in r. orbit.
163.	**758**	n/a	tomb 76	0-0.5	l. prox. femur, l. radius, r. ulna, r. humerus & rib frag.	No observable pathology
164.	**759**	n/a	tomb 76	5-6	l. humerus &l. radius, 4 vert, r. ilium, rib frags, prox. tibia	No observable pathology
165.	**764**	?	missing	13-14	Maxilla, scapulae, ribs, humeri, r. tibia & fibula	No observable pathology
166.	**767**	n/a	tomb 40 forma B/8	1-2	Some teeth, cranial fragments, diaphyses of femora, tibiae	No observable pathology
167.	**770**	n/a	tomb 40 forma B/8	5-6	r. femur, diaphyses of tibiae, fragments of fibulae	No observable pathology
168.	**773**	n/a	tomb 45	0.5-1	Mandible minus r. ramus, clavicles, r. scapulae, r. humerus, distal pt of l. humerus, l. ilium, r. radius, prox. pt of r. ulna, midshaft of r. femur, prox pt of r. fibula	Posterior/medial bending of l. mandibular ramus (r. missing)
169.	**774**	n/a	tomb 46 Forma 1/8	1-2	r. scapula, r. prox. humerus, l. ilium, l. femur, pts of tibiae	No observable pathology
170.	**777**	n/a	tomb 41 forma 3	0-0.5	Femora & tibiae	No observable pathology
171.	**778**	n/a	tomb 41 forma 3	0.5-1	Some cranial bones, l. humerus & shaft of r. humerus, missing distal end of r. ulna, shaft of r. femur, r. tibia, shaft of fibulae	No observable pathology
172.	**780**	n/a	tomb 43 forma 4	1-2	Occipital, scapulae, humeri, ulnae, l. radius, r. ilia, femora, tibiae, fibulae & fragmentary vertebrae.	Bending & flaring of fibulae
173.	**781**	n/a	tomb 43 forma 4	4-5	Portions of most long bones present	No observable pathology
174.	**783**	n/a	tomb 46b	10-11	Portions of mandible and maxilla, left humerus, radius & ulna, head of l. femur pox. End of l. fibula	No observable pathology
175.	**788**	n/a	tomb 43 forma 12	2-3	R & l humeral shafts, r & l fibula frags, 1 rib frag	No observable pathology
176.	**791**	n/a	tomb 42	4-5	Cranial frags, long bones, rib frags, pelvis mandible	No observable pathology

177.	**803**	?	missing	7-8	Complete skeleton – long bone ends broken	No observable pathology
178.	**810**	?	missing	13-14	Complete skeleton in good condition	Periostitis on lateral tibiae shafts. Harris lines visible in tibiae. Transverse fossa lesions on thoracic vert bodies
179.	**811**	?	missing	2-3	Humerii, femora, radius & ulna shafts, mandible, maxilla	Pitting –open in antero-medial part of l. orbit
180.	**812**	?	missing	14-15+	Complete skeleton in good condition	Slight pitting in antero-medial portions of orbits. Transverse siit-like fossae/lesions on T4-T8 anterior bodies of vert
181.	**815**	?	missing	7-8	Complete skeleton but brittle & fragmentary, no bone ends preserved	No observable pathology
182.	**839**	13	cappuccina	14-15+	Complete skeleton, good condition, most epiphyses fused	No observable pathology