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The "scapula sign": a new indicator for rickets?

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The “scapula sign”: a new indicator for rickets?

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Master Thesis

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Introduction

1.1 Rickets, scurvy, and the ‘scapula sign’

Rickets is a metabolic disease caused by a vitamin D deficiency, which mainly occurs in growing individuals. It is characterized by minerally compromised newly formed bone and weakened existing bone tissue due to lacking availability of necessary calcium salts, as vitamin D plays a crucial part in the body’s calcium metabolism (Mays and Brickley, 2022, p.36). Although the lack of vitamin D can be hereditary or have other causes, the majority of rickets cases finds its etiology in nutritional causes and lack of exposure to sun (Diaz-Thomas & Iyer, 2023, p.646). Nutritional rickets occurs globally – not only in areas with geographically low concentrations of UV exposure rickets cases are present, but also socio-cultural and socio-economic conditions that might cause differences in (the availability of) health care are factors in the occurrence of rickets (Diaz-Thomas & Iyer, 2023, p.644).

Although nutritional diseases are hard to detect in the archaeological record, another nutritional disease that shows lesions in human skeletal remains is vitamin C deficiency or scurvy (Snoddy et al., p.877). As one of the main uses of vitamin C is the production of collagen, a deficiency will cause not only hemorrhaging due to the weakened structure of connective tissue, but also reduced osteoblastic activity of extra-cellular bone matrix, which can eventually lead to osteopenia (low bone mineral density) (Brickley & Ives, 2020, p.46,50; Snoddy et al., 2018, p.878; Klaus, 2017, p.97).

The study of vitamin D deficiency related diseases in skeletal remains dates back to the 19th century, although then, the literature mostly consisted of individual case studies. It was not until recently that paleopathologists started to shift their interests towards the study of rickets; probably due to a greater interest in childhood paleopathology (the study of disease in the past) (Mays & Brickley, 2018, p.1; Hadley & Hemer 2014, p.1). The identification of rickets was primarily based on the presence of (residual) bending deformities in the long bones of older children and adults as there were very few studies on rickets occurring in infants, because the assessment of the presence of rickets in dry bone was quite difficult (Mays & Brickley, 2018, p.2). Only in the late 1990s, physical anthropologist Donald J. Ortner († 2012) was among the first to incorporate clinical literature on rickets and radiographic imaging to support his research into the vitamin D deficiency

disease. His 1998 article initiated the publication of a plethora of papers describing new cases of vitamin D deficiency and paved the way into restructuring the diagnostic assessment of rickets in paleopathological research (Mays & Brickley, 2018). It was also Ortner who first tried to lay down a framework of macroscopically visible lesions to assess diagnostic certainty of scurvy in dry bone (Snoddy et al., 2018, p.876). Other researchers after him have added to this framework, although this framework is – as it is for rickets – still growing and developing because nutritional diseases such as rickets and scurvy are difficult to diagnose, as the majority of the lesions associated with both diseases are also viewed as non-specific markers of stress or anemia (Zuckerman et al., 2014, p.27).

Furthermore, in recent years, bioarchaeologists have begun to research the co-occurrence of rickets and vitamin C deficiency or scurvy, which can be seen as part of the increased interest in recognizing and diagnosing co-occurring conditions (Brickley & Morgan, 2023, p.638). Although it is possible to assume the phenomenon of co-occurrence also occurred in the past, detailed analyses of affected individuals are very rarely found in paleopathological literature (Schattmann et al., 2016, p.63). After all, is not unthinkable that individuals suffering from malnutrition are suffering from more than one nutritional deficiency simultaneously. The co-occurrence of scurvy and rickets has been studied by clinicians, but few reports exist in paleopathological literature. The symptoms of both rickets and scurvy affect each other: for example, a vitamin C deficiency result in reduced osteoblastic activity and might therefore suppress the long bone bending deformities as is characteristic of rickets (Brickley & Ives 2020, p.232).

In 1971, radiologist and pediatrician Alfred Weiss from the Beilinson Hospital in Petah-Tiqva, Israel, introduced what he called ‘the scapular sign’; a rachitic induced change in the inferior angle of the scapula in growing individuals, which to date, has been getting little attention in rickets focused clinical research or in palaeopathology (Weiss, 1971, p.633; Ives, Swan & Humphrey, 2023, p.61). In 2023, a second paper discussing the scapula sign was published by R. Ives, K. Swan, and L. Humphrey, wherein the question was posed if the scapula sign could be an indicator of rickets. Their study found that the scapula sign as posed by Weiss mainly occurred in individuals with severe rickets, albeit not exclusively. This finding is a great contribution to the

framework of lesions visible in rickets, but the scapula sign has not nearly been studied enough to speak of a new diagnostic criterium for rickets (Ives, Swan & Humphrey, 2023, p.60).

Although research into the co-occurrence of rickets and scurvy so far is promising, not many osteoarcheological articles have been published yet. Additionally, detection and assessing diagnostic certainty of nutritional diseases in non-adults still needs to be further studied, as it remains challenging to assess nutritional diseases in the bioarchaeological record (Lewis, 2009, p.133). Because of the lack of research into Alfred Weiss' scapula sign with regards to diagnosing rickets, it is interesting to study whether the presence of the scapula sign might have a correlation with scurvy or the co-occurrence of rickets and scurvy, as results in the article by R. Ives, K. Swan, and L. Humphrey (2023) do not give further detail as to why the scapula sign did not exclusively occur in individuals with severe rickets. As vitamin C is also a crucial building stone for the production of bone matrix and cartilage, it could be worthwhile to study whether the scapula sign can be found in individuals with scurvy, or in individuals with scurvy and rickets. Furthermore, as porosity on the supra- and infra-spinous fossae of the scapula is already considered as a lesion associated with scurvy, it is not farfetched to explore if other parts of the scapula would be affected as well (Geber & Murphy, 2012, p.516). This master thesis will therefore focus on exploring whether the scapula sign is indeed an indicator for rickets like Weiss (1971) poses, or also for scurvy, or if the sign might be unrelated after all.

1.2 Research questions

The main research question reads as follows:

“To what extent is the scapula sign as identified by Weiss (1971) an indicator for rickets or a possible (new) indicator for scurvy and/or the co-occurrence of rickets and scurvy in the post-medieval non-adults of Middenbeemster?”

The answer to this question will be explored through the following sub-questions:

- What is the prevalence of rickets and scurvy in the Middenbeemster population?

- What is the prevalence of the scapula sign? How many of the individuals in the sample have the scapula sign, and have rickets and/or scurvy?
- What is the distribution per age group of the prevalence of rickets, scurvy, and the scapula sign?

1.3 Approach

To answer these questions, a sample of 47 growing individuals between the age of 0-12 from the Middenbeemster population currently housed at the Human Osteoarchaeology Lab in Leiden, with one or both scapulae intact will be analyzed to assess the presence of rickets and/or scurvy based on the paper by Brickley and Morgan (2023). The Leiden collection of the Middenbeemster population is comprised by individuals excavated from the cemetery of Keyser church (1617-1866) in 2011 (Lemmers et al., 2013, p.35). Subsequently, their scapulae will be analyzed for presence of the ‘scapula sign’ as described by Weiss (1971) and further defined by Ives, Swan, and Humphrey (2023). Furthermore, it will be assessed whether there is a correlation between the diagnosis of rickets and/or scurvy, and the presence or absence of the scapula sign in either or both scapulae.

1.4 Thesis outline

First, chapter 2 will be comprised of an overview of the characteristics of both vitamin deficiencies and how to assess them, and the possibility of co-occurrence of the two. Then, the problematics of diagnostic certainty will be discussed, and Weiss’ scapula sign will be reviewed. In chapter 3, the methods and materials for this study will be discussed. Chapter 4 will consist of an overview of the results, and in chapter 5 these results will be discussed, followed by a conclusion.

Chapter 2: Assessing rickets and scurvy: Theory

2.1 Vitamin D deficiency: rickets

Although not the single cause, the majority of rickets cases are a result of a deficiency in vitamin D. Sufficient levels of vitamin D are either acquired through ingestion of vitamin D-rich food (e.g. fish oil or egg yolks), or somatically produced through skin exposure to UV radiation (Mays & Brickley, 2022, p.36; Veselka, 2019, p.13, Lewis, 2009, p.119). This is why cases of rickets often occur in areas where sunlight is scarce such as densely populated urban areas with smog or areas with cold, wet climates such as the Netherlands or the United Kingdom (Diaz-Thomas & Iyer, 2023, p.644). There are several forms of vitamin D, the most common of which are vitamin D3 (cholecalciferol), which is produced in the skin, and vitamin D2 (ergocalciferol), which can be derived from plants. Both vitamin D3 and D2 undergo two hydroxylation processes before the most active form of the vitamin, 1,25(OH)₂D, is produced (Veselka, 2019, p.14).

Vitamin D has many functions in the body, the most known is that of calcium homeostasis, whereby vitamin D is needed for the mineralization of newly formed bone (osteoid) and the development of cartilage (Ives, Swan & Humphrey, 2023, p.59). Normally, during the growth and development stage of a human's life, osteoid is deposited after which mineralization is required to provide the bone with strength and hardness (Veselka, 2019, p.14). For infants, the process of calcium homeostasis changes within weeks after being born from a passive homeostasis (through the placenta) to an active one where the infant becomes dependent on vitamin D from nutrition and UVB exposure. As breastmilk is not very vitamin D rich, it is often seen that infants have a higher risk of acquiring rickets if being breastfed for too long – which can be the case when other possible food sources are scarce. Vitamin D deficiency thus can result in defective mineralization of newly formed osteoid, especially during growth and development phases (Ives, Swan & Humphrey, 2023, p.59). Furthermore, if the calcium intake continues to be insufficient during childhood, growing individuals will still be at risk (Diaz-Thomas & Iyer, 2023, p.649).

Vitamin D deficiency may lead to bending, metaphyseal flaring, and thickening deformities in the skeleton, because mineralization of the newly formed osteoid is inadequate or impaired (fig.1). This is most visible in the ribs and the weight bearing bones, such as the arm and leg bones, as these bones will bend more

easily due to gravity and/or muscular tension (fig.2) (Veselka, 2019, p.14; Ives, Swan & Humphrey, 2023, p.59). The appearance and manifestation of rickets in growing individuals is dependent on general nutrition, age, growth rate and mobility of the individual (Lewis, 2009, p. 121). Furthermore, growing individuals that are not necessarily underfed, but are not outside enough or do not eat enough food stuffs containing vitamin D are more likely to show bowing defects and thickened cortices (the outer shell of compact bone matrix), while malnourished growing individuals will more likely develop general atrophy and thinned cortices (Lewis, 2009, p.122). Growing individuals that were able to stay mobile during their disease period show more intense bowing than individuals who were more stationary. Also, bowing of the upper limbs can be observed in infants who develop rickets during their crawling stage (Lewis, 2009, p.123).



1. Metaphyseal flaring/porosity on two tibias. Taken from Schattmann et al., 2016, p.70. Copyright A. Schattmann.



2. Image a) shows the difference between a 'normal' left humerus (left), and a bending deformity in a left humerus (right). Image b) shows a clear biomechanical defect in another left humerus. Taken from Schattmann et al., 2016, p.70. Copyright A. Schattmann.

The identification of rickets has a long history: the first study on rickets was published in 1645 and focused on English children who worked indoors as wool spinners and did not frequently come outside. As a result of the

Industrial Revolution in Europe (17th – 20th century), more and more cases of rickets are described, often case studies of poor children in dense urban areas in northern Europe (Diaz-Thomas & Iyer, 2023, p.646). As previously mentioned in the introduction, before Donald J. Ortner presented a framework for identifying other lesions regarding the manifestation of rickets in dry bone in 1998, the presence of rickets was usually assessed through residual bowing defects in long bones of older children and adults (Mays & Brickley, 2018, p.2). However, although rickets can be easier identified in growing individuals, signs of rickets can disappear just as easily when somatic stores of vitamin D are replenished (Lewis, 2009, p.124). Additionally, the first symptoms of rickets are subtle but can result in whooping coughs and gastrointestinal infections, usually before bowing defects occur. This means a possibility exists that growing individuals may not survive the disease long enough before the more easily detectable lesions develop in their skeletons (Lewis, 2009, p.125). If non-adults do survive the disease, a distinction can be made between active rickets and healed rickets. As non-adults grow rapidly, replenishment of vitamin D results in the fading of some signs, such as the flaring of the costochondral rib ends and metaphyseal ends of the long bones. The thickening of the cranial vault and thickening of the long bones due to the restored levels of vitamin D and possible residual biomechanical defects such as bowing indicate an individual might have had rickets and has subsequently healed (Brickley & Morgan, 2023, p.640).

For this thesis, the lesions that will be examined to identify the presence of rickets will be limited to skeletal, macroscopically visible lesions associated with rickets: in other words, all lesions associated with rickets that result from defective mineralization and/or biomechanical defects.

2.2 Vitamin C deficiency: scurvy

Scurvy, or vitamin C deficiency, is often omitted in clinical literature on metabolic (bone) diseases because humans are not able to synthesize their own vitamin C and therefore does not classify as a “true” metabolic bone disease. Unlike most mammals, humans need to obtain this through diet, and the deficiency can therefore be interpreted as a result of some form of malnutrition (Brickley & Ives, 2020, p.43). First observed as a sailor’s disease, scurvy was not considered as a disease that could occur in non-adults as well until the emergence of industrialization (Lewis, 2009, p.127). The metabolism of vitamin C ensures the production of collagen Type 1,

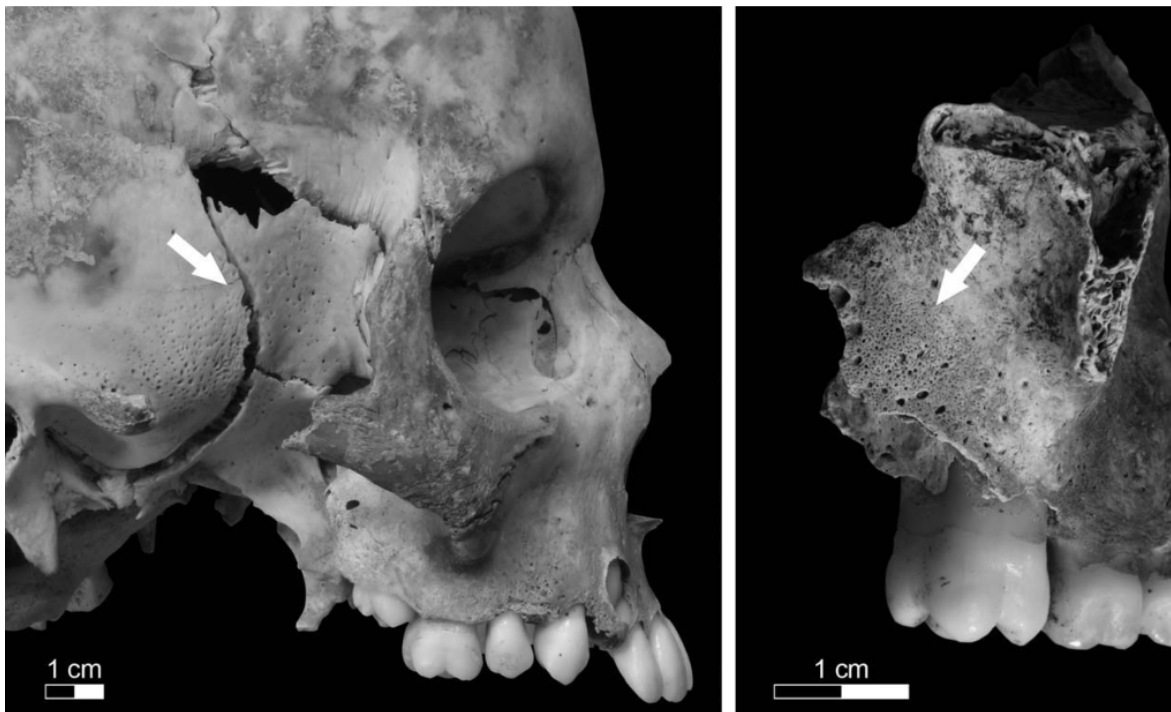
which the body needs to form connective tissues in the skin, blood vessels, cartilage, and bone (Lewis, 2009, p.127). A lack of ascorbic acid – synonymous to vitamin C – primarily results in the formation of defective collagen, and secondarily causes poor osteoid formation (Klaus, 2017, p.97; Lewis, 2009, p.127). If the bodily storage of ascorbic acid is lower than 350mg, blood vessels will become fragile and might rupture. Furthermore, periosteal membranes grow weaker, and the immune system begins to fail – which causes poor metabolism of iron and folate. The consequences of a deficiency in ascorbic acid are expressed in the form of hemorrhaging – an inflammatory response of the blood outside the circulatory system – and is one of the most common characteristics of scurvy (Klaus, 2017, p.97). However, when hemorrhages form around bone tissue, skeletal signs can be observed; here, lesions will be expressed as increased porosity and/or the formation of subperiosteal new bone (SPNB) formation near the site of hemorrhaging (Klaus 2017, p.97; Brickley & Ives, 2020, p.52).

Since hemorrhaging in bodily tissue is the primary result of vitamin C deficiency, only secondary lesions are visible in skeletal remains when a hemorrhage appears near bone (Brickley & Ives, 2020, p.52.). This is most often occurring near cranial bones. Scurvy does not occur *in utero* or at birth; even if the mother herself does not have adequate levels of ascorbic acid, does it take at least four months until a vitamin C deficiency becomes observable, and most cases of scurvy occur in infants between six months and two years old (Ortner & Putschar, 2003, p.270, Lewis, 2009, p.127).

Scurvy causes depressed osteoblastic activity, resulting in reduced or arrest deposition of osteoid. This is also why osteopenia is frequently reported in scurvy cases. Furthermore, a stagnation of new bone formation can be observed in growing individuals, as well as reduced levels of bone within existing tissue (Brickley & Ives, 2020, p.50). The lesions caused by scurvy are most visible on skeletal elements that grow fast, such as the costochondral ends of ribs, distal metaphysis of femur and the cranial bones (Ortner & Putschar, 2003, p.270). The lesions associated with scurvy are often also associated with other metabolic diseases such as anemia or rickets, which resulted in a “under-diagnosing” of many scurvy cases for many years. (Zuckerman et al., 2014, p.27; Lewis, 2009, p.127). However, D.J. Ortner and his colleagues developed a framework of criteria, wherein he argued that bilateral porosity on the greater wing of the sphenoid bone (GWS) could be taken as a

pathognomonic lesion for scurvy (fig.3) (Zuckerman et al., 2014, p.27; Klaus, 2017, p.107). Lesions on the GWS are a result of hemorrhaging on the temporalis muscle underlying the sphenoid bone, which is used during underjaw movement and mastication (Ortner & Ericksen, 1997, p.213,215; Geber & Murphy, 2012, p.515; Snoddy et al., 2018, p.878). As discussed above, vitamin C is crucial for the production of collagen and tissues such as skin, gums, mucus membranes (and bones) are comprised of a higher concentration of collagen, therefore the first to be affected by ascorbic acid deficiency. This explains why many of the scurvy lesions are found mainly in cranial bones such as the GWS, posterior surface of the maxillae, and medial surface of the mandibular ramii (Geber & Murphy, 2012, p.515,516; Léger, 2008, p.1404).

Other lesion sites that are associated with scurvy include the zygomatic bone, inferior surface of the palatine processes of the maxillae, orbital walls, infraorbital foramen of the maxilla, foramen rotundum of the sphenoid bone, long bones, the supra- and infraspinous fossa of the scapulae and the ilia. On all these sites, porosity and/or the formation of subperiosteal new bone (SPNB) due to (primary) hemorrhaging is expected (Brickley & Morgan 2023, p.639).



3. Left: example of porosity on the greater wing of the sphenoid bone. Right: Porous bone formation on the posterior surface of the maxillary body. Taken from Geber & Murphy, 2012, p.516. Copyright J. Geber and E. Murphy.

2.3 Co-occurrence: a relationship between scurvy and rickets?

The term co-occurrence in paleopathology is used when discussing the presence of at least two (chronic) conditions (Brickley & Ives, 2020, p.227). With the co-occurrence of two (or more) diseases, an etiological relationship is inferred, which is difficult to determine based on skeletal remains alone. It is therefore important to review which potential biological interactions or socio-cultural factors have caused each disease present in an individual, because it is possible that they do not have a link after all (Brickley & Ives, 2020, p.227-229). When two conditions might co-occur, it is possible that lesion expression in skeletal remains will be influenced by one another (Brickley and Ives, 2020, p.229). As for rickets and scurvy, their co-occurrence has been a topic of study among clinicians and paleoarcheologists. Because they are both diseases relating to malnutrition, it is not uncommon that both could be present at the same time (Lewis, 2009, p.97). Although vitamin deficiencies can develop at any time, it has been reported so far that co-occurrence of rickets and scurvy usually develops in individuals between 6 months and 5 years old (Schattmann et al., 2016, p.63).

When two or more diseases co-occur, their interaction can affect each other's (lesion) expression (Schattmann et al., 2016, p.64). In the case of rickets and scurvy, the reduced osteoblastic activity due to the lack of ascorbic acid could hinder any bowing defects caused by rickets because less osteoid will form. Furthermore, lower levels of vitamin D causes poor bone mineralization, which can influence the formation of subperiosteal new bone, a lesion common in scurvy (Brickley and Ives, 2020, p.232). Hemorrhaging is not affected by vitamin D deficiency, but the weakened bone that is subsequently produced is not likely to survive in archaeological contexts very often (Brickley and Morgan, 2023, p.642). However, it must be said that the interaction between the effects of rickets and scurvy are not black and white and are completely dependent on the order of onset of the diseases, severity, and duration (Schattmann et al., 2016, p.67). It is not unlikely that the first deficiency will 'mask' symptoms of the second, as the first onset disease often dominates, which can be the case in rickets-scurvy co-occurrence (Schattmann et al., 2016, p.67). Medical literature suggests that it is often scurvy that is the primary disease in rickets-scurvy co-occurrence, and rickets the secondary disease (Schattmann et al., 2016, p.67). Although lesions of active rickets and healed rickets can be differentiated – the flaring of metaphyseal and costochondral ends often seen as signs of active rickets because of impaired

mineralization, and thickening of the cranial vault and long-bones attributed to healed rickets due to restoration of sufficient vitamin D – this is not yet the case for cases of healed and active scurvy, as it seems that skeletal evidence for scurvy can be observed only after vitamin C levels are replenished after a deficiency period, and scorbutic lesions appear after osteoid production resumes (Schattmann et al., 2016, p. 68; Brickley and Morgan 2023, p.640; Geber & Murphy, 2012, p.515). Therefore, rickets-scurvy co-occurrence can be difficult to diagnose; signs of malnutrition are usually only visible after the individual has recovered (Lewis, 2009, p.103). Furthermore, the body of paleopathological literature on the co-occurrence of rickets and scurvy remains slim so far, and comparable case studies are limited in number (Schattmann et al., 2016, p.63). For this thesis, both diseases will be assessed individually and separate from one another, after which the presence or absence of co-occurrence will be concluded.

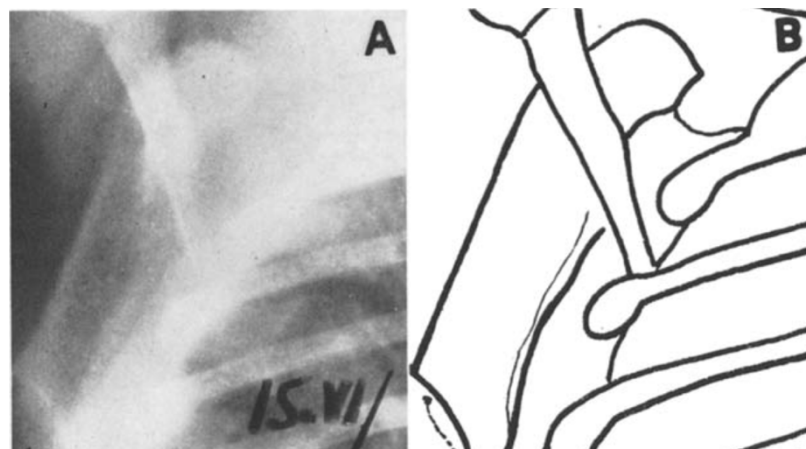
2.4 The ‘scapula sign’: overlooked or irrelevant?

As previously mentioned in the introduction, radiologist Alfred Weiss of the former Beilinson Hospital in Petah-Tiqva, Israel, introduced the ‘scapula sign’ in 1971, after seeing notably visible changes in the inferior angle of the scapula of children with active rickets on their radiographs (Weiss, 1971, p.633). Unfortunately, both in clinical literature as well as in paleopathological literature, this scapula sign has been quite overlooked so far.

To put into perspective, the original article by Weiss has been cited by only five other sources according to Google Scholar, all from before 1990 except for an article by Rachel Ives, Karen Swan, and Louise Humphrey (2023), which will be discussed further below. In his article, Weiss (1971) discusses changes in the inferior angle of the scapula caused by rickets. It is already quite commonly known that rickets is primarily visible in radiographs in the epiphyseal plates of the long bones as well as in the ribs and skull. However, the scapula is rarely examined because it was deemed to not be seen clearly enough on radiographs (Weiss, 1971, p.633). Weiss argues in his article that the rachitic changes in the scapula actually can be seen on radiographs if the individual’s arms are placed over the head and displacing the angle of the scapula away from the lungs (Weiss, 1971, p.633). Furthermore, he argues that changes in the scapula are seen in quite early stages of the disease,

especially in infants. According to Weiss, there are three stages: first, the border of the scapular angle becomes “indistinct” as a result of the failure in calcification of the cartilaginous cells. As the rickets progresses, the metaphyseal (inferior) angle alters from a convex to a concave shape, with the indistinct border remaining indistinct. Lastly, only seen in severe cases, the growing buildup of non-calcified cartilaginous cells at the metaphyseal border of the inferior angle continues but no new bone is formed, leaving the angle to appear “cut off and brush-like” (fig.4) (Weiss, 1971, p.634).

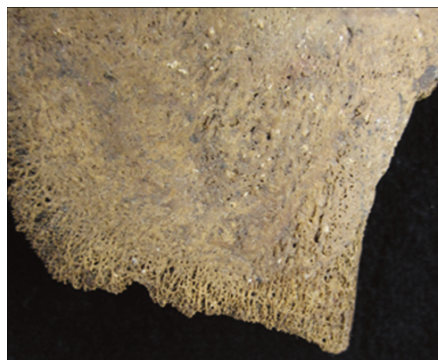
Noteworthy about this article is the fact that Weiss only mentions thirteen cases of rickets, and almost all of them infants. There is no further research on different age groups, and his original sample is very small. Furthermore, it is interesting that this scapular sign was accidentally discovered in unsuspected cases of rickets: these infants were brought into the hospital with other, often respiratory related symptoms, and were only diagnosed with rickets after these changes in the scapula were observed in radiographs (Weiss, 1971, p.633). Lastly, Weiss mentions that the nature of the observed scapular changes should result in considering the scapula as a “flattened long bone”, which can be taken as an argument as to why the scapula sign should be considered as an indicator for rickets (Weiss, 1971, p.636). Unfortunately, Weiss’ other publications – if any – are hard to find. The other five articles that cite Weiss’ paper do not mention anything on his discovery, but merely reference him when arguments are made on the importance of certain bones – the scapula – being examined when diagnosing rickets, but no reference to the use or diagnosis of the scapular sign specifically.



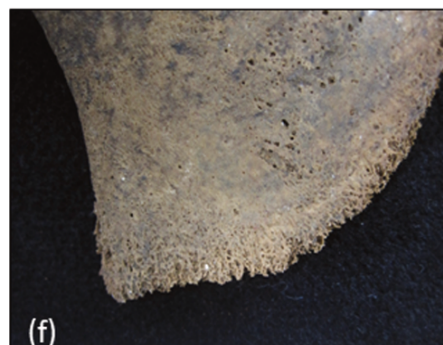
4. A: X-ray of one of Weiss' patient's scapulae, showing a "cut-off" inferior angle. B: Schematic drawing of the cut-off inferior angle shown in A. Taken from Weiss (1971), p. 635. Copyright Alfred Weiss.

In 2023, bioarcheologists Rachel Ives, Karen Swan, and Louise Humphrey published an article reposing the question whether the scapula sign could be an indicator for rickets, based on Alfred Weiss' discovery in 1971, and if this would be a valuable addition to diagnostic criteria for paleopathological research for rickets. Two post-medieval British assemblages from two large cemeteries – Christ Church Spitalfields and Bethnal Green – were evaluated to see if the changes at the inferior angle were macroscopically observable (Ives, Swan & Humphrey, 2023, p.61). The authors too, confirmed that there was very little research regarding rachitic scapular changes and only discuss two other papers from 1998, one from Ortner and Mays (1998), who mention the discovery of two juvenile cases with active rickets and scapulae with irregular, porous cortex “adjacent to the growth plates”, and a study by Littleton (1998) discussing “a porosity layer and a roughened bone surface at the inferior angle of the scapula” of an infant (Ives, Swan & Humphrey, 2023, p.61).

In their study, Ives, Swan, and Humphrey (2023) analyzed a total of 527 juveniles, 0-12 years old, with one or both scapulae observable. They macroscopically evaluated all inferior angles of all scapulae and studied the presence of changes in shape to concavity, porosity, coarseness, blunting and the presence of “slit-strut morphology”: vertical struts in the cortical bone surface of long bones and ribs, of which Ortner and Mays (1998) have argued these represent the presence of unmineralized osteoid during life (fig.5 and fig.6) (Ives, Swan & Humphrey, 2023, p.60). Radiographs were taken to help visualize the suggested traits. The authors also measured all scapulae to see if individuals with rickets tended to have smaller scapulae than individuals without rickets.



5. Anterior surface of the left scapula of Individual NHMUK PA UNREG 3599 CAS84 2772 showing slit-strut morphology, loss of the solid border and the concavity of the inferior angle. Taken from Ives, Swan, and Humphrey (2023), p.64. Copyright Trustees of the National History Museum.



6. Anterior view of right scapula of Individual NHMUK PA UNREG 3702 CAS84 2876 showing slit-strut morphology and concavity of the inferior angle. Taken from Ives, Swan, and Humphrey (2023), p.64. Copyright Trustees of the National History Museum.

The study of Ives, Swan, and Humphrey (2023) showed that the scapula sign was not identified in individuals that did not have other skeletal changes caused by rickets; only individuals with “severe cases of rickets” presented a scapula sign (Ives, Swan & Humphrey, 2023, p.62). Furthermore, the authors concluded that the highest prevalence of scapula signs was found in individuals between 1 and 5 years old (Ives, Swan & Humphrey, 2023, p.62). They also observed that, for all individuals that had the scapula sign, the criteria of blunting, flattening, or squaring of the inferior angle was visible macroscopically as well as radiographically (Ives, Swan & Humphrey, 2023, p.62). Slit-strut morphology was mainly visible macroscopically and looked like “fraying of the border” of the inferior angle on radiographs (Ives, Swan & Humphrey, 2023, p.62). Furthermore, the maximum or minimum length of all scapulae did not reveal a consistent pattern; some individuals with rickets did indeed have smaller scapulae than expected for their age, but this was not found everywhere in the sample. Unfortunately, the authors do not mention whether smaller scapulae could have anything to do with the severity of the rickets lesions (Ives, Swan & Humphrey, p.62).

In conclusion, Ives, Swan, and Humphrey (2023) argued that the scapula sign as described by Weiss (1971) is visible in archaeological remains, mostly in individuals with severe cases of rickets, although not exclusively. Furthermore, the scapula sign was most prevalent in individuals between the age of 1 and 5 years old. However, although the prevalence of the scapula sign in their whole sample of individuals with rickets was not higher than 22%, the authors argue that the scapula sign can be added to the lesions identifiable for rickets (Ives, Swan & Humphrey, 2023, p.67).

Chapter 3: Materials and Methods

3.1 Materials

3.1.1 The Middenbeemster population

The sample for this thesis consists of individuals from the Middenbeemster collection currently stored at the Human Osteoarcheology Lab in Leiden. In 2011, the Laboratory for Human Osteoarcheology and the archaeological company Hollandia Archeologen conducted an excavation near the Keyser church in the town of Middenbeemster (fig.7). The town of Middenbeemster was founded after the Beemster lake was reclaimed and turned into a polder between 1609 and 1613



7. In red: the excavation area of the cemetery of the Keyser church. Image taken from Hakvoort, A. et al. (2013). *De begravingen bij Middenbeemster. Hollandia (464), p.11.* Copyright A. Hakvoort/Hollandia Archeologen.

designed by Hendrick de Keyser) was eventually constructed and initiated in 1623. The town of Middenbeemster lies directly in the center of the Beemster polder, and the cemetery of Middenbeemster near the Keyser church acted as the final resting place for all Beemster inhabitants living in its vicinity (Hakvoort, 2013, p.14; Leiden University, 2011). Due to its complex layout and planning, the Beemster polder eventually gained the status of World Heritage Site ordained by UNESCO in 1999 (Leiden University, 2011).

When the Protestant church wanted to build an annex to the Keyser church, the cemetery of Middenbeemster had to be excavated. During the summer of 2011, the University of Leiden in collaboration with Hollandia Archeologen brought a surprising number of 412 skeletons to the surface although the preliminary analysis done by Hollandia Archeologen predicted no more than some 250 graves (Lemmers et al., 2013, p.40,59; Leiden University 2011; Van Nuland, 2019). The graves in the cemetery of Middenbeemster can be dated between 1617 and 1866 but the majority of the interred remains most likely date to the late 18th and 19th centuries. Every individual was buried in a wooden coffin, some of which decorated with iron or brass handles or lettering made of copper nails on the lid (Leiden University, 2011). Furthermore, a map of the

cemetery documenting all names of the people buried in each plot from 1829 onwards was found, and after the deciphering of a name on one of the coffins, the archaeologists were able to comprehend the old layout of the cemetery (Van Nuland, 2019). Members of the Historical Association of Middenbeemster helped the archaeologists clean the excavated remains and aided in the archival part of the research, exploring the ancestry of the old inhabitants (Van Nuland, 2019).

3.1.2 Sample selection

The sample chosen for this thesis is comprised of 47 individuals between the age of 0 – 12 years old with the inferior angle of one or both scapulae still intact. The sample consists of 21 infants (0-2 years), 11 children (2-5 years) and 15 juveniles (5-12 years). The classification of age groups is based on Lemmers et al. (2013), but slightly modified for this thesis as the estimated ages in the database give a broad estimation of the exact age of the individuals. For example, an individual aged 2 years +/- 6 months is classified as a child and not as an infant, and an individual aged 4.5 years +/- 6 months is classified as a juvenile and not as a child. The complete distribution and ages for this sample can be found in Appendix 1.

3.2 Methods

3.2.1 Estimation of age

After the excavation, the age of the individuals was estimated through a combination of methods: dental measurements of both deciduous and permanent teeth by Liversidge et al. (1998), dental development of deciduous teeth by Demirjian et al. (1973), dental development of permanent teeth by Moorrees et al. (1963), and dental eruption by Ubelaker (1979). For individuals with no preserved teeth, age was estimated through measurements of long-bone length by Maresh (1970), clavicle length by Black and Scheuer (1996), and stage of bone and epiphyseal fusion by Schaefer et al. (2009) (Lemmers et al., 2013, p.37-39). For the scope of this thesis, the age of the analyzed individuals was not determined again and is taken directly from the database of the Laboratory of Human Osteoarchaeology, based on the report by Lemmers et al. (2013).

3.2.2 Diagnostic criteria for rickets and scurvy

As the identification of nutritional diseases such as rickets and scurvy can create a better understanding of the general health and variability therein of past groups, striving towards better diagnostic certainty is imperative. (Brickley and Morgan, 2023, p.638; Snoddy et al., 2018, p.877). Already mentioned in the introduction, Donald J. Ortner was the first to lay down a framework of macroscopically visible lesions in dry bone, for both rickets and scurvy. Over the years, studies have increased in number, and many more researchers have contributed to the framework of Ortner. Unfortunately, it seems that the subjectivity and interpretation of the researcher still plays a big factor in the assessment of lesions. This is also visible in the papers that are being published; there is a lack of consensus on the language used to express diagnostic certainty (Brickley & Morgan, p.638). Ambiguous diagnostic language is not only a problem in scurvy and rickets research but occurs in all paleopathological research and diagnosis. In 2015, Jo Appleby et al. published a paper on increasing the confidence in paleopathological diagnosis by using the Istanbul terminological framework in differential diagnosis (Appleby et al., 2015, p.20). This framework was originally designed by the VN to assess trauma and torture cases in court and forensic medicine research but was modified by Appleby et al. to fit the needs in paleopathological diagnosis. Table 1 shows the adapted terminology from Appleby et al. (2015) and how this could help to increase the confidence and, most importantly, consensus, in paleopathological diagnosis and research.

Table 1. Diagnostic framework as proposed by Appleby et al. (2015), p.20.

Category	Explanation
1. Not consistent	The lesions could not have been caused by the conditions described.
2. Consistent	The lesions could have been caused by the conditions described, but it is non-specific and there are many other possible causes.
3. Highly consistent	The lesions could have been caused by the conditions described, and there are few other possible sources.
4. Typical of	The lesions are usually found with this type of conditions, but there are other possible causes.
5. Diagnostic of	The lesion could not have been caused in any other way than by the conditions described (i.e. pathognomonic).

Like the Istanbul protocol, Appleby et al. argue that “the terminology should be representative of the overall evaluation of the appearance, extent, and anatomical distribution of all lesions” (Appleby et al., 2015. p.20,21).

In 2023, Brickley and Morgan devised a version of this terminological framework specifically for diagnosing rickets and scurvy. In their article, they use the term ‘diagnostic of’ when discussing “unambiguous lesions that can only have been caused by the disease under consideration” (Brickley and Morgan, 2023, p.641). ‘Highly consistent/typical of’ is used when “there is a strong probability the lesions represent an instance of the disease in question, but there are other possible causes of the observed lesions that have been considered and can largely be ruled out” (Brickley and Morgan, 2023, p.642). Finally, the term ‘consistent with’ is used in cases where “the observed lesions may be seen in cases of rickets and scurvy but cannot be excluded from being related to another cause, either through lesion appearance, location or lack of corroborating evidence” (Brickley and Morgan, 2023, p.642). For this thesis, the framework as proposed by Appleby et al. (2015) and further altered by Brickley and Morgan (2023) will be used to assess rickets and scurvy on the chosen sample. The lesions will be scored as ‘present’, ‘absent’, or ‘unobservable’ if the lesion cannot not visually be observed due to poor preservation status. The lesions that will be scored for rickets and the lesions scored for scurvy can be found in table 2. For this thesis, only macroscopically visible lesions are considered, and microscopic (dentine) lesions are excluded from the scoring sheets for the sake of time.

Furthermore, table 3 shows which lesions are considered to be diagnostic, typical, or consistent with rickets and scurvy according to Brickley and Morgan (2023) and the work of Schattmann et al. (2016); in their paper, Schattmann et al. (2016) provide diagnostic values to each lesion identifiable for rickets and/or scurvy. The authors have divided the lesions into categories ‘probable’, ‘possible’ and ‘non-diagnostic’ in their article (Schattmann et al., 2016, p.66,67). To keep consistent with the Brickley and Morgan (2023) paper on assessing diagnostic certainty, this thesis uses the terms ‘diagnostic’ instead of ‘probable’, ‘highly consistent’ instead of ‘possible’ and ‘consistent’ instead of ‘non-diagnostic’.

Lesions for Rickets	Lesions for Scurvy
Cranial vault porosity (result of impaired/defective mineralization).	Sphenoid, external surface, greater wing (GWS).
Orbital roof porosity (result of impaired/defective mineralization).	Zygomatic bone, medial surface/posterior-medial surface of maxillary zygomatic process.
Cranial vault thickening (occurs when sufficient vitamin D becomes available again, seen in healed cases of rickets).	Posterior surface of maxillae.
Deformed mandibular ramus (a biomechanical deformity).	Coronoid process of mandible, medial surface.
Rib bending deformity (biomechanical).	Inferior surface, palatine processes of maxillae.
Costochondral rib flaring (combination of impaired mineralization and biomechanical deformity). *	Maxillary bone and/or alveolar bone.
Costochondral rib porosity (result of impaired/defective mineralization).	Orbital walls.
Ilium concavity (biomechanical deformity).	Cranial vault.
Upper limb long bones bending deformity (biomechanical deformity).	Infraorbital foramen of maxillae.
Lower limb long bones bending deformity (biomechanical).	Foramen rotundum of sphenoid bone.
Long bone metaphyseal flaring/cupping of ends. *	Long bones.
Superior flattening femoral metaphysis.	Scapulae: supra- and infraspinous fossa.
Coxa vara.	Ilia.

*Table 1. Macroscopic lesions for rickets and scurvy. *= Also reported in scurvy, although not specifically scored for in this thesis. For all scurvy lesion sites, porosity or subperiosteal new bone formation (SPNB) is expected. Table is based on table 1 and 2 in the paper by Brickley and Morgan (2023), p.639 and p. 640.*

3.2.3 Assessing rickets and scurvy

For the assessment of the lesions that were macroscopically scored, a division was made of the relative 'hierarchy' of lesions that could have been possibly scored. The lesions were divided into three diagnostic categories, based on Appleby et al. (2015) and Brickley and Morgan (2023): 'Diagnostic of', 'Highly consistent with/typical of', and 'Consistent with'. To assess which individuals could be put in each category, a definition for each category was defined based on the work by Harper et al. (2011). In this paper, a scoring system for the diagnosing of syphilis and treponemal disease was devised. Their framework was based on a scoring system, scoring from 5-0 based on the severity and frequency of lesions, shown in table 4. As for the assessing of rickets and scurvy, the categories by Harper et al. (2011) were redefined to fit the diagnostic categories presented and

proposed by Brickley and Morgan (2023): “Diagnostic of”, “Highly Consistent with/Typical of”, “Consistent with” and “Not Consistent with”.

Diagnostic lesions for rickets	Highly consistent lesions for rickets	Consistent lesions for rickets	Diagnostic lesions for scurvy	Highly consistent lesions for scurvy	Consistent lesions for scurvy
Costochondral rib flaring	Cranial vault porosity	Orbital roof porosity	Porosity or SPNB (bilateral) on greater wing of sphenoid bone (GWS)	Porosity or SPNB on foramen rotundum sphenoid bone	Porosity or SPNB on orbital walls
Long bone metaphyseal flaring	Costochondral rib porosity	Coxa vara	Porosity or SPNB on posterior surface of the maxillae	Porosity or SPNB on zygomatic bone	Porosity or SPNB on maxillary and/or alveolar bone
Deformed mandibular ramus	Bending deformity long bones upper limb	Superior flattening femoral head	Porosity or SPNB on the scapula; supraspinous and/or infraspinous	Porosity or SPNB on medial coronoid process of the mandible	Porosity or SPNB on long bones
Rib bending deformity	Bending deformity long bones lower limb			Porosity or SPNB on infraorbital foramen	Porosity or SPNB on inferior surface of the palatine process of the maxillae
Cranial vault thickening	Ilium concavity				Porosity or SPNB on ilium

Table 3. Lesions for scurvy and rickets divided based on the diagnostic values given by Schattmann et al. (2016), p.66 and p.67.

For rickets, an individual falls in the “Diagnostic of” category when the presented lesions are specific for rickets, found on multiple skeletal elements or in the presence of lesions suggestive of rickets disease on other skeletal elements. In other words, an individual needs to present more than three diagnostic lesions or one diagnostic lesion in the presence of at least two lesions of the ‘highly consistent’ category. An individual will fall in the “Highly consistent with/Typical of” category when they present lesion(s) specific to rickets on a single skeletal element and/or lesions suggestive of rickets on multiple skeletal elements. This means that the criteria for the “Highly consistent with/Typical of” category are one diagnostic lesion and/or at least three lesions from the ‘highly consistent’ category. The “Consistent with” category will consist of individuals presenting lesions suggestive of rickets on multiple skeletal elements, i.e. at least three lesions falling in the ‘consistent’ category, and no lesions from higher categories. Lastly, individuals will be assessed as “Not consistent with” when the lesions presented are not consistent with rickets disease processes, and present less than three lesions of the ‘consistent’ category and no other lesions from higher categories.

For scurvy, an individual will be assessed as “Diagnostic of” when they present lesion(s) pathognomonic for scurvy, i.e. bilateral porosity or SPNB on the greater wing of the sphenoid bone (GWS) or lesions specific to scurvy in the presence of lesions suggestive of scurvy on multiple skeletal elements. In other words, individuals need to present the GWS-lesion or one diagnostic lesion with at least three lesions of the ‘highly consistent’ category. Individuals comprising the “Highly consistent with/Typical of” category need to present lesion(s) specific to scurvy on a single skeletal element and/or lesions suggestive of scurvy disease on multiple skeletal elements. This means one diagnostic lesion that is not the GWS-lesion and/or in the presence of at least three lesions from the ‘highly consistent’ and/or ‘consistent’ category. An individual falls in the “Consistent with” category when the lesions presented are suggestive of scurvy disease, found on multiple skeletal elements; at least three lesions of the ‘consistent’ category, and no lesions from higher categories. Finally, an individual will be diagnosed as “Not consistent with”, if the lesions presented are not consistent with scurvy disease processes; less than three lesions from the ‘consistent’ category, and no lesions from higher categories.

In the event it is not possible to score lesions because bones are not preserved, it is not possible to diagnose an individual with enough certainty. Therefore, an extra category “Unobservable” was added, comprised of all individuals without long bones and ribs (for rickets), or less than 3 cranial bones (for scurvy).

Definition	Score
Lesions specific to the disease found on multiple skeletal elements, or in the presence of lesions suggestive of the disease on other skeletal elements.	5
Lesions specific to the disease on a single element.	4
Lesions suggestive of the disease on multiple skeletal elements.	3
Lesions suggestive of the disease on a single element.	2
Lesions consistent with the disease on one or more skeletal elements.	1
Lesions not consistent with the disease process under study.	0

Table 4. Framework of diagnostic categories based on Harper et al. (2011), p.119.

3.2.4 Co-occurrence assessment

As discussed in the previous section, there are five categories in which an individual could be diagnosed. To ascertain which individuals had both diseases, the outcomes for both diseases were assessed and accumulated, based on the different combinations shown in table 5. These outcomes will then be cross-referenced with the prevalence of the scapula sign in the sample.

Rickets diagnostic	Scurvy diagnostic	Disease Status
Diagnostic of Rickets	Diagnostic of Scurvy	Both
Diagnostic of Rickets	Highly Consistent/Typical Scurvy	Rickets and possible Scurvy
Diagnostic of Rickets	Consistent with Scurvy	Rickets and maybe Scurvy
Diagnostic of Rickets	Not Consistent with Scurvy	Rickets
Diagnostic of Rickets	Unobservable	Rickets, Scurvy Unobservable
Highly Consistent/Typical Rickets	Diagnostic of Scurvy	Scurvy and possible Rickets
Highly Consistent/Typical Rickets	Highly Consistent/Typical Scurvy	Possibly both
Highly Consistent/Typical Rickets	Consistent with Scurvy	Possible Rickets, maybe Scurvy
Highly Consistent/Typical Rickets	Not Consistent with Scurvy	Possible Rickets
Highly Consistent/Typical Rickets	Unobservable	Possible Rickets, Scurvy Unobservable
Consistent with Rickets	Diagnostic of Scurvy	Scurvy and maybe Rickets
Consistent with Rickets	Highly Consistent/Typical Scurvy	Possible Scurvy, maybe Rickets
Consistent with Rickets	Consistent with Scurvy	Maybe both
Consistent with Rickets	Not Consistent with Scurvy	Maybe Rickets
Consistent with Rickets	Unobservable	Maybe Rickets, Scurvy Unobservable
Not Consistent with Rickets	Diagnostic of Scurvy	Scurvy
Not Consistent with Rickets	Highly Consistent/Typical Scurvy	Possible Scurvy
Not Consistent with Rickets	Consistent with Scurvy	Maybe Scurvy
Not Consistent with Rickets	Not Consistent with Scurvy	Neither
Not Consistent with Rickets	Unobservable	No Rickets, Scurvy Unobservable
Unobservable	Diagnostic of Scurvy	Scurvy, Rickets Unobservable
Unobservable	Highly Consistent with/Typical Scurvy	Possible Scurvy, Rickets Unobservable
Unobservable	Consistent with Scurvy	Maybe Scurvy, Rickets Unobservable
Unobservable	Not Consistent with Scurvy	No Scurvy, Rickets Unobservable
Unobservable	Unobservable	Both diseases unobservable

Table 5. Possible combinations of diagnostic categories for both scurvy and rickets, and the "concluded" Disease Status, for tracking the prevalence of co-occurrence in the sample.

3.2.5 Assessment of the scapula sign

Although the article by Ives, Swan, and Humphrey (2023) poses a new criterium for rickets, the assessment of the criterium itself is not very well described. In their article, the authors mention five, arguably quite broad, criteria: coarseness, blunting, concavity/cupping, slit-strut morphology, and porosity. However, it is not

described when a scapula sign is indeed a scapula sign. For this reason and for the purpose of this thesis, the criteria were scored on ‘present’, ‘absent’, and ‘unobservable’. The presence of the scapula sign will be indicated by if there are three or more criteria scored as present. When not all five criteria were able to be assessed and the final score ended in a “draw”, a ‘possible’ was attributed to the scapula in question. Additionally, a scapula sign is deemed ‘unobservable’ when an individual does not have both scapulae but for one scapula, the scapula sign is deemed absent. In this case, it is considered that the scapula sign possibly could not have been observed due to the lack of preservation. On the contrary, when only one scapula is present and the scapula sign is present, the individual is recorded as having a scapula sign. Furthermore, combinations of the outcomes of both scapulae were made to cross reference them with the disease status.

3.2.6 Radiographic assessment

Following the research of Ives, Swan, and Humphrey (2023), additional radiographic images of all scapulae were made to follow the argument of Ives, Swan, and Humphrey that lesions of the scapula sign would be more visible in radiographs, especially the mineralization and slit-strut morphology of the growth plates (Ives, Swan & Humphrey, 2023, p.61). Furthermore, Schattmann et al. (2016) and Brickley and Morgan (2023) argue the added value of radiographic imaging in diagnosing the presence of rickets and scurvy – in the case of this thesis this is especially relevant for rickets, as most scorbutic lesions observable on radiographs are lesions occurring in adult cases of scurvy, and not as much for growing individuals (Schattmann et al., 2016, p.64; Brickley & Morgan, 2023, p.639).

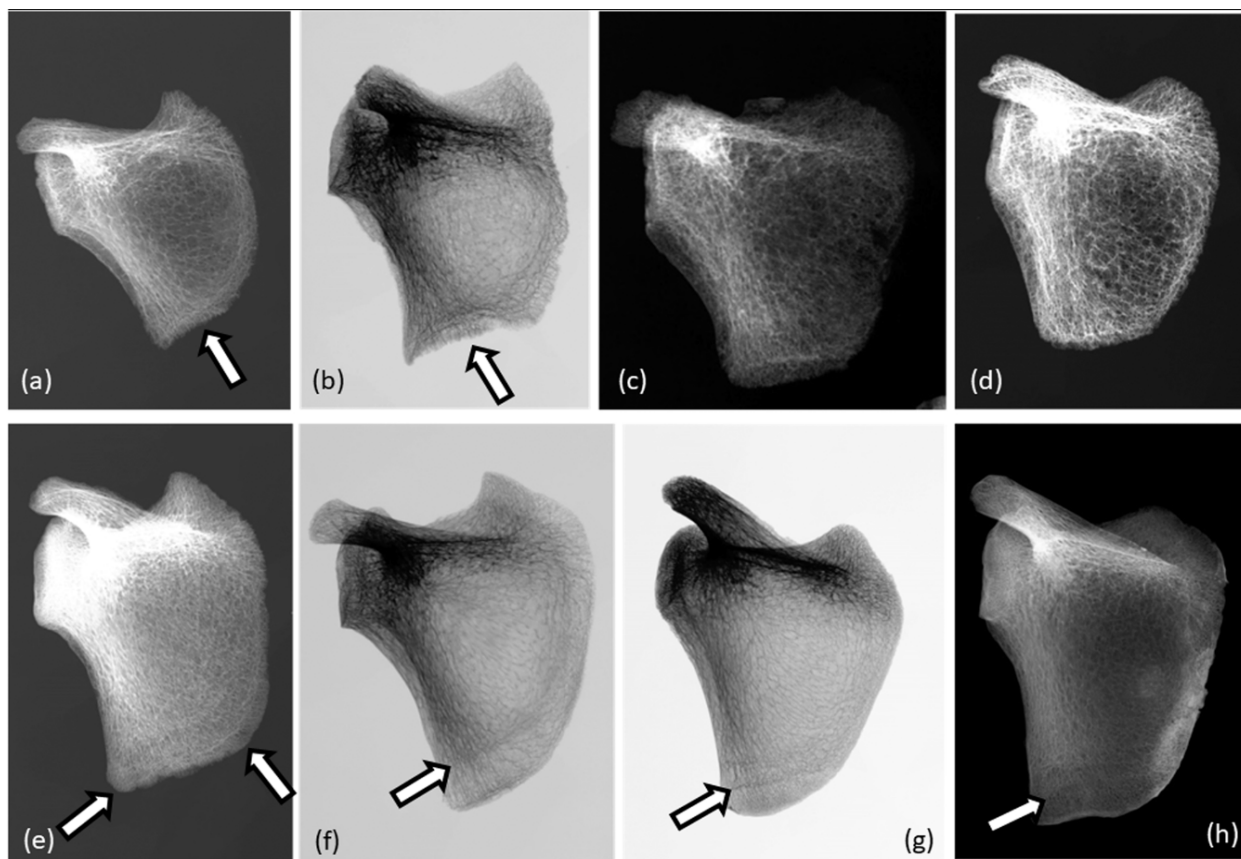


8. Image of "normal" shape of inferior angle, with a solid bone margin. Credit: Ives, Swan & Humphrey (2023), p. 60. No other credit mentioned in original paper.

If the scapula sign is visible on radiographs, it is expected that growth lines demarking a distinction between poorly mineralized bone and sufficiently mineralized bone will be observable. The radiographic images provided in the paper of Ives, Humphrey, and Swan (2023) were taken for reference to compare the scapulae in the Middenbeemster sample with scapulae from their sample that they identified as having a scapula sign.

Figure 9 shows eight scapulae identified by Ives, Humphrey, and Swan (2023), and figure 8 shows a scapula from their sample with a normal inferior angle. This image is also used as a base reference for this thesis. The scapula signs in this sample are registered with a ‘no’, ‘yes’ or ‘possible both’ if the scapula sign is bilaterally absent, present, or possible, and by a ‘no’, ‘yes L/R’ or ‘possible L/R’ if unilaterally observed.

The X-ray machine used for this thesis is an Aribex Nomad Dental X-ray machine, with 70kv/1,5mA. All radiographs were taken with an exposure time of 0,4 seconds at the Human Osteoarchaeology Lab in Leiden under supervision by faculty lab assistant M.L. Irving-Langevoort.



9. The scapulae of 8 individuals from the study by Ives, Swan, and Humphrey (2023), p.63. For the identification of the individuals, and credit for the images, the original caption is enclosed as well: “Examples of the pathological changes observed at the inferior angle of the scapula in this sample. Clearly identifiable examples of blunting, flattening, or squaring of the inferior angle in active rickets (Fig. 2a Individual NHMUK PA UNREG 3702 CAS84 2876; 2b Individual NHMUK PA UNREG 3599 CAS84 2772; 2c. Individual PGV10 2520; 2d Individual NHMUK PA UNREG 3282 CAS84 2440; 2e Individual NHMUK PA UNREG 3371 CAS84 2532). A cupping deformity causing compression and concavity of the inferior angle was identified macroscopically, and the extent of the compression was clearly visible in radiographs (Fig. 2a, b). Coarsening and loss of definition of the normal border was identified (Fig. 2a, b,c,d). Increased medio-lateral breadths were also identified at the inferior angle in some active cases (e.g. see arrows showing flattening and wide breadth of the inferior angle Fig. 2e). Growth arrest lines demonstrating the outline of former defects were observed in juveniles with evidence of healed rickets are shown with arrows (Fig. 2f Individual NHMUK PA UNREG 3562 CAS84 2734; 2g Individual NHMUKPA UNREG 3283 CAS84 2441; 2h Individual NHMUK PA UNREG 3069 CAS84 2206) and one possible case of cyclical phases of rickets (Fig. 2e). Copyright Trustees of the Natural History Museum.”

Chapter 4: Results

4.1 Sample demography

The sample taken from the Middenbeemster population consisted of 47 individuals, with the inferior angle of one or two scapulae still intact. As discussed in section 3.1.2., the age groups for the individuals were classified as follows: the infants range between 0-2 years, children are grouped between 2-5 years, and individuals between 5-12 years are seen as juveniles. Within the sample, there are 11 children, 21 infants and 15 juveniles. Of the infants, 71% is younger than 6 months, and 19% are 1.5 years or older. The mean age at death of the infants is 7,25 months old. Furthermore, 36% of the children are 3 +/- 1 years old, 27% of the children are 3.5 +/- 1 years old, 9% are 3 +/- 0.5 years old, 9% 4.5 +/- 0.5 years old, 9% 4 +/- 1.5 years old, and 9% 4 +/- 1 years old. The mean age at death among the children in the sample is 3,5 years old. The age distribution in the juvenile group ranges from 5 +/- 2 years old (20%), 7.5 +/- 2 years old (13%), 8 years old (20%), 10 years old (13%), and 27% is above 10 years old. The mean age at death for the juveniles is 8,5 years old. For the detailed age distribution, see Appendix 1.

4.2 Rickets

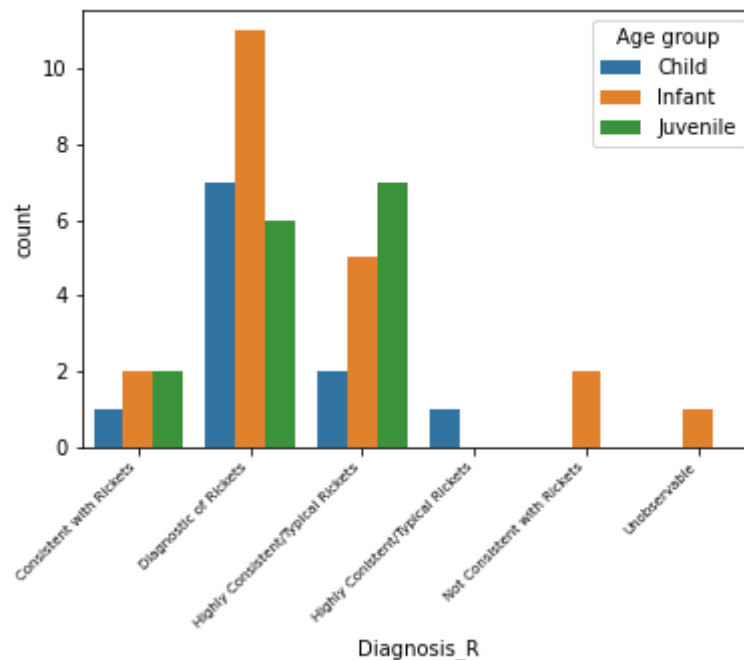
Based on the diagnostic categories as defined in section 3.2.3, 24 of all individuals were categorized as “Diagnostic of Rickets”, 15 were concluded to have lesions “Highly Consistent with/Typical of Rickets”, 5 individuals presented lesions “Consistent with Rickets”, 2 individuals were deemed “Not Consistent with Rickets”, and for one individual, it was “Unobservable” to determine rickets, as this individual missed their long bones from both upper as well as their lower limbs (fig.10). See also Appendix 2 for all scored individuals.

The age of at death of the individuals in the infant group varies between 3 weeks +/- 1mo old and 2.5 years +/- 0.5yr old. The distribution of rickets in the infant group shows that 11 individuals were presenting lesions “Diagnostic of Rickets”, whereas 5 presented lesions “Highly Consistent with/Typical of Rickets”, 2 “Consistent with Rickets” and 2 “Not Consistent with Rickets”. Only one individual was deemed “Unobservable”, as their long bones were not preserved.

The age at death of the individuals in the children group varies between 3 years +/- 0.5yr and 4.5 years +/- 0.5yr old. Of all children, 7 were labeled as “Diagnostic of Rickets”, 3 were “Highly Consistent with/Typical of Rickets” and 1 was deemed “Consistent with Rickets”. None of the children in the sample were “Not Consistent with Rickets” or “Unobservable”.

Finally, the age at death in the juvenile group varies between 5 years +/- 2yr and 12 years +/- 1yr old. Within the juvenile group, 6 presented lesions “Diagnostic of Rickets”, and 7 presented lesions “Highly Consistent with/Typical of Rickets”. Only 2 individuals were scored for lesions “Consistent with Rickets”. There were no individuals categorized as “Not Consistent with Rickets” or “Unobservable”.

In conclusion, the category “Diagnostic of Rickets” was comprised by 7 children, 11 infants and 6 juveniles. The category “Highly Consistent with/Typical of Rickets” was comprised by 3 children, 5 infants and 7 juveniles. Additionally, the category “Consistent with Rickets” consists of 1 child, 2 infants and 2 juveniles. The category “Not Consistent with Rickets” consists of 2 infants, and no children or juveniles. Furthermore, the category “Unobservable” for rickets consists of 1 infant, and no children or juveniles.



10. Graph of diagnostic categories for rickets per age group.

4.3 Scurvy

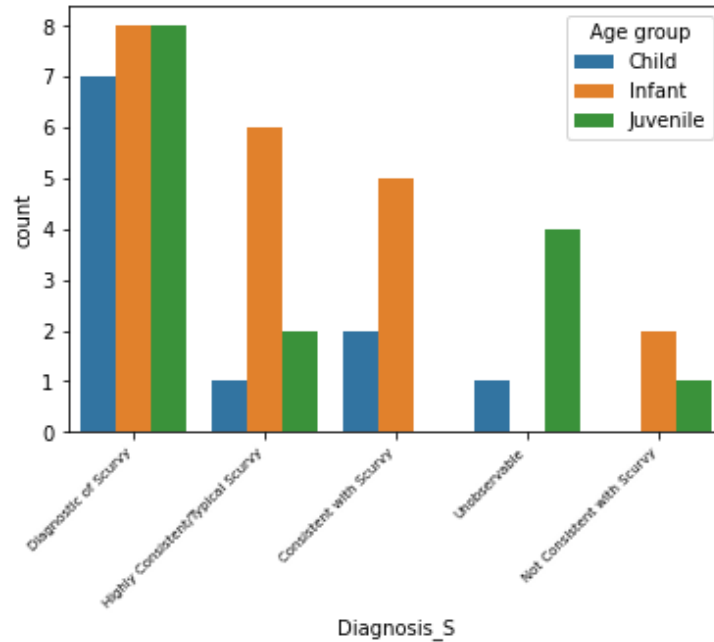
As for diagnosing scurvy, within the whole sample population, 23 individuals had lesions “Diagnostic of Scurvy”. Furthermore, 9 individuals presented lesions “Highly Consistent with/Typical of Scurvy”, 7 individuals were categorized as having lesions “Consistent with Scurvy”, and 3 individuals were deemed “Not Consistent with Scurvy”. Of the sample, 5 individuals are categorized as “Unobservable”, as they all had less than three cranial bones preserved (fig.11). See also Appendix 3 for all scored individuals.

For the infant group, 8 individuals presented lesions “Diagnostic of Scurvy”, 6 individuals showed lesions “Highly Consistent with/Typical of Scurvy”, and 5 were categorized as “Consistent with Scurvy”. Finally, 2 individuals were categorized as “Not Consistent with Scurvy”. There were no individuals deemed “Unobservable”.

Out of the children in the sample, 7 presented lesions “Diagnostic of Scurvy”, and 1 was scored for “Highly Consistent with/Typical of Scurvy”. Furthermore, 2 individuals were categorized as being “Consistent with Scurvy”. None of the individuals was categorized as “Not Consistent with Scurvy”, and 1 was deemed “Unobservable”.

Of the juveniles, 8 individuals presented lesions “Diagnostic of Scurvy”, and 2 individuals were categorized as having lesions “Highly Consistent with/Typical of Scurvy”. None of the juveniles were categorized as “Consistent with Scurvy”. There was 1 individual categorized as “Not Consistent with Scurvy”, and 4 individuals were deemed “Unobservable”.

In conclusion, the category “Diagnostic of Scurvy” was comprised by 7 children, 8 infants and 8 juveniles. The category “Highly Consistent with/Typical of Scurvy” consists of 1 child, 6 infants and 2 juveniles. Furthermore, the category “Consistent with Scurvy” was comprised of 2 children, 5 infants and no juveniles. The category “Not Consistent with Scurvy” consists of 2 infants, 1 juvenile and no children. Finally, the category “Unobservable” for scurvy was comprised of 1 child, 4 juveniles and no infants.



11. Graph of diagnostic categories for scurvy per age group.

4.4 Co-occurrence

To assess which individuals of the sample presented lesions of both rickets and scurvy, combinations of the diagnostic categories for both diseases were made, shown in table 5 (chapter 3). In the whole sample, 14 individuals presented lesions that were diagnostic for both rickets and scurvy and were therefore classified as “Both”. Furthermore, 5 individuals were categorized as having scurvy and presenting lesions highly consistent with rickets, classifying as “Scurvy and possible Rickets”. Another 4 individuals were diagnosed as having scurvy and presenting lesions consistent with rickets, in the category “Scurvy and maybe Rickets”. The disease status “Rickets and possible Scurvy” (“Diagnostic of Rickets” + “Highly Consistent/Typical Scurvy”) occurred also in 4 individuals. The same goes for the category “Rickets and maybe Scurvy”, indicating that 4 individuals were “Diagnostic of Rickets” and had lesions “Consistent with Scurvy”. Additionally, 4 individuals had lesions for “Possible Rickets, Scurvy Unobservable” (“Highly Consistent with Rickets” + “Unobservable Scurvy”). Only 3 individuals were “Highly Consistent with/Typical of Rickets” + “Consistent with Scurvy”: categorizing as “Possible Rickets, maybe Scurvy”. Moreover, 1 individual was categorized as “Diagnostic of Rickets” + “Unobservable Scurvy” (i.e. category “Rickets, Scurvy Unobservable”), and 1 as having lesions for “Neither”

(“Not Consistent with Rickets” + “Consistent with Scurvy”). The category “Possible Scurvy”, i.e. “Highly Consistent with/Typical of Scurvy” + “Not Consistent with Rickets” was also only found in 1 individual, as well as “Rickets” (“Diagnostic of Rickets” + “Not Consistent with Scurvy”), and “Maybe Rickets” (“Consistent with Rickets” + “Not Consistent with Scurvy”) (tab.6). All other combinations of diagnostic categories were not found in this sample.

The infant group is comprised of a wider variety of categories: 4 individuals were classified as having lesions for “Both”. The categories “Rickets and maybe Scurvy”, “Rickets and possible Scurvy” were found in 3 individuals each. The categories “Scurvy and possible Rickets”, “Possibly only Rickets” and “Scurvy and maybe Rickets” were all found in 2 individuals per category. For the disease statuses “Neither”, “Possible Scurvy”, “Rickets”, “Possibly both” and “Possible Scurvy, Rickets Unobservable”, 1 individual per category was found. No other combinations were detected for the infant group.

In the children group of the sample, 4 individuals were categorized as “Both”, and 2 individuals were put in the category “Scurvy and possible Rickets”. Furthermore, the categories “Scurvy and maybe Rickets”, “Rickets and possible Scurvy”, “Rickets and maybe Scurvy”, “Diagnostic of Rickets, Scurvy Unobservable” and “Possibly only Rickets” were all found for 1 individual each. Other categories were not found.

Finally, in the juvenile group, 6 individuals were deemed to have lesions diagnostic of rickets and of scurvy, categorized as “Both”. Another 4 individuals classified as “Possible Rickets, Scurvy Unobservable”. The category “Possibly both” was found in 2 individuals, and the for the categories “Scurvy and maybe Rickets”, “Maybe Rickets” and “Scurvy and possible Rickets”, 1 individual per category was found. No other combinations were detected.

The combinations for disease statuses “Maybe both”, “Maybe Rickets, Scurvy Unobservable”, “Scurvy”, “Possibly only Scurvy”, “Maybe Scurvy”, “No Rickets, Scurvy Unobservable”, “Scurvy, Rickets Unobservable”, “Maybe Scurvy, Rickets Unobservable”, “No Scurvy, Rickets Unobservable”, “Possible Rickets” and “Both diseases Unobservable” were not found in the entire sample. For the sake of relevance, these categories will not be discussed further in this thesis.

Categories co-occurrence	Counts
Both	14
Scurvy and possible Rickets	5
Scurvy and maybe Rickets	4
Rickets and possible Scurvy	4
Rickets and maybe Scurvy	4
Possible Rickets, Scurvy Unobservable	4
Possible Rickets, maybe Scurvy	3
Possibly both	3
Diagnostic of Rickets, Scurvy Unobservable	1
Neither	1
Possible Scurvy	1
Rickets	1
Possible Scurvy	1
Rickets	1
Possible Scurvy, Rickets Unobservable	1
Maybe Rickets	1

Table 6. Categories of co-occurrence found in the sample for this thesis and their prevalence.

4.5 The scapula sign

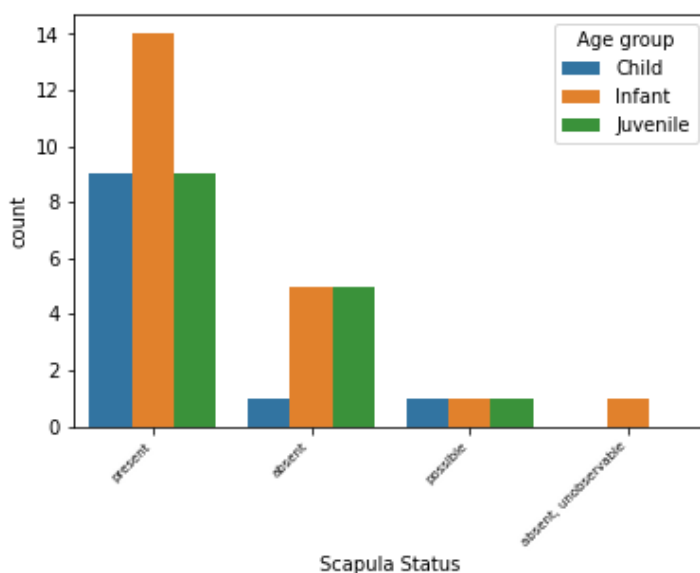
As discussed in chapter 3, the scapula sign was macroscopically assessed based on five criteria: blunting, concavity/cupping, porosity, coarsening and slit-strut morphology. The presence of three or more criteria resulted in a present scapula sign. As mentioned in section 3.2.5., the scapula sign was deemed present in an individual if one or both scapulae presented three or more lesions. Within the whole sample (n=47), there were 40 left, and 41 right scapulae with its inferior angle intact: 34 of all individuals had both scapulae, 6 individuals only had a right scapula, and 7 individuals only had a left scapula. Of all left scapulae, 16 scapulae were deemed of having a scapula sign. Also, 3 scapulae were categorized as having a possible scapula sign; this means that some criteria were present, but others unobservable; see also Appendix 4. Furthermore, 21 scapulae did not have the scapula sign. As for all right scapulae, 22 had a present scapula sign, 3 had a possible scapula sign and 16 scapulae did not have the scapula sign (fig.12).

As for the infants, 14 individuals had both scapulae intact, 4 individuals had only a left scapula and for 3 individuals only their right scapula was preserved. The scapula sign was found in 14 of the individuals. Also, 5 individuals did not show a scapula sign. Only one individual showed an ‘absent and unobservable’ scapula sign, meaning this individual only had one scapula, and the presence of the sign could not be scored because their other scapula could not be analyzed.

Of the children in the sample, 7 individuals had both scapulae intact, 2 individuals only had a left scapula, and 2 individuals had a right scapula. The scapula sign was found in 9 of the individuals, and 1 had a possible scapula sign. Additionally, 1 individual had no scapula sign.

In the juvenile group, 13 individuals had both scapulae intact, 4 individuals had only their left scapula, and 3 individuals only had their right scapula preserved. The scapula sign was found in 9 individuals. Another 5 juveniles presented no scapula sign, and 1 presented a possible scapula sign.

In conclusion, a present scapula sign was found in 9 children, 14 infants, and 9 juveniles. The scapula sign was absent in 1 child, 5 infants and 5 juveniles. A possible scapula sign was found in 1 child, 1 infant and 1 juvenile. An unobservable scapula sign was found in 1 infant, and not in other age groups.



12. Graph of distribution of scapula sign per age group.

4.5.2 The scapula sign and rickets

In this section, it will be discussed for which rickets diagnostic category the scapula sign is present or absent. For the individuals in the category “Diagnostic of Rickets” (n=24), 17 individuals had a present scapula sign, and 6 individuals did not have a scapula sign. Only one individual had an unobservable scapula sign. Of the individuals in the category “Highly Consistent with/Typical of Rickets” (n=15), 11 individuals had a present scapula sign, and 1 individual had no scapula sign. The remaining 3 individuals all had a possible scapula sign.

Furthermore, in category “Consistent with Rickets” (n=5), 1 individual had a present scapula sign, and 4 individuals did not have scapula sign. Lastly, in the category “Not Consistent with Rickets” (n=2), 2 individuals had a present scapula sign, and in the “Unobservable” (n=1) category, the one individual had a present scapula sign (fig.13).

The infant group (n=21) showed to have 7 individuals with a present scapula sign in the category “Diagnostic for Rickets”, 3 individuals had no scapula sign, and 1 individual had an unobservable scapula sign. For the category “Highly Consistent with/Typical of Rickets”, 4 infants showed a present scapula sign, and 1 individual had a possible scapula sign. Furthermore, the 2 individuals categorized as “Consistent with Rickets” did not present a scapula sign. In the category “Not Consistent with Rickets”, 2 individuals had a scapula sign. Lastly, the infant categorized as “Unobservable” for rickets showed a present scapula sign.

Within the children group (n=11) of the sample, 6 individuals in the “Diagnostic of Rickets” category had a present scapula sign, and 1 individual had no scapula sign. In the category “Highly Consistent with/Typical of Rickets”, 2 children presented a scapula sign, and 1 individual had a possible scapula sign. For the categories “Not Consistent with Rickets” and “Unobservable”, no children were categorized and therefore no scapula signs recorded.

As for the final group, the juveniles (n=15), 4 individuals in the category “Diagnostic of Rickets” showed a present scapula sign, and 2 individuals did not show a scapula sign. In the category “Highly Consistent with/Typical of Rickets”, 5 individuals had a scapula sign, 1 individual had a possible scapula sign, and 1 individual did not show a scapula sign. For the 2 juveniles classified as “Consistent with Rickets”, they did not show a scapula sign. For the categories “Not consistent with Rickets” and “Unobservable”, no juveniles were categorized and therefore no scapula signs recorded.

4.5.3. The scapula sign and scurvy

To assess the prevalence of the scapula sign in individuals scored for scurvy, the results will be discussed in this section. Of the whole sample group, 23 individuals were categorized as “Diagnostic of Scurvy”. Of those individuals, 12 showed a scapula sign, and 2 showed a possible scapula sign. Additionally, 8 individuals in the

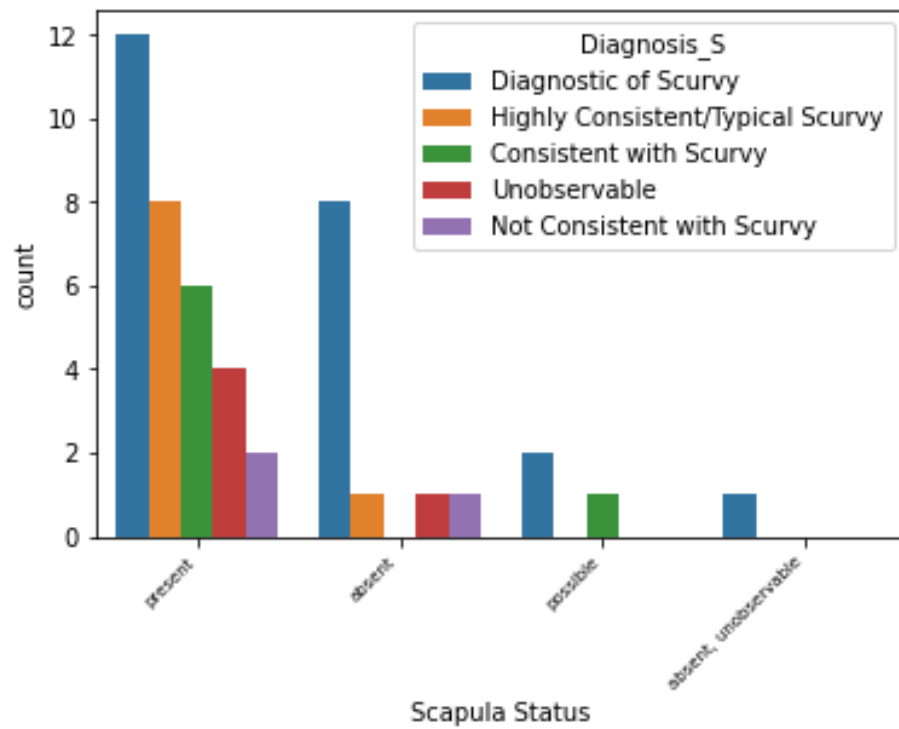
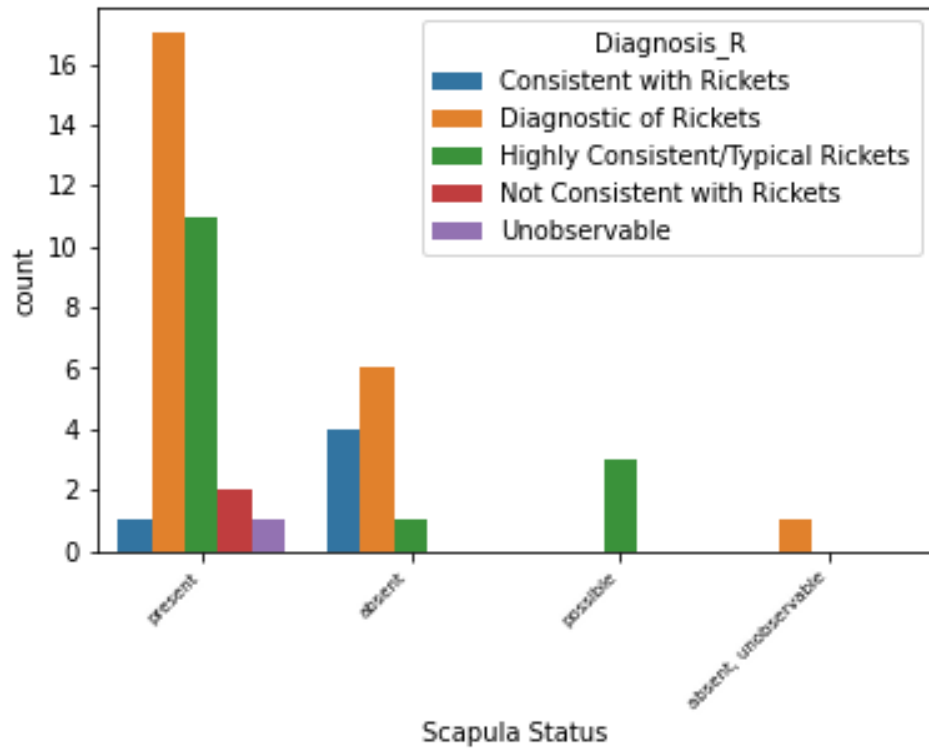
“Diagnostic of Scurvy” category did not show a scapula sign, and for 1 individual, the scapula sign was unobservable. Of 9 the individuals classified as “Highly Consistent with/Typical of Scurvy”, 8 had a scapula sign, and 1 did not show a scapula sign. For the 7 individuals categorized in “Consistent with Scurvy”, 6 had a scapula sign, and 1 had a possible scapula sign. The 3 individuals in the category “Not Consistent with Scurvy” showed that 2 had a scapula sign, and 1 did not have a scapula sign. For the 5 individuals in the category “Unobservable”, 4 showed a scapula sign and 1 individual did not have a scapula sign (fig.14).

Within the infant group, for the 8 infants classified as “Diagnostic of Scurvy”, 3 showed a scapula sign, 4 individuals did not show a scapula sign, and 1 individual had an unobservable scapula sign. Of the 6 individuals categorized as “Highly Consistent with/Typical of Scurvy”, 5 individuals presented with a scapula sign and 1 did not have a scapula sign. For the 5 individuals in the “Consistent with Scurvy” category, 4 showed a scapula sign, and 1 showed a possible scapula sign. Finally, in the category “Not Consistent with Scurvy” (n=2), both individuals showed presence of a scapula sign. For the category “Unobservable” for scurvy, no infants were categorized and therefore no scapula signs recorded.

For the children group of the sample, in the category “Diagnostic of Scurvy”, 5 individuals showed a scapula sign, 1 individual had a possible scapula sign, and 1 individual did not present a scapula sign. Furthermore more, the only child categorized as “Highly Consistent with Scurvy” showed a scapula sign, and only one child was classified as “Unobservable” for scurvy and showed a scapula sign. For the category “Not Consistent with Scurvy”, no children were categorized and therefore no scapula signs recorded.

In the juvenile group, of the 8 individuals categorized as “Diagnostic for Scurvy”, 4 showed a scapula sign, 1 showed a possible scapula sign, and 3 individuals did not show a scapula sign. Of the 2 individuals classified as “Highly Consistent with/Typical of Scurvy”, both showed a scapula sign. The single juvenile that was categorized as “Not Consistent for Scurvy” did not present a scapula sign. Finally, in the category “Unobservable” for scurvy (n=4), 3 individuals presented a scapula sign, and 1 individual did not have a scapula sign. For the category “Consistent with Scurvy” no juveniles were categorized and therefore no scapula signs recorded.

13. Visualisation of distribution of scapula sign per diagnostic category for rickets.



14. Visualisation of distribution of scapula sign per diagnostic category for scurvy.

4.5.4. The scapula sign and co-occurrence of rickets and scurvy

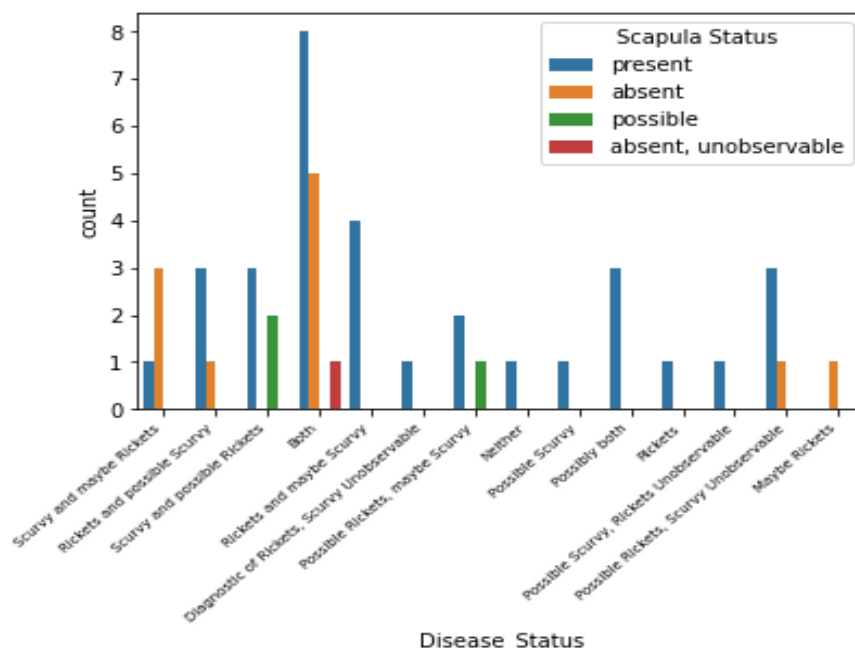
To assess whether the scapula sign might have a possible correlation for the co-occurrence of rickets and scurvy, it was analyzed which disease status category showed present or absent scapula signs (fig.15). The individuals in the whole sample were divided into fourteen categories, based on the lesions they presented. The categories are shown in table 6. Of the individuals categorized as “Both” (n=14), 8 individuals showed a present scapula sign, 5 individuals did not have a scapula sign and 1 individual had a (possibly) unobservable scapula sign. For the individuals in the category “Scurvy and possible Rickets” (n=5), 3 individuals showed a scapula sign, and 2 individuals showed a possible scapula sign. Between the individuals in the category “Rickets and possible Scurvy” (n=4), 3 individuals presented a scapula sign, and 1 individual did not have a scapula sign. Furthermore, all 4 individuals classified as “Rickets and maybe Scurvy” presented a scapula sign. For the category “Scurvy and maybe Rickets” (n=4), 1 individual showed a scapula sign, and 3 individuals did not have a scapula sign. Of the 4 individuals in the category “Possible Rickets, Scurvy Unobservable”, 3 individuals showed a scapula sign, and 1 individual did not have a scapula sign. Between the individuals in category “Possibly only Rickets” (n=3), 2 individuals had a scapula sign, and 1 individual had a possible scapula sign. Of the category “Possibly both”, all 3 individuals presented a scapula sign. All individuals in categories “Diagnostic of Rickets, Scurvy Unobservable” (n=1), “Neither” (n=1), “Possibly only Scurvy” (n=1), “Possible Scurvy, Rickets Unobservable” (n=1), “Rickets” (n=1) presented a scapula sign. Lastly, the individual classified as “Maybe Rickets” did not show a scapula sign.

Of the infants classified as “Both” (n=4), 1 individual presented a scapula sign, 2 individuals did not present a scapula sign and 1 individual presented an unobservable scapula sign. Of the category “Rickets and maybe Scurvy” (n=3), all individuals presented a scapula sign. In the category “Rickets and possible Scurvy” (n=3), 2 individuals presented a scapula sign, and 1 did not have a scapula sign. Both infants classified as “Scurvy and possible Rickets” had a scapula sign, whereas both infants classified as “Scurvy and maybe Rickets” did not have a scapula sign. Between the individuals in the category “Possibly only Rickets” (n=2), 1 individual had a scapula sign, and 1 individual had a possible scapula sign. All infants in categories “Neither” (n=1), “Possible Scurvy” (n=1), “Possible Scurvy, Rickets Unobservable” (n=1), “Possibly both” (n=1) and “Rickets” (n=1)

presented a scapula sign. There were no other categories recognized in this age group and therefore no other scapula signs recorded.

From the children group, 4 individuals classified as “Both”, 3 of which presenting a scapula sign and 1 individual not presenting a scapula sign. Of the children categorized as “Scurvy and possible Rickets” (n=2), 1 individual had a scapula sign, and 1 individual had a possible scapula sign. Finally, all the individuals in the remaining categories, “Rickets, Scurvy Unobservable” (n=1), “Possibly only Rickets” (n=1), “Rickets and maybe Scurvy” (n=1), “Rickets and possible Scurvy” (n=1), “Scurvy and maybe Rickets” (n=1) presented a scapula sign. There were no other categories recognized in this age group and therefore no other scapula signs recorded.

In the last group, of the juveniles classified as “Both” (n=6), 4 individuals showed a scapula sign and 2 did not show a scapula sign. In the category “Possible Rickets, Scurvy Unobservable” (n=4), 3 individuals presented a scapula sign, and 1 individual did not have a scapula sign. All individuals in the category “Possibly both” (n=2) showed a scapula sign. The juvenile classified as “Scurvy and possible Rickets” (n=1) showed a possible scapula sign. Finally, the individuals in the categories “Maybe Rickets” (n=1) and “Scurvy and maybe Rickets” (n=1) did not show scapula signs.



15. Visualisation of scapula sign per disease status category.

4.6 Radiographic assessment of the scapula sign

To see if the defects of the scapula were also radiographically visible, X-rays were taken of all scapulae with its inferior angle still intact. All radiographs were compared with radiographic images from the paper by Ives, Swan, and Humphrey (2023). Based on the imaging provided by Ives, Swan, and Humphrey (2023) (fig.6&7) it was observed that in the whole sample, 5 individuals showed a radiographically visible scapula sign. Of these individuals, 1 showed scapular defects in both scapulae, 2 individuals had a scapula sign in their right scapula, and 2 individuals had a left scapula sign. Of the 2 individuals with a left scapula sign, one of them did not have a right scapula preserved. Of the 2 individuals that had a right scapula sign, both did not have their left scapula preserved. Furthermore, 11 individuals had possible scapula signs, 4 of which had a possible scapula sign in both scapulae, 4 individuals had a left possible scapula sign, and 3 individuals had a right scapula sign. All but one individual with a possible bilateral or unilateral scapula sign had both scapulae preserved. Finally, 31 individuals did not have a scapula sign based on their X-rays. Between them, 23 individuals had both scapulae preserved, 4 individuals only had a right scapula preserved, and 4 only had a left scapula preserved.

According to the radiographic imaging, within the group of infants, 1 infant had a right scapula sign (unilaterally preserved), and 1 infant had a left scapula sign, with both scapulae preserved. Furthermore, 3 infants had a possible bilateral scapula sign. Finally, 16 individuals did not have a scapula sign; 10 of which had both scapulae, 3 had only their right scapula preserved and 3 infants had only their left scapula.

Among the children, 1 individual showed a scapula sign bilaterally. Another 3 individuals had a possible scapula sign in their right scapula; only 1 individual did not have both scapulae preserved. Additionally, 1 individual had a possible left scapula sign, and did not have a right scapula preserved. Lastly, 6 children did not have scapula sign, 1 of which had only their left scapula preserved, and 1 only their right scapula.

Of all juveniles, 1 juvenile had a left scapula sign (unilaterally preserved), and 1 juvenile had a right scapula right (also unilaterally preserved). Additionally, 3 individuals had a possible left scapula sign, and all had both scapulae intact, and 1 individual had a possible bilateral scapula sign. Finally, 9 juveniles did not present a scapula sign, and all of them had both their scapulae intact.

Chapter 5: Discussion

This thesis has attempted to explore whether a defect of the inferior angle of the scapula, described as the scapula sign by Alfred Weiss (1971) could be an indicator not only for rickets, but also for scurvy or only appears when the two diseases co-occur. To answer this, three sub questions were posed. First, the prevalence of both rickets and scurvy in the sample of the Middenbeemster population needed to be assessed. Subsequently, the prevalence of the scapula sign needed to be assessed, as well as how the prevalence of the scapula sign was distributed per age group and disease status.

The results from the diagnostic assessment of rickets and scurvy show that 50% of the sample is diagnostic of rickets, and 49% of the sample is diagnostic of scurvy. Furthermore, 30% of the sample is diagnostic of rickets and scurvy. Also, 57% of the individuals diagnosed as having both scurvy and rickets, have a present scapula sign. Additionally, the prevalence of the “Diagnostic of Rickets” category in infants was 52%, 64% in children and 47% in juveniles. For scurvy, the “Diagnostic of” category had a prevalence of 38% in infants, 64% in children, and 53% in juveniles. Of the whole sample, 68% was assessed to have a present scapula sign. The prevalence per age group shows a distribution of 66% in infants, 81% in children and 60% in juveniles.

Although these prevalence rates seem quite high, the study by Ives, Swan, and Humphrey (2023) also shows 30% of their sample (n=527) having signs of rickets (Ives, Swan & Humphrey, 2023, p.62). However, they do not elaborate further whether this group also contains individuals in a ‘highly consistent’ or ‘consistent’ category. Furthermore, a study by Geber and Murphy (2012) researching the Irish Famine (n=970) shows a prevalence for scurvy of 16% diagnostic of scurvy, probable scurvy in 14% and possible scurvy in 21% (Geber & Murphy, 2012, p.517). Although Geber and Murphy (2012) did divide diagnostic certainty in different categories similar to this thesis, the difference in prevalence here is still considerable. Because both Geber and Murphy (2012) and Ives, Swan, and Humphrey (2023) have much larger samples than was used in this study, it would be expected that the prevalence of at least scurvy would be much lower in this sample – as it seems that the prevalence for (diagnostic of) rickets in this thesis is comparable to the results from Ives, Swan, and Humphrey (2023).

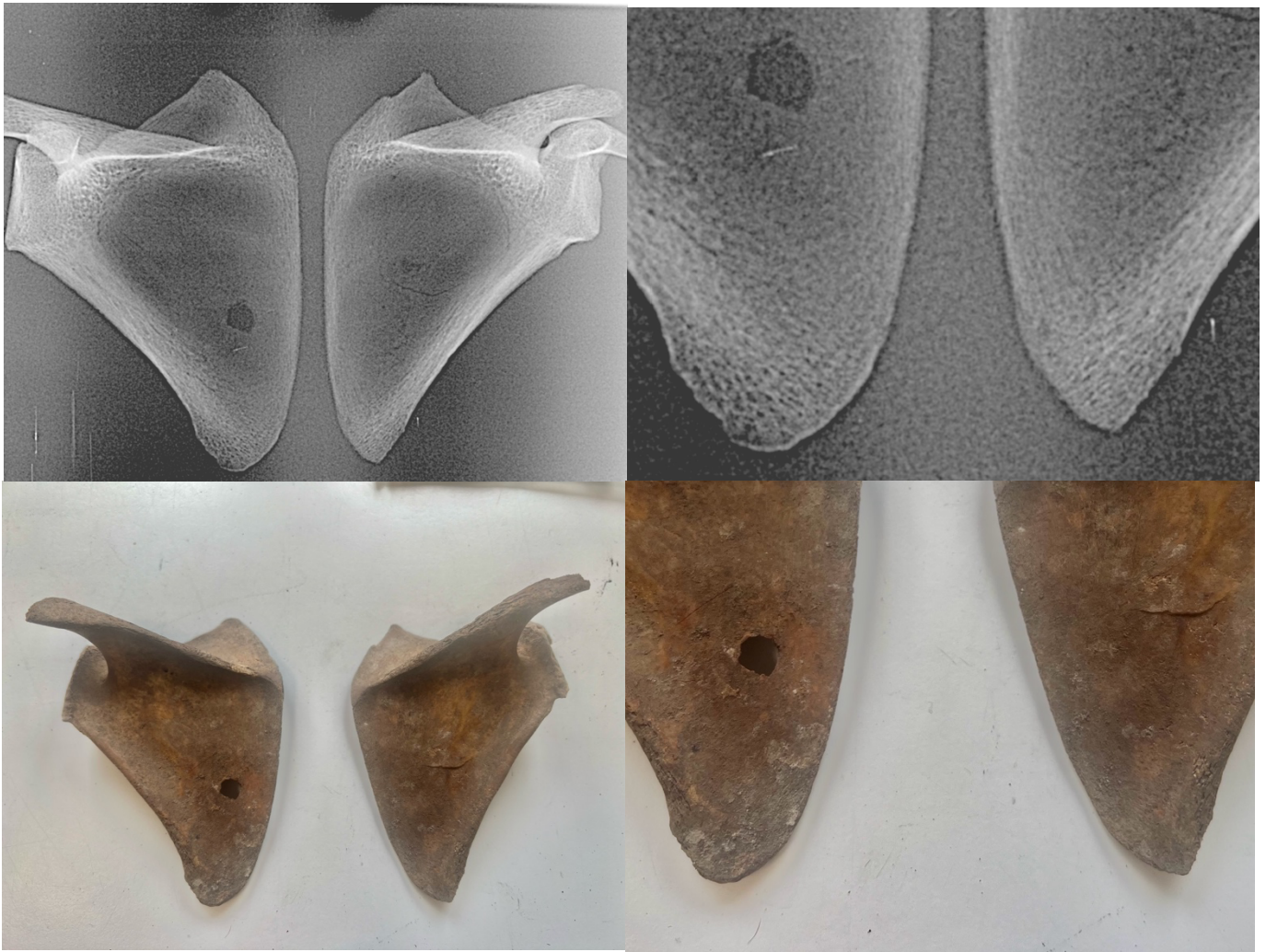
The high prevalence in this thesis could be due to several reasons. First, it is a relatively small sample, which instantly makes a ratio greater or smaller when one or two individuals end up in a specific category. This could indicate that the diagnostic framework used in this thesis might not be suitable for small samples. Secondly, this sample contains a much higher number of infants, almost half of the sample, most of them not older than a few months. The studies mentioned above have samples containing more children and more juveniles, where the possibility of healed cases is much higher. As mentioned in chapter 2, due to the rapid growth rate of non-adults, it can be assumed that rickets and scurvy prevalence in samples with a higher number of older non-adults than infants would be lower as restoration of the vitamin deficiencies could result in the invisibility of lesions (Lewis, 2009, p.133). Furthermore, the high prevalence of both diseases scored in this thesis could also be due to the fact that non-adults tend to have more porous bone – growing bones are often a lot more porous than bones of adults – and the distinction between expected porosity and unexpected porosity was difficult to observe (Lewis, 2009, p.135). As mentioned before, porosity, rickets, scurvy, non-specific markers of stress are tricky to distinguish from one another; although their etiology might be different, their visual expression is often very similar (Zuckerman et al., 2014, p.27; Klaus, 2017, p.97).

However, the results of this thesis still show that the prevalence of a present scapula sign is most common in individuals that are either diagnostic of rickets and/or diagnostic of scurvy, although the ratio between present-absent scapula sign is higher in the diagnostic category of scurvy than it is for rickets. Nonetheless, if the prevalence of the scapula sign is evaluated in amongst all disease status categories, it is still evident that individuals categorized as having both scurvy and rickets have the highest number of present scapula signs. This finding does suggest the possibility to consider defects of the inferior angle of the scapula as an addition to the criteria proposed by Ortner and his colleagues. Yet, there are still some things to reflect on in light of this suggestion. First, according to the results of this thesis, the prevalence of the present scapula sign is high in all diagnostic categories and is even present in the “Not Consistent” categories. It is understandable that there could be a possible margin of error, although it is unclear as to how large this margin would then be. However, it seems that for the categories “Diagnostic of” and “Highly Consistent with/Typical of” for rickets, prevalence of the scapula sign is highest and partly contradicts the finding of Ives, Swan, and

Humphrey (2023) that the scapula sign is almost exclusively visible in severe cases of rickets. As for scurvy, there is a high number of absent scapula signs in the category “Diagnostic of” as well, albeit lower than the number of present scapula signs. Interestingly for scurvy, however, is the relative high number of individuals with “Unobservable” scurvy, the majority of which has a scapula sign. It must be said that in preparation for this thesis, all but three sources, namely Brickley and Morgan (2023), Schattmann et al. (2016) and Geben and Murphy (2012), do not mention the scapula at all when discussing scurvy, although porosity due to hemorrhaging near the supra- and infraspinous fossa is a known sign for scurvy. It is interesting to observe that, in the literature analyzed for this thesis, almost all lesions for rickets are consequently mentioned but in the case of discussing lesions for scurvy, the scapula is not always named. This begs to question the relevance of the presence of porosity in the scapula when assessing scurvy; although the infrequency could also be due to the fact the known difficulties of distinguishing scurvy, rickets, anemia, and non-specific markers of stress (Klaus, 2017, p.96).

It is also noteworthy that the macroscopic assessment of the scapula sign and the radiographic assessment of the scapula sign differ considerably. The scapula sign proposed by Weiss (1971) was not very extensively described, as his paper was only four pages long and focused mainly on the radiographic pattern that he found in the scapulae of sick children that happened to have rickets. It could be expected from the paper by Ives, Swan, and Humphrey (2023) that the criteria of said scapula sign would be further expanded upon and described in more detail. However, the authors also did not engage much with the proposed criteria for the scapula, not more than the naming of what they are: porosity, blunting, concavity/cupping, coarsening and slit-strut morphology. Furthermore, although Ives, Swan, and Humphrey (2023) argued that they used their radiographic assessment as more of a visual aid, it seems that they did base their conclusion of the scapula sign primarily on radiographic assessment, and secondarily on macroscopic assessment. This begs the question as to whether they have observed cases in which the scapula sign was macroscopically visible, but not on the X-rays, or vice versa, just as is the case in this thesis. The results of the X-rays for this thesis show that many of the scapula signs that were macroscopically assessed as present, are not visible on the radiographs. However, there are also a few scapula signs that are assessed as absent macroscopically, but present in the X-rays. For

example, individual S286V0469, a 10-year-old juvenile diagnostic of scurvy and consistent with rickets, has a possible left scapula sign visible on a radiograph, but was not macroscopically assessed because this scapula only showed two out of five criteria: coarsening and a slight bending deformity that was scored under cupping/concavity (fig.16).

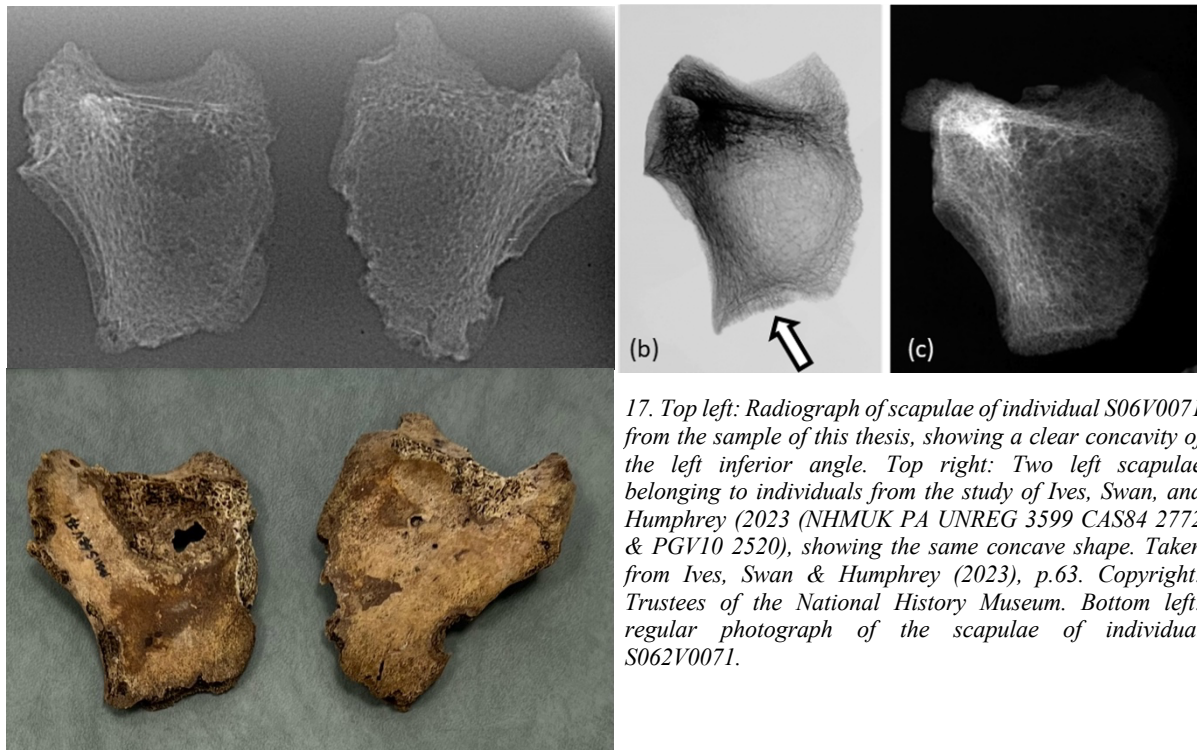


16. Top left: Radiographic image of the scapulae of individual S286V0469. Top right: closeup of the inferior angles, showing a clear fraying of the left lateral border of the inferior angle. Bottom left: Photograph of macroscopic visibility. Bottom right: closeup of the inferior angles, macroscopically showing only (probably taphonomic) coarsening and a slight bending deformity.

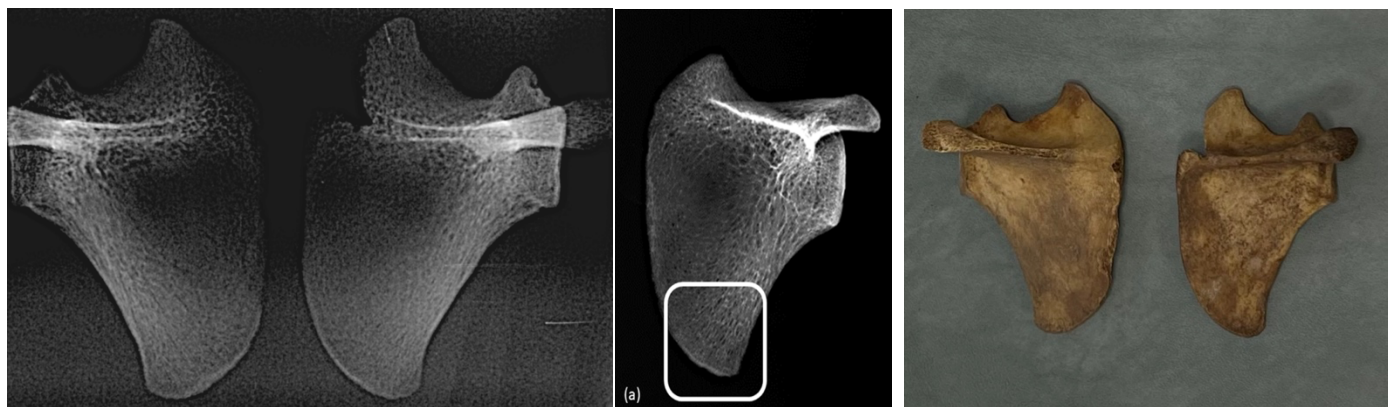
Furthermore, there was only one individual in the sample that had a scapula sign almost identical to the signs presented by Ives, Swan, and Humphrey (2023) (fig.17). In this case, it was a 3,5-year-old child with a severe case of rickets and diagnostic of scurvy. Seeing as this is the only individual mimicking the same visible alterations to the inferior angle of the scapula as the study by Ives, Swan, and Humphrey (2023) but many more individuals in the sample for this thesis seem to have a present scapula sign according to the criteria given by the same study, it should open the discussion as to the appearance of the scapula sign and its possible variations. A third example is a 3-year-old child that is both diagnostic of rickets and of scurvy, presented with a scapula sign that was macroscopically visible, but not visible on X-ray (fig.18). Macroscopically, this child scored present for coarsening, cupping, and porosity, although the cupping was not visible on X-ray, but macroscopically observed. It seems that most visible in X-rays are the loss of border definition due to porosity and slit-strut morphology, and that the criteria of concavity should be defined further, as a slight bending of the inferior angle inwards was not visible on the X-rays taken for this study.

This high contrast of results between this thesis and the work by Ives, Swan, and Humphrey (2023) does bring forth the question of when a scapula sign is a scapula sign. Currently, at the moment this thesis is being produced, there exist but two sources regarding the scapula sign: Weiss (1971) and Ives, Swan, and Humphrey (2023). The first solely based his findings on radiographic assessment, and the second heavily relied on it as well. After all, the assessment of the vitamin deficiencies as per the framework proposed in this thesis is also not black and white, and combinations of lesions should be evaluated as a whole, and not by form of a “tick-list” (Brickley and Morgan, 2023, p.643). The results of this thesis suggest that, in order to map the scapula

sign further, it should be explored whether the scapula sign is almost exclusively visible on radiographs, or if macroscopic observation should be used as the dominant manner of assessment.



17. Top left: Radiograph of scapulae of individual S06V0071 from the sample of this thesis, showing a clear concavity of the left inferior angle. Top right: Two left scapulae belonging to individuals from the study of Ives, Swan, and Humphrey (2023 (NHMUK PA UNREG 3599 CAS84 2772 & PGV10 2520), showing the same concave shape. Taken from Ives, Swan & Humphrey (2023), p.63. Copyright: Trustees of the National History Museum. Bottom left: regular photograph of the scapulae of individual S062V0071.



18. Left: X-ray of scapulae of individual S293V0495 from the sample of this thesis. Middle: a reference radiograph provided by Ives, Swan, and Humphrey (2023) for a normal shaped inferior angle. Taken from Ives, Swan & Humphrey (2023), p.60. Copyright R. Ives, K. Swan, L. Humphrey. Right: normal photo of the scapulae of S293V0495.

After all, although this thesis shows that it could be argued that defects of the inferior angle of the scapula can be linked to (nutritional) deficiencies that affect bone formation, it is also possible that the scapula sign might

be ‘just another’ non-specific marker of stress. As cartilage – and especially cartilage of non-adults – are notoriously poor preserved, and the scapula is a quite thin bone to begin with, it could also be suggested that the macroscopically visible criteria for the scapula sign, such as coarsening and porosity, are due to taphonomic reasons, in which case, the criteria of the scapula sign would need further study (Ives, Swan & Humphrey, 2023, p.63). As for the significance of the scapula sign as a diagnostic criterion, it should be further studied if rickets and/or scurvy are recognizable through this lesion. In this thesis, the individuals were assessed on the nutritional diseases first, and the scapula sign second. It would be interesting to test the relevance of the scapula sign by reversing this order of assessment.

With regards to the co-occurrence and disease assessment: although the created framework based on the work by Morgan and Brickley (2023), Schattmann et al. (2016), and Harper et al. (2011) has shown to be a well-supported framework for the assessment of rickets and/or scurvy and has the potential to be used in future research, it can be suggested that the large diversity of diagnostic categories might not work for small samples and might even have a blurring effect on results. In this thesis, there were many categories containing only one or two individuals. However, it can be argued that the diagnostic certainty did not lose its preciseness, even though the results were more spread out than initially expected upon creating of this framework. Yet, it does predict its usefulness in future research with larger samples and supports a standardized way of assessing lesions. Additionally, in this thesis it has proven most useful to only review the individuals in the “Both” category because of the size of the sample. It is expected that the usefulness of the other categories will increase once the sample size increases as well.

Furthermore, as scurvy is often only visible in individuals older than four months, it is interesting that many of the infants younger than four months in this sample have also been deemed to have lesions diagnostic or highly consistent with scurvy. It is also not likely that the majority of the infants have survived their disease long enough to develop lesions (Lewis, 2009, p.125). A margin of error is suspected here, and it would be valuable if this would be further explored in the future. Subsequently, with regards to the whole age distribution within the sample, the infant group was the largest, comprising 45% of the sample. As mentioned before, it remains challenging to distinguish lesions for rickets, scurvy, anemia, or non-specific markers of stress,

especially in skeletal remains of non-adults, as some level of porosity is to be expected anyway (Klaus, 2017, p.96; Lewis, 2009, p.135). For future research, it would be interesting to study whether the framework of this thesis would be better applicable on samples with more evenly distributed groups, so far as the archeological record permits.

Conclusion

This thesis has explored whether defects of the inferior angle of the scapulae could be associated to vitamin D and/or vitamin C deficiency diseases. To answer this, a sample (n=47) of the post-medieval Middenbeemster population currently stored in the Human Osteology Lab in Leiden of non-adults between 3 weeks and 12 years old with one or both scapulae intact were analyzed. The sample was assessed for the prevalence of rickets, the prevalence of scurvy and the prevalence of the so-called scapula sign, initially proposed by Weiss (1971) and expanded upon by Ives, Swan, and Humphrey (2023). For this thesis, the questions were structured as follows:

“To what extent is the scapula sign as identified by Weiss (1971) an indicator for rickets or a possible (new) indicator for scurvy and/or the co-occurrence of rickets and scurvy in the post-medieval non-adults of Middenbeemster?”

The answer to this question has been explored through three sub questions. First, it was necessary to study the prevalence of both rickets and scurvy in the Middenbeemster population. Every individual was analyzed for lesions associated with rickets and lesions associated with scurvy. For each disease, macroscopic lesions were scored and subsequently assessed in a disease status based on their diagnostic, highly consistent or consistent character. A framework for assessing the diagnostic certainty of both diseases was created in pursuit of a more standardized way of assessing rickets and scurvy. The results show that 30% of the sample population was diagnostic of both rickets and scurvy. The remaining 70% was divided in thirteen other categories ranging from “Highly Consistent with/Typical of” to “Unobservable”, for both rickets and scurvy. The results are possibly affected by a ‘porosity-bias’, as it remains challenging to distinguish between expected and unexpected porosity in skeletal remains of growing individuals.

Secondly, the presence of the scapula sign was assessed based on criteria given in the paper by Ives, Swan, and Humphrey (2023). Also, a new framework was constructed to assess the scapula sign based on these criteria. Additionally, radiographs were taken to help visualize the scapula sign. The results of this thesis show

that 68% of the whole sample population presented a scapula sign and 57% of the sample that was categorized as “Both” (i.e. diagnostic of scurvy and rickets) presented a scapula sign. In the discussion it was further elaborated that this high prevalence could possibly be due to the fact that although the assessment framework was not flawed, the definitions of the criteria for the scapula sign could have been explored further, most likely in future research, as the source material for the assessment in this thesis was slim.

Thirdly, it was explored what the distribution of the prevalence of rickets, scurvy and the scapula sign was per age group. The results show that the highest prevalence of rickets (“Diagnostic of”) was in children (64%), although this was also the smallest group, followed by 52% of infants, and 47% of juveniles. Additionally, the prevalence of scurvy (“Diagnostic of”) was also highest in the children group with 64%, followed by 53% of juveniles and 38% of infants. Lastly, the prevalence of the scapula sign was also highest in the group of children, with 81%. Furthermore, 66% of infants showed a scapula sign, and 60% of juveniles showed a scapula sign.

The results show a high prevalence for the co-occurrence of scurvy and rickets, namely 30% of the sample. Furthermore, half of the individuals scored as both diagnostic for rickets as well as for scurvy presented with a scapula sign. This thesis does therefore argue that there is a strong correlation between rickets and scurvy, and the presence of a defect in the inferior angle of the scapula. However, it becomes evident that questions need to be raised regarding the definition and criteria of the scapula sign. Many of the scapula signs that were assessed macroscopically were not visible on X-rays although it was expected the radiographic assessment would have a confirming function of the lesions that were macroscopically assessed. The results of this thesis suggest that it is imperative more research needs to be done in pursuit of defining the scapula sign and its appearance more clearly. Furthermore, it has clearly shown that the scapula sign can be a significant lesion and should be further studied. Finally, the framework constructed for the assessment of rickets and scurvy shows great promise, although the differences in visible pathology for adults and non-adults needs to be more carefully considered; nutritional diseases can disappear from the skeletal record almost as quickly as they appear when it comes to non-adults and must be kept in mind. For future research, it would be interesting to explore whether the used frameworks in this thesis prove to be successful in studies with larger samples with more even

distributions of age groups. To summarize, although the results of the disease assessment seem to be affected by a 'porosity-bias', and the results of the scapula sign assessment heavily rely upon not entirely well-thought-out criteria, the frameworks created for this thesis are very promising and should be a strong starting point to study the relation between rickets, scurvy, and the scapula sign even further.

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Appendix 1: Disease Status and Scapula Sign Overview

ID	Age group	Diagnosis_R	Diagnosis_S	Disease_Status	Scapula Status	X-ray	conclusion_L	conclusion_R	B/LR
S141V0223	Child	Consistent with Rickets	Diagnostic of Scurvy	Scurvy and maybe Rickets	present	no	unobservable	present	R
S343V0732	Child	Diagnostic of Rickets	Highly Consistent/Typical Scurvy	Rickets and possible Scurvy	present	possible L	present	unobservable	L
S127V0204	Child	Highly Consistent/Typical Rickets	Diagnostic of Scurvy	Scurvy and possible Rickets	present	no	absent	present	B
S062V0071	Child	Diagnostic of Rickets	Diagnostic of Scurvy	Both	present	yes, both	present	present	B
S293V0495	Child	Diagnostic of Rickets	Diagnostic of Scurvy	Both	present	no	present	absent	B
S316V0641	Child	Diagnostic of Rickets	Consistent with Scurvy	Rickets and maybe Scurvy	present	no	absent	present	B
S443V1525	Child	Diagnostic of Rickets	Unobservable	Diagnostic of Rickets, Scurvy Unobservable	present	possible R	unobservable	present	L
S326V0708	Child	Diagnostic of Rickets	Diagnostic of Scurvy	Both	absent	possible R	absent	absent	B
S353V0739	Child	Highly Consistent/Typical Rickets	Diagnostic of Scurvy	Scurvy and possible Rickets	possible	no	possible	possible	B
S181V0407	Child	Diagnostic of Rickets	Diagnostic of Scurvy	Both	present	no	present	unobservable	L
S412V0888	Child	Highly Consistent/Typical Rickets	Consistent with Scurvy	Possible Rickets, maybe Scurvy	present	possible R	present	absent	B
S284V0446	Infant	Diagnostic of Rickets	Diagnostic of Scurvy	Both	present	no	unobservable	present	R
S421V0940	Infant	Highly Consistent/Typical Rickets	Consistent with Scurvy	Possible Rickets, maybe Scurvy	possible	possible both	absent	possible	B
S320V0662	Infant	Highly Consistent/Typical Rickets	Diagnostic of Scurvy	Scurvy and possible Rickets	present	no	absent	present	B
S158V0230	Infant	Diagnostic of Rickets	Diagnostic of Scurvy	Both	absent	no	absent	absent	B
S165V0242	Infant	Diagnostic of Rickets	Diagnostic of Scurvy	Both	absent	no	absent	absent	B
S058V0092	Infant	Diagnostic of Rickets	Consistent with Scurvy	Rickets and maybe Scurvy	present	yes R	unobservable	present	R
S082V0084	Infant	Diagnostic of Rickets	Diagnostic of Scurvy	Both	absent, unobservable	no	absent	unobservable	L
S187V0267	Infant	Consistent with Rickets	Diagnostic of Scurvy	Scurvy and maybe Rickets	absent	no	absent	absent	B
S046V0023	Infant	Consistent with Rickets	Diagnostic of Scurvy	Scurvy and maybe Rickets	absent	no	absent	absent	B
S037V0021	Infant	Not Consistent with Rickets	Not Consistent with Scurvy	Neither	present	no	absent	present	B
S493V1069	Infant	Diagnostic of Rickets	Highly Consistent/Typical Scurvy	Rickets and possible Scurvy	absent	no	absent	absent	B
S048V1520	Infant	Not Consistent with Rickets	Highly Consistent/Typical Scurvy	Possible Scurvy	present	no	present	unobservable	L
S335V0711	Infant	Diagnostic of Rickets	Consistent with Scurvy	Rickets and maybe Scurvy	present	possible both	present	absent	B
S314V0655	Infant	Highly Consistent/Typical Rickets	Highly Consistent/Typical Scurvy	Possibly both	present	no	absent	present	B
S227V0297	Infant	Highly Consistent/Typical Rickets	Diagnostic of Scurvy	Scurvy and possible Rickets	present	possible both	absent	present	B
S152V0244	Infant	Highly Consistent/Typical Rickets	Consistent with Scurvy	Possible Rickets, maybe Scurvy	present	no	present	present	B
S352V0747	Infant	Diagnostic of Rickets	Not Consistent with Scurvy	Rickets	present	yes L	present	present	B
S050V0042	Infant	Unobservable	Highly Consistent/Typical Scurvy	Possible Scurvy, Rickets Unobservable	present	no	unobservable	present	R
S072V0033	Infant	Diagnostic of Rickets	Highly Consistent/Typical Scurvy	Rickets and possible Scurvy	present	no	present	unobservable	L
S287V0450	Infant	Diagnostic of Rickets	Consistent with Scurvy	Rickets and maybe Scurvy	present	no	unobservable	present	L

S323V0650	Infant	Diagnostic of Rickets	Highly Consistent/Typical Scurvy	Rickets and possible Scurvy	present	no	present	present	B
S044V0027	Juvenile	Diagnostic of Rickets	Diagnostic of Scurvy	Both	present	possible L	possible	present	B
S269V1506	Juvenile	Highly Consistent/Typical Rickets	Unobservable	Possible Rickets, Scurvy Unobservable	absent	no	absent	absent	B
S286V0469	Juvenile	Consistent with Rickets	Diagnostic of Scurvy	Scurvy and maybe Rickets	absent	possible L	absent	absent	B
S549V1181	Juvenile	Diagnostic of Rickets	Diagnostic of Scurvy	Both	absent	no	absent	absent	B
S367V0803	Juvenile	Consistent with Rickets	Not Consistent with Scurvy	Maybe Rickets	absent	possible L	absent	absent	B
S282V0417	Juvenile	Highly Consistent/Typical Rickets	Diagnostic of Scurvy	Scurvy and possible Rickets	possible	yes L	possible	unobservable	L
S104V0136	Juvenile	Highly Consistent/Typical Rickets	Highly Consistent/Typical Scurvy	Possibly both	present	no	present	present	B
S140V0207	Juvenile	Diagnostic of Rickets	Diagnostic of Scurvy	Both	present	no	present	absent	B
S389V0857	Juvenile	Highly Consistent/Typical Rickets	Unobservable	Possible Rickets, Scurvy Unobservable	present	no	present	absent	B
S301V0583	Juvenile	Highly Consistent/Typical Rickets	Unobservable	Possible Rickets, Scurvy Unobservable	present	yes R	unobservable	present	R
S396V0877	Juvenile	Diagnostic of Rickets	Diagnostic of Scurvy	Both	present	no	present	possible	B
S334V0716	Juvenile	Diagnostic of Rickets	Diagnostic of Scurvy	Both	present	possible both	present	present	B
S248V0393	Juvenile	Highly Consistent/Typical Rickets	Unobservable	Possible Rickets, Scurvy Unobservable	present	no	absent	present	B
S503V1099	Juvenile	Diagnostic of Rickets	Diagnostic of Scurvy	Both	absent	no	absent	absent	B
S365V0773	Juvenile	Highly Consistent/Typical Rickets	Highly Consistent/Typical Scurvy	Possibly both	present	no	absent	present	B

Appendix 2: Rickets Lesions Overview

ID	cran_vault_por	orb_roof_por	cran_vault_thick	def_mand_ramus	rib_bend_def	cc_rib_flar	cc_rib_por	ilium_concav	ul_longb_bend_def	il_longb_bend_def	longb_metaph_flar	supr_flat_femor_me_taph	coxa_vara	Diagnosis_R
S353V0739	absent	present	absent	absent	absent	absent	absent	absent	absent	present	present	absent	absent	Highly Consistent/ Typical Rickets
S326V0708	absent	present	absent	absent	present	absent	absent	absent	absent	present	present	present	absent	Diagnostic of Rickets
S443V1525	unobservable	unobservable	unobservable	unobservable	absent	unobservable	present	unobservable	present	present	present	absent	unobservable	Diagnostic of Rickets
S316V0641	present	present	absent	absent	present	absent	present	absent	absent	present	present	unobservable	absent	Diagnostic of Rickets
S293V0495	absent	present	absent	absent	absent	present	absent	absent	absent	absent	present	present	present	Diagnostic of Rickets
S062V0071	present	unobservable	present	unobservable	present	present	present	present	absent	present	present	present	present	Diagnostic of Rickets
S127V0204	absent	present	absent	absent	absent	unobservable	absent	absent	absent	present	present	present	present	Highly Consistent/ Typical Rickets
S343V0732	absent	present	absent	absent	present	present	absent	present	absent	present	absent	present	present	Diagnostic of Rickets
S141V0223	absent	present	absent	absent	absent	absent	absent	absent	absent	absent	absent	present	present	Consistent with Rickets

S046V0023	S187V0267	S082V0084	S058V0092	S165V0242	S158V0230	S320V0662	S421V0940	S284V0446	S412V0888	S181V0407
absent	present	present	present	present	absent	absent	absent	absent	absent	absent
present	present	present	present	absent	present	unobservable	unobservable	present	unobservable	present
absent	absent	absent	absent	absent	absent	absent	absent	absent	absent	absent
absent	absent	absent	unobservable	absent	present	absent	absent	absent	absent	absent
absent	absent	absent	present	present	absent	absent	absent	absent	absent	absent
absent	absent	present	present	present	present	present	absent	absent	absent	present
absent	absent	present	absent	present	absent	present	absent	absent	absent	absent
absent	absent	absent	absent	present	absent	absent	unobservable	absent	absent	absent
present	absent	absent	absent	present	present	absent	present	present	absent	absent
present	absent	unobservable	absent	present	present	absent	absent	present	unobservable	present
absent	present	present	absent	present	present	absent	present	present	present	present
present	unobservable	unobservable	absent	absent	unobservable	absent	unobservable	present	unobservable	absent
absent	present	unobservable	absent	absent	absent	absent	absent	absent	unobservable	absent
Consistent with Rickets	Consistent with Rickets	Diagnostic of Rickets	Diagnostic of Rickets	Diagnostic of Rickets	Diagnostic of Rickets	Highly Consistent/ Typical Rickets	Highly Consistent/ Typical Rickets	Diagnostic of Rickets	Highly Consistent/ Typical Rickets	Diagnostic of Rickets

S287V0450	S072V0033	S050V0042	S352V0747	S152V0244	S227V0297	S314V0655	S335V0711	S048V1520	S493V1069	S037V0021
absent	present	absent	absent	absent	present	unobservable	present	absent	present	absent
absent	unobservable	absent	absent	unobservable	present	unobservable	absent	unobservable	absent	unobservable
absent	present	absent	absent	absent	absent	unobservable	absent	unobservable	absent	absent
absent	unobservable	unobservable	unobservable	absent	absent	absent	absent	unobservable	absent	absent
present	absent	absent	present	absent	absent	absent	absent	absent	absent	absent
present	present	present	present	present	absent	present	present	unobservable	absent	unobservable
present	present	absent	absent	absent	absent	present	absent	absent	present	absent
present	unobservable	unobservable	absent	unobservable	absent	absent	absent	unobservable	absent	absent
present	absent	unobservable	absent	present	absent	absent	absent	absent	absent	absent
present	unobservable	unobservable	present	unobservable	absent	absent	present	absent	absent	absent
present	unobservable	unobservable	present	absent	present	absent	absent	absent	present	unobservable
unobservable	unobservable	unobservable	unobservable	unobservable	unobservable	unobservable	unobservable	unobservable	unobservable	unobservable
absent	unobservable	unobservable	absent	unobservable	absent	present	absent	absent	absent	unobservable
Diagnostic of Rickets	Diagnostic of Rickets	Unobservable	Diagnostic of Rickets	Highly Consistent/ Typical Rickets	Highly Consistent/ Typical Rickets	Highly Consistent/ Typical Rickets	Diagnostic of Rickets	Not Consistent with Rickets	Diagnostic of Rickets	Not Consistent with Rickets

S301V0583	S389V0857	S140V0207	S104V0136	S282V0417	S367V0803	S549V1181	S286V0469	S269V1506	S044V0027	S323V0650
unobservable	unobservable	absent	absent	absent	absent	present	absent	unobservable	unobservable	absent
unobservable	unobservable	present	present	present	absent	absent	present	unobservable	present	absent
unobservable	unobservable	absent	absent	absent	absent	absent	absent	unobservable	unobservable	present
absent	unobservable	present	absent	absent	absent	absent	absent	unobservable	present	absent
absent	absent	absent	absent	absent	absent	unobservable	absent	absent	absent	present
unobservable	absent	present	present	present	unobservable	present	absent	absent	present	present
absent	absent	absent	absent	absent	absent	absent	absent	absent	absent	present
unobservable	absent	absent	absent	present	absent	absent	absent	absent	present	absent
absent	absent	present	absent	absent	absent	absent	absent	absent	absent	present
absent	absent	absent	present	absent	absent	absent	absent	present	present	present
present	present	present	present	present	present	present	present	present	present	present
present	present	absent	present	absent	present	present	present	present	present	absent
present	absent	absent	present	absent	present	absent	absent	absent	present	present
Highly Consistent/ Typical Rickets	Highly Consistent/ Typical Rickets	Diagnostic of Rickets	Highly Consistent/ Typical Rickets	Highly Consistent/ Typical Rickets	Consistent with Rickets	Diagnostic of Rickets	Consistent with Rickets	Highly Consistent/ Typical Rickets	Diagnostic of Rickets	Diagnostic of Rickets

S365V0773	S503V1099	S248V0393	S334V0716	S396V0877
unobservable	absent	unobservable	present	present
unobservable	present	unobservable	present	present
unobservable	present	unobservable	absent	absent
absent	absent	absent	absent	present
absent	present	present	present	present
unobservable	absent	absent	present	present
absent	present	present	absent	absent
unobservable	absent	absent	absent	absent
absent	absent	absent	absent	absent
absent	present	present	absent	absent
present	present	absent	present	present
present	present	absent	absent	absent
present	present	present	absent	present
Highly Consistent/ Typical Rickets	Diagnostic of Rickets	Highly Consistent/ Typical Rickets	Diagnostic of Rickets	Diagnostic of Rickets

Appendix 3: Scurvy Lesions Overview

ID	ext_surf_GWS	zbone_or_mzproc	post_surf_max	coro_proc_mand_medsurf	pal_proc_max_inf_surf	max_ao_mand_al_veob	orb_wall	cran_vault	inforbfora_max	foramroidm_sphen	long_bone	scap_supr_infr_spinfos	ilia	Diagnosis_S
S353V0739	present	present	absent	present	present	present	present	absent	present	unobservable	present	present	present	Diagnostic of Scurvy
S326V0708	present	unobservable	present	present	present	present	present	absent	unobservable	unobservable	present	present	absent	Diagnostic of Scurvy
S443V1525	unobservable	unobservable	unobservable	unobservable	unobservable	unobservable	unobservable	unobservable	unobservable	unobservable	absent	absent	unobservable	Unobservable
S316V0641	unobservable	unobservable	unobservable	absent	present	present	present	absent	unobservable	unobservable	absent	possible	absent	Consistent with Scurvy
S293V0495	present	absent	present	present	present	present	present	absent	absent	present	absent	absent	absent	Diagnostic of Scurvy
S062V0071	present	unobservable	unobservable	unobservable	unobservable	unobservable	unobservable	present	unobservable	unobservable	present	absent	present	Diagnostic of Scurvy
S127V0204	absent	absent	present	present	present	present	present	absent	absent	present	present	present	absent	Diagnostic of Scurvy
S343V0732	absent	absent	unobservable	present	present	present	present	present	present	absent	absent	absent	absent	Highly Consistent/ Typical Scurvy
S141V0223	present	absent	present	present	present	present	present	absent	present	absent	absent	present	present	Diagnostic of Scurvy

S046V0023	S187V0267	S082V0084	S058V0092	S165V0242	S158V0230	S320V0662	S421V0940	S284V0446	S412V0888	S181V0407
absent	present	present	unobservable	absent	present	present	unobservable	present	absent	present
absent	present	present	unobservable	unobservable	unobservable	unobservable	present	absent	present	present
present	unobservable	unobservable	unobservable	present	present	unobservable	unobservable	present	unobservable	present
absent	absent	absent	unobservable	absent	absent	absent	absent	absent	present	present
present	present	present	unobservable	present	present	present	unobservable	present	unobservable	present
present	present	present	unobservable	present	present	present	unobservable	absent	unobservable	present
present	present	present	present	absent	present	present	unobservable	present	unobservable	absent
unobservable	unobservable	unobservable	unobservable	present	absent	unobservable	present	present	unobservable	present
present	present	present	present	present	present	present	unobservable	present	absent	unobservable
absent	present	absent	absent	present	absent	absent	absent	absent	absent	absent
present	present	absent	absent	absent	present	absent	absent	present	absent	present
absent	absent	absent	present	present	absent	absent	unobservable	absent	absent	absent
Diagnostic of Scurvy	Diagnostic of Scurvy	Diagnostic of Scurvy	Consistent with Scurvy	Diagnostic of Scurvy	Diagnostic of Scurvy	Diagnostic of Scurvy	Consistent with Scurvy	Diagnostic of Scurvy	Consistent with Scurvy	Diagnostic of Scurvy

S287V0450	S072V0033	S050V0042	S352V0747	S152V0244	S227V0297	S314V0655	S335V0711	S048V1520	S493V1069	S037V0021
absent	absent	absent	absent	unobservable	present	unobservable	absent	unobservable	absent	absent
absent	unobservable	unobservable	absent	present	unobservable	unobservable	absent	absent	present	present
unobservable	unobservable	unobservable	absent	unobservable	unobservable	unobservable	unobservable	unobservable	unobservable	unobservable
present	unobservable	unobservable	unobservable	present	absent	unobservable	absent	unobservable	absent	absent
present	unobservable	unobservable	absent	absent	present	unobservable	absent	unobservable	absent	absent
absent	unobservable	unobservable	absent	unobservable	absent	present	present	unobservable	present	present
absent	unobservable	absent	absent	unobservable	present	unobservable	absent	unobservable	absent	unobservable
absent	present	absent	absent	absent	present	unobservable	present	absent	present	absent
unobservable	unobservable	unobservable	unobservable	unobservable	unobservable	unobservable	unobservable	unobservable	unobservable	unobservable
absent	present	absent	absent	unobservable	present	unobservable	present	unobservable	unobservable	absent
absent	unobservable	unobservable	absent	absent	present	present	absent	absent	present	absent
absent	present	present	absent	absent	present	present	absent	present	present	absent
absent	unobservable	unobservable	absent	unobservable	absent	present	present	unobservable	present	absent
Consistent with Scurvy	Highly Consistent/ Typical Scurvy	Highly Consistent/ Typical Scurvy	Not Consistent with Scurvy	Consistent with Scurvy	Diagnostic of Scurvy	Highly Consistent/ Typical Scurvy	Consistent with Scurvy	Highly Consistent/ Typical Scurvy	Highly Consistent/ Typical Scurvy	Not Consistent with Scurvy

S301V0583	S389V0857	S140V0207	S104V0136	S282V0417	S367V0803	S549V1181	S286V0469	S269V1506	S044V0027	S323V0650
unobservable	unobservable	present	absent	present	absent	present	present	unobservable	present	absent
unobservable	unobservable	absent	absent	unobservable	unobservable	absent	absent	unobservable	absent	present
unobservable	unobservable	unobservable	present	present	absent	unobservable	present	unobservable	present	unobservable
absent	unobservable	absent	absent	absent	absent	absent	absent	unobservable	absent	present
unobservable	unobservable	present	present	present	absent	present	present	unobservable	present	present
unobservable	unobservable	present	absent	present	unobservable	present	present	unobservable	present	present
unobservable	unobservable	present	present	present	absent	absent	present	unobservable	present	absent
unobservable	unobservable	absent	absent	absent	absent	present	absent	unobservable	unobservable	absent
unobservable	unobservable	present	absent	absent	unobservable	absent	absent	unobservable	absent	unobservable
unobservable	unobservable	present	absent	absent	absent	absent	present	unobservable	absent	absent
present	present	absent	absent	present	absent	absent	absent	absent	absent	present
absent	absent	present	absent	present	absent	absent	absent	absent	present	present
unobservable	absent	absent	present	present	absent	absent	present	absent	absent	present
Unobservable e	Unobservable e	Diagnostic of Scurvy	Highly Consistent/ Typical Scurvy	Diagnostic of Scurvy	Not Consistent with Scurvy	Diagnostic of Scurvy	Diagnostic of Scurvy	Unobservable e	Diagnostic of Scurvy	Highly Consistent/ Typical Scurvy

S365V0773	S503V1099	S248V0393	S334V0716	S396V0877
unobservable	present	unobservable	present	absent
absent	present	unobservable	present	absent
present	present	unobservable	present	present
present	present	present	absent	absent
present	present	unobservable	present	present
present	present	unobservable	present	present
unobservable	present	unobservable	present	present
unobservable	absent	unobservable	present	present
absent	unobservable	unobservable	absent	absent
unobservable	absent	unobservable	present	present
absent	present	present	absent	present
present	absent	absent	absent	absent
unobservable	present	absent	present	present
Highly Consistent/ Typical Scurvy	Diagnostic of Scurvy	Unobservable	Diagnostic of Scurvy	Diagnostic of Scurvy

Appendix 4: Scapula Sign Lesions Overview

ID	coarsening_L	blunting_L	concavity_or_cupping_L	slit_strut_morph_L	porosity_L	conclusion_L	coarsening_R	blunting_R	concavity_or_cupping_R	slit_strut_morph_R	porosity_R	conclusion_R
S353V0739	present	unobservable	absent	absent	present	possible	unobservable	absent	absent	absent	present	possible
S326V0708	absent	present	absent	absent	present	absent	present	absent	absent	absent	present	absent
S443V1525	unobservable	unobservable	unobservable	unobservable	unobservable	unobservable	present	absent	present	absent	present	present
S316V0641	present	absent	absent	present	absent	absent	present	present	absent	present	present	present
S293V0495	present	absent	present	absent	present	present	present	absent	absent	absent	absent	absent
S062V0071	absent	unobservable	present	present	present	present	absent	present	unobservable	present	present	present
S127V0204	present	absent	absent	absent	present	absent	absent	absent	present	present	present	present
S343V0732	absent	present	absent	present	present	present	unobservable	unobservable	unobservable	present	unobservable	unobservable
S141V0223	unobservable	unobservable	unobservable	unobservable	unobservable	unobservable	present	absent	present	absent	present	present

S046V0023	S187V0267	S082V0084	S058V0092	S165V0242	S158V0230	S320V0662	S421V0940	S284V0446	S412V0888	S181V0407
present	absent	absent	unobservable	absent	absent	absent	absent	unobservable	absent	present
absent	absent	absent	unobservable	absent	absent	absent	unobservable	unobservable	present	present
absent	present	absent	unobservable	absent	absent	absent	absent	unobservable	present	absent
absent	absent	absent	unobservable	present	absent	present	present	unobservable	absent	absent
present	present	present	unobservable	present	present	present	absent	unobservable	present	present
absent	absent	absent	unobservable	absent	absent	absent	absent	unobservable	present	present
present	absent	unobservable	present	present	absent	present	present	present	absent	unobservable
absent	absent	unobservable	unobservable	absent	absent	present	unobservable	present	absent	unobservable
present	present	unobservable	absent	present	absent	absent	unobservable	present	present	unobservable
absent	absent	unobservable	present	absent	present	present	absent	present	absent	unobservable
absent	present	unobservable	present	present	present	absent	present	present	present	unobservable
absent	absent	unobservable	present	absent	absent	present	possible	present	absent	unobservable

S287V0450	S072V0033	S050V0042	S352V0747	S152V0244	S227V0297	S314V0655	S335V0711	S048V1520	S493V1069	S037V0021
unobservable	present	unobservable	present	absent	absent	absent	absent	absent	absent	absent
unobservable	present	unobservable	absent	present	absent	absent	present	present	absent	absent
unobservable	unobservable	unobservable	present	absent	absent	absent	absent	absent	absent	absent
unobservable	absent	unobservable	present	present	present	present	present	present	present	present
unobservable	present	unobservable	present	present	present	present	present	present	present	absent
unobservable	present	unobservable	present	present	absent	absent	present	present	absent	absent
present	unobservable	present	present	present	absent	present	absent	unobservable	absent	present
absent	unobservable	absent	unobservable	present	present	absent	present	unobservable	absent	absent
present	unobservable	present	unobservable	absent	absent	absent	absent	unobservable	present	present
present	unobservable	present	present	absent	present	present	absent	unobservable	absent	absent
absent	unobservable	present	present	present	present	present	present	unobservable	present	present
present	unobservable	present	present	present	present	present	absent	unobservable	absent	present

S301V0583	S389V0857	S140V0207	S104V0136	S282V0417	S367V0803	S549V1181	S286V0469	S269V1506	S044V0027	S323V0650
unobservable	present	present	present	absent	present	present	present	absent	present	absent
unobservable	present	present	present	unobservable	unobservable	present	absent	absent	unobservable	absent
unobservable	present	present	present	present	absent	absent	present	present	unobservable	present
unobservable	absent	absent	absent	absent	absent	absent	absent	absent	absent	present
unobservable	present	present	present	present	absent	absent	absent	present	present	present
unobservable	present	present	present	possible	absent	absent	absent	absent	possible	present
present	present	absent	present	unobservable	present	present	present	unobservable	present	absent
present	absent	present	absent	unobservable	absent	present	absent	absent	present	present
absent	absent	absent	present	unobservable	absent	absent	absent	absent	unobservable	present
present	absent	absent	present	unobservable	absent	absent	absent	absent	present	present
present	present	present	present	present	present	absent	present	present	present	present
present	absent	absent	present	unobservable	absent	absent	absent	absent	present	present

S365V0773	S503V1099	S248V0393	S334V0716	S396V0877
present	present	present	present	present
absent	absent	absent	present	absent
absent	present	absent	absent	present
absent	absent	absent	present	absent
present	absent	present	absent	present
absent	absent	absent	present	present
present	present	present	absent	present
present	absent	absent	present	absent
absent	absent	present	absent	present
absent	absent	absent	present	absent
present	present	present	present	present
present	absent	present	present	possible