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A BIOARCHAEOLOGICAL AND HISTORICAL ANALYSIS OF SCURVY IN EIGHTEENTH AND NINETEENTH CENTURY ENGLAND

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Supervisor: Dr Andrew Shortland
Dr Simon Mays
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Abstract

The identification of metabolic diseases is a crucial aspect of osteoarchaeological analysis and of paleopathological studies. This study is specifically concerned with the study of scurvy and its bony manifestation. This investigation considers the recognition of the bony lesions of scurvy in adult skeletons that originate from English archaeological contexts dating to the Post Medieval period. In order to identify scorbutic bony lesions, assemblages were analysed that derived from the Georgian period Navy that were known to suffer from endemic scurvy, namely Haslar hospital near Portsmouth and Stonehouse hospital in Plymouth. These assemblages were complemented by two Non-Naval skeletal collections of a broadly contemporaneous time period, one of which was a prison assemblage from Oxford Castle in Oxford and the other was from Darwen, Lancashire and consisted of a Primitive Methodist cemetery.

For the purpose of this study, an extensive literature review was carried out and a specially modified scurvy recording form was created. In total three hundred and fifty-eight skeletons were analysed using the scurvy recording form on which a total of twenty-one potential scorbutic indicators were scored. The data was then subject to statistical analysis and a set of primary and secondary scorbutic indicators was established. The primary scorbutic lesions were femur, sphenoid, posterior maxilla, scapula, endocranial and mandible. Nine secondary lesions were also established and these were lesions of the foot, humerus, ulna, radius, hand, clavicle, innominate, fibula and the ectocranial surface of the skull. In total, 66.7% of the Haslar assemblage was found to have suffered from scurvy, followed by Plymouth with 20.6%, Darwen with 16.4% and Oxford Castle with 7.9%.

It was found that scurvy could be identified in adult skeletal material through the recognition of a number of lesions that could not be attributed to any other disease process. The results indicated that scurvy was present in all of the skeletal collections studied but was more common in the Naval assemblages. This is an important development in the detection of scurvy in the archaeological record and is crucial in the reconstruction of past diets and metabolic disease patterns.

Keywords:

Post Medieval, Royal Navy, paleopathology, haemorrhage, Vitamin C, periostitis, potato eating.

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Chapter 1: Introduction

1.1 Introduction

Bioarchaeologists aim to reconstruct past life patterns through the examination of archaeological human skeletal remains (Roberts and Manchester 2005). This allows bioarchaeologists to reconstruct past health and disease models, a vital aspect of which is the study of metabolic diseases (Brickley and Ives 2008). Metabolic bone diseases are those disorders that affect the process of bone modelling and remodelling (Albright and Reifenstein), which under normal circumstances allow the skeleton to grow and respond to stimulus. Metabolic bone diseases are an extremely worthwhile aspect of palaeopathological study was they can provide valuable data on past lives due to the multifactorial causation of metabolic conditions. These factors can include diet, environment, culture and hormones. However the majority of metabolic bone diseases are related to nutritional deficiencies and lack of nutrients in the diet which affect the bone modelling and remodelling process. There are many well-known metabolic bone diseases which include; osteoporosis, iron-deficiency anaemia, rickets, osteomalacia, Paget's disease and scurvy. Many bioarchaeologists refer to the metabolic diseases as 'indicators of stress', consequently this term has become commonly used in osteoarchaeological studies (Brickley and Ives 2008).

This research is concentrating on the occurrence and the skeletal manifestation of scurvy. It is an ancient metabolic disease, thought to have been first recorded in the Ebers papyrus approximately 1550BC and still known to occur occasionally today in individuals that are undernourished (Dolberg et al. 2010; Fain 2005; Hirschmann and Raugi 1999). Scurvy is a disease of malnutrition that is particularly associated with the Age of Exploration and the

advent of long sea voyages to exotic locations like India, Europe and the East Indies (Cook 2004, 224). It is caused by a lack of Vitamin C in the diet, as humans cannot produce their own Vitamin C (Fain 2005, 124). Vitamin C is also known by the term 'ascorbic acid', however throughout this thesis, Vitamin C will be used. Vitamin C is commonly found in fresh fruit and vegetables (Carr et al. 1999, 1086). This disease has been largely neglected in palaeopathological studies, particularly when it occurs in adults (Geber et al. 2012; Van der Merwe et al. 2010 b). This is primarily because scurvy is a disease of the soft tissue like blood vessels, muscles and ligaments (Popovich et al. 2009). However in the more advanced stages of the disease it can affect the musculo-skeletal system (Fain 2005, 125) and it can therefore cause skeletal lesions.

Scurvy is a chronic metabolic disease that can manifest as a whole suite of clinical symptoms. The symptoms of scurvy are directly related to the primary function of Vitamin C, which is that of stable collagen production. Collagen is the primary component of many essential bodily structures for example skin, bones, tendons and ligaments. Humans typically need to maintain a constant body pool of Vitamin C of 1500-2000mg and this can be sustained by a daily Vitamin C intake of approximately 30mg. Scurvy occurs when the human body pool of Vitamin C falls below 350mg. When the human body is in a scorbutic state, no collagen can be produced and therefore no healing or no new collagenous structures can be produced. This typically leads to haemorrhageing caused by resulting frailty of blood vessels. Initially this haemorrhageing may be mild but over time it becomes chronic and causes musculo-skeletal symptoms such as pain, periosteal haemorrhageing, osteoporosis and hemarthrosis (Fain 2005, 126). Over time as scurvy worsens it can lead to death due to severe bleeding and myocardial infarction.

There is significant documentary evidence to suggest that the primary study populations suffered from chronic recurring scurvy (Rogers 1988; Lloyd 1963; ADM 102). The two primary study assemblages both originate from Georgian period Royal Naval Hospital cemeteries. The Georgian era covers the period from 1714 to 1837. The individuals buried in these cemeteries would have served in the Royal Navy and would have been in active service when they died. It was during this time that the power of the Royal Navy was at its height and long voyages were routine. It is these long voyages and a lack of fresh provisions that caused scurvy to occur. These individuals are an ideal archaeological study population to assess the skeletal manifestation of endemic scurvy in dry human skeletal material because it was so widespread in the Georgian period Royal Navy (Rogers 1988, 100).

1.2 Aims and Objectives

The study aims to establish the presence of scorbutic lesions and to investigate environmental trends in the occurrence of scurvy in four post-medieval skeletal assemblages. The first aim of this research is to establish whether or not scurvy was commonplace in the post medieval period by examining these skeletal assemblages and complementing this data with historical documentation. This research then aims to establish whether or not adult scurvy can be identified in dry human bone through the macroscopic analysis of four contemporaneous post medieval skeletal assemblages. These assemblages all derive from working classes dating from the eighteenth and nineteenth century in England. The two Naval skeletal assemblages represent individuals that would have worked on the lower decks in the Royal Navy (Boston et al. 2005) and died between 1753 and 1826. These two groups derive from the cemeteries at Haslar Naval hospital near Portsmouth and Stonehouse Naval hospital in Plymouth. The non-Naval comparative collections derive from

a poor labouring Primitive Methodist group from Darwen, Lancashire (1832-1861) and the last is a group of prisoners that were imprisoned and died in Oxford Castle prison (17-19th century). The objective is also to determine whether scurvy was as present in land populations as it was in the Navy of the eighteenth and nineteenth century. This research will also confirm if scurvy was as prevalent in the Georgian period Navy as popular opinion would suggest. Another of the primary objectives is to establish a methodology which can be used as a standard in the analysis of skeletal assemblages by ascertaining which, if any, bony lesions are the best indicators of scurvy in dry adult human bone.

The primary objective of this research is to establish a methodology in order to identify the lesions of adult scurvy through macroscopic analysis. It also aims to establish whether or not these lesions can be attributed unambiguously to just scurvy. This is because many of the lesions of scurvy such as periostitis can also be caused by other disease processes and are therefore usually referred to as 'non-specific lesions' (Roberts and Manchester 2005; Ribot and Roberts 1996). It is thought that there must be something distinctive about the patterning and location of the lesions of scurvy and this can be used to aid scurvy identification. It was decided that a scurvy recording form should be produced and used for analysis of each skeleton. The scurvy recording methodology will be generated from an extensive literature review by collating all known lesions that are associated with scurvy. Lesions will be recorded as being present/absent and described in detail. These will then be subjected to statistical analyses and compared to the clinical manifestation of scurvy. All of these lesions will have to be definitively linked to the clinical manifestation of scurvy and the pathophysiology of the scorbutic human body.

The secondary objective of this research is to examine the historical documentation to establish if scurvy was prevalent in the study populations. It is clear that there is a direct link between diet and the development of scurvy. In the published cases of palaeopathological adult scurvy, all have been associated with severely malnourished populations, for example those affected by famine, miners, explorers and the Tudor Navy (Geber et al. 2012; Van der Merwe et al. 2010 a and b; Crist et al. 2005; Stirland 2005). However no studies to date have tried to link the skeletal indicators of scurvy to an assemblage of individuals known to originate from the lower decks of the Georgian period Royal Navy. It is expected that the Naval assemblages being studied will show evidence of scurvy, as it is well known from historical documentation that the Navy of this period had very limited access to fresh fruit and vegetables on long voyages (Carpenter 1986). It was this dietary restriction that led to the development of scurvy in Naval crews. The hypothesis being examined in this research is that the Naval assemblages will show evidence of scurvy in their bones whereas the non-Naval collections would not show scurvy.

The next chapter will introduce scurvy, its symptoms and clinical manifestation. Vitamin C in food will also be discussed. Chapter three will examine the historical data available for both the Naval and the comparative assemblages and will examine the typical diet that would have been consumed by each group. Chapter four will present each of the study assemblages and also the methodology established for this research and the scoring system that will be used to assess each lesion. This chapter will also set out the methodology for evaluating preservation, completeness, age and sex demographic for each assemblage. In this chapter, primary data from the hospital logs from Haslar and Plymouth will be presented which gives the percentages of men suffering from scurvy in the study period.

Chapter five will present results that were obtained from the analysis using the scurvy recording form, along with full demographic profiles for each assemblage. Chapter six will discuss the results of the lesion analysis in light of the historical and medical data available and will establish the reliability of the methodology. Chapter seven will present the conclusions reached and further work proposed for this project.

This study will for the first time bring together osteological, medical and historical data in order to examine the complete picture of scurvy in the study assemblages. This is the first time that this kind of research has been undertaken in Georgian period Royal Naval skeletal assemblages and then compared to contemporaneous Non-Naval groups. This is also the first time that a post medieval prison population and a non-conformist Methodist population have been examined for evidence of scurvy. This study is important because of the potential benefits of a practical methodology for recognising scurvy in adult skeletal material since there is no current methodology for this that is accepted and used by all osteologists. This research has the potential to change the way scurvy is identified and recorded in adult skeletal remains and has great potential for use in archaeological and forensic contexts.

"The medical profession itself took a very narrow and very wrong view. Lack of ascorbic acid caused scurvy, so if there was no scurvy there was no lack of ascorbic acid. Nothing could be clearer than this. The only trouble was that scurvy was not the first symptom of a lack but a final collapse, a premortal syndrome and there is a very wide gap between scurvy and full health"

Albert Szent-Gyorgyi, Discoverer of Vitamin C and Nobel Prize Winner, taken from his Nobel Prize Acceptance Speech 1937. Reproduced from Stone 1972.

Chapter 2: The Role of Vitamin C in the body and the medical symptoms of scurvy

2.1 The Role of Vitamin C in the body and the medical symptoms of scurvy

In this chapter, the aetiology of scurvy will be considered first, along with the body's need for Vitamin C and its physiological functions within the human body. The manifestation of scurvy throughout the human body will be reviewed and the effect of scurvy on other disease processes and pathological changes.

2.1.1 What is Scurvy?

Scurvy is a metabolic disease defined as a deficiency of Vitamin C (ascorbic acid) in the human body (Fain 2005, 124; Halligan et al. 2005, 688; Pimentel 2003, 328). Scurvy is considered to be a metabolic bone disease (Brickley and Ives 2008). Metabolic bone diseases are typically defined as those diseases that affect the processes of remodelling and bone formation in the skeleton (Albright and Reifenstein 1948). Scurvy is a particularly enigmatic disease as the complexity of the disease process is still not fully understood and it

is known to affect only a few living creatures. Humans, guinea pigs, primates and some species of fish such as salmon and trout and some fruit bats cannot synthesise Vitamin C in their bodies and therefore have to consume an exogenous source of this vitamin in order to maintain health (Brickley and Ives 2008, 41). It is because of this that guinea pigs were commonly used to research the effects of Vitamin C deficiency (Chatterjee 1967; Manchester 1998, 170). The ability to synthesise Vitamin C that is held by most animals negates the need for a dietary source of Vitamin C (Stone 1961, 345). Most animals can produce quantities of Vitamin C far beyond their own bodily needs using enzymes in the glucuronic acid pathway to metabolise glucose that is ingested (Hirschmann et al. 1999, 898; Carr et al. 1999, 1086). In evolutionary history the site of Vitamin C synthesis seems to have migrated (Stone 1965), many birds have a transitional site of production; many mammals produce Vitamin C in the liver (Chatterjee et al. 1961), whereas in reptiles the production occurs in the kidney (Brickley and Ives 2008, 41). It is thought that in human evolutionary history the ability to synthesise Vitamin C was lost by humans as part of a developmental trade-off (Stone 1965). This is thought to have been because of high Vitamin C levels within the human diet in the past, so the ability to synthesise Vitamin C was lost.

The word scurvy itself seems to have a number of origins. One theory is that it gets its name from the Latin ‘scurbutus which means to rupture or to lacerate (Halligan et al. 2005, 688). The word scurvy itself only appeared in English in the sixteenth century (Hirschmann et al. 1999, 896). Others believe that the word originated from the low German ‘schorbuk’ which means ‘break belly’ because of the belief that scurvy was a disease that led to rupture (Hirschmann et al. 1999, 896). It is also true that the word scurvy was commonly used by Shakespeare to mean a discreditable or vile individual (Hirschmann 1999). He used it in King

Lear Act 4, Scene 6 when Lear implores the Earl of Gloucester to “Get thee glass eyes, and like a scurvy politician, seem to see the things thou dost not” (Shakespeare and Foakes 1997, 340).

2.1.2 What is Vitamin C?

Vitamin C is also known as ascorbic acid (AA) and is a naturally occurring acidic compound. Vitamin C is a water soluble vitamin (Naidu 2003, 7) with the chemical symbol of $C_6H_{12}O_6$, the chemical structure of Vitamin C can be seen in Figure 2.1. It is available from a wide

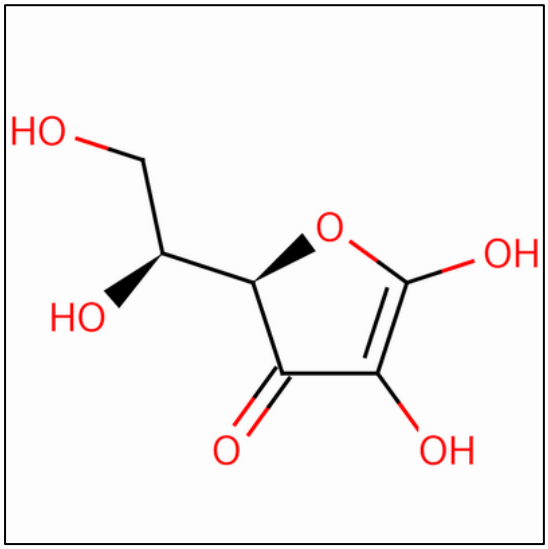

Figure 2.1 Structure of Vitamin C. Reproduced from www.medicalook.com.

range of fruit and vegetables and it is readily absorbed in the intestine (Manchester 1998, 169). It was discovered by Szent-Gyorgyi in 1932, (Dolberg et al. 2010, 183) it was artificially synthesised for the first time in 1933. It cannot be stored in the human body (Naidu 2003, 11) but by sustaining a constant dietary intake every day the maximum body pool of Vitamin C can be maintained (Fain 2005, 124). Vitamin C is artificially manufactured today and is available in a wide variety of tablets and powders.

2.1.3 Vitamin C Food Sources

Vitamin C is commonly found in fresh vegetables and fruit particularly citrus fruits, melons, berries and green leafy vegetables like broccoli and cabbage (Léger 2008, 1404), the content of Vitamin C for many fruits and vegetables can be seen in Table 2.1. Fruit and vegetables are the richest source of Vitamin C that are available to most people and provides 90% of all Vitamin C in Western diets (Hirschmann et al. 1999, 899). Vitamin C is found in highest content in vegetables and fruit with a high water percentage. It is also a naturally occurring antioxidant, these are molecules that inhibit the oxidation of other molecules such as free radicals, in the case of Vitamin C, it may help prevent cancer and atherosclerosis (Paddayatty et al. 2003). Other food stuffs do contain Vitamin C include some meats especially offal (Gabay 1993) but many everyday dietary staples like grains, fish, eggs and pasteurised dairy products contain little or no Vitamin C (0-2mg/100g) (Fain 2005, 124). It has become commonplace in many modern commercial food processes to fortify foods with Vitamin C, for example breakfast cereals. Dietary supplementation of Vitamin C through vitamin tablets and powders is also commonplace. Due to common fortification and dietary supplementation of Vitamin C, scurvy does not commonly occur in modern society (Hirschmann 1999, 899). Human milk contains particularly high amounts of Vitamin C which is why breastfeeding infants do not develop scurvy (Rajakumar 2001). Human breast milk contains 0.05mg of Vitamin C per cubic centimetre (cc), whereas cow's milk contains 0.02mg per cc (Selleg and King 1936).

Modern pasteurised milk has further diminished quantities of Vitamin C because of the high temperatures that are used in the pasteurisation process. Holmes et al. (1943, 340) found an 18% loss of Vitamin C when cow's milk was pasteurised. In spite of it being quite

commonly occurring, Vitamin C is an extremely sensitive vitamin and is easily destroyed by heat, oxygen, ultraviolet light, exposure to metal, alkaline and long term storage (Fain 2005, 124). Highly processed and cooked foods contain significantly lower levels of Vitamin C

Table 2.1 Typical Values of Vitamin C content in common foods of European diets. Adapted from Carpenter 1987 and Johnson et al. 1998.

Food Type	Weight of Item (g)	Vitamin C (mg)
Brussel Sprout – Raw	100g	85
Brussel Sprout – Boiled	100g	65
Broccoli- Boiled	155g	140
Cabbage- Boiled	170g	40-50
Potato- Raw	200g	40
Potato- Baked in Skin	200g	30
Potato- Boiled and Reheated	200g	5-15
Green Peas- Sold frozen and then boiled	160	20
Tomato- Raw	100	20
Carrots- Raw	155	9
Carrots- Boiled	155	6
Lettuce- Chopped	55	4
Bread, flour, margarine, chocolate, dried peas and beans	-	Zero
Strawberries- Raw	150	88
Orange -Raw and whole	180	66
Banana- with peel	175	12
Apple- Raw and whole	180	7
Liver- Beef or Lamb cooked	85	15-30
Cow's Milk(pasteurised) or Yogurt	250	2
Butter, cheese, eggs or well cooked meat or fish	-	Zero
Rose-Hips –Raw	100	250-800
Blackcurrants- Raw	100	150-200
Onion- Raw	100	10-15
Lemon- Raw	100	40-50

Vitamin C than their uncooked equivalent (Blee et al. 2002, 410). This loss is partly due to the water solubility of Vitamin C, leading easily into cooking water (Tressler et al. 1936, 909). In reference to the Table 2.1, the loss of Vitamin C between cooked and uncooked food is visible. For example raw potato contains approximately 20mg of Vitamin C per 100g, but

when potatoes are cooked and reheated, this is reduced to between 2 and 7mg per 100g. In a cup of raw carrot there is approximately 9mg of Vitamin C, in a cup of boiled carrot, it is reduced to 6mg. The process of cooking is complex and it is the combination of heat, water and exposure to metal cooking vessels that causes the destruction of Vitamin C. Cooking of fruit and vegetables usually reduces the amount of Vitamin C by between 20-40% (Levine et al. 1999).Cooking in large quantities of water, in copper pots (Jones and Hughes 1976) and for long periods of time further reduces the Vitamin C content. Therefore it is best to consume fresh raw fruit and vegetables as these contain the highest levels of Vitamin C.

2.1.4 Vitamin C Recommended Allowance

Most governments and the World Health Organisation (WHO) have published dietary guidelines for vitamin and mineral intakes for adults in order to maintain good health. The United Kingdom government stipulates that the recommended daily allowance (RDA) for Vitamin C is 40mg per day (www.nhs.co.uk). The RDA is defined as the average daily dietary intake level that is enough to meet most healthy individuals nutritional requirements .This is roughly equivalent to the current government recommended five servings of fruit and vegetables (Halligan et al. 2005, 692). In actuality, the human body needs approximately 30mg of Vitamin C a day to maintain a healthy metabolic balance and to sustain the body pool of between 1500-2000mg (Cheung et al. 2003, 248). It is important to ingest this much Vitamin C every day because the half-life is so short at between 10-20 days (Naidu 2003, 10). There is a saturation level of Vitamin C in the human body which is about 20mg per kg, where the body simply cannot absorb anymore (Gabay et al. 1993; Sauberlich 1990; Shamsaddini et al. 2001). When this saturation point is reached and Vitamin C is then excluded from the diet it will take longer for the clinical symptoms of scurvy to appear,

conversely scurvy will appear very quickly in individuals who have a low body pool and are then placed on a restrictive diet. Scurvy will occur when the body's total pool of Vitamin C falls below 350mg (Pop-Jordanova 2008, 2; Fain 2005, 124). The bodily absorption of Vitamin C is usually balanced by the bodily need for Vitamin C and any excess is excreted by the body. Absorption of Vitamin C occurs in the ileum via active transport and it seems that the maximum amount that can be absorbed is 180mg/day (Fain 2005, 124). There are no known major consequences of excess Vitamin C and some medical professionals advocate megadoses (1000s mg/day) which they believe improve mortality in diseases like cancer and "many biochemical, clinical and epidemiologic studies have indicated that Vitamin C may be of benefit in chronic diseases such as cardiovascular disease, cancer and cataract" (Carr et al. 1999,1087). Linus Pauling, the American chemist and winner of two Nobel prizes, was a staunch advocate of Vitamin C megadoses and he himself took 3g of Vitamin C every day and died at the age of 93 (Dunitz 1996). Pauling believed that Vitamin C was crucial for cold prevention and that it could increase quality and length of life for the sufferers of terminal cancer (1979, 1976). The results of his clinical studies have been disputed, but the fact remains that excess Vitamin C has no ill consequences and may be beneficial beyond its well-known physiological functions (Naidu 2003).

The clinical symptoms of scurvy will appear after 3-6 months on a diet totally deficient of Vitamin C (Hirschmann et al. 1999, 895). The daily allowance and bodily requirement is dependent on a number of factors including pregnancy/lactation, smoking, activity levels, age, alcohol consumption and health status (Pimentel 2003, 331). Stress, smoking, fever and infections can cause rapid declines in body levels of Vitamin C (Naidu 2003, 9) and therefore scurvy can set in quickly when the body is harassed by disease or stress. Smoking has a

particularly adverse effect on the body pool of Vitamin C, since it increases catabolic turnover and means smokers need on average an additional 130mg of Vitamin C in their daily diet (Schectman et al. 1989, 162). It is important to note that smoking was introduced to England in the period being studied in this research and this will be discussed further in Chapter three. Alcohol is also known to increase Vitamin C metabolism and alcoholics are among the few groups that regularly present with scurvy in modern clinical contexts (Léger 2008). However this is not to say that scurvy is rare and in modern clinical contexts scurvy has also been known to occur in faddy eaters, homeless individuals, adults living alone, the institutionalised elderly, refugees and those with psychiatric disorders or long term illness and infection (Hirschmann et al. 1999, Pimentel 2003, Desenclos et al. 1989, Léger 2008). Vitamin C depletion can also be found in apparently healthy individuals. In the early 1990's Hercberg (1994) found that 5% of women and 12% of men had less that 2mg/l of Vitamin C in their blood from a French group of over 1100 healthy non-hospitalised patients (Hercberg 1994, 230). A healthy individual should have between 8-10mg/l in their blood (Galan et al. 2005). A similar study carried out in America by Johnston (1998) found that nearly 37% of the study group had Vitamin C depletion. These studies suggest that many people do not have adequate levels of Vitamin C in their bodies and may suffer from some of the effects of sub-clinical scurvy and even a small percentage may unknowingly be suffering from the early effects of scurvy. However it is clear that these studies do not take into account other factors that may deplete the body pool of Vitamin C. Vitamin C depletion also increases the risk of developing myocardial infection, cataracts and infection (Fain 2005, 127).

When scurvy does occur, Vitamin C is one of the best examples of a medical 'magic bullet' (Glouberman 2009, 555). It is referred to as a 'magic bullet' because when someone is

suffering, even from severe chronic scurvy, a very small amount of Vitamin C can act like a miraculous cure and even the acute symptoms can be cured in a short period of time. Modern clinical practice is to treat those suffering with scurvy to intravenous or oral megadoses of 1-2 grams of Vitamin C every day for two weeks (Fain 2005, 127). Improvements will be visible within a few days to a week and scorbutic bleeding has been known to stop in 48 hours (Leger 2008, 1404; Halligan et al. 2005, 692).

2.1.5 Essential Bodily Functions of Vitamin C

Vitamin C has a multitude of biochemical and physiological functions within the human body and the multitude of its clinical symptoms reflect these roles. What is frequently considered

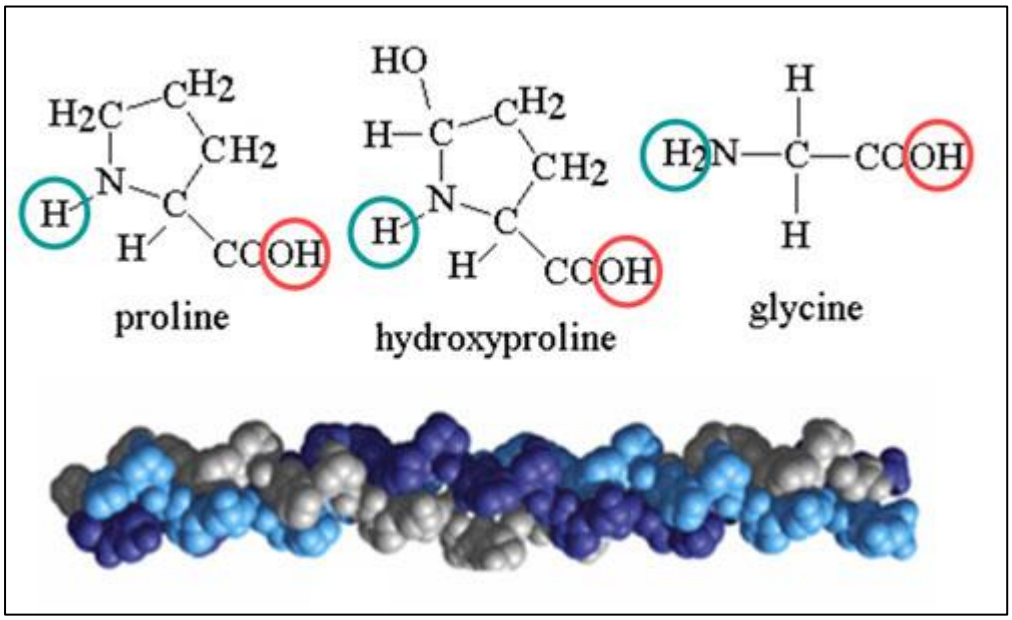

Figure 2.2 Diagram showing the triple helical structure of collagen. Reproduced from www.chemistryland.com.

to be the most important function of Vitamin C is the production of collagen (Barnes 1975). Vitamin C is essential for the hydroxylation of collagen (Arrigoni et al. 2002, 5). Vitamin C is an essential co-factor of the synthesisation of the polypeptide molecules lysyl and prolyl, which are hydroxylated using Vitamin C to hydroxyprolyl and hydroxylysl (Englard 1986).

This change in the collagenous structure ensures that the triple helical collagen structure can be maintained which is vital for its fixed arrangement (Hirschmann et al. 1999, 898).This triple helical structure that makes collagen to structurally stable can be seen in Figure 2.2.

When no Vitamin C is present there can be no production of structurally stable collagen, therefore the collagen that is produced is weak and defective and cannot fulfil its function (Blee et al. 2002, 409; Dolberg et al. 2010, 184). Collagen is the building block that holds the human body together, it is one of the most abundant proteins present in the body and makes up a large part of mesenchymal tissue which includes connective tissues such as skin, tendons, inter-vertebral discs, muscle, ligaments, blood vessels and the organic component of bone (McCormick 1954, 29). Some of these tissues such as muscles, skin and gums contain a greater concentration of collagen and this makes these more susceptible to Vitamin C deficiency (Léger 2008, 1404). It has also been shown that Vitamin C plays a role in the structural maintenance of intercellular 'cement' which is vital in the organisation and composition of endothelial blood vessels and other endothelial structures such as lymphatic vessels (Fain 2005, 126; Popovich 2009, 411).

The collagen formation function makes Vitamin C a particularly vital component in the formation of bone and teeth especially in childhood and is essential for maintenance and repair of collagenous structures in adults. If adequate levels of Vitamin C are not present then osteogenesis cannot occur and bone growth completely ceases, which is a major consequence of scurvy in subadults (Brickley and Ives 2006, 163). This also affects dentinogenesis (tooth growth) which can lead to enamel hypoplasia if the individual recovers from a scorbutic episode, which has been known since the 1700s (Bunon 1746 in Hillson 1992, 461). "Defective bone matrix , cessation of bone growth, and failure to ossify

result in musculoskeletal limb pain, limping, swelling over long bones, progressive leg weakness , pseudoparalysis and fractures" (Popovich et al. 2009, 411) are some of the very serious effects of sub adult scurvy. Osteoporosis and osteopenia can also occur in adults as "Vitamin C depletion results in defective osteoid matrix formation and in increased bone resorption" (Fain 2005 126).

Vitamin C is also a co-factor in the production of carnitine, norepinephrine and some peptide hormones (Hirschmann et al. 1999, 899). Carnitine is an essential ammonium compound that is synthesised from amino acids. Its function is the transport of fatty acids into cell mitochondria to manufacture metabolic energy and is important in the functioning of the cardiovascular system (Sinatra et al. 1999). It is not surprising then that early symptoms of scurvy include fatigue and lassitude. Norepinephrine is a catecholamine which functions as a hormone and neurotransmitter. It has a whole host of functions including controlling blood pressure, controlling the 'fight or flight reflex', decision making, attention span, mood, regulating heart rate and blood pressure (Tanaka et al. 2000, Ordway et al. 2007). There are some peptide hormones that depend on Vitamin C as a co-factor for synthesis, these proteins have important immunologic and endocrine functions that produce effects on many body tissues including bone and muscle (Blalock et al. 1985, 858).

2.1.6 Vitamin C and Anaemia

Vitamin C is also closely linked to the formation of iron-deficiency anaemia which is another complex metabolic disease (Leger 2008, Halligan et al. 2005, and Dolberg et al. 2010). Anaemia is caused by a lack of dietary iron and leads to underproduction of haemoglobin which carries oxygen around the body. The signs of anaemia are pallor, fatigue and can be clinically recognised by low iron levels in the blood. The link between scurvy and anaemia is

complex one, as scorbutic haemorrhages can cause heavy blood loss which exacerbates anaemia, Vitamin C also aids the absorption of dietary iron in the intestines by reducing it from its ferric to ferrous form, which in turns increases its obtainability in the body. An important point to note is that Vitamin C is essential for the absorption of non-haem iron (i.e. iron from vegetable sources) which is not as easily absorbed as haem iron from animal sources (Roberts and Manchester 2005, 226). Anaemia may be present in up to 75% of patients that present with scurvy (Ozturk et al. 1999 245) and often this serves to complicate the clinical picture of scurvy.

2.1.7 Vitamin C and Immunity

Vitamin C is also vital for immune system integrity; this vital function is twofold and consists of its antioxidant role and that of pathogen destruction (Thomas and Holt 1978, 370). The role of Vitamin C is primarily as an antioxidant and it has also been shown that Vitamin C stimulates immune response and enhances T-cell production in response to infection; T-cells are white blood cells that have a wide variety of functions in immunological function (Wing et al 2008). It has been frequently been noted that scorbutic individuals are more susceptible to infectious disease (Hess 1920).

2.1.8 Summary

In the preceding section, the aetiology of scurvy has been discussed along with the specific physiological functions of Vitamin C. Food sources rich in this vitamin were also discussed, along with the recommended daily allowance of Vitamin C. This vitamin is essential to a great number of physiological functions in the human body and its deficiency is detrimental to health because of these. The most important of these functions is that of collagen formation, which is necessary to maintain integrity of vital bodily infrastructures like skin,

bone, muscles and tendons. The pathological symptoms of scurvy will be described in detail shortly and these all fit with its multitude of roles in the body from collagen production, energy production, immune maintenance and hormone regulation. It is clear that even sub clinical scurvy can cause health problems and can adversely affect vital physiological processes.

2.2 The role of Vitamin C in overall body health

2.2.1 Introduction to the role of Vitamin C in overall body health

It is clear that Vitamin C has a great number of very specific functions within the human body and is universally beneficial to health even excluding these roles (Arrigoni 2002, 1). Scurvy is an extremely complex disease process. In a scorbutic state, the body is much more vulnerable to infections, other metabolic diseases and poor wound healing (Blee et al. 2002, 410; Olmedo et al. 2006, 912). This effect has even been noted by historical authors such as James Lind who believed that those with that suffer from disease were more likely to suffer from scurvy "when the fever leaves the patient very low, especially if he has had a flux, with which the scurvy associates itself more readily than with most other disorders, this return of the scurvy often proves fatal" (Lind 1777, 507).

Vitamin C is related to numerous aspects of immune response and of particular note is the high ascorbate content of white blood cells (Thomas and Holt 1978, 370). Vitamin C deficiency can cause the defective production of interferon. Interferons are proteins made in response to pathogens in response to bacteria and viruses. There is significant evidence that the interferon produced in scorbutic individuals is defective and is produced in lower quantities than normal (Scrimshaw and SanGiovanni 1997). There are several disease

processes associated with scurvy, these are listed in Table 2.2 along with their potential skeletal manifestation, these will be examined in the next paragraphs or will be considered later as a scorbutic lesion.

Table 2.2 Conditions associated with scurvy and their potential skeletal manifestation.

Conditions Associated With Scurvy	Potential Skeletal Manifestation
Vitamin D Deficiency	Rickets in Children and Osteoporosis/Osteopenia in Adults
Infection/Infectious Diseases	Osteomyelitis, Periostitis
Degenerative Joint Disease	Degenerative changes of the joints and Schmorl's Nodes

There is a complex relationship between Vitamin C deficiency and diseases impacting on the body. Some of these are caused directly by the deficiency, others indirectly, in general these are a result of a number of causative circumstances interacting with each other. Those diseases include; osteoporosis, rickets, non-specific infections, Schmorl's Nodes and degenerative disc disease and increased risk of fracturing and delayed epiphyseal fusion.

2.2.2 Osteoporosis and Osteomalacia

Deficiency of Vitamin C is highly likely to be contributory to osteoporosis in adults (Barratt et al. 1996, 702) because of the bone turnover abnormalities caused by scurvy. This is because when the human body is scorbutic, there is still bone remodelling taking place, i.e. bone is being removed but it cannot be adequately replaced because of the lack of viable bone collagen. Osteoporosis is metabolic disease that is usually concerned with lack of Vitamin D and calcium in the diet and is associated with decreased bone mineral density (Brickley and Ives 2008, 151). Osteoporosis is defined by the WHO as a bone mineral density two and a half standard deviations lower than peak bone mass (World Health Organisation 1994). Osteoporosis frequently leads to increased susceptibility of bone fractures (Brickley and Ives

2006, 188). The relationship between Vitamin C deficiency and osteoporosis was examined in a study conducted on the Johannesburg Bantu in the 1960's looking at patients that presented in hospitals with the clinical and radiological characteristics of scurvy (Seftel 1966, 642). All of the individuals presenting with scurvy also displayed radiological signs of severe spinal osteoporosis and in two cases this was confirmed by autopsy findings (Seftel 1966, 644). Biopsy samples were taken and there was a significant decrease in bone trabeculae, absence of osteoblasts, higher than normal osteoclasts and larger than normal marrow cavities. The two individuals that were autopsied showed extreme brittleness and fragility of the highly trabecular bones of the ribs, vertebrae, sternum and pelvis and many of the vertebral bodies had collapsed (Seftel 1966, 643). The samples in this study had a mean age of 47 which may indicate that some of the osteoporosis may be age related, however some of the findings were observed in individuals as young as 30. Seftel's (1966) showed similar patterns to another Bantu study which showed that 69% of their osteoporotic patients were also scorbutic (Grusin et al. 1957, 647). In a large American study, it was found that women with high Vitamin C dietary intake had greater bone mineral density (Simon and Hudes 2001, 432). Modern clinical case studies also report scurvy and osteoporosis concurrently (Barratt and Summers 1996; Pimentel 2003; Barratt 1996). Joffe (1961) considered osteoporosis of the spine and compression fractures to be the commonest radiographic finding in scorbutic individuals. Hall et al. (1998, 187) conducted a study on Vitamin C supplementation of post-menopausal women and showed that when Vitamin C was combined with an adequate calcium intake, it resulted in a substantial increase in bone mineral density. It is also likely that scurvy is also linked to the occurance of osteomalacia, which is the adult form of rickets that is caused by Vitamin D deficiency, however no references to these occurring together in adults could be found in the medical literature.

These examples illustrate the complexity of the relationship between Vitamin C, calcium and bone mineral density and it also demonstrates the complicated link between different metabolic diseases.

2.2.3 Schmorl's Nodes and Degenerative Disc Disease

Schmorl's Nodes are herniations of the collagenous structure that are found between the vertebrae, that then enters the vertebral body (Faccia et al. 2008, 29). Between each vertebra is a cushion composed of an external ring known as the annulus fibrosis and an

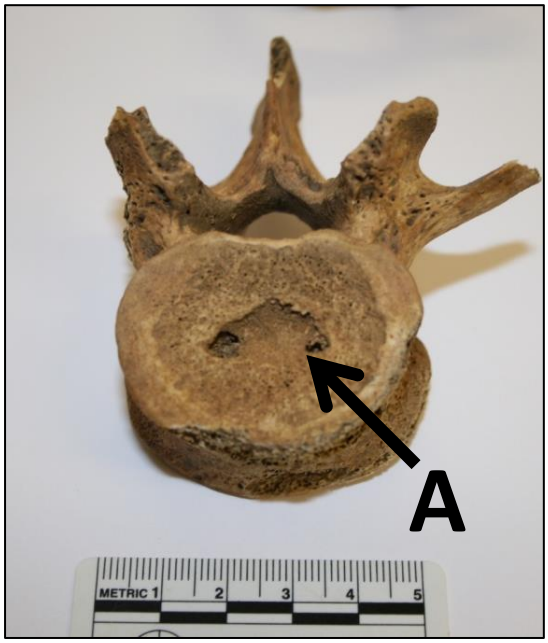

Figure 2.3 Photograph Showing Schmorl's Node, characterised by a depression in the vertebral body and marked by A.

internal pillow of jelly known as the annulus pulposis (Roberts and Manchester 2005, 139). A Schmorl's node occurs when the annulus fibrosis ruptures and the pulposis leaks out and is commonly known as a slipped disc (Wagner et al. 2000). Since these are collagenous structures their integrity is compromised when the body is in a scorbutic state "it is only logical to conclude that lesions ...such as we now find in the intervertebral discs...may be etiologically related to deficiency of Vitamin C" (McCormick 1954, 33). The osseous marker

of this herniation is a small depression or indentation in the vertebral body as shown in Figure 2.3. McCormick supports his association of scurvy with Schmorl's Nodes with scurvy with some interesting results from his own case studies where all of those patients that presented with intervertebral disc lesions were heavy smokers and had very low Vitamin C blood levels (McCormick 1954, 32). There is thought to be a similar link between degenerative disc disease (DDD) and low Vitamin C levels. With its onset, DDD causes inflammation which necessitates the need for more Vitamin C which in turn leads to a positive feedback model that causes to more degeneration (Smith 2010, 696). It has also been suggested that dietary deficiency of Vitamin C can also lead to increased joint degeneration and osteoarthritis. There have been studies of the links between these and the results seem somewhat contradictory. McAlindon et al. (1996) reported benefits of taking large doses of Vitamin C in reducing arthritic pain and slowing joint degeneration. Other studies have reported no advantage to taking Vitamin C supplement in degenerative joint disease (Wluka et al. 2002). In the guinea pig model, it has been shown that Vitamin C deficiency can increase the severity in the occurrence of spontaneous knee arthritis (Kraus et al. 2004, 1822). It is obvious that there are complex interactions between vertebral disc and joint degeneration and Vitamin C deficiency, but it is clear that it is necessary for cartilage maintenance (McCormick 1954).

2.2.4 Delayed Epiphyseal Fusion

Delayed epiphyseal fusion caused by scurvy in teenagers and young adults is something that was recorded by Dr James Lind at Haslar Hospital (Lind 1772). During the course of autopsies carried out at Haslar, he noted "crepitus" which is a grating or crackling sound in the joints and during autopsies he saw that the epiphyses had become separated from the diaphysis

(Lind 1772, 243). Lind's treatise cites earlier works such as Willis' Tractus *de Scorbuto* (1667) who also notes grating and crackling of the joint. He associated this with an incurable stage of the disease (McCormick 1954, 30). Crepitus is also something that has been frequently noted in infantile scurvy (Hartman and Friedman 1931, 337). In young adults, this crepitus can lead to delayed fusion of the long bones and can potentially lead to discrepancies in the osteological ageing of these individuals.

2.2.5 Disease Processes Influenced by Vitamin C Deficiency

Table 2.2 shows a number of pathologies that are affected by Vitamin C deficiency that can manifest in the bones. Probably the most significant of these is a greatly increased susceptibility to infections (Fain 2005, 127, Halligan et al. 2005, 691). It is difficult to infer how often this occurred from the osteological record as acute infections cannot usually be recognised through bony changes. However it is clear that many diseases that do manifest in the bones are indirectly linked to Vitamin C deficiency such as Schmorl's Nodes. This were not directly considered in this study due to poor overall preservation of vertebrae across all four assemblages. Osteoporosis was not examined as radiography was not consistently used. However, it is hoped that future studies may be able to assess these processes and their link to Vitamin C deficiency.

2.3 The Pathological Symptoms of Scurvy/Clinical Features of Scurvy

The pathological symptoms of scurvy are widespread and systematic, have a negative influence on mental state and are a reflection of, and directly related to, essential functions of Vitamin C (Olmedo et al. 2006, 912). The manifestation of scurvy will be described from its initial symptoms right through to the most severe expression of the disease. As

mentioned previously, scurvy tends to affect certain economic groups particularly economically disadvantaged populations, alcoholics, the elderly, faddy eaters, those with eating disorders, convalescents and those with underlying chronic diseases (Popovich et al. 2009,413; Halligan et al. 2005, 688) and even those with cancer (Fain 1998, 1661). Scurvy affects infants and adults differently as the growth process is complete in adults. Infant scurvy is a well understood paleopathological process and will not be described here, since this thesis deals with adult populations.

2.3.1 Physiological Manifestation of Adult Scurvy

The physiological indications of scurvy are primarily as a consequence of the faulty production of collagen within the body while it is in a scorbutic state. Scurvy affects the whole body because "loss of Vitamin C affects all tissues of mesodermal origin, thus there is an impairment of the cohesive property of matrix connective tissue and endothelium" (Shorbe 1953, 49). The clinical manifestation of scurvy is usually apparent in between 1-3 months with no dietary Vitamin C intake (Fain 2005) The associated non-specific symptoms of scurvy include weight loss, pallor, lassitude, weakness and muscle and joint pains (Halligan et al. 2005, 691). The onset of scurvy has been studied in prison populations by Hodges et al. (1971). In this study, the first signs of scurvy appeared when blood serum levels of Vitamin C fell to between 0.13-0.24mg/100ml (Hodges et al. 1971, 435). Pathognomonic symptoms of scurvy often include multiple haemorrhagic sites throughout the body (Hirschmann and Raugi 1999, 898). These haemorrhagic manifestations throughout the body are due to the fragility of the blood vessel walls and weakening of the endothelium, these lesions are usually symmetrical in nature (Brickley et al. 2008). These haemorrhages can be exacerbated by even minor trauma due to the extreme fragility of the

blood vessels (Hirschmann and Raugi 1999, 902). Other indicative signs of scurvy include; corkscrew hairs, petechiae, ecchymoses, gingival bleeding and tooth loss, hemarthroses and pupura (Popovich et al. 2009, 406). All of these lesions will be each discussed in detail and have been divided into four broad categories; behavioural, dermatological, oral and musculoskeletal manifestations.

2.3.2 Behavioural Symptoms

It is broadly believed by many of those in the medical profession that the first non-specific symptoms of scurvy are subtle behavioural signs. In the Kinsman and Hood (1971) Iowa Prison study, where a small group of prisoners were placed on a scorbutic diet (lacking Vitamin C), it was found that prisoners became un-cooperative, depressed, lazy, and easily fatigued. There were also huge personality changes recorded and all the test subjects became very depressed (Kinsman and Hood 1971). The personality changes were followed by a degeneration of psycho-motor response and decreased work output (Kinsman and Hood 1971, 455). Lind (1772, 99) also states that a 'universal lassitude' is an early symptom of scurvy accompanied by laziness and a distinct disinterest and listlessness. These symptoms have also been reported by many other authors in modern medical case studies of scurvy (Leger 2008, Halligan et al. 2005, and Dolberg et al. 2010). This is probably linked to the detrimental affect the Vitamin C deficiency has on neurotransmitter function in the brain (Katsuki 1996) and the fatigue is due to fatty acids not being transported to the cell mitochondria for energy production (Leger 2008, 1404).

2.3.3 Cutaneous or Dermatological Symptoms

The first externally visible symptoms of Vitamin C deficiency are those that occur in the skin (Hirschmann and Raugi 1999, 901). Firstly the skin becomes dry and rough, known as "follicular hyperkeratosis" which usually occurs on the legs and buttocks (Popovich et al. 2009, 408). This is often accompanied by corkscrew hairs and peri follicular haemorrhages or petechiae (Vitler 1980, 486). The petechiae are tiny pinprick haemorrhages which result from increased capillary vessel fragility and the breakdown of intra cellular cement substance which is especially important in the endothelium of blood vessels (Popovich et al. 2009, 411). Petechiae can occur all over the human body but are usually most visible on the limbs and trunk. Petechiae are very small haemorrhages, usually 1-2mm in size; these will progress to ecchymoses which are much larger in diameter at over 1cm in size (Olmedo et al. 2006, 912). Ecchymoses are also known as hematomas and look very similar to large bruises, these can occur under the skin, in muscle tissue, on top of the periosteum or less commonly underneath the periosteum as a result of haemorrhage (Frankel 2006, 149).

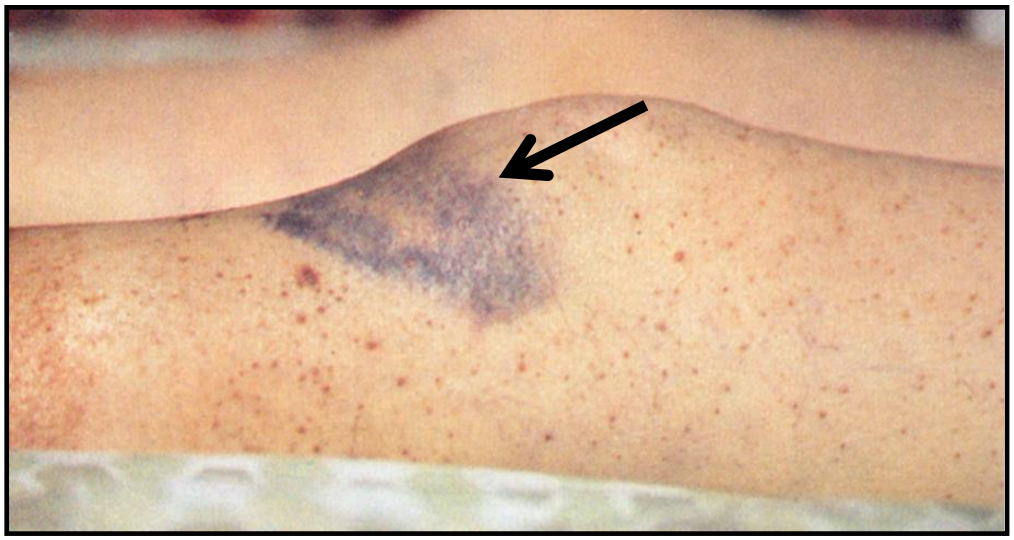

Figure 2.4 Photograph of ecchymosis and petechiae with the arrow pointing to a large ecchymosis surrounded by the petechiae. Reproduced from McLaren 1992.

Figure 2.4 shows a large ecchymosis marked by a large black arrow and the petechiae are visible as tiny pinprick discolorations all over the skin. Any skin lesions present such as wounds and ulcers will also begin to display poor healing (Olmedo et al. 2006, 912) and in very severe cases there may be spontaneous wound opening of old damaged skin due to softening and thinning of the scar tissue (Popovich et al. 2009, 411). Edema is another condition that can be associated with scurvy (Hurlimann et al. 1994); it consists of an abnormal accumulation of fluid under the skin due to leakage from capillaries. This may be due to the breakdown of the endothelium of lymphatic vessels. Lind (1777) is particularly adamant that edema was a classic symptom of scurvy and he frequently describes it as a finding in his autopsies of men that died of scurvy. In scurvy, it seems that edema usually occurs in the legs probably due to the effects of gravity on fluid accumulation (Silverman et al. 1993).

2.3.4 Oral and Ophthalmic Symptomology

Oral indicators will only occur in individuals that have teeth and therefore do not occur in edentulous individuals and babies without teeth (Hirschmann et al. 1999, 902). One of the early signs is halitosis, bleeding and inflammation of the gingival area (Lo Russo et al. 2008, 480). Bleeding may occur even after only slight trauma or friction. The gums become bright red, swollen, soft and spongy masses grow and extrude from the mouth (Halligan et al. 2005, 689). As part of this process the periodontal ligament may also become loose and teeth may be lost ante-mortem. This process may also be due to the resorption of the underlying alveolar bone (Hirschmann et al. 1999, 902). It is widely accepted that the oral abnormalities that accompany scurvy are more striking in those people that already have very poor dental health and hygiene (Fain 2005, 126). The oral changes are one of the most

recognisable indications of scurvy. One of the recurring themes in descriptions of scurvy since the earliest times has been spongy, red gums and very bad breath. The chaplain of Ferdinand Magellan's fleet in the 1520s described it as "most revolting of all, a strange plethora of gum tissue sprouting out of the mouth, which immediately rotted and lent the victims breath an abominable odour" (Lamb 2001). This description has been replicated by many authors up to the present day, in the modern medical context these are commonly referred to as "significant gingival hyperplastic lesions" and can be clearly seen Figure 2.5 (Halligan et al. 2005 689). It is unclear from medical perspective how these lesions are formed if the body cannot produce collagen and an extensive literature review produced no results and the author can provide no explanation. These lesions of the gums are accompanied by significant periodontal disease and a darkening and bleeding of the gums (Brickley et al. 2008). The bleeding is caused by a loss of the marginal epithelium which

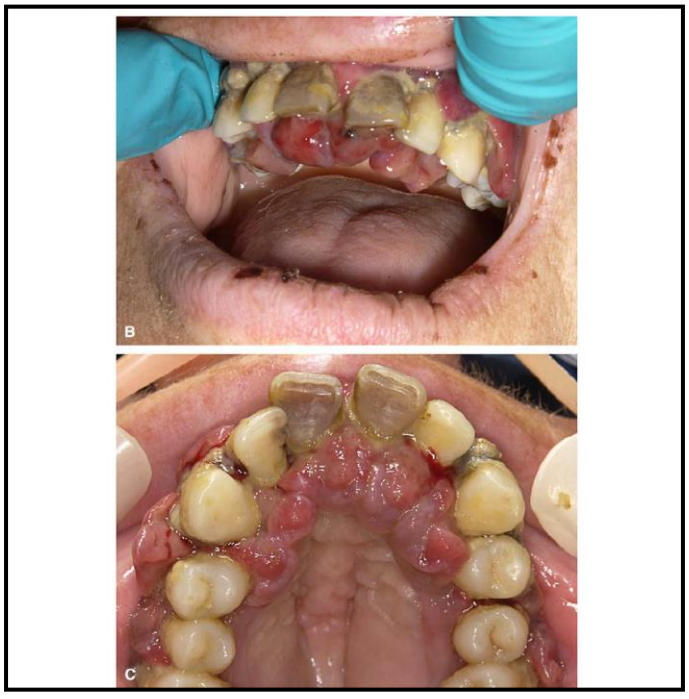

Figure 2.5 Photograph of oral scurvy showing outgrowths . Reproduced from Halligan et al. 2005.

normally protects the gums (Hirschmann et al. 1999, 902).The gums become inflamed and red and then progress to becoming loose and slack. The periodontal ligament which holds each tooth in place becomes lax and teeth may be lost ante-mortem (Danzieser-Wols et al. 2004).

Ophthalmic manifestations of scurvy seem to be again relatively common. The most common manifestation is haemorrhageing and spontaneous hematomas behind and around the eyes which can lead to severe pain, swelling, proposes or protruding eyes (Hirschmann and Raugi 1999, 902). These lesions are generally intraorbital but sometimes these are subperiosteal (Griffeth et al. 1997, 680). In Hood and Hodges (1969) study, ocular lesions were studied in nine scorbutic individuals and five of these presented with ocular vascular lesions which appeared between 74 and 95 days on a Vitamin C free diet (Hood and Hodges 1969, 566). In general, Hood concludes that ocular haemorrhages usually only appear in severe cases of scurvy and he attributed this to the relatively high concentration of Vitamin C in the eye, which can take a long period to deplete (Hood et al. 1969 564).

2.3.5 Musculoskeletal Symptoms

The musculoskeletal symptoms of scurvy are a distinguishing feature of this disease and are thought to occur in 80% of adult individuals with scurvy (Leone et al. 1997). The osseous and muscular abnormalities tend to occur later than the cutaneous and behavioural and are a sign of chronic scurvy as the haemorrhages get progressively worse (Fain 2005, 125). The initial musculoskeletal symptoms consist of myalgia (muscle pain) and arthralgia (bone pain), which are caused by bleeding leading to inflammation of the nerves and subsequently causing pain (Hirschmann et al. 1999 902). This is then followed by haemorrhages into the joints, the periosteum and the muscles. The haemorrhagic manifestation of scurvy is

strongly linked to mechanical trauma and particularly that of the muscles of mastication and in the leg areas that are easily affected by trauma (Van der Merwe et al. 2011). Haemorrhage into muscles commonly occurs in the posterior thigh and in the calf

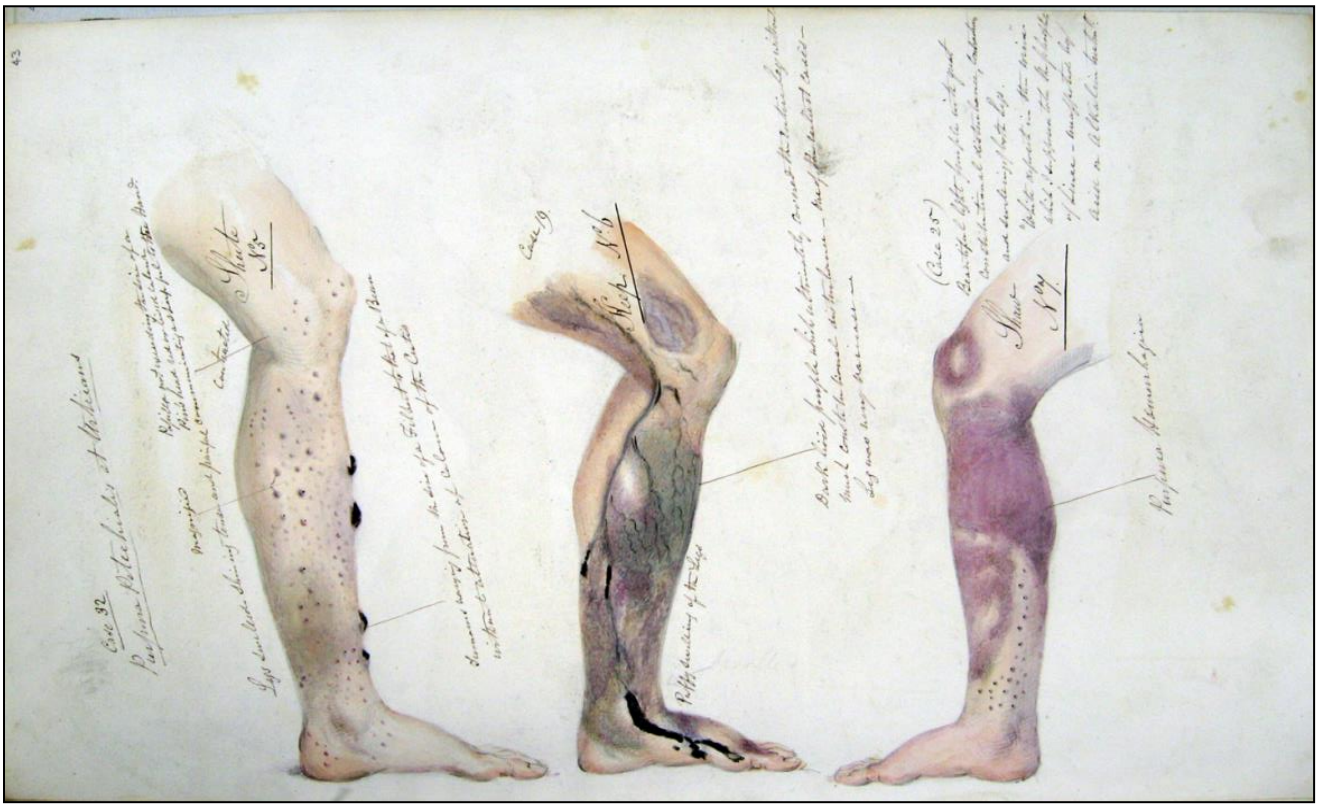

Figure 2.6 Drawing of the manifestation of scurvy in the legs showing petechiae and ecchymoses, from the journal of Henry McMahon 1841-1842 on-board the Convict Ship *Barossa*. Reproduced from the National Archives Collection ADM 102/7/8/53.

(Seftel et al. 1966, 643); this can be clearly seen in the watercolour illustration Figure 2.6. From this illustration the intramuscular hematomas can clearly be seen indicated by the severe bruising and blackness. Intramuscular hematomas have also been known to lead to compartment syndrome (Walters et al. 2007, S48). Compartment syndrome occurs where there is insufficient blood supply to a 'compartment' such as the leg or arm, it causes nerve damage, leading eventually to necrosis. It is haemorrhageing from a capillary that leads to this widespread bleeding as capillary vessels are just one cell wall thick compared to denser, well-muscled veins and arteries which are less likely to haemorrhage. Joint haemorrhages or

hemarthroses occur most commonly in the ankle, knee and sometimes in the hip joint (Fain 2005, 125; Bevelaqua et al. 1976, 1874). It is the musculoskeletal scorbutic abnormalities that can lead to osseous changes, for example haematomas, in the area of the periosteum can cause bony periosteal reaction (Brickley and Ives 2008, 62). Sub-periosteal haemorrhages can lead to the lifting of the periosteum from the bone surface which leads to the formation of ossified sub-periosteal haematomas, this most commonly occurs in the legs (Van der Merwe et al. 2009, 2010). Defective wound and fracture healing and the opening of old wounds and fractures have been frequently recorded in historical references to scurvy (Lind 1772, 253). If collagen is not being produced it can lead to severely impaired wound healing, can weaken old scars and can soften new scar tissue (Popovich et al. 2009, 411; Nannestad-Jorgensen et al. 1998). It has also been recorded in medical contexts that sometimes the worst affected bodily areas are those that were previously injured (Walters et al. 2007). There are several historical records that describe the breakdown of scar tissue and bone calluses in scorbutic individuals. James Lind reported that on Anson's Voyage that there were reported cases of callus breakdown, "where the callus of broken bones which had been completely formed for a long time, were found dissolved and the fracture seemed as if it had never been consolidated" (Lind 1772, 253). Callus breakdown is caused by defective osteoid formation and an imbalance in bone remodelling, with higher rates of bone resorption and a suspension of bone formation (Fain 2005, 124). This makes the bony trabeculae more brittle and significantly more prone to fracturing (Ozturk et al. 1999, 245).

2.3.6 End Stages of Scurvy

When an individual is suffering from scurvy and does not ingest any Vitamin C, death will result (Stone 1966, 345). In the majority of cases it is thought that scurvy sufferers die from

an infection brought on by the effects Vitamin C deficiency on the immune system. In Follis' study of scurvy in children he found that nearly 60% of children studied with scurvy also had an infection (Follis et al. 1950). In some cases however, scurvy can cause sudden death from heart problems usually myocardial infarction (Fain 2005, 127). In many historical accounts, sudden death occurs unexpectedly. In Armstrong's; *Naval Hygiene and Scurvy* 1858*,* he records the case of a seaman that died under his care on a voyage to the East Indies in 1849. The individual had been at sea since he was a boy and presented with scurvy, the same day he was sent to bed, he appeared to be in good form, he ate at 3pm , at 10pm Armstrong saw and spoke to him and "a few minutes later he was heard to breathe somewhat heavily and although not five minutes had elapsed from the time his breathing became oppressed until I was in the sick bay…he had ceased to exist before I reached him" (Armstrong 1858, 51). This is an occurrence that is frequently noted by other historical writers who remark on scurvy sufferers who suddenly expire even after a very small exertion, such as rising from their hammock (Lind 1772).

2.3.7 Scurvy Today

Scurvy is rarely seen and reported on by medical practitioners today. Scurvy is typically diagnosed through blood tests because scurvy may mimic a number of medical conditions including vasculitis, systemic bleeding disorders and deep vein thrombosis (Firth and Marvan 2001). Many of the single cases studies of scurvy have been published in order to create awareness of scurvy among the medical community (Halligan et al 2005; Dolberg et al 2010) and to create familiarity of its symptoms, differential diagnosis and treatment. The symptoms and of scurvy have been discussed in the previous sections and these may occur in any combination.

2.3.8 Summary of the Pathological Manifestation of Adult Scurvy

Adult scurvy is a very distinctive disease process and its manifestation is characterised by a number of key clinical and systemic phenomenon (Dolberg et al. 2010, 184). Initially these symptoms are limited to the skin (Olmedo et al. 2006, 912) and psychological symptoms, but as the body pool of Vitamin C depletes even further, the musculoskeletal symptoms appear (Fain 2005, 125). It is these that lead to the osseous changes that are the subject of this research and represent the chronic stages of this disease process.

Chapter 3 Scurvy in Post Medieval England

3.1 Cultural and Historical context to Eighteenth and Nineteenth Century England.

3.1.1 Life and society in the eighteenth century

This chapter aims to provide a cultural and historical framework in which to place the study assemblages. It is important to understand the society of the eighteenth and the early to mid-nineteenth century in England to put this research in context. There is rich historical documentation surviving from this time which can provide us with an idea of the kind of economic, social and cultural circumstances that these people would have lived through. The period of the Georgian and early Victorian era was particularly transformative for all aspects of English society (Williams 2004, 1). The Georgian era began in 1714 with the reign of George I and ended in 1830 with the death of death of George IV (Richardson 2008, 1). The Victorian era began in 1837 and ended in 1901, needless to say, this research is only concerned with the first thirty years of the Victorian era. It was during the eighteenth century that the Industrial Revolution began in Britain and subsequently spread throughout Europe and the world (Duiker and Spielvogel 2010, 549). The Industrial Revolution meant the mechanisation of processes such as textile production, iron working and mining and farming (Plumb 1963, 77-80). The social changes of the Industrial Revolution were also all encompassing (Porter 1990, 15). For the first time population exploded in the towns and cities due to urban industry and the combined population of England and Wales increased from 6.5 million in 1750 to 16 million in 1841 (Hobsbawn 1999, 5). The industrialisation of England was a major turning point and it changed English society irreversibly (Plumb 1963, 84). The England of this period was also very much turning into a class led society (Olsen

Figure 3.1 Hogarth's Gin Lane 1751. Depicting the alcoholism and poverty that characterised the eighteenth century. Reproduced from www.britishmuseum.org.

1999, 13) and for the first time there was a chasm emerging between the upper classes and the lower laboring classes and people began to identify themselves with a class (Williams 2004, 311). Before the eighteenth century there was no working class, which is thought to be linked to advent of the Industrial Revolution. More than 20% of all poor families needed charitable assistance in order to live (Olsen 1999, 15) and over 30% of Londoners were living below the Poverty Line (Rees et al. 2001, 4). There were massive social problems (Plumb 1963, 84), infant mortality rates were very high (Sherwin 1946, 169), crime rates were relentlessly rising (Hay and Rogers 1997, 147), and slums were commonplace in overcrowded cities which led to diseases like typhus, dysentery and cholera (Porter 1991, 70). This is epitomised in Hogarth's Gin Lane which is reproduced in Figure 3.1. Gin Lane in particular depicts the ill effects of excessive alcohol consumption on society.

The eighteenth century also marked the birth of the British Empire. During the Georgian era, Britain was almost constantly at war. Its empire was expanding and the Royal Navy began to maintain a regular fleet overseas (Hudson 2007, 14). The Seven Years War lasted from 1754 to 1763 and was the first war that was fought on a truly global scale (Marston 2001). Britain was continually victorious except in its loss of the American colonies as a result of the American War of Revolution, in the long term colonies were retained in Canada, the Caribbean, Australia and India (Lavery 1989, 31-32). These overseas colonies were of course protected and maintained by the Royal Navy (Freemont-Barnes 2007, 4). Throughout the Georgian and Victorian period, England was a global power on land and sea due to the might of the army but particularly the Royal Navy. This military prowess was powered by the advent of industry. Hobsbawm (1992, 2) stated that "ships and overseas trade were the lifeblood of Britain, the Navy its most powerful weapon".

All of these factors had a huge effect on agriculture and the provision of foodstuffs, which directly impacted on the diet of the entire population (Allen in Floud (ed) 1994, 96). It was also during this period that people are being exposed for the first time to new and exotic foods like the potato and citrus fruits (Olsen 1999, 232). Diet is directly linked to the development of scurvy, as scurvy is a dietary deficiency of Vitamin C (Hirschmann et al. 1999, 895; Ostler 2004, 383) and the following sections will examine diet and health of the Navy, the working classes and that of prisoners. Particular emphasis will be placed on the staple foods being consumed at this time and their role in the development of scurvy.

3.1.2 Health and Disease in Post Medieval England

Medicine in the Georgian period bears little resemblance to modern medicine and knowledge of the causation of disease and illness during this time was rudimentary at best

(Court 1987, 6). The Georgian period has often been referred to as the 'Age of Agony' due to the common outbreaks of deadly diseases and famines (Hudson et al. 2007, 10). There was some medical progress in this period due to Enlightenment medicine with the advent of vaccinations (Plotkin 2011, 18) and increased anatomical and pathological knowledge (Mitchell 2012). However medical practitioners were woefully inadequate at diagnosing and curing disease (Olsen 1999, 265). Quack remedies such as bleeding, chemical preparations' and induced vomiting were commonplace (Lane 2001, 2), but these quacks did come under a lot of criticism particularly when *The Lancet* were established in 1823 (Porter 2001, 250). Herbal remedies were also used to cure many complaints (DiMarco 2010, xxi). Most doctors could scarcely accurately diagnose disease and they had extremely limited means by which to treat ailments, in short they "lacked science" (Porter 1993, 8). Surgeons were even less highly regarded (Olsen 1999, 263), but it was the surgeons who performed lifesaving operations, amputations, excisions and earned them the reputation as "sawbones" (Goddard 2005, 413). An amputation is shown in Figure 3.2. It was also the surgeons who were expected to cater for the military, navy and those individuals in government institutions such as prisons (Olsen 1999, 263). Diseases such as cholera, smallpox, typhus and tuberculosis were commonplace and for the most part incurable (Porter 1991, 70; Rees et al. 2001, 112). In most cases, people relied on home remedies to cure illness, of which the majority were unsuccessful. However by 1800, there were sixteen dispensaries open in London which provided free medicine and intended to provide free home visits for the poor (Porter 1991, 73). Of most relevance to this research is the recognition of metabolic diseases, which were not formally recognised until the 1880s with the work of Thomas Barlow who wrote extensively about juvenile scurvy and infantile rickets, also known as Barlow's Disease (Barlow 1883, 1894). It was not till the modern era that the precise

causation of these diseases was ascertained and the term ‘metabolic disease’ first used in 1948 (Albright and Reifenstein 1948).

Despite these shortcomings, the Georgian a period was one of great development in medicine and it was here that progress was made particularly in the area of Naval medicine

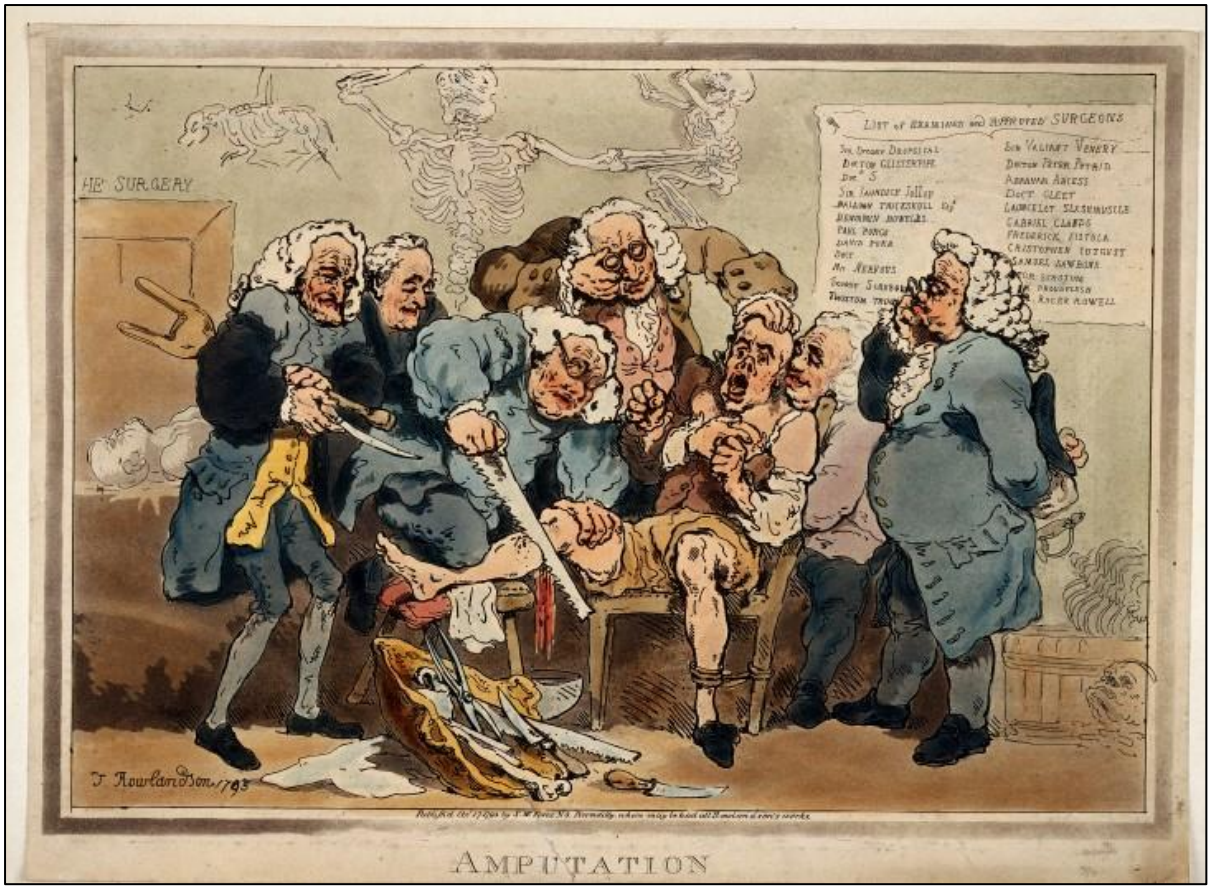

Figure 3.2 Print by Thomas Rowlandson (1793) showing five surgeons perform an amputation whilst being overseen by another surgeon. Courtesy of www.wellcomeimages.org.

and hygiene. The Fathers of Naval Medicine implemented innovative new principles of hygiene and issued directives on diet and disease prevention (Hudson 2007, 14). It is also in this period that dissection and post mortem autopsy became commonplace (Hurren 2011, 4-5). Dissection was the primary method by which medical practitioners learned their craft and bodies were therefore a valuable commodity (Mitchell 2012, 2). By the end of the Victorian period the practice of medicine would have been recognisable to the modern

observer along with the corresponding infrastructure of hospitals and an increasingly institutionalised medical service (Hurren 2011, 77).

3.2 Post Medieval Diet

There is a direct link between the development of disease and diet; and particularly that of scurvy (Hirschmann and Raugi 1999, 895). It is therefore crucial to understand dietary patterns and their link with the occurrence of adult scurvy. In post medieval England, fruit and vegetables made up a very small proportion of the daily diet (Olsen 1999). Our knowledge of post medieval diet is based on historical documentation such as contemporary cookbooks (Beeton 1861; DiMarco 2010) and the writings of historians and health reformers. This is supplemented by archaeological evidence, however in archaeological studies there has been tendency to concentrate on the meat and fish aspects of diet as these are best represented in the archaeological record from animal bone analysis and isotopic analysis (O'Connell and Hedges 1999; Klein and Cruz-Uribe 1984). Archaeobotanical analysis can shed some light on vegetable and fruit consumption, but this evidence is not plentiful, as vegetable matter does not commonly survive in the archaeological record (Greig 1989).

It is also critical to consider the impact of the Industrial Revolution on food production and diet in this period. It has been claimed that diet and therefore health has steadily decreased from prehistoric times up to the Post Medieval period (Roberts and Cox 2007). These health trends are expressed in the palaeopathological assessment of human skeletal remains of archaeologically provenanced skeletal assemblages (Steckel 2001). The Post Medieval period is characterised by the Industrialisation of England and the increasing commercialisation of agriculture along with rising levels of poverty (Roberts and Cox 2007).

Despite the improvements in agricultural production which directly resulted in higher productivity and quality, famines and crop failures were commonplace (Carpenter 1986).

3.2.1 Diet Staples, Food Preparation and Smoking

Poverty and subsequent poor diet were seen as a fact of life in eighteenth and nineteenth century England (Rees et al. 2001, 6). A poor diet can be defined as one that does not meet daily nutrition requirements or one that is imbalanced. It is thought that the poorer classes on average consumed less than 2,000 calories a day, which taking into account daily bodily needs, only allows for only one hour of heavy labour per day (Hay and Rogers 1997, 78). The staple diet of the time for the majority of society was bread (Wilbraham and Drummond 1957) and the newly introduced potato. Marshall (1956, 169) maintains that "the unskilled labourer in the towns and the agricultural labourer in the country lived chiefly on bread, cheese, small beer, with meat perhaps, once a week". This would have been supplemented by other fresh vegetables and fruit which were available on a seasonal basis. There is scant archaeobotanical evidence for consumption of plant products. There is medieval evidence for fruit such as grape, damson, plum, apple, pear, raspberries and blackberries from cesspits in Leicester (Monckton 1995, 36). There is also evidence from other sites for vegetables such as pea, leek, beans and asparagus (Monckton 1995, 37). Excavation of a post medieval tenement in Staffordshire has provided evidence for grains such as wheat, rye and oat along with common fruits and vegetables, like cabbage, turnip, carrot, beet, apple and grape (Moffett et al. 1996). So there is evidence for the consumption of fruit and vegetables but it must be kept in mind that these supplies would have been seasonal. It should be also be remembered that expanding colonial trade meant that for the first time exotic fruits such as oranges, pineapples and figs were becoming popular but these were

still extremely expensive and were only within the reaches of the wealthier classes (Olsen 1999, 232; Mason 2004, 30). The poor in the nineteenth century suffered quite badly from poor diet and were extremely susceptible to food shortages and malnutrition (Roberts and Cox 2007). During the winter month's fruit and vegetables would have been scarce and it is likely that many people may have developed sub clinical scurvy (Stark 2009, 105). Falk, Gedda, and Gothlin (1932) recorded the occurrence of latent scurvy in Sweden throughout the year and showed that latent scurvy was most likely to occur in April. This fits well with this theory because if vegetables run out during December or January, then it will take until March or April for latent scurvy to develop. Latent or subclinical scurvy is Vitamin C depletion before the appearance of clinical symptoms of scurvy (Stark 2009).

It is essential to consider the staple diet of the lower classes in the development of scurvy. The staple diet of the laboring classes of England was bread and in the Northern counties oatmeal and potatoes were commonplace, the laboring class could not afford much meat, but bacon was popular when available (Olson 1999, 235). Tea was drunk by all social classes; it was drunk black and was of no real nutritional value. Other basics of a laborers diet were cheese, treacle, broths, stews and beers (Olsen 1999, 235). It is however difficult to properly ascertain the precise dietary practices of the poorer classes in eighteenth and nineteenth century Britain. The earliest food survey was undertaken by Dr. Edward Smith in 1862/1863, who surveyed 370 English families of the poor laboring classes (Carpenter 1991, 1517). Smith found that bread was the most common food consumed at just over 12lbs a week on average per person and then potatoes at 6lbs a week (Carpenter 1991, 1518). Most significant to this research is that he surveyed unemployed cotton workers in the Northern counties in 1862/1863 during the Lancashire Cotton Famine. This is crucial because one of

the four assemblages being examined in this research is a population of cotton workers from Lancashire that was contemporaneous with the Cotton Famine. Carpenter (1991), analysed the weekly diet of two unemployed cotton workers which are reproduced in Table 3.1. These two workers were simply recorded as No 9 and No 39. These two cotton workers subsisted primarily on a diet of bread, supplemented by oatmeal, onions, treacle, cheese and some bacon. Tea was the most common drink, which was drunk by both workers, coffee and beer was also consumed. It should be noted that there was no potato input

Table 3.1 Showing Lancashire Cotton Workers Diet data that was collected by Dr. Edward Smith in 1861/1862. Adapted from Carpenter 1991, 1515.

	Bread	Oatmeal	Onions	Treacle	Bacon	Cheese	Tea	Coffee	Beer
No 9	**8lb.**	**-**	**2lb.**	**1lb.**	**1/2lb.**	**1/2lb.**	**1/2oz.**	**-**	**-**
No 39	**12 lb.**	**2lb.**	**-**	**1lb.**	**1/2lb.**	**-**	**1/2oz.**	**2oz.**	**2oz.**

because during this time, Smith notes that potatoes and animal products were too expensive to purchase (Smith 1864). When further questioned the interviewees explained that these were former common purchases.

Smith made many recommendations to improve the diet of the poor laboring classes by encouraging higher vegetable and milk consumption (Smith 1864). These recommendations had little effect; however at the time Smith's research was pioneering in trying to improve the diet of the poor laboring classes (Carpenter 1991, 1518).

It was previously mentioned in Chapter 2 that smoking significantly influences the body's need for Vitamin C and can exacerbate Vitamin C depletion. Smoking was introduced to England during this period and it is possible that this may have had an effect on scurvy rates

in this period. It is unlikely to have had a huge effect on the Naval populations as smoking was banned on board Naval vessels and chewing tobacco was much more popular (Rogers 1988). However it is an important factor to consider in scurvy occurrence.

The move from a primarily rural population to a population that was concentrated in towns also directly affected the diet of the laboring class. For pre-Industrial rural families vegetable patches would have been customary, in towns this was impossible and urban families were forced to purchase vegetable items which were not always affordable (Olsen 1999). Overall, it appears that the laboring classes relied upon usually inexpensive bread as their staple and vegetables were included only when they were within their means (Wilbraham and Drummond 1957).

3.2.2 Vitamin C content of the Post Medieval Diet

Vitamin C content is highest in fresh fruit and vegetables, this can be seen in Table 2.1 in subchapter 2.1.3. When these are cooked, preserved or stored for long periods of time, the Vitamin C content declines significantly (Butz et al. 2003; Gil et al. 1999, 2216). In Post Medieval England the available fruit and vegetables were usually only consumed when cooked due to a common misconception that in their uncooked state they were deleterious to health (Lane 1995, 67). Mrs Beeton's well known 'Book of Household Management' published in 1861 devotes a small section to vegetable preparation and recipes. In her recipe for cooking carrots, she recommends they be cooked for up to two and a half hours. For broccoli she recommends twenty five minutes and boiled beetroot is to be cooked for up to three hours (Beeton 1861). This method of long cooking effectively destroys any Vitamin C that is present and if any Vitamin C did survive it would seep out into the cooking water (Erdman and Klein 1982, 499). Root vegetables do contain a small amount of Vitamin

C when raw and lightly cooked, but it was the introduction of the Vitamin C rich potato that changed the face of everyday food in the British Isles and Europe (Salaman 1949). It is thought that the introduction of the potato significantly improved populational health (Carpenter 1986) due to its all-round nutritional value. The potato became widely grown in Britain at the beginning of the nineteenth century and proved particularly popular in Northern counties like Lancashire where the soil was ideal for their cultivation (Mason 2004, 30). The potato was popular due to its high yield and the fact that it would flourish in soil unsuitable for growing cereals (O'Grada 1995, 16). The potato is a vegetable that is rich in Vitamin C when consumed in large quantities even in its cooked state (Salaman 1949, 123). There is 26mg of Vitamin C present in a 175g serving of boiled new potatoes in their skins (The Potato Council 2012). Therefore if the poorer classes were consuming 5.25lbs of potatoes a week per person (Salaman 1949, 123) this would have provided more than half of the recommended daily allowance of Vitamin C. August Hirsch has written that he believed that scurvy had become much less common in Europe with the spread of potato cultivation (1885). For the poor of England in the eighteenth and nineteenth century the potato would have been the ideal staple diet, but its standing in the diet of the poor was far outstripped by the popularity and cheapness of bread (Thompson 1992, 256).This was true for most of England except some of the Northern regions, where the potato soon gained popularity, possibly due to the Irish populations living there (Lomax 1986, 72). In Ireland the potato was the staple food of the whole country (O'Grada 1995). In neighbouring England, white bread was preferred by all classes (Mason 2004, 29) and when made from any grain source such as rye, barley or wheat, of all which are virtually Vitamin C free due to the paucity of vitamins in grains (Serna-Saldivar 2012,68). When the potato was removed from the diet due to seasonal unavailability or famine, it also eliminated a vitally important

source of Vitamin C. So for the majority of the poor, the only anti-scorbutic foods that were available were the potato and a very limited seasonal selection of fruit and vegetables.

It is clear that the main foodstuffs consumed by the poor laboring classes were a poor source of Vitamin C and definitely posed a risk for the development of scurvy. This risk was exacerbated in times of famine and hardship (Carpenter 1986, 102). It must also be kept in mind that the supply of fruit and vegetables would have been seasonal (Thompson 1992, 251). Stark (2009,105) suggests a cyclical pattern of scurvy with individuals developing scurvy in the winter and recovering in the spring and summer with the reintroduction of anti-scorbutic foods to the diet.

3.2.3 Scurvy in the Laboring classes of Post Medieval England

Scurvy cannot be considered to be a new disease in this period. Approximately 100 scurvy epidemics occurred throughout Europe between 1556 and 1857 (Prevention Magazine 1996, 501). 'Land Scurvy' had been known for hundreds of years and was associated with famines, wars and pestilences (Susman and Deller 1955, 966). It is very difficult to establish rates of scurvy among the working classes of England in eighteenth and nineteenth century as there is no official figures. The reasons for this are manifold. The main issue was likely to have been lack of medical care towards these classes, few could afford medical assistance even in the most dire of situations (Lane 2001, 2) and therefore if scurvy was present, it was rarely documented. This was compounded by there being very few physicians and very large city populations, for example in Birmingham in 1767 the ratio of population to practitioners was 10,299 people for just one physician (Lane 2001, 22). Secondly, even if medical assistance was sought few 'land' surgeons or physicians had any experience of scurvy and in all likelihood would have been unable to diagnose it. Indeed, it is thought that scurvy was

frequently mis-diagnosed. A Dr. Barrett working in a Bath workhouse in the 1840s notes high rates of pupura hemorrhagica 'diagnosed' by fellow practitioners, which he believes was actually scurvy due to the failure of the potato (Barrett 1847, 147-150). Pupura hemorrhagica is a disease process characterised by reduction in platelet count, subcutaneous haemorrhages and weakening of the capillary vessels (Eliason and Ferguson 1932, 801). These symptoms are identical to that of clinical scurvy.

For the purpose of this research this documentary review of scurvy will concentrate in the mid-1800s as this corresponds with the date of main non-Naval and non-Prison skeletal assemblage in this study. Drummond and Wilbraham (1957), in their compendium; 'The Englishman's Food' go so far as to suggest that throughout medieval times sub clinical scurvy was commonplace in the winter time until the introduction of the potato in the seventeenth century (Drummond and Wilbraham 1957). The potato had such a huge effect on English diet that, Salaman (1949) argues the poor English relied on the potato so extensively "by the middle of the century (this) was shown by the tragic effects of the blight…the mortality figures rose by 30%...scurvy was once more prevalent throughout Great Britain" (Salaman 1949, 525). Salaman is referring to the years of potato blight in the 1840s that led to an extensive potato famine in Ireland and across some parts of Northern Europe (Carpenter 1986, 102). There is also documentary evidence for scurvy in England during this period (Lomax 1986, 71). In 1847, the aforementioned Dr. John Barrett records scurvy in Bath and he also records cases from Tisbury (Wiltshire), Frome, Camely and Keynsham (Somerset) and Bradford (Barrett 1848, 148). A Dr. Shapter also records scurvy occurring in a lunatic asylum and hospital in Exeter in 1847 (Shapter 1847, 281). In Glasgow in 1847, 122 cases of scurvy were treated at the Glasgow Royal Infirmary (Lomax 1986,71)

Scurvy also became common in Lancashire and “a haemorrhagic tendency has been noted in several towns, actual scurvy has been seen among cotton workers in Stockport, Preston,

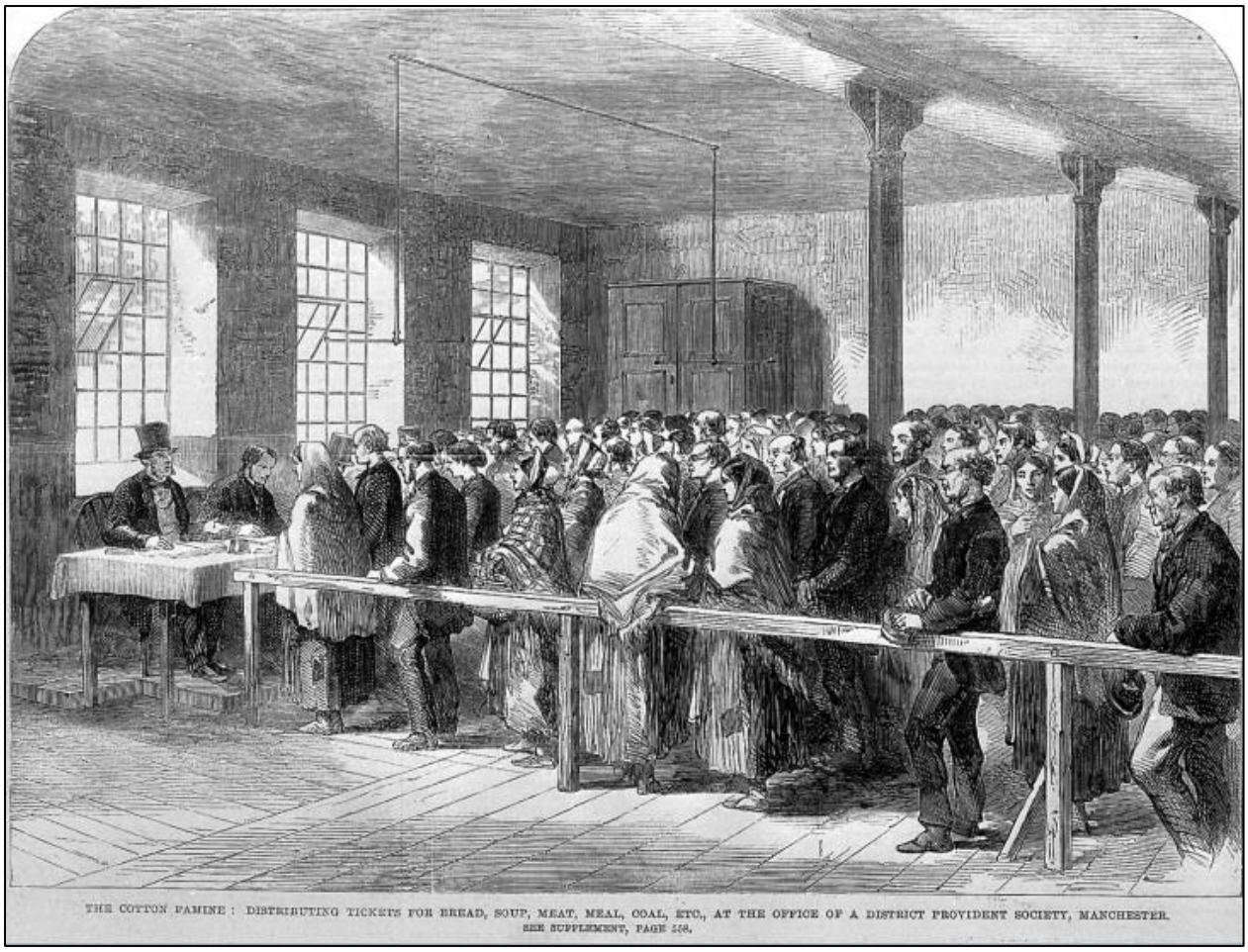

Figure 3.3 Cotton Workers queuing for meal and fuel tickets during the Cotton Famine. Reproduced from www.wellcomeimages.org.

Blackburn and Salford” during the Cotton Famine of 1861-1865 (Henderson 1934, 104). Figure 3.3. depicts poor cotton workers queuing for meal and fuel tickets during the Cotton Famine in Manchester. A Dr. Buchanan who was working as a medical officer reporting to the Privy Council also recorded scurvy in Lancashire and Cheshire during the Cotton Famine and he attributed this to a diet that was primarily composed of bread and tea and a lack of potatoes (Buchanan 1863, 309).

These historical sources not only show that scurvy was present in England during the mid-eighteenth century, but that the potato is intrinsically linked in with the occurrence of

scurvy in this period and this fact was recognised by at least some contemporary writers (Buchanan 1863; Shapter 1847; Barrett 1848). It must also be remembered that just because there was not a large scale famine on the magnitude of the Irish famine, it does not mean the failure of the potato did not adversely affect the everyday diet of working class (Salaman 1949).

3.3 The Post Medieval Prison System

"The misery of gaols is not half their evil, they are filled with every corruption that poverty and wickedness can generate between them, with all the shameless and profligate enormities that can be produced by the impudence of ignominy, the rage of want and the malignity of despair. In prison the awe of the public eye is lost, the power of the law is spent; there are few fears, there are no blushes. The lewd inflame the lewd and the audacious harden the audacious. Everyone fortifies himself as he can against his own sensibility, endeavours to practice on others the arts which are practiced on himself and gains the kindness of associates by similitude of manners"

Samuel Johnson in The Idler Magazine 1759 no. 38

3.3.1 Introduction to the prisons of the period.

From medieval times most English towns had at least one gaol for the confinement of debtors and criminals (Lane 2001, 114). For the most part the county prisons were not used as a form of punishment, but as a place to detain debtors and those awaiting trial for

criminal activities, short-term prisoners and those awaiting hanging or transportation (Priestley 1985). The first state run prison was opened in 1816 at Millbank in London which reflects the rising criminality in society in this time; it was opened primarily as a holding place for prisoners awaiting transportation (Priestley 1985). At this time transportation was a common form of criminal punishment (Willis 2005, 173) and up until 1776 transportation to America, particularly Virginia and Maryland was commonplace. This practice ceased during the War of Independence and prisoners were instead exiled to Australia and Van Diemen's Land, which is modern day Tasmania (Sherwin 1946, 185). Between 1718 and 1775, 50,000 British convicts were transported to the American colonies (Ekirch 1987, 27) and when transportation was stopped in 1868, a total of 160,000 had been sent to Australia (Hughes 1986, 2). Along with prisons, ships or prison hulks were also commonly used for holding prisoners prior to transportation. The primary prison hulks were moored in Portsmouth, Plymouth and Chatham and were reputed to be extremely unhealthy and overcrowded; these were however abolished by the mid-1850s (Creese 1995, 60).

In the eighteenth century, prisons were considered to be havens of misery, disease and wickedness by the commentators of the time (Gray 2009, 3). Buildings that were being utilised as prisons were often old castles or stone built defensive structures that were ill-suited to the confinement of large numbers of people (Lane 2001, 114). During the eighteenth century, the majority of people were imprisoned for committing a crime or being unable to pay one's debts (Olsen 1999, 214). In some cases debtors and criminals were separate, but for most county goals this was not possible. In 1776, there were 2,500 debtors in the prison system in England, 994 felons and 653 petty offenders (Howard 1777).

3.3.2 Prison Conditions

One of the chief historical sources for prison conditions at this time are the writings of John Howard (1777, 1784, 1786). During the 1770s and 1780s John Howard visited prisons all over England and Wales. He is considered to be the earliest prison reformer in England, he himself being taken prisoner by the French in Brest in the 1750's (Farrar 1833, 16), and this incident is thought to have led to his interest in Georgian prison standards. Howard's observations are recorded in his book, *The state of the prisons in England and Wales, with preliminary observations on some foreign prisons*. This book was published in 1777, detailing his first tour of the national prison system and later of the European prison system (Lane 2001, 114). Most pertinent to this research, John Howard recorded his observations about the general state of prisons, but most importantly he comments on the state of Oxford Castle which is one of the study groups being investigated here. Howard's remarks are detailed in the site background of Oxford Castle in the methodology chapter.

In general, John Howard notes that "many prisons are scantily supplied and some almost totally unprovided with the necessaries of life" (1777, 7). Men, women and youngsters were all crowded together in small unsanitary cells often with little or no straw for bedding (Howard 1777). Overcrowding in gaols became a massive problem in the late eighteenth century (Lane 2001, 114). The rise in convicts was linked in part to the advent of the Industrial Revolution and rising poverty, debt and crime levels (Plumb 1963, 84).

3.3.3 Prison Diet and Disease

Prison conditions were extremely poor and poor prison diet was also a pressing problem, there was no uniformity in the standard and size of prison food rations across the country

(Howard 1777). In the late eighteenth century at the time of Howard's writing he states that many bridewells (house of correction/prison) have no food allowance for prisoners, those that did have an allowance varied, in some it was a penny worth a day, others two pence and others again a shilling a week (Howard 1777, 9). This shows absolutely no standardisation or regulations in place to regulate national prison diets. Those in prison awaiting trial had no legal right to food at all (Sherwin 1946, 184). It would however seem that the main food consumed by prisoners was bread accompanied by water or gruel porridge and bread (Howard 1777, 9). Howard did note that a standard pennyloaf bread ration weighed 7-8 ounces (approx. 200g) and this was one of the few aspects of prison diet that was identical across the country (Howard 1777, 11). Often the bread was boiled in water to make a "water soup". John Howard is extremely critical of the 'ordinary prisoners' diet and compares it to that of a prisoner of war. The daily allowance to six prisoners of war was nine pounds of bread, four and a half pounds of beef, three pints of pease and six quarts of beer along with plenty of water (Howard 1777, 21). It should be noted that there was absolutely no provision of fresh fruit or vegetables in the standard prisoner of war diet, but Howard still believed it to be better than a normal prisoner's diet. A standard prison diet for one person later in the mid nineteenth century consisted of gruel and porridge and either six ounces of meat and 8 ounces of potato or 1.5 litres of soup on alternate days (Carpenter 1991, 99). Eight ounces of cooked potato would contain 15-20mg of Vitamin C. It must be concluded that a standard prison diet is very low in Vitamin C and in many cases it was totally lacking.

Disease was another major problem with ailments such as gaol distemper or fever (typhus), tuberculosis and smallpox that were rife (Lane 2001, 114; May 2006, 8). In the 1750 'Black

Sessions', Newgate prisoners infected with typhus caused the death of more than 40 people at the Old Bailey including jurors and judges (Olsen 1999, 267). A similar event happened at the Oxford Assizes which was known as the 'Black Assizes' (Davies 2005, 91). Prisons epidemics were especially common when inmates were fed only bread and water (Lane 2001, 116). In response to outbreaks of disease, the Gaol Distemper Act was passed in 1774 encouraging prison health reforms; it promoted cleaner conditions, ventilation and the segregation of sick and well prisoners (Creese 1995, 35). Howard (1777) suggested that every gaol should have a surgeon, but this was only fully implemented with the advent of the Pentonville Prison System in 1842 (Lane 200, 116).

3.3.4 Scurvy in Prisons

Scurvy was not uncommon in English prisons due to the extremely low Vitamin C content of the prison rations (Carpenter 1986, 99). The first documentary evidence for scurvy in prisons is from the late 1700s, the prison reformer John Howard notes that "some prisoners are grievously affected with scorbutic distempers" (Howard 1777, 39). Documentary sources referring to scurvy in prisons increase in the nineteenth century and in 1823 there was a massive outbreak of scurvy recorded in Millbank prison (Lane 2001, 116). This scorbutic epidemic was due to a change in diet and large reduction in ration sizes, particularly as the daily allowance of one pound of potato per inmate was totally eliminated as it was deemed to be too 'luxurious' (Carpenter 1986, 99). Scurvy was seen in over half of the 860 inmates in the prison (Carpenter 1986, 99). Scurvy was not just a problem in London city prisons but in over twenty county gaols all over England in the previous years (Baly 1843, 701-702). Baly found a strong link between scurvy outbreaks and absence of potatoes in the diet of the

inmates and “where diets had been subsequently changed to include a regular ration of potatoes, no more was heard of the disease” (Baly 1843, 702). This again attests to the anti-scorbutic power of the potato which would have been a cheap and nutritious foodstuff but when eliminated from the diet led to scurvy.

3.4 The Georgian Period Navy

It was during the Georgian period that the Royal Navy became a force to be reckoned with and began to confirm its status as one of the largest military powers in the world (Freemont-Barnes 2007, 4). This era was one of substantial expansion and development for the British Empire and the Royal Navy was fundamental to this campaign (Goddard 1991, 212). This imperial growth was steered by a series of significant conflicts throughout the period beginning with the Seven Years War (1756-1763) and ending with the Napoleonic Wars (1803-1815). The Royal Navy grew incrementally in order to cope with the need to defend the British Empire and to control overseas colonies. The Navy was also effectively “the largest industrial organisation in the western world” (Rogers 1988, 29), as well as being a very complex society within itself. Nicholas Rogers’s seminal text concerning the anatomy of the Georgian Navy is aptly called ‘The Wooden World’. This title defines the Georgian Navy and explains the attitude and mind-set of civilians and those within the ‘wooden world’. Onboard ship was a peculiar society with its own rules and values (Rogers 1988, 37). It was a heavily ranked group, deriving its commissioned officer set from the middle classes of society and seamen generally from the lower classes (Lloyd 1968, 233).

The size of the Navy varied greatly over the Georgian period with its largest growth being towards the end of the Napoleonic Wars; in 1794 it numbered approximately 65,000 men increasing to 140,000 men in 1815 (Fremont-Barnes 2007, 9; Goddard 1991, 213). In the eighteenth century the Royal Navy was probably by far the single biggest employer in England (Bardolph et al. 1998). These numbers were achieved by supplementing volunteers with impressments during wartime (Kemp 1970, 162). Conditions in the Navy were good by the standards of the time with satisfactory food rations, wages and excellent opportunity to rise within the ranks. The well-being and capability of the Navy was however heavily dependent on victualling and the resulting health of sailors (Rogers 1988, 82). Victualling is the provision of food to ships and is used in particular reference to Navies.

3.4.1 Naval Hospital Establishment

Since the health of the navy was one of the most important factors that governed its success and was directly linked to the manpower available (Rogers 1988; Lloyd 1968), it comes as a surprise then that before 1753 there were no specialised naval hospitals to cater for the sick mariners. Sick men were landed at hospital ships which were usually old warships that were completely unfit for purpose (Kemp 1970, 123). There were non-clinical hospitals prior to 1753 established at Greenwich and Chatham in the late seventeenth century (Stevenson 2007, 229), but these were not hospitals in the modern sense of the word, more like retirement homes for military and naval veterans (Boston et al. 2008) Two Greenwich pensioners are depicted in Figure 3.4, these pensioners were frequently disabled as can be seen in this image. These hospitals were established by funds taken from the Chatham chest (Tait 1906, 1), a charitable fund established in 1590 by Sir Francis Drake and Sir John Hawkins to provide pensions for injured naval retirees (Buchanan 2005, 221). The funds

from the Chatham chest were received from individual parishes for the seamen born in the area and a proportion was also taken from sailors' wages (Kemp 1970, 22). The Chatham

Figure 3.4 Painting depicting the Greenwich Pensioners, with numerous amputees depicted. Reproduced from www.rmg.co.uk

chest is currently on display on Chatham Historic Dockyard and can be seen in Figure 3.5. The first purpose built clinical naval hospitals were those built at Gosport and Plymouth (Lane 2001, 171). Prior to the building of the Naval Hospitals there were sick quarters in the Naval port towns that consisted of rented rooms which were frequently run by local landladies that took care of the sick. However these women often had their own interests at heart and sick sailors were often taken advantage of (Rogers 1988, 109; Tait 1906, 9). This contract system was open to numerous abuses and the standard of the lodgings was very poor (Crimmin 1999, 55). A big problem for the Admiralty was that these men hardly ever recovered or returned to Naval service, which meant a massive loss of manpower (Rogers 1988, 109). By the beginning of the1750's, the opinion of the Admiralty was that it would be

best to found purpose built Naval hospitals. There were many advantages to this new system; the old 'contract' system was simply unable to cope with large numbers of sick, and

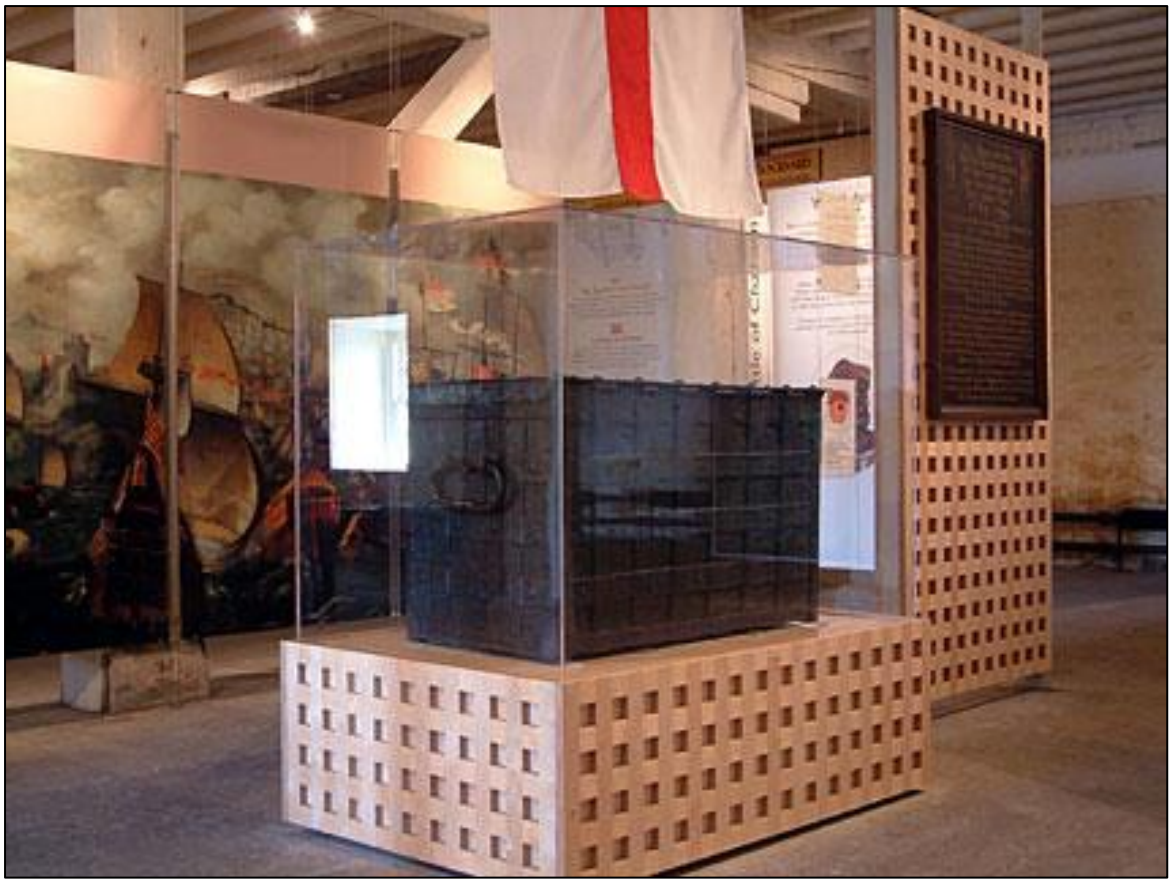

Figure 3.5 Photograph of the Chatham Chest on display at Chatham Historic Dockyard, reproduced from www.englishheritage.co.uk

it could not provide the medical care that was needed (Lane 2001, 171). The contract hospital system was inefficient, expensive, encouraged desertion, alcoholism and bad habits. It is also true that the medical care being provided by the contract hospitals was poor and it is clear from the documentary sources that the Admiralty recognised the need for a new more proficient system. This difficulty was particularly apparent in war time when numbers of injured and sick sailors increased many times over. The new hospitals were also carefully situated in order to deter desertion, but also had to be close enough to the coast to facilitate easy transportation of ailing sailors from ship to hospital and vice versa (Tait

1906). The development of these revolutionary Naval hospitals were incredibly innovative and influenced hospital architectural styles internationally (Buchanan 2005). It also brought the health of naval personnel to the forefront and it bolstered the importance that the Admiralty assigned to healthcare.

The next subchapters will deal specifically with the establishment of Royal Hospital Haslar in Gosport and Stonehouse Hospital, Plymouth. This will include the background to their construction, and their importance in the development of Naval healthcare. The historical background of both sites will be presented, however the archaeological excavations of these sites will be discussed in chapter four.

3.4.1.1. Royal Hospital Haslar

Royal Hospital Haslar (hereafter, just Haslar) was the first naval hospital to be built at a home port by the Admiralty. It is located in Gosport, Hampshire, very close to the port city of Portsmouth. The first evidence of a hospital at Gosport was that of the Fortune hospital in 1713 which was a small contract hospital, it is likely that this was located near where Forton Barracks is today (Tait 1906; White 1989, 38). In 1745 the Admiralty purchased the 95 acre site at Haslar as it was deemed to be "the most convenient place for a hospital for the Sick and Hurt Seamen at Gosport" (Admiralty Paper 98/2 1745). There were a number of reasons for this, the first being the relative isolation of the site which was surrounded by water and estuarine mud and was separated from the town of Portsmouth (Lavery 1989, 216). It was also ideal for the reception of sick sailors; the sick could be conveniently transported by boat from the ships anchored at Spithead to a jetty at Blockhouse Lake (Buchanan 2005, Tait 1906). The Admiralty commissioned Theodore Jacobson who designed the Foundling Hospital in London, to design the hospital at Haslar. Jacobson planned for a

palatial style hospital deciding on a massive quadrangular structure with an internal piazza. From Jacobson's plan it is possible to see that the each wing of the hospital is actually made of two parallel three storey ranges of wards, this can be seen in Figure 3.6. This design was implemented in order to allow freer circulation of air. During this time ventilation and pure air were considered key to disease prevention and the miasmatic theory of disease causation was at its height. The foundations for Haslar were laid in 1746 and during the construction Jacobson's plan was modified to a simpler three sided structure. The scale of Haslar was still very large, the front façade of the building is 567 feet long and the two receding wings 553 feet long (Tait 1906, 16). This change in plans made Haslar a 'U' shaped structure which was similar to the pre-existing Naval hospitals at foreign bases at Gibraltar

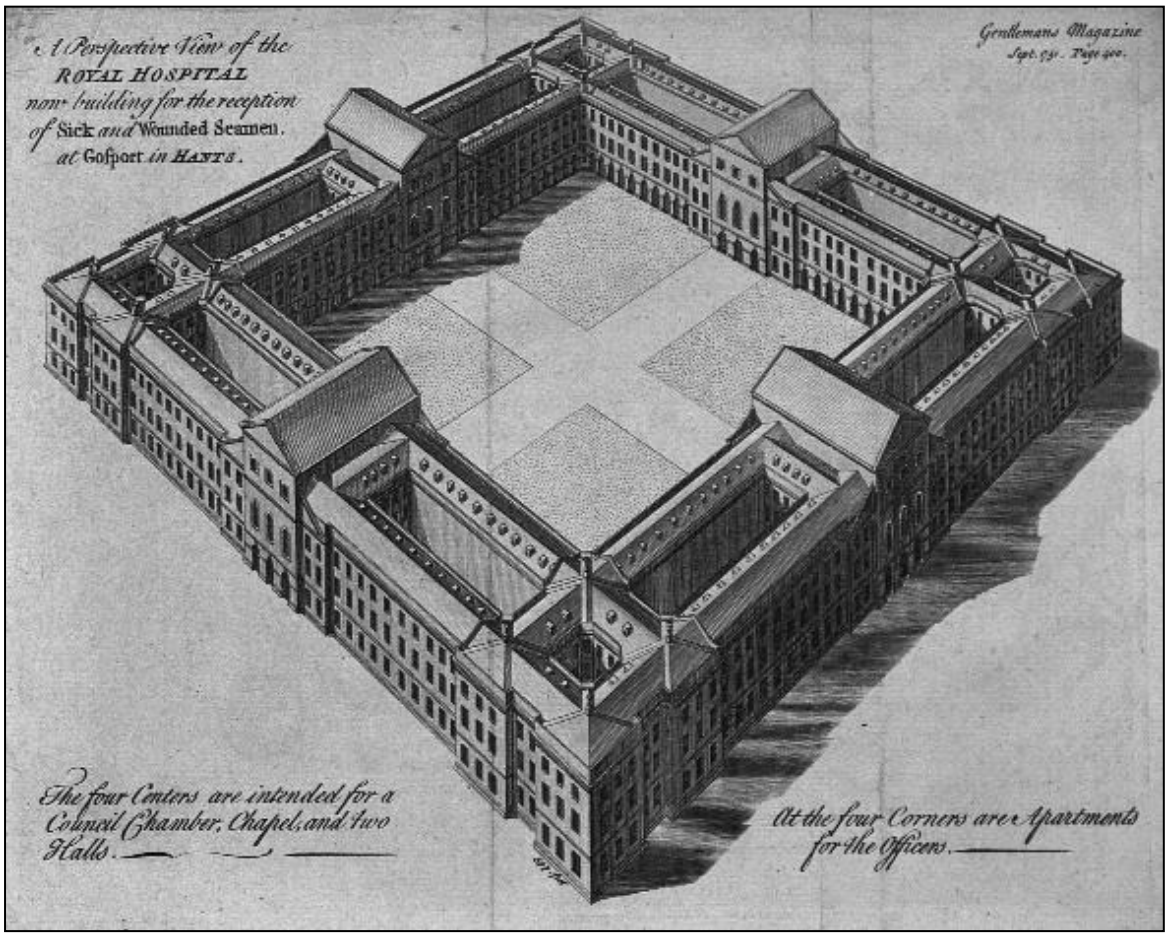

Figure 3.4 Theodore Jacobson's original four sided plan of Haslar hospital from 1744 as published in the Gentleman's magazine and then reproduced in Stevenson 2007.

and Jamaica. This 'U' shaped structure did mean that desertion was a rather large problem at the hospital, throughout 1794 between ten and thirty-two men deserted from the hospital every month (ADM 102/273), this was remedied in 1796 by building a twelve feet high iron railing to complete the quadrangle (Tait 1906). The plans were also changed to increase patient lodgings, which were increased from an original figure of 1500 up to 1800 patient capacity. Haslar was finally completed in 1761 (Crimmin 1999, 191) and by this time had cost over £100,000 to build and was particularly notable as the largest red brick building in Europe (Tait 1906, 9).

When Haslar opened in 1754 it was a state of the art prototype and it reflected importance that the Admiralty placed on the health of the navy , it was one of the most expensive projects commissioned by the Admiralty in the eighteenth century (Rogers 1988). By 1778 it could house 2,100 patients, and by 1787 it contained 84 separate medical and surgical wards (Buchanan 2005, 224). Haslar was also progressive, having the best physicians of the time working there, James Lind was the chief physician at Haslar hospital for almost twenty-five years (Dunn 1997, F64), see subchapter 3.5.4.2. It was also supplied with the latest medicines and implementing the latest research into disease causation, prevention and hygiene (Tait 1906; Rogers 1988, 111). Haslar was open for over two hundred and fifty years until it was closed by the Ministry of Defence in 2007.

3.4.1.2 Stonehouse Hospital, Plymouth.

Stonehouse Naval Hospital in Plymouth was the second naval hospital to be built at a home base that was planned and constructed by the Admiralty. Stonehouse Hospital was deliberately built several years after Haslar was completed and open to patients. The main reason for this can be attributed to the nerviness of the Admiralty in regards to the success

of Haslar hospital and for a period there were also alternative plans to convert a nearby prison which would have proved significantly cheaper for the Admiralty at a cost of only £300 (Buchanan 2005). The construction of Stonehouse hospital was begun in 1758 but not completed till 1762, however the hospital was open to the sick from 1760. Prior to the construction of the Stonehouse, the sick were transferred to the hospital ship the Canterbury which was moored off Plymouth. When Stonehouse was constructed a landing jetty in Stonehouse Creek was used to transfer patients to the hospital.

The short delay in time between the planning and the building of Stonehouse is reflected in its architecture which echoes new 'Enlightenment Clinical Theory' (Buchanan 2005). Stonehouse hospital was designed by Alexander Rovehead who was helped by William Robinson who was Clerk of the Works to Greenwich Hospital. Rovehead's plan was to plan a hospital built "for the purpose of admitting freer circulation of air and also of classing several disorders in such a manner, as may best prevent spread of contagion" (Brebbia et al.

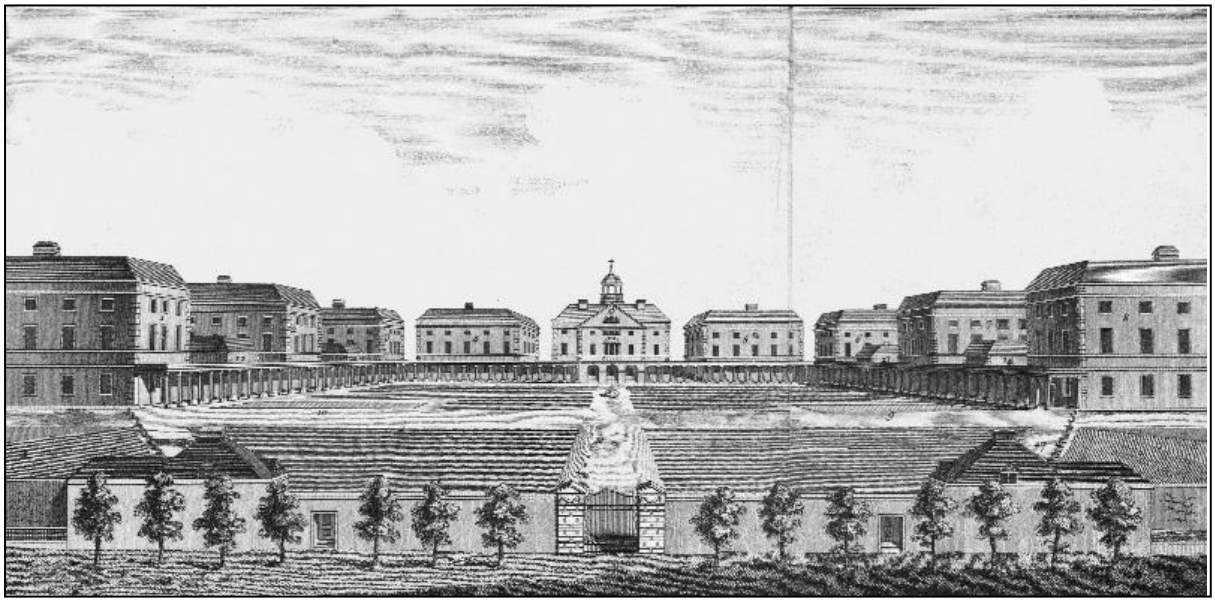

Figure 3.5 The Royal Naval Hospital at Stonehouse, Plymouth from the French edition (1788) of Howard's State of the Prisons. Two ward blocks at the front are omitted; note that the hospital blocks are made up of separate buildings. Reproduced from Stevenson 2007.

2010 461). This design was emphasised in the pavilion plan of Stonehouse hospital which helped to prevent the spread of disease between each ward and can be seen in both Figure 3.7 and 3.8. Stonehouse hospital consisted of ten separate buildings that were connected using a colonnade which was roofed and connected each block to the other and to the church. The ten pavilions were each 65 feet long and 46 feet wide, three storeys high and contained six wards. The pavilions were laid out on three sides of a square and the whole area was surrounded by high walls. Cross ventilation was essential in this plan and the spacing between each block helped to prevent cross infection. In total the hospital could accommodate 900 patients but in extraordinary circumstances up to 1500 could be housed in Stonehouse. What is of particular note is that infectious patients were separated from non-infectious which was an important step towards modern medical practices (Buchanan 2005, 226). Many contemporary writers were very impressed by the Stonehouse plan, including the prison reformer John Howard and the French writer Jacques Rene Tenon (Buchanan 2005, 226). John Howard was particularly impressed by the separation of each block which prevented the spread of infectious disease (Howard 1784). Lecointe's subsequent design of French hospitals was visibly influenced by Stonehouse's pavilion structure (Buchanan 2005, 227). The pavilion style plan was very effective at preventing disease spread as each pavilion was used as 'quarantine' for a particular disease (Buchanan 2005, 226). The pavilion style plan of Stonehouse was completely different to the palatial style employed at Haslar and it embodies the major changes in medical theory that were coming to pass at the time. This efficacy is shown in the death rates of Stonehouse hospital when compared with Haslar (Blane 1799 and ADM 102 Hospital Muster records), which will be discussed later in chapter five. Several contemporary observers commented on

Stonehouse hospital including John Howard the prison reformer who was very impressed by the architecture and running of the hospital. Jacques Rene Tenon and Charles Augustine

Figure 3.6 Modern aerial photograph of Plymouth Naval Hospital, showing an aerial view of the separate hospital buildings. Reproduced from www.plymouthdata.info.

Coulomb who were prominent French scientists commented on Stonehouse and they adapted the pavilion hospital plan in France where it was very successful. The cleric John Wesley also visited Stonehouse in 1785 when he was 82 years of age. John Wesley spoke highly of Stonehouse; "I took a walk through the Royal Hospital for sick and wounded sailors, I never saw anything of the kind so complete; every part is so convenient, and so admirably neat"...he does however note that it is found wanting "a man of faith and zeal to watch over the souls of the poor patients and teach them to improve their affliction" (Wesley 1916).

It is important to consider the significance of Stonehouse in the development of military architecture as it was one the first hospitals in the world to emphasise ventilation and disease segregation. Stonehouse marked an evolution from Haslar where seclusion and imprisonment were at the forefront of planning to an increased emphasis on health and wellbeing (Buchanan 2005). This architectural revolution transformation was advantageous to the overall status of the Navy as it decreased the amount of time that seamen spent in Stonehouse and it lowered the overall mortality rate when compared to Haslar. Stonehouse marked a massive progression in international healthcare architecture and it was widely regarded as the finest hospital in Europe (Pugh 1972).

3.4.2 Diet in the Royal Navy

The diet of the Royal Navy was of vital significance to its overall capacity and success (Crimmin 2007, 192). Victualling the Navy was a difficult business as rations had to be limited to foodstuffs that were relatively inexpensive and easily preserved for long periods of time onboard (MacDonald 2004, 9). Food was issued and food rations were regulated by the Victualling Board (Davies 2002, 35). There was also a massive logistical obstacle for the Victualling Board since during wartime, they had to supply rations for up to 147,000 men (Bannerman 2011, 169). Table 3.2 specifies the weekly ration for each man but these could be easily substituted when standard rations were unavailable (MacDonald 2006, 176). Once a week, flour and raisins were swapped for beef (Pope 1997, 151) and pease were replaced with fresh vegetables where possible “when they can be procured and not at any time exceeding the value of pease saved, at the Pursers credit price” (1808 Victualling Regulations in Lloyd and Coulter 1961, 81). Seamen were notoriously conservative and “did

Table 3.2 Standard Naval Rations for each man shown by days of the week, adapted from Rogers 1988, 83.

	Bread	Beer	Beef	Pork	Pease	Oatmeal	Butter	Cheese
Sunday	1 lb.	1 gal.	-	1 lb.	½ pt.	-	-	-
Monday	1 lb.	1 gal.	-	-	-	1 pt.	2 oz.	4 oz.
Tuesday	1 lb.	1 gal.	2 lbs.	-	-	-	-	-
Wednesday	1 lb.	1 gal.	-	-	½ pt.	1 pt.	2 oz.	4 oz.
Thursday	1 lb.	1 gal.	-	1 lb.	½ pt.	1 pt.	-	-
Friday	1 lb.	1 gal.	-	-	½ pt.	1 pt.	2 oz.	4 oz.
Saturday	1 lb.	1 gal.	2 lbs.	-	-	-	-	-

not like the foreign substitutes for their regular diet" (Rogers 1988, 86). During wartime, little time was spent in port and therefore fresh vegetables were uncommon and were rarely procured (Davies 2005, 36). Whilst at sea 'bread' rations refer to the ship biscuit or hardtack, these hard biscuits were impossible to eat without being soaked first, each biscuit was usually broken into pieces and added to soup (MacDonald 2004, 17). The biscuit could be kept fresh for months at a time when stored in an airtight container. The standard meat rations usually consisted of beef and pork which was salted and stored in casks (Rogers 1988, 83). The historical records also indicate that live animals were also kept on board, which were slaughtered as needed. These animals included pigs, cattle, goats and fowl, but this was only practical for the short term, as many animals didn't cope well at sea (MacDonald 2004, 86; Davies 2002, 34). Fresh fish was also available and it has been recorded that seamen even ate exotic animals like turtles, tortoises, dolphins and sharks when available (MacDonald 2004, 40). Raisins were the only fruit that was officially part of naval rations, however pursers and officers did buy extra fruit and vegetables at their

discretion and these were considered to be supplementary to standard rations. Popular vegetables that were frequently purchased were onions and cabbages which were sometimes used as anti-scorbutics, however these were never eaten raw (Rogers 1988, 86). Carrots, turnips, pumpkins and kale are also mentioned in the ships logs as being occasionally eaten onboard (MacDonald 2004, 36).

Rations may have been somewhat monotonous by today's standards, but are extremely generous by the standards of the eighteenth and nineteenth century and much better than the diet of the general populace who rarely had access to meat (Ashton 1955, 177; Crimmin 2007, 192). By looking at these rations, it is possible to calculate the average daily calorific intake for every seaman, which was somewhere between 4500 - 5000 calories (MacDonald 2006, 177), twice the modern daily guideline for an active man (NHS Guidelines 2012-www.nhs.co.uk). This shows how active these seamen were and it also demonstrates the value that the Admiralty placed on keeping the belly of the Navy full.

3.4.3 Vitamin C content of the Naval Diet

The basic rations allocated to each sailor are all quite low in Vitamin C (Davies 2002, 35). Basic Vitamin C content of food has been discussed in subchapter 2.1.3. Bread contains no Vitamin C (Serna-Saldivar 2012, 68). The standard naval meat ration was preserved by the addition of copious amounts of salt, then stored, which depleted Vitamin C content to a negligible level (MacDonald 2004, 21). It would therefore appear that Vitamin C was only available through animal protein when animals were slaughtered onboard and the offal was consumed when fresh. It is a similar situation with the vegetable rations, which were dried, and then stored for a long period of time, destroying the Vitamin C. This process is particularly damaging for peas, the type of dried peas used by the Navy had to be soaked

overnight and then cooked for several hours which is deleterious to Vitamin C (MacDonald 2004, 33-34). When fresh vegetables were available they were usually boiled or added to soups or stews which diminished the Vitamin C content. It is also important to note that on board most Naval vessels copper pots were commonly used for cooking (Allen et al. 2006). Jones and Hughes (1976, 81) showed a significant difference between the Vitamin C content of vegetables cooked in copper cooking pots versus vegetables cooked in iron pots. In their study they used cabbages that were boiled in a copper pot for twenty to thirty minutes and as a result the cabbage lost 65% of its Vitamin C content, whereas cabbage cooked in an iron pot for the same amount of time only lost about 18% of its Vitamin C (Jones and Hughes 1976, 81). This is a significant difference in Vitamin C loss which is caused by something as simple as using a different type of cooking pot. There has also been a small amount of research investigating the link between alcohol consumption and Vitamin C absorption. Faizallah and colleagues (1986) showed that in the hours after alcohol consumption that Vitamin C excretion from the human body increases significantly. In their study both lager and whiskey were tested, it was found that there was a 47% increase in Vitamin C excretion after the consumption of these drinks (Faizallah et al. 1986, 186). The daily ration of drink for each seaman was one gallon of beer (Rogers 1988, 83). This is not the equivalent of a modern gallon but is a wine gallon which about five-sixths of a modern gallon, this equates to about three and a half litres today (MacDonald 2004, 41). It must be remembered that their beer was quite weak at about 2-3% alcohol (MacDonald 2004, 40), but in such large quantities would have most certainly had an effect on Vitamin C elimination from the body. It has been suggested that Vitamin C actually aids the natural elimination of alcohol from the body. Chen et al. (1990,185-186) showed that healthy men that were given megadoses (2g/day) of Vitamin C for two weeks prior to consuming alcohol, cleared alcohol from their

blood much quicker than a control group. When beer ran out wine was drunk at the rate of about a pint a day, this would have been about the same strength as modern wine (11-13% alcohol content) (McDonald 2004, 41) and would have had a detrimental effect increasing further Vitamin C excretion. Alcoholic drinks were the only liquids that seamen were drinking on a daily basis, since water quickly turned sour when kept in casks, and alcoholic drinks did not (Davies 2002, 39).

Therefore, the standard rations that were provided by the Admiralty were for the most part woefully deficient in Vitamin C and the low levels of Vitamin C present were diminished by a number of other factors. These include the prolonged storage and cooking of food in copper pots and the high levels of alcohol that were consumed. This would have led to the development of scurvy quite soon into a voyage and would not have been helped by the sub clinical scorbutic state of many recruits prior to joining the Navy (Rogers 1988, 101).

3.4.4 Health and Scurvy in the Royal Navy

3.4.4.1 General Health in the Navy

The Georgian Navy was infamous for being very unhealthy fleet and particularly those squadrons assigned to the West Indies station (Rogers 1986, 98). During the Napoleonic Wars, eight times more men died of disease than of battle injury (Cook 2001, 95) Diseases such as fevers, fluxes, scurvy and sexually transmitted disease are commonly recorded in both the hospital admissions and the surgeon's logs (ADM 102). These diseases are directly related to the size of the fleet and the massive increase in naval manpower at this time led to a manifold increase in ill health. This is caused by many factors. The first of these is that

contagious diseases like the 'bloody flux' (diarrhoea) spread rapidly in the cramped conditions where the sailors slept (Cook 2001, 96). Also when the Navy was at its largest, squadrons in the tropics were common, which meant that men were struck with a whole range of tropical diseases such as malaria that they were unprotected against. Another major factor is the effect of scurvy on the general health of the Navy (Davies 2002, 1; Thomas 1991, 51). When the human body is in a scorbutic state the immune system is significantly reduced (Thomas et al. 1978, 370; Kennes et al. 1983, 305) which increases the likelihood of contracting other infectious diseases. Also due to weakened collagenous structures, it is possible that rupture (hernia) was more common in scorbutic individuals. It is frequently noted by the naval surgeons that those men with scurvy often suffered with fluxes and fevers (Lind 1772).

Scurvy is considered to be synonymous with the Royal Navy, the nature of the 'Wooden World' meant that it was isolated on sea voyages and could therefore not be easily supplied with fresh provisions (Rogers 1988, 100). As discussed in subchapter 3.4.3, the typical naval rations were woefully deficient in Vitamin C and this was further worsened by long term storage and cooking in copper pots. It has also been suggested that heavy exertion and a very physical lifestyle which is typical of that in the Navy can also hasten the onset of scurvy (Norris 1983). Scurvy has been recorded since the earliest Navies and could disable a Navy as easily as enemy attack (Carpenter 1986). One of the most famous examples of massive mortality caused by scurvy is that Anson's Voyage in the 1740's (Anson 1748), this is a key historical case study that will be examined in the next subchapter.

3.4.4.2 Commodore Anson's Voyage and Scurvy

In 1740, 1955 men embarked on a voyage with Commodore George Anson to complete a circumnavigation of the globe in order to seize Spain's territories in the Pacific, the route taken can be seen in Figure 3.9. Of all the men that set out, 1051 men died in the course of

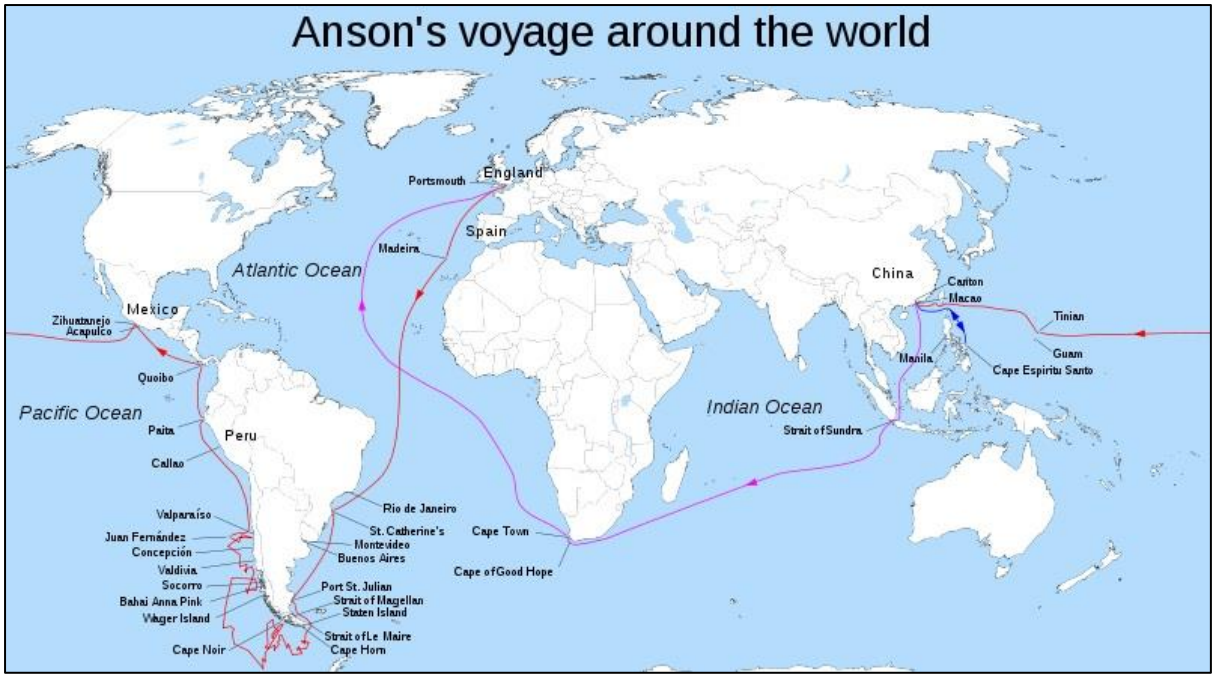

Figure 3.9 Map showing the route of Anson's voyage ill-fated around the world. Reproduced from www.doveglion.com

the journey, primarily due to scurvy (Anson 1749). This case study epitomises poor victualling, bad crew choices and the provision of inappropriate medicines (Lloyd 1963, 359). The only medicines that was provided was Elixir of Vitriol along with the 'Pill and Drop of Dr. Joshua Ward', which was composed of wine, balsam and antimony, its only medical property being that of a violent diuretic (Lloyd 1963). Scurvy "raged with great violence" (Anson 1781) throughout the journey. Five hundred Chelsea pensioners were chosen to join Anson's voyage, of this number only two hundred and fifty nine arrived in Portsmouth. All of

whom were old and unhealthy, subsequently not one made it back to England. Scurvy appeared soon into the voyage and is described by Anson in the passage below;

'The common appearances are large discoloured spots, swelled legs, putrid gums and above all an extraordinary lassitude of the body, especially after any exercise whatsoever; this lassitude at last degenerates into a proneness to swoon and even die on the least exertion of strength.This disease is likewise attended with a strange degeneration of spirits, which shiverings, tremblings and a disposition to be seized with the most dreadful terrors on the slightest accident.' Commodore George Anson 1749, 101.

These symptoms also accompany modern scurvy cases and have been described in chapter two. The significance of Anson's voyage is that it served to highlight the fatal consequences of scurvy on long voyages where squadrons are ill-equipped and ill-victualled to deal with it (Cook 2004, 224). It was the outrage at the high mortality of this voyage that served to motivate young surgeons like James Lind to conduct experiments into scurvy (Lloyd 1963, 360). In fact James Lind dedicated his *Treatise on Scurvy* to Anson as it was Anson's account of the voyage that initially interested Lind in scurvy (Tröhler 2005, 519).

3.4.5 Naval Surgeons and the Introduction of Lemon Juice Rations

3.4.5.1 Introduction to the Naval Surgeons and Introduction of Lemon Juice Rations

The lifestyle adopted by the men of the Navy made them extremely susceptible to the development of scurvy. Naval surgeons were intimately acquainted with the symptoms of scurvy and its symptomology is extensively described by many naval surgeons (Lind 1772; Blane 1799). These descriptions are identical to that provided by modern medical case

studies that have been described in chapter two. The Naval physicians were particularly concerned with the cause and potential cures of scurvy and their observations on this disease process were extensively recorded in their theses and books. There were a handful of very influential Naval writers in the early Georgian era including Thomas Trotter, Gilbert Blane and the Father of Nautical Medicine; James Lind. It was the influence and research of these men that led to the eradication of scurvy through the use of Vitamin C, this will be discussed in subchapter 3.4.6.5.

3.4.5.2 James Lind M.D (1716-1794)

James Lind is commonly referred to as the Father of Nautical Medicine (Lloyd 1963, 360) and did considerable work improving naval hygiene (Cook 2001, 96). Lind was a physician in the Navy from the 1740s when he became surgeon on the *Salisbury* in the Channel Fleet, where he conducted his now famous experiments on lemon juice and scurvy (Tröhler 1997, 50; Sutton 2003, 603). Whilst on a cruise on the *Salisbury* in the Channel after about a month men began to suffer from scurvy. Lind took twelve men aside and divided them into groups of two and allocated each a 'cure', they all had the typical symptoms of scurvy "they all in general had putrid gums, the spots and the lassitude, with weakness of the knees" (Lind 1772, 149). Lind created six test groups of which one group received a quart of cider, one received sulphuric acid, one received vinegar, one received seawater, one got a mixture of herbs and spices and the last group received oranges and lemons (Lind 1772, 149-150). Unsurprisingly Lind found that the men who received oranges and lemons soon recovered and were fit for duty (Lind 1772, 150). When Lind initially published these results little notice was taken, which is at least partly due Lind's own lack of faith in this cure. Even in his *Treatise*, Lind suggested that the main cause of scurvy was environment and that it was

caused by cold weather which led to a lack of sweating (Lind 1753). Despite Lind's lack of perseverance in promoting his results, it was his results and the perseverance of Trotter and Blane that in the long term led to the Admiralty adopting their lemon juice policy in 1795,

Figure 3.10 Portrait of James Lind with Haslar Hospital in the background. Reproduced from www.jameslindlibrary.org.

discussed in more detail later in subchapter 3.4.6.5. (Lloyd 1963, 360). In 1758, Lind was a surprise candidate to the prestigious position of Chief Physician at the newly opened Haslar (Bartholomew 2011, 9). At the end of his career at Haslar in 1783, Sir George Chalmers painted Lind's portrait with Haslar in the background, as can be seen in Figure 3.10. Lind continued in this role for twenty-five years and recorded many of his observations and work at Haslar in the final edition of his *Treatise on Scurvy* in 1772. During his time at Haslar from 1758 until 1783, Lind was exposed to extraordinarily high rates of scurvy in those men being

admitted (Lind 1772). It was at Haslar during the period of the Seven Years War that Lind was attending to between three and four hundred scorbutic patients every day (Lind 1772). The Channel Fleet was devastated by scurvy throughout the second half of the 1700s, in 1780 after a six week cruise with the Channel Fleet, 2400 men were transferred to Haslar hospital suffering from scurvy (Lloyd 1965, 178). Lind himself wrote extensively about scurvy and conducted autopsies on those men that died of scurvy in the hospital (Lind 1772). Lind's Treatise was the only comprehensive historical and medical review of scurvy until Alfred Hess published his book in 1920 (Hess 1920). The Treatise is of particular importance because it is a very wide-ranging historical literature review of scurvy, its medical symptoms and potential cures which related to both land populations and seamen. Lind's research was instrumental in the eradication of scurvy.

3.4.5.3 Gilbert Blane (1749-1834)

Gilbert Blane is widely considered to be "the most distinguished and influential of all naval physicians" (Lloyd 1965, 132). At the start of his career he was profoundly influenced by Lind and by the time he was appointed to the position of Physician of the Fleet in 1780 he was committed to improving its health (Ellis 1969, 192). As the first Physician of the Fleet to receive monthly mortality and illness returns from the surgeon of each ship, Blane was probably one of the first naval physicians to have a well-rounded idea of mortality and illness in the whole Navy. Blane recognised that mortality caused by disease was too high and he critically estimated that if the mortality rate that existed in 1779 continued then the supply of seamen would have run out before the defeat of Napoleon (Lloyd 1965, 133). He published extensive mortality figures in his book; *Observations on the Diseases of Seamen* (Lloyd 1965, 132). Blane was one of the first people to recognise the magnitude of the work

of James Lind on the efficacy of lemon juice in the cure of scurvy. Blane had a lot of faith in the effectiveness of lemon juice but not in the boiled 'rob' that Lind recommended (MacDonald 2004). Blane conducted his own experiments on lemon juice on the *Suffolk* which was on passage to India. For this trip Blane mixed lemon juice with sugar and grog and for the most part it prevented scurvy. It was Blane's influence that helped to reduce rates of scurvy in the Navy and it was him that convinced the Admiralty to introduce lemon juice rations to all seamen. The implementation of Blane's policies reduced mortality significantly and made the Royal Navy the powerful military force that defeated Napoleon.

3.4.5.4 Thomas Trotter (1760-1832)

Thomas Trotter is by far the lesser known of the key Naval surgeons. In 1793 he became second to the chief physician at Haslar hospital and the next year was appointed the chief physician to the Channel Fleet. Trotter was best known as a medical reformer and was particularly interested in scurvy and published his *Observations on Scurvy* in 1786 and a very influential three volume book *Medicina Nautica* (Cook 2001, 96). Both of these books were only concerned with the diseases of seamen and did not really place these diseases in their wider context. Trotter was very much an advocate of the work of Lind, but like Blane thought lemon juice should be served fresh (Trotter 1804). However, Trotter did not see lemon juice as a preventative for scurvy but as a cure when scurvy occurred. Trotter was of the opinion that lemon juice was not good for the constitution "it cures scurvy and preserves human life, but at the same time it weakens the digestive powers, consumes fat, and lessens muscular vigour" (Trotter 1804, 74). Trotter was a supporter of providing salads and apples and also he was one of the promoters of lemon crystals that were produced from lemon juice to cure scurvy. Trotter must also claim some credit in introduction of

lemon juice rations (Tröhler 2005, 522) and ultimately he had the health of ordinary seamen at the heart, he states in his *Medicina Nautica* that "the reader may smile at the idea of a physician to the Fleet, attending to stalls at a vegetable market, or perambulating the country to calculate the produce; but it never seemed to me below the dignity of the profession, nor did I consider it a mean task to serve the sallad(sic) with my own hands from the *Charon's* quarter deck" (Trotter 1804, 129).

3.4.5.5 Introduction of Lemon Juice Rations

The introduction of lemon juice rations in the Royal Navy effectively heralded the age of Nelson and British success at sea (Lloyd 1963, 360). It was men like Blane, Lind and Trotter that strove to improve the health of ordinary sailors in the Royal Navy, and their work is particularly apparent in the lowering of scurvy rates and its eventual eradication from both the Merchant and Royal Navy which occurred in the 1850's (Harvie 2002, Lloyd and Coulter 1961). It was Blane in 1795 that persuaded the Admiralty to make lemon juice regulation issue, after six weeks at sea relying on salt provisions (Lloyd 1963). Blane was by this period a commissioner on the Sick and Hurt Board (Tröhler 2005). In 1796, lemon juice was issued in small cylindrical bottles that held half a gallon of juice, lemons were imported to Britain, where they were squeezed, packed and then issued (MacDonald 2006, 161-162). This was not however the end of the scurvy, as lemon juice was provided 'at the discretion of the ships surgeon'; therefore if that particular surgeon did not believe in the efficacy of lemon juice, then he may choose to carry another antiscorbutic or nothing at all. This will be discussed further in subchapter 6.3.

3.5 Religion in the Eighteenth and Nineteenth Century

One of the primary comparative groups studied was a group of Primitive Methodists and to further understand this context, eighteenth century religion and in particular Methodism will be discussed in the following sections. Eighteenth century England was a place of great change, the Industrial Revolution changed the country beyond recognition. This transformation affected all aspects of people's life and society (Plumb 1963, 84). Urbanisation exacerbated high infant mortality rates, low life expectancy and malnutrition. For poor individuals', life was arduous, monotonous and uncertain (Plumb 1963, 89). Alcoholism was a huge problem, "alcohol was an essential narcotic which anaesthetised men against the strains of contemporary life" (Tomas 2003, 49). There were also massive society problems with gambling and violence. There was a need among ordinary working people for something to live for and for a sense of salvation. This came in the form of religion and specifically John Wesley and the formation of the Methodist Movement (Kent 2002, 1). John Wesley was a fervent theologian, who preached extensively and was rejected by the Anglican Church, where he was trained. He later went on to found Methodism, it appealed to the masses as "it contained so much that was capable of satisfying the deepest needs of human nature" (Plumb 1963, 95). Methodism was particularly attractive to the poorer echelons of society as they not considered welcome in the established church and particularly those living in industrial areas (Yates 2008, 91). Methodism was believed to be the only successful religious movement in a period where "the nation seemed to linger in a state of spiritual inertia" (Olsen 1999, 279). Methodism was also a very attractive religion because it was much more democratic, ordinary people having a say in how the church was run and taking a much more active role (Plumb 1963).

John Wesley and his brother founded their religious non-conformist movement in 1729, it however remained within the Anglican movement until the 1780s when John Wesley began to ordain his own Methodist priests (Olsen 1999, 286). This event marked the split between Methodism and Anglicanism and marked the formation of new religious movement. Methodism was a simple evangelical religion that emphasised systematic worship and an all-inclusive membership and a more democratic structure (Kent 2002, 96). This attitude made it a religion for the poor (Plumb 1963, 94) and its membership grew from 24,000 in 1767 to 77,000 by 1796 (Olsen 1999, 286; Yates 2008, 91). Methodism had a unique 'emotional core' that was lacking from the other religious institutions that emphasised quiet and restrained worship. Methodists were frequently loud, fervent and expressive in their religion and when led by Wesley this faction increased in size and vigour (Plumb 1963, 95; Stevens 2012, 63).

3.5.1 Primitive Methodism

The group that forms part of this study was that of a collection of skeletal remains exhumed from a Primitive Methodist cemetery. Primitive Methodists were a sub-group of the larger Methodist movement. This substantial group was led and established by the preacher Hugh Bourne, who was a poor farmer born near Stoke-on-Trent (Kendall 1906). He and his followers felt disillusioned, believing by the early 1800's Methodism had lost its way from the original ideas and zeal of John Wesley (Price 2012, 36). Primitive Methodists were expelled by the mainstream Methodist movement and broke away from the mainstream in 1810 they were, in general very poor socioeconomic group with their strong idealistic principles (Patterson 2012, 4), not far removed from main ideals of Wesleyan Methodism,

but with certain differences. Primitive Methodist preachers preferred simple language that lay people could easily understand and they encouraged female preachers , Sarah Kirkland becoming their first female preacher in 1812 (Price 2012, 42). This emphasis on a more 'primitive' form of Methodism endeared poor labourers to their cause and led to large groups of Primitives being established in the Northern counties of England (Oxford Archaeology Interim Report 2011, 9). It has been said that "Primitive Methodists was a religion of the poor, orthodox Wesleyanism remained as it had commenced a religion for the poor" (Thompson 1991, 41). One specific Methodist ideal provides a stark contrast to the Naval lifestyle is that of alcohol consumption. Prior to 1987 all Methodists were expected to be totally abstinent from alcohol, in fact at the Wesleyan Conference in 1874, a Temperance Committee was appointed to suppress intemperance and close public houses (chippenhammethodistcurcuit.co.uk). For a time, Methodists did not even use Eucharist wine and replaced it with grape juice (Abraham and Kirby 2009).

The Primitive Methodists of Darwen are typical as a poor rural group of cotton mill labourers; this group epitomises the ideals of Methodism. Methodists also became strongly involved in the Trade Union movement (Ritson 1909, 277-8). Primitive Methodism was first recorded in Darwen in 1823 (Wright and Colling 1908, 28), but was not until 1832 that the first Primitive Methodist chapel was built on Redearth Road, the cemetery of which is the basis of this research (Oxford Archaeology Report 2011, 9). The location of Darwen within the county of Lancashire can be seen in Figure 3.11. The chapel was built by the Primitive Methodist parishioners of the area and the cemetery was adjacent. Burial registers survive for the cemetery from 1847 but it is thought that the burial ground began its use at the same time as the chapel (Gifford 2006). Diseases that would have commonly affected the

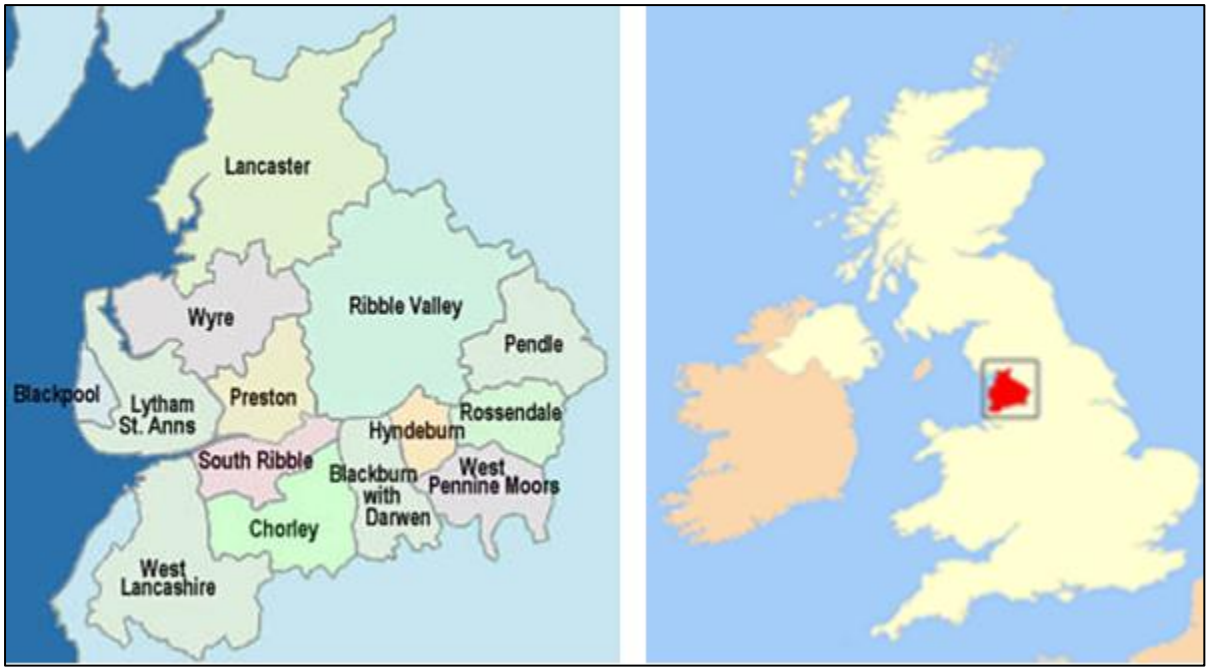

Figure 3.11 Map showing the location of Darwen which is labeled as 'Blackburn with Darwen'. Reproduced from wiki.fot-net.eu.

Darwen population would have been typhus, fever and dysentery (Oxford Archaeology Interim Report 2011). It has also been shown from historical sources that this population suffered from dietary deficiency diseases such as rickets and scurvy in times of food shortage (Lomax 1986, 71).

3.6 Historical Context Summation

Adult scurvy during the post medieval period was not just confined to the Royal Navy, but also affected land populations (Carpenter 1986). The four assemblages that form the basis of this study come from three distinct societal groups of a generally contemporaneous time period. The two primary groups are those from the two Naval hospital burial grounds, one originates from a post medieval prison and the other a religious burial ground of a working class population. These groups do in general represent those people from the lower echelons of society, but are geographically varied and come from very different cultural contexts with varying dietary and food intakes. Scurvy in these assemblages broadly results

from lack of fruit and vegetables in the diet due to differing cultural reasons such as occupation, poverty and imprisonment.

The study of scurvy in these collections allows us to infer the occurrence of scurvy in Naval and non-Naval groups in post medieval England during the study period. The historic and cultural backgrounds to these sites are fundamental to the interpretation of the skeletal lesions of scurvy and the recognition of scurvy in these archaeological populations. It is essential to investigate the historical evidence of scurvy and then explore whether or not this is also evident in the skeletal material.

3.7 Review of Adult Scurvy in the Palaeopathological Record

3.7.1 Introduction to Disease Identification in Palaeopathology

Osteoarchaeology concerns the creation of an osteobiography for skeletons or skeletal assemblages which will usually include assessment of age, sex, health, stature and if relevant; ethnic ancestry (Mayes et al. 2008; Buikstra and Ubelaker 1994, Brickley and McKinley 2004). This research is primarily concerned with palaeopathology, which is an essential subgroup within osteoarchaeology. Palaeopathology is the study of disease in archaeological human remains and is one of the primary methods by which we gain knowledge about diet, health patterns, trauma and disease in the past and are often the main focus of osteoarchaeological studies (Mays 1997). Within the study of palaeopathology, the study of metabolic bone diseases is of fundamental significance as these diseases specifically reflect diet (Brickley and Ives 2008). Palaeopathology is somewhat limited in its use to bioarchaeologists in that it can only provide us with information on long-term diseases (Roberts and Manchester 2005, 7). Acute diseases that

cause rapidly ensuing death usually leave no visible mark or lesions in living human bone. Palaeopathology is also somewhat limited by bone remodelling, as bony lesions will heal over time and if the time of death is many years later, there may be no traces visible in the human skeleton. However, the very nature of metabolic diseases lies in their chronicity, it is a protracted period of malnutrition or imbalanced diet that leads to the development of metabolic diseases and subsequent imbalances in bone modelling (Brickley and Ives 2008, 11).

3.7.2 Introduction to Metabolic Disease Identification in Palaeopathology

Metabolic bone diseases are defined as those diseases that affect the processes of remodelling and bone formation in the whole skeleton (Albright and Reifenstein 1948). This group of diseases includes; rickets, iron deficiency anaemia, osteomalacia, osteoporosis, Paget's disease of bone, and of course, scurvy or Vitamin C deficiency (Brickley and Ives 2008). The majority of these diseases arise from an imbalanced diet that is deficient in vital nutrients (Roberts and Manchester 2005, 223). Metabolic diseases are very complex and they rarely occur on their own, often several metabolic diseases occur alongside each other and they also frequently occur with infectious disease (Barlow 1883). The reason for this is that metabolic diseases lower immunity which makes individuals more susceptible to infections. This does make it difficult to identify and distinguish metabolic diseases in the skeleton. Metabolic diseases are the expression of many social, cultural, genetic and dietary factors on the human body. The interactions between these influences are detailed in Figure 3.12. The study of metabolic diseases is vital to build a complete picture of health in past societies and is particularly important to chart changing health patterns since the advent of agriculture (Brickley and Ives 2008).

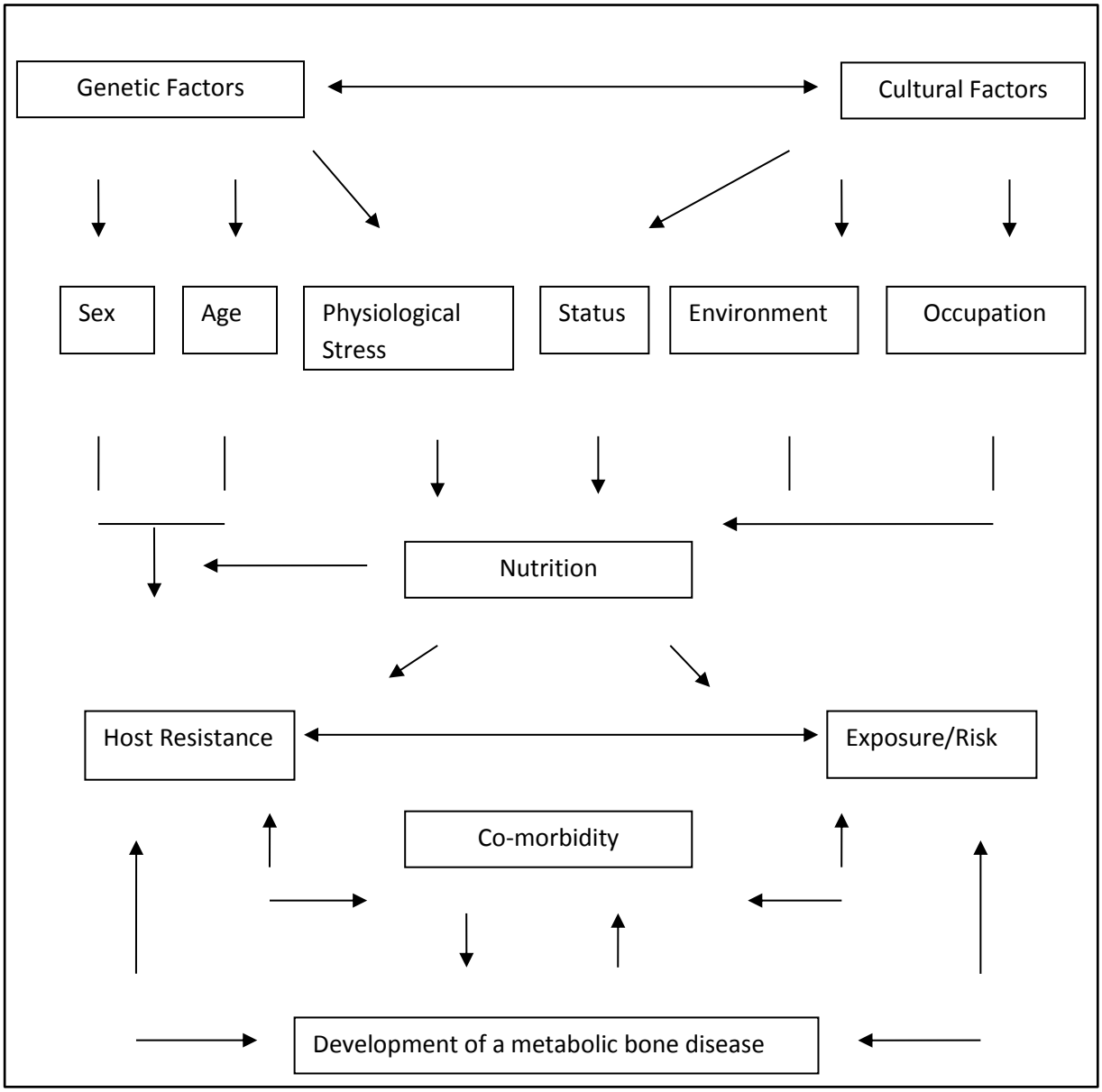

Figure 3.12 Outlining factors that affect the development of metabolic diseases, adapted from Brickley and Ives 2008.

3.7.3 Bone Modelling and Remodelling

Bone is a living tissue, as such needs nourishment and is in a constant state of turnover and replacement. Bone is composed of a mixture of organic collagen fibres and non-organic mineral component consisting primarily of a form of hydroxyapatite (Sommerfelt and Rubin 2001). All bones are surrounded externally by an osteogenic layer of tissue known as the periosteum and internally by the endosteal membrane (Baron 1999). In the course of life,

bone is continually being taken away and replaced by newly produced bone. This process is known as modelling and remodelling.

Modelling and remodelling in the skeleton occurs to allow bone growth and changes in bone mechanics and consists of mature bone being resorbed and new bone being formed (Scheuer and Black 2000). This process continues throughout life as between 2-5% of the human adult skeleton is replaced every year (Bonewald 2006, 331). This also repairs bones after fracture and responds to activities such as mechanical loading.

Modelling and remodelling are conducted by two primary bone cells; osteoclasts and osteoblasts (Gerhard et al. 2009). Osteoclasts are a cell that destroy bone cells by resorption, the osteoclast portions off a small section of bone and dissolves it using biological enzymes. Histologically speaking, this process results in a scalloped appearance (Brickley and Ives 2008). Osteoblasts then exude new organic matrix and adjust its mineralisation, all controlled by hormone processes. These osteoblasts then die or become osteocytes and incorporated within the bone matrix (Doblaré et al. 2004). The function of these osteocytes is to communicate to osteoblasts and osteoclasts in order to stimulate further bone modelling and remodelling. All of these processes are controlled by a multitude of proteins, hormones and growth receptors (Lemairea et al. 2004). It is an imbalance in the modelling and remodelling process described here that characterise a metabolic disease (Brickley and Ives 2008).

3.8 Introduction to Scurvy in the Archaeological Record

There is significant documentary evidence of scurvy occurring in historical contexts. Most authors cite an ancient Egyptian document known as the Ebers Papyrus as being the earliest historical reference to this disease (Brickley and Ives 2008, Fain 2005, Dolberg et al. 2010).

The Ebers Papyrus is a medical papyrus dating from 1550BC, which contains numerous medicinal remedies and incantations to cure disease. Scurvy was also particularly commonly recorded in post medieval documentation, however there has been relatively little in this area, which has resulted in an increase in the reporting of cases of both adult and archaeological evidence to back up the significant amount of historical references (Geber 2012, 512; Roberts and Cox 2003, 306). Recently there has been significant progress identifying juvenile scurvy in archaeological assemblages (Geber et al. 2012, Bourbou 2003a and 2003b, Brickley and Ives 2008, Mays 2008, Ortner et al. 2001, Van der Merwe et al. 2009, 2010). This can be attributed to a number of factors, but the primary cause has simply been an increasing awareness of the identification of scorbutic lesions, especially in infants, among osteologists. The following sections will concentrate on the macroscopic osseous lesions of scurvy followed by a review of the published archaeological cases of scurvy, concentrating on cases of adult scurvy. As part of this, there will be an effort to link the pathological manifestation in the living human body to that of the palaeopathological manifestation in dry human skeletal material.

3.8.1 Introduction to Archaeological Literature Review of the Macroscopic Lesions of Scurvy

One of the fundamental goals of bioarchaeologists is to determine the occurrence of metabolic disease in the past and in the case of this study Vitamin C deficiency. The most important point to consider in the study of scurvy is that the skeletal lesions of scurvy are in most cases indicative of the healing stage of this disease, when at least some Vitamin C has been re-introduced into the diet and the body is recovering or recovered (Brickley and Ives 2008, 48). When the body is recovering, healing can take place quickly and Follis et al. (1950) proved that in infantile scurvy there will be no histological evidence of scurvy in as little as 3 months after a scurvy episode. The next section will address how the lesions of scurvy manifest in human body and how bone reacts to scorbutic haemorrhageing and extravasation of blood throughout the body.

3.8.2 The Biological Reaction of Living Human Bone to Haemorrhage and Inflammation

The primary pathological manifestation of scurvy in the human body is a mixture of haemorrhagic and inflammatory processes. Living human bone reacts to stimulus in two main ways; these are bone removal and bone formation or a mixture of both of these processes (Brickley and Ives 2008). These processes are vital to the identification of pathological lesions in dry archaeological bone. In scurvy the main bony lesions are porosity or porous lesions and also bone formation which may be haematomas or periosteal reaction. Porosity or abnormally porous bone surfaces are thought to be caused by inflammation resulting from haemorrhageing. This can be explained as one of markers of scurvy is the extravasation of blood from damaged capillaries into the surrounding tissue and muscle. This then leads to the propagation of new capillaries at the site, which reach

the site through the increased vascularity and pores in the bone which can be identified macroscopically (Ortner et al. 1997). The second type of osseous lesion associated with scurvy is new periosteal bone formation or ossification of haematomas. The periosteum itself is a dense layer of connective tissue that is formed of two distinct layers. The outer layer is the 'fibrous' layer and the inner is 'osteogenic' layer. It is the osteogenic layer that contains osteoblasts and these lie in contact with cortical bone surface (Dwek 2010). The periosteum supplies the bone with nutrition and oxygen so that new bone can be produced when trauma occurs. When inflammation and/or haemorrhageing occurs in the soft tissues there is often no direct effect on bone but if these processes occur adjacent to the periosteum then bony reaction can occur. When the periosteum is inflamed or traumatised it can be raised from the bone which is normally adheres very tightly to, by structures known as Sharpeys Fibres. Periosteal lifting and haemorrhageing can also be caused by the rupture of a subperiosteal blood vessel (Griffeth et al. 1997). This can lead to sub periosteal bone reaction or ossification of blood clots that accumulate underneath the periosteum. Initially this new bone will be new 'woven' or immature bone and will appear as being very porous but this will over time become lamellar or mature bone (Ortner and Erickson 1997). Ossified hematomas will be recognisable as a usually oval area of appositional new bone.

It is a mixture of porosity, vascularity and new bone reaction that make up the cranial and postcranial bony lesions of scurvy. The following paragraphs will examine archaeological cases studies, the lesions that are said to result from adult scurvy and the anatomical areas where these lesions commonly occur will also be discussed.

3.8.3 Published Cases of Archaeological Scurvy

There has been an increase in the publications of infantile scurvy in recent years with the work of Ortner et al. (1997, 1999, 2001, and 2004); however there still remains scant published evidence of adult scurvy. Adult scurvy is very much underrepresented in the palaeopathological literature and in the majority of palaeopathological texts just gloss over the subject of adult scurvy. In the following sections, the handful of published case studies of archaeological adult scurvy will be discussed. The majority of these case studies can be clearly linked to historical groups and specific time periods which are linked to periods of famine which subsequently led to deprived and malnourished populations. The diagnosis of scurvy is perceived as being more plausible if there is a definite association between a skeletal group and a historical event when we know that scurvy was present due to certain factors. The historical and archaeological background for each site will be discussed and the lesions that led to the diagnosis of scurvy in the skeletal material.

3.8.3.1 Spitsbergen Whalers

In the 1980s, the first detailed study of a scorbutic population was published (Maat 1987, 1984). These articles detailed a group of whalers that were buried at Spitsbergen in the seventeenth century. Spitsbergen is within the Arctic Circle and forms part of the Svalbard Archipelago. Fifty whalers that died here were buried in permafrost and as a result the soft tissue preservation was exceptional. The documentary sources indicate the whalers often suffered from scurvy due to the restrictive diet on board whaling ships. Lack of plant life in the cold conditions meant that even when men reached land there was no chance to consume Vitamin C rich foods. When these remains were excavated the preserved hematomas were found in the form of black maculae. These preserved stains were primarily

found on the lower limb bones and especially within with the joint surfaces (Maat and Uytterschaut 1984). There were also stains found on the tooth roots which were interpreted as gingival bleeding and some individuals had suffered from ante-mortem tooth loss and periodontal disease. Only one individual presented with any osseous changes that can be interpreted as scorbutic, this individual had a tibial ossified haematoma (Maat 1987). Maat also referred to what he described as 'bony infarctions' which are fractures in the cortical bone which he thought may be due to scurvy, however this might also have been caused by the Artic temperatures. What is fascinating about this study is that there would have been hardly any macroscopic evidence of scurvy, had not these exceptionally cold conditions allowed the preservation of blood and soft tissue, which poses interesting questions about scurvy in the osteological record. It appears possible for an individual on an extremely limited diet to have scurvy, die as a result and not display any osteological changes (Maat 1982). The Spitsbergen whalers provide an exceptional case study of adult scurvy where it is thought that all of these sailors died of the effects of scurvy. However no images of the pathological phenomena described here have been published so it is difficult to draw information from this case study when researching adult scurvy.

3.8.3.2 Kimberley Diamond Miners

Another recent publication which also involved George Maat was a population of nineteenth century miners from Kimberley, South Africa (Van der Merwe et al. 2010a, 2010b). This population consists of migrant black workers that moved into Kimberley to mine the rich diamond resources that were discovered in this new city (Van der Merwe et al. 2010a, 308). These workers were employed in large mines run by commercial companies. When these workers became unwell they were treated in compound hospitals in Kimberley

and those that died they were buried in a pauper's burial ground. Historical documentation indicates that these men suffered from many diseases including, syphilis, pneumonia, tuberculosis and scurvy. One of the diseases that was most commonly recorded was scurvy due to the lack of fresh fruit and vegetables in the city of Kimberley. In total, 107 skeletons were diagnosed with scurvy (Van der Merwe et al. 2010(a), 315). There were three criteria that were used in the possible diagnosis of scurvy; these are ossified hematomas on the anterior tibia, periodontal disease and sub-periosteal reaction. As part of the diagnosis of ossified haematomas, these lesions were histologically examined in order to determine if they were appositional in nature (Van der Merwe et al. 2010(b), 237). The most distinguishing criteria was that the cortical bone was to be unaffected by the overlying.

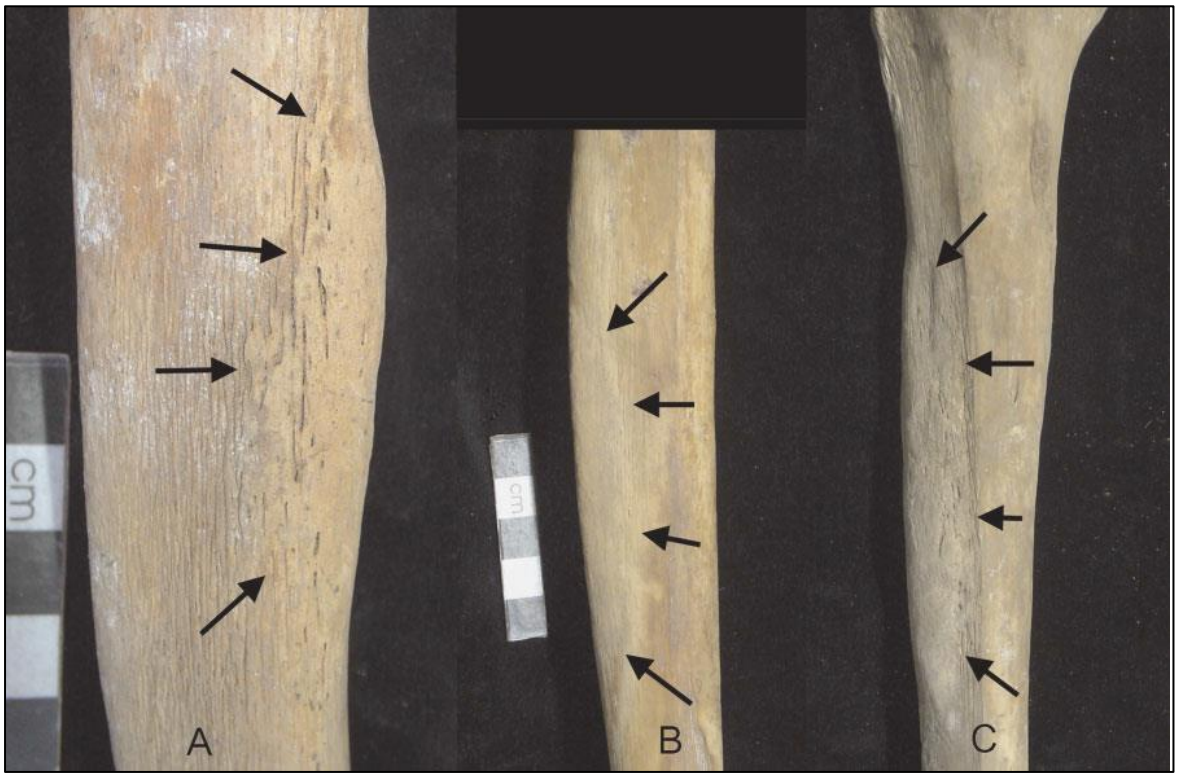

Figure 3.13 Examples of tibial ossified hematomas as marked by the black arrows from the Kimberley Diamond Miners. Reproduced from Van der Merwe et al 2010.

hematoma and was indeed as a result of an ossifying blood clot. These ossified hematomas are reproduced in Figure 3.13. This again is another excellent example of scurvy in a

specialist population with a limited dietary intake of fruit and vegetables due to location and circumstances

3.8.3.3. Saint Croix Island

Another exceptional site that has provided evidence of scurvy in adult skeletal remains is that of Saint Croix Island. Saint Croix Island is located in the modern day state of Maine, on the Canadian-American border. The Saint Croix River forms the border between the two countries. It was on this frozen island in the middle of the Saint Croix River that a group of explorers led by Samuel de Champlain were trapped in 1605 (Crist et al. 2005,5). Samuel de Champlain is known as the 'The Founder of New France' and was instrumental in the founding of the modern Canadian city of Quebec (Charles 2011, 26). De Champlain and his group experienced a bitterly cold Canadian winter that they were ill prepared for. They were forced to overwinter on La Croix Island which was only about six acres in size and was not big enough to support the group of about 79 men who were left on the island (Crist et al. 2005). The results were well documented and de Champlain later described the events in his book; *Les Voyages*, which was published in 1613. De Champlain records that 35 of the 79 men died due to what seems to be scurvy. He provides some graphic descriptions of the symptoms that the men suffered from;

"During the winter a certain malady attacked many of our people. It is called 'mal de la tere' otherwise 'scorbut'... There was engendered in the mouths of those that have it large pieces of superfluous fungus flesh which caused a great putrefaction; and this increased to such a degree that they could hardly take anything except in liquid form. Their teeth barely held in their places and could be drawn out with the fingers without causing pain. Afterwards they were taken by great pains in their arms and legs which became swollen and very hard and

covered in spots like flea bites; and they could not walk on account of the contraction of the nerves; consequently they had almost no strength and suffered intolerable pains"

Champlain 1922 from the 1622 original text reproduced from Crist et al. 2005.

These symptoms are identical to other historical descriptions of scurvy and to modern medical cases. Those men that died were all buried on the island, twenty-four of these have been excavated and recorded in two seasons of archaeological excavation (Crist et al. 2005). Many of these individuals that have been analysed exhibited what have been interpreted as the bony signs of scurvy. The majority of this evidence was concentrated on lesions of the cranial region and particularly that of the oral cavity. These lesions have all been attributed to osseous reaction to haemorrhageing. In the cranial region, lesions of the hard palate and tori were identified as being scorbutic in nature and porosity of the sphenoid. Other cranial lesions noted were porosity of the alveolar bone and new bone on the mandible near the mandibular foramen. Periosteal bone reaction was also observed on the femora and tibiae of nine individuals. A rare lesion that was recorded at the Saint Croix site was lytic lesions on the cervical vertebrae of at least seven individuals, which Crist et al. (2005) interpreted as being due to haemorrhageing of the vertebral arteries. These pathologies are accompanied by very strong evidence for mouth surgery which has been interpreted as being when the 'superfluous fungus flesh' was removed. The site of Saint Croix is unusual in that it has direct documentary evidence of scurvy provided by a reliable historical witness and the site can be dated very precisely. This is accompanied by an excellent osteological analysis that reports possible scorbutic lesions that have never previously been identified, along with a very promising diagnosis of scurvy based on lesions from both the cranial and

postcranial skeleton. However no photographs of any scorbutic lesions were included within this publication which is frustrating as the rest of the publication is excellent.

3.8.3.4 Puy St Pierre, Briançon France.

Puy St Pierre is a small community located on the slopes of Mount Prerol in the western Alps, this inhospitable location can be seen in Figure 3.14. The site itself is located on a rocky promontory and is subject to very harsh weather conditions and in particular long cold winters. The assemblage that forms this study was excavated from the church cemetery, which has been dated to between the 15th and 18th century (Berge 1999). The remains of 14 males, 22 females and 7 unsexed individuals were identified. Only the skulls were studied

Figure 3.14 Photograph of Puy St Pierre showing the Alps in the background. Reproduced from www.cartesfrance.fr.

for evidence of scorbutic lesions. In the skulls, several lesions were attributed to scurvy (Salis et al. 2005). These include palatine tori, cribra orbitalia, cribra cranii (porotic hyperostosis) and diffuse microporosity. In the study, they identified 12 individuals with

these lesions and diagnosed them with scurvy (Salis et al. 2005). This study inadequately address how the diagnosis of scurvy is reached and it is also unclear why the postcranial elements were not utilised in this analysis and no images were included in the publication.

3.8.3.5 Greenwich Naval Hospital

An assemblage of human remains that were excavated from Greenwich Naval hospital near London has also displayed possible signs of scurvy. Greenwich is a famous district in south east London that has long been associated with the Navy, and is the current home of the National Maritime Museum. This assemblage consists of a collection of skeletons from the hospital itself. This hospital was more akin to a retirement home for Naval veterans than a clinical hospital (MacQueen-Buchanan 2005), it was essentially a care home for old or disabled Naval seamen who were retired from the Royal Navy. Many of the individuals were invalided and discharged from the Navy due to amputations and there was an extremely high rate of trauma recorded (Boston et al. 2005). Since many of the men were many years retired from the navy and the likelihood is that any signs of scurvy in their bones would be well healed and remodelled. However, there were some well healed subtle lesions noted that could be attributed to scurvy. Sphenoid lesions were seen in twelve individuals and healed multiple element periostitis along with ante-mortem tooth loss and cribra orbitalia. 'Orange peel' ectocranial porosity was also noted in the assemblage. These lesions likely represent healed scurvy in this collection, which is not surprising. These seamen would have served in the Royal Navy of the eighteenth and nineteenth century and were therefore subject to the same on-board conditions as the primary assemblages being studied in this research. They would have almost certainly suffered from the effects of scurvy, but due to

their retired status it is difficult to confirm this in the skeletal remains and no specific images of possible scorbutic lesions were included in the monograph.

3.8.3.6 Mary Rose

An earlier comparative Naval assemblage is that of the Mary Rose, which was King Henry VIII's flagship that sank off Portsmouth in 1545. At the time the French Naval Fleet were moored off the Isle of Wight and the English Fleet were sailing to meet them (Marsden 2003). This was the maiden voyage of the Mary Rose and when she attempted to raise sail, it seems she was caught by a gust of wind, she keeled over and sunk in the Solent. There were four hundred and fifteen men on-board the Mary Rose, of which only thirty survived by swimming to shore. Most drowned, trapped by anti-boarding netting (Stirland 2005). The Mary Rose remained buried in the silt of the Solent until it was excavated in 1982 by the Mary Rose Trust. Significant quantities of human remains were recovered from the excavation, however all of these remains were extremely commingled (Stirland 1993, 19). Despite the unusual taphonomic conditions, all of the human remains were in excellent condition and the bone surface preservation was exceptional. The bone was studied as part of a large scale research study and a minimum number of individuals of one hundred and seventy nine individuals was established (Stirland 2005). Out of this there were ninety-two almost complete individuals. In all likelihood this represents about a quarter of the original crew. Amongst this exceptional assemblage, Ann Stirland puts forward a number of lesions which she thought might be indicative of scurvy, one of these was an 'orange peel' appearance of the external skull like porotic hyperostosis but thicker. She also observed cribra orbitalia, ossified hematomas and periostitis on the lower limb bones of some individuals which she concluded may be attributed to scurvy (Stirland 2000, 90). Stirland

urges caution in the diagnosis of scurvy due to the nature of non-specific lesions associated with this disease (Stirland 2005). It is hard to ascertain if these individuals do indeed show scorbutic lesions because of the lack of published data on the metabolic lesions in this assemblage. However, because this is a Tudor Naval assemblage it is likely that these individuals did suffer from scurvy due to the dietary restrictions caused by a seaboard life in previous voyages, as many of the crew were seasoned sailors. In Tudor times it is likely that scurvy was less severe because of the shorter sea journeys that were undertaken in this period. However, it is evident that a re-analysis of the remains in the light of new research would contribute significantly to the study of scurvy in the Tudor Navy.

3.8.3.7 Kilkenny Workhouse

The most recent and comprehensive study into the osteology of scurvy is Geber et al. (2012) who has published a study into the osteological manifestation of scurvy in an Irish Famine population. This population was excavated from the cemetery of the Kilkenny Workhouse in the South-East of Ireland. The individuals buried here were those that died in the Workhouse during the Irish Potato Famine in the 1840s. In this period the majority of the Irish population relied on the potato and in the mid-1840s when the potato crop failed the country was plunged into a large scale famine. One quarter of the country's population was lost due to death and emigration. To deal with this mass poverty and starvation, workhouses were established with the passing of the Poor Law Act of 1838 (Ferriter 2005, 31). Workhouses were government institutions were people worked for very basic food rations and were seen as being the very last resort for the poor. These buildings were overcrowded and disease ridden and as a result many who entered, died (Cousens 1960). When the Vitamin C rich potato was unavailable people began to develop scorbutic

symptoms and were more susceptible to disease and infections. In the workhouses there were very strict rations which in 1848 consisted of; 8 oz. of Indian Meal, 1 oz. of rice, ½ pint of milk, ¾ pound of brown bread and ½ pint of soup (Anon 1848 in Geber et al. 2012). However it is thought that in desperate times during the extreme food shortages, the diet would have simply consisted of bread and water. In the best case scenario of the 1848 rations, the only sources of Vitamin C would have been the milk which would have contained small amounts of Vitamin C plus the vegetable soup, which might contain a little, depending on which vegetables are being used, the cooking time and the cooking vessel. There is no reference to scurvy in the accompanying historical documentation of the workhouse; however it is thought by Geber (et al. 2012) that scurvy was mis-diagnosed by the workhouse physicians as gastric or typhoid fever. During the excavations in the cemetery of the workhouse, the remains of nine hundred and seventy people were excavated with all age groups were represented. Geber's study looked at both juvenile and adult scurvy, but for the purpose of this study, the emphasis will be placed on his research into adult scurvy. In his study he established a suite of lesions and created three categories of scurvy diagnosis; definite, probable and possible. These three groupings are reached by assessment of the number of scorbutic lesions or variables that are present. These variables are also categorised into three groups; definite, indicative and suggestive. These variables are split into those for subadult and adult scurvy. Some of the definite variables for adult scurvy are lesions of the sphenoid, palatine surface and maxilla. Examples of posterior maxilla and sphenoid lesions can be seen in Figure 3.15. Some of the indicative variables include multiple element periostitis on the femora, fibulae, tibiae and infraorbital area new bone reaction. Some suggestive variables put forward were sphenoid lesions of the greater wing, endocranial lesions of the parietal and periostitis of the femur and scapula.

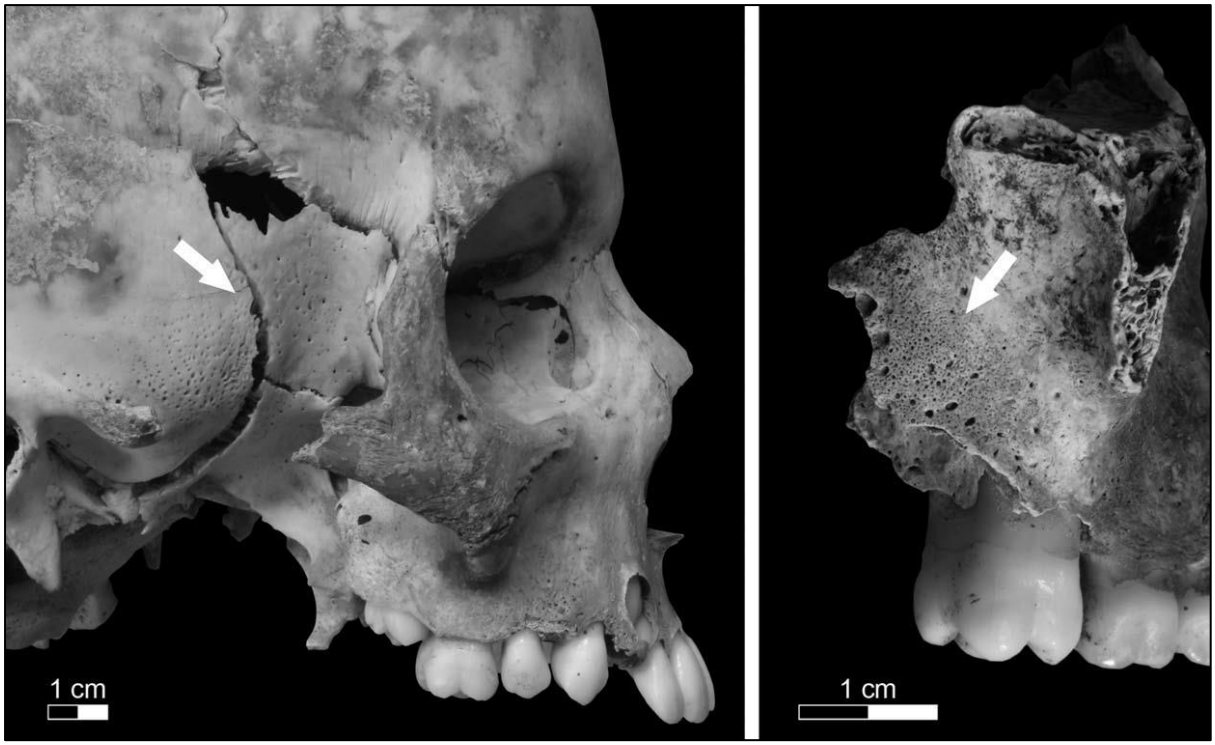

Figure 3.15 Photographs showing posterior maxilla and sphenoid lesions that Geber identified as scorbutic, reproduced from Geber et al. 2012.

Geber's approach is quite specific in this aspect and diagnosis of scurvy is directly linked to the manifestation of lesions. If definite variables are present then the skeleton is assigned a diagnosis of definite scurvy, indicative lesions for probable scurvy and so forth. The data put forward in Geber (et al. 2012) is very persuasive and provides an excellent basis for scurvy diagnosis in dry skeletal bone. Geber also found some fascinating correlations between height, sex and scorbutic lesions. Non-scorbutic men averaged at approximately 170cm and scorbutic men averaged at approximately 172cm, a statistically significant difference. Geber attributed this difference to physical size being the differentiation between scorbutic and non-scorbutic individuals in this population, the implication being that taller people need more Vitamin C for the same reason that males require more Vitamin C than females, so much so that in Canada the RDA OF Vitamin C for males in 15mg more than females (Health Canada 2005). Males were also 1.7 times more likely to be diagnosed with scurvy than

women in the Kilkenny population. It must be kept in mind that the Kilkenny Workhouse population is rather unique and is a prime example of a malnourished population that was particularly badly affected by consequences of Vitamin C deficiency. A study of this kind is exceptional as few skeletal assemblages can be directly associated with a historical famine event of such magnitude and provides a rare chance to study the effects of severe scurvy in a malnourished population.

3.8.3.8 Summation of Archaeological Cases of Scurvy.

All of these published instances of adult scurvy in the archaeological record are linked to a specific historical event or circumstance which in most cases is substantiated by significant historical documentation. These case studies all consist of populations affected by dietary restrictions and subsequent malnourishment due to economic and social conditions. It is difficult to establish cases of adult scurvy where there is no historical evidence for the disease. The problem with a lot of these studies is that each one uses a different set of criteria to establish the diagnosis of scurvy and there seems to be little agreement on the number of scorbutic lesions that should be used in order to establish a diagnosis in a single set of skeletal remains. Another major problem with these studies is that many are quite outdated and frequently the publications do not contain any images which may hinder the use of their criteria in the identification of scurvy.

3.9 Review of the Macroscopic Features of Adult Scurvy

It is agreed that the lesions of adult scurvy are quite subtle in their form. There are very few pathognomonic lesions that can be definitively linked to scurvy and many individuals will not have bony lesions even when they have scurvy (Mays et al. 2012; Brickley and Ives 2008,

62). Also due to the subtlety of these lesions it is possible that these lesions have gone un-noticed by osteologists because of the rapid recording that is so frequently required in the commercial archaeological sector. It is also true that many osteologists are apprehensive about possible over-diagnosis of diseases especially those that manifest through non-specific lesions. The emphasis should be placed on the interpretation of all present lesions and the historical background is also a vital aspect to consider. This section will concentrate on the suite of scorbutic lesions which have been compiled from an extensive literature review.

These potential scorbutic lesions have many differential diagnoses as they are non-specific by their nature (Lewis and Roberts 1997, 581). As part of this research, an effort has been made to differentiate between those periostitic lesions caused by trauma, infection etc. to those that are the result of scorbutic haemorrhageing. One of the primary methods of achieving this is a thorough examination of the patterning of skeletal lesions and making an effort to link anatomy and osteology. It is also important to consider other disease processes that may cause lesions similar to scurvy.

Most bioarchaeologists distinguish between infantile and adult scurvy, as it is thought these diseases have vastly different manifestations in their dry bone state (Geber et al. 2012, 515). As such they have received different treatment in osteological publications and it is thought that the bony manifestation of scurvy in subadults is much more severe than that in adults (Jaffe 1972). However, it is possible that there is not such a vast palaeopathological difference between adult and infantile scurvy. As such the Ortner et al. (1997; 1999; 2001) suite of lesions which was designed for infantile remains has been drawn on extensively to aid the recording of scurvy in dry bone in adult remains. Both diseases manifest as abnormal

haemorrhageing, the only difference is that the juvenile skeleton has not finished growth. The Ortner et al. (1997; 1999; 2001) suite of lesions is widely accepted as a reliable way of diagnosing scurvy in juvenile bone.

3.9.1 Identifying Scurvy in Skeletal Material

At the moment, there are two reliable choices for the identification of scurvy in dry bone material. The first of these is macroscopic analysis and the second is histology. There has been some work done in the area of collagen testing by Dr. Hannah Koon of Bradford University, however this research remains unpublished. There are several benefits to using macroscopic analysis for the identification of scurvy in dry bone. The first of these is the obvious disadvantage of using destructive sampling technique. The other main problem is that histology can only ascertain if new bone is present by examining cross sections but often this is visible to the naked eye of the trained observer anyway. Macroscopic analysis is also much more cost and time effective than destructive sampling, which is particularly important in the commercial archaeological sector. At the moment, there are no other methods of identifying scurvy but it is hoped that further work in the area of collagen testing will increase the reliability of this technique. For the purpose of this research, it was decided that macroscopic analysis would be best as permission could not be obtained for destructive sampling of the study material.

3.9.2 Macroscopic Lesions of Scurvy

The macroscopic lesions of scurvy in adults are detailed in the following sections and are divided into cranial and postcranial lesions. These lesions are classified by skeletal location, lesion type and a brief physiological/anatomical explanation of lesion causation. These

lesions have been compiled from a literature review of archaeological scurvy studies and medical studies.

3.9.3.1 Cranial Anatomy and Lesions

In the assessment of cranial scorbutic lesions, it is vital to consider the soft tissue anatomy of the cranium. The cranium and facial region is covered in complex layers of muscles and

Table 3.3 Showing cranial lesions of adult scurvy and their anatomical assessment.

Cranial Skeletal Location	Lesion Type	Anatomical Explanation
Ectocranial	Porosity of the external skull extending primarily over the parietals and ending at the temporal lines, looks like 'orange peel' and can be rough and shiny	These lesions are limited to the areas of the skull covered by the epicranial aponeurosis, and may represent haemorrhage of blood vessels under this structure or haemorrhage of the scalp.
Endocranial	Porosity, vascularity and new bone on the bones of the internal cranium, which may include the parietals, occipital, frontal and temporal.	These lesions are likely to be caused by haemorrhageing of the blood vessels of the meninges and leakage of blood from the cavernous sinus
Sphenoid	Porosity and new bone formation on the greater wing of the sphenoid, the foramina of the sphenoid and the pterygoid plates.	These are likely caused by bleeding of the muscles of mastication especially the pterygoids and haemorrhageing of the deep temporal arteries.
Orbits	Porosity, vascularity and new bone formation on the orbital roof	Haemorrhageing of the ophthalmic artery
Hard Palate	Abnormal porosity and new periosteal bone reaction	Could be caused by the outgrowth of flesh from the mouth and also the haemorrhageing of the alveolar and palatine branches of the third maxillary artery.
Posterior Maxilla	Abnormal porosity and new bone formation	Haemorrhageing due to the action of the pterygoid muscles and bleeding of the temporal and maxillary arteries
Zygomatic	Porosity and new bone formation on the anterior and posterior aspects and often adjacent to the zygomatico-facial foramen	Haemorrhageing from the blood vessels emerging from the zygomatico-facial foramen and proximity to the temporalis muscle

Dentition and Alveolar Bone	Ante-mortem tooth loss and abnormal porosity of the alveolar bone	Ante-mortem tooth loss is caused by loosening of the periodontal ligament. Abnormal porosity can be caused by haemorrhageing from the alveolar and palatine branches of the maxillary artery
Mandible	Porosity and new bone near the coronoid process, under the mylohyoid foramen and ramus and sometimes on the external mandible adjacent to the mental foramen	Porosity and new bone can be caused by haemorrhageing into the temporalis and masseter muscle groups.

many of these have been implicated in haemorrhageing , especially those muscles involved in mastication. All of these muscle groups can be seen in Figure 3.16 .It is also essential to consider the blood supply in this region. It has been suggested that it is the haemorrhageing of the blood vessels that supply these muscles that causes the ecchymoses and petechiae

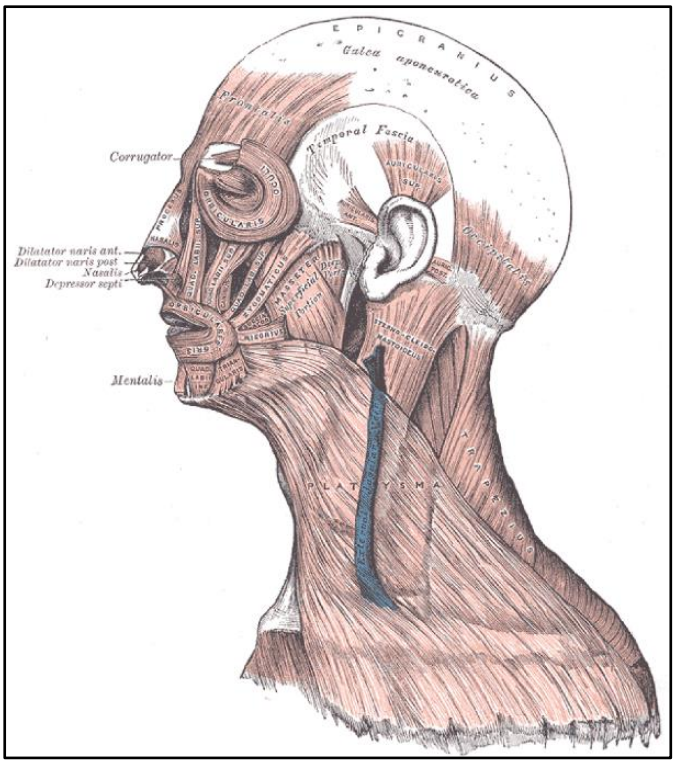

Figure 3.16 Diagram of cranial anatomy showing musculature. Reproduced from Gray's Anatomy.

that are so characteristic of scurvy. It is thought that the extravasated blood can accumulate in muscle bellies and at their attachment or origins. It is possible that anatomical structures are key to the patterning of the osseous lesions of scurvy. This is due to the fact that haemorrhages may come up against a soft tissue anatomical structure which may stop the progress of the haemorrhage. It is vital to consider the soft tissue structures and blood vessel supply in the cranial region in order to understand the osseous manifestation of scurvy in this area. The complex blood supply to the cranial area can be observed in Figure 3.17. This is also important when distinguishing between scorbutic and non-scorbutic reaction.

As mentioned previously, there seems to be a strong link between the lesions of scurvy and the muscles of mastication. In his Treatise on Scurvy, James Lind cites several historical authors who wrote about this. The first was the sixteenth century physician Solomone Alberto, who linked a contraction of the temporalis muscle to scurvy. He says this is most

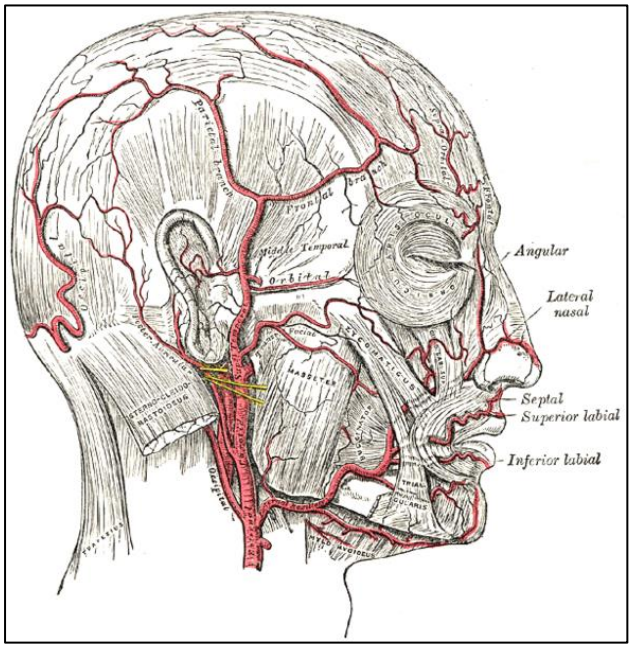

Figure 3.17 Diagram showing the complex blood supply to the cranial area which may be linked to scorbutic lesions. Reproduced from Gray's Anatomy.

common in children with scurvy; Lind also mentions a doctor who was working in the 1730's in the Russian Army, who stated the temporal muscle often swells in scurvy. Ortner (1997) in his work on juvenile scurvy argues that sphenoid lesions result from haemorrhageing caused by the mechanical trauma of mastication (Ortner 1997, 213). Therefore if there are defined, localised lesions of the skull, which are delineated by a soft tissue structure such as a muscle, then it may be possible to attribute these lesions to scurvy.

3.9.3.2 Postcranial Anatomy and Lesions

In the postcranial skeleton, the most commonly reported indicator of scurvy in archaeological populations is either ossified haematomas or periosteal reaction in the long bones (Geber et al. 2012; Van der Merwe et al. 2010, and Stirland 2000). Table 3.4 shows the anatomical explanation of postcranial lesions. The living bone reacts to both of these phenomenona in similar ways, with the production of new bone, and there is great debate as to whether one can distinguish between new bone caused by haemorrhagic or inflammatory processes (Mays *pers. comm*. 2012; Roberts and Manchester 2005; Aufderheide and Rodriguez Martin 1998). Either way new bone is a non-specific indicator of stress and in most cases cannot be attributed to a single cause. However, if the individual does not recover from scurvy and receives no Vitamin C then there will be no bony reaction (Mays et al. 2012). When scorbutic new bone is produced it is restricted by soft tissue. This is where anatomical recording as part of the scurvy recording form comes into use. The pathognomonic feature of scurvy in living individuals is bleeding and bleeding into muscles is common which often leads to muscle contraction and swelling.

Table 3.4 Showing Postcranial Scorbutic lesions by bone element, lesion type etc.

Postcranial Skeletal Location	Lesion Type	Anatomical Explanation
All long bones and the feet, hands and innominates	Periosteal new bone reaction and abnormal porosity	Porosity and new osseous reaction can be caused by inflammation and/or haemorrhageing.
Long Bones especially tibiae and femora	Ossified Haematomas	Ossified blood clots represent haemorrhaged blood that comes into contact with bone
Vertebrae	Compression Fractures	Caused by osteoporosis which can sometimes be attributed to scurvy

Muscle contracture is caused by blood leaking into muscles which leads to severe pain. However this blood is restricted in its passage and spread by the gross anatomy of the musculature. The blood is essentially 'trapped' by the muscle. Most importantly if this extravasated blood occurs in musculature adjacent to a bone the area of the bone affected is limited by the muscle that lies the deepest and the area/s of the bone that the muscle originates from/attaches to. James Lind (1772, 493) describes this process "the stiffness of the knee (in scurvy) may be occasioned by coagulated blood lying deep in the ham (hamstring), between the flexor tendons, or upon the tendon of biceps tibiae, frequently extending themselves to the belly of the gastrocnemius muscle". Ossified haematomas can be differentiated from inflammatory periosteal lesion through a number of points. The first is that ossified haematomas tend to be located on the anterior tibia, are well demarcated and are usually ovoid in shape. They are lumpy, thicker than periosteal reaction and are differentiated clearly from the cortical bone surface. Whereas periosteal reaction is not often ovoid in shape, is usually quite a thin, delicate layer and can occur nearly anywhere in the skeleton. Often differentiating between inflammatory reaction and ossified

haematomas can be difficult since both result from haemorrhagic processes. It is hoped that further research into adult scurvy can resolve this issue and it is possible to use histological methods in suitable cases.

It should always be remembered that scurvy is not a disease of the bone, but a soft tissue disease that can affect living bone. As part of the scurvy recording form and skeletal analysis, there will be an effort made to record any lesions that are specifically anatomical in nature. A lesion that is directly related to anatomy is defined as that reaction/lesion that is directly associated with a soft tissue anatomical structure.

3.9.4 Summary of Scorbutic Dry Bone Lesions

As can be seen from the archaeological literature, it is apparent that there has been an increase in the publication of adult scurvy in recent years. These studies have used a variety of different lesion types and methodologies to establish the diagnosis of scurvy.

The problem that all osteologists face is a lack of the presence of the soft tissue and anatomical structures that once surrounded the skeleton. This does not mean that we should forget the soft tissue and how it affects living human bone. In the case of most skeletal pathologies, they are in fact not directly related to bone. It is in majority of cases, a disease of the soft tissue that subsequently causes a bony lesion. When considering scurvy, this is particularly important as scurvy is not strictly a disease of the bone but it is known to cause bony lesions. This is one of the reasons why scurvy is not widely identified in the archaeological record unlike other bone diseases such as rickets or osteoporosis. The lesions of scurvy are for the most part non-specific as they could be caused by a number of disease processes, the aim of this research is to distinguish between these and lesions caused by

scurvy. The main way of doing this is by observing and recording the nature of the lesions present and whether or not these are associated with anatomical structures.

Chapter 4: Materials and Methods

4.1 Introduction to Materials and Methods

The aim of this research is to explore whether scurvy can be identified in Post Medieval English skeletal collections and to potentially establish a set of reliable indicators to identify scurvy in dry human bone. Previous chapters have concentrated on the palaeopathological literature review and the general historical context to the study sites. The first section of this chapter will provide a very specific background on the historical and archaeological context for each site. Relevant historical data will be discussed briefly and the findings of the archaeological excavations will be presented in detail. The four assemblages that will be examined are Royal Haslar Hospital, Plymouth Hospital, Darwen and Oxford Castle.

The main focus of this study is the examination of pathological lesions that occur in adult scurvy. However this research would not be complete without age at death and sex data for all skeletal assemblages that were studied. This section will establish the methodologies used to collate this data. This information provides us with a complete osteobiography for each individual that was analysed as part of this research. Preservation and completeness classification systems will also be given. A full methodology of how this data is gathered and recorded is presented in a later section of this chapter.

This chapter will also present the scurvy lesions that were selected from the literature review as being those that best represent the manifestation of adult scurvy in the skeleton. Each lesion is also represented by a scoring system that was established in order to determine the reliability of scorbutic lesions. Cranial and postcranial bones will be examined

separately and a scoring system produced which will be accompanied by physical description of the lesions and photographs.

4.2 Materials

4.2.1 Introduction to Materials

The assemblages that form the main body of this study are broadly dated from the mid eighteenth century to the early to mid-nineteenth century. The principal assemblages are those from the Naval Hospital cemeteries at Stonehouse Hospital, Plymouth and Haslar Hospital, Gosport. To supplement these, it was important to select comparative assemblages of a broadly similar date. The comparative assemblages originate from the burial ground associated with Oxford Castle Prison, Oxford and from the cemetery of a former Primitive Methodist church, Redearth, Darwen in Lancashire. From the examination of Table 4.1 it is possible to see that all four assemblages studied are broadly

Table 4.1 Showing the study groups, date ranges and the number of skeletons excavated.

Study Group	**Total Number of Adult Skeletons**	**Time Period**
Haslar Naval Hospital	45	1753-1826 AD
Stonehouse Naval Hospital	189	1761-1824 AD
Darwen, Redearth	61	1831-1862 AD
Oxford Castle	63	17-18th C AD
Total Number	**358**	**17-19th C AD**

contemporaneous and are therefore ideal for this comparative study. This table also shows the number of skeletons represented in each collection. All of these assemblages have been excavated in the past ten years and this research is the first instance of a large scale, geographically and culturally varied study specifically in the palaeopathology of scurvy in Post Medieval England. The archaeological excavation background for each site will be discussed in this chapter and some specific historical data relating to each site. The general historical background data has been presented in a previous section (see Chapter 3).

4.2.2 Oxford Castle, Oxford City, Oxfordshire.

Oxford Castle is located on the western side of the modern city of Oxford to the east of the river Thames and to the west of the river Cherwell and has been occupied since Anglo-Saxon times (Jope 1952, Poore et al. 2009). A keep was first constructed on the mound in the Anglo-Norman times when the site was in the control of Robert D'Oyley and the ditch and walls were all incorporated in a large defensive structure (Joy 1831). The site at Oxford Castle was first utilised as a gaol during the 1480s and one of the first recorded gaolers was John Crookston from 1486 to 1529 (Davies 2005, 3). From this period debtors and criminals were imprisoned at Oxford Castle tower along with individuals deemed mentally unstable (Davies 2005, 90). For the purpose of this research the emphasis will be placed on the post-medieval occupation of the site from the seventeenth through to the eighteenth centuries. From the 1750's onwards a lot of detail is known about the gaolers and offenders from the contemporary historical sources. These include John Howard's account of prisons in the eighteenth century*, Jackson's Oxford Journal* and other sources such as pamphlets and newspapers of the time (Boston and Webb 2012, 48). Prisoners facing serious charges were commonly kept in the castle for long periods of time as the assizes took place rarely. When

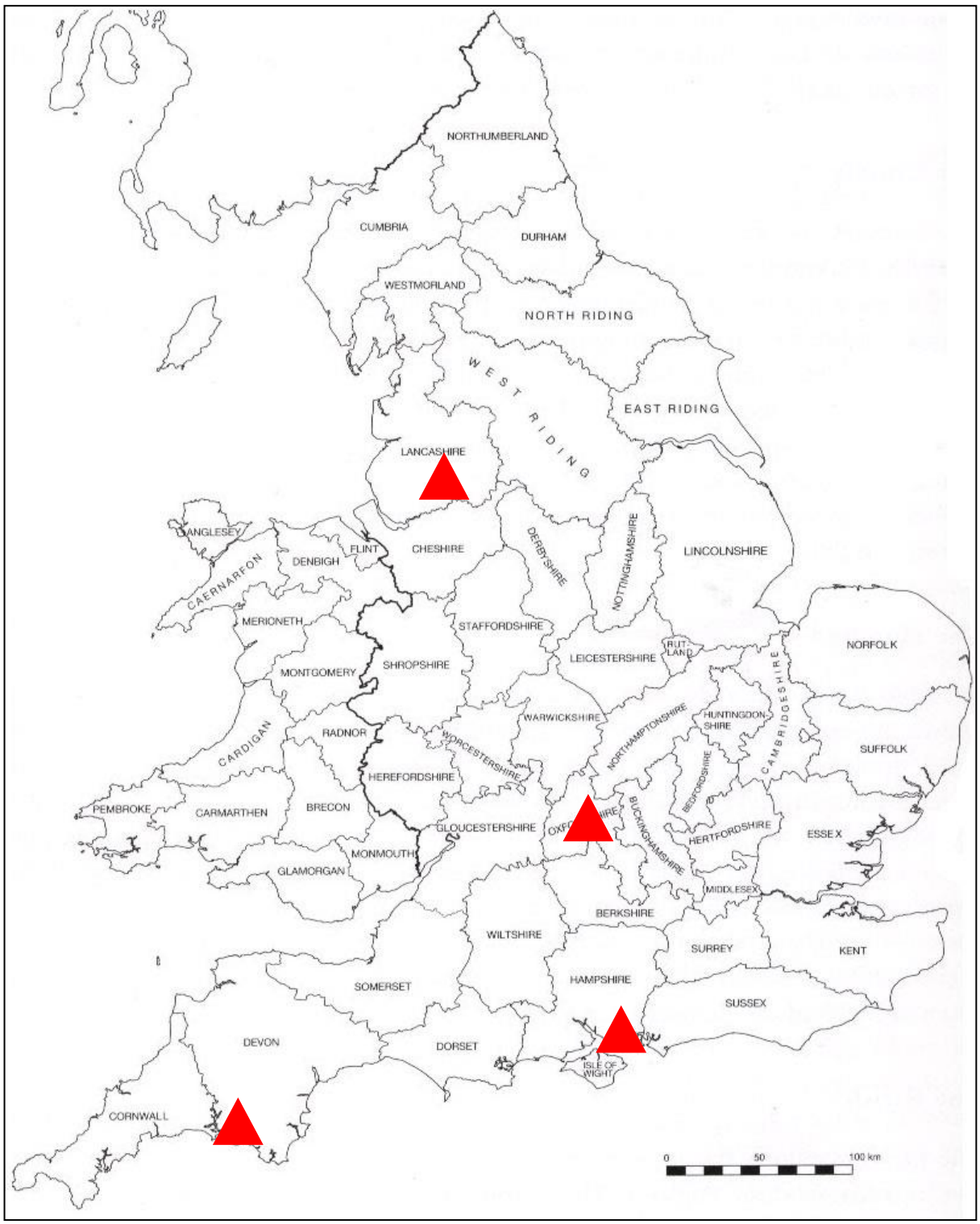

Figure 4.1 Map of England showing each study site marked by a red triangle

found guilty, many were executed or transported especially prior to 1776 when transportation to America was commonplace (Davies 2005).

One of the chief historians that refer frequently to Oxford Castle in the post medieval period was John Howard. In the 1777 edition of his book he recorded the numbers of prisoners and the prison conditions. Howard described the debtor's cells, women's cells and dungeons as being inadequate and too small for the amount of prisoners being held there. He described the courtyard as being "much too small... (however) the gaoler has a spacious garden" (Howard 1777, 315). Howard also stated that there was no straw on the floors of the cells, there was no infirmary and sanitary conditions there were quite poor. In 1773 alone eleven out of thirteen prisoners in the gaol died of smallpox, 'distemper' and 'gaol fever' (typhus) which reveals a high mortality rate of 85% (Howard 1777).There is also evidence for the burial of deceased prisoners in the grounds of the prison;

"Mr Wisdom (the Gaoler) told me that about nine years ago wanting to build a little hovel, and digging up the stones for the purpose, from the ruins of the court, which was formerly in the castle he found under them a complete skeleton with light chains on the legs; the links being very small. These were, probably, the bones of a Malefactor who died at the Court of Distemper at the Black Assize" Howard 1777, 317

This historical reference is confirmed by the archaeological evidence. The executioner's gallows were present at Oxford Castle and was commonly used as site for hanging criminals that were found guilty at the Assizes, it was the only place of execution in Oxford city after 1787(Davies 2005). It would seem from the historical documentation that criminals hung at Oxford castle were buried there in the infilled moat, or first anatomised in the local medical schools and then returned to the castle for burial (Boston and Webb 2012). Oxford Castle

site was redeveloped and added to right through the nineteenth and twentieth century, was used as a prison until modern times and was only closed in 1996.The site is currently the location of a luxury hotel.

4.2.3 Archaeological Excavations at Oxford Castle

Oxford Castle was first subject to archaeological excavations in the 1950s and more recently in 2002. In 1952 Jope excavated underneath a section of the castle mound and recorded the remains of Anglo-Saxon houses and late Saxon pits and pottery (Jope 1952, 85). Jope was largely uninterested in the post-medieval aspects of the site, but does record that the ditch was in-filled and that there were the remains of a seventeenth century road adjacent to the ditch (Jope 1952, 83). In 2002 Oxford Archaeology undertook rescue excavations in the area of the Oxford Castle mound and ditch prior to redevelopment of the site. During this excavation the remains of 62 individuals were excavated and were subject to full osteological analysis. It is believed that these remains constitute those of criminals that were executed at the Castle. The skeletons were buried in the moat ditch that was backfilled in the post medieval period. The graves were quite shallow with the deepest being 0.6m deep, in some cases the bodies were thrown carelessly on the moat and just covered with soil (Poore et al. 2009), there was no indication that coffins were used, but small pins were found, which may represent the remains of burial shrouds. There is some evidence to suggest that the individuals were hanged, some had their hands tightly placed together over the stomach suggesting binding of the hands and many of the hands were tightly clenched which can happen during hanging. The date of the skeletons is based on pottery typology from the contexts containing the skeletons. It is thought that the majority of the skeletons probably date to before the introduction of the Murder Act in 1752 which

made the dissection of criminals compulsory (Poore et al. 2009) and meant that the remains would have been retained and not buried back at Oxford Castle.

Figure 4.2 Photograph of four Oxford Castle skeletons in-situ during excavation. Courtesy of Oxford Archaeology.

In general these skeletons were very well preserved but many were incomplete which is shown by the high rate of skeletons that cannot be either aged or sexed due to lacking either pelvis or skull. High levels of truncation and re-use of space at this site are to blame for high levels of incomplete skeletons. These skeletons were analysed for this thesis in April and May 2012. The majority of these skeletons were young males (See Table 5.9) which fits in well with John Howard's description of the typical convicts.

"Convicts are generally stout, robust, young men who have been accustomed to free diet, tolerable lodgings and vigorous exercise...these are ironed and thrust into close offensive

dungeons some of them without straw or other bedding in which they continue in winter fifteen or sixteen hours out of twenty four in utter inactivity" Howard 1792, 467.

All of the historical evidence from the prison documents would indicate that in general the health of prisoners was very poor, sanitation was non-existent and disease was commonplace. It likely that many of the prisoners kept at Oxford Castle would have suffered from nutritional deficiencies. The skeletal assemblage from Oxford Castle gives us an excellent opportunity to assess the occurrence of scurvy in a typical post medieval prison population.

4.2.4 Redearth Primitive Methodist Cemetery, Darwen, Lancashire

Redearth Primitive Methodist cemetery is located on the Redearth Road in the town of Darwen in Lancashire in the North of England. The area of Redearth is located to the south of the centre of Darwen. Darwen is a large market town and was well known in the 1800s as being a major textile manufacturing centre. Cotton processing was the primary industry in the area until the period of the Lancashire Cotton Famine in the 1860s. Figure 4.3 shows the 'India Mill' constructed in Darwen at the height of the cotton-spinning boom.

Primitive Methodism began to gain a foothold in this area in the late 1820s and a preacher named John Verity was one of the first to give sermons in Redearth (Wright and Colling 1908). The first Primitive Methodist church was built in Redearth in 1832, prior to this open air preaching was commonplace (Ritson 1909, 191). At this time the church site was located in the rural countryside and was extremely representative of the character of the Primitive Methodist church. The church itself was a co-operative project and was built by voluntary

workers and controlled by a board of Trustees who were all working class individuals (Oxford Archaeology Interim Report 2011). It would seem that the congregation of the

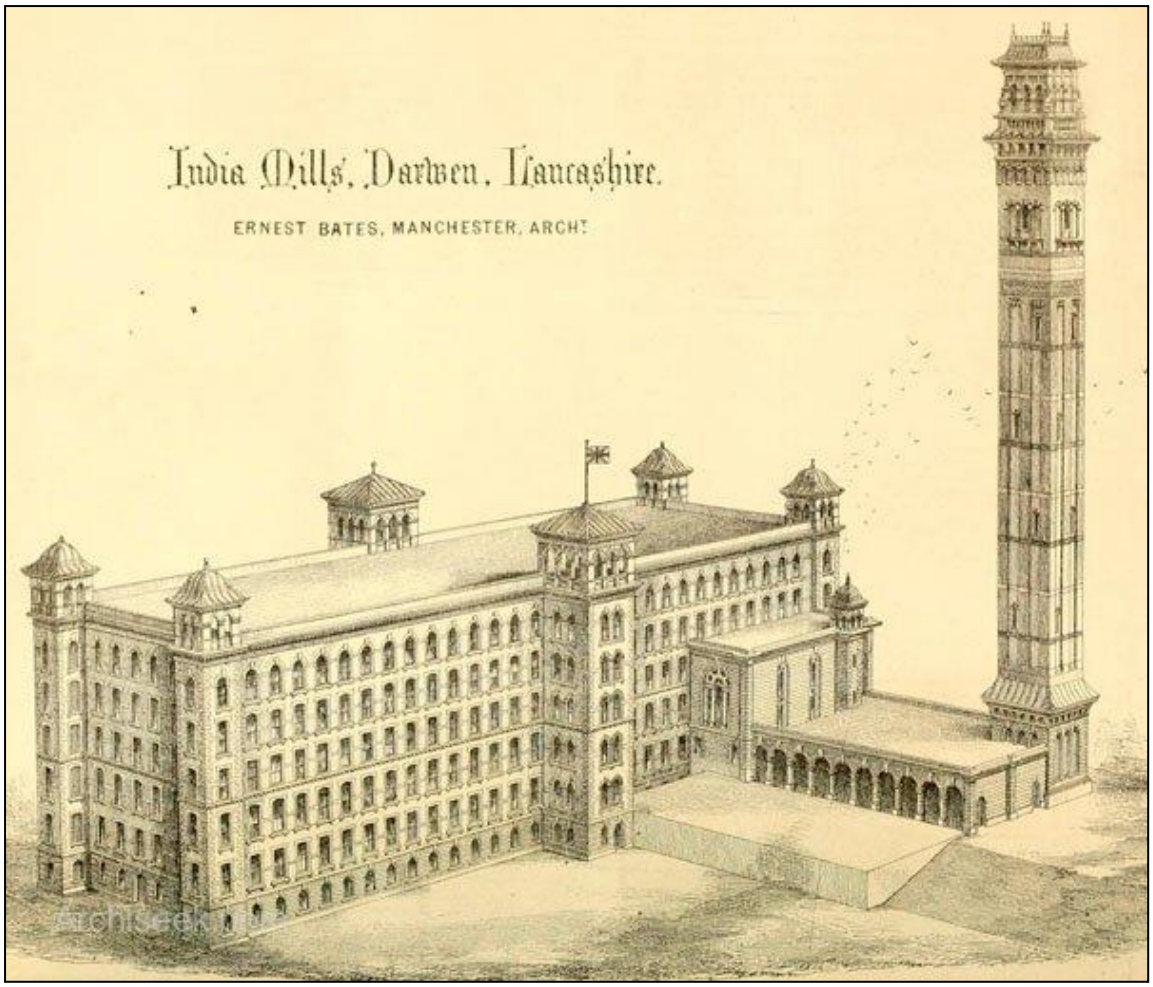

Figure 4.3 Image of the India Mill constructed in Darwen. Image reproduced from www.archiseek.com.

Redearth Primitive Methodist church was a very poor labouring class and the church registers indicate that the primary occupations were cotton spinners and weavers, miners, labourers and carters (Oxford Archaeology interim report 2011).

4.2.5 Archaeological Excavations at Redearth, Darwen.

The site on Redearth Road was commercially redeveloped in 2006 and it was then that the remains of the former Primitive Methodist chapel and burial ground were revealed. An

initial watching brief with some limited excavations was carried out by Gifford Ltd and then a full scale excavation conducted by Oxford Archaeology followed. In the course of these excavations the foundations of the original church was located along with the adjacent burial ground, in total 142 individuals were excavated. Of 133 grave cuts, 111 of these provided evidence of coffins and in some cases breastplates, grips and fabric were preserved (Oxford Archaeology Interim Report 2011). The death registers indicate that there was a very high infant mortality rate, 60% of all children died before the age of 10 (Darwen Local Historical Society 1987, 10). In total sixty-one adult skeletons from the Darwen assemblage were assessed for age, sex, pathology and for the macroscopic signs of scurvy. A significant number of the skeletons were very poorly preserved and therefore a high number of individuals could not be easily assigned an age or sex, Oxford Archaeology indicated that this pattern of poor preservation was due to the poor soil conditions and high water table and flow of water through the site (Oxford Archaeology Interim Report 2011). The Redearth Assemblage covers a time period of approximately 1832 to 1861 which is significant as the cemetery is still in use for the first year of the Lancashire Cotton Famine which lasted from 1861-1865, when widespread malnutrition and food shortages were present in Lancashire and would have been in use throughout the potato famine of 1845-1849 which is known to have affected Northern England and this population were also known to be quite poor (Oxford Archaeology Interim Report 2011). Of the one hundred and forty two skeletons excavated, eighty-one individuals were excluded from the scurvy analysis as the remains represented those of infants and sub-adults under the age of eighteen which do not form part of this study.

4.2.6 Royal Naval Hospital Haslar, Gosport, Hampshire and its Burial Ground.

Haslar Naval Hospital is located in Gosport, Hampshire which is very close to Portsmouth which was a key Naval base throughout the post-medieval period. Royal Haslar was the first purpose built clinical Naval hospital built at a home base by the Admiralty, the history of Haslar has been extensively discussed in Chapter 2. However what is of importance here is the burial of the dead at Haslar. The dead seamen and marines that died within Haslar hospital were buried in the adjacent burial ground which was commonly referred to as the Paddock. Hospital workers such as washerwomen may also have been buried in the Paddock (Tait 1906). The Hospital Admission Musters (Admiralty Records held at the National Archives, Kew) also indicate that many men were brought onshore 'a corpse' and were subsequently interred in the Paddock. The Paddock is a direct source of valuable data about the life, infirmities and death of the seamen in the service of the Royal Navy of this period.

The Paddock was the unconsecrated burial ground that was used for dead Naval personnel that were transferred to the hospital for burial and for those seamen and marines that died in the hospital. Tait (1908, 69) states that "the whole land to the south-west of the hospital including the enclosed ground now known as the paddock and the old cemetery, as well as the ground on which the Terrace stands, was used discriminately as a burial ground , in the early days of the hospital". Tait also affirms that any excavation in the area of the Terrace exposes human remains. The Paddock began its use in 1753, the same year that the hospital opened and it was used right up till 1826 when a new consecrated cemetery took over. This consecrated cemetery is now known as the Garden of Remembrance and is located in the north eastern section of the Paddock. The Paddock itself consists of the area to the south west of the hospital. Between 1756 and 1765 approximately 5045 men died in Haslar and

were buried in the Paddock (Death of Seamen Register ADM102/374- National Archives). Unfortunately, there are no other surviving burial registers for the Paddock but the Hospital Musters record if individuals were interred elsewhere. It would seem from these burial registers that the high ranking Naval officers were generally not buried in the Paddock but were instead buried elsewhere at their family's expense. The rest of the burials were paid for by the Royal Navy and/or friends or shipmates of the deceased. At the end of each month the burial costs were detailed, for example in June 1794 there were 59 funerals at a cost of £21 7s 9d (ADM 102/274).

4.2.7 Excavations in the Paddock

There are numerous accounts of human bone being found during works in the grounds of Haslar hospital stretching as far back as the construction of the Terrace in 1798 (Oxford Archaeology 2005). In 2005, Oxford Archaeology was commissioned to undertake archaeological evaluations in the Paddock for the Ministry of Defence in order to determine the extent of the burial ground prior to the site being sold. During the course of this evaluation nineteen trenches were opened and human remains were uncovered. In each trench that human remains were uncovered, a sample of between 1-3 skeletons was recorded in-situ. By and large the excavations by Oxford Archaeology showed that the area of the Paddock had a dense distribution of burials (Oxford Archaeology Report 2005).In 2007, Cranfield University began a series of research excavations at Haslar that lasted till 2010, all of the trenches opened during this time can be seen in Figure 4.4. During the course of these excavations 47 skeletons were lifted and these were all subject to full osteological analysis. The four seasons of excavations provided valuable information on the

burial practises at Haslar including evidence for wooden coffins from the remains of pine wood and iron nails recovered from the grave cuts. Out of the forty-seven skeletons

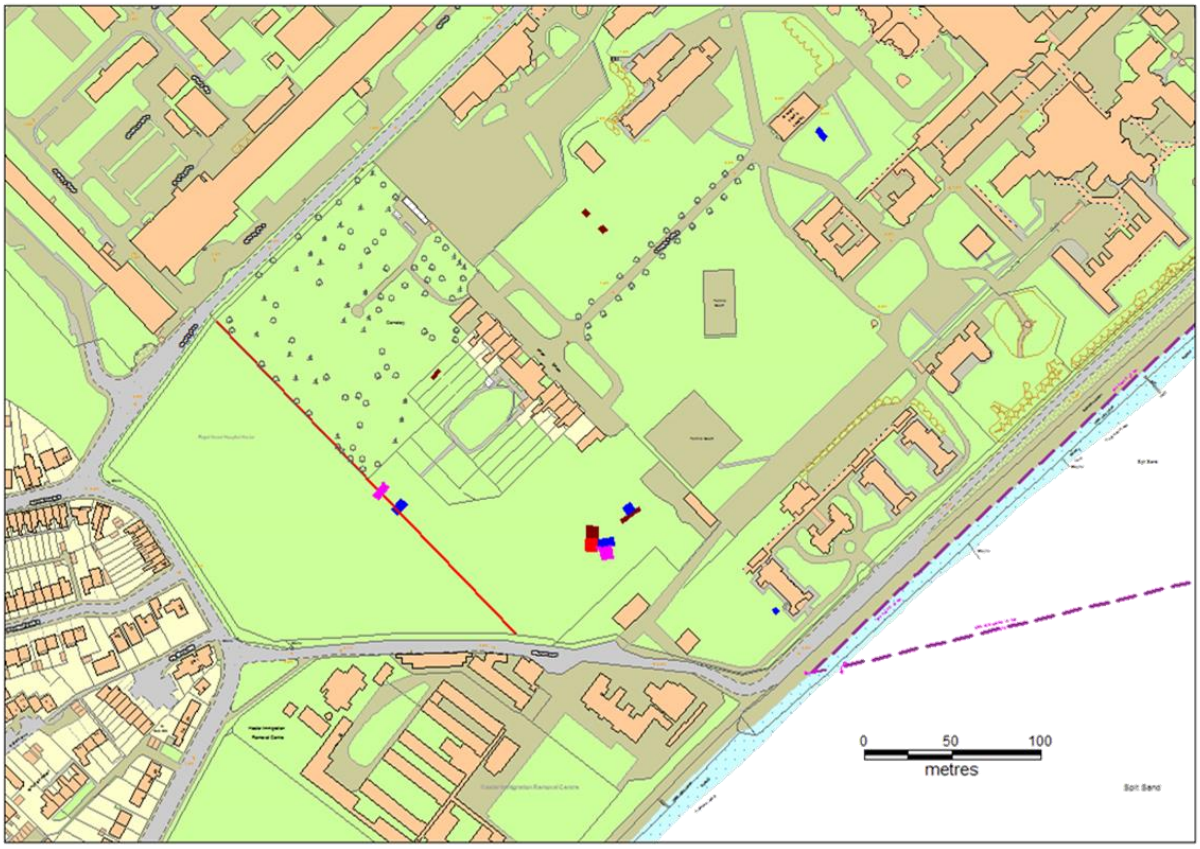

Figure 4.4 Showing the trenches dug in the Paddock by Cranfield University 2007-2010. The maroon areas were excavated in 2007, the red trenches in 2008,the blue in 2009 and the pink in 2010. The red line marks the boundary of the Paddock. Courtesy of Mr. Peter Masters.

lifted, there was evidence for a wide variety of pathologies and for post-mortem anatomisation. The central part of the Paddock provided evidence for very dense burials but no evidence for intercutting or multiple burials. In this area all burials were aligned on a north-west south-east orientation. The Haslar assemblage provides us with a valuable opportunity to assess scorbutic lesions in a collection of skeletons that are securely dated and provenanced from a unique group of people that lived in a period where scurvy was endemic and their occupation predisposed them to this disease.

4.2.8 Stonehouse Hospital Burial Ground, Plymouth

Stonehouse Hospital, Plymouth was the second Naval hospital to be built at a home base and the history of the site has been discussed extensively in Chapter 3. When the Commissioners for the Sick and Hurt purchased the land at Stonehouse they also acquired an area known as 'No Place Field'. In the period 1760 to 1826 an area of land known as 'Stray Park' was used as a unconsecrated burial ground for those that died in the hospital which was then replaced by 'No Place Field' which was consecrated in 1826 by the Bishop of Exeter (Reese 1962). Stray Park was not an official Naval burial ground and Thomas Trotter the eminent Naval physician criticised this fact in his *Medica Nautica*; "amidst the deficiencies of Plymouth hospital a contiguous burial ground is much wanted. The present one is a bit of waste ground belonging to the corporation of Plymouth , where the seamen have such a dislike to be laid, that on dying, if they have any money as will defray the expense of being carried to a churchyard, they will leave a Will to that effect" (Trotter 1803).

4.2.9 Excavations at Stray Park, Plymouth

In 2007, part of the 'Stray Park' cemetery was excavated prior to a proposed development. This area is now known as the Crescent on Notte Street and was the site of the former Crescent Car Showroom. The cemetery was excavated between April and August 2007 by Exeter Archaeology and human remains were recovered. In total approximately one hundred and ninety two skeletons were excavated and a significant amount of disarticulated bone was also recovered. Figure 4.5 shows the excavation of an amputee seaman from the Stray Park cemetery. The area that was excavated was quite densely utilised for burials, there were up to three burials interred in each cut and there was also significant evidence for intercutting. At least some of the burials were coffined, but by no means were all coffined and it would seem that the graves were all unmarked (Exeter

Archaeology 2008). The small finds recovered during the excavation confirm the known dates of the cemetery being used from 1760-1824. The small finds include sherds of English

Figure 4.5 Image of skeleton being excavated Stray Park burial ground. Courtesy of Exeter Archaeology.

stoneware 1720-1750 which were thought to be re-deposited when the graves were dug and also a coin dating to the time of King George III (1770-1797) which was also recovered and provides some solid dating evidence for the use of the graveyard and fits well with what is known from the documentary sources (Exeter Archaeology 2008).

4.3 Historical Analysis of Scurvy Rates.

There are a number of primary historical sources that were utilised to provide an indicator of scurvy rates in the Royal Navy throughout the time that both the Paddock at Haslar and Stray Park at Plymouth were in use (1753-1826). These include the medical treatises and dissertations written by Naval doctors, the Surgeons Logs from on-board Royal Naval ships and the Admission Records relating to the Naval Hospitals. This research is particularly concerned with the rates of scurvy before and after the Admiralty introduced lemon juice rations in 1795.

4.3.1 Scurvy Rates in the Royal Naval Hospital Admission Musters.

Since the establishment of the Naval hospitals at Haslar and Plymouth records were taken of all the men that entered the hospital. These records included the name of each man admitted, his ship, date of entry into the hospital, illness and date of death or discharge back to their ship. These records are now kept at the National Records Office at Kew, London. Unfortunately the records from the early years at both Plymouth and Haslar do not survive. The surviving records begin in the year 1792 and continue through until the end of the use of the cemeteries at Haslar and Plymouth. The Paddock at Haslar and Stray Park at Stonehouse ceased to be used in 1826. What is particularly useful about these records is that they cover the period when the Admiralty introduced lemon juice rations to cure the scurvy that was so endemic within the Navy in 1795 (Tröhler 2005, 522). The records themselves detail the hospital admissions on a monthly basis and at the end of each month collate all the individuals that were received, discharged, died and those that remain in the hospital from the previous month. These musters are an invaluable source of information about all disease rates but of most relevance to this study are the scurvy rates in the hospitals. In the musters, scurvy is recorded in several different ways: the most common is

'scurvy', followed in varying amounts by 'scorbutic', 'scorbutic ulcers' , 'scorbutic eruptions', 'scorbutic flux', 'scorbutic leg' or 'scorbutic arm', and 'scorbutus'. Scurvy is also frequently recorded with other diseases like rheumatism, ulcers and fluxes and fevers. From

Table 4.2 Rates of Scurvy recorded at Haslar and Plymouth hospitals.

Haslar			**Plymouth**		
Year	**Total Admitted**	**Total with Scurvy as a Percentage of Total Admitted**	**Year**	**Total Admitted**	**Total with Scurvy as a Percentage of Total Admitted**
1792	2319	21(0.9%)	**1792**	1614	46(2.8%)
1793	11887	206(1.7%)	**1793**	10514	141(1.3%)
1794	20171	292(1.4%)	**1794**	7795	126(1.6%)
1795	17846	711(4%)	**1795**	7093	1040(14.7%)
1796	14869	182(1.2%)	**1796**	5596	174(3.1%)
1797	17305	166(1%)	**1797**	10320	280(2.7%)
1798	11141	39(0.4%)	**1798**	12560	83(0.7%)
1799	10154	34(0.3%)	**1799**	11249	63(0.6%)
1800	10285	27(0.3%)	**1800**	12454	95(0.8%)
1804	4007	22(0.5%)	**1804**	8801	78 (0.9%)

1792 up till 1800 every muster for both hospitals was analysed and every case of scurvy was noted with the Excel spread sheets detailing all months attached in Appendix D. Table 4.2 shows the combined figures for both Haslar and Plymouth from 1792 until 1800 and also for 1804. This period is particularly important as it covers the period of time immediately prior to and after the introduction of lemon juice rations in 1795. A special case study was also made of 1804, as by this time lemon juice rationing should have been enshrined in the mind-set of Royal Naval surgeons and the Admiralty. Table 4.2 shows the rates of scurvy as

a percentage of the overall number of men admitted for each year. At the beginning of record taking in 1792, scurvy rates were low and accounted for only 0.9% of admissions at Haslar and 2.8% at Plymouth. At its height in 1795, before the introduction of lemon juice rations, scurvy accounted for 4% of all overall Haslar admissions and 14.7% of all Plymouth admissions. This decreased to 1.2% at Haslar and 3.1% at Plymouth in 1796. It would seem

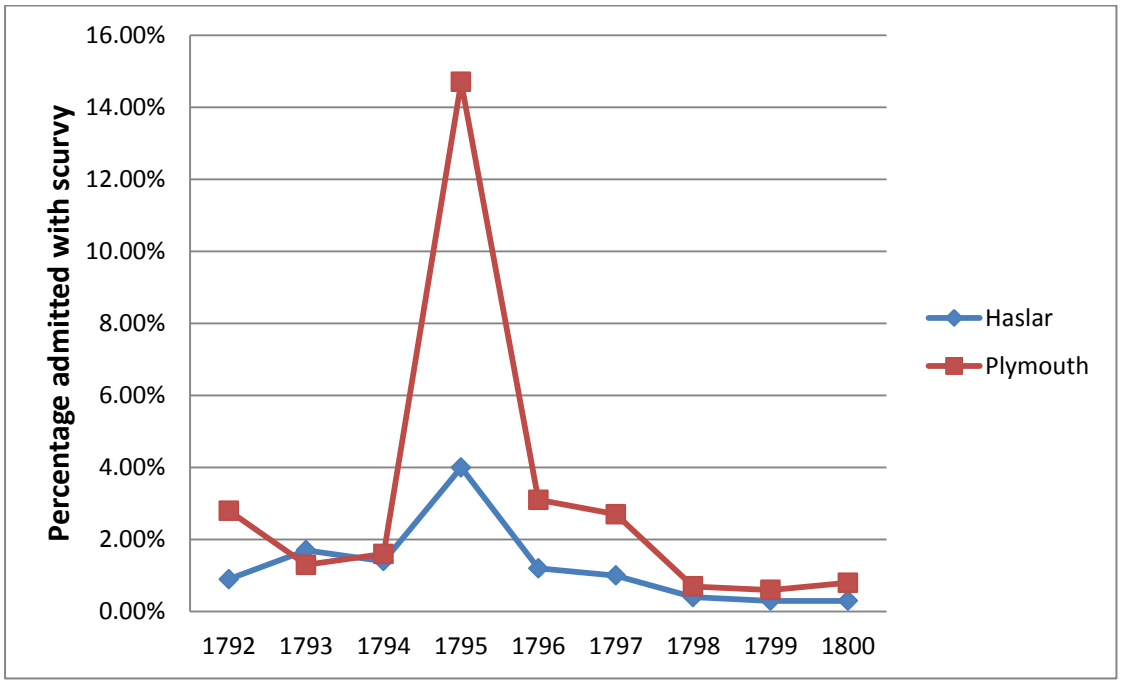

Figure 4.6 Scurvy numbers by year at Haslar and Plymouth hospital from 1792-1800.

by examining Table 4.2 that after 1798 scurvy does become scarcer in the hospital admissions, decreasing to only 0.3% at Haslar in 1800 and 0.8% at Plymouth. At Plymouth the average yearly figure is slightly lower than Haslar but the overall scurvy figure from 1792-1800 is quite high at 1,861 scurvy cases compared to 1,678 scurvy cases at Haslar which was the larger of the two hospitals. One of the primary aims of analysing the hospital musters is to ascertain if there is a discernible difference between the period before and after the introduction of lemon juice rations by the Admiralty in 1795 and the reason for the apparent scurvy epidemic in 1795. The second aim is to see if there is a difference in scurvy

rates between Haslar and Plymouth. The graph in Figure 4.6 clearly illustrates the peak in scurvy rates at Haslar and Plymouth in 1795 and the subsequent decrease in rates until 1800. If the year 1795 is taken as a whole, it can be seen that in total 1040 men were admitted into Plymouth Hospital with scurvy compared to 711 men admitted with scurvy to Haslar. The outbreak of scurvy in 1795 might be attributed to a heightening of hostilities in the wars of the French Republic (MacDonald 2004). This appears to have caused a disruption in lemon juice supplies to the Royal Navy in 1796, it is likely that that this disruption began in 1795 and could be associated with the large outbreaks of scurvy seen at Haslar and Plymouth. Prior to 1796 most of the Navy's supply of lemons came from Spain, but because of the war, the supply was switched to Lisbon, this supply was however extremely unreliable and unpredictable (Henderson-Smith 1918).

What is particularly interesting is that peaks in scurvy in Haslar hospital seem to occur in the summertime, with the highest numbers occurring in May which is unexpected as most authors have suggested that scurvy is a disease that most commonly occurs in the winter due to a lack of fruit and vegetables (Stark 2009). A similar pattern is visible at Stonehouse,

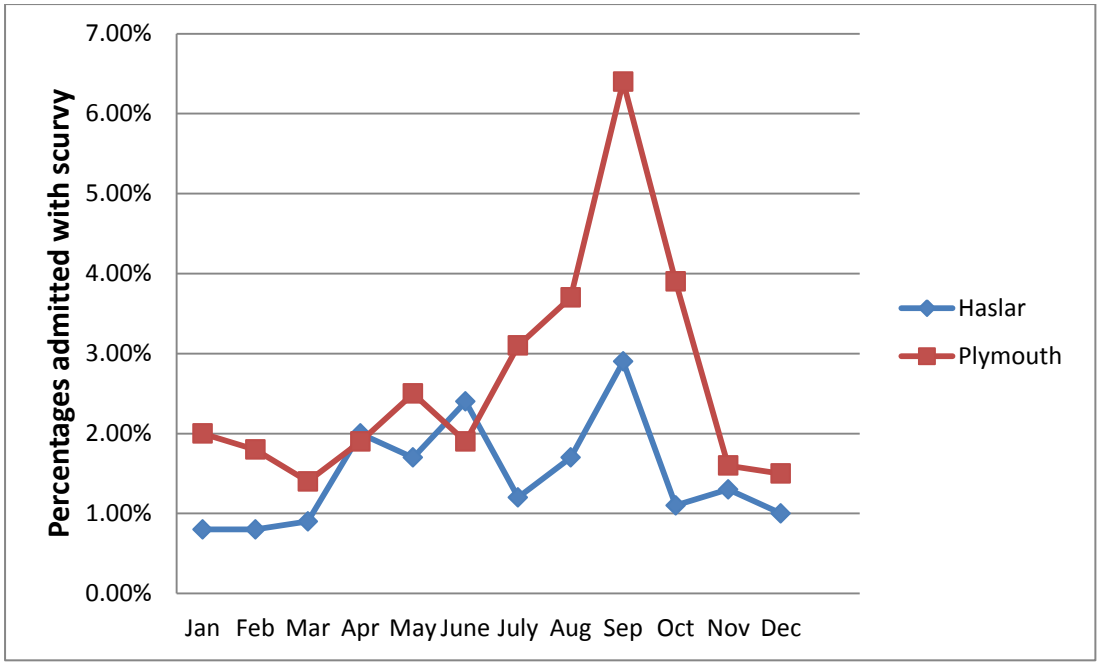

Figure 4.7 Monthly percentages of scurvy admissions at Haslar and Plymouth from 1792 to 1800.

Plymouth, however in Plymouth the highest scurvy intakes are in September. For example, if the scurvy admission figures in 1795 are examined, 40% of all men admitted to Stonehouse in September had scurvy, at Haslar there was a peak in April where nearly 23% of all admissions were scurvy. These percentages are based on data that is presented in Appendix D. The monthly pattern from 1792 to 1800 can be seen in Figure 4.7, it is clear from this table that the lowest rates of scurvy are occurring in the winter months and the highest throughout the early summer through until November. This is a fascinating pattern and it is unclear why this is so. However it is not known how long these men would have had scurvy before they were admitted to the Naval hospitals. From examination of Figure 4.7, it is also plain to see that scurvy was more common at Plymouth hospital throughout the period examined. This is particularly interesting as Blane (1799) stipulates that Plymouth was by far the healthier hospital during this period. It would also seem that even after the introduction of lemon juice rations that scurvy rates at Plymouth were higher than Haslar. The year 1804 can be taken as a case study, there were just 22 cases of scurvy in Haslar during this time, in

Plymouth there was 78 cases (ADM 102/614-617), this can be seen in Table 4.2. In 1804, the unusual seasonal pattern of scurvy was again noted. The highest rates of scurvy in both Plymouth and Haslar occurred in the summertime. It is difficult to ascertain why scurvy was more common at Plymouth. It had a significantly smaller capacity and was in general much quieter in terms of admission rates in the 1790s and 1800s. If one looks at the entire number of men that were admitted to both hospitals in the period 1794 to 1795, there were a total of 38,000 men admitted to Haslar where only 7,000 were admitted to Plymouth. Considering the massive difference in admission numbers there is only a small difference in mortality rates, at Haslar 2.56% of those admitted died, at Plymouth it was 2.15%. What must be said however is that very few men actually died just from scurvy after being admitted to the hospitals in the 1790's. Even in Stonehouse at the height of the scurvy epidemic only 1-2 men a month are recorded as having actually died from scurvy (ADM 102: 601-610). This is may be an underestimation of the mortality of scurvy and the reasons for this are complex. The first being that just because an individual was admitted for another disease did not mean they didn't have scurvy, secondarily many individuals developed scurvy after suffering from the flux and other debilitating infections and trauma (Lind 1772).

These musters provide a wonderful source of primary data from the two primary Naval hospitals used throughout the Georgian period. It is possible to directly compare the two hospitals in terms of scurvy and mortality rates on a yearly and monthly basis. These records are also an excellent way to assess the success of the introduction of lemon juice rations. What should also be mentioned is that we do not have the logs to indicate scurvy rates prior to 1792. James Lind does indicate that scurvy was particularly bad in the late 1750's and 1760's, he states that "I have frequently visited during the five years of the late war with

France (1758-1763), three or four hundred scorbutic patients a day" (Lind 1772, Aiii). It is not known if the 1792-1795 rates are representative of pre 1792 rates, therefore they must be interpreted with caution.

4.3.2 Surgeon's Logs of the Royal Naval Ships

The surgeon's log was a written record that gave a general account of the health of the ships company whilst at sea which included the daily sick lists and any medical or surgical interventions that were undertaken. The logs were kept by the ships surgeon and often include fascinating observations on various matters from weather patterns, to disease patterns, statistics and cures. These logs are also an excellent source of data about scurvy rates at sea. The logs are also an interesting contrast to the hospital admission records and were returned to Physician General of the Navy (Cock and Rogers 2008, 155). These logs were made compulsory by Blane who wanted to receive monthly returns of disease in the fleets and it is this information that he analyses in his book *Observations on the Diseases Incident to Seamen* (Blane 1782). After 1796, there are still significant outbreaks of scurvy in the squadrons at sea, however these cases were for the most part cured by the surgeon at sea without the need to discharge the individual to a hospital. Scurvy is still a common occurrence on-board Royal Navy ships but, the provision of lemon juice rations on-board cures it quickly when it does occur. For example, when looking at Nelson's squadron in the Mediterranean in 1803 and specifically at the ships Gibraltar and *Triumph (*ADM 102-52/3616). The logs have been examined and it found that on *Triumph* in early 1804 that there were 37 cases of scurvy (MacDonald 2004 163). However Nelson himself ordered special lemon juice rations and the scurvy soon cleared up. When *Gibraltar* moored at the Maddalena Islands on the 6th of August 1803, where she sent 135 scurvy cases on-shore

(McDonald 2004, 164). Fresh provisions were obtained for the ship and scurvy was not recorded by the surgeon during the remainder of her voyage. It was not just these specific ships that were affected by scurvy outbreaks, but the whole Mediterranean fleet during 1803 and 1804 because the war had caused a disruption in the fresh food supplies from Spain. Sicilian lemons had been used by the Navy since 1796 since an earlier disruption in the Lisbon lemon supply and Nelson sent his personal physician, Dr Snipe, to Sicily to secure 50,000 gallons of lemon juice for the use of the Navy (Henderson-Smith 1918, Crimmin 1997).

The author has made a study of scurvy rates on the *Vanguard* that was patrolling the West Indies from 1802 to 1804. The surgeon on board was William Cather and he made extensive notes in his log regarding scurvy. Cather gave those suffering from scurvy a mixture of lime juice and bark along with some onions. When *Vanguard* arrived into Halifax in 1802 there were 113 men suffering from scurvy on-board. The *Vanguard* was a third rate ship of the line which means it would have had more than 500 men, but less than 720 men on board (Rogers 2004). Another fascinating case study is that of *Albion* which was employed in the East Indies in 1806 and 1807. The surgeon on board was Andrew Elphinstone and the ship suffered a massive outbreak of scurvy in January 1807 which reached 154 cases of scurvy out of a full crew of 590 men. The surgeon ordered a large amount of soil be brought onto the ship, an earth bath was constructed and the scorbutic men buried in this up to their necks. Elphinstone also records using sulphuric acid on thirty-one men, nitric acid on eight men and lime juice on fourteen men to treat scurvy (ADM 102/82/3). What this does show is that surgeons as late as 1807 are not relying solely on lemon and lime juice to cure scurvy and that scurvy outbreaks at sea are still commonplace. It is curious to think that years

earlier Lind and Blane were extolling the use of citrus fruits in the cure of scurvy and that well into the 1800's they were not universally accepted, with other unproven and unsuccessful methods still being used. It is this that particularly emphasises the gulf between modern and Georgian naval medicine (MacDonald 2004).

On initial inspection of the historical records and books written subsequently, it would appear that scurvy in the navy disappeared after 1796. In the naval hospitals for the most part this is true but it is a very different situation at sea, scurvy is still occurring in the squadrons well into the 1800's. From the case studies presented it is also apparent that lemon juice was still not being used exclusively as a cure for scurvy and the naval surgeons were using alternative 'cures' like sulphuric acid. When the ships logs are examined, there were no regular receipts for the delivery of lemon juice to ships and certainly not enough to provide standard rations that the Admiralty recommended (MacDonald 2004, 165). MacDonald attributes this to the Admiralty policy that lemon juice should be used at the discretion of each ships surgeon and that many surgeons did not use lemon as a curative therapy rather than a preventative. Many surgeons of the fleet were extremely conservative and continued with the use of traditional anti-scorbutics and were unwilling to accept the efficacy of lemon juice. It would seem that scurvy cases didn't progress to such a severe stage that they had to be transported to home base hospitals like Haslar or Stonehouse, Plymouth. However it is also true that scorbutic men may have been transported to hospitals at foreign bases where they were cured and later discharged back to service. It also seems that the majority of these scorbutic individuals are recovering quickly from their illness due to more efficient victualling and better healthcare.

4.3.3. Summary of Historical Scurvy

It is clear from the historical records that scurvy was a problem in the Naval hospitals in the years leading up to 1795 and it was then that Gilbert Blane persuaded the Admiralty to take action and introduce lemon juice rationing to the entire Royal Naval fleet. Despite the evidence indicating that lemon juice rationing was not accepted wholesale, there is still evidence that it reduced the rate of scurvy at both Haslar and Plymouth to near insignificant levels. It almost certainly saved many lives and led to most cases of scurvy being cured at sea. However despite the known efficacy of lemon juice rations, not all surgeons were convinced by its effectiveness and still resorted to 'quack' remedies. At no stage did the Admiralty force naval surgeons to carry lemon juice rations, it was at their own discretion. This can be seen as a lack of faith in the overall worth of lemon juice and the Admiralty's lack of power over the everyday actions of the surgeons of the fleet. It is because of this lack of absolute action that scurvy continued to occur at sea and to diminish the overall well-being and health of the Royal Naval fleet.

4.4 Osteological Methodology.

This and following sections will present all methodologies for the analysis of the study assemblages. The primary method of analysis utilised on the dry human bone was macroscopic analysis, which was carried out on all adult skeletons. Good bone surface preservation was also an important criterion, if there was excessive trabecular bone exposure then the remains were excluded from analysis. Only skeletons that were 25% or more complete with both cranial and postcranial remains present were analysed. Only adult skeletons (aged over 17/18) were analysed as this study is concentrating only on adult scurvy. Every skeleton was analysed using a general osteological and skeletal inventory

recording form adapted from Buikstra and Ubelaker (1996) which was employed to record age, sex, height, preservation, completeness and pathological conditions. They were then analysed using a specific scurvy recording form which will be discussed later. The Oxford Castle and Darwen assemblages were both analysed in the human remains laboratory in the Burials Department of Oxford Archaeology, the Haslar assemblage was analysed at Cranfield University Shrivenham and the Plymouth collection was studied at the Institute of Archaeology in Archaeology Department of Oxford University.

4.4.1 Recording Completeness and Preservation

For each skeleton both completeness and preservation was recorded on the full osteological recording form. A visual recording form was used to grade the completeness of each skeleton, this is included in the osteological recording form in Appendix B. Each bone present was shaded in, this form was also useful for recording fragmentation. Completeness was also evaluated on a four point scale, which were 5-25%, 26-50%, 51-75% and then 76%- 100%. Surface preservation was assessed with a three point grading system which is shown in Table 4.3 (MOLA Human Osteology Statement 2008, 9).

Table 4.3 Grading System used for assessing bone preservation. Adapted from the Museum of London Osteology Method Statement 2008, 9.

Grade	Description
1-Good	Bone surface in good condition with no erosion; fine surface detail such as coarse woven bone deposition would (if present) be clearly visible to the naked eye
2-Moderate	Bone surface in moderate condition; some post-mortem erosion on long

	bone shafts. Erosion of articular surfaces and some prominences
3- Poor	Bone surface in poor condition; extensive post-mortem erosion resulting in pitted cortical surfaces; articular surfaces missing or severely eroded

4.4.2 Ageing Adult Individuals

Adult age was established by the presence of a full dentition and an age of over 17-18 years of age from ageing techniques. Adult ages were determined through a combination of macroscopic methods. There are many methods of ageing available to the osteologist, however some are more reliable than others. This is because there is a difference between biological and chronological age, this disparity can be caused by many factors including lifestyle (Schwartz 1995, 185). Different ageing methods were used for young and mature adults. Older adolescents and young adults can also be aged using epiphyseal fusion, which was used where appropriate. The primary ageing methods that were chosen were assessment of the degeneration of the pubic symphysis (Suchey and Brooks 1986) and auricular surface (Buckberry and Chamberlain 2002). These were chosen as they are the most reliable methods when used in British populations (Molleson and Cox 1993). When the pelvis was not present, sternal rib ageing (Işcan 1984; Işcan and Loth 1985) was used as it is known to be useful for European populations (Lovejoy et al. 1985). Where possible all ageing methods were used, as this increases accuracy of the age range obtained. Table 4.4 shows the age at death categories that were used for this research (Boston et al. 2008). Please note that whilst an adolescent age category is used, only individuals over eighteen years of age were included in this research, individuals below this age were excluded.

Table 4.4 Age-at-death categories that were used in this research. Adapted from Boston et al. 2008, 32.

Skeletal Age at Death	Age Range
Adolescent	13-18 years of age
Young Adult	19-25 years of age
Middle Adult	26-35 years of age
Mature Adult	36-45 years of age
Older Adult	45+ years of age
Adult	Age undetermined

4.4.3 Sex Estimation in Adult Skeletal Remains

In this study, an effort was made to establish sex for all adult individuals studied. Sexually dimorphic features in the cranium and pelvis along with sexually dimorphic measurements in the postcranial skeleton were all used. Previous osteological studies have proved that the pelvis is the best indicator of sex in adult remains (Sutherland and Suchey 1991), followed by the skull (Mays 1998). It is thought however that a combination of methods will provide the most accurate sex estimation. Cranial and pelvic morphologies adapted from Buikstra and Ubelaker (1994) were used as standard. When the results of these methods were taken together, one of five sex categories was attained. These five categories are:

- Definite Male
- Possible Male

- Definite Female
- Possible Female
- Sex indeterminate

These categories were attained by using a combination of both metric and morphological sexing methods.

4.5 Methodology for Macroscopic Analysis of Scorbutic Lesions

All of the skeletons that met the criteria for general osteological analysis were also macroscopically analysed for scorbutic lesions. These lesions were recorded on a specially developed scurvy recording form, itself generated from a comprehensive review of possible scorbutic lesions compiled using both archaeological and medical literature. The aim of this macroscopic analysis was to identify, record and quantify any lesions that may be scorbutic in nature. All pathological lesions that could be caused by scurvy were recorded on a present/absent basis and in the subsequent sections each lesion will be examined in detail and classification systems will be presented. The lesions recorded were those visible to the naked eye without the use of magnifying lens. The scurvy recording form is divided into cranial and postcranial lesions and the categorisation of lesions will also be presented in this fashion.

There has been significant work done by Ortner and his colleagues (1997, 2001, and 2004) in order to establish a suite of lesions in subadult remains that can be attributed to scurvy. Adult skeletal remain pose a much bigger challenge than subadults as the pathological changes are much more subtle, but there are some rather obvious overlaps as it is the same pathological process. Therefore, the collection of lesions proposed here is a combination of the Geber et al. (2012), Ortner et al. suite (1997, 2001, 2004) and lesions that have been

adapted from the limited palaeopathological evidence for adult scurvy and the medical literature. It is important to remember that all of the lesions being discussed result from a haemorrhagic or inflammatory disease process that is so characteristic of scurvy (Carpenter 1987; Fain 2005). Lesions such as Schmorl's Nodes and degenerative disc disease, which were discussed in Chapter 2, were also excluded as they are only indirectly related to scurvy. It is also crucial to be able to distinguish what is pathological from what is normal. In the following sections, each pathological lesion will be presented and these will be divided into cranial and postcranial categories.

4.5.1 Scorbutic Cranial Lesion Identification and Classification

Cranial lesions are central to the positive diagnosis of scurvy. The only lesions that can be considered to the pathognomonic of infantile scurvy are those that occur in the cranial region (Ortner 1997). These lesions are those that can be considered to be directly caused by scorbutic haemorrhageing, particularly that in the muscles of mastication. Many cranial lesions are identified as they manifest through abnormal porosity. Throughout this research abnormal porosity has been defined as "a localised, abnormal condition in which fine holes, visible without magnification, but typically less than 1mm in diameter penetrate a lamellar bone surface" (Ortner and Ericksen 1997, 212). An important point to consider when macroscopically analysing the skull is that any porosity observed must be consistent. Scorbutic lesions are physiologically predisposed to be uniform, regular, patterned and to occur bilaterally (Stark 2009). Bilateral pattering is also essential when considering differential diagnoses. This is because in clinical case studies and medical reports, scorbutic haemorrhages occur in a specific pattern which has been discussed in Section 3. Trauma and other pathological processes do not tend to produce bilateral lesions which are useful to

distinguish these from scurvy. What is intrinsic to this study is the creation of a new category of lesion, which is an 'anatomically linked' lesion. The hypothesis behind this research is that scurvy is a distinctive disease process that affects soft tissue and the musculoskeletal system in a systemic and predictable pattern and therefore should produce lesions that are specific in their nature. This has been touched on by Ortner (1997) by his work on the muscles of mastication but this work aims to establish a similar physiological aetiology and classification for all scorbutic cranial lesions.

4.5.1.1 Ante-Mortem Tooth Loss

Ante-mortem tooth loss was scored using the tooth recording scheme established by Buikstra and Ubelaker (1994). This is important to distinguish ante-mortem tooth loss from other types of tooth loss, for example teeth lost during excavation. The 1-8 scale was used for all skeletons with alveolar bone present.

1. Present, but not in occlusion
2. Present, development completed, in occlusion
3. Missing, with no associated alveolar bone
4. Missing with alveolus resorbing or fully resorbed: pre-mortem loss
5. Missing, with no alveolar resorption: post-mortem loss
6. Missing: congenital absence
7. Present, damage renders measurement impossible, but other observations are recorded
8. Present , but unobservable (e.g. deciduous or permanent tooth in crypt)

Pre or Ante mortem tooth loss (AMTL) is the loss of teeth during life; it is of particular importance as tooth loss in life can be caused by scurvy (Hess 1920; Lind 1777; Popovich et al. 2009). AMTL can be caused by a number of factors including poor dental health,

periodontal disease, carious lesions, peri-apical abscesses, excessive calculus, trauma and scurvy (Roberts and Manchester 2005). Tooth loss has long been associated with scurvy in the historical documentation, this is due to the laxity of the periodontal ligament when the human body is in a scorbutic state. This leads to loosening of the teeth and their subsequent loss (Hess 1920, 89). Tooth loss associated with scurvy has been commonly recorded in medical cases of scurvy (Fain 2005; Olmedo et al. 2006; Velandia et al. 2008). In the vast majority of cases, AMTL cannot be directly be related to scurvy however if it is associated with new periosteal bone on the alveolar bone adjacent to the tooth socket then this may be related to gum haemorrhages. This only occurred in cases where the tooth loss was relatively recent. Where this occurred, it was recorded as being a scorbutic anatomically linked lesion.

4.5.1.2 Periodontal Disease

For all individuals with teeth present and in occlusion, periodontal disease was also recorded. Periodontal disease is defined as inflammation of the periodontal tissues which can include the periodontal ligament, the gums and the alveolar bone (Hillson 2005). Periodontal disease is a group of diseases which can be caused by a number of genetic, environmental, dietary and oral hygiene factors including calculus deposits, levels of oral bacteria and diseases like scurvy (Hillson 1996).This study is concerned mainly with periodontitis which is an inflammation of the periosteum in the area of the tooth sockets which is caused by a number of factors, one of which can be bleeding caused by scurvy. It is well known that the oral symptoms are among the earliest manifestation of scurvy (Firth et al. 2001) and that gingivitis is a characteristic symptom of scurvy (Fain 2005). Lind is particularly resolved in his knowledge that the gum symptoms are a clear indicator of

scurvy, he mentions these numerous times, he re-iterates that in scorbutic individuals, the gums are livid, red, spongy, putrid and often ulcerated. He also states that petechiae are often present on the gums when there are no petechiae elsewhere (Lind 1772, 242). In medical case studies, these scorbutic changes have been known to cause periodontal disease which is visible radiographically (Firth et al. 2001; Halligan et al. 2005; Charbeneau et al. 1983; Pimental 2003).

However it is difficult differentiating between periodontal disease caused by bleeding, poor oral hygiene or age related factors. Periodontal disease was recorded for each tooth on a 0-4 scale devised by Ogden (2008) which differentiates between healthy (1) and unhealthy (4) alveolar bone based on simple visual assessment and is detailed here:

0. Unable to score
1. Tooth meets alveolar margin at acute knife edged margin
2. Alveolar margin is blunt and flat topped with slightly raised rim
3. Alveolar margin is rounded, porous, with a trough of 2-4mm depth between tooth and alveolus
4. Alveolar margin is ragged and porous with an irregular trough or funnel >5mm between tooth and alveolus.

Occasionally new periosteal reaction of the alveolar bone associated with periodontal disease was recorded, this was recorded as being a scorbutic anatomically linked lesion.

4.5.1.3 Cribra Orbitalia and other lesions of the orbit.

Cribra Orbitalia is defined as pores, sieve-like pores or outgrowth in the bone of the orbital roof and has long been associated with anaemia (Roberts and Manchester 2005; Wapler

2004) and the factors that can cause it (Stuart-MacAdam 1985, 1987; Fairgrieve and Molto 2000). This lesion is thought to occur due to the expansion of the diploë of the skull, due to the increased need for red blood cells in anaemia (Roberts and Manchester 2005, 229). It has also been attributed scurvy by some authors (Schlutz 2001; Geber et al. 2012), this is because the two diseases often occur concurrently in the same individual (Cohen et al. 2001). Vitamin C is necessary to absorb iron in the human body, this is discussed further in section 2.1.6.

In the course of this study, plaques of periosteal bone were recorded in the eye-sockets. This lesion cannot be attributed to cribra orbitalia, as there is no outgrowth, porosity and usually no other evidence for anaemia. It is possible that such lesions could be caused by haemorrhaging into the bony orbits caused by scurvy (Ortner et al. 2011, 202). Hess (1920), states that subperiosteal haemorrhage of the orbital plate of the frontal bone occasionally occurs in severe scurvy and often causes exophthalmos and bruising of the eyelids (Hess 1920, 84). Bruising of the eyelids is something that Lind (1772) records also "not unusually on one or both eyelids…which appears swelled and of a deep red colour (Lind 1772, 425). Scurvy is a well recorded consequence of infantile scurvy (Dunnington 1931; Parsons and Smallwood 1935; Richardson 1948) and scurvy has long been considered a differential diagnosis in the cause of exophthalmos and ocular haemorrhage (Blake 1921). Exophthalmos caused by ocular haemorrhage was found to have occurred in 49 of 379 (12.9%) reported cases of infantile scurvy collated by the American Paediatric Society in 1898 (Blake 1921, 308). In adults there are some references to this occurance, Palmer (1963) reported an older man with scurvy who presented with ocular haemorrhages caused by the trauma of sneezing (Palmer 1963, 693). Hodges and Hood (1969) in the Iowa prison

scurvy study stated that ocular lesions appear early, between 74-95 days on a Vitamin C free diet and manifest in the form of haemorrhages. Hirschmann and Raugi also suggest ocular changes are not un-common in adult scurvy (Hirschmann and Raugi 1999, 902).

There are a handful of bioarchaeological studies that suggest that bony orbit pathology may be caused by scurvy (Mogle and Zias 1995; Brown and Ortner 2011; Geber et al. 2012; Mays 2008). It is clear that ocular haemorrhagic lesions are not uncommon in adults with scurvy and it is possible that these lesions may manifest as bony reaction in the eye socket. There has been significant research conducted into cribra orbitalia and the most commonly used classification system is that of Stuart-Macadam (1991), which categorises on a 0-5 scale and is detailed below;

0. Normal bone surface
1. Capillary-like impressions on the bone
2. Scattered fine foramina
3. Large and small isolated foramina
4. Foramina have linked into a trabecular structure
5. Outgrowth in trabecular form from outer table surface

On the scurvy recording form, cribra orbitalia was recorded using the Stuart-Macadam system and whether it occurred unilaterally or bilaterally. All individuals with observable eye-sockets were visually assessed for any pathological changes of the eye-socket. Stuart

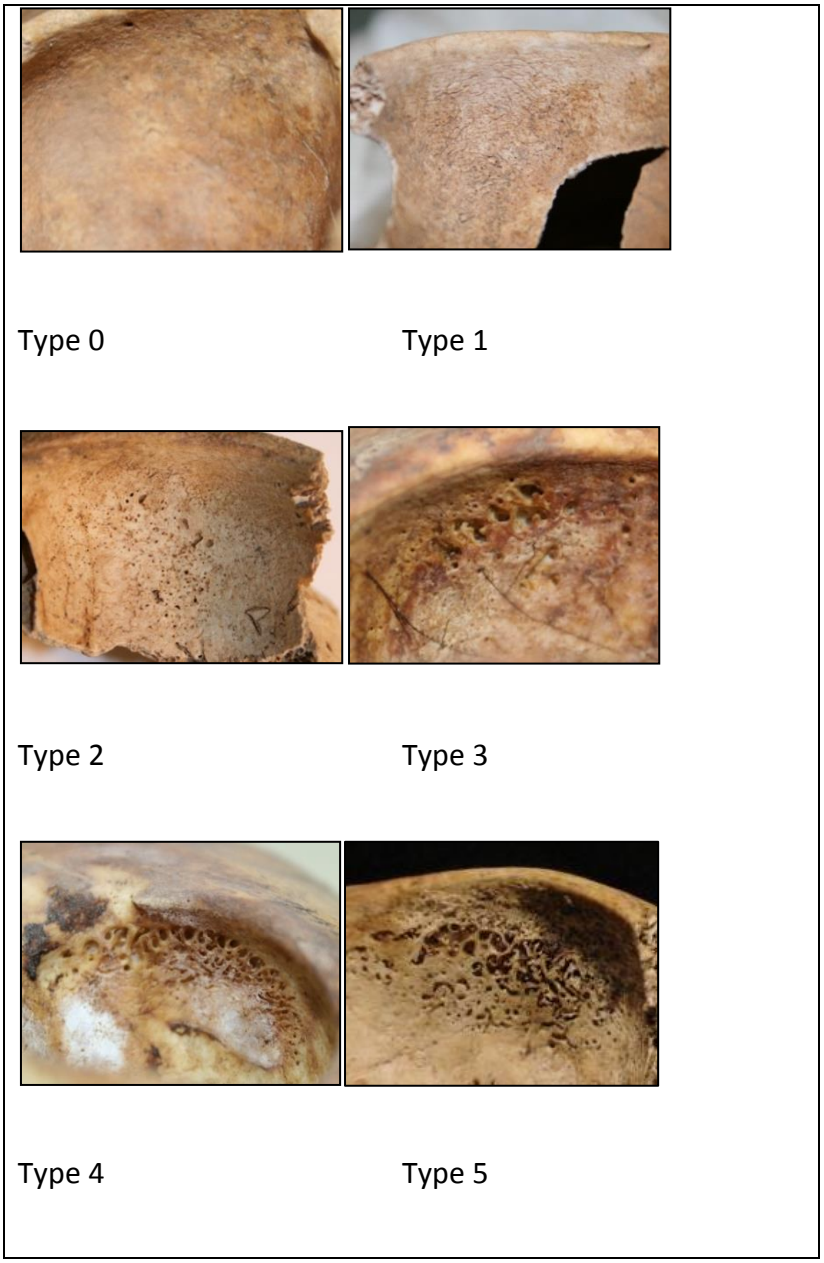

Figure 4.8 Classification system for cribra orbitalia. All photographs taken by author except Type 5, which is reproduced from http://archive.museumoflondon.org.uk/

Macadam's classification system is displayed in Figure 4.8 which shows the pathological /changes that described. Occasionally lesions not covered by Stuart-Macadam's classification were identified. These lesions frequently consisted of plaques of periosteal bone which were usually observed in the orbital roof. These lesions are recorded under the heading of cribra orbitalia, however it is likely that they have a very different aetiology to cribra. It is thought that these plaques of bone may result from haemorrhaging in the area

of the eye socket. It was these 'plaque like' lesions that were recorded as potentially being scorbutic in nature and were recorded as anatomical lesions. All orbital lesions were recorded the form reproduced in Table 4.5 and were fully described in a comments section along with a diagram or sketch where necessary.

Table 4.5 Recording form for orbital lesions.

	Left	Right
Code		
Additional Notes		

4.5.1.4 Ectocranial skull changes

The external skull or ectocranial skull may be subject to a number of pathologies, the best known of which is porotic hyperostosis (PH). It is an abnormal porosity of the external skull that is often found in association with cribra orbitalia especially in sub-adult skeletons (Roberts and Manchester 2005). However this was not commonly recorded in this study. Instead a little reported ectocranial lesion was recorded. This lesion has been referred to as cranial vault porosity by Geber (2012) and Stirland (2005) and as 'orange peel' by Boston et

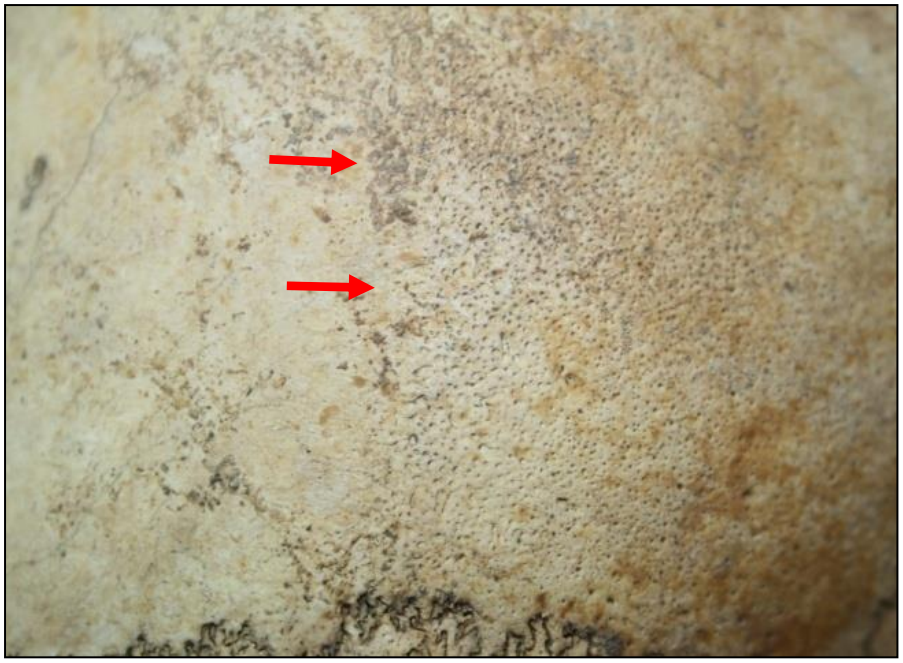

Figure 4.9 Ectocranial porosity on the skull.

al (2008, 57) and can be seen in Figure 4.9. It can be characterised by a lumpy 'orange peel' like porosity which is often rough to feel and shiny in appearance and is localised by the lines of attachment of the cranial aponeurosis and is totally visually distinct from PH. It was recorded on a present /absent basis on the scurvy recording form and its location on the skull. Figure 4.10 shows a distinct contrast above the temporal line and in line with edge of the cranial aponeurosis. The arrows point out a boundary which separates distinct thickened 'orange peel' porosity on one side compared to the normal bone surface on the left.

It is easy to differentiate between P.H and 'orange peel' lesions. With porotic hyperostosis one would expect a thickening of the diploë and a subsequent distinct porosity of the external skull, along with thinning of the cortical bone and exposure of the trabeculae due

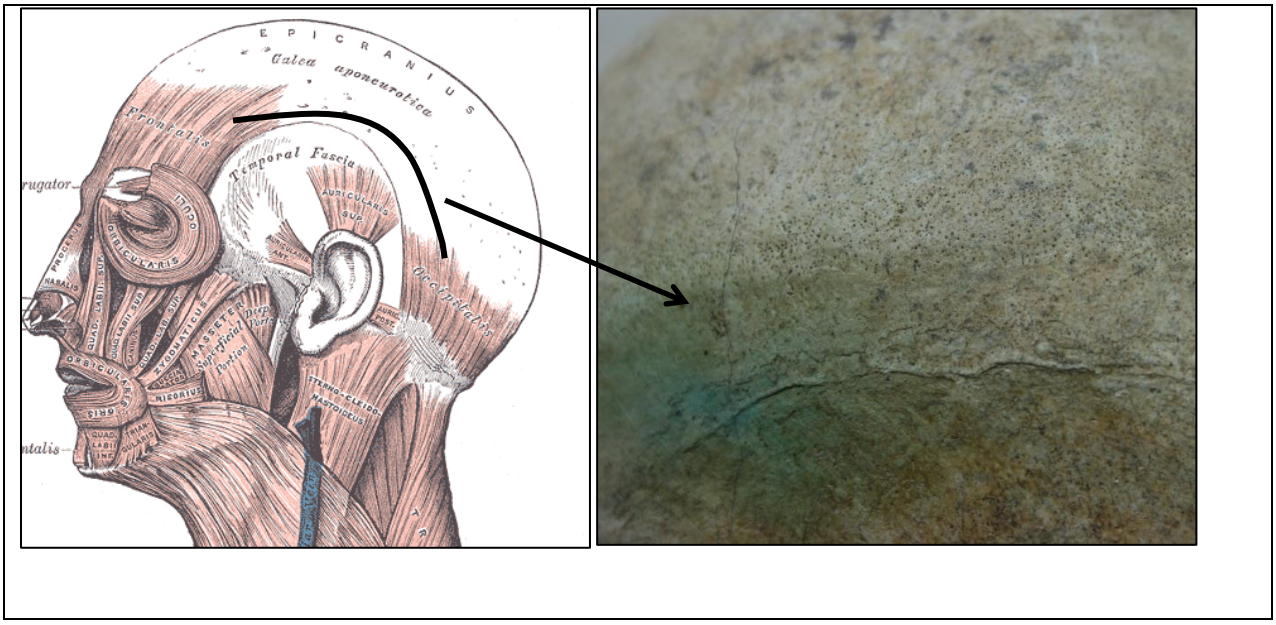

Figure 4.10 A- anatomical diagram showing the galea aponeurotica, adapted from Gray's Anatomy. B- Ectocranial lesion limited in its extent by the area of the galea aponeurotica.

to higher haematopoietic need (Keenleyside and Panayotava 2006, 373; Vercellotti et al. 2009, 358; Stuart- Macadam 1985, 1987). These features are not present with ectocranial lesions in this study, which are thickened, very porous and 'orange peel' like in their appearance (Stirland 2005). There is also no evidence for expansion of the diploë, another distinctive feature of these lesions is that their occurrence is restricted to the area of both parietal bones, medial to the temporal lines and would seem to be defined by the epicranial aponeurosis and is external to the temporal fascia. This can be seen clearly in Figure 4.10 A and B, the line of bony reaction stops above the temporal line where the temporal fascia begins. These 'orange peel' lesions may often be lumpy and rough and appear as though a layer of bone has been laid down on top of the external skull cortex. This is even visible macroscopically where the parietals are broken post-mortem.

Assuming that these 'orange peel' lesions are different to traditional porotic hyperostosis, what can cause these lesions? After literature review references were found to sub-aponeurotic hematomas and scalpal edema associated with scurvy in children (Ahuja and

Karande 2002; Lowenburg and Shields 1937; Scott 1936). James Lind in his Treatise (1772) references a surgeons letter from 1762, which states that the swelling of scurvy sometimes spread to the head "the disease most commonly began with a soft swelling of the legs…extending…up to the breast and…even to the head" (Lind 1772, 278). Hess (1920, 95, 36) also states that sub periosteal haemorrhaging of the cranium and scalp was not unknown in adults and that scorbutic headaches were common. A sub-aponeurotic haemorrhage or edema could certainly cause lesions on the external skull that are restricted by the area of the aponeurosis. Diseases such as syphilis can cause ectocranial lesions but these are markedly different form the typical gummatous lesions associated with syphilis. These lesions are unique in that they cover the whole parietals and therefore could not be caused by typical trauma patterns. In the bioarchaeological literature, some authors have suggested that ectocranial porosity may be caused by a minor scalp infection (Jennings 2010), however it is unlikely that scalp infections would affect such a high proportion of these adult skeletal assemblages. Tinea capitis is one the most common fungal scalpal infection and this is prevalent in children, but is uncommon in adults (Barlow 1988). However such lesions could be caused bacterial scalp infections and it is not known how common these would have been in the past. Shang and Trinkaus (2008), in a case study suggest that ectocranial lesions may be caused by subaponeurotic haematomas caused by trauma such as hair pulling to the scalp, which could cause periostitis in this region which is limited to the area covered by aponeurosis (Shang and Trinkaus 2008, 434) In a Nubian case study, Wood-Jones (1910, 285-286) attributes skull periostitis to trauma caused by carrying water jars on the head, which is quite a reasonable explanation. Stirland (2005) suggests the porosity of the external skill may be indicative of scurvy (Stirland 2005, 97), however she does not suggest an aetiology. It is most likely that these lesions are caused by scurvy but

exacerbated by minor trauma. It is well known that in the Royal Navy that hair was of great importance to seamen, their hair was often very tightly tied into a pigtail or queues with a ribbon (Haythornthwaite 2012, 28). In scorbutic individuals it is possible that the minor trauma to the scalp incurred during hair styling would have been enough to cause scalp haemorrhage.

4.5.1.5 Sphenoid

There are several anatomical regions in the cranial region that can display localised porosity that may be associated with scurvy. Ortner and Erickson (1997) consider the sphenoid to be the key anatomical location for porosity, see section 4.4.2 for definition of abnormal porosity. Porosity and new bone formation can occur on the greater wing of the sphenoid, the lesser wings, pterygoid plates and fossae and the sphenoid body (Geber et al. 2012; Ortner and Ericksen 1997; Crist et al. 2005). It is thought that these lesions occur bilaterally in scurvy cases (Ortner 1999, 325). The arrow in Figure 4.11A is pointing to a distinct area of

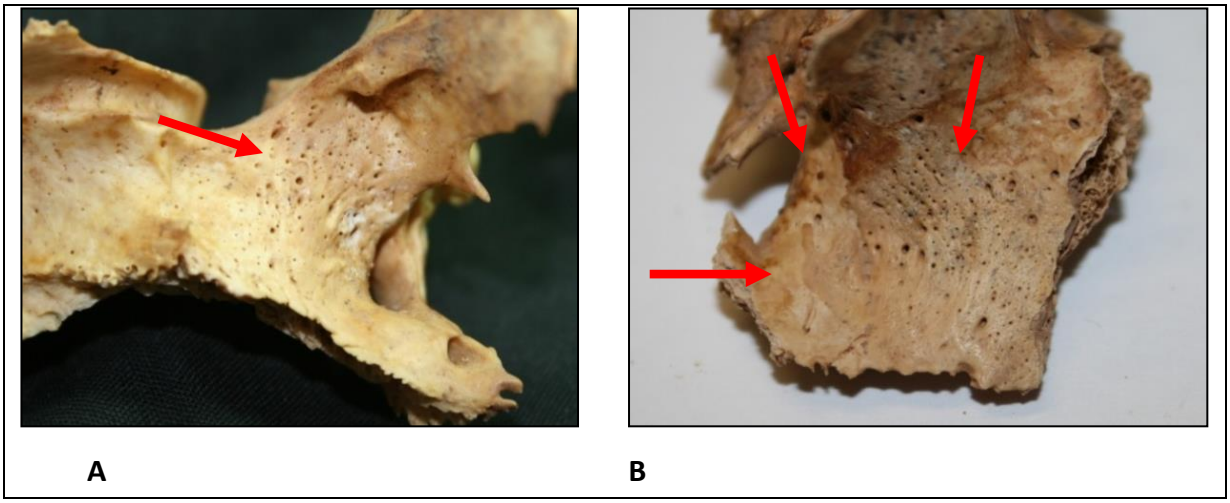

Figure 4.11A+B A-abnormal porosity of sphenoid, B-plaque of bone on sphenoid.

fine porosity on the body of the sphenoid. Figure 4.11B shows a plaque of new porous bone which is on top of the original bone cortex adjacent to the area of the foramen ovale. Lesions on the sphenoid were recorded on a present/absent basis, the anatomical area where the lesions occur and whether these were of a porous type or a reactive new bone formation. It is suggestive that these lesions may be indicative of scurvy considering sphenoid anatomy and the bilateral nature of such lesions (Ortner and Ericksen 1997, 212). This bone is the area for attachment for some of the muscles of mastication such as the pterygoids and it is a bone through which several arteries pass. This has for several years been recognised as a key area in the diagnosis of infantile scurvy due to the potential for scorbutic haemorrhaging in the temporalis musculature in the area due to mastication (Ortner and Erickson 1997; Brickley and Ives 2008; Ortner et al. 2001). There are a small suite of lesions occurring in the sphenoid that have been attributed to scurvy, the include porosity or reactive bone occurring on the greater wings, lesser wings, and the foramina. The foramen rotundum is the foramen that is most often affected, see Figure 4.12. Scorbutic lesions in the area of the sphenoid are not something that is found in the medical or historical data. In the historical data there are some references to swelling of the temporalis muscle which overlies the sphenoid bone. James Lind references two other medical writers who both observed swelling of the temporalis muscle in scurvy sufferers, in his chapter discussing scurvy in the Russian army, he states that “sometimes the temporal muscle is swelled and hardened under the zygomatic process” (Lind 1772, 427). This is very likely to cause sphenoid lesions, along with haemorrhaging from the blood vessels passing through the sphenoidal foramina and the attachment site of the pterygoid muscles on the

Figure 4.12 Image of sphenoid with new bone in area of the foramen rotundum.

pterygoid plate which has been attributed to scurvy in archaeological cases of infantile scurvy (Brown and Ortner 2001). There is no other known differential diagnosis for sphenoidal lesions, except perhaps traumatic damage to the sphenoid and the associated musculature, however this is highly unlikely to have affected so many individuals.

4.5.1.6 Endocranial Lesions

Endocranial lesions are those that occur on the internal surface of the human skull. Endocranial lesions have in recent times received more attention in the palaeopathological literature (Lewis 2004; Geber et al. 2012; Schultz 2001; Buckley 2000) and numerous possible causes have been put forward including meningitis, tuberculosis, trauma and metabolic diseases such as scurvy. Endocranial lesions have attracted little attention in the study of adult and infantile scurvy but they have received a mention from Buckley (2000) and Geber et al. (2012) as a possible indicator of scurvy in subadults and occasionally in

adults. Lewis (2004) does also consider scurvy to be a possible cause for endocranial reaction in subadults.

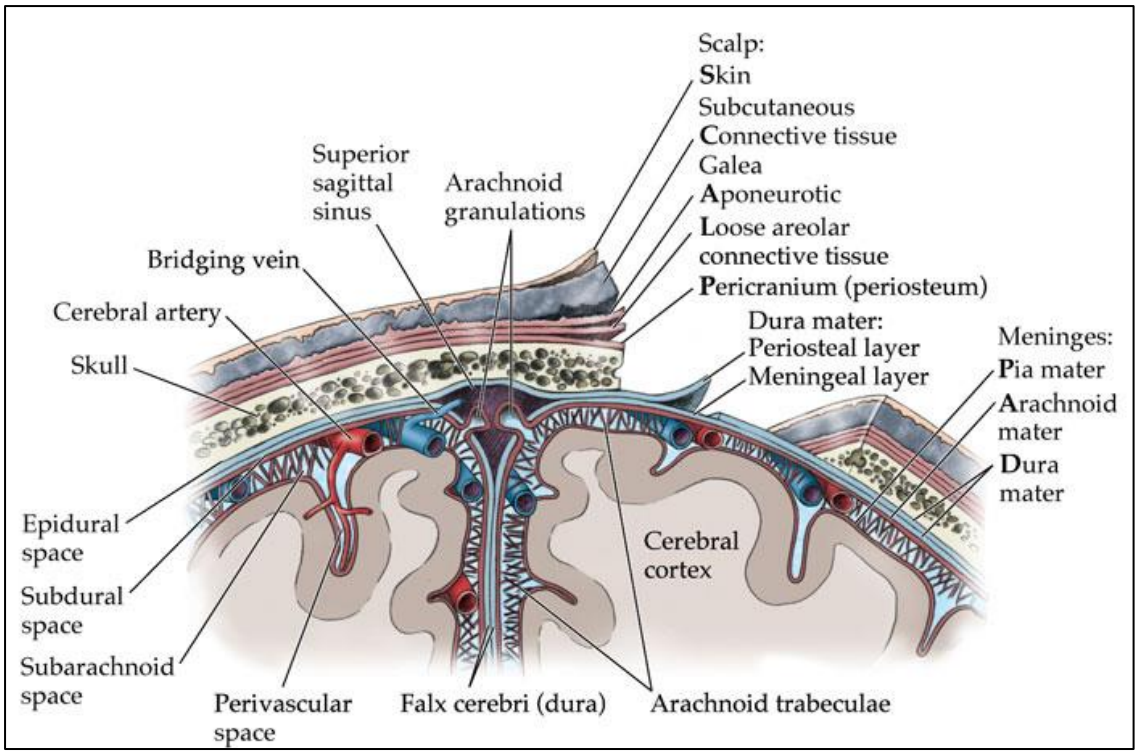

Figure 4.13 Cross-section through cranium, showing meningeal structure. Reproduced from www.missinglink.ucsf.edu.

This research proposes that endocranial lesions maybe in fact be highly suggestive of adult scurvy. In the course of this study, the types of endocranial lesions that were recorded include porosity in the grooves for the meningeal vessels, periosteal reaction commonly in the cruciate eminence of the occipital, in the sigmoid sulcus of the temporal bone, the sagittal sulcus and occasionally elsewhere on the internal skull. These lesions may be indicative of haemorrhaging in the dural area and several clinical case studies have discussed the scorbutic aetiology of both extra, sub dural and sub arachnoid haemorrhage (Ahuja and Karande 2002; Ingalls 1936; Hess 1920; Hempl and Wagner 1940; Sutherland 1894; Gilman and Tanzer 1932). The dura and their relationship to the periosteum and skull can be seen in Figure 4.13. In a clinical study conducted by Scherer (1913), it was recorded

that a quarter of their infantile scurvy study group presented with cerebral bleeding. Gilman and Tanzer in 1932 found seven reported cases of scurvy that was associated with meningeal bleeding (Gilman and Tanzer 1932). If a haemorrhage is caused by a small tear in the meningeal vessels, it can become chronic and may not become apparent for many weeks which could allow bony changes to occur. Hempl and Wagner (1940, 106) record that it took forty days for a subdural hematoma to be healed in a scorbutic infant. It has been suggested by Clemetson (2004a, 2004b) that histaminemia caused by Vitamin C depletion "causes capillary and venular fragility and can lead to retinal petechiae and bleeding from the bridging veins between the brain and the dura" (Clemetson 2004b 535). This is a likely scenario in chronic scurvy and could be caused or even exacerbated by minor trauma (Ahuja and Karande 2002, 442). It is clear that meningeal haemorrhaging is not unknown in scurvy particularly in subadults. There is also evidence for this occurring in adults. Lind records that in one autopsy that he conducted that he found 'four ounces of water under the dura mater and a small quantity of it in the right ventricle of the brain" (Lind 1772, 499). This could be similar to oedema which is commonly recorded in scurvy due to the frailty of the blood vessels in scurvy. It is possible that this could also cause inflammatory changes associated with endocranial lesions. " Lind (1772) often records that men with scurvy had severe headaches; these could have been caused by haemorrhage or oedema of the brain. Feigenbaum (1917) records a subdural haemorrhage in a scorbutic German soldier that died of the disease. Hess (1920, 86) in his compendium of scurvy states that scorbutic haemorrhaging often spares the central nervous system but "the meninges are somewhat more frequently involved". It would seem that meningeal involvement in adult scurvy

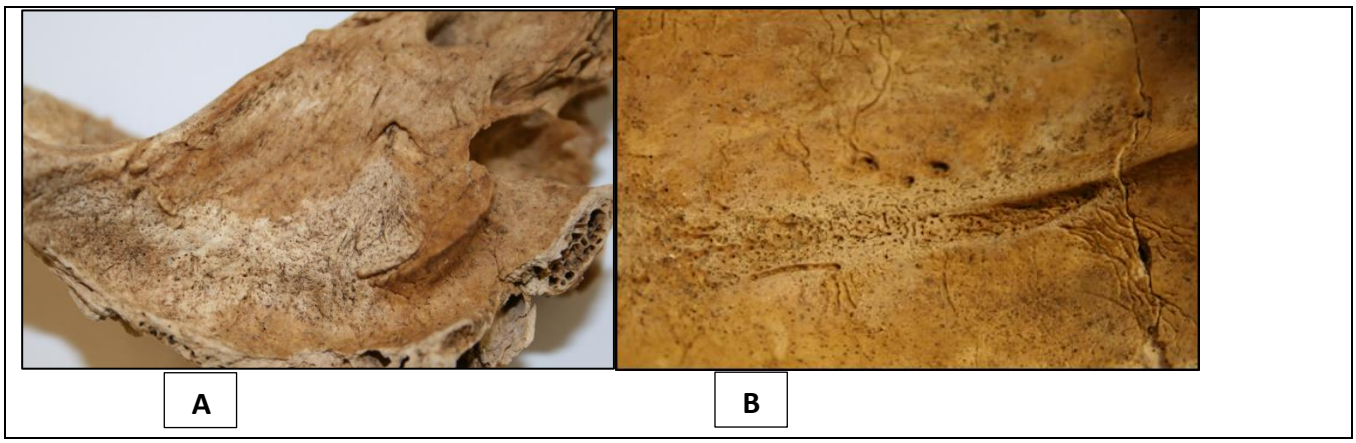

Figure 4.14 A- New bone in area of sigmoid sulcus of temporal, B- Bony reaction in area of sagittal sulcus of frontal.

is not unknown but is reported uncommonly, however it is possible that that scurvy could be the cause of endocranial lesions recorded in this study. The other proposed causative factors for endocranial lesions include tuberculosis, trauma and meningitis. Tuberculosis (TB) can cause erosive (lytic) endocranial lesions, along with other skeletal signs of TB such as rib and vertebrae lesions (Mays et al. 2002, 33). Very few erosive endocranial lesions were noted in this study, which excludes TB as a differential diagnosis. In total, 3 individuals were found to have TB. Meningitis could be cause of these lesions, however for the changes noted in this study to occur, it would have to be chronic meningitis. This is very rare in adults today (Boos et al. 2004), however we do not know its prevalence in the pre-antibiotic age. Therefore it must be considered an unlikely but potential cause of these lesions. It is possible that trauma could cause such lesions. In this case however, it is more likely that it is a combination of scurvy and minor trauma of the blood vessels causing intra-cranial haemorrhage and inflammation may be the cause of such lesions. Possible exacerbating factors that may lead to these lesions could include minor head trauma such as small blow to the head which could result from numerous circumstances. The surgeon of the *Terrible* in 1854 records the case of a marine who died after a 'slight blow' to the head, an autopsy revealed a blood clot between the dura mater and the skull but no trace of external injury

was found (ADM 102/122/5B/2). Alexander McKechnie of the *Layton*, a convict ship, records a brain effusion in a scorbutic man, with no evidence for trauma. Considering the patterning of the lesions which was discussed earlier, this aetiology is extremely plausible. This is because during the osteological analysis, it was noted that all the lesions occurred in places where extravasated blood would naturally pool if haemorrhaging did occur. The most common locations for lesions are the sigmoid sulcus, the cruciate eminence of the occipital and sagittal sinus see Figure 4.14A and 4.14B. These locations all contain sinuses which drain blood from the brain and are therefore likely sites for haemorrhaging of frail scorbutic blood vessels.

Lewis (2004) has classified endocranial lesions into four categories based on their appearance, structure and their stage of healing. These categories were used in this research and are detailed here;

1. Pitted lesions
2. Deposits of white or grey, fiber or immature new bone
3. Capillary lesions (new bone organised with or around vascular structures)
4. Hair-on-end formation characterised by frosting, thickening and remodelling

The endocranial surface of the skull of each skeleton was assessed for lesions where possible. In cases where the skull was intact, the endocranial surface was not visible for inspection, so only fragmented or broken skulls could be visually assessed for endocranial reaction. Lesions were found on the endocranial surfaces of all cranial vault bones and were recorded on a present/absent basis, healed/active and the Lewis (2004) category that it falls into.

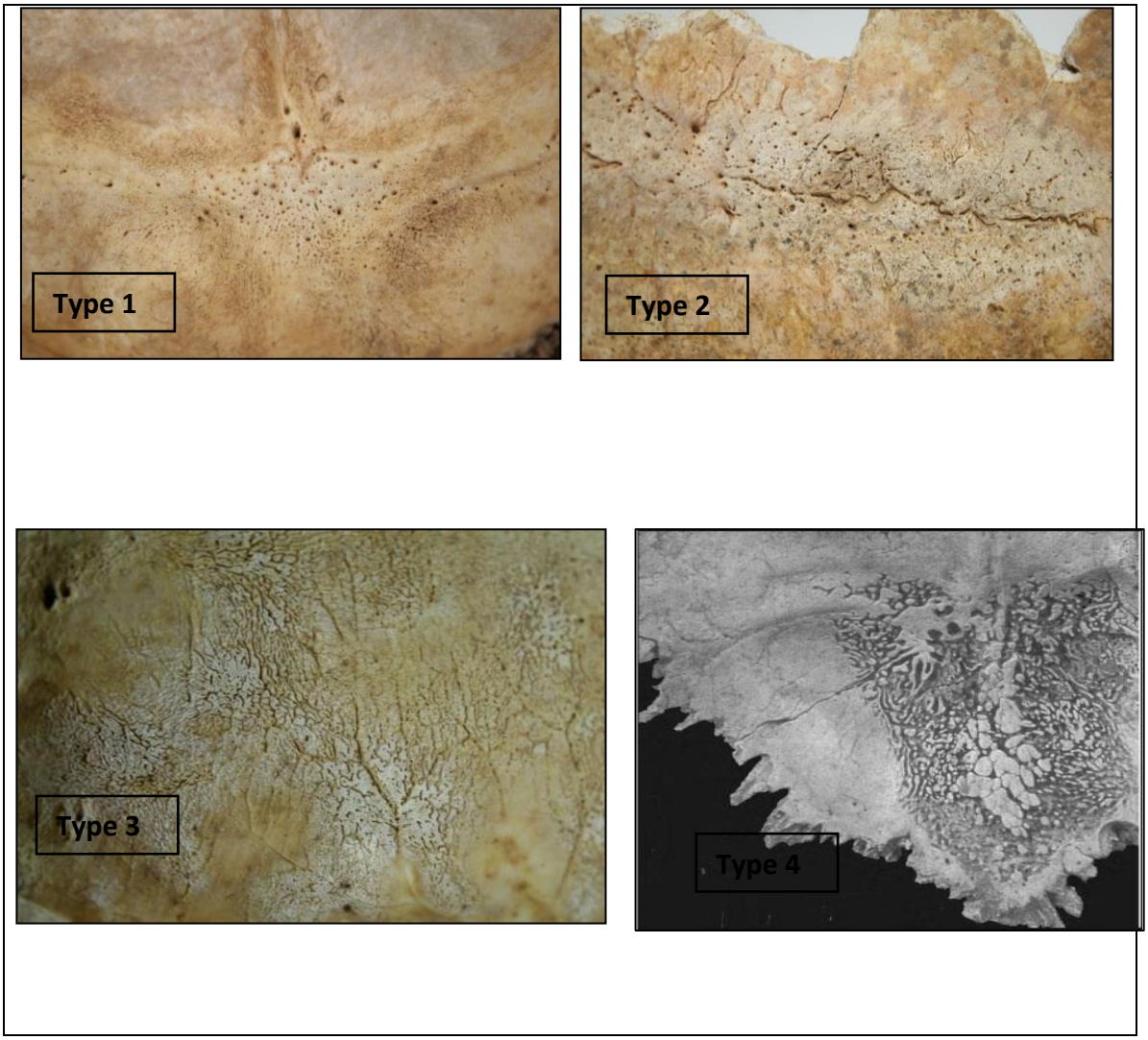

Figure 4.15 Endocranial lesion classification, all photos taken by author, except Type 4 which was reproduced from Lewis 2004.

4.5.1.7 Hard Palate

The hard palate was examined for porous lesions, spicules and remodelling on its surface. In healthy individuals there may also be some porosity of the hard palate particularly along the palatine suture (Crist 2005) and sometimes around the border of the dental arch forming a U shape (Brickley and Ives 2006). Porous lesions of the palate have been little discussed in the paleopathological literature, due to the acceptance that a small amount of porosity of the hard palate is customary and is often associated with tooth eruption and periodontal

disease (Brickley and Ives 2006, 165). However if hard palate porosity is marked or there is evidence of remodelling it may be indicative of scurvy. Ortner et al. (1999) proposes that if the porosity is outside the U-shaped arc that surrounds the alveolar bone, then it is atypical and may be pathological. Abnormal porosity and inflammation of the hard palate can be attributed to irritation, swelling or haemorrhaging of the mucosal tissues covering the hard palate or bleeding of the blood supply to the area. It has also been attributed to severe periodontal disease. Some of the most distinctive and commonly recorded changes of scurvy are that of the oral cavity (Halligan et al. 2005; Firth and Marvan 2001). Hess (1920, 193) records that "petechiae may not only be found in the skin but in the mucosa of the mouth especially overlying the hard palate". Lind (1772) also records that the hard palate and the back of the mouth is frequently ulcerated and black in colour (due to haemorrhages) in scurvy sufferers (Lind 1772, 122). This demonstrates that haemorrhaging in this area is commonplace in scurvy and is therefore likely to cause bony lesions. One of the most dramatic symptoms of scurvy is the sprouting of spongy gum tissue from the area of the hard palate inside the mouth; this has been frequently recorded in both modern medical and historical cases of scurvy (Lind 1772; Hess 1920; Halligan et al. 2005; Firth et al. 2001). This has been vividly described by De Champlain in his expedition "there was engendered in the mouths of those who had it large pieces of superfluous fungus flesh, which caused great putrefaction and this increased to such a degree, that they could hardly take anything but in liquid form (Champlain 1613, 303 in Crist et al. 2005). This pathognomonic symptom has been long recorded historically and it is likely that this would cause lesions of the hard palate, particularly remodelling and porosity caused by subsequent inflammation (Crist et al. 2005, 48), this could have also been accompanied by haemorrhaging of the palatine artery (Brown and Ortner 2001, 203) which could lead to

pathological lesions. It has been established to be an indicator of infantile scurvy (Ortner et al. 2009; Brickley and Ives 2006, Molleson and Cox 1993). Due to this lack of attention in the paleopathological literature, there are no alternative aetiologies put forward for lesions of the hard palate but in theory a severe infection or haemorrhaging could cause lesions. Considering that oral haemorrhagic and inflammatory lesions are characteristic of scurvy, it can be considered the most likely cause.

For an individual to be classified as having a pathological hard palate there had to be abnormal microporosity over 30% of the hard palate. This may be accompanied by spiculation and Figure 4.16 shows the three scale classification system for recording lesions

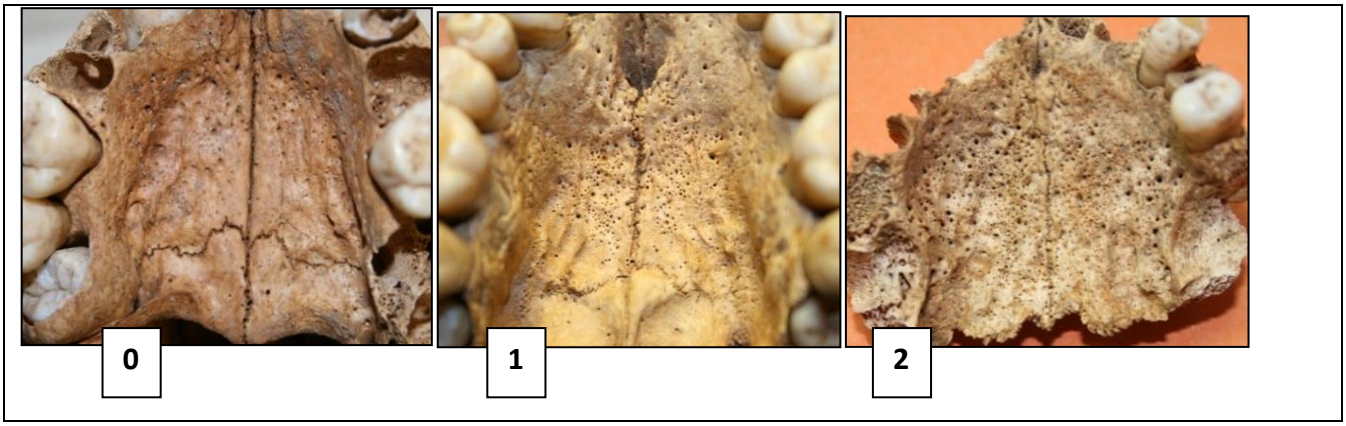

Figure 4.16 Showing classification scale of hard palate lesions.

of the hard palate, this classification was decided by the author. Number '0' is a normal non-pathological hard palate, there are some pores visible macroscopically but these are well within the normal palate, there are some pores visible macroscopically but these are well within the normal range. Number '1' palatine is moderately porous and displays a mixture of micro and macroporosity and spiculation (roughness). The '2' palatine is extremely porous and spiculated all over its surface and extending up towards the alveolar bone and sockets. In order to classify the palate lesions a scoring system was established by the author;

0. No atypical porosity, spiculation or remodelling

1. Mild to moderate porosity with or without spiculation and remodelling

2. Extreme porosity extending over the majority of the palate surface often accompanied with roughness, spiculation and remodelling.

Where there was extreme microporosity, associated with remodelling and periosteal reaction all over the hard palate, then these lesions were recorded as being possibly related to scurvy.

4.5.1.8 Maxilla and Zygomatic

There a several anatomical regions in this area that are thought to display scorbutic lesions these include; the infraorbital foramen, posterior maxilla and the anterior zygomatic, often in the area of the zygomatico-orbital foramen. The infraorbital foramen can display plaques of periosteal bone as shown in Figure 4.17A, these can be very subtle especially in the healed state. The posterior maxilla can also show abnormal porosity which is graded on Ortner's scale of porosity (1997). This lesion site is usually well defined and may also consist of a small raised plaque of bone, see Figure 4.17B. The posterior maxilla is considered to

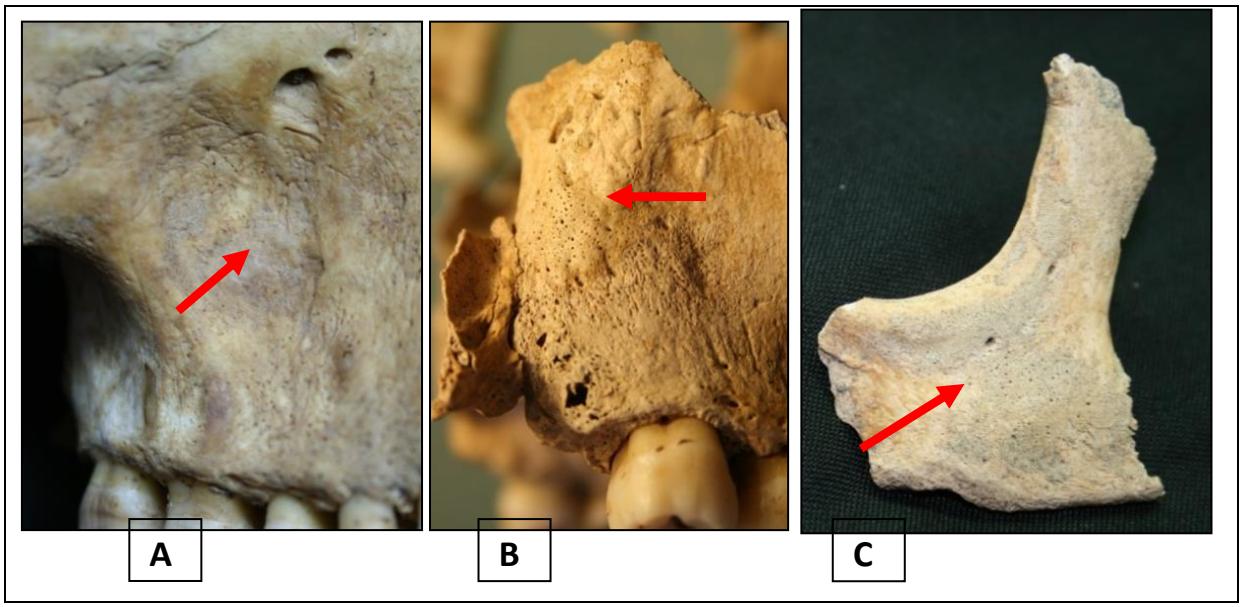

Figure 4.17 A- plaque of bone under infraorbital foramen, B- porosity of posterior maxilla, C-plaques of bone on zygomatic.

indicative of scurvy in paleopathological cases of infantile scurvy (Mays 2008; Ortner and Erickson 2007, Brickley and Ives 2008). The posterior maxilla is a key site for muscle attachment, the medial pterygoid muscle originates from the area of the maxillary tuberosity in this area, the muscle is vital in masticatory movements. It is thought that during mastication, this movement can cause haemorrhaging of the delicate blood supply to this muscle which is that of the inferior alveolar artery (Brown and Ortner 2001, 203). This is likely to manifest as swelling, haemorrhage and subsequent bony reaction in this area. Head, mouth and cheek swelling was of course not an uncommon occurrence in historical cases of scurvy "the muscles of the… cheeks became greatly swelled and hard" (Lind 1772, 410). Swelling of the muscles of the jaw has also been commonly recorded in experimental guinea pig scurvy (Hess 1920, 122). There has been no other proposed differential diagnosis for lesions of the posterior maxilla.

The zygomatic has been examined as a bone that may display scorbutic lesions in infantile skeletal material (Geber 2012; Brown and Ortner 2009). A vein and an artery run through the infraorbital foramen of the anterior zygomatic and these blood vessels are very susceptible to trauma as they are quite exposed to potential minor trauma on the front of the face. Possible scorbutic lesions of the zygomatic may consist of abnormal porosity adjacent to the infraorbital foramen and new reactive bone on the anterior zygomatic and hypertrophy of the bone (Brown and Ortner 2009, 199). New bone and inflammation in this area is uncommon and can be attributed to a small number of causes which may include scurvy (Jardine 2006), trauma and even tuberculosis (Agrawal et al. 1977). Hagmann (1937) records swelling over the zygomatic in an infant with scurvy, he was unsure whether the swelling resulted from oedema or haemorrhage (Hagmann 1937, 482). No specific medical

cases of this kind have been found in adults; however it is likely that this kind of lesions can be attributed to scurvy due to the frailty of the blood vessels emerging from the foramen, in the historical medical literature a swelling of the area around the eyes has been noted in scurvy sufferers (Hess 1920, Lind 1772).There are no references to adult zygomatic scorbutic lesions in the literature but the author has frequently noted abnormal porosity and new bone in this area and it was for this reason included on the scurvy recording form. However whilst this research was in progress, Geber et al (2012) published their research which did implicate infraorbital zygomatic lesions as indicative of scurvy in adults.

4.5.1.9 Mandible

Lesions that are linked to scurvy have long been thought to occur on the internal surface of the mandibular ramus adjacent to the mandibular condyle and coronoid process (Ortner 2001). In the course of this research lesions were also identified on the external surface of the mandible and extending down as far as the mental foramen. These lesions are frequently found bilaterally and may consist of porous lesions and plaques of periosteal

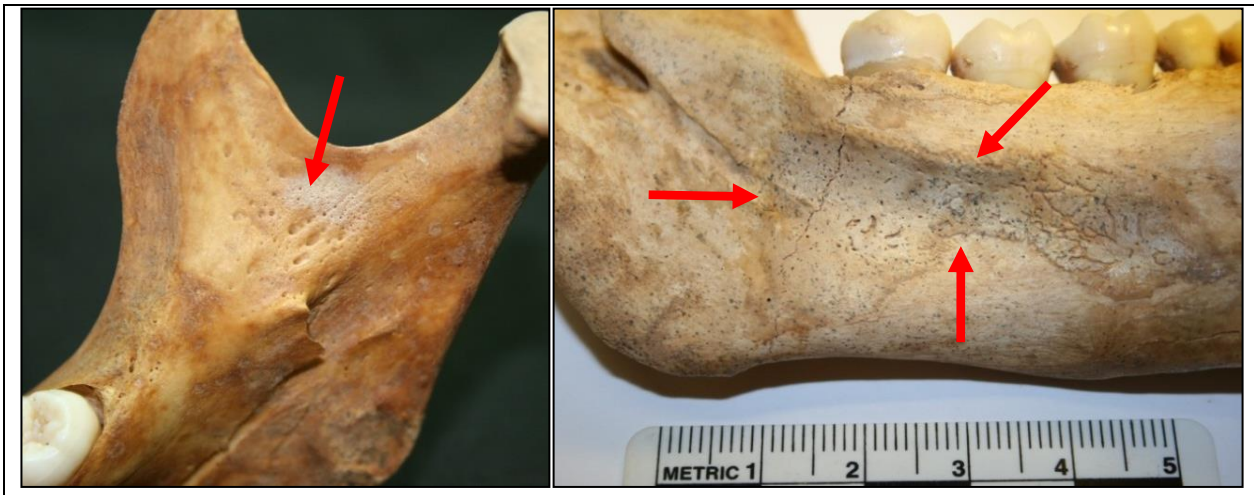

Figure 4.18 A- porosity on the internal mandible adjacent to the mandibular condyle, B- Plaque of periosteal bone on the internal body of the mandible below the alveolar bone.

bone. The two above photographs both show osseous scorbutic lesions on the internal surface of the mandible body and ramus. Figure 4.18A shows a distinct area of porosity adjacent to the mylohyoid groove. Figure 4.18B illustrates a large plaque of vascular periosteal bone running along the body of the internal mandible. The mandible is important as it is an attachment site for some of the muscles of mastication which are thought to be particularly susceptible to the effects of scorbutic haemorrhaging (Crist et al. 2005; Ortner and Erickson 1997; Ortner et al. 2001). The muscles of mastication that attach on the mandible are the masseter, temporalis and the lateral pterygoids. Therefore there are several anatomical areas of the mandible that should be examined for the effects of scorbutic haemorrhaging. The first of these is the area of the mandibular condyle where the lateral pterygoid muscle attaches, many authors have attributed porosity in this area to a scorbutic aetiology (Geber and Murphy et al. 2012; Brown and Ortner 2011; Brickley and Ives 2006, Ortner et al. 1999). The second anatomical region that can be considered is the coronoid process which is where the temporalis muscle attaches. The medial coronoid may display abnormal vascularity and porosity (Ortner et al. 1999). A third area that may display the effects of scorbutic haemorrhaging is the external mandibular body where the masseter lies adjacent to the mandibular ramus and attaches at the mandibular angle and the base of the mandibular ramus. Masseter is a massive thick muscle and previous experiments into guinea pigs have shown this muscle frequently degenerates in scurvy (Dalldorf 1929, 293) and is prone to haemorrhage and swelling (Hess 1920). This has also been noted in humans with severe scurvy, Lind (1772) specifically mentions that the masseter muscle becomes indurated (Lind 1772, 428) and “mortification spread to the cheeks and the lower jaw and the jaw bone in some fell down upon their breast” (Lind 1772, 259). Hess specifically mentions that the ramus of the lower jaw is often badly affected (Hess 1920, 178). During

this research it was also noted that the internal body of the mandible may be affected, below the area of the mylohyoid muscle. This type of lesion is shown in Figure 4.19.

Figure 4.19 Plaque of healing bone under mylohyoid line of internal mandible.

A fourth area that can also be affected with scurvy is the area around the mental foramen. The mental foramen occurs bilaterally usually below the second premolar on the external mandible. These foramina permit the exit of the mental nerve and blood vessels that supply the area. These blood vessels occur very close to the surface and are therefore susceptible to haemorrhage from minor trauma. This haemorrhage can be recognised as reactive bone or abnormal porosity. There have been no differential diagnoses put forward for these mandibular lesions, again it is possible that these lesions could be caused by trauma, but the pattering of lesions does not fit with this. All of the lesion sites are associated with the musculature of the mandible and occur in locations where bleeding is known to occur from the medical and historical references.

When recording mandibular lesions they are recorded on a present/absent basis and whether porous lesions or new bone is present. In both cases the extent of the reaction is sketched on an anatomical diagram of the mandible. When periosteal bone reaction is

present in the mandible it is important that is not as a result of an abscess or tooth infection.

4.5.2 Postcranial Scorbutic Pathology Classification

Every postcranial bone that had an intact cortical bone surface was assessed for the macroscopic lesions associated with scurvy. In general the vertebrae and the ribs were not expected to display any scorbutic lesions but virtually every other element present was examined for scorbutic periosteal reaction. Every bone present was visually examined for periosteal reaction, porosity and ossified haematomas. Periosteal reaction is defined as a non-specific inflammation of the periosteum which is the protective covering around the bone. This manifests itself as pitting/porosity, longitudinal striations and plaques of new woven or older healed lamellar bone on top of the cortical bone surface. Periostitis was recorded as being present/absent and healed/active. The type of bone formation was recorded, which bone it occurred on and a full description of the anatomical area where it

Figure 4.20 Ossified Haematoma distinguished by a 'lump' of crumbly white new bone on the anterior tibia.

occurred. Ossified haematomas were identified as being very localised, well demarcated areas of bone apposition (Van der Merwe et al. 2010). Ossified haematomas tend to be oval in shape and the key point is that they 'sit' on top of the original cortical bone surface. Figure 4.20 is showing a typical ossified haematoma, it is a different colour (they usually tend to vary from a white to grey colour) to the original cortical bone and is a typical ovoid shape and it is often 'raised' and 'lumpy'. It can be distinguished from generalised periosteal reaction in a number of ways. Periosteal reaction is not usually demarcated in this fashion and tends to be much more porous and 'fluffy' than hematomas which tend to more dense and 'crumbly' (Maat pers comm. 2012). This can be seen when comparing Figure 4.20 which is a dense concentration of white bone compared to a thin layer of porous bone in Figure 4.21. Periosteal bone is also much more plaque-like; it tends to be quite thin in nature. This can be seen in Figure 4.21, this brown periosteal bone is on-top of the cortical

Figure 4.21 Periosteal Reaction (non-anatomical) on tibia.

bone surface. This lesion shown in Figure 4.21 is not associated with a soft/bony anatomical feature, these will be discussed in the next section.

4.5.2.1 Anatomical Scorbutic Lesions

Most lesions in the cranial region that are associated with scurvy are associated with a soft tissue or bony anatomical region and this topic has been adequately addressed by Ortner in relation to the muscles of mastication (Ortner and Ericksen 1997). Haemorrhage due to the stress of chewing can occur in the masticatory musculature and it is this that is thought to cause lesions on the temporal, sphenoid and mandible (Ortner and Ericksen 1997). For the identification of postcranial lesions, this physiological explanation is being used as a template to identify the bony lesions of scurvy in the postcranial skeleton. This research hypothesises that the lesions of scurvy in the postcranial skeleton would have been limited by anatomical structures, similar to that in the cranial region. This will be discussed next.

4.5.2.2 Muscle Attachment Sites

It is being put forward that the bony lesions of scurvy are localised and delineated by muscle attachment areas, commonly recorded sites include the linea aspera of the femur,

Figure 4.22 Periosteal Reaction on the femur, related to anatomical feature.

the soleal line of the tibia, the gluteal lines of the innominates and others. Figure 4.22 is an image of a proximal third of the femur. In the image, a trochanter can be seen and the proximal linea aspera, there is grey/white periosteal bone present on this femur which is clearly defined by the linea aspera. This is vital in the diagnosis of an anatomical lesion. This is shown again in Figure 4.23 which is two images of a humerus. The first of these is a diagram from Gray's anatomy showing the muscle attachment areas, the second is a photograph of a humerus with extensive periosteal bone and by comparing the two it can be seen that the periosteal bone fits exactly the areas where muscles are lying and then it can be seen where the muscles attach there is no periosteal bone. A similar image can be seen in Figure 4.24A, which is a photograph of an innominate with delineated periosteal bone reaction. The periosteal reaction in this case is limited by the gluteal line and this is shown by comparing 4.24A to 4.24B, which is an anatomical diagram of the innominate

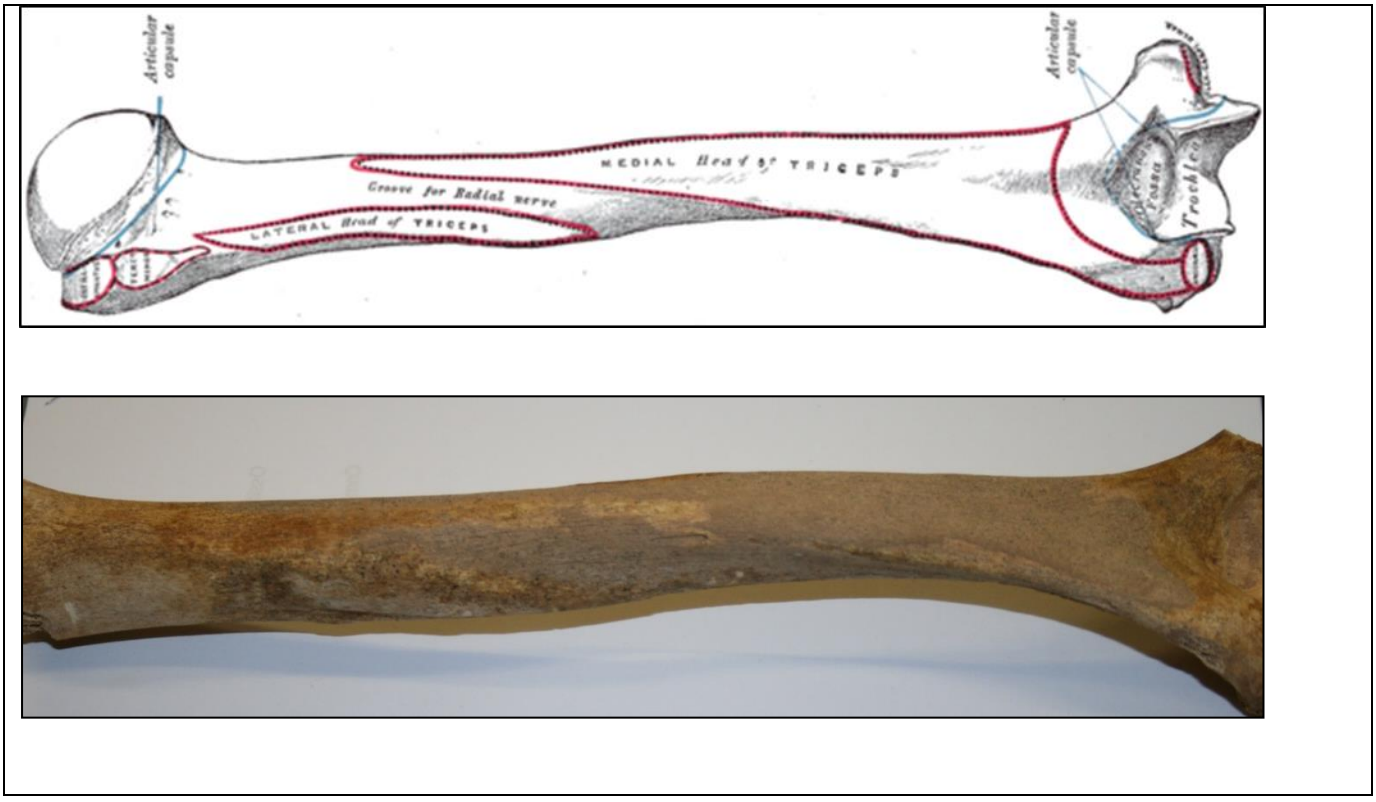

Figure 4.23 A- Anatomical Diagram of humerus showing muscle attachments, B- humerus with periosteal reaction. First image is reproduced from Gray's Anatomy.

bone showing the muscles that attach in this area. In this case, the periosteal reaction occurred in the area that is covered by the gluteus minimus muscle and is delineated by the extent of this muscle. When recording periosteal lesions, it was of particular importance to note these lesion limitations caused by musculature on the scurvy recording form and they were drawn on bone diagrams in an effort to link the reaction to a particular muscle or group of musculature. It is also important to differentiate new periosteal bone from hematoma formation, this has been discussed in section 3.9.3.2.

4.5.2.3 Other Anatomical Structures

During the recording of lesions, other periosteal and reactive lesions were recorded in association with anatomical structures, separate from the musculature. Primarily these

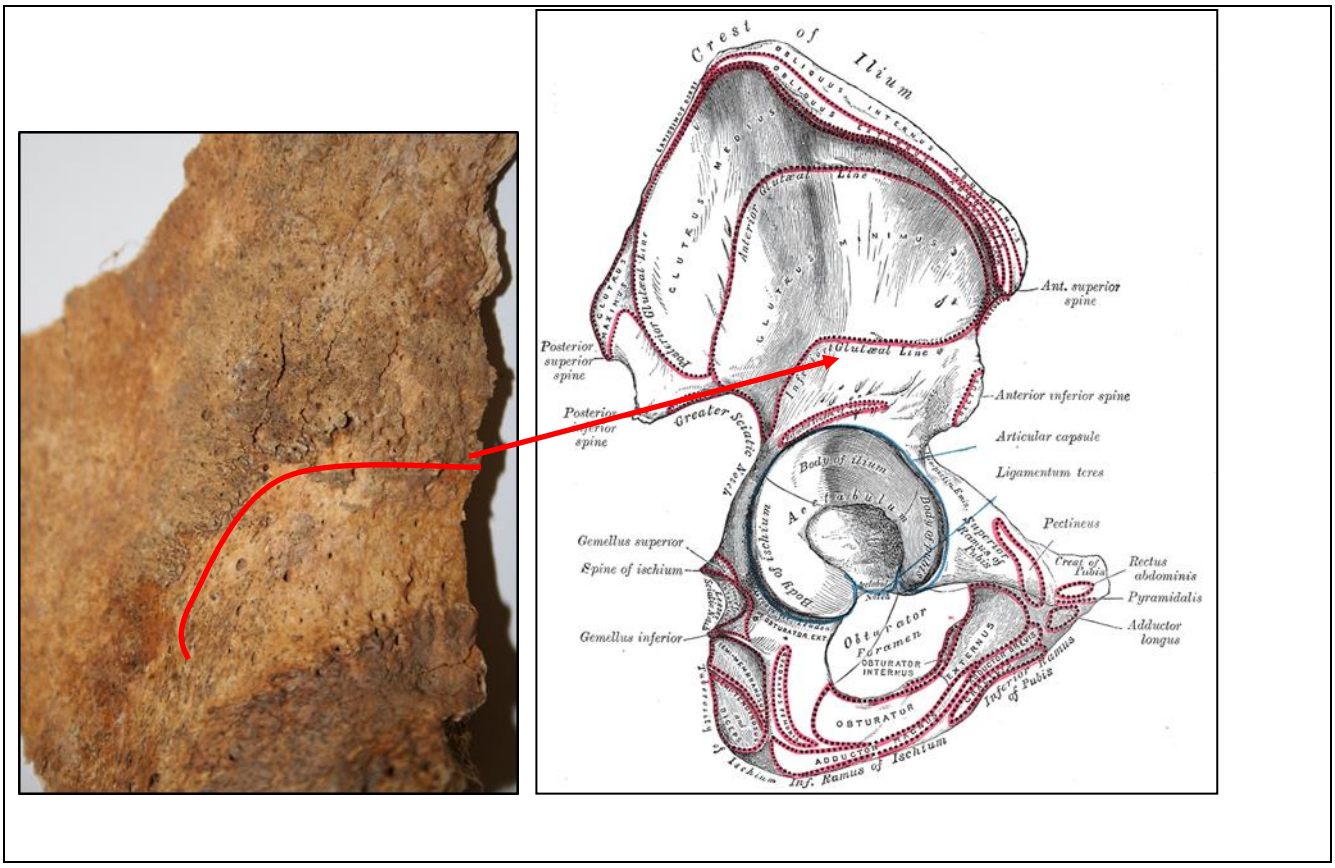

Figure 4.24 A- Innominate with periosteal reaction, B- anatomical diagram of innominate.

lesions occurred in association with postcranial foramina. The main purpose of foramina is to allow the passage of blood vessels and nerves through bone (Seeley et al. 1998, 182).Specifically periosteal lesions tend to occur adjacent to nutrient foramina, whose purpose it is to allow arteries and veins into bones to provide essential nutrients to the bone itself. In

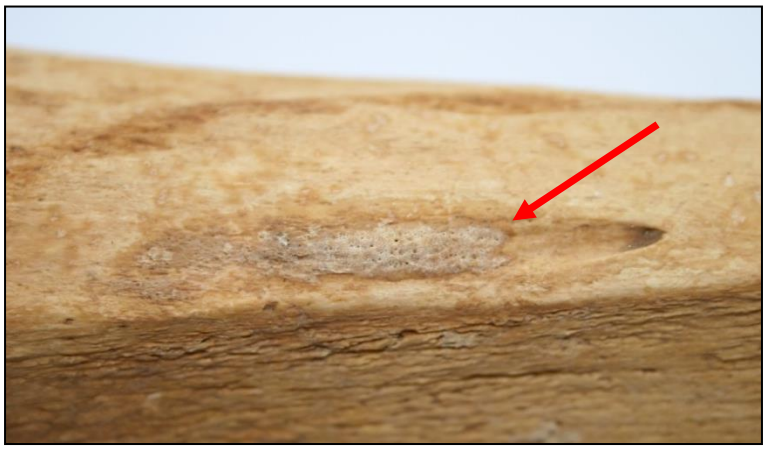

Figure 4.25 Periosteal bone in association with nutrient foramen of tibia.

Figure 4.25 a patch of periosteal bone is located in the groove for the nutrient foramen of the tibia. In Figure 4.26 a similar lesion is seen in association with the nutrient foramen for

Figure 4.26 Periosteal bone in association with the nutrient foramen of the fibula.

the fibula. This lesion can also occur on other bones in association with foramina for example the innominate. These lesions were recorded in the scurvy recording form according to location and type of pathology.

The preceding sections in this chapter have presented lesions of the cranial and postcranial skeleton that are likely to be associated with scurvy. When these lesions occurred, they were recorded in the scurvy recording form, with particular note being made of the location of the lesion. In the next subchapter, the statistical methodology will be presented.

4.6 Statistical Methodology

The following chapter summarises the analysis of all three hundred and fifty-eight skeletons by site, skeleton number and scorbutic bony lesion occurrence. All data was originally recorded by hand using the scurvy recording form and the standard human remains recording form. This data was then input into Microsoft Excel 2010. This data was visually checked for any mistakes or anomalies and was then imported into IBM Statistics Programme for the Social Sciences (SPSS) 20. This primary excel database is attached in Appendix C. A coding system for the recorded scorbutic lesions was established which is detailed below:

1. Bone Present but no pathological bony lesions present
2. Bone Present with pathological non-anatomical bony lesion present
3. Bone Present with anatomically defined lesion present
9. Bone Missing or bone surface unobservable

In total the data for three hundred and fifty-eight skeletons was selected and then input into the database. For each skeleton thirty-two variables were scored. Of these thirty-two

variables, ten of these from the cranial region were scored and then eleven from the postcranial skeleton which were scored for both left and right sides. The cranial variables were: endocranial, ectocranial, ante-mortem tooth loss, zygomatic, mandible, periodontal disease, posterior maxilla, sphenoid, cribra orbitalia and hard palate. The postcranial variables were: femur, tibia, fibula, foot, innominate, humerus, ulna, radius, clavicle, scapula and hand. From this data it was possible to obtain the true prevalence (TPR) rates for each bone and site. A true prevalence rate is the number of bones/elements affected by pathological lesion as a proportion of the total number of observable elements present. This is important, as it takes into account bone preservation and completeness. This provides a more accurate picture than crude prevalence rates (CPR) which do not take bone preservation into account. A crude prevalence rate is the total rate of lesion occurrence over the total number of individuals in an assemblage. The true prevalence rates were determined for each population and are presented in the tables in the following section. For each site, the total number of bones present, those with anatomical lesions and those with non-anatomical lesions were calculated. In total 358 skeletons formed part of this study, however due to the preservation threshold, this number was reduced to 284 suitable skeletons which were then examined for lesions that may be associated with scurvy.

Table 4.6 Total final study group shown by age and sex.

Combined Assemblage	?	Male	Male?	Female	Female?	Total
Adolescent	3	14	3	4	0	24
Young Adult	10	50	13	4	0	77
Middle Adult	8	25	9	4	1	47
Mature Adult	10	16	17	2	1	46
Older Adult	6	11	5	2	0	24
Adult	13	27	22	4	0	66
Total	50	143	69	20	2	284

These 284 skeletons shown by age and sex can be seen in Table 4.6. It is clear that there is a massive underrepresentation of females in the final study group. In total, only 22 females made the final study group. In general there is a mix of age groups however young adults are the largest age group and older adults are a very small proportion of the overall group.

4.6.1 Statistical Analyses Conducted

Statistical analyses were conducted both within and between the Naval and non-Naval combined collections. Chi-squares were conducted between the left and right sides of the postcranial bones. It was also conducted to establish difference between the rates of lesions recorded in the combined Naval vs. Non Naval Assemblage for all lesions and for anatomical compared to non-anatomical lesions. Statistical analyses looking at the relationship between all the recorded variables were also carried out. Chi- square tests were used to test for differences in all of these statistical comparisons. When P values were calculated in statistical tests, it was decided that $p = < 0.01$ with a 99% significance level should be used as the standard for any significant correlations. The alpha level was defined at 1% to guarantee that the differences and correlations identified between the populations and variables were not random for at least 99% of cases. This was decided in order to make the evidence to reject the null hypothesis stronger. When the expected chi-square was less than 5, then Fishers Exact test was used employed to test for probable non-random relationships between variables. Several hypothesises were put for forward for testing. The first of these was that there would be higher rates of lesions and in particular anatomical lesions in the combined Naval assemblage. This is due to the strong historical evidence for scurvy in the combined Naval assemblage. The first null hypothesis was that there would be no difference between combined Naval and non-Naval assemblages. The second null hypothesis was that

there would be no difference between the observed rates of anatomical compared to non-anatomical lesions between the Naval and non-Naval assemblages.

Chapter 5: Results

5.1 Introduction to Results

The following chapter presents the osteological and historical results of the analysis conducted as part of this research. The first part of this chapter will introduce the results for the standard osteological analysis of all four assemblages. This will include the demographic profiles for each site and a description of the preservation and completeness of the skeletal material analysed. This is important as preservation and completeness has direct implications on the identification of scorbutic pathological lesions. The second section will introduce the results for the analysis of scorbutic lesions which will include the prevalence rates and the statistical analysis for each lesion.

5.2 Introduction to Osteological Analysis Results

In this section, the osteological analysis of each skeletal assemblage will be presented. The demographic profile of each group will be displayed in table form. The preservation and completeness of each group will also be outlined.

5.2.1 Royal Hospital Haslar

In total forty-five individuals were analysed from this site. Preservation varied across the site. The majority of the skeletons were excavated from the area of the central Paddock and of these, many were poorly preserved (44.4%), as can be seen in Table 5.2. This is due to the gravelly soil conditions that encouraged significant water drainage and subsequently severe surface damage and leaching of the bone. In contrast, the skeletons excavated from the area in the west of the Paddock were buried in clay and were in near perfect condition and these account for some of the well preserved skeletons. Despite the overall poor preservation, the majority (88.9%) of skeletons were more than 51% complete as there was

little to no truncation or disturbance of graves in the main Paddock trenches, this can be seen in Table 5.1.

Table 5.1 Completeness of the skeletons from Haslar.

Completeness	Skeletons No.	Percentage
76-100%	22	48.9%
51-75%	18	40%
26-50%	4	8.9%
0-25%	1	2.2%
Total	45	100%

The completeness of the Haslar skeletons meant that most of the skeletons from this site were aged and sexed as can be seen in Table 5.3. Only 1 (2.2%) Haslar skeleton could not be

Table 5.2 Preservation of skeletons from Haslar.

Preservation	Skeletons-No	Percentage
Good – 1	22	48.9%
Moderate- 2	3	6.7%
Poor- 3	20	44.4%
Total	45	100%

aged or sexed due to poor preservation. From inspection of Table 5.3, it can be seen that just one female was identified, the rest of the skeletons were all classified as males or

Table 5.3 Age and Sex of Haslar skeletons.

Haslar	?	Male	Male?	Female	Female?	Total
Adolescent	1	1	7	0	0	9 (20%)
Young Adult	0	8	3	0	0	11 (24.4%)
Middle Adult	0	9	2	0	0	11 (24.4%)
Mature Adult	0	9	0	1	0	10 (22.2%)
Older Adult	0	1	1	0	0	2 (4.4%)
Adult	0	0	2	0	0	2 (4.4%)
Total	1(2.2%)	28(62.2%)	15(33.3%)	1(2.2%)	0	45

probable males. Out of these, 71% of these were either young, middle or mature men aged between 19 and 44. This is unsurprising, in that men in active naval service were mostly

young or middle aged due to the physical demands of this trade. Few active seamen would have been older, only 2 older men (4.4%) were recorded in the Haslar assemblage.

5.2.2 Stonehouse Hospital, Plymouth

In total one hundred and eighty nine skeletons were analysed from Stonehouse hospital, Plymouth. This site was excavated as part of a commercial rescue excavation and the completeness of the skeletons suffers as a result of the overuse of mechanical diggers on-site to uncover skeletal remains, this can been seen in Table 5.4. This was also due to some

Table 5.4 Completeness of Stonehouse, Plymouth skeletons.

Completeness	Skeletons No.	Percentage
76-100%	65	34.4%
51-75%	40	21.2%
26-50%	45	23.8%
0-25%	39	20.6%
Total	189	100%

truncation of burials by overlying buildings and other structures. The majority of the Plymouth skeletons (87.8%) were in good or moderate preservation, which can be seen in Table 5.5, this was due to the soft 'shillet clay' at the burial site (Plymouth City Council 2007). The good preservation meant that the majority of the skeletons could be aged and sexed accurately. Only 15 (7.8%) skeletons could not be sexed, this was despite many not

Table 5.5 Preservation of the Stonehouse, Plymouth skeletons.

Preservation	Skeletons-No	Percentage
Good – 1	96	50.8%
Moderate- 2	70	37%
Poor- 3	23	12.2%
Total	189	100%

having a skull or pelvis. Many of the sexually dimorphic measurements proved to be useful as a lot of the skeletons were very masculine in their measurements. However 57 (30.2%) of the Plymouth could not be aged accurately. This is again likely to be due to the heavy use of

Table 5.6 Age and Sex demography of the Stonehouse, Plymouth assemblage.

Plymouth	?	Male	Male?	Female	Female?	Total
Adolescent	2	10	7	0	0	19 (10%)
Young Adult	3	45	12	0	0	60 (31.7%)
Middle Adult	2	13	4	1	0	20 (10.6%)
Mature Adult	3	16	4	0	0	23 (12.2%)
Older Adult	0	7	3	0	0	10 (5.3%)
Adult	5	41	11	0	0	57 (30.2%)
Total	15(7.9%)	132(69.8%)	41(21.7%)	1(0.6%)	0	189

mechanical excavators at this site. This is because as a skeleton lies in the ground the front of the pelvis is slightly higher than the rest of the skeleton and is more likely to be damaged by mechanical diggers. The pelvis is vital for aging adult individuals and if it is not present then aging is made very difficult. Out of the individuals that could be sexed, all but one were assigned as males or possible males. The males that were analysed were mostly young or middle adults (42.3%) aged between 19 and 35 years of age, which fits in well with what was found with Haslar and what is known about men in active service in the Royal Navy. The number of adolescents is high at nineteen individuals (10%) and indicates the young age that men were recruited into the Navy, which was sometimes as young as 12 (Rogers 1988). On the whole, the Plymouth skeletons represent a group of young males, unsurprising considering the make-up of Royal Navy in this period.

5.2.3 Oxford Castle, Oxford City.

In total sixty three skeletons were analysed from Oxford Castle. Almost one third (28.6%) of skeletons from Oxford Castle were less than 25% complete, this was due to high levels of

truncation and anatomisation at this site, as can be seen in Table 5.8. Despite this high level of incomplete skeletons, the overall preservation of the collection was good (85.7%) as can be seen in Table 5.7. The good preservation of the Oxford Castle skeletons meant that high levels of pathology were recognised in this assemblage. Despite the good preservation, a

Table 5.7 Preservation of the Oxford Castle skeletons.

Preservation	Skeletons- No	Percentage
Good – 1	54	85.7%
Moderate- 2	9	14.3%
Poor- 3	0	0%
Total	63	100%

good deal of the Oxford Castle skeletons could not be sexed and/or aged due to missing pelvic or cranial elements. It can be seen in Table 5.9, that a significant amount of skeletons (34.9%) could only be assigned as adults and that eleven individuals (17.5%) of individuals could not be assigned a sex. From the examination of Table 5.9, it can be seen that the

Table 5.8 Completeness of Oxford Castle Skeletons.

Completeness	Skeletons No.	Percentage
76-100%	29	46%
51-75%	8	12.7%
26-50%	8	12.7%
0-25%	18	28.6%
Total	63	100%

majority (63.5%) of the skeletons from Oxford Castle were assigned as either male or possible males. Out of both males and females, the largest group was that of adults that could not be accurately aged (34.9%) followed by middle adults (17.5%) aged between 26 and 35 years of age and adolescents (17.5%) aged between 13- 18 years of age. Overall the Oxford Castle assemblage is predominately a collection of young males, which tallies well with John Howards (1777) description of typical convicts in this period.

Table 5.9 Age and Sex of the Oxford Castle skeletons.

Oxford Castle	?	Male	Male?	Female	Female?	Total
Adolescent	1	4	3	3	0	11 (17.5%)
Young Adult	0	4	2	1	0	7 (11.1%)
Middle Adult	0	7	2	2	0	11 (17.5%)
Mature Adult	0	4	0	3	0	7 (11.1%)
Older Adult	0	3	0	2	0	5 (7.9%)
Adult?	10	5	6	1	0	22 (34.9%)
Totals	11(17.5%)	27(42.9%)	13(20.6%)	12(19%)	0	63

5.2.4 Redearth, Darwen.

In total sixty-one skeletons were analysed from Redearth, Darwen. The taphonomic conditions at Darwen were most unusual and twenty-five (41%) of skeletons were poorly preserved. Table 5.10 and 5.11 detail the preservation and completeness of the adult

Table 5.10 Preservation of Redearth, Darwen assemblage

Preservation	Skeletons-No	Percentage
Good – 1	8	13.1%
Moderate- 2	28	45.9%
Poor- 3	25	41%
Total	61	100%

skeletons excavated from Redearth. The taphonomic conditions at Redearth were very unusual, there was frequent significant warping and cortical bone flaking, especially of the long bones (Oxford Archaeology Interim Report 2011). Often the bones were discoloured to

Table 5.11 Completeness of Redearth, Darwen assemblage.

Completeness	Skeletons No.	Percentage
76-100%	23	37.7%
51-75%	12	19.7%
26-50%	10	16.4%
0-25%	16	26.2%
Total	61	100%

a pink colour and there was heavy post-mortem fragmentation and cracking of bones. This is thought to have been caused by the changing water levels in the site, the bones were subject to repeated wetting and drying over a long period of time. Only eight (13.1%) of the adult skeletons were in good condition, as can be seen in Table 5.10. Despite the poor condition of most skeletons, thirty-five (57.4%) of the skeletons were 51% or more complete. This was because there was little to no intercutting of graves and most missing elements could be attributed to the unusual taphonomic conditions. Due to poor preservation of the Redearth assemblage, accurate aging and sexing was not always possible. In total, twelve individuals (19.7%) of the total assemblage could not be sexed, as can be observed in Table 5.12. Of the remaining skeletons that could be sexed, it was found

Table 5.12 Age and Sex demographic of the Darwen, Redearth assemblage.

Redearth-Darwen	?	Male	Male?	Female	Female?	Total
Adolescent	4	1	0	1	0	6 (9.8%)
Young Adult	0	1	1	4	0	6 (9.8%)
Middle Adult	0	4	1	7	2	14 (23%)
Mature Adult	0	4	0	4	1	9 (14.8%)
Older Adult	2	2	0	6	0	10 (16.4%)
Adult-Age?	6	2	3	2	3	16 (26.2%)
Totals	12(19.7%)	14(22.9%)	5(8.2%)	24(39.3%)	6(9.8%)	61

to be a predominantly female group, with thirty (61.2%) skeletons assigned as female or possible female. Age could not be obtained for sixteen (26.2%) of the total population, when aging was possible, there were peaks at death for middle aged to older adults. A small number of the Redearth skeletons died in their sixties and possibly even seventies. Overall this is a typical familial assemblage, that dominated by middle aged to older females and the males that were present had died at a younger age than the females, peaking at middle and mature adults.

5.2.5 Summary of Osteological Analysis Results

From examination of all the demographic data available for each assemblage, it is clear to see that both Haslar and Plymouth are atypical of archaeological skeletal assemblages. These are male dominated populations consisting of specialist individuals, who all died whilst in the service of the Royal Navy. Oxford Castle represents a group of people that died whilst awaiting trial for criminal activities or those that were convicted and subsequently executed following trial. Most of these individuals were young to middle-aged men. Redearth, Darwen was the only familial assemblage that was examined and it represents the 'normal' death curve expected of an Industrial era population (Roberts and Cox 2003). There was significant infant mortality, with those women that did not die in childbirth living longer than men who commonly died in their middle age (Oxford Archaeology Interim Report 2011).

5.3 Cranial Lesions

In total there were ten elements that were assessed for lesions in the cranial region, these include: 1. ectocranial 2. endocranial, 3. ante-mortem tooth loss, 4. hard palate, 5. sphenoid, 6. cribra orbitalia, 7.posterior maxilla, 8. zygomatic, 9.mandible and 10. periodontal disease. During the recording of cranial lesions, lesions were recorded as being present if they occurred bilaterally, which one would expect with scurvy. Therefore in the skull, lesions were not recorded as left and right but as absent or present.

5.3.1. Ectocranial Pathology

Ectocranial pathology was commonly recorded in all of the study assemblages. Table 5.13 shows all the values for ectocranial pathology for each assemblage. In the total combined assemblage 80.6% (229/284) had observable ectocranial skull surfaces. Overall 78.6%

(180/229) skeletons displayed evidence of pathological lesions of which the majority of these 60.3% (138/229) were related to anatomical ectocranial features. Haslar showed by far the highest rate of anatomically related ectocranial pathology (88.6%), followed by Plymouth (73.9%). The comparative assemblages had very similar rates with fourteen skeletons from each site displaying anatomically related lesions, Oxford Castle had a TPR of 35.9% (14) and Darwen had a TPR of 28% (14). Chi-square tests were used to determine if there was any difference between the combined Naval and combined Non-Naval collections. When these are compared, there is a statistically significant difference [$\chi 2$ (1) = 15.621, p = <0.001]. When anatomical lesions are compared to non-anatomical lesions, there is also a statistically significant difference observed [$\chi 2$ (1) = 38.558, p = <0.001]. Chi-squares were also used to establish if there was any statistically significant difference

Table 5.13 Ectocranial Lesion rates by site.

Lesion Coding	Darwen	Oxford Castle	Haslar	Plymouth	Total
1.-no lesion	21 (42%)	10 (26.4%)	3 (6.8%)	15 (15.6%)	49
2.non-anatomical lesion	15 (30%)	15 (38.5%)	2(4.5%)	10 (10.4%)	42
3.-anatomical lesion	14 (28%)	14 (35.9%)	39 (88.6%)	71 (73.9%)	138
Total	50	39	44	96	229

between ectocranial and other lesions recorded. The only comparative lesions that there was no significant association with was cribra orbitalia, posterior maxilla, zygomatic, mandible, and fibula at the level of P=>0.01.

5.3.2. Endocranial Pathology.

Endocranial pathology was commonly recorded for the Plymouth and Haslar collections. Table 5.14 shows the rates of this lesion occurance across all assemblages. In the overall group, 80.6% (229/284) of skulls were fragmented but in good condition which allowed examination of the internal skull surface. The overall true prevalence rate of endocranial lesion occurrence was 47.2% (108/229) and out of this 41% (94/229) showed lesions that were anatomically linked. The highest true prevalence rate recorded was at Plymouth, where 60.4% (58) of skeletons displayed anatomically related lesions, followed by Haslar with 43.2% (19). Out of the comparative groups, 17.5% (7) of the Oxford Castle skeletons

Table 5.14 Endocranial lesion rates by site

Lesion Coding	Darwen	Oxford Castle	Haslar	Plymouth	Total
1.-no lesion	38 (77.6%)	26 (65%)	24 (54.5%)	33 (34.4%)	121
2.-non anatomical lesion	1 (2%)	7 (17.5%)	1 (2.3%)	5 (5.2%)	14
3.- anatomical lesion	10 (20.4%)	7 (17.5%)	19 (43.2%)	58 (60.4%)	94
Total	49	40	44	96	229

and 20.4% (10) of the Darwen skeletons were affected. Chi-square tests were used to determine if there was any difference between the combined Naval and combined Non-Naval collections. When these are compared, there is no statistically significant difference [$\chi 2$ (1) = 3.773, p = 0.0521]. When anatomical lesions are compared to non-anatomical lesions, there is a statistically significant difference observed [$\chi 2$ (1) = 10.449, p = 0.001].

Chi-squares were also used to establish if there was any statistically significant difference between endocranial and other lesions recorded. There was no statistical correlation with hard palate, femur and tibia at P= >0.01.

5.4.3 Ante-Mortem Tooth Loss

Ante-mortem tooth loss was recorded for all assemblages but was rarely recorded as being associated with an anatomically linked feature. Across the overall combined collections 77.8% (221/284) of individuals had retained sufficient alveolar bone to assess tooth presence. In total 58.4% (129/221) individuals showed ante-mortem tooth loss. Only 1.8%

Table 5.15 Ante-Mortem Tooth Loss rates by site.

Lesion Coding	Darwen	Oxford Castle	Haslar	Plymouth	Total
1.-no lesion	9 (20.5%)	20 (57.1%)	20 (47.6%)	43 (43%)	92
2.-non-anatomical lesion	35 (79.5%)	15 (42.9%)	20 (47.6%)	55 (55%)	125
3.-anatomical lesion	0	0	2 (4.8%)	2 (2%)	4
Total	44	35	42	100	221

(4/221) of these total cases were anatomical lesions. These four skeletons originated from Haslar and Plymouth. Fisher's Exact test were used to determine if there was any difference between the ante-mortem tooth loss rates of the combined Naval and combined Non-Naval collections. When these are compared, there is no statistically significant difference [p = 0.3193]. When anatomical lesions are compared to non-anatomical lesions, there is no

statistically significant difference observed [p = 0.1574]. Chi-squares were also used to establish if there was any statistically significant difference between ante-mortem tooth loss and other lesions recorded. There was no significant association between AMTL and sphenoid, zygomatic and mandible lesions at P=>0.01.

5.3.4 Hard Palate

Table 5.16 shows the rate of hard palate lesions recorded for each site. In the combined assemblage there were 71.5% (203/284) of individuals that had observable hard palates. Overall it was established that 72.4% (147/203) of individuals presented with pathological

Table 5.16 Hard palate lesion rates by site.

Lesion Coding	Darwen	Oxford Castle	Haslar	Plymouth	Total
1.-no lesion	23 (37.7%)	21 (67.7%)	1 (2.4%)	11 (11.9%)	56
2.-non anatomical lesion	15 (24.6%)	6 (19.4%)	22 (53.7%)	55 (59.8%)	98
3.- anatomical lesion	1 (1.6%)	4 (12.9%)	18 (43.9%)	26 (28.3%)	49
Total	39	31	41	92	203

lesions of the hard palate. Of these 24.1% (49/203) were deemed to be anatomically pathological in nature. The highest rate was recorded at Haslar where 43.9% (18) individuals had anatomical lesions, of the Plymouth skeletons 28.3% (26) showed these lesions. Both Darwen and Oxford Castle had lower rates recorded, only 12.9% (4) individuals at Oxford Castle and 1.6% (1) at Darwen. Chi-square tests were used to determine if there was any difference between the rates of hard palate lesions in the combined Naval and combined

Non-Naval collections. When these are compared, there is a statistically significant difference [χ2 (1) = 66.537, p =< 0.001]. When anatomical lesions are compared to non-anatomical lesions, there is no statistically significant difference observed [p = 0.1114]. Chi-squares were also used to establish if there was any statistically significant difference between hard palate and other lesions recorded. Sphenoid, endocranial, tibia and femur were did not correlate at P= >0.01.

5.3.5 Sphenoid

Table 5.17 details the sphenoid lesion rates for each site. When the four assemblages were combined, there were 195 sphenoid bones that were of research quality and could be assessed for lesions. Overall 64.1% (125/195) individuals displayed pathological lesions of the sphenoid. Of these 52.8% (103/195) displayed anatomically related lesions. This rate was highest at Haslar 74.4%, followed by Plymouth with 60.7% (51) skeletons affected, Darwen also displayed a high rate 38.5% and 17.2% (5) were affected at Oxford Castle. Chi-square tests were used to determine if there was any difference between the combined Naval and combined Non-Naval collections. When these are compared, there is a statistically significant difference [χ2 (1) = 45.738, p =< 0.001]. When anatomical lesions are compared to non-anatomical lesions, there is no statistically difference between the combined Naval and combined Non-Naval collections. When these are compared, there is a statistically significant difference [χ2 (1) = 45.738, p =< 0.001]. When anatomical lesions

Table 5.17 Sphenoid lesion rates by site.

Lesion Coding	Darwen	Oxford Castle	Haslar	Plymouth	Total
1.-no lesion	22 (56.4%)	24 (82.8%)	4 (9.3%)	20 (23.8%)	70
2.-non anatomical lesion	2 (5.1%)	0	7 (16.3%)	13 (15.5%)	22
3.- anatomical lesion	15 (38.5%)	5 (17.2%)	32 (74.4%)	51 (60.7%)	103
Total	39	29	43	84	195

are compared to non-anatomical lesions, there is no statistically significant difference observed [p = 0.3600]. Chi-squares were also used to establish if there was any statistically significant difference between sphenoid and other lesions recorded. There was a significant difference with ante-mortem tooth loss and hard palate at P= >0.01.

5.3.6 Cribra Orbitalia

Overall 68.3% (194/284) individuals had their left orbit intact and observable. For cribra orbitalia there was a high true prevalence rate at 41.2% (80/194) and a very low rate of potential anatomical lesions at 6.7% (13/194). Anatomically related lesions were only recorded at Haslar and Plymouth. In total 19.5% (8) skeletons at Haslar were affected and 6.2% (5) at Plymouth. Chi-square tests were used to determine if there was any difference between the combined Naval and combined Non-Naval collections. When these are compared, there is no statistically significant difference [p = 0.6524]. When anatomical

Lesion Coding	Darwen	Oxford Castle	Haslar	Plymouth	Total

1.-no lesion	**26 (61.9%)**	**18 (60%)**	**24 (58.5%)**	**46 (56.8%)**	**114**
2.-non-anatomical lesion	**16 (38.1%)**	**12 (40%)**	**9 (22%)**	**30 (37%)**	**67**
3.-anatomical lesion	**0**	**0**	**8 (19.5%)**	**5 (6.2%)**	**13**
Total	**42**	**30**	**41**	**81**	**194**

Table 5.18 Cribra Orbitalia lesion rates by site.

lesions are compared to non-anatomical lesions, there is a statistically significant difference observed [$\chi 2$ (1) = 8.358, p = 0.003]. Chi-squares were also used to establish if there was any statistically significant difference between cribra orbitalia and other lesions recorded. There was no association with posterior maxilla, mandible, zygomatic, fibula and ectocranial at P= >0.01.

5.3.7 Posterior Maxilla

In total there were one hundred and ninety observable left maxillae available for examination. For lesions of the posterior maxilla there was a true prevalence lesion rate observed of 39.5% (75/190), the vast majority of these lesions were thought to be anatomical in nature at 33.7% (64/190). Haslar again displays the highest rate of anatomically linked lesions at 61.9% (26), followed by Plymouth with 30.9% (26), Darwen with 23.7% (9) and 11.5% (3) at Oxford Castle. Chi-square tests were used to determine if there was any difference between the combined Naval and combined Non-Naval collections. When these are compared, there is a statistically significant difference [$\chi 2$ (1) =

10.388, p = 0.0013]. When anatomical lesions are compared to non-anatomical lesions, there was no statistically significant difference observed [p = 0.6831]. Chi-squares were also

Table 5.19 Posterior Maxilla lesion rates by site

Lesion Coding	Darwen	Oxford Castle	Haslar	Plymouth	Total
1.-no lesion	27 (71%)	22(84.6%)	12 (28.6%)	54 (64.3%)	115
2.-non anatomical lesion	2 (5.3%)	1 (3.8%)	4 (9.5%)	4 (4.8%)	11
3.- anatomical lesion	9 (23.7%)	3 (11.5%)	26 (61.9%)	26 (30.9%)	64
Total	38	26	42	84	190

used to establish if there was any statistically significant difference between posterior maxilla and other lesions recorded. There was no association with zygomatic, mandible, cribra orbitalia, fibula and ectocranial at P= >0.01.

5.3.8 Zygomatic

In total there were one hundred and sixty-three zygomatic bones for which observations could be taken. A true prevalence rate of 47.2% (77/163) was recorded for zygomatic lesions, the vast majority of these 39.9% (65/163) were recorded as being anatomical in nature. The highest rate was at Haslar where 74.3% (26) of skeletons with zygomatics present were affected, at Plymouth it 37.7% (23) this was followed by Darwen 24.3% (9) and 23.3% (7) at Oxford Castle. Chi-square tests were used to determine if there was any difference between the combined Naval and combined Non-Naval collections. When these

are compared, there is a statistically significant difference [χ2 (1) = 11.533, p = 0.0007]. When anatomical lesions are compared to non-anatomical lesions, there was no statistically significant difference

Table 5.20 Zygomatic lesion rates by site

Lesion Coding	Darwen	Oxford Castle	Haslar	Plymouth	Total
1.-no lesion	27 (73%)	19(63.3%)	8 (22.9%)	32 (52.5%)	86
2.-non anatomical lesion	1 (2.7%)	4 (13.3%)	1 (2.8%)	6 (9.8%)	12
3.-anatomical lesion	9 (24.3%)	7 (23.3%)	26 (74.3%)	23 (37.7%)	65
Total	37	30	35	61	163

observed [p = 0.2910]. Chi-squares were also used to establish if there was any statistically significant difference between zygomatic and other lesions recorded. There was no association with cribra orbitalia, posterior maxilla, ante-mortem tooth loss, ectocranial, mandible and fibula at P= >0.01.

5.3.9 Mandible

In total 195/284 individuals had mandibles that were present and observable. The true prevalence rates of mandibular lesions was found to be 50.2% (98/195), out of this 36.4% (71/195) of lesions were recorded as anatomically related lesions. The Haslar skeletons displayed the highest rate of pathological anatomical lesions of the mandible with 55% (22) skeletons being affected, followed by Plymouth with 36.1% (31), Darwen with 32.5% (13)

and Oxford Castle with 17.2% (5). Chi-square tests were used to determine if there was any difference between the combined Naval and combined Non-Naval collections. When these are compared, there is a statistically significant difference [$\chi 2$ (1) = 14.418, p = 0.0001].

Table 5.21 Mandible lesion rates by site.

Lesion Coding	Darwen	Oxford Castle	Haslar	Plymouth	Total
1.-no lesion	27 (67.5%)	20 (17.2%)	11 (27.5%)	39 (45.3%)	97
2.-non anatomical lesion	0	4 (13.8%)	7 (17.5%)	16 (18.6%)	27
3.-anatomical lesion	13 (32.5%)	5 (17.2%)	22 (55%)	31 (36.1%)	71
Total	40	29	40	86	195

When anatomical lesions are compared to non-anatomical lesions, there was no statistically significant difference observed [p = 0.4163]. Chi-squares were also used to establish if there was any statistically significant difference between mandible and other lesions recorded. There was no association between ectocranial, ante-mortem tooth loss, zygomatic, cribra orbitalia and posterior maxilla at P= >0.01.

5.4.10 Periodontal Disease

In total 78.2% (222/284) of the combined study group had half or more of their alveolar bone present in order to identify periodontal disease. The true prevalence rate of periodontal disease across all four assemblages was established to be 90.1% (200/222). This is extremely high but not unexpected, a very small proportion of these lesions were recorded as grade 4 with bony reaction, 1.3% (3/222). 4.8 % (2) of these originated from

Haslar and 0.9 % (1) from Plymouth. Chi-square tests were used to determine if there was any difference between the combined Naval and combined Non-Naval collections. When these are compared, there is a statistically significant difference [$\chi 2$ (1) = 10.858, p = 0.0001]. When anatomical lesions are compared to non-anatomical lesions, there was no

Table 5.22 Periodontal Disease lesion rates by site.

Lesion Coding	Darwen	Oxford Castle	Haslar	Plymouth	Total
1.-no lesion	9 (23.1%)	5 (15.2%)	0	8 (7.4%)	22
2.-non anatomical lesion	30 (76.9%)	28 (84.8%)	40 (95.2%)	99 (91.7%)	197
3.- anatomical lesion	0	0	2 (4.8%)	1 (0.9%)	3
Total	39	33	42	108	222

Statistically significant difference observed [p = 0.5579]. Chi-squares were also used to establish if there was any statistically significant difference between periodontal and other lesions recorded. All other lesions were statistically associated at P= >0.01.

5.4.11 Summary of Statistical Analyses of Cranial Lesions

For the most part, the Haslar assemblage recorded the highest true prevalence rates of anatomical scorbutic lesions over all four assemblages, followed by Plymouth. Overall the Darwen and Oxford Castle collections had lower prevalence rates of potential scorbutic cranial lesions, with Oxford Castle rates being the lower of the two overall. The prevalence rates have all been presented as TPR's which controls for the preservation and completion of the skeletal remains that were analysed as part of this study.

5.4 Statistical Analyses of Postcranial Lesions

Eleven elements were assessed in the postcranial skeleton for both left and right sides. The eleven elements are tibia, fibula, femur, humerus, radius, ulna, scapula, clavicle, innominates, feet and hands. Chi-square tests were used to assess if there was a difference between lesions of the left and right sides, when the expected count was less than 5 then Fishers Exact test was used. It was therefore decided to proceed using only the left side as there was no difference observed whether right or left sides were used. The results of the chi-square tests between lesions are presented in the form of a contingency table in Table 5.35. In this table, insignificant results are in red, the rest were found to be significant, for example when femur is tested against ectocranial then a significant difference.

5.4.1 Femur

Fisher's Exact test was used to determine if there was a difference between left and right femoral lesions. There was no difference observed between the two, P value was established at [p=1.000]. The left femur was then selected for statistical analyses. In total there were two hundred and sixty-eight 74.9% (268) skeletons that had a left femur present and observable. The true prevalence rate of femoral lesions was established at 75% (201/268) and anatomically related lesions were 26.9% (72/268). Haslar had the highest rate of anatomically related lesions with 34.9% (15), followed by Plymouth with 32.3% (43) and smaller figures noted at Oxford Castle with 16.7% (6) and Darwen with 14.3% (8). Chi-square tests were used to determine if there was any difference between lesions of the femur in the combined Naval and combined Non Naval collections.

Table 5.23 Femoral lesion rates by site.

Lesion Coding	Darwen	Oxford Castle	Haslar	Plymouth	Total
1.-no lesion	22 (39.3%)	14 (38.9%)	5 (11.6%)	26 (19.6%)	67
2.-non anatomical lesion	26 (46.4%)	16 (44.4%)	23 (53.5%)	64 (48.1%)	129
3.-anatomical lesion	8 (14.3%)	6 (16.7%)	15 (34.9%)	43 (32.3%)	72
Total	56	36	43	133	268

When these are compared, there is a statistically significant difference [$\chi 2$ (1) = 14.918, p = 0.0001]. When anatomical lesions are compared to non-anatomical lesions, there was no statistically significant difference observed [p = 0.0505]. Chi-squares were also used to establish if there was any statistically significant difference between femur and other lesions recorded. There was no statistical association found with tibia, endocranial and hard palate at the level of P= >0.01.

5.4.2 Fibula

Chi-square test was used to establish if there was a difference between left and right fibula lesions. There was no statistically significant difference observed between the two [p=0.5500]. The left fibula was then selected for statistical analyses. In the total combined assemblage 62.6% (224) of individuals had a left fibula that was of research quality. The true prevalence rate of fibular lesions was calculated as 36.6% (82/224), the rate for anatomical lesions was 10.7% (24/224). Haslar had the highest true prevalence rate of anatomical fibular lesions with 17.1% (6) being affected, followed by Plymouth with

skeletons 10.9% (5), Darwen with 10.4% (5) and Oxford Castle with two 5% (2). Chi-square tests were used to determine if there was any difference between lesions of the fibula in the combined Naval and combined Non-Naval collections. When these are compared, there was no statistically significant difference [p = 0.5717]. When anatomical lesions are compared to non-anatomical lesions, there was no statistically significant difference observed [p = 0.4541]. Chi-squares were also used to establish if there was any statistically significant

Table 5.24 Fibula lesion rates by site.

Lesion Coding	Darwen	Oxford Castle	Haslar	Plymouth	Total
1.-no lesion	33 (65.8%)	25 (62.5%)	18 (51.4%)	66 (65.3%)	142
2.-non anatomical lesion	10 (20.8%)	13 (32.5%)	11 (31.4%)	24(23.8%)	58
3.-anatomical lesion	5 (10.4%)	2 (5%)	6 (17.1%)	11 (10.9%)	24
Total	48	40	35	101	224

difference between fibula and other lesions recorded. There was no statistical association found with ectocranial, cribra orbitalia, posterior maxilla and zygomatic at the level of P= >0.01.

5.4.3 Tibia

Chi-square test was used to establish if there was a difference between left and right tibia lesions. There was no difference observed between the two [P=0.4630]. The left tibia was then selected for statistical analyses. In total there was a total of two hundred and fifty three left tibiae represented in the total combined study group. The true prevalence rate

was calculated as 77.9% (197/253), the rate for anatomical lesions was 21.7% (55/253). Of the fifty-five left tibiae with anatomical lesions, thirteen 30.2% (13) of the Haslar were affected, 24.6% (29) of the Plymouth skeletons, 15.7% (8) of Darwen skeletons and 12.2% (5) of the Oxford Castle skeletons. Chi-square tests were used to determine if there was any difference between lesions of the tibia in the combined Naval and combined Non-Naval collections. When these are compared, there was a statistically significant difference at [$\chi 2$ (1) = 7.392, p = 0.0006].When anatomical lesions are compared to non-anatomical lesions,

Table 5.25 Tibia lesion rates by site.

Lesion Coding	Darwen	Oxford Castle	Haslar	Plymouth	Total
1.-no lesion	15 (29.4%)	14 (34.1%)	3 (7%)	24 (20.3%)	56
2.-non anatomical lesion	28 (54.9%)	22 (53.7%)	27 (62.8%)	65 (55.1%)	142
3.-anatomical lesion	8 (15.7%)	5 (12.2%)	13 (30.2%)	29 (24.6%)	55
Total	51	41	43	118	253

there was again no statistically significant difference observed [P = 0.1288]. Chi-squares were also used to establish if there was any statistically significant difference between tibia and other lesions recorded. There was no statistical association found with hard palate, endocranial and femur at the level of P= >0.01.

5.4.4 Humerus

Chi-square test was used to establish if there was a difference between left and right humerus lesions. There was no difference observed between the two, [P=1.000]. The left humerus was then selected for statistical analyses. In total there were two hundred and sixty-seven intact left humeri examined from the study sites. The true prevalence rate was calculated as 12.3% (33/267), the rate for anatomical lesions was 10.5% (28/267).Nearly half of these lesions occurred in the Plymouth assemblage with 10.6% (15) individuals affected. Darwen had 13.3% (6), Oxford Castle with 13.5% (5) and 4.5% (2) at Haslar. Chi-square tests were used to determine if there was any difference between lesions of the humerus in the

Table 5.26 Humerus lesion rates by site.

Lesion Coding	Darwen	Oxford Castle	Haslar	Plymouth	Total
1.-no lesion	38 (84.4%)	31 (83.8%)	42 (95.5%)	123 (87.2%)	234
2.-non anatomical lesion	1 (2.2%)	1 (2.7%)	0	3 (2.1%)	5
3.- anatomical lesion	6 (13.3%)	5 (13.5%)	2 (4.5%)	15 (10.6%)	28
Total	45	37	44	141	267

combined Naval and combined Non-Naval collections. When these are compared, there was no statistically significant difference at [P=0.3130]. When anatomical lesions are compared to non-anatomical lesions, there was no statistically significant difference observed [P = 1.000]. Chi-squares were also used to establish if there was any statistically significant

difference between humerus and other lesions recorded. There was no statistical association found with radius, ulna, innominate and foot at the level of P= >0.01.

5.4.5 Radius

Chi-square test was used to establish if there was a difference between left and right radius lesions. There was no statistically significant difference observed between the two [P=0.5006]. The left radius was then selected for statistical analyses. In the total combined assemblage two hundred and fifty-five (71.2%) skeletons had a left radius present and observable. The true prevalence rate was calculated as 6.7% (17/255), the rate for anatomical lesions was 5.9% (15/255). The prevalence of anatomical lesions was spread across all four assemblages, 9.5% (4) of the Haslar assemblage was affected, 0.8% (5) of Plymouth, Darwen and Oxford Castle was equally affected with three skeletons each. Chi-

Table 5.27 Radius lesion rate by site.

Lesion Coding	Darwen	Oxford Castle	Haslar	Plymouth	Total
1.-no lesion	39 (92.9%)	35 (89.7%)	38 (90.5%)	126 (95.4%)	238
2.-non anatomical lesion	0	1 (2.6%)	0	1 (3.8%)	2
3.-anatomical lesion	3 (7.1%)	3 (7.7%)	4 (9.5%)	5 (0.8%)	15
Total	42	39	42	132	255

square tests were used to determine if there was any difference between lesions of the radius in the combined Naval and combined Non-Naval collections. When these are compared, there was no statistically significant difference at [P=0.4231]. When anatomical

lesions are compared to non-anatomical lesions, there was no statistically significant difference observed [P = 1.000]. Chi-squares were also used to establish if there was any statistically significant difference between radius and other lesions recorded. There was no statistical association found with humerus, ulna, innominate, scapula, clavicle and hand at the level of P= >0.01.

5.4.6 Ulna

Chi-squared test was used to establish if there was a difference between left and right ulna lesions. There was no difference observed between the two [P=0.8754]. The left ulna was then selected for statistical analyses. In the total combined assemblage, 70.9% (254/358) skeletons had a left ulna that was present and observable. The overall true prevalence rate was calculated as 9.1% (23/254), the rate for anatomical lesions was 7.5% (19/254). Out of these 19 skeletons, there was four 9.3% (4) of the total Haslar skeletons affected,

Table 5.28 Ulna lesion rates by site.

Lesion Coding	Darwen	Oxford Castle	Haslar	Plymouth	Total
1.-no lesion	36 (87.8%)	31 (83.8%)	39 (90.7%)	125(94%)	231
2.-non anatomical lesion	0	2 (5.4%)	0	2 (1.5%)	4
3.- anatomical lesion	5 (12.2%)	4 (10.8%)	4 (9.3%)	6 (4.5%)	19
Total	41	37	43	133	254

12.2% (5) of the total Darwen assemblage, 4.5% (6) of the total Plymouth skeletons and 10.8% (4) of the total Oxford Castle skeletons. Chi-square tests were used to determine if

there was any difference between lesions of the ulna in the combined Naval and combined Non-Naval collections. When these are compared, there was no statistically significant difference at [P=0.0943]. When anatomical lesions are compared to non-anatomical lesions, there was no statistically significant difference observed [P = 1.000]. Chi-squares were also used to establish if there was any statistically significant difference between ulna and other lesions recorded. There was no statistical association found with humerus, radius, innominate and scapula at the level of P= >0.01.

5.4.7 Innominate

Fisher's Exact test was used to establish if there was a difference between left and right innominate lesions. There was no difference observed between the two [p=1.000]. The left innominate was then selected for statistical analyses. In the total combined assemblage of 66.5% (238/358) of skeletons had a present and observable left innominate. The true prevalence rate was calculated as 10.1% (24/238), the rate for anatomical lesions was 8.8% (21/238). Out of the total of twenty-one left innominates affected by anatomical

Table 5.29 Innominate lesion rates by site

Lesion Coding	Darwen	Oxford Castle	Haslar	Plymouth	Total
1.-no lesion	39 (92.9%)	30 (90.9%)	28 (65.1%)	117 (97.5%)	214
2.-non anatomical lesion	0	1 (3%)	1 (2.4%)	1 (0.8%)	3
3.-anatomical lesion	3 (7.1%)	2 (6.1%)	14 (32.5%)	2 (1.7%)	21
Total	42	33	43	120	238

lesions, the majority originated from Haslar, of which 32.5% (14) of the skeletons displayed these lesions, only 1.7% (2) skeletons from Plymouth and Oxford Castle 6.1% (2) showed these lesions and 7.1% (3) from Darwen. Chi-square tests were used to determine if there was any difference between lesions of the innominate in the combined Naval and combined Non-Naval collections. When these are compared, there was no statistically significant difference at [p=0.6437]. When anatomical lesions are compared to non-anatomical lesions, there was no statistically significant difference observed [p = 0.7216]. Chi-squares were also used to establish if there was any statistically significant difference between innominate and other lesions recorded. There was no statistical association found with humerus, radius, ulna and scapula at the level of P= >0.01.

5.4.8 Scapula

Chi-squared test was used to establish if there was a difference between left and right scapula lesions. There was no statistical difference observed between the two [p=0.8347].

Table 5.30 Scapula lesion rates by site.

Lesion Coding	Darwen	Oxford Castle	Haslar	Plymouth	Total
1.-no lesion	39 (95.1%)	29 (82.9%)	40 (97.6%)	114 (98.3%)	222
2.-non anatomical lesion	0	0	0	0	0
3.- anatomical lesion	2 (4.9%)	6 (17.1%)	1 (2.4%)	2 (1.7%)	11
Total	41	35	41	116	233

The left scapula was then selected for statistical analyses. In the total combined assemblage 65.1% (233/358) of skeletons had a present and observable left scapula. The true prevalence rate was calculated as 11/233 (4.7%), the rate for anatomical lesions was the same. When calculated by site, Oxford Castle demonstrated the highest true prevalence rate with 17.1% (6) of skeletons indicating anatomical scapula lesions, only 1.7% (2) were affected at Plymouth, 4.9% (2) at Darwen and 2.4% (1) at Haslar. Chi-square tests were used to determine if there was any difference between lesions of the scapula in the combined Naval and combined Non-Naval collections. When these are compared, there was a statistically significant at [$\chi 2$ (1) =8.451, p=<0.01]. When anatomical lesions are compared to non-anatomical lesions, there was no statistically significant difference observed [P = 1.000]. Chi-squares were also used to establish if there was any statistically significant difference between scapula and other lesions recorded. There was no statistical association found with radius, ulna, innominate, clavicle and hand at the level of P= >0.01, the rest were correlated.

5.4.9 Clavicle

Chi-square test was used to establish if there was a difference between left and right clavicle lesions. There was no statistically significant difference observed between the two [p=0.3472]. The left clavicle was then selected for statistical analyses. In the total combined assemblage 65.4% (234/358) of skeletons had a present and observable left clavicle. The combined true prevalence rate was calculated as 3% (7/234), the rate for anatomical lesions was the same. When calculated by site, Darwen demonstrated the highest overall true prevalence rate with (3.3%) of skeletons affected, Plymouth had three (1.6%). Both Oxford Castle and Haslar had only one skeleton each affected. Chi-square tests were used to determine if there was any difference between lesions of the clavicle in the combined

Table 5.31 Clavicle lesion rates by site.

Lesion Coding	Darwen	Oxford Castle	Haslar	Plymouth	Total
1.-no lesion	41 (95.3%)	33 (97.1%)	39 (97.5%)	114 (97.4%)	227
2.-non anatomical lesion	0	0	0	0	0
3.-anatomical lesion	2 (4.6%)	1 (2.9%)	1 (2.5%)	3 (2.6%)	7
Total	43	34	40	117	234

Naval and combined Non-Naval collections. When these are compared, there was no statistically significant difference [P = 0.6867]. When anatomical lesions are compared to non-anatomical lesions, there was no statistically significant difference observed [P = 1.000]. Chi-squares were also used to establish if there was any statistically significant difference between clavicle and other lesions recorded. All lesions were associated except for radius, hand and scapula at the level of P= >0.01.

5.4.10 Foot

Chi-square test was used to establish if there was a difference between left and right foot lesions. There was no difference observed between the two [P=0.4321]. The left foot was then selected for statistical analyses. In the total combined assemblage 56.1% (201/358) of skeleton had the majority of their foot bones intact and observable. The overall true prevalence rate was calculated as 18.9% (38/201), the rate for anatomical lesions was 17.4% (35/201). When calculated by site, Plymouth had the highest true prevalence rate with

twenty 21.7% (20) skeletons with anatomical lesions of the foot, Haslar had 16.1% (5) skeletons.

Table 5.32 Foot lesion rates by site.

Lesion Coding	Darwen	Oxford Castle	Haslar	Plymouth	Total
1.-no lesion	36 (87.8%)	31 (83.8%)	26 (83.9%)	70 (76.1%)	163
2.-non anatomical lesion	0	1 (2.7%)	0	2 (2.2%)	3
3.-anatomical lesion	5 (12.2%)	5 (13.5%)	5 (16.1%)	20 (21.7%)	35
Total	41	37	31	92	201

Both Darwen and Oxford Castle had five skeletons each affected. Chi-square tests were used to determine if there was any difference between lesions of the foot in the combined Naval and combined Non-Naval collections. When these are compared, there was no statistically significant difference [P = 0.1977]. When anatomical lesions are compared to non-anatomical lesions, there was no statistically significant association observed [P = 1.000]. Chi-squares were also used to establish if there was any statistically significant association between foot lesions and other variables recorded. All other lesions were associated except humerus at the level of P= >0.01.

5.4.11 Hand

Chi-square test was used to establish if there was a difference between left and right hand lesions. There was no statistically significant difference observed between the two

[P=1.000].The left hand was then selected for statistical analyses. Looking at the overall combined collection, there were 58.9% (211/358) left hands available for analysis. The true prevalence rate of lesions was calculated as 2.8% (6/211), the rate for anatomical lesions was the same across the combined assemblages. When sites were looked at individually, Plymouth presented with three individuals affected, and then one each exhibited lesions at Haslar, Darwen and Oxford Castle. Chi-square tests were used to determine if there was any association between lesions of the hand in the combined Naval and combined Non-Naval collections. When these are compared, there was no statistically significant difference [P = 1.000]. When anatomical lesions are compared to non-anatomical lesions, there was no statistically significant difference observed [P = 1.000]. Chi-squares were also used to establish if there was any statistically significant association between hand and other lesions recorded. There was no statistical association with radius, clavicle and scapula at the level of P= >0.01.

Table 5.33 Hand lesion rate by site

Lesion Coding	Darwen	Oxford Castle	Haslar	Plymouth	Total
1.-no lesion	41 (97.6%)	33 (97.1%)	30 (96.8%)	101 (97.1%)	205
2.-non anatomical lesion	0	0	0	0	0
3.- anatomical lesion	1 (2.4%)	1 (2.9%)	1 (3.2%)	3 (2.9%)	6
Total	42	34	31	104	211

5.5 Diagnosis of Scurvy in Skeletal Material

From examination of the literature review and statistical analysis, it was established that some of these lesions were more indicative of scurvy than others and some can even be considered to be virtually pathognomonic scorbutic traits in adults, these include anatomical lesions on the femur, sphenoid, posterior maxilla, scapula, endocranial and mandible (Geber et al. 2012, 515; Crist et al. 2005). It was decided to use statistical analysis to test all non-specific scorbutic indicators against these primary diagnostic lesions. This was done to establish the diagnostic use of all non-specific lesions. Lesions that were associated with at least five out of six of these primary diagnostic lesions were then considered to be secondary diagnostic lesions. These secondary diagnostic lesions that were proven to correlate with the primary lesions were anatomically related lesions of the foot, humerus, ulna, radius, ectocranial, hand, clavicle, innominate and fibula. Periodontal disease correlated with all the primary lesions, however this was disregarded due to its lack of a definite scorbutic aetiology. The rest of the lesions were disregarded due to lack of correlation.

When scurvy diagnosis is examined on the level of the individual skeleton, it was decided that for a positive diagnosis to be made, there had to be a combination of primary diagnostic and secondary diagnostic lesions present. For a diagnosis of probable scurvy to be made then three primary lesions had to be present and at least one secondary lesion, for a diagnosis of possible scurvy, then there had to be two primary lesions present and at least two secondary lesions. However if any one skeleton showed four or more primary diagnostic lesions and one or more secondary lesions, then a diagnosis of probable scurvy was assumed. These criteria were chosen to reduce the risk of over diagnosis of scurvy in

skeletons being assessed for scurvy. These results are presented in Table 5.34. When the true prevalence rate of individuals affected by scurvy was calculated, it was found to be was five (7.9%) in Oxford Castle, followed by Darwen with thirteen (16.4%), Plymouth with forty-eight (20.6%) and Haslar is by far the highest with thirty-three (66.7%) of the total collection displaying diagnostic scorbutic lesions.

Table 5.34 Calculated scurvy rates for each study assemblage.

	Oxford Castle	Darwen, Redearth	Haslar	Plymouth
Probable Scurvy No.	4/63 (6.3%)	9/61 (14.8%)	22/45 (48.9%)	34/189 (18%)
Possible Scurvy No.	1/63 (1.6%)	1/61 (1.6%)	8/45 (17.8%)	5/189 (2.6%)
Totals	5/63 (7.9%)	10/61 (16.4%)	30/45 (66.7%)	39/189 (20.6%)

In total, there are more total scurvy cases in the combined Naval assemblage when compared to the combined Non-Naval assemblage, this can be seen in Figure 5.3. In total,

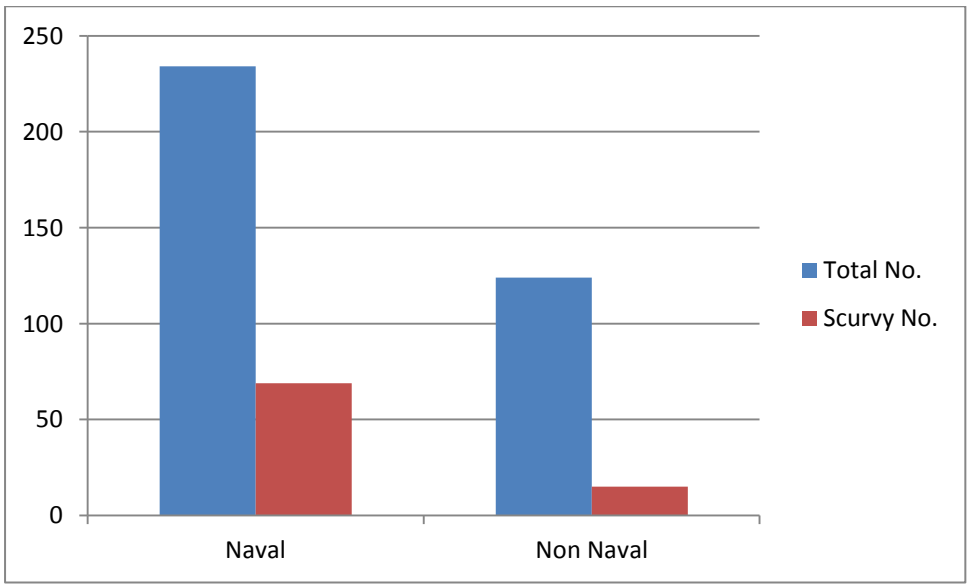

Figure 5.3 Graph showing total combined number of skeletons with and without scurvy in combined Naval and Non-Naval assemblages

	EN	EC	AM	HP	SP	CR	PM	ZY	MN	PD	FE	FI	TI	HU	UL	RA	CL	FT	HN	IN	SC
EN	-	<0.01	<0.01	0.145	<0.01	<0.01	<0.01	<0.01	<0.01	<0.01	0.394	<0.01	0.844	<0.01	<0.01	<0.01	<0.01	<0.01	<0.01	<0.01	<0.01
EC	<0.01	-	<0.01	<0.01	<0.01	0.239	0.137	1.000	0.559	<0.01	<0.01	0.022	<0.01	<0.01	<0.01	<0.01	<0.01	<0.01	<0.01	<0.01	<0.01
AM	<0.01	0.017	-	<0.01	0.267	<0.01	<0.01	0.038	0.114	<0.01	<0.01	<0.01	<0.01	<0.01	<0.01	<0.01	<0.01	<0.01	<0.01	<0.01	<0.01
HP	0.145	<0.01	<0.01	-	0.084	<0.01	<0.01	<0.01	<0.01	<0.01	0.527	<0.01	0.190	<0.01	<0.01	<0.01	<0.01	<0.01	<0.01	<0.01	<0.01
SP	<0.01	<0.01	0.267	0.084	-	<0.01	<0.01	<0.01	<0.01	<0.01	<0.01	<0.01	<0.01	<0.01	<0.01	<0.01	<0.01	<0.01	<0.01	<0.01	<0.01
CR	<0.01	0.239	<0.01	<0.01	<0.01	-	0.755	0.284	0.083	<0.01	<0.01	0.332	<0.01	<0.01	<0.01	<0.01	<0.01	<0.01	<0.01	<0.01	<0.01
PM	<0.01	0.137	<0.01	<0.01	<0.01	0.755	-	0.161	0.040	<0.01	<0.01	0.359	<0.01	<0.01	<0.01	<0.01	<0.01	<0.01	<0.01	<0.01	<0.01
ZY	<0.01	1.000	0.038	<0.01	<0.01	0.284	0.161	-	0.596	<0.01	<0.01	0.037	<0.01	<0.01	<0.01	<0.01	<0.01	<0.01	<0.01	<0.01	<0.01
MN	<0.01	0.559	0.114	<0.01	<0.01	0.083	0.040	0.596	-	<0.01	<0.01	<0.01	<0.01	<0.01	<0.01	<0.01	<0.01	<0.01	<0.01	<0.01	<0.01
PD	<0.01	<0.01	<0.01	<0.01	<0.01	<0.01	<0.01	<0.01	<0.01	-	<0.01	<0.01	<0.01	<0.01	<0.01	<0.01	<0.01	<0.01	<0.01	<0.01	<0.01
FE	0.394	<0.01	<0.01	0.527	<0.01	<0.01	<0.01	<0.01	<0.01	<0.01	-	<0.01	0.470	<0.01	<0.01	<0.01	<0.01	<0.01	<0.01	<0.01	<0.01
FI	<0.01	0.022	<0.01	<0.01	<0.01	0.332	0.359	0.037	<0.01	<0.01	<0.01	-	<0.01	<0.01	<0.01	<0.01	<0.01	<0.01	<0.01	<0.01	<0.01
TI	0.844	<0.01	<0.01	0.190	<0.01	<0.01	<0.01	<0.01	<0.01	<0.01	0.470	<0.01	-	<0.01	<0.01	<0.01	<0.01	<0.01	<0.01	<0.01	<0.01
HU	<0.01	<0.01	<0.01	<0.01	<0.01	<0.01	<0.01	<0.01	<0.01	<0.01	<0.01	<0.01	<0.01	-	0.258	0.036	<0.01	0.067	<0.01	0.481	<0.01

UL	<0.01	<0.01	<0.01	<0.01	<0.01	<0.01	<0.01	<0.01	<0.01	<0.01	<0.01	<0.01	<0.01	0.258	-	0.328	<0.01	<0.01	<0.01	0.759	0.074
RA	<0.01	<0.01	<0.01	<0.01	<0.01	<0.01	<0.01	<0.01	<0.01	<0.01	<0.01	<0.01	<0.01	0.036	0.328	-	0.091	<0.01	0.083	0.193	0.437
CL	<0.01	<0.01	<0.01	<0.01	<0.01	<0.01	<0.01	<0.01	<0.01	<0.01	<0.01	<0.01	<0.01	<0.01	<0.01	0.091	-	<0.01	1.000	<0.01	0.317
FT	<0.01	<0.01	<0.01	<0.01	<0.01	<0.01	<0.01	<0.01	<0.01	<0.01	<0.01	<0.01	<0.01	0.067	<0.01	<0.01	<0.01	-	<0.01	<0.01	<0.01
HN	<0.01	<0.01	<0.01	<0.01	<0.01	<0.01	<0.01	<0.01	<0.01	<0.01	<0.01	<0.01	<0.01	<0.01	<0.01	0.083	1.000	<0.01	-	<0.01	0.333
IN	<0.01	<0.01	<0.01	<0.01	<0.01	<0.01	<0.01	<0.01	<0.01	<0.01	<0.01	<0.01	<0.01	0.481	0.759	0.193	<0.01	<0.01	<0.01	-	0.034
SC	<0.01	<0.01	<0.01	<0.01	<0.01	<0.01	<0.01	<0.01	<0.01	<0.01	<0.01	<0.01	<0.01	<0.01	0.074	0.437	0.349	<0.01	0.333	0.034	-

Table 5.35 Contingency Table showing the chi-squared results for comparison of all lesions. Insignificant results are shown in red. EN is endocranial, EC is ectocranial, AM is ante-mortem tooth loss, HP is hard palate, SP is sphenoid, CR is cribra orbitalia, PM is posterior maxilla, ZY is zygomatic, MN is mandible, PD is periodontal disease, FE is femur, FI is fibula, TI is tibia, HU is humerus, UL is ulna, RA is radius, CL is clavicle, FT is foot, HN is hand, IN is innominate, SC is scapula.

29.5% of the Naval assemblage were recognised to show signs of scurvy and 12.1% of the Non-Naval assemblage show signs of scurvy.

Cases of probable scurvy was found across all four assemblages, in the next subchapter, a number of case studies will be discussed in order to give a better idea of the lesion suite that needs to be present in order to diagnose scurvy.

5.5.1 Scurvy Case Studies

In the following subsection three case studies from the combined study group will be presented. This has been done to show the potential manifestation of scurvy on the level of each individual skeleton. For each skeleton, standard osteobiographical data will be presented along with all possible scorbutic lesions present.

5.5.1.1 Darwen SK206

SK206 was a young male aged between 18 to 20 years of age at death. This individual presented with multiple element active periostitis. Patches of new periosteal reaction was present bilaterally on the femora, tibiae, fibulae, scapulae, humeri, ulnae, radii, os coxae

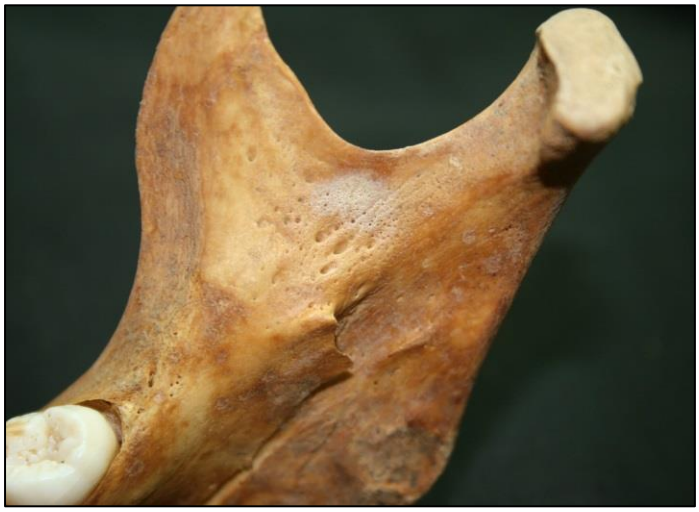

Figure 5.4 Mandible of SK206 showing abnormally porous are adjacent to the mylohyoid.

and calcaneii. All of these lesions were anatomically linked and were very clearly delineated by muscle attachments. Non bilateral lesions were also present on the left clavicle, a left fifth metatarsal, along with an unidentified left metacarpal. The internal mandible adjacent to the mylohyoid foramen was abnormally porous and can be seen in Figure 5.4, this was seen on both sides of the mandible. Extensive endocranial lesions were also noted.

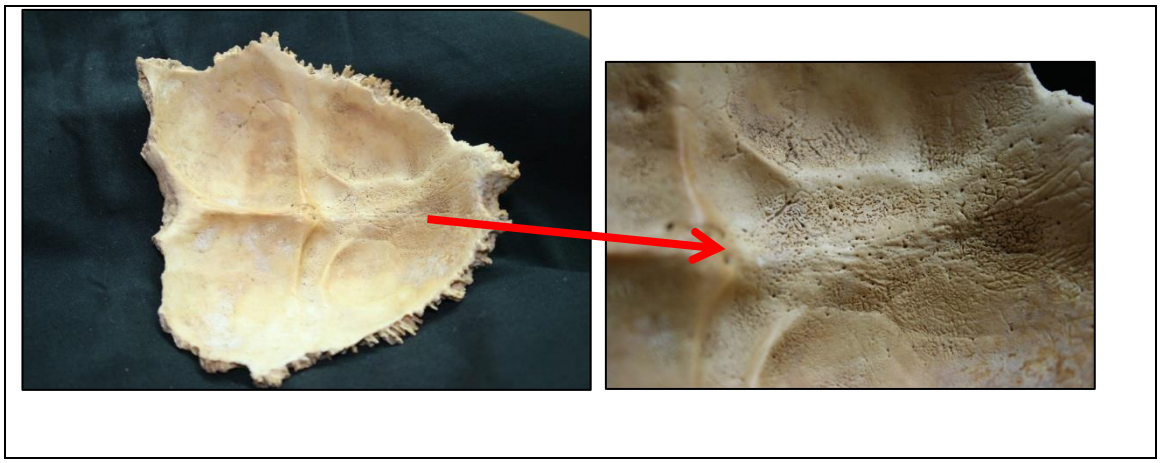

Figure 5.5 Endocranial surface of SK206 showing Type 3 endocranial lesions.

These lesions were classed as Type 3 endocranial reaction and consisted of capillary type lesions, these were observed on the internal surface of the frontal, parietals, occipital and temporal. Lesions on the internal occipital can be seen in Figure 5.5. Active ectocranial porosity was noted and this was distinctly differentiated by the aponeurosis. Type 4 active cribra orbitalia was also noted in both eye sockets. This combination of primary and secondary lesions in this individual was decided to be a case of probable scurvy.

5.5.1.2 Plymouth SK520

Skeleton 520 represented the remains of an older adult male aged between 40 and 50 years of age at death. In total twelve elements of this individual presented with periosteal reaction. Both the left and right femora, left tibia, both humeri, both radii, both ulnae along

with the left foot and both hands. Both the femora displayed new periosteal bone running alongside the linea aspera. The right tibia was not present but the left tibia showed a

Figure 5.6 Left tibia of SK520 showing possible ossified hematoma

possible ossified haematoma which can be seen in Figure 5.6. The author was unsure if this lesion represented a defined area of thick periosteal growth or a possible ossified haematoma. The lesion on the tibia is clearly defined by the soleal line. All of the lesions in this individual seemed to be in an advanced stage of healing, none of the periosteal bone was new and 'fluffy' but was more structured and lamellar. Possible lesions of scurvy were also noted in the cranial region. Some ectocranial porosity was noted along with ante-

Figure 5.7 Left sphenoid of SK520 showing abnormal porosity

mortem tooth loss. Sphenoid lesions were also noted and both zygomatic bones were noted to be particularly porous on the anterior surfaces. The left sphenoid is shown in Figure 5.7. The cranial vault was intact; therefore the endocranium could not be viewed. Due to the combination of cranial and postcranial lesions, this individual was decided to be a case of probable case of scurvy from the Plymouth assemblage.

5.5.1.3 Haslar SK212

Haslar SK212 represents the remains of a young man aged between 20 and 25 years of age at death. In total thirteen elements of this individuals postcranial skeleton were affected by active periosteal lesions. Both tibiae were affected along with both femora, fibulae, radii and Os coxae, right ulna and elements of both left and right feet. All of the periosteal reaction present looked immature, woven and porous in nature. An example of this very immature periosteal bone on the left tibia is shown in Figure 5.8, this was typical of the kind of reaction that was found throughout SK212.

Figure 5.8 Non-anatomical immature periosteal reaction on the left tibia of SK212.

The lesions on both tibiae were not considered to be anatomical lesions, however anatomical lesions adjacent to the linea aspera of both femora were noted. Periosteal lesions on both calcaneii were also noted and can be seen in Figure 5.9.

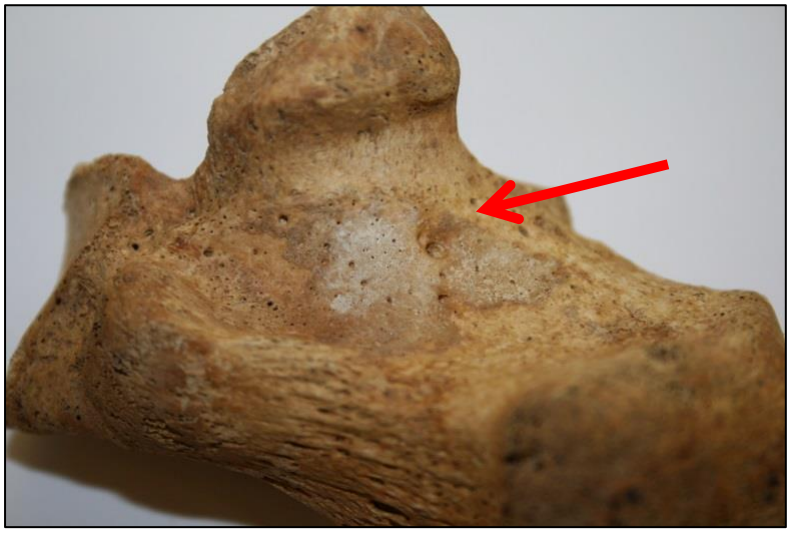

Figure 5.9 Periosteal reaction on calcaneus of SK212.

Cranial lesions were also noted in SK212, ectocranial porosity which was limited by the lines of the aponeurosis was noted, along with grade 2 endocranial lesions, porosity of the zygomatics and quite marked abnormal porosity of the hard palate. The combination of these lesions meant that this skeleton was decided upon as a case of probable scurvy.

5.6 Summary of Scorbutic Lesion Analysis

Every scorbutic lesion recorded was statistically analysed to determine its use in the identification of scurvy in adult skeletal material. These were then were divided into those lesions that could be considered to be primary diagnostic lesions of scurvy and those that were secondary diagnostic lesions and those that were completely non-specific. These were then used to establish the overall rates of scurvy for each study group. In addition to overall rates of scurvy, three case studies of scurvy were also presented. In the next chapter each

lesion, will be discussed from the viewpoint of the physiological manifestation of scurvy and the results of the statistical analysis.

Chapter 6: Discussion

6.1 Introduction

The results presented in the previous chapter suggest some interesting inferences about the usefulness of macroscopic lesions in the diagnosis of scurvy in skeletal material. In this section, the results presented in chapter five will be examined. This section will also bring together medical and historical evidence for the manifestation of scurvy in the human body. This is important, as an effort will be made to establish the exact cause of possible scorbutic bony lesions by examining lesion patterning and occurance and linking that to the physiological expression of scurvy. Potential differential diagnoses for the lesions will also be discussed. This will help to establish the reliability of using skeletal lesions to identify scurvy in skeletal assemblages. This section will also attempt to explain how scorbutic lesions can be identified based on their distinct features and characteristics and how they can be differentiated from the lesions of other specific and non-specific disease processes. The results of the statistical analysis of the scorbutic variables will also be examined and how this can be used to determine the reliability of the lesions in the diagnosis of scurvy. The last section will examine the prevalence rates of scurvy for each collection and discuss if the Naval assemblages were more affected by scurvy than the comparative groups.

6.2 Cranial Scorbutic Lesion Discussion

6.2.1 Introduction to Cranial Scorbutic Lesion Discussion

There were ten regions in the cranial area that were identified as being fundamental to the musculoskeletal manifestation of scurvy. These ten are endocranial, ectocranial, hard palate, ante-mortem tooth loss, cribra orbitalia, sphenoid, posterior maxilla, zygomatic region, mandible and periodontal disease. It is apparent that the presence/absence of one

of these lesions alone is not enough to confirm the presence of scurvy. Therefore it was decided that a number of lesions must be present in order for scurvy to be diagnosed. The following section will present each lesion separately, along with how it can be caused by scurvy, how exactly it manifests and the results of statistical analysis. This then will then be used to determine the use of these lesions in the diagnosis of scurvy.

6.2.1.1 Ectocranial Lesions

Ectocranial lesions were recorded in all of the study assemblages. Most pertinent to this study are those 'orange peel' lesions that were defined by the area of the galea aponeurotica, i.e. those lesions that were scored a '3'. Most of these lesions occurred in Haslar and Plymouth and a smaller number in Darwen and Plymouth. In Haslar 88.6% (39/44) of skeletons displayed anatomically related ectocranial lesions and in Plymouth 74% (71/96) were affected. The Non- Naval groups presented with lower rates, in Darwen 28% (14/50 of skulls were affected and in Oxford Castle 35.9% (14/39) were affected. No other comparative studies could be found where a TPR of orange peel lesions could be found as these lesions are not well known in the paleopathological literature. Ectocranial pathologies were associated with five out of six of the primary diagnostic lesions established for adult scurvy. Due the higher rate of endocranial lesions in the Naval assemblages, the aetiological explanation for this lesion in scorbutic individuals and the strong association with the other primary diagnostic features of scurvy, ectocranial was deemed to be a primary secondary feature of scurvy in adults.

6.2.1.2 Ante-Mortem Tooth Loss

Ante mortem tooth loss was commonly recorded in this study. Ante-mortem tooth loss has also been examined in some other paleopathological studies of scurvy. In the Greenwich Naval assemblage, the rate of ante-mortem tooth loss was 44.73% which is quite high, but this population is aged. In the study assemblages, the combined rate of ante-mortem tooth loss across all four sites was 58.4%. The Greenwich study demonstrates the problems inherent in using AMTL to aid in scurvy diagnosis, as this population also suffered from numerous other causes of AMTL. Therefore it is not possible to ascribe these high AMTL rates to scurvy. Overall the combined assemblage had 58.4% (129/221) individuals that suffered from AMTL. Darwen had a particularly high TPR of 79.5% but it is thought that this is due to the old age of this population and the high prevalence of edentulous individuals, followed by Plymouth with 55%, and then similar rates were recorded at Haslar (47.6%) and Oxford Castle (42.8%). It is thought that in the Post Medieval period, there is a general increase in AMTL levels due to the greater availability of sugar and refined flour, leading to caries and tooth loss (Roberts and Cox 2003, 396). When AMTL was tested against other reliable cranial and postcranial lesions, there was some association. However due to multiple causation factors, AMTL was disregarded as not being a reliable indicator of adult scurvy.

6.2.1.3 Endocranial Lesions

When examining endocranial lesions from the study sites using statistical analysis, it was found that 41% (94/229) individuals exhibited anatomically related proliferative or porous endocranial lesions. Of the 14 individuals that were graded as '2', the majority of these were attributed to TB as they occurred with pathognomonic postcranial TB lesions such as lytic

spinal and rib lesions. Out of the 94 individuals with anatomical endocranial lesions, 58 of these were in Plymouth, followed by 19 at Haslar, 10 at Darwen and 7 at Oxford Castle. These rates are almost certainly an underestimation as the occurance of this lesion could not be assessed if the skull was intact. This was a particular problem with Darwen and Haslar because they had a high proportion of intact skulls. The only other study of adult scurvy that also recorded endocranial lesions was Geber and Murphy (2012); they recorded parietal endocranial lesions at 2.53% (9/356). When endocranial lesions were tested against the other primary diagnostic lesions, there was a significant association with four of these. It was decided that endocranial lesions should be classified as a primary diagnostic feature of scurvy due to the statistical analysis, the aetiology of these lesions and the pattering of the lesions within the endocranium.

6.2.1.4 Sphenoid

Few other paleopathological studies have considered sphenoid lesions in adult remains. However three will be examined here. Geber and Murphy (2012) record an overall rate of 4.64% (13/280) of lesions of the greater wing of the sphenoid and 0.71% (2/282) of the lesser wing. Boston et al. (2008) in the Greenwich sailors record a crude prevalence rate of 12.37% of the male population (Boston et al. 2008, 57). Crist et al. (2005) also examined sphenoidal lesions in the St. Croix skeletons and recorded a true prevalence 4/7 (57.1%) of observable skeletons displaying abnormal porosity and new bone. When sphenoid lesions in this study group were examined, they were found to occur in (125/195) of the total combined assemblage in this study that had sphenoid bones present. Again, Haslar 32/43 (74.4%) and Plymouth 51/84 (60.7%) presented with the highest true prevalence rates. In the Darwen skeletons, 15/39 (38.5%) exhibited anatomically related sphenoid lesions, with

5/29 (17.3%) at Oxford Castle. The sphenoid was significantly associated with all other primary diagnostic cranial lesions and one primary postcranial indicator and was therefore considered to be a primary diagnostic indicator of scurvy in adult bones.

6.2.1.5 Hard Palate Lesions

Hard palate lesions were recorded as being present in 72.4% of the total assemblage examined as part of this study, however only 24.1% (49/203) of these lesions displayed evidence of remodelling and significant abnormal porosity of '2' as presented in the methodology chapter. The highest rate of these lesions occurred in the Naval assemblages; Plymouth with 26/92 (28.3%) individuals affected and 18/41 (43.9%) at Haslar. Oxford Castle had 4/31 (12.9%) and Darwen 1/39 (2.6%) affected. After an extensive literature review, only one other site has examined the prevalence of hard palate lesions in scorbutic dry adult bone. Crist et al. (2005) found that 44% (7/16) of individuals showed areas of remodelling and raised and abnormal porosity across the hard palate. Hard palate lesions in this study correlated significantly with four of the primary diagnostic lesions and therefore were not considered to be an indicator of scurvy. However this lesion may be promising in the study of scurvy and remodelled and abnormal lesions of the hard palate should be recorded as a non-specific scorbutic lesion.

6.2.1.6 Posterior Maxilla

Posterior maxilla lesions have been rarely recorded as part of the suite of cranial scorbutic lesions in adult skeletal remains. In Geber and Murphy (2012, 519) the posterior maxilla was considered to be a definite indicative lesion and it occurred in 6/339 (1.77%) of adult individuals. Scorbutic posterior maxilla lesions occurred in 64/190 (33.7%) of adults

examined as part of this research. Again the majority of these lesions occurred in Plymouth (26/92) and Haslar (18/41). In Oxford Castle just 4/31 and in Darwen only 1/39 was recorded. Posterior maxilla was significantly associated with five other primary diagnostic lesions. Due to this and the clear link between the posterior maxilla and scorbutic haemorrhaging of the muscles of mastication, it was classified as a primary diagnostic cranial indicator of adult scurvy.

6.2.1.7 Periodontal Disease

In archaeological studies, periodontal disease has been used occasionally to aid in the identification of scurvy (Van der Merwe et al. 2010b; Maat et al. 1984; Crist et al. 2005). Van der Merwe et al. (2010b) used periodontal disease as one of the three indicators of scurvy in their study, in that group 43.7% (7/16) of individuals with scurvy also had periodontal disease. In this study, extremely high levels of periodontal disease were recorded as 200/222 (90.1%) of the combined group showed signs of periodontal disease. However, only three of these cases could be linked to scorbutic haemorrhaging and were manifested as periosteal bone reaction. All three of these cases were found in the Naval assemblages. There was an excellent association between periodontal and other lesions. However due to the multi-causal aetiology of periodontal disease and the lack of a definite scorbutic indicator it was dismissed as being a non-specific indicator of scurvy.

6.2.1.8 Zygomatic

In the course of this research abnormal porosity and new bone growth adjacent to the infraorbital foramen of the zygomatic was noted frequently and totalled 65/163 (39.9%) of all present and complete left zygomatic bones. The majority of these lesions did occur in

Haslar 74.3% (26/35) and Plymouth 37.7% (23/61), with 24.3% (9/37) recorded at Darwen and 23.3% (7/30) at Oxford Castle. There are no other studies that have conducted research on the potential of scorbutic zygomatic lesions, so it is impossible to make any comparison with other sites. Trauma could be a causative factor for these lesions, but only a small number of fractured zygomatic (two) were noted in the combined assemblage so it is unlikely. It is most probable that the lesions could be caused by scurvy and possibly minor trauma. In the Navy, inter-personal violence was commonplace; "quarrels and fighting" were commonplace below deck (Lavery 2010, 274). Trauma to the zygomatic is commonly implicated in examples of inter-personal violence. Lesions of the zygomatic were linked with only four out of six of the primary diagnostic indicators and therefore were not classified as a secondary diagnostic lesion of adult scurvy but can be considered to be a possible non-specific indicator of scurvy.

6.2.1.9 Mandible

In total 71/195 (36.4%) individuals in the combined assemblage presented with anatomical mandibular lesions. Again this type of lesion occurred most commonly in the Naval assemblages, the rate at Haslar was 55% (22/40) and 36% (31/86) at Plymouth, the non-Naval collections had lower rates, Darwen had a rate of 32.5% (13/40), followed by Oxford Castle with 17.2% (5/29). One other paleopathological study has studied this phenomenon, Crist et al. (2005) who found that five out of seventeen individuals in their study presented with reactive new bone near the mental foramen. The Naval rates are quite high, it is interesting to consider the diet of the Navy which consisted primarily of tough preserved foods such as salted meat and hardtack. This may have had adverse consequences on the muscles of mastication and the chewing of tough foods may of have the effect of

exacerbating haemorrhaging and inflammation in this area. Mandibular lesions can be clearly linked to the physiological manifestation of scurvy and were classified as a pathognomonic lesion by Geber et al. (2012). As such, mandibular lesions were classified as a primary diagnostic lesion.

6.2.1.10 Cribra Orbitalia and lesions of the Orbit

In the analysis of the study assemblages, 34.5% (67/194) of the total combined assemblage presented with cribra orbitalia, of which there were 13 cases that consisted of plaques of new bone in the orbit which may have been associated with sub-periosteal haemorrhage of the bony orbit. These 13 cases all occurred in the naval assemblages known to suffer from scurvy, Haslar presented with 8/44 cases and Plymouth with 5/81. When cribra orbitalia was correlated with the other lesions, it correlated positively with four primary diagnostic lesions. Periosteal lesions of the orbit were assigned as non-specific indicators of adult scurvy. However the orbital plaques have great potential for future studies but the numbers in this study are too small to make any definite inferences about this type of lesion.

6.2.2 Summary of Cranial Scorbutic Indicators

All of the cranial lesions that have been assigned as primary or secondary diagnostic indicators of scurvy can all be attributed to the soft tissue indicators of scurvy and scorbutic haemorrhaging in the cranial region. Differential diagnoses for the lesions have been put forward, however most of these can be dismissed due to the location, type and patterning of the lesions. It must also be considered that these lesions do not often occur in isolation and when a suite of lesions caused by a chronic haemorrhagic disease process are observed

in an adult skeleton, then scurvy is the most likely candidate. In the next section, the postcranial indicators of scurvy are discussed.

6.3 Introduction to Postcranial Scorbutic Indicators Discussion

The postcranial indicators will be discussed by body part affected and will therefore be divided into three categories. These categories are the lower extremity (femur, tibia, fibula and foot), the upper extremity (humerus, ulna, radius and hand) and the irregular bones (clavicle, scapula and innominate). These regions will again be discussed in the light of the results presented and will be placed in the context of the medical manifestation of scurvy. Differential diagnoses will also be discussed.

6.3.1 Lower Extremity- Femur, Tibia, Fibula and Foot

The lower limbs are the second most commonly affected element of the human body affected by scorbutic haemorrhaging after the cranial region (Fain 2005; Lind 1772; Bevelaqua et al. 1976; Hodges et al. 1969). It is thought that scorbutic haemorrhaging in this area can lead to sub-periosteal haemorrhaging and ossified haematomas (Van der Merwe et al. 2010b; Crist et al. 2005; Geber et al. 2012). The most common bones presenting with periostitis are the tibia and fibula (Roberts and Manchester 2005; Connell and Miles 2010, 44). This is because both the fibula and particularly the tibia are quite exposed to trauma and have a lower surface temperature than other bones which can precipitate bacterial growth in this area (Roberts and Manchester 2005). This area can also be badly affected by venous stasis which can lead to varicose veins and ulcers. The femur does not often present with typical non-specific periosteal reaction due to its relatively thick protection under dense layers of fat and musculature (Ortner 2003).

In historical cases of scurvy, the legs are often worst affected by scorbutic haemorrhaging such as large ecchymoses and hematomas. Lind states that the commonest site of haemorrhaging and swelling is the lower extremity, "the legs are swelled and hard, chiefly at the calves and sometimes they are greatly indurated…the muscles of the thighs are often rigid and painful" (Lind 1772, 461). Hess agrees that the legs are the most commonly affected by scorbutic haemorrhaging; he specifically states that "trauma plays an important role in determining the location of deeper as well as of the superficial haemorrhages. In adults especially in soldiers, in whom the greater number of cases has been recorded, the lower extremity is the commonest site, between the knee and the ankle, the area most exposed to blows as well as acted upon by static congestion" (Hess 1920, 85). This is an important observation and particularly relevant in his study because of the similarity between the life of army and the navy and the parallels between the types of conditions sustained by the two groups. These haemorrhages of the legs have often been generally referred to as the 'black leg' (Hampl et al. 2001). These observations are mirrored in modern clinical cases which often refer to large ecchymoses and swelling of the legs which can be exacerbated by minor trauma and cause the legs to turn back and bruised (Velandia et al. 2008; Pimental 2003; Allen at al 1982; Lau et al. 2009; Smith et al. 2011; DeLuna et al. 2003).

The tibia is commonly affected by these ecchymoses and in particular the calf muscles (Joffe 1961; Smith et al. 2011; Grusin et al. 1957). Lind (1772) explicitly states that the flexor tendons are often 'stuffed' full of blood (Lind 1772, 498). In the course of this study, defined areas of periosteal bony reaction were noted in relation to that are covered by soleus, tibialis posterior, tibialis anterior and also flexor digitorum longus. In Figure 6.1,

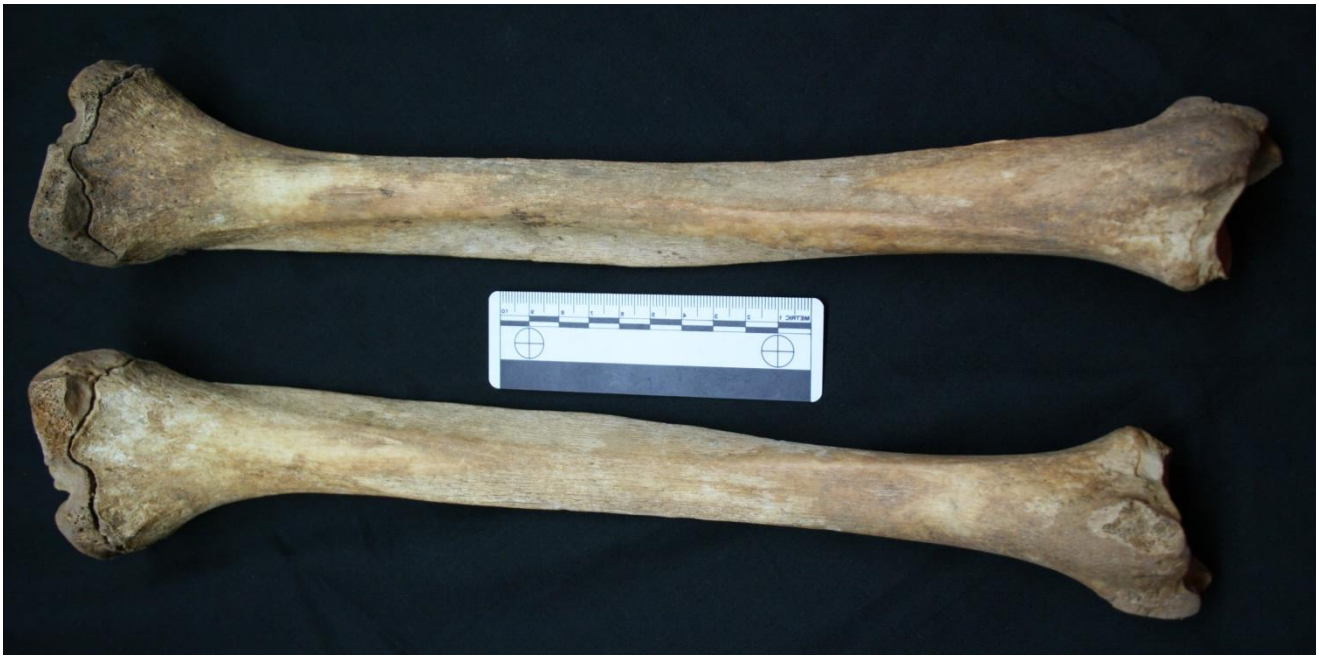

Figure 6.1 Image of tibiae showing periosteal reaction limited to the areas of tibialis anterior and flexor digitorum longus.

there are two tibiae belonging to the same individual showing periosteal reaction limited to the areas covered by tibialis anterior and flexor digitorum longus. It is thought that one of the few places that typically display ossified haematomas is the anterior tibia. The reason for this is that there is no muscular covering of the anterior tibia and therefore no restriction for a haematoma to form especially if subcutaneous haemorrhaging occurs. However in the areas of the tibia, that are covered by muscle, then it is more likely to cause periosteal reaction if haemorrhaging occurs. Periosteal reaction and ossified haematomas of the tibia were commonly recorded during this analysis and 197 out of 253 observable tibiae presented with non-specific lesions. Anatomically related lesions were noted in 55/253 skeletons which included 6 ossified haematomas. The majority of these anatomically related lesions originated from the Naval assemblages, Haslar with 13/43 (30.2%) present and Plymouth had 29/118 (24.6%) skeletons with anatomically related reaction of the left tibiae. In Darwen, there were 8/51 (15.7%) recorded and 5/41 (12.2%) in Oxford Castle. Tibial periostitis can be attributed to a number of causes and can be caused by a non-specific

infectious disease, a specific disease process, venous pathology or even trauma (Pinheiro et al. 2004, 141; Roberts and Manchester 2005; Ortner 2003). These aetiologies can easily be ruled out because of the appearance, location and bilateral nature of these lesions. Most specific disease process such as syphilis leave pathognomonic lesions which are often lytic, these are easily distinguished from non-specific lesions. Trauma can be ruled out as a result of these lesions due to their bilateral and widespread occurrence. Despite the non-specific nature of most tibial lesions, anatomical scorbutic lesions can be easy distinguished by their demarcation which can be attributed to haemorrhaging in the lower leg musculature. Tibial lesions were associated with four primary diagnostic lesions and were then classified as a non-specific lesion. However anatomical tibial lesions can still be useful in the diagnosis of scurvy.

The fibula can also be marked by the effects of scorbutic haemorrhaging due to its intimate relationship with the musculature of the tibia. In particular, the fibula is close to the calf muscles which are particularly susceptible to scorbutic haemorrhaging (Choh et al. 2009; Grusin et al. 1957). It is thought that haemorrhaging in the area of the fibula can be demarcated by the haemorrhaging of soleus, the fibularis muscles and flexor digitorum longus. Out of 224 intact left fibulae, 82 displayed periosteal lesions, of which 24 of those were related to the musculature of the fibula. Out of the 24, the highest rate occurred in Haslar with 6/35 (17.1%) fibulae affected, this was followed by Plymouth with 11/101 (10.9%). In Darwen the rate was also high with 5/48 (10.4%) of present fibulae affected, at Oxford Castle the rate was much lower with just 2/40 (5%). This prevalence rate indicates the rate of anatomical fibular lesions is much higher in the assemblages known to have suffered from scurvy and is noticeably lower in Oxford Castle. The fibula correlated with the

primary variables and was therefore classified as a secondary diagnostic lesion. The fibula is subject to the same differential diagnoses as the tibia.

The femur is the main bone of the upper thigh and is susceptible to the effects of scorbutic haemorrhaging. In numerous medical and historical cases, the thigh is specifically referenced as the location of scorbutic pathologies. Hess (1920, 95) states that "subperiosteal haemorrhage has long been recognised as a lesion characteristic of scurvy, it occurs most commonly at the lower end of the femur". Many medical studies also indicate that the posterior femur is a common place for haemorrhaging to occur (Smith et al. 2011; Jaffe 1961). Specifically, vastus intermedius, vastus lateralis and vastus medialis, all part of the quadriceps muscle group, are implicated in haemorrhaging and edema here (Smith et al. 2011; Popovich et al. 2009). Any femoral lesions recorded during this study, were recorded as anatomical if the periosteal reaction was defined by the attachment of these muscles, periosteal reaction defined by iliapsoas was also recorded in this study. Lind specifically mentions psoas as a muscle that he observed to be subject to scorbutic haemorrhaging (Lind 1772, 498).

Overall 201/268 skeletons in the total combined assemblage displayed lesions of the left femur, of these 72/268 (26.9%) were defined by the musculature of the femur and were therefore thought to be scorbutic in nature, of these 11 possible ossified haematomas were recorded. The highest proportion of these lesions occurred in Plymouth and Haslar. In the Haslar skeletons, 15/43 (34.9%) of intact left femora presented with these lesions, Plymouth had a very similar rate with 43/133 (32.3%) affected. Both non- Naval assemblages showed much lower rates, Oxford Castle had 6/36 (16.7%) affected and Darwen had 8/56 (14.3%). Overall this would support the argument that this the groups affected by scurvy have a

significantly higher rate of anatomically related femoral lesions. There are few differential diagnoses that can be put forward as causative factor for these lesions. Trauma cannot be considered, as the minor injury that so often affects the tibia, does not often affect the femur because of its thick muscle and fat protection (Ortner 2003, 209). The femur is also not as affected by venous stasis and the lower temperature that predisposes the tibia to non-specific periostitis. The muscle linked lesions of the femur can be considered to be pathognomonic of scurvy as they tally perfectly with the medical and historical descriptions of scurvy and cannot be attributed to any other cause. Femoral lesions were strongly linked with the other primary and secondary diagnostic features and due to this were classed as a primary diagnostic lesion.

The area of the ankle and foot is another part of the lower extremity that can be vulnerable to the haemorrhagic effects of scurvy. Like the other parts of the lower limb, this is partly the case due to gravity and the effect of venous stasis in the area (Hess 1920). Lind asserts that often the first sign of scurvy can occur in the ankles (Lind 1772, 499). Similarly several medical studies have noted edematous and haemorrhagic changes to the ankle area in scorbutic individuals (Hirschmann 1999; Pangan and Robinson 2001; Jennings et al. 1938; Pimentel 2003).

During this analysis, periosteal new bone in the foot was frequently noted on the medial calcaneus and occasionally the metatarsals especially number one and five. In the total combined assemblages, there was 38/195 skeletons with periosteal bone on left feet, of these 35/195 were assessed to be related to foot musculature. The highest proportion of lesions occurred in Plymouth with 20/92 (21.7%) of left feet affected, followed by Haslar with 5/31 (16.1%). In the comparative groups, rates were slightly lower, 5/37 (13.5%) of the

Oxford Castle skeletons were affected and 5/41 (12.2%) of the Darwen skeletons. The new bone and reaction on the calcaneus was always seen on the medial aspect under the sustentaculum tali and was restricted to the area where the medial head of quadratus planta originates and can be seen in Figure 6.2. New bone was usually noted on the lateral dorsal surface of metatarsal five and medial surface of metatarsal one. The periosteal bone on the calcaneus has not been recorded by any previous study on the skeletal lesions of scurvy. The only other aetiology that can be put forward for this lesion is trauma to the foot (Matt pers. comm 2012). This is unlikely in that the medial aspect of the foot joint is well protected from trauma and none of the lesions recorded occurred with visible trauma. The lesions are also clearly demarcated by the area covered by quadratus planta. Foot lesions were deemed to be a secondary diagnostic indicator of scurvy.

Scorbutic manifestations of the lower limb have long been recorded in association with

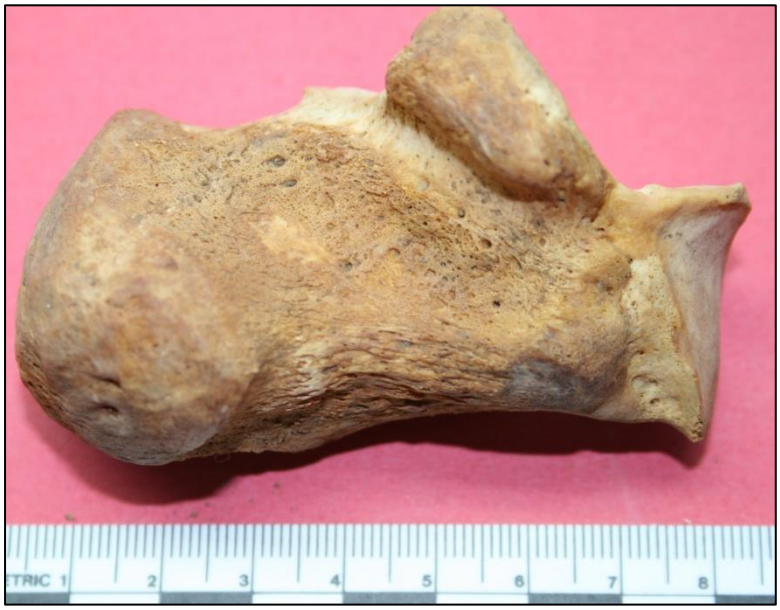

Figure 6.2 Periosteal reaction of the medial calcaneus.

scurvy (Lind 1772; Hess 1920) and are still recorded today (Pangan and Robinson 2001; Fain 2005; Hirschmann 1999). Haemorrhaging into the muscles of the legs is restricted by the

extent of the muscles and their attachment and relationship to bone. It is haemorrhage into the deep muscles that causes bony changes, which can be identified through bony changes limited to areas covered by muscle structures.

6.3.2 Upper Extremity - Humerus, Ulna, Radius and Hand.

The upper limbs are somewhat less often affected by scorbutic haemorrhage than the lower limbs, however has been known to occur. This is thought to be the case, because there is less stasis of blood in the arms and gravity has less effect on blood pooling in the muscles of this region. However, Lind describes a man with scurvy, where haemorrhaging in the arm manifested before any changes in the legs or elsewhere (Lind 1772, 275), he also notes another case of a scorbutic man whose “arm indeed was quite black from elbow to wrist” (Lind 1772, 134), Lind also mentions contraction of flexor tendons of the arm in this case. When Lind examined the dissection notes of Lord Anson’s surgeon, it was noted that the arms of those that died from scurvy were red and when dissected the interstices of the arms were full of purulent matter and coagulated blood (Lind 1772, 242). Bartholomew (1972) records the case of an adult with scurvy who exhibited subperiosteal haemorrhages of the ulna, radius and humerus. Jennings and Glazebrook (1938) record an individual with scorbutic haemorrhaging of the forearm associated with previous injuries, in this article it was also noted that scorbutic haemorrhaging occurred in a discrete area of the arm after the application of a tourniquet. References to scorbutic haemorrhage in the upper arm, forearm and elbow have been found in modern medical reports (Hirschmann and Raugi 1999; Shattuck 1928; Cleland and Fry 1930). Scorbutic bleeding in the area of the arm does occur and therefore could easily cause periosteal lesions on the arm bones. The prevalence of lesions in the region will be discussed in the next section.

The humerus forms the main part of the upper arm and was examined for scorbutic lesions in this study. In total 33 out of 267 humeri had periosteal lesions, 28 of these were limited to the areas covered by the deep arm muscles. When looking at the Naval and non-Naval groups, it was found that the highest proportion of anatomical lesions occurred in Darwen with 6/61 (9.8%). Similar rates occurred in both Oxford Castle with 5/63 (7.9%) skeletons affected and Plymouth with 15/189 (7.9%). Haslar had the lowest overall rate with 2/44 (4.5%) of individuals affected. This is interesting as the highest rate does not occur in the Naval assemblages, however it does support the occurrence of scurvy in Darwen. The main muscles affected by scorbutic haemorrhaging were medial and lateral heads of triceps, brachialis and teres minor. Figure 6.3 shows new periosteal bone formation on a distal humerus, this bone formation is demarcated by the origin of the brachialis muscle, which can be particularly clearly seen on the lateral aspect. The forearm was also examined for lesions, both the radius and ulna displayed periostitic lesions. The ulna displayed lesions that were outlined and defined by the muscles which include pronator teres, pronator quadratus, flexor digitorum profundus, brachialis and supinator. These are all deep muscles that directly overlie the bone surface of the ulna. 23 out of 254 left ulnae displayed periosteal lesions, of which 19 of these were related to one of the muscles mentioned earlier. Out of these, Darwen showed the highest rate 5/41 (12.2%), followed by Oxford Castle 4/37 (10.8%), Haslar with 4/43 (9.3%) and Plymouth 6/133 (4.5%). The radius displayed a similar rate of lesions, 17 out of 155 left radii exhibited periosteal lesions. Out of these 15 lesions were related to the musculature of the forearm. In this case, the most lesions occurred in Haslar with 4/42 (9.5%), followed by Oxford Castle 3/39 (7.7%),

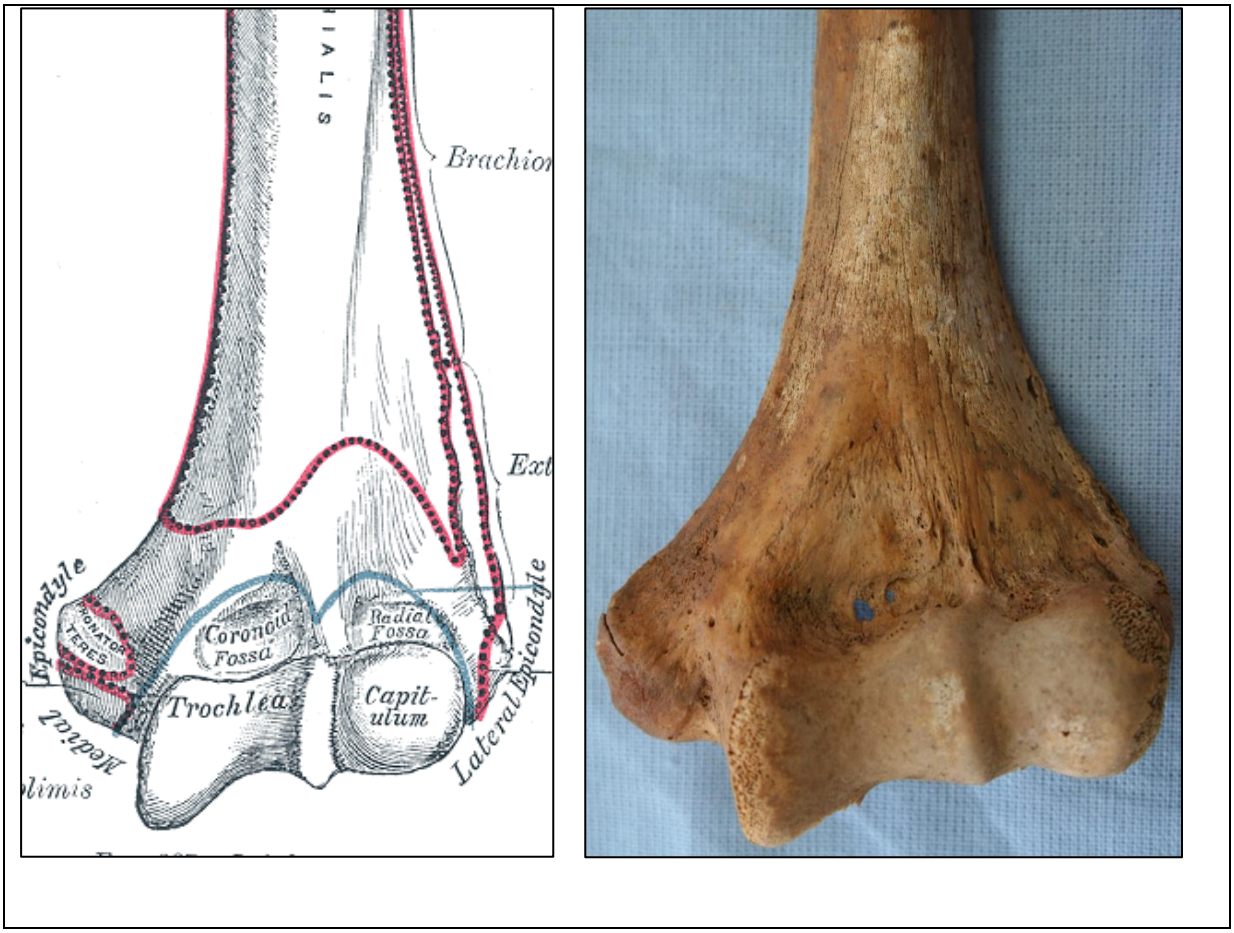

Figure 6.3 Anatomical diagram of anterior distal arm compared to photo of anterior distal arm.

Darwen 3/42 (7.1%) and least of all in Plymouth 5/132 (3.8%). Figure 6.4 shows the extent of new bone on the radius, as defined by the muscle sheath of flexor pollicus longus. Differential diagnoses that can be explored for lesions of the arm include trauma. In one case at Haslar, an individual displayed new bone associated with remains of a textile which may be the remains of bandages and also a copper pin. This lesion was clearly not defined as a scorbutic lesion and as such was classed as a non-specific lesion, caused probably by trauma. However, the majority of those arm lesions recorded were classed as anatomical and therefore specific to scurvy because of their clear connection to the musculature of the upper limb. Hand lesions were recorded extremely rarely throughout this research, only

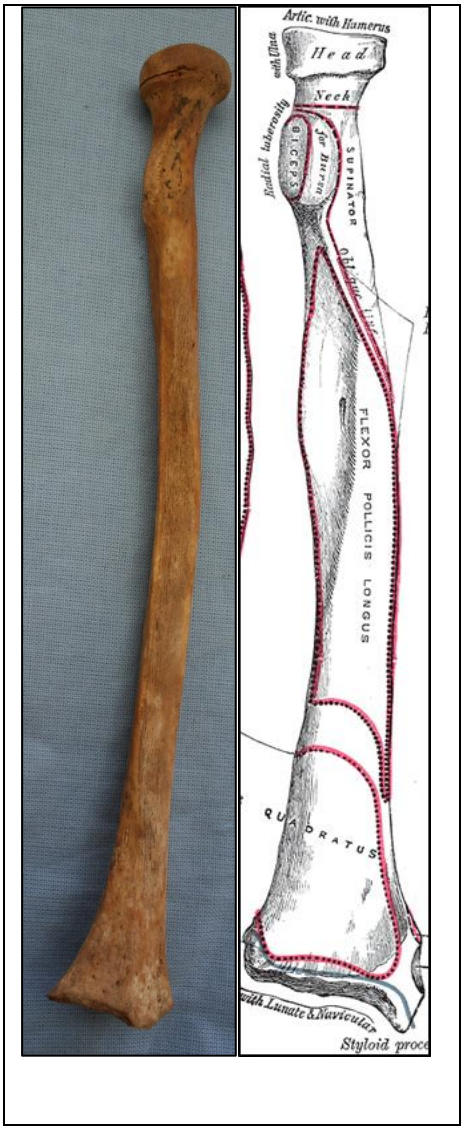

Figure 6.4 Photograph of radius compared to anatomical diagram of radius.

6/211 cases were recorded overall. Every site had 1 case, except for Plymouth that had 3. It is possible that these lesions are the result of scurvy. However due to the small amount of lesions recorded, no lesion patterning was noted. It was however noted that those individuals with multiple scorbutic lesions were those that presented with periosteal bone on the hands. Hand lesions didn't correlate with the primary diagnostic features of scurvy, probably due to lack of numbers. It is the feeling of the author, that periosteal lesions of the metacarpals are too easily caused by trauma and that should only be seen as indicative of scurvy when the occur with a large number of diagnostic lesions. Hand lesions were therefore considered to be a non-specific indicator of scurvy.

6.3.2 Irregular Bones- Scapula, Innominate and Clavicle

The irregularly shaped bones will be discussed in this section. These bones have received varying attention in the dry bone diagnosis of scurvy. The following section will establish if these lesions can be useful in the diagnosis of scurvy.

The scapula has received some attention in the identification of infantile scurvy and periosteal lesions of the scapula in this context are considered to be quite diagnostic (Brickley and Ives 2006; Geber and Murphy 2012). In adults no such link has been

established. It would seem that the shoulder is at least occasionally involved in adult scurvy. Lind (1772) states that pain in the shoulder is known in scorbutic individuals and that on occasion, scurvy can 'seize' the shoulders more than any other part (Lind 1772; 333). Occasionally shoulder haemorrhaging is noted in adult sufferers of scurvy (Cleland and Fry 1930; Jennings and Glazebrook 1938). In the assemblages studied, periosteal reaction was noted in the area of deltoidus, supraspinatus, infraspinatus, subscapularis and teres minor. In total only 11 out of 233 individuals in the combined assemblage showed periosteal reaction of the left scapula. Out of these, the majority were in Oxford Castle with 6/35 (17.1%) affected. Only 1/44 (2.3%) at Haslar was affected, 2/41 (4.8%) at Darwen and 2/73 (2.7%) at Plymouth. It is interesting that Oxford Castle shows the highest rate and it is somewhat unexpected, however the overall rates are so low, it is difficult to generalise. Despite the small number of skeletons affected, such lesions of the scapula cannot be attributed to any other disease process and can be considered to be a primary diagnostic lesion.

The clavicle was also examined for scorbutic lesions. It is thought that the clavicle would be subject to the same haemorrhages that affect the scapula. Only 7/234 clavicles presented with periosteal bone, however none of the lesions could be definitively linked to the musculature of the clavicle. Proportionally the highest rate occurred in Darwen 2/43 (4.6%), followed by Oxford Castle 1/34 (2.9%), then Plymouth 3/117 (2.6%) and Haslar 1/40 (2.5%). However owing to the small numbers it is difficult to discern any pattern. However, clavicle lesions was strongly linked with the primary lesions and were therefore classified as a secondary diagnostic lesion.

The innominate is the last bone to be discussed in relation to scorbutic lesions. Again it is not a bone that has received any attention in the study of adult scurvy. The only author that mentions the hip as being a site of interest in scurvy is Lind (Lind 1772, 506). No medical references could be found specifically to link hip haemorrhaging and scurvy. However due to the large muscle masses in this area, it is highly unlikely that haemorrhaging never occurs here. In the course of this analysis, 24 out of 238 left innominates displayed periosteal bone reaction, out of these 21 showed lesions that were definitely related to muscle attachments of the hip area. Two-thirds (14) of these anatomical lesions occurred in Haslar alone, which accounted for 32.6% of the present left innominates in the assemblage. Plymouth had 2/120 (1.6%), Darwen had 3/42 (7.1%) and Oxford Castle had 2/30 (6.7%). In the overall assemblage, the muscle sites that displayed reaction were illiacus, gluteus medius and gluteus medius. It was strongly associated with the primary lesions and the innominate was then classified as a secondary diagnostic lesion.

6.3.4 Differential Diagnosis of Postcranial Lesions

The main lesion that has been discussed in relation to postcranial scorbutic lesions is that of periostitis. As previously discussed, periostitis is a non-specific lesion that can be caused by a number of causative factors that can include trauma, ulcers, vascular problems, cancer, infection, hypervitaminosis A and numerous disease processes (Roberts and Manchester 2005; Ortner 2003). Ortner (2003) has suggested that periostitis be divided into primary and secondary forms. The primary form referring to periostitis as a process by itself and

secondary referring to periostitis associated with a separate disease process or syndrome such as syphilis (Ortner 2003, 209). Secondary periostitis will be discussed here first. Periostitis is known to occur with many specific disease processes such as syphilis and tuberculosis. Disease diagnosis in this case is based on the pathognomonic lesions of these infections. For example with syphilis, it typically manifests with as periosteal lesions with gummatous lesions which have a lytic (eaten away) appearance. The characteristic feature of tuberculosis is osteomyelitis (Roberts and Manchester 2005) and this is a lytic process, which is easily distinguished from periosteal reaction. Similarly carcinomas can manifest in the human skeleton with periosteal reaction but lytic lesions and tumours are more characteristic of this disease. It is also possible that multiple element periostitis could be caused by hypertrophic osteoarthropathy (HOA). This disease process is characterised by periosteal proliferation of the tubular long bones, which is usually bilateral and symmetrical (Christensen et al. 2013). The lesions of disease are thickest mid diaphysis and are centripedal. This disease is uncommonly reported in the archaeological populations, because it is usually associated with chronic lung conditions such as lung cancer and tuberculosis (Ortner 2003). However it is though that this disease may be underdiagnosed due to the prevailing opinion that periosteal reaction is caused by infection and often HOA is ignored as a differential diagnosis (Mays per comm 2013). It is however somewhat unlikely that the lesions in this study are caused by HOA, as it is improbable that such a high proportion of skeletons would be affected by this uncommon disease. Also visceral rib periostitis is typical of HOA and the rates of rib periostitis in the combined assemblages are very low. The periosteal patterning is also different, in HOA only long bones are affected but in scurvy both long bones and irregular bones are affected. Hypervitaminosis A has also been suggested as a cause for periosteal reaction. This is unlikely to be the cause in this

case, particularly in the Naval assemblages. It is well known that deficiency of vitamin A was common in the Navy and was often recorded as occurring with scurvy (Carpenter 2003). Vitamin A is found in highest concentration in oily fatty foods and it is known that none of the study populations had access to the quantity of fatty rich foods needed to cause Hypervitaminosis. In essence, none of these disease processes are the cause of the type of periosteal lesions that were seen in this study.

Primary periostitis is thought to have two main causes which are trauma and infection/inflammation and it can be extremely difficult to distinguish between the two. Traumatic periostitis can be caused by a one-off or chronic traumatic insult, not necessarily associated with a fracture. Periostitis can also be caused by an infection/inflammation that cannot be identified. The aim of this study is to be able to specifically identify the periosteal lesions of scurvy and differentiate them from the periosteal lesions caused by other aetiologies. During the course of the skeletal analysis, specific periosteal lesions were recorded that were associated with muscles overlying the bone and are delineated by the muscle attachment areas. These kinds of scorbutic 'anatomical' lesions have never been reported in the postcranial skeleton before, but do occur in a very specific pattern and are certainly delineated by the muscle sheaths. It is evident that the deep muscle haemorrhages of scurvy can be quite substantial and are limited by the muscle sheath and occur within the muscle fibres (Hess 1920, 85) and it is thought to cause the lesions recorded in this study. It is described best by Lind; "the quantity of the effused stagnating blood was sometimes amazing; we have opened bodies in almost a fourth part of this vital fluid has escaped from its vessels. It often lay in large concretions on the periosteum; and in the legs and thighs, the bellies of them muscles seemed generally as if they were stuffed with it" (Lind 1772, 496).

From this description, it is easy to envisage how deep musculature of the legs and other locations can fill with blood and cause bony lesions that are limited by the boundaries of the muscles.

Another type of new anatomical lesion that was recorded was periosteal bone related to the nutrient foramina of bones such as the innominate, tibia and fibula. In these cases, there is clearly a distinct, usually small patch of new bone, which clearly follows the track of the blood vessel into the bone. It is thought that bleeding from the nutrient foramina blood vessels could be caused by minor trauma, which is instigated by scorbutic frailty. It has also possible that normal muscle contraction can cause scorbutic blood vessels to rupture and cause bleeding (Brickley and Ives 2006). This process could just as easily affect the blood vessels passing through foramina.

Both lesions of bone defined by the musculature and the lesions of the nutrient foramina can be considered to be indicative of a chronic haemorrhagic process, namely scurvy. This analysis has proved that scorbutic periosteal lesions can be differentiated from non-specific periosteal lesions in the postcranial skeleton.

6.3.5 Age and Sex Considerations

Other considerations that need to be taken in account when looking at possible scorbutic lesions include age and sex differences. Sex differences are particularly interesting in this research in that the two primary assemblages are completely male dominated. Only one female was present in Plymouth and one in Haslar, these were both excluded from analysis as they were not expected to show scorbutic lesions. However females in Oxford Castle and Darwen did show evidence of scurvy.

Age was another important consideration when looking at rates of many lesions including ante mortem tooth loss, external skull lesions, posterior maxilla and lesions of the hard palate. It was decided to look at each age range and look at lesion rates to see if there was a correlation between older age and lesion occurance, these results can be seen in Figure 6.1. For ectocranial lesions, 40.6% of all individuals displaying these anatomical lesions were under the age of 25, only 13.8% of affected adults were over the age of 45. This would seem to indicate that there is not a

Table 6.1 Showing possible age related lesions and the age categories associated with these.

	Ectocranial	Ante Mortem Tooth Loss	Hard Palate	Posterior Maxilla
Adolescent	17 (12.3%)	6 (4.6%)	4 (8.2%)	8 (12.5%)
Young Adult	39 (28.3%)	30 (23.3%)	15 (30.6%)	21 (32.8%)
Middle Adult	21 (15.2%)	15 (11.6%)	9 (18.4%)	11 (17.2%)
Mature Adult	28 (20.3%)	37 (28.7%)	10 (20.4%)	13 (20.3%)
Older Adult	19 (13.8%)	24 (18.6%)	7 (14.3%)	8 (12.5%)
Adult?	14 (10.1%)	17 (13.2%)	4 (8.2%)	3 (4.7%)
Total	138	129	49	64

a pattern of ectocranial lesions only occurring in older individuals. Ante mortem tooth loss was also examined and this was more common in mature and older adults, which accounted for 47.3% of all cases in the combined assemblage. Ante mortem tooth loss can also be related to posterior maxilla lesions. These lesions were more common in younger individuals, with young adults and adolescents accounting for 45.3% of these lesions, whereas older and mature adults accounted for 32.8% of lesions. Hard palate lesions were also examined and similar rates were seen for both older/mature individuals and for young/adolescent individuals.

6.4 Scurvy Rates and the Historical Context

In section 5.5 ,the rate of scurvy for each site was established, these will be discussed here. Similarly in previous chapters, historical evidence for scurvy at the study sites was presented and in particular yearly figures of scurvy rates in the Naval hospitals from 1792-1800 was presented. This subsection will attempt to link the historical evidence for scurvy to the results of the skeletal analysis. As previously discussed a diagnosis of scurvy was based on a combination of primary and secondary diagnostic lesions, individuals with a combination of 3 or more primary lesions and 1 or more secondary lesions were classified as having probable scurvy. Individuals with 2 primary lesions and 2 or more secondary lesions were defined as having possible scurvy. Any less than 2 primary lesions along with two secondary lesions and scurvy was not diagnosed. These criteria were chosen as it was thought that this would lead to less over-estimation of scurvy rates and would give a more representative picture than other studies. Van der Merwe et al. (2010b) diagnosed scurvy using three lesions, namely ossified haematomas, periodontal disease and subperiosteal reaction. This would almost certainly over-estimate scurvy rates as periodontal disease is a non-specific indicator and the sub-periosteal reaction could have been caused by a number of causes. Geber et al. (2012) diagnosed scurvy in skeletons that showed any one of his definite scorbutic traits. For this study a minimum of four diagnostic scorbutic lesions had to be present. The scurvy rates for each assemblage were established in section 5.6 and will be discussed here. For the purpose of this discussion, the individuals with probable and possible scurvy will be combined. The Naval collections will be discussed first, the prevalence rate established for Haslar was 30/45 (66.7%) of the total population, and

39/189 (20.6%) of the Plymouth assemblage. The percentage noted at Haslar is quite high but this is thought to be quite representative considering the preservation of this assemblage. The rate of scurvy at Plymouth is thought to be an underestimation due to the high rate of skulls that are missing. When the Plymouth data is corrected for the missing crania, then this figure doubles to 46.2%, which is much more comparable to the Haslar data.

There does seem to be a large discrepancy between the calculated rates of scurvy in the skeletal assemblages when compared to the observed rate in historical records. The osteological rates seem extremely high in comparison. There are many issues that may have contributed to discrepancies in the rates that have been obtained and these will be discussed in the following paragraphs.

The main difficulty is reliance on limited historical records to provide an indication of scurvy rates in the study period. Historical records are by their very nature biased. There are several biases that need to be examined in relation to the historical accounts. The first of these is that we do not have historical records for the period 1753-1791 and it is not known how many men suffered or died from scurvy in this time in the two Naval hospitals. Only records from 1792 onwards were available for both hospitals and we are therefore missing records from the time period when scurvy would have been at its height in the Navy. The overall number of individuals that were admitted to Haslar and Plymouth with scurvy in the 1790s is in reality very low. The year of 1795 records the highest rates of scurvy, however in this year only 4% of men admitted to Haslar and 14.7% of men admitted to Plymouth are recorded as suffering from scurvy, of whom very few died. These low recorded rates may be down to a number of reasons. The first of these is that seamen suffering from mild scurvy

may not have been routinely admitted to hospital and only the most severe cases were received into hospital. Endemic sub-clinical scurvy was common in the post medieval period, especially among the poor and in Naval recruits (Rogers 1986, 101), so perhaps mild scurvy was not seen as a disease worthy of hospital admission. Also many of the individuals may not have been admitted to the hospital with just scurvy, it may have co-occurred with another disease process. This has been briefly mentioned in section 5.8.2, it was frequently noted in the musters that scurvy was recorded with fevers and fluxes and it is possible that these diseases were recorded as priority over scurvy. Most importantly, it should be considered that scurvy was almost eradicated by the 1790s and this is the earliest period that we have hospital admission musters for Haslar and Plymouth. A literature review was conducted to find an indication scurvy rates in the Navy pre 1792 and only rates for sick quarters and contract hospitals in Deal and Plymouth were found. It must be noted that these records do not include the hospital at Stonehouse, Plymouth. These scurvy rates are presented in Table 6.1 and show the number of sick in sick quarters and the contract hospitals in this time, the number with scurvy and those that died of scurvy in 1757 and 1758. The rates of scurvy recorded at the contract hospitals in the 1750's at Plymouth and

	Plymouth			**Deal**		
Year	**Sick**	**Scurvy**	**Died**	**Sick**	**Scurvy**	**Died**
1757- 1st Qtr.	566	145 (26%)	7 (5%)	367	57 (16%)	2 (4%)
1757- 2nd Qtr.	523	211 (40%)	10 (5%)	327	23 (7%)	1 (4%)
1757- 3rd Qtr.	168	49 (29%)	2 (4%)	430	42 (10%)	1 (2%)
1757- 4th Qtr.	264	61 (23%)	2 (3%)	360	28 (8%)	3 (11%)
1758- 1st Qtr.	390	59 (15%)	6 (10%)	263	36 (14%)	0
1758- 2nd Qtr.	161	23 (14%)	1 (4%)	142	33 (23%)	1 (3%)
1758- 3rd Qtr.	n.a	n.a	n.a	115	17 (15%)	0

Table 6.2 Scurvy rates at Plymouth and Deal sick quarters in 1757 and 1758 reproduced from Rogers 1988, 102.

Deal are a great deal higher than those recorded at Haslar and Plymouth in the 1790's and may provide an indication of scurvy rates in the 1750's. Even still, these rates are much lower that the three or four hundred scorbutic patients that Lind is seeing every day in the 1750's at Haslar (Lind 1772). Due to the lack of historical records from the early years of the Naval hospitals, we cannot determine how many individuals suffered from scurvy during these years. However what can be said, is that the rates of scurvy in the 1790's do not seem give an accurate picture of scurvy rates from the 1750's-1780's in the Naval hospitals and are almost certainly an underrepresentation.

Another reason for the inconsistency between skeletal rates of scurvy and historical documentation may be due to bone biology and bone remodelling. It is not known how long it takes for scurvy lesions to heal and disappear. Therefore it is possible that an individual may have suffered an episode of scurvy at sea, been cured and perhaps months or even years later suffered from another disease and may have been admitted to the hospital and died. It is also true that scurvy was often cured on-board and therefore they may be no record of the individual having entered the hospital with scurvy. In this case, it is likely that the scurvy will still be visible in the skeleton but this scorbutic individual would not appear in the hospital logs. Another important consideration in relation to bone remodelling and scorbutic lesions is that we must remember that scurvy is a metabolic disease process. A metabolic disease process by its definition affects the way human bone models and remodels and it is not known the effect that a recurring metabolic disease may have on the length of healing of pathological scorbutic lesions. It is apparent that there can be no clear association derived from the observed skeletal rate of scurvy and the historical

documentation due to the incomplete and inconsistent nature of the Naval hospital records especially from the early use of the hospitals.

There were also some differences noted between the rates of scurvy recorded between Haslar and Plymouth. One of the reasons suggested for this difference is a disparity in the diet of the two assemblages. There have been isotopic studies conducted on the skeletal remains from both hospitals and suggest that there may have been a slight difference in the victualling of the two. In Plymouth, the isotopic data indicates that there was a higher marine component in the diet of those men buried at Plymouth and also a higher proportion of C4 plants such as maize, sugar-cane, sorghum and millet (Roberts et al. 2012). This is thought to be due to Plymouth hospital receiving men that were on patrol in the Atlantic, Caribbean and Americas whereas Haslar mainly received men from the Channel Fleet. This would seem to indicate that the men are receiving slightly different rations and possibly different rations of fresh fruit, vegetables and lemon juice which could have had an effect on the occurrence of scurvy. Also due to the different patrols that were untaken from Haslar and from Plymouth, there is thought to be a difference between the average journey distances. It is possible that the men leaving Plymouth would have been more likely to get scurvy due the longer journeys to the Caribbean and the Americas compared to ships leaving Portsmouth that were patrolling for shorter periods in the Channel. This difference is not reflected in the osteological record, where Haslar has much higher rates of scurvy than Plymouth. However this difference is thought to be due to the high number of incomplete skeletons in the Plymouth assemblage, for example when the Plymouth data is corrected just for missing skulls, the rate of scurvy doubles. It is also important to consider the social status of the Naval assemblages, when on-board the seamen were well fed. However whilst

on land, these seamen were subject to the same conditions as the lower classes of the time period.

In the comparative assemblages, Darwen has quite a substantial rate of 16.4% (10/61) individuals and Oxford Castle was found to be 7.9% (5/63) of the total assemblage. The evidence for scurvy in the skeletal assemblages seems to broadly fit in with the historical documentation. It is thought that the high rate recorded at Darwen may have been caused by the malnutrition that is widely recorded during the Potato Famine and later the Cotton Famine in Lancashire, combined with other famines recorded during the period the cemetery was in use and also likely due to malnutrition caused by poverty. Darwen is a particularly interesting comparative assemblage as Primitive Methodists did not drink alcohol. This is a stark contrast to the Naval assemblages that drank large quantities of alcohol on a daily basis. However, it is unlikely that this alcohol free diet had a big influence of scurvy rates and malnutrition caused by poverty is more likely to have been a major factor. The low rate recorded at Oxford Castle is again not unexpected in that there are no direct references to scurvy at the site but it is possible that small numbers of this assemblage did suffer from scurvy due to the restricted prison diet. It was previously mentioned that the Oxford Castle skeletons were subject to anatomisation, this made the identification of scurvy difficult as many of the skeletons were incomplete.

6.5 Summary

There are several conclusions that can be drawn from the results presented. The first is that each anatomical scorbutic lesion that has been assigned as a primary or secondary diagnostic lesion can be positively linked to the physiological manifestation of scurvy. Numerous clinical and medico-historical sources have been cited that support their

interpretation. This is important progress in the development of a pathognomonic suite of scorbutic lesions that can be definitely attributed to a haemorrhagic disease process, namely scurvy.

The second conclusion is that scurvy was in fact present in all of the assemblages that were studied. The Naval assemblages were badly affected but there was a notably high rate noted at Darwen and a small number at Oxford Castle. It is evident that scurvy was not just endemic in the Royal Navy of the post medieval period, but also in the general populace in times of hardship and subsequent malnutrition. This is an important observation and provides us with an insight into the prevalence of scurvy in post medieval England.

Chapter 7: Conclusion

The aim of this study was to ascertain if adult scurvy can be identified in adult skeletal remains and if so, to establish a reliable methodology to recognise adult scurvy in dry bone through macroscopic analysis. This aim was achieved by the analysis of three hundred and fifty-eight skeletons from four distinct skeletal assemblages. Two of these skeletal collections derived from the cemeteries of Georgian period Naval hospitals and two from a Non-Naval context of a contemporaneous temporal and a similar geographical context.

Four assemblages were analysed as part of this research, all of these assemblages originated from post medieval English contexts. It is clear from the historical research that in particular the Naval assemblages suffered from scurvy, this has been discussed in detail in chapter 3. This historical framework is vital to this study, as in the majority of osteoarchaeological studies we have no historical sources to indicate important contextual data such as occupation, socio-economic status and the specific diseases they suffered from. With the Naval assemblages there is a wealth of historical documents available, which specifically indicate that active Naval servicemen suffered badly from scurvy due to lack of fresh fruit and vegetables on long voyages. It is this evidence that makes the Plymouth and Haslar collections ideal for a bioarchaeological study investigating the bony manifestation of scurvy. It is also now clear that comparative assemblages may have suffered from the effects of scurvy. In particular, the Darwen assemblage is likely to have endured the consequences of scurvy during the Lancashire Cotton Famine. It is not known from the historical records if Oxford Castle specifically suffered from scurvy, but it is clear that scurvy was a problem in the prisons of the time.

It was proposed that scurvy should be observable in skeletal material because the chronic haemorrhagic and inflammatory lesions that characterise scurvy should manifest in dry bone (Fain 2005). It was thought that these lesions should be restricted due to the soft tissue and musculature of the human body, in the same way that haemorrhaging blood is restricted by the same structures in scorbutic individuals. This has been proved by the recognition of a new class of lesion known as an anatomically related lesion. Anatomically related lesions have never been described in the paleopathological literature in conjunction with the postcranial skeletal remains of adults. This new lesion classification has the potential to simplify the diagnosis of scurvy in adult remains and cannot be attributed to any other disease process.

The data that has been collected in the course of this research proved the hypothesis that adult scurvy can be recognised in the archaeological record. As expected, significant higher rates of scorbutic lesions were found in the skeletons that originated from the burial grounds of the Naval hospitals, which fits in well with the historical documentation. 66.7% of the final Haslar assemblage was found to have scurvy, along with 20.6% of Plymouth. However the results were not totally clear-cut, in that scurvy was also present in both of the comparative collections. In particular, the Darwen assemblage presented with a high rate of scurvy in its adults (16.4%) and Oxford Castle was lower with 7.9%. No other studies of this kind in England have been carried out so there are no comparative rates to compare.

7.1 Limitations to Research

There are numerous limitations that have restricted this research and indeed restrain most bioarchaeological studies. The first is the availability of suitable skeletal material available for research and the inherent biases that come with excavated archaeological material. Both

Naval assemblages represent a very small proportion of skeletal material excavated from very extensive burial grounds. In particular, the Haslar material consisted of only forty-five skeletons excavated from a burial ground that may contain tens of thousands of burials. There were also issues with poor preservation and excavation damage. In particular the Darwen assemblage was exceptionally poorly preserved and this limits the quality of bone that was available for analysis. This research is not concentrated on the examination of just one bone, the diagnosis of scurvy is based on a suite of lesions from the entire skeleton and in order for scurvy to be identified both cranial and postcranial lesions must be present.

Another limitation of this research is lack of knowledge about precise timings for bone remodelling and lesion healing and how this is affected by scurvy. This also has direct implications for the length of time that the lesions of scurvy will be 'visible' in the human skeleton after an episode of scurvy. At the moment, it can be said that the scorbutic lesions are characteristic of the healing stage of scurvy, i.e. when some vitamin C is re-introduced into the diet, however timings cannot be ascertained. This also means that an individual may have suffered from scurvy and it is no longer visible in their skeleton due to remodelling. It is also true that an individual may die from scurvy and there will be no bony reaction present due to no vitamin C being re-introduced in the diet (Maat 1984). The skeleton can therefore only provide us with a narrow window of scurvy 'status' around the time of death.

Another limitation was a lack of hospital musters and subsequent scurvy rates from the early use of both Haslar and Plymouth hospitals. This may have led to the large difference between the observed skeletal rates of scurvy and the historical rates of scurvy.

Despite these limitations, the assemblages that was analysed in this research were ideal populations for an investigation into the dry bone manifestation of scurvy, particularly the Naval collections.

7.2 Directions for Future Research

This study has highlighted the capacity for research on the recognition of scurvy in adult human remains. This study was small, but potential future work should be carried out on a much larger scale and should be extended to other time periods. In this course of this study, a number of lesions that were not directly caused scurvy but were linked to its manifestation were discussed, for example, Schmorl's Nodes and Degenerative Disc Disease. This study did not have the scope to examine the link between these lesions and the skeletal manifestation of scurvy, however this could be an interesting avenue for future studies. Radiographic analysis could also be potentially useful to look at the link between scurvy and osteoporosis. There is also the possibility to use more destructive testing techniques such as histology to identify lesions such as hematomas. There is also the hope that techniques such as collagen testing will be further defined and this could be used in combination with macroscopic analysis. Another interesting possibility would be analysing populations that are known from historical documentation to have suffered from a period of malnutrition or famine and subsequently may have suffered from scurvy. It is vitally important to analyse further skeletal populations using the same research methodology. It would then be possible to compare results and determine whether this methodology is useful on larger scale multi-period archaeological assemblages.

7.3 Conclusion

Vitamin C deficiency and resultant scurvy would have been a detrimental problem for past populations that were exposed to periods of malnutrition due to famine, war and even prolonged winters. This research has proved the hypothesis that adult scurvy can be identified in dry bone and has established a reliable methodology by which this diagnosis can be achieved. It is fundamental to the study of paleopathology to be able to identify this disease process in past populations as it can provide us with a more complete picture of the health status of past populations and the complex interface between health and disease interaction.

Glossary

Bridewell- is a term for a prison or a house of correction, may have come from Bridewell prison which was opened in London in the 16th century.

Collagen- protein that forms most of connective tissues, such as kin, bone, tendons, muscles, and cartilage

Ecchymoses-large bruise, greater than 1cm in size

Hemarthrosis- haemorrhage or bleeding into a joint space.

Periosteum- is a membrane that tightly adheres to the outer surface of all bones.

Petechiae- tiny pinprick haemorrhages that often look like a rash

Pupura- red/purple bruises caused by bleeding just under the skin.

Victualling-to provide with food or stores, particularly used in reference to ships.

Vitamin C-also known as ascorbic acid, is an essential nutrient for humans, it is a naturally occurring organic compound with antioxidant properties

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