A Bioarchaeological Approach to Stress and Health in Medieval Denmark: Dental Enamel Defects and Adult Health in Two Medieval Danish Populations

By

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Abstract

This research engages in a bioarchaeological analysis of two medieval Danish sites using combined microscopic and macroscopic methods in order to investigate three objectives. The first was to consider the relationship between childhood stress and adult health through the joint consideration of microscopic enamel defects and adult health indicators. Given the context of these populations over a period of history characterized by changing climate and socioeconomic conditions, punctuated by famine and plague, this research also sought to examine temporal patterns in health and stress. Given the increased urbanization over the medieval period, the final objective was to consider health patterns between rural and urban populations. The results showed that the number of stress events did have an impact on later life mortality, and that there was differential expression of this relationship between males and females and between surface and internal enamel defects. A statistically significant decrease in stature was apparent after 1350 A.D. as well as an insignificant increase in tuberculosis and treponema, but an insignificant increase in age at death over time. The inter-site comparison showed higher rates of infectious disease at the rural site of Sejet, with tuberculosis in particular being significantly higher in females at Sejet. Mean age at death was also significantly lower at Sejet for the study sample, but a consideration of the broader cemetery sample showed no significant site differences, suggesting that this might be a sampling phenomenon. These patterns likely reflect the complex nature of the rural and urban interaction during this period, but also emphasize the need for further sampling. This research points to the complex relationship between stress and health and outlines the importance of developing more comprehensive etiological models and operational definitions for identifying stress indicators in dental enamel.

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Chapter 1 Introduction

Scope, purpose, importance, overview

The field of bioarchaeology integrates developments in multiple disciplines, drawing from areas such as archaeology, human osteology, clinical medicine, biomolecular research, and chemistry. It broadly encompasses a number of research areas unified by their common interest in gaining insight into past human populations. Bioarchaeology investigates questions relating to human health, genetics, biomolecular evolution, migration, and diet, to name a few. The strength of this field lies in its multidisciplinarity, and it is capable of providing insight into areas which are relevant to past, present and future human populations. The current research is situated within the background of bioarchaeological research, and as such it draws from human osteology, archaeology, history, and clinical medicine.

The purpose of this project is to investigate the relationship between childhood physiological perturbations (stress) and adult health and longevity through a consideration of dental enamel defects in conjunction with the skeletal evidence. This research is further situated within the context of a particularly volatile time in medieval Denmark, characterized by changing climate, shifting socioeconomic conditions, disease, and population pressure (to be discussed in more detail in chapter 2). As such, the present investigation will also consider the effects on health of these conditions through a temporal analysis and will provide information on two recently excavated cemetery populations (Sejet and Ole Wormsgade) which have not previously been the subjects of extensive study. Sejet was a rural cemetery and Ole Wormsgade was an urban parish cemetery located in the town of Horsens. As such, the consideration of individuals from these two sites will also allow the assessment of patterns of health between urban and

rural settings as a third primary objective of this research. Finally, by investigating both surface and internal dental microstructures, this research seeks to elucidate the relative health impacts on later life of stress events as indicated by these respective structures. Studies frequently focus on either internal or external structures, but seldom on both simultaneously (exceptions being Condon, 1981; Condon and Rose, 1992; Goodman and Rose, 1990). As such, this project represents an attempt to consider both levels of dental enamel defect in the same samples.

Based on the three objectives of this research, three hypotheses and their accompanying null hypotheses can be proposed:

1) It is proposed, based on current clinical, epigenetic, and epidemiological understandings of patterns in stress and health, that an increased frequency of childhood stress events will have a negative impact on adult health indicators. The null hypothesis proposes that the childhood stress events recorded in the dental enamel will have no impact on later life health as seen in the survivors of these childhood stress episodes.
2) Based on understandings of temporal changes over the course of the medieval period, with increasing population pressure through the early medieval period followed by a relief in population pressure after the Black Death, it is expected that health will be seen to deteriorate in the periods prior to the mid-14th century, but will subsequently improve. The null hypothesis here proposes that there will be no temporal change in health visible in the skeletal remains.
3) There has been ample research to indicate the impact of increased population density and urbanisation on health (Lewis et al., 1995; Lewis, 2002; Roberts

and Cox, 2003; Steckel, 2004; Koepke and Baten, 2005; Roberts and Manchester, 2005). It is proposed here that the urban population in Ole Wormsgade will present with a higher frequency of stress indicators and with poorer adult health than the rural population at Sejet. Given past research on patterns in leprosy (Boldsen, 2009), however, it is expected that prevalence rates for this disease will be higher at Sejet than at Ole Wormsgade due to differential patterns in disease control. Namely, leprosy hospitals were heavily instituted in towns, but it seems that leprosy in the rural population may have been left relatively unchecked for longer leading to greater disease exposure outside of the urban setting (Boldsen, 2009). The null hypothesis in relation to urban versus rural health proposes that there will be no statistically significant differences in stress levels and health profiles.

These hypotheses are based on extensive previous research which will be explored in further detail over the course of the introduction and in the literature review (chapter 2).

Concepts of Stress

A focal point of this research is the investigation of childhood stress events in relation to adult health. As such, 'stress' is a fundamental concept which must be defined for the purposes of this study. Stress can, simply put, be understood as the body's response to any stimulus (stressor) which can evoke physiological changes in the brain or body (namely, a stress response) (Dallman 2003:18). The concept of stress is, however, highly complex, having multiple meanings and being somewhat circular in nature (Romero et al., 2009). The very definition of stress reflects this multiplicity, with the term 'stress' being used to characterize the stimulus that causes a stress reaction, the

physiological response to this stimulus, and the pathological result from overstimulation of natural physiological responses (Romero et al., 2009, p 376).

The appearance of a defect / stress marker in the enamel will be variably expressed depending on the particular stage of activity that the ameloblasts are at when the stressor occurs (Witzel et al., 2008) and upon factors such as the morphology of the enamel at that particular point on the tooth surface (Goodman and Rose, 1990). Goodman and Rose (1990) have proposed a model which maintains that a defect either will or will not appear depending upon whether a stress threshold is surpassed (disrupting ameloblast activity). Fitzgerald and Saunders (2005) emphasized a continuum of expression for dental enamel microstructures, with no set threshold between normal and pathological. Witzel et al. (2008), on the other hand, examined ameloblast patterns in relation to particular defect types and proposed a three threshold model based on three different ameloblast response types, each resulting in a slightly different enamel histological pattern. Ultimately, these combined efforts speak to developments which can help elucidate the nature and duration of stress responses as they relate to enamel defects.

The approach taken with this project works within the framework of Fitzgerald and Saunders (2005) emphasis on the lack of a minimum defined threshold in the definition of stress markers but at the same time works from the basis that it is essential in the study of stress to define possible stress episodes. It is maintained, as per Goodman and Rose's (1990) model, that stress incidents appear differently depending upon such factors as the region of the crown affected, and the nature of a particular individual's response to any given stressor. In taking this approach, the project also integrates Hillsons' (1992) emphasis on even the finest enamel defect being a response to

physiological disruption along the same spectrum as more severe defects. Witzel et al's (2008) research and also comparative research on surface macroscopic and microscopic techniques conducted by Hassett (2012) justifies the use of a microscopic approach which can further identify enamel defects that might be invisible to a macroscopic surface examination. Unfortunately, as a population approach on a microscopic level was a key objective of this thesis, it was determined that a more detailed integration of Witzel et al's (2008) distinctions in ameloblast disruption was beyond the scope of the current research. Thus, it was not possible in the current thesis to conduct a detailed analysis of each defect to determine the point of ameloblast disruption. However, the consideration of enamel defects on the level described by Witzel et al's (2008) findings would be an interesting point for future exploration.

When considering responses to stress in skeletal remains, such concepts as those outlined in the above literature can be useful in gaining a better understanding of the osteological response. The extent to which such factors leave permanent marks on the skeleton (including in dental enamel) must be understood within the context of individual susceptibility within a developmental framework (since individual susceptibility or frailty may be expected to change over the course of their development). Models such as those developed by Witzel et al. (2008), Goodman and Rose (1990) therefore provide useful frameworks within which skeletal indicators or responses to stressors and the variable levels of expression of these defects might be better understood.

Childhood Stress and Adult Health

The above approach can be greatly informed by the integration of the results from clinical and experimental research using modern human populations and animal models. The current investigation is interested in the relationship between childhood stress incidents as indicated by dental enamel defects (DED's) and adult health (as understood from adult skeletal health indicators). The developmental range captured by these teeth runs from just after one year of age to around 6 years of age (as represented by the anterior dentition selected) (Reid and Dean, 2006).

While a critical period of rapid development occurs during the first three years of life, being the continuation of the foetal period of accelerated growth (Bogin, 1999), there are a number of fundamental systems which continue to grow during the period captured by dental enamel development (MacGregor, 2008). It has long been recognised that child development can be heavily impacted by experience through the mechanism known as developmental plasticity or ontogenetic modification (Lasker, 1969; Schell, 1995). Exposure to different nutritional loads, pathogens, and stressors, can effectively 'shape' the developing system into its final form, and this final form can either be adaptive or maladaptive in adulthood (Ulijaszek, 1996). The impact can occur at any point during development, being particularly severe during periods of rapid development (Ulijaszek, 1996; Bogin, 1999). It is for this latter reason that the foetal period is the most critical period from a developmental perspective. However, it is the former point which forms the basis for the current investigation. An examination of the body systems which develop over the first six years of life, and which might be impacted by the same stress events which leave marks on the concurrently developing teeth, is therefore pertinent to the current examination and will be discussed in the literature review.

The purpose of the current project is thus not to conclusively identify the mechanisms behind the relationship between early life stress and later life health, as far more extensive clinical and epigenetic research is still required in this area. Rather, the focus of this aspect of the project will be to examine whether correlation is visible between the occurrence of early life stress experiences and later life health. As outlined above, the sample available for this research captures a post-natal period from roughly 1 to 6 years of age. In some cases, teeth were worn down to approximately 2 years of age. As such, the period studied does not capture the fetal period which is the main focus of the literature on developmental origins. However, since critical systems are still developing over the period covered, it is argued that stress experiences have the potential to disrupt development. As such, the developmental origins literature and concepts of developmental plasticity are still highly applicable to our understanding of any patterns which emerge.

In an archaeological context, it can be particularly challenging to account for differential exposure to stressors. While it is possible to identify probable exposures within the environment based on cumulative evidence from the archaeological and skeletal analyses, it is impossible to determine whether a particular individual was exposed to specific stressors unless the stressor is reflected in the skeletal remains, and frequently skeletal markers are non-specific. The experience of chronic or systemic stress can leave marks on the skeleton. It is possible in some instances to detect pathogens in association with individual remains whether through DNA or palaeopathological analyses. There are other forms of evidence which can be recovered about diet (for example from stable isotopes or from the microscopic analysis of food particles in dental

calculus), and it is possible to learn about dietary deficiencies if diseases such as rickets or scurvy can be identified. However, such information may be difficult to pinpoint or may not be preserved on a representative enough scale.

We are thus faced with a number of challenges. Despite mounting evidence supporting a connection between childhood stress and adult health, the nature of this relationship is poorly understood and the key players in the stress relationship (exposure and frailty) are often hidden. The literature surrounding this topic will be reviewed in more detail in chapter 2. As part of that examination possible stressors present in the environment will be identified and temporal patterns in these stressors will be examined. This will in itself point to possible varying levels of exposure over time and at the different sites. When used in conjunction with the historical literature, it is hoped that some insight into the socio-environmental factors at play will be gained. Ultimately, there is no way at present to tell the cause of a particular stress-producing incident from the dental enamel defects (Neiburger, 1990), but it will be proposed that such a defect reflects a certain level of impact on the system (regardless of cause). The presence of such a change in the dental enamel reflects an incident which could have impacted other systems developing at that point in time. It is hypothesized here that a relationship between childhood stress and adult health will be visible in the medieval Danish populations under study. Support for this hypothesis would be expected in a correlation between higher frequencies of enamel defects and adult indicators of ill health (such as lower stature and decreased longevity).

Dental Enamel and Dental Enamel Defects

Dental enamel provides a useful source of evidence for such research on a number of levels. The process of enamel formation (amelogenesis) takes place with a regular periodicity (Antoine et al., 2009), which in effect creates a record of development over the course of crown growth. Furthermore, the process of amelogenesis can be disrupted by systemic stress (caused by factors such as infection and nutritional deficiency), and this results in permanent morphological changes (Goodman and Rose, 1988, 1990; Mellanby, 1929, 1930, 1934; Schwartz et al., 2006). Enamel is also one the hardest biological substance in the human body, and thus amongst the most likely to survive in the archaeological record. When the tooth crown is complete, the enamel will be noncellular, and will consist of approximately 87% inorganic material by weight (or at least 95% by volume), most of which is composed of hydroxyapatite (Brudevold and Söremark, 1967; Hillson, 1996, 2005; Risnes, 1998; FitzGerald and Rose, 2008). Once enamel has been formed in this regular pattern, it is not subject to further remodelling, unlike the rest of the skeleton, and thus it preserves a record of events occurring through the course of its formation which can then be identified and plotted chronologically (FitzGerald and Rose 2008a; Condon and Rose 1992).

Health in Medieval Denmark

The late medieval period in Europe has historically been portrayed as one of significant unrest, fluctuating between times of prosperity and crisis. This period was punctuated by changing climatic conditions, which were associated with socioeconomic changes and shifts in subsistence (Fagan, 2000; Benedictow, 2004; Hybel and Poulsen, 2013). Accompanying these patterns were demographic shifts with increasing urbanization and changing population sizes (Benedictow, 2004, 2012; Hybel and Poulsen, 2013). These were influenced not only by environmental factors but also by epidemiological changes seen in shifting frequencies of diseases such as leprosy and tuberculosis, and in the occurrence of epidemics such as Black Death.[1] The most severe of these epidemics hit Denmark in 1349 A.D., and annihilated an estimated 30-50% of the population in Europe (DeWitte and Wood, 2008).

These events are suggested by the historical literature, and in studying two Medieval Danish populations, the extent to which population health was affected by these changes and any differences in patterns between town and countryside will be investigated. It is hypothesized that health will improve over time, and that this improvement will be visible in the skeletal health indicators. These questions were investigated by Yoder (2006) in her PhD thesis, but the only possible change she found

[1] There is still a great deal of controversy surrounding the epidemiology of Black Death. Included in this is discussion surrounding the possible impact of climate change on patterns of transmission (Stothers, 1999; Stenseth et al., 2006; Büntgen, 2009; Kausrud et al., 2010; McMichael, 2010). While the most likely causative agent currently seems to be *Yersinia pestis* (the cause of later Asian and Indian plagues) (Raoult et al., 2000; Benedictow, 2004; Wiechmann and Grupe, 2005; Haensch et al., 2010; Bos et al., 2011, 2012; Cui et al., 2012), the situation was likely extremely complex and influenced by many factors (Stenseth et al., 2008; Walløe, 2008; Welford and Bossak, 2009; Bos et al., 2011; Büntgen et al., 2011). A full discussion of this would be beyond the scope of this project, but it is an avenue in which a great deal of research is yet required to elucidate patterns. For the purposes of this project, it suffices to recognize the timeline of Black Death in Denmark (likely entering in 1349 A.D. and having the greatest impact over 1350 A.D.) (Benedictow, 2004), and to acknowledge the tremendous demographic impact of this epidemic (Benedictow, 2004).

was increased incidence of periostitis in females for the later period, which she attributed either to increased rates of infection or to individuals being healthier and therefore surviving infection longer, as formulated in the Osteological Paradox (Wood et al., 1992). Furthermore, while there was a growth of towns in Denmark during the medieval period, there was likely still a high level of movement between town and countryside (Hybel and Poulsen, 2013), and so the extent to which populations from each would have been differentially exposed to any diseases and stressors is unclear. By considering stress events on a very fine scale through the microscopic investigation of enamel defects, this study will attempt to gain further insight into these patterns.

Summary

This research is thus quite broad in scope, and it incorporates a holistic approach which draws both from traditional techniques in skeletal analysis and from microscopic techniques. It is by nature a bioarchaeological study set in a historic period, and so it will incorporate historical documentation and information drawn from archaeology alongside the osteological investigation. It offers methodological developments, theoretical insights into human health, and information about a particular period of Denmark's past.

The analysis will be conducted through the microscopic examination of defects on both the surface and the internal enamel. Such an approach will also make it possible to develop a better understanding any differential patterns of stress response and health as reflected by these different microstructures. Up until this point, the consideration of surface enamel defects in connection with stress events in archaeological populations has largely been conducted macroscopically (Bennike 1985; Obertová 2005; Palubeckaitė *et al*. 2002; Wright 1997). It has been shown, however, that even the smallest defects which

are indistinguishable without a microscope can represent stress events (Hillson, 1992a). The integration of a higher degree of precision in this study will lead to a greater understanding of stress in this population.

This dissertation will progress through a literature review which will provide background information into the historical and regional context of Denmark during this period, an overview of the study of health from human skeletal remains with a focus on the use of dental enamel defects, and a discussion of the theory surrounding the relationship between childhood stress and adult health. The third chapter will provide an overview of the materials and methodological approaches used in this study. The fourth chapter presents the results of this investigation, while these are discussed further in chapter five. Chapter six will present final conclusions and suggestions for future research.

Chapter 2 Literature review

Introduction

The current research builds on a vast field of literature, drawing from areas as diverse as clinical research to climatology to history. This body of information provides the context in which the populations being studied lived. It furthermore provides valuable insight into the relationships and patterns which might be represented in their skeletal remains. Through a review of this literature, the fundamental knowledge foundations will be established and the potential contributions of the current study will be highlighted. This chapter will discuss the period of development covered by the dental enamel used in this study. It will furthermore review the research which has led to current understandings of the relationship between childhood stress and adult health. This will be followed by a review of the specific sources of evidence available for studying health in the human skeletal remains, along with the problems and limitations entailed by these sources of evidence. The review of this background literature will then proceed to outline the context in which the people in this study lived, drawing from historical, climatic, archaeological, and epidemiological perspectives. This will be followed by an outline of past research that has informed our understanding of health during this period, drawing from the osteological literature.

Evidence for the impact of early life stress on later life health

While particularly rapid development occurs during the prenatal period, this accelerated pace of growth continues into the infant and childhood periods (Bogin, 1999). Dental enamel in the teeth reviewed through this research develops from roughly one to six years of age, based on analysis of a nearby Northern European sample (Reid and Dean, 2006). It is therefore pertinent to review the numerous body systems which

continue to develop over the course of the first six years of life, and which might consequently be impacted by stressors resulting in systemic stress responses during this period (Table 2-1).

Table 2-1 Table outlining the developmental patterns which occur during the period of enamel development captured by this study. Information taken from (2008) [1] and Chamley et al. (2005)[2], and Bee and Boyd (2010)[3].

System	Development from 1 to 6 years with some Associated Implications
Nervous[1,3]	✓ The brain is largely formed by birth, but requires further development after birth. ✓ From birth to 5 years of age the brain grows rapidly, doubling its weight in the first year and attaining 90% of the adult brain size by the age of 5. [1] This period is characterize by slow growth from 2 to 4 years of age and a subsequent growth spurt at the age of 4.[3]
Cardiovascular[2]	✓ The heart doubles in weight in the first year. ✓ By 9 years of age this weight has become six times greater.
Respiratory[1]	✓ There is continued growth and development until 8 years of age. ✓ The number of alveoli increases until 3 years of age, after which time they continue to grow in size until 8 years of age. ✓ The bronchiole pathways continue to develop until 9 years of age. ✓ The lungs triple in weight from 5 years of age until puberty.
Renal[1]	✓ Young children have a larger surface area to volume ratio.
Digestive[1]	✓ Acid secretion in the gastrointestinal tract is lower until about 10 years of age.
Immune[1]	✓ The infant and child immune system requires many years to develop. ✓ IgG reaches adult levels by 4 years of age.

The cardiovascular system is one of those developing during these years. The six-fold increases in the weight of the heart from one to nine years of age (MacGregor 2008: 80) leaves ample room for developmental changes and impacts to take place. The nervous system is also engaged in rapid development over this period. While fully

formed by birth, the brain doubles in size within the first year and continues to develop at an accelerated rate until the age of five, making these first five years critical to development (Chamley et al., 2005). During this period, synaptic connections continue to develop (effectively building the neural connections) and are shaped according to individual experiences, making the period spanning these first years critical for child development (Bee and Boyd, 2010).

Lack of development in a number of systems has been shown to be tightly connected with childhood health and susceptibility to environmental stressors (Bogin, 1999). Elements of renal / urinary and digestive systems which are not fully developed during early childhood make children more susceptible to digestive issues and infection via that route (MacGregor 2008). The immune system, which is not fully developed during early childhood, also leaves a child more susceptible to infectious disease. Furthermore, MacGregor (2008: 184) notes that this system is particularly severely affected by chronic stress. The release of cortisol can negatively impact the development of the immune system, leading to varying levels of compromised immunity. The extent of this impact will be dependent upon the level, duration, and timing of the stressor. It will also be variable dependent upon a child's stress response to the situation (MacGregor 2008).

There is a growing body of clinical literature which is providing insight into the relationship between early childhood stress incidents and adult health (Barker and Osmond, 1986; Barker, 2001, 2004a; b; Gillman, 2005; Gluckman et al., 2005; De Boo and Harding, 2006; Simmons, 2009). This literature predominantly outlines the impact of particularly early life stress events (during the fetal period and early childhood); for

example the *Fetal and Infant Origins of Adult Disease* hypothesis) (Barker, 2001). Floyd and Littleton (2006) also found that stress in the first 18 months (as indicated by DEH) seemed to be particularly correlated with height for age, emphasizing that individuals living during this period of rapid growth are likely heavily susceptible to stress. However, the literature surrounding the connection between early life stress and later life health is relevant to the current investigation which considers slightly later childhood periods of development as it outlines a model by which later adult health can be impacted by early life stress. Cumulatively, the literature concerned with developmental origins and developmental plasticity suggests that health later in life is affected by environmental factors in early life, acting through the processes connected with developmental plasticity (Lasker, 1969; Bogin, 1999). The basis for these investigations lies in the susceptibility of rapidly developing systems to disruption due to the experience of stress. Continued rapid postnatal development during the period captured by dental enamel growth in the present study creates a context in which systemic disruption through stress experiences can continue to have a physiological impact. As such, the literature surrounding developmental plasticity is relevant to critical periods of development during childhood as well as to the fetal and infant periods.

Developmental or phenotypic plasticity is defined as "the ability of a single genotype to produce more than one alternative form of morphology, physiological state, and/or behavior in response to environmental conditions" (West-Eberhard, 1989, p 249). This has been observed in diverse contexts, from horned beetles (Moczek, 2010) to human sweat glands (Barker, 2007). Developmental plasticity can occur through epigenetic changes such as DNA methylation or histone acetylation, through tissue

differentiation during development, and through changes in homeostatic control mechanisms (Gluckman and Hanson, 2004a; b). Ultimately, these changes may have positive or negative consequences for later life health depending on a number of factors (including later life environment) (Gluckman and Hanson, 2004a; b).

An aspect of this relationship which has been heavily investigated both within the field of biological anthropology and beyond the connection between anthropometrics, morbidity, and mortality(Kaplan, 1954; Seckler, 1980, 1982; Beaton, 1989; Stinson, 1992; Saunders and Hoppa, 1993; Bogin, 1995, 1999; Bogin et al., 2002; Bogin and Varela-Silva, 2010). Specifically, shorter stature has traditionally been associated with growth stunting and may be taken as an indicator of potentially deleterious circumstances interfering with optimum development (Kaplan, 1954; Beaton, 1989; Stinson, 1992; Saunders and Hoppa, 1993; Bogin, 1995, 1999; Bogin et al., 2007). A complicating factor here derives from the interpretation of short stature, with the observation that people of short stature may not be negatively impacted in other aspects of their health. This observation led to the development of the 'small but healthy' hypothesis (Seckler, 1980, 1982, 1984), which has met with significant debate (Beaton, 1989; Stinson, 1992; Bogin et al., 2007).

In response to this, it might be suggested that a more nuanced understanding of the relationship between body height (and proportions) and health impacts is demanded (Bogin, 1999; Bogin and Varela-Silva, 2010). Specifically, we must once again turn to a threshold understanding of stressors and stress responses. The impact of a stressor on an individual results from a complex interaction between the nature of the stressor (type, duration, and severity) and the nature of the individuals' response to stress (in itself the

result of a unique interaction between individual biological / genetic factors, past experiences, and developmental timing, amongst other things) (Saunders and Hoppa, 1993; Kuzawa, 2005; Bogin and Varela-Silva, 2010). While the 'small but healthy' hypothesis effectively proposes that reduced height may be interpreted as an adaptation to environmental stressors (which may ultimately enable a person to survive better in a resource and energy deficient environment), such a reduction in size might be better seen as a compromise between developing systems, and an accommodation made in the absence of sufficient resources, which may be deleterious or neutral in ultimate health impact depending on the specific circumstances involved (Stinson, 1992).

The relationship between growth stunting and health is thus part of the same suite of changes in relation to stressors as dental enamel defects. It might therefore be expected that stress indicated in changing anthropometrics (stature, body proportions) might correlate with the specific indicators of stress events (such as dental enamel defects). It should be noted that this may be tempered in the current study which focuses on the survivors of childhood stress events, since it has been noted that the strongest correlation between mortality and growth disruption is visible in the first years of life (Saunders and Hoppa, 1993, p 140). Nevertheless, as already discussed, sufficient stress occurring at any point in development may interfere with any systems growing at the time of the stress, and this includes osteological growth. In this respect, Bogin and Varela-Silva's (2010) examination of anthropometrics in relation to the experience of stress events, with particular emphasis of proportional differences is relevant, as it draws attention to the importance of developmental timing. More specifically, those parts of the body growing at the time of the stress experience stand the risk of being most heavily

impacted. Boldsens' (1998) consideration of body proportions in the Danish medieval populations is also highly relevant in this scenario, and cautions against a simplistic consideration of the relationship between DEH and health by identifying an apparent impact of DEH on some proportional measurements but not on cumulative adult height. Bolden (1998) suggests that the influence of catch up growth is reflected in these different results. Similarly, Floyd and Littleton (2006) identify differential patterns between size and proportions in their study of DEH. This suggests a complex interaction between the nonspecific stress experiences expressed by DEH and growth, and in particular Floyd and Littleton (2006) emphasize the importance of timing of a stress experience in the outcome in developmental patterns.

The study of dental enamel also in itself has the potential to make useful contributions to our understanding of these relationships between stress events and any corresponding health impacts. The extended lifespan of humans makes studies of the relationship between childhood and adult health highly problematic. Within the medical community, only animal models and cohort studies have thus far been able to contribute to the picture of these relationships. Work in biological anthropology has also frequently demonstrated a connection between indicators of childhood stress (such as enamel hypoplasia) and mean age at death (White, 1978; Cook and Buikstra, 1979; Goodman and Armelagos, 1988; Armelagos et al., 2009). This area of research makes it possible to collect longitudinal information on patterns in individual life history from potentially very large samples (Armelagos et al., 2009).

These cumulative studies have contributed to a growing body of evidence for the impact of early life physiological perturbations on later life health in various forms. Such

an impact seems to occur if the stress is incurred during a range of developmental ages (Ulijaszek, 1996; Gluckman and Pinal, 2001). Effectively, the etiology of such mechanisms seems to be extremely complex, and may involve anything from nutritional deficiency and infectious disease at some stage during growth and development to environmental factors (Blackwell et al., 2001). The effects of these factors are variable and this relationship is therefore difficult to decipher. It has thus been proposed that stress during growth and development will impact whichever systems are developing at that point in time and that this will have an effect on health later in life in relation to the systems thus affected (Gluckman and Pinal, 2001; Roseboom et al., 2001). The developmental period under consideration in this thesis ranges from approximately 2 years of age to 6 years of age. While the relationship between stressors, individual frailty and stress response, and later life health outcomes is complex and not fully understood, the study of teeth developing during these years of life has the potential to provide insight on a longitudinal level into developmentally critical periods.

Looking at health

There are a number of characteristics which have been used to assess the other side of the question, namely assessing adult health in past populations from osteological remains. These include pathological changes associated with infection, activity-related skeletal changes, osteoarthritic changes, dental health, changes associated with metabolic conditions, and general parameters such as stature and longevity. While it might seem, from this, to be a straightforward process to identify these health indicators and to derive conclusions regarding the health of both individuals and the population from these forms of evidence, such a procedure is in fact fraught with difficulty, and has been challenged on a number of fronts. Of central importance to this discussion is the fact that the

individuals and population under consideration are not living individuals and populations (Wood et al., 1992). These populations are by nature fundamentally different and cannot be considered in exactly the same ways as modern, living populations. A brief discussion of the particular complications associated with this sort of study is therefore essential.

Some of the most fundamental theoretical challenges to palaeoepidemiology were those outlined by Wood et al. (1992) in the *Osteological Paradox* - namely those of demographic nonstationarity, selective mortality, and hidden heterogeneity of risks. Demographic nonstationarity refers to the incompatibility of skeletal samples with traditional demographic models which assume stationarity. Effectively, a more realistic state of affairs might involve a non-stationary population, i.e., one exhibiting some level of migration and fluctuating rates of age-specific fertility and mortality, and non-zero rates of growth. It is, however, the latter two challenges which are most directly critical to the current study. Selective mortality was based on the fact that of the people at risk at any given time in a population, we are only able to observe those who died at a particular age – those who were selected out of the population at risk to die (Wood et al., 1992). Hidden heterogeneity of risks means that different individuals in a population will be differentially susceptible to falling ill or to dying, but that we cannot know the frailty (or susceptibility) of everyone in the living population given the evidence available from the skeletal material. In connection with this arose problems of interpretation of skeletal remains, since not all ailments affect bone and since any illness has to be established for a more extended period of time before it becomes visible osteologically (Wood et al., 1992).

Roth (1992) suggests that heterogeneity of risk is the most problematic of the three issues outlined in the *Osteological Paradox*. Wood et al. (1992) point out that unless we have some way of telling how frailty differs between individuals, the determination of individual frailty from the mortality patterns in the population is extremely tenuous at best. Ultimately, the extent to which individuals are more or less susceptible to morbidity or mortality is something which cannot be known given the osteological evidence and current theoretical understandings. Indeed, even in modern populations this is something which is not fully understood! However, as per the discussion earlier in this chapter on the impacts of childhood stress on adult health, clinical research is beginning to suggest that early exposure to certain circumstances (malnutrition, allergens, respiratory infection, etc.) can have an impact on individuals' frailty (their risk of morbidity and mortality later in life). Unfortunately, within the archaeological setting it is challenging to identify which individuals in a population were exposed to which risks. Unless those risks left marks on the skeletal remains, all it may be possible to do is establish which risks might have been present in the environment. A bioarchaeological approach which integrates information on the context in which the people in the samples lived, might hope to inform this situation with some understanding of the possible risk factors to which they were exposed, but the direct connection will in many cases remain elusive.

Dental Enamel and Dental Enamel Defects

Teeth are formed in an incremental fashion, beginning at the enamel-dentin junction (EDJ). Odontoblasts (the dentin forming cells) are the first to differentiate, and as they begin to form dentin along the EDJ they stimulate the formation of ameloblasts (the enamel forming cells) (FitzGerald and Rose, 2008). Both odontoblasts and

ameloblasts move outwards in opposite directions from the EDJ (odontoblasts towards the pulp chamber and ameloblasts towards the crown) (FitzGerald and Rose 2008). Ameloblasts lay down enamel to form a lacework of prisms which extend towards the surface of the tooth, although a layer of prism-free enamel is found at the crown surface. Once the matrix secretion phase is complete, the process of maturation begins as the ameloblasts convert to break down the organic substance of the enamel, thus allowing the hydroxyapatite crystals to grow (FitzGerald and Rose, 2008; Hillson, 2005). When the tooth crown is complete, the enamel will be noncellular, and will consist of approximately 87% inorganic material by weight (or at least 95% by volume), most of which consists of hydroxyapatite (Brudevold and Söremark, 1967; Hillson, 1996, 2005; Risnes, 1998; FitzGerald and Rose, 2008).

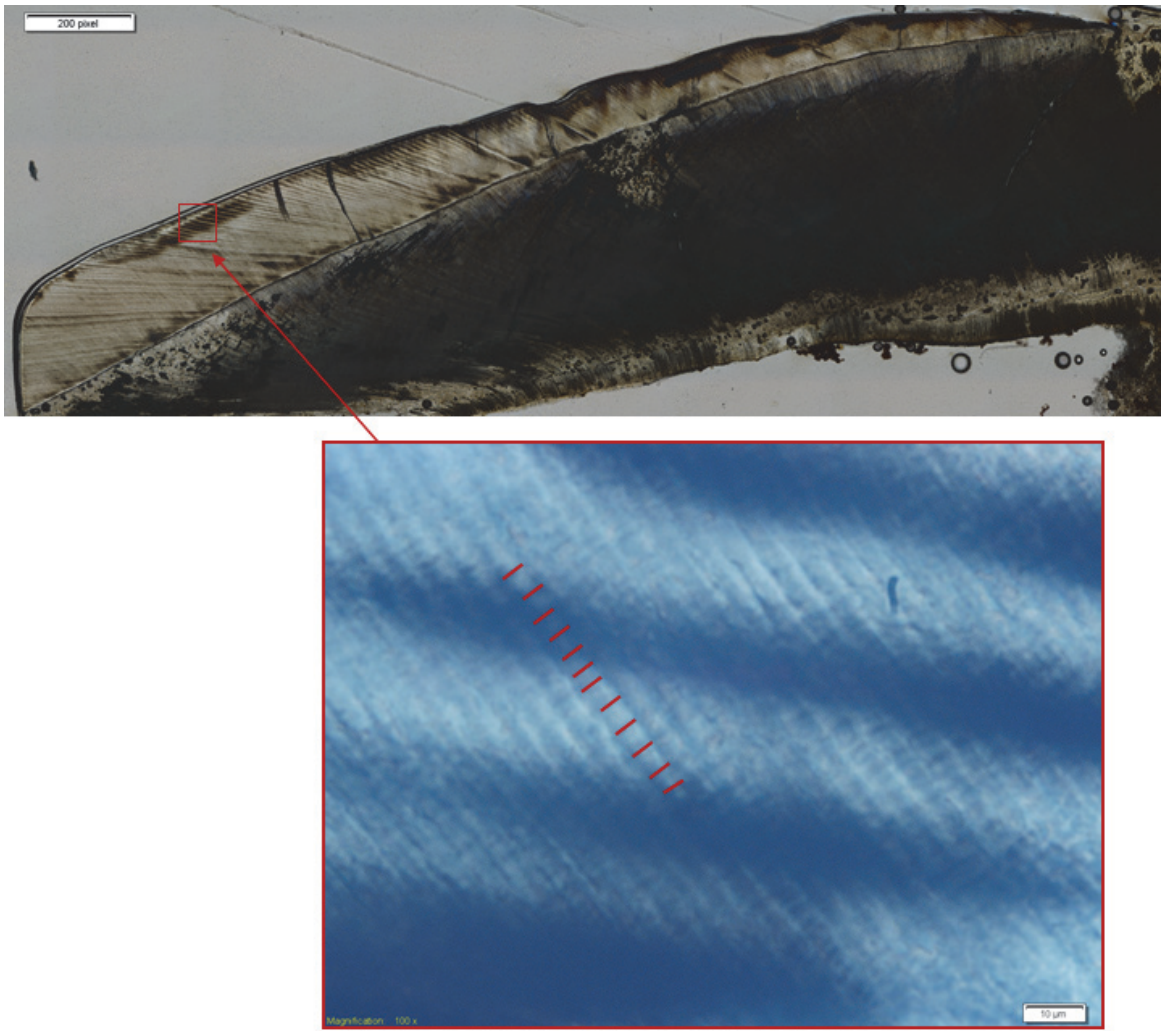
Enamel formation occurs at a much faster pace than dentin formation, since the process is more complex with dentin. The formation of dentin involves the initial deposition of a predentin matrix, and there is an intermediate process of matrix adjustment which occurs before mineralization can commence (FitzGerald and Rose, 2008). As such, enamel has been the focus of much of the research into tooth development. The faster pace at which enamel is formed makes it ideal for establishing a chronology (such as that established by Reid and Dean [2000, 2006]). Furthermore, enamel is more accessible for the researcher and tends to be simpler to interpret (given its less complex formation patterns) (FitzGerald and Rose, 2008). Understanding the details of enamel formation is essential for any study involving patterns in tooth development, including those which consider developmental defects.

The chronological markers in dentin and enamel can be divided into short and

long period features. The short period markers, known as cross-striations in enamel, are formed according to a circadian rhythm (Bromage and Dean, 1985; FitzGerald, 1998; Antoine et al., 2009) (Figure 2-1). These are effectively bands which run transversely across the prisms. As a result of their circadian rhythms, cross striations can be used to establish the age of an individual quite accurately when observed under a polarized light microscope or a scanning electron microscope[2]. The long period markers in enamel are known as brown or accentuated striae of Retzius after Anders Retzius (1837). Striae of Retzius are "incremental lines indicating the position of the ameloblast layer and of the developing enamel surface at different points of time" (Risnes, 1998, p 332). While individual prisms are continuously lengthening, the speed of this fluctuates in a rhythmic fashion, and in synchrony for all of the ameloblasts. As such, the striae of Retzius represent the pattern of movement of the growth front rather than strictly an incremental layering of material (FitzGerald and Rose 2008: 245).

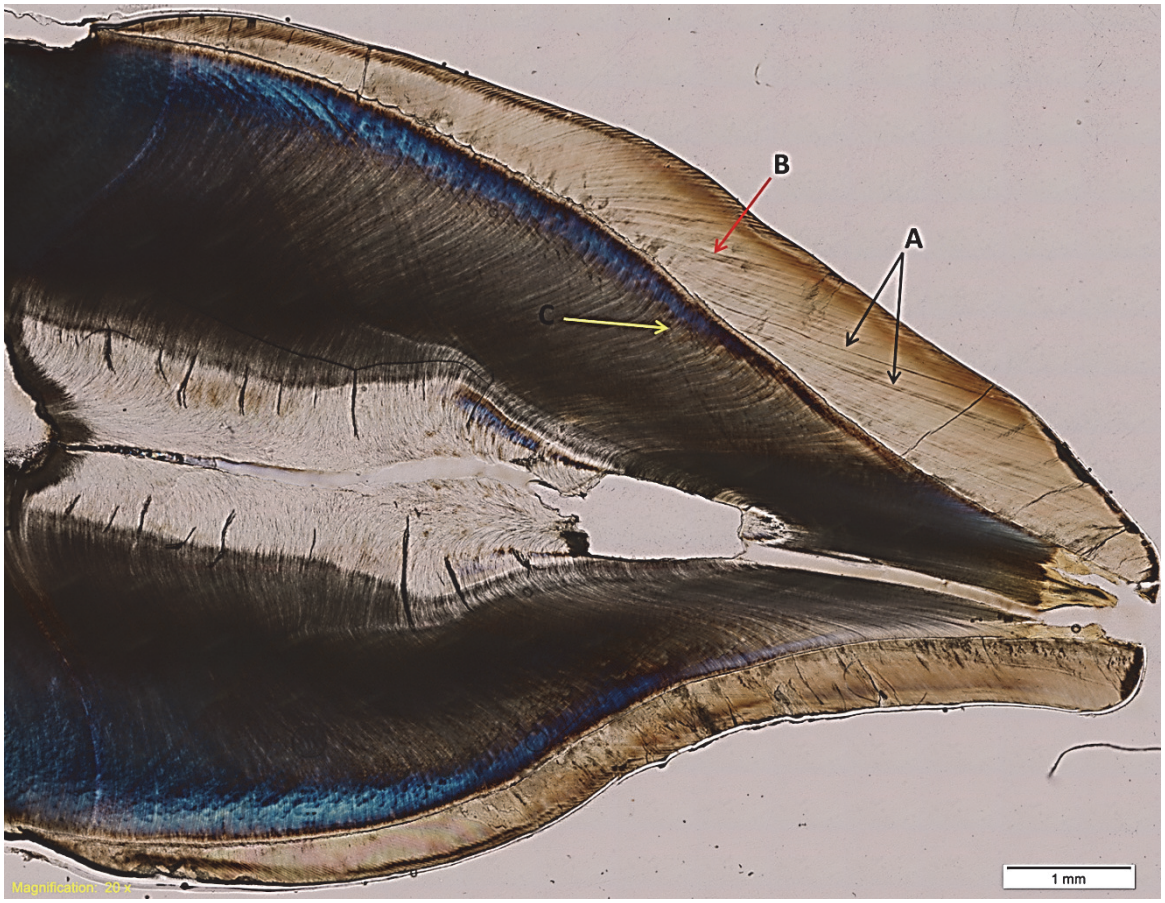
[2] This requires an anchoring point for the chronology, such as the neonatal line which can situate the rest of the markers in relation to birth.

Figure 2-1 Image depicting cross striations on the inset image (marked by short red parallel lines). (Sejet X116).



Striae of Retzius run outwards towards the surface of the crown, cutting across the prisms. They are evident on the surface of the imbricational (cervical) zone as ridges and grooves which collectively form wavelike patterns known as perikymata (FitzGerald and Rose, 2008; Hillson, 1996). The parallel structures in the dentin are known as peri-radicular bands (Bromage and Dean, 1985) (Figure 2-2).

Figure 2-2 Accentuated Structures with accentuated striae of Retzius marked (A, black arrows), along with an accentuated striae of Retzius (B, red arrow) with an associated peri-radicular line in the dentin (C, yellow arrow). (Ole Wormsgade X1019)



Towards the tooth cusp, in the appositional zone, the striae curve around in a domed pattern and are not apparent on the surface of the tooth. Bromage and Dean (1985) note that the first twenty to thirty striae (representing approximately six months of growth) are not visible on the surface of the enamel in the appositional zone in the human permanent incisors (FitzGerald and Rose, 2008; Hillson, 2005). This will vary according to both tooth type and individual, and must be taken into account when attempting to reconstruct chronology based on the enamel surface. Any research involving estimation of age from enamel microstructures must also consider the different rates of formation and spacing both within and across teeth (Hillson, 2005; FitzGerald and Rose, 2008).

The rate of enamel production also affects both the spacing and the angle of the striae in relation to the EDJ. Those located closer to the cusp exhibit a more acute angle (resulting in the domed pattern of striae in the appositional zone) and are generally farther apart (Hillson, 2005; FitzGerald and Rose, 2008).

In order to associate these structures with a chronological framework, it is essential to establish the length of time it takes each to form. While cross striations follow the circadian rhythm noted above (Hillson, 1996; FitzGerald, 1998; FitzGerald and Rose, 2008; Antoine et al., 2009), these are only visible upon close inspection of a section of enamel under microscope. Attempts have also been made to utilize striae of Retzius and perikymata to determine the age of an individual (FitzGerald and Rose, 2008; Hillson, 2005; Risnes, 1998; Antoine et al., 2009). When the number of cross-striations are counted between two adjacent striae of Retzius, a certain level of variability is found to exist between individuals (Risnes, 1998; Hillson, 2005; Reid and Dean, 2006; FitzGerald and Rose, 2008). At the most extreme ends of the scale, values have been recorded from five to fourteen, but they most commonly range from seven to ten (FitzGerald and Rose, 2008; Hillson, 2005; Reid and Dean, 2006; Risnes, 1998). Since the number of cross-striations between striae of Retzius average broadly around seven days, they are known as circaseptan rhythms or intervals (FitzGerald and Rose, 2008; Hillson, 2005; Risnes, 1998). The reason for this variability is unclear, but the count is consistent within a particular individual (FitzGerald and Rose, 2008). Ideally, this means that if an individuals' count can be established, it can be used across their entire dentition. If this is not possible, then it is best to calculate the possible range based on the outer limits of known variation (FitzGerald and Rose, 2008). In order to establish the exact age

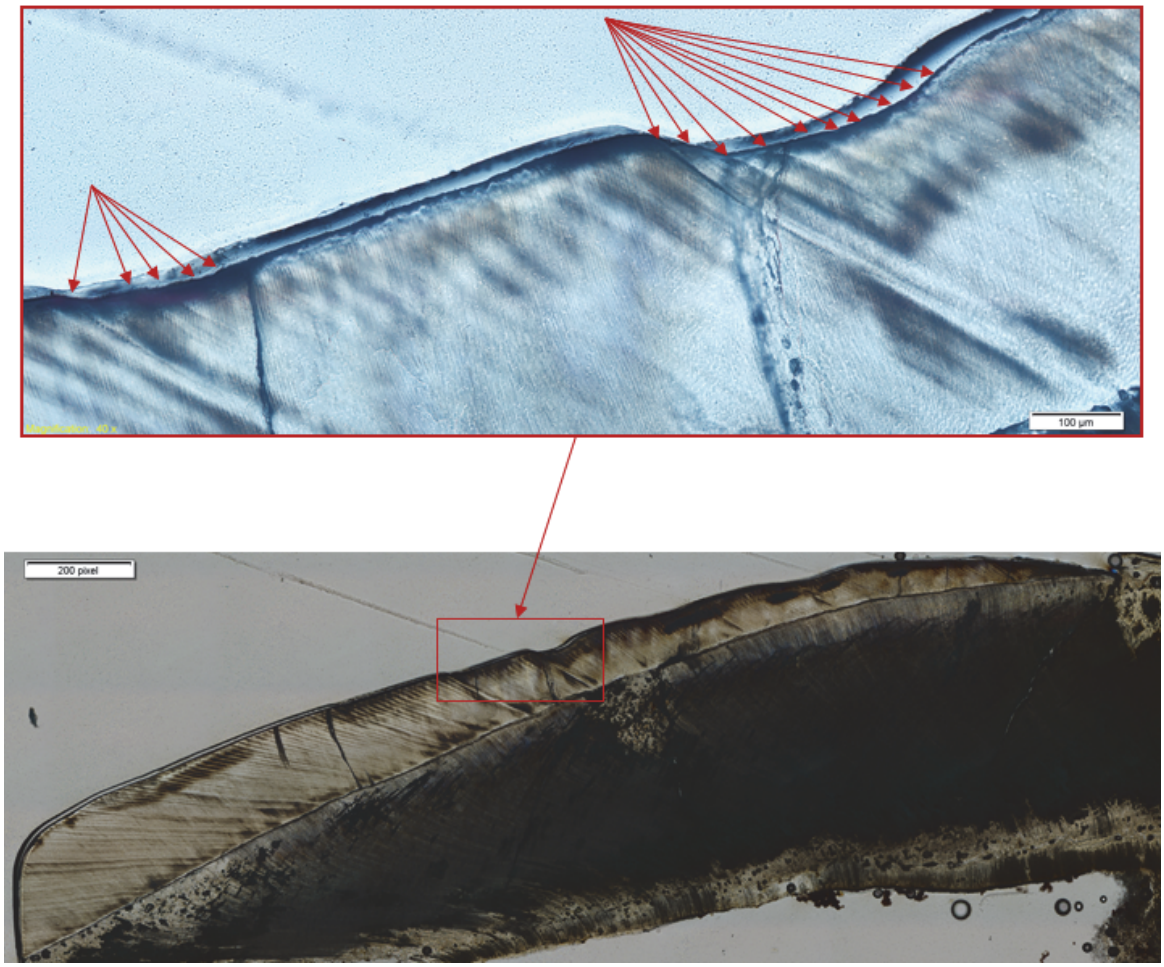
of occurrence of a defect in an individual, however, it is further necessary to be able to anchor the chronology at a certain point of time. In the case of dental enamel, a pronounced striae (known as the neonatal line) forms at birth and can be used as a point of reference. This may be preserved in the first permanent premolar cusp in adults if it is not extensively worn (Hillson, 1996; Hillson et al., 1999; Antoine et al., 2009).

Through the above account, it is clear that dental enamel is formed in an incremental fashion which can be measured in order to reconstruct the chronology of tooth development. Of central importance in this respect is the circadian rhythm of cross-striations and the relationship of these cross-striations to striae of Retzius in the form of circaseptan intervals (Reid and Dean, 2000, 2006). Common patterns of abnormal development include dental hypoplasias and Wilson bands (or accentuated striae of Retzius), hypocalcifications (or opacities), and discolourations (Hillson, 1996). All of these features are found in the enamel. Due to the higher visibility of these features in enamel and to the durability of enamel in archaeological remains, enamel defects have received the most attention and will be the focus of this thesis.

In general terms hypoplasias involve disruption during the enamel formation process while hypocalcifications are linked to abnormal mineralization (Hillson, 1996, 2005). Discolourations can result from either metabolic disorder during development leading to the deposition of different pigments, or from the staining of enamel deficiencies during mineralization (Hillson, 1996; Pindborg, 1982). Hypoplasias are effectively caused by disruptions of the perikymata at the enamel surface leading to "exaggeration of the perikymata [pkg], with a greater than normal pkg spacing down their occlusal wall" (Hillson, 1996, p 167) (Figure 2-3). One difficulty which has plagued

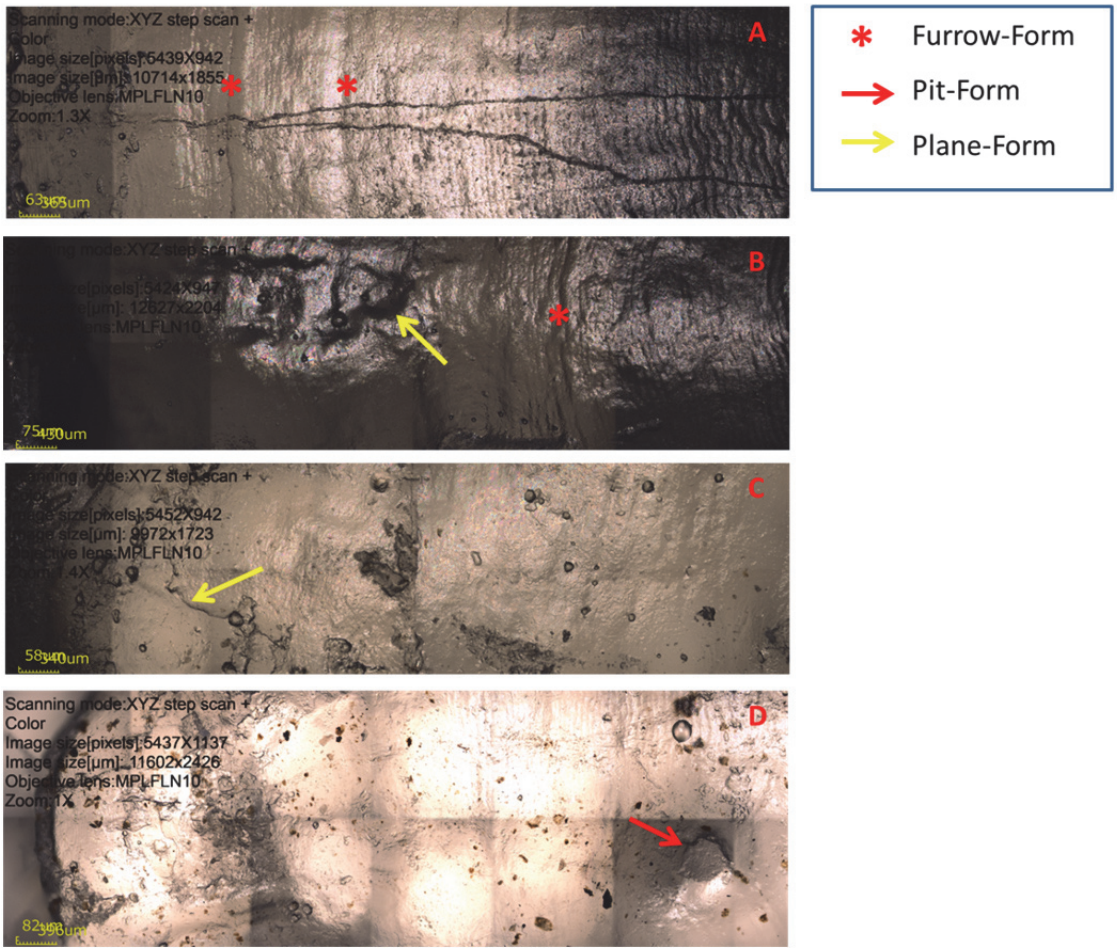
research involving enamel hypoplasia in the past involves a lack of agreement on the minimal expression of hypoplasia which should be considered (Cucina and Işcan, 1997, p 220). Goodman and Rose (1990) maintain that unless the hypoplasia is detectable macroscopically and can be identified using a probe, it should not be considered significant enough to reflect a specific health incident. Berti and Mahaney (1995) on the other hand, utilized an arbitrary scoring method of severity from A to D, and cautioned that a failure to recognize and record the most minor defects can result in the elimination of too many possible episodes of growth disruption. Hillson (1992, 1996) further substantiates this concern after examining dental microstructure and assessing spacing between perikymata. He suggests that even hypoplasias which affect only a single perikyma can be linked with systemic developmental disruption (Hillson, 1992a). It is this definition which will be utilized in the current study, in order to achieve the highest level of precision possible.

Figure 2-3 Image showing two hypoplastic events with perikymata involvement visible in the inset image. The most prominent accentuated striae of Retzius marking the beginning of each episode is visible towards the left of each defect (marked by the first arrows in each cluster). (Sejet X116)



Hypoplasias can be classified into furrow-type, pit-type, and plane-type defects depending on the extent and the nature of ameloblast involvement (Hillson 1996) (Figure 2-4). The furrow-type defects (linear enamel hypoplasias), are the most commonly found and are the simplest to interpret chronologically (Hillson 1996). They have received the most attention of all dental defects in the fields of bioarchaeology, palaeopathology, and palaeoanthropology and are the focus of the present research.

Figure 2-4 LEXT images showing examples of the three types of enamel defect. Two furrow-form defects (linear enamel hypoplasias) are marked by red asterisk in image A and two more occurring in close succession are marked by the asterisk in image B. Plane-form defects are visible as the edges of enamel fronts marked by the yellow arrows (image B and image C). A pit-form defect is visible in image D, marked by the red arrow). (Image A: Sejet X575; Image B: Sejet X477; Image C: Sejet X527; Image D Sejet X504)



Most work to date has concerned linear enamel hypoplasias (or furrow-type defects), which are "hypoplastic defects ... arranged in a band around the circumference of the crown, following the trend of the perikymata, and representing episodic disruptions to matrix secretion throughout the growing dentition" (Hillson, 1996, p 165). This form of defect only occurs in the imbricational zone of enamel (Hillson, 1996). Microscopically, they are usually apparent as an increased spacing of perikymata along the occlusal boundary (or wall) of the defect (Hillson, 1992a, 1996; Hillson and Bond,

1997; Guatelli-Steinberg et al., 2004; King et al., 2005), and they may affect anywhere from one to thirty perikyma (Hillson, 1996). This depends on a variety of factors, including the location on the tooth, the type of tooth affected, the severity and duration of the stressor, the nature of the stress factor, and the immune response of the individual (Brook et al., 1997). Witzel et al. (2008) suggest three thresholds of ameloblast response which can result in differential expression of enamel defects. They propose that factors which control which threshold is breached are the duration and intensity of the impact and the ameloblast susceptibility (dependent on the age and stage of the cell lifecycle).

Wilson bands (accentuated striae of Retzius) are generally accepted to be the histological manifestations of hypoplasias (Goodman and Rose, 1990; Hillson, 1996)[3], and have been defined as "any striae of Retzius exhibiting abnormal prism bending and absence or distortion of prism structure" (Rose et al., 1978, p 513). Goodman and Rose (1990) and Goodman et al. (1992) also differentiate between "Wilson" bands and "Cluster" bands. The latter is defined as "a Wilson band like stria … that extends from the enamel surface to one-half or less of the enamel width" (Goodman and Rose, 1990, p 93). The name which Goodman and Rose (1990) assign to this class of defect is derived from their observation that they normally appear in clusters. Rose (1977) further distinguished three subtypes of Wilson band. Distorted structure bands were effectively striae of Retzius which exhibited abnormal distortion of prism borders, while black spot pathological bands were similar to the distorted structure bands, but also had black spaces throughout the enamel. Finally, structureless pathological bands were bands which had "no normal structure along the striae" (Rose, 1977, p 443). Thomas (2003) distinguishes

[3] Although there is still some disagreement regarding the regularity of this association(Goodman and Rose, 1990)

between ‘weak’ and ‘strong’ accentuated striae. Strong AS were those obviously accentuated striae which are similar to the Wilson bands defined by Goodman and Rose (1990). Weak AS on the other hand were slightly less accentuated striae which still stood out against non-accentuated striae. In reality, the identification of AS or Wilson Bands is extremely complicated, since they exist along a continuum making the establishment of a threshold for identification extremely difficult. Their variable appearance across the crown length also makes the employment of a single definition problematic. The current thesis will integrate a nuanced approach which identifies AS based on their accentuated appearance in relation to surrounding striae in the particular crown region. The extent to which they extend to the surface and result in hypoplastic defects will also be taken into account.

In general terms, all dental developmental disruptions appear to result from stress during development. However, this stress can derive from three general sources (Pindborg, 1982). Developmental defects can result from trauma during development (Suckling, 1980; Suckling and Purdell-Lewis, 1982), can signify a hereditary response (Pindborg, 1982; Winter and Brook, 1975), or can be indicative of a systemic stress factor (Goodman and Rose, 1990; Hillson, 1996, 2005, 2008). Enamel disruptions arising from each of these different categories manifest in a variety of ways. Trauma-induced defects are primarily restricted to a localized area of the dentition (depending on where the stress occurred) and systemic disruptions typically affect all enamel which was in the process of development at the time of disturbance (Hillson, 1996). Hereditary defects (amelogenesis imperfecta), on the other hand, are typically severe and are often accompanied by other symptoms in the individuals affected (Pindborg, 1982; Winter and Brook, 1975).

It has been proposed that localized defects would have been relatively rare in past populations, a suggestion which has thus far been borne out in the literature (Goodman et al., 1980; Goodman and Rose, 1990; Hillson, 1996). Hereditary defects were also likely uncommon since their prevalence rate in modern populations is less than 1% (Winter and Brook, 1975; Witkop 1957 as cited by Goodman and Rose, 1990; Hillson, 1996). Systemic hypoplasias are the most frequently found in both archaeological remains and the modern population, and are therefore of the most relevance for research considering health in past populations (Goodman et al., 1980; Goodman and Rose, 1990). While it is necessary to establish a defect across all teeth developing concurrently to be sure that it is related to systemic stress, this can unfortunately be complicated in archaeological populations when studying internal microstructures. Namely, this would require the thin sectioning of multiple teeth in each individual, and is therefore highly destructive. The low rates of localized defects which are apparent from past studies make a diagnosis of systemic defect based on a single tooth more likely to be accurate, but without sampling multiple teeth the possibility of the defect being localized cannot be ruled out.

Research generally points to a causal association of dental defects with nonspecific systemic stress during development (Sweeney et al., 1969, 1971; Pindborg, 1982; Goodman and Rose, 1990). These stress episodes can result from nutritional deficiency (Mellanby, 1930, 1934; Klein, 1931; Sweeney et al., 1969, 1971; Pindborg, 1982; Seow, 1991) or from a systemic infection (including syphilis, measles, and any number of childhood illnesses leading to high fevers) (Sarnat and Schour, 1941; Sweeney et al., 1969, 1971; Pindborg, 1982; Goodman and Rose, 1990; Seow, 1991) or even with psychosocial stress (Schwartz et al., 2006). Sweeney *et al.* (1971) found a correlation

between hypoplastic defects and malnutrition in Guatemalan children, and a study by Infante and Gillespie (1974) further indicated that rates of hypoplasia varied according to season and familial association in a population of 429 Guatemalan children from four villages. They suggest that factors operating at the family level had an impact on hypoplasia. A later study by Goodman et al. (1991) found that children who received nutritional supplementation from birth were nearly half as likely to display hypoplastic defects as those from the control group who had not received supplementation. However, while these findings support a causal connection between hypoplasia and nutritional status, it remains a nonspecific relationship as it is unclear whether specific nutritional factors have a greater impact than others.

The apparent connection to nutritional intake is unlikely to be as straightforward as inadequate consumption resulting in disruption in dental development. Research into other etiological connections to dental defects suggests that any factor which can impede metabolic activity might have an impact. In a study of Guatemalan children by Sweeney et al. (1969), a correlation was identified between rates of LEH and rates of infection during the first month of life. More specifically, higher rates of faeces-virus isolations and premature births were identified amongst children with hypoplastic defects. Suckling et al. (1983, 1986) have conducted a series of experimental studies on sheep which showed associations between hypoplasia and parasitic infection. Further research by Suckling and Pearce (1984) on children in New Zealand showed a significant correlation between enamel defects and history of either serious illness or accident. An association between psychological conditions and brain damage (Judes et al., 1985) is also indicative of a more complex etiology for dental defects, and Schwartz et al. (2006) found an

association between dental enamel defects and stressful situations (surgery and follow-up visits) in a captive gorilla.

Dental developmental defects have had a significant impact in the field of bioarchaeology as a result of their potential as windows into the health and nutritional status of past populations. The recognition of these features as nonspecific indicators of stress trapped in a chronological sequence which can theoretically be reconstructed has sparked a series of investigations (Goodman et al., 1980; Armelagos et al., 2009). Furthermore, the fact that dental defects are retained throughout life makes it possible to apply the same standards to multiple sectors of the population – in this case to both adults and subadults. As such, researchers are able to investigate patterns in both survivors and non-survivors (Goodman and Armelagos, 1988; Goodman, 1996; Thomas, 2003). This may make it possible to identify patterns in the relationship between stress factors occurring earlier in life and health at a later stage, and it is this concept which is central to the current investigation.

Adult indicators of health

The above section highlights the key component used in this investigation to track childhood health experiences in the form of nonspecific markers of systemic stress. Dental enamel structures are useful in that they may offer insight into childhood health in adult remains where remodelling has otherwise obscured much of this information. However, in order to gain insight into the impact of early life stress incidents on later life health, it is necessary to look at other health parameters which can be identified from the adult skeletal remains. This thesis is focused on looking at those individuals who survived any childhood stress experience and lived to adulthood. Health indicators that

can be recovered from adult skeletal remains include stature and body proportions, age at death, and signs of infectious disease. The evidence for these health indicators will be briefly reviewed in this section.

Stature and Body Proportions

One feature which has been heavily linked to environmental factors such as nutritional deficiency and disease is stature and patterns in human growth (Tanner, 1963, 1981a; Eveleth and Tanner, 1976; Fogel et al., 1983; Mensforth, 1985; Boldsen and Iregren, 1990; Floud et al., 1990; Lovejoy et al., 1990; Saunders and Hoppa, 1993; Roberts, 1995; Steckel, 1995, 2004; Boldsen, 1998; Bogin, 1999; Hoppa and Fitzgerald, 1999; Larsen, 1999; Lewis, 2002; Steckel and Rose, 2002; Bogin and Varela-Silva, 2010; Watts, 2011; DeWitte and Hughes-Morey, 2012). Stature is an multifaceted feature, with final adult stature representing a complex interaction between genetic and environmental variables (Eveleth and Tanner, 1976; Tanner, 1981b; Floud et al., 1990; Bogin, 1999; Steckel and Rose, 2002). While there is a genetic component to stature which has been considered in relation to past populations (Boldsen, 1990b; Roberts and Manchester, 2005), the observation that individuals from more affluent backgrounds tend to be taller has led to an association being made between stature on the one hand and lifestyle, health and other environmental factors on the other (Eveleth and Tanner, 1976; Addyman, 1989; Floud et al., 1990; Komlos, 1990, 1993; Steckel, 1995; Bogin, 1999; Larsen, 1999; Steckel and Rose, 2002; Steckel et al., 2002; Cardoso and Gomes, 2009). Stature can be impacted by stress during growth and development, although it is also subject to catch-up growth (Tanner, 1963, 1981 a, c; Steckel and Rose, 2002). Prolonged nutritional deficiency (which can be influenced by disease load and activity patterns) can cause more permanent growth stunting which may be visible in adult skeletal remains (Bogin, 1999;

Steckel and Rose, 2002; Steckel, 2004). Ultimately, as has already been discussed in the section on childhood stress and adult health, such stress incurred during growth and development and any subsequent catch-up growth will result in a final stature and alteration of other body proportions in association with those specific growth components which were impacted by these two processes (i.e., growth disruption and catch-up growth).

Given this background, stature estimation has become a common component of osteological analyses which seek to learn about patterns of health in the past (Mensforth, 1985; Elo and Preston, 1992; Steckel, 1995, 2004, 2005; Boldsen, 1998; Gunnell et al., 2001; Steckel et al., 2002; Roberts and Cox, 2003; Kemkes-Grottenthaler, 2005; Koepke and Baten, 2005; Roberts and Manchester, 2005; Watts, 2011). These studies have considered secular trends (Trotter and Gleser, 1951; Fogel et al., 1983; Boldsen, 1990b; Komlos, 1993; Jantz and Jantz, 1999; Roberts and Cox, 2003; Kemkes-Grottenthaler, 2005; Koepke and Baten, 2005; Giannecchini and Moggi-Cecchi, 2008; Cardoso and Gomes, 2009; Kaupová et al., 2013), differences between urban and rural settings (Boldsen, 1990b; Steckel, 1995; Komlos and Baten, 2004; Koepke and Baten, 2005; Yoder, 2006; Kaupová et al., 2013), possible evidence for social patterns within societies (Boldsen, 1990b; Koepke and Baten, 2005), and connections between stature and other health indicators (Boldsen and Iregren, 1990; Gunnell et al., 2001; Kemkes-Grottenthaler, 2005; DeWitte and Wood, 2008; DeWitte and Hughes-Morey, 2012).

While these studies have identified correlations between stature and various health indicators (such as longevity and enamel defects), such patterns are not always apparent. Blackwell et al. (2001) found no connection between adult height and childhood health

conditions in a longitudinal clinical study. Boldsen (1998) found no connection between adult height and enamel hypoplasia but did find a connection to body proportions. Likewise Jantz and Jantz (1999) identified changes in body proportion in their consideration of secular change 19th and 20th century American samples, and proposed that body proportions and in particular long bone proportions change in relation to environmental stress. Bogin et al. (2002) in their comparison of American born versus Guatemalan born children of Mayan descent also found that the proportion of leg length to overall stature was a strong indicator of environmental stress. This relationship is explored further by Bogin and Varela-Silva (2010), who note that a divergence from the expected ratio of leg length, for example to trunk length (specifically a high sitting height ration with shorter legs in proportion to sitting height) is a marker of negative health impacts during early life.

These results reflect the complex nature of growth and development, with the duration and timing of an environmental stressor having an effect that is in turn dependent on the developmental stage of the different elements at the time of occurrence (Steckel and Rose, 2002). While the various osteological elements represent slightly different periods of growth in subadults, the final stature of an individual will reflect the cumulative growth stunting of those elements contributing to the final stature measurement. Thus, different measurements and comparisons will reflect different processes and snapshots into growth and development. Any interpretation must take these factors into consideration.

An important component to any consideration of stature and body size in relation to health is the mode of measurement used in the study. One methodology which is not

population specific and which provides quite accurate results (Maijanen, 2009) is the anatomical method, in which skeletal elements which contribute to the height of an individual (axial skeleton, lower limbs, cranium) are all measured and then added together with a soft tissue factor applied (Fully, 1956; Fully and Pineau, 1960; Raxter et al., 2006, 2007). However, this method is problematic since archaeological skeletal remains are frequently too fragmentary to acquire the requisite measurements (Giannecchini and Moggi-Cecchi, 2008). A common way of considering stature from skeletal remains is through the application of regression formulae to long bone lengths (Dupertuis and Hadden, 1951; Trotter and Gleser, 1958; Genovés, 1967; Trotter, 1970; Formicola and Franceschi, 1996), which is less demanding in terms of the level of preservation required. These regression formulae are based on the calculated relationship between stature and the respective long bone (White and Folkens, 2005; White et al., 2012).

Unfortunately, since there are population differences in body proportions as they related to height (Genovés, 1967; Ruff, 2002; White and Folkens, 2005), the application of such formulae to populations outside of the one from which the formula was developed has been shown to introduce bias by a number of researchers (Dupertuis and Hadden, 1951; Trotter and Gleser, 1952; Boldsen, 1984a; Sciulli et al., 1990; Formicola, 1993; Konigsberg et al., 1998). Formicola (1993) found that while some regression formulae (Pearson, 1899; Trotter and Gleser, 1952, 1977; Olivier et al., 1978) produced relatively compatible results with the anatomical method (Fully and Pineau, 1960) (although there were still sex differences in accuracy), others were more problematic (Breitinger, 1937 as cited by Formicola 1993; Bach, 1965 as cited by Formicola, 1993). In relation to

medieval Danes, Petersen (2005) found that the Trotter and Gleser (1952, 1958) regression formula showed a 4 cm bias on a Danish sample, cautioning against the use of such standard formula for this population. One feature which is apparent with a number of regression formulae is the tendency for underestimation of the tall range and overestimation of the short range, leading to a concentration towards more central stature estimates (Formicola and Franceschi, 1996; Maijanen and Niskanen, 2006).

Konigsberg et al. (1998) demonstrate that traditional physical anthropological techniques for stature estimation based on long bone lengths use inverse calibration which is based on regressing stature on long bone length, effectively taking a Bayesian approach to the problem and employing an informative prior. In doing this, the stature estimates based on long bone lengths are influenced by the stature distribution of the model population, which is problematic if this distribution is not a compatible fit with that of the study population.[4] An alternative approach is to regress long bone length on stature and solve for stature using a uniform prior. This is consistent with a classical calibration, employing maximum likelihood principles (Konigsberg et al., 1998). This calculation is shown to produce unbiased stature estimates, and is thus more appropriate or an archaeological sample does not have a known stature distribution (Konigsberg et al., 1998). Furthermore, Konigsberg et al. (1998) show that the more commonly used inverse calibration, while more accurate if the true stature is close to the mean of the distribution, becomes less accurate farther away from this mean while classical calibration is more accurate. It is these patterns for inverse calibration that result in the observed biases in

[4] Inverse calibration is favourable if the population distribution of the model population is compatible with that of the study population, as it does generate the lowest mean squared error (Konigsberg et al., 1998).

regression estimates for situations where the model population does not necessarily fit the study population.

There have been various attempts to deal with the issues associated with regression formulae. Sjøvold (1990) has developed "*the weighted line of organic correlation*" as a broad-spectrum line of fit to adjust for unknown population of origin (i.e. this is a method which is broadly applicable regardless of genetic background and sex). Such an approach is particularly helpful when it is not possible to generate population-specific formulae and where population is unknown (as may be the case in forensic cases and for ancient remains). Konigsberg et al. (1998), however, caution that this approach, which represents a compromise between inverse and classical calibration, might be inappropriate in situations requiring significant extrapolation to estimate stature. Giannecchini and Moggi-Cecchi (2008) used the Delta of Gini parameter to test applicability of different regression formulae to their sample by testing compatibility of stature estimates derived from different long bone lengths. Other population specific regression formulae have also been developed for a number of populations around the world (Telkkä;, 1950; Genovés, 1967; Boldsen, 1984a; Sciulli et al., 1990; Sciulli and Giesen, 1993; Sciulli and Hetland, 2007; Auerbach and Ruff, 2010; Maijanen and Niskanen, 2010; Béguelin, 2011).

In response to these issues with regression formulae Boldsen (1990) suggested that any consideration of height in medieval Danish populations should utilize either the direct measurement of height in the grave or a consideration of untransformed long bone lengths (specifically the femur). Later research by Petersen (2005, 2011) has further supported the accuracy of stature in the grave measurements over Trotter and Gleser's

(1952) sex-specific formulae, and has added a correction factor of 2.5 cm to this measurement (Petersen, 2011). This is echoed by Maijanen and Niskanen (2006) and (Maijanen, 2009) who recommend the use of anatomical methods over regression formulae wherever possible. Other researchers have also incorporated raw long bone measurements into their analyses (Gunnell et al., 2001; Kemkes-Grottenthaler, 2005; DeWitte and Wood, 2008; Jatautis et al., 2011; Meiklejohn and Babb, 2011; DeWitte and Hughes-Morey, 2012; DeWitte and Slavin, 2013).

Infection

The consideration of infectious disease prevalence rates in cemetery populations is a highly complex issue, as outlined in the Osteological Paradox (Wood et al., 1992). While it is possible to identify from the skeletal remains a limited number of diseases which are present in the population (such as leprosy, treponema, rickets, and tuberculosis) (Aufderheide and Rodriguez-Martin, 1998), care must be taken in diagnoses. Extending this beyond the diagnosis of individual cases to infer disease prevalence rates in the population introduces further layers of difficulty. Once again, we come back to the issues highlighted by Wood et al. (1992). In the *Osteological Paradox*, it is noted that due to heterogeneity of risk and selective mortality, the frequency of lesions in a skeletal sample will always be higher than in the living population. This is because death itself is a selective process in which people who are at the most risk of dying at any given age due to any particular factor (whether it is visible osteologically or not) are the most likely to enter the sample.

Furthermore, given that at least a portion of the lesions visible on the skeleton will be related to the respective frailty of the individuals in the skeletal sample, it is expected

that there will be a higher proportion of lesions amongst the dead than amongst the living (less frail individuals will persist to a greater extent). This is particularly the case for active lesions, since they are more likely to be related in a direct way to the death of an individual. As such, Wood et al., (1992) emphasize the importance of distinguishing between active and inactive lesions. They also note that since we do not know the actual risk of any given lesion to the living population, "it is impossible to predict from modern population prevalences ... the proportion of a skeletal sample that would be expected to show a hard-tissue response" (Wood et al., 1992, p 349). A biocultural approach may be able to inform us further as to the types of disease and potential risks of exposure within a population, but this can only ever be an estimate. We can infer from demographic patterns whether population density or exposure to environmental conditions conducive to zoonotic or parasitic diseases would have influenced the health profile of a population, but we cannot decipher differences in individual risk of exposure, nor in differential individual frailty.

While Wood and colleagues (1992) engage in an exercise to reconstruct the lesion frequency in the living population from a skeletal population, they also caution that a number of assumptions (such as stationarity) are necessary in order to come to any such estimation, and that if these cannot be upheld it is not possible to estimate lesion prevalence in the living population from the skeletal remains. It is clear that lesion frequencies in the skeletal sample do not therefore directly reflect those in the living population. Instead,

> if a skeletal lesion – or the condition responsible for it, or a trait predisposing to that condition, or another trait highly correlated with that condition – has any

> relationship whatsoever to the risk of death, the skeletal collection must be a biased sample for the living population (Wood and Milner, 1994, p 635).

Furthermore, any attempt to compare relative lesion frequencies across different skeletal samples is problematic (Wood et al., 1992). Such estimates reflect the frequency of a particular lesion in relation to other lesion types in a particular sample. They therefore innately contain information with respect to the particular proportions in a given sample, i.e. they are sample-specific. This sample specificity may relate to differential individual frailty, differential exposure within that population due to any number of factors, or even differential preservation. Of extreme importance here is that unless the skeletal samples being compared have the same cause of death distributions, we are not comparing like with like and any conclusions can be problematic (Wood et al., 1992). The situation is especially complex when it comes to the consideration of changing patterns of health over time, such as during an epidemiologic transition. Since it is rather difficult to be absolutely certain of all competing causes of death to which a mortuary sample would have been exposed, we cannot be sure that we are comparing populations with the same distributions.

For the purposes of the current research, the consideration of differences in disease between the two cemeteries, and over time, will therefore be approached with caution. Waldron (2009) advocates the use of a common odds ratio in order to compute a summary statistic for prevalence rates within a particular population. This is a simple way to look at differences in disease prevalence rates across populations, but once again any interpretations should be treated with caution consistent with the limitations of these data and with the biases identified by Wood et al. (1992). There are also complications regarding what the presence or absence of skeletal lesions means. This question was

addressed by Ortner (1991), received particular attention in relation to porotic hyperostosis by Stuart-Macadam (1991) and was further considered by Wood et al. (1992). The difficulty in interpretation here lies in the nature of osteological response to disease. Bones can only remodel in a limited number of ways, and so a particular osteological reaction may be exhibited by different types of illness, i.e. bones demonstrate low diagnostic specificity (Wood and Milner, 1994). Furthermore, bones often register a delayed response to infection due to the speed of bone remodelling. As such, a disease episode has to last long enough for the bone to be affected (thus exhibiting low sensitivity to disease), and there are a number of diseases which do not affect the bone at all (Wood and Milner, 1994).

Ultimately, the extent of the osteological involvement and the nature of the response will depend on a number of factors, including an individuals' immune response. As a result of this, an individual who displays no skeletal pathology may represent a healthy individual, they may represent an individual who was never exposed to the disease, or they may have been an individual whose immune response was not sufficient to ensure survival long enough for their bones to develop pathological formations (Ortner, 1991). In other words, the absence of lesions may equally represent high or low frailty (Wood et al., 1992). Alternatively, the opposite may be true if lesions are present (Ortner, 1991; Wood et al., 1992).

The two samples considered in this thesis will be assessed for lesions characteristic of three main diseases, namely leprosy, tuberculosis, and treponematosis. The results will be considered in light of the varying interpretations suggested under the osteological paradox, and the temporal and regional context will be used to help inform

the interpretation of the results based on factors such as differential exposure to disease over time and according to rural versus urban settings. Furthermore, the consideration of dental enamel defects in association with disease manifestation will be engaged in to help inform potential differential frailty of individuals. Ultimately, the consideration of overall stature will capture cumulative growth over the course of development while signs of infectious disease will reflect the potential impact on health of stress experiences during growth and development (in the form of disease response).

Age at Death

While patterns in infectious disease might reflect particular conditions experienced by individuals in a population, age at death might be used to further contribute to our understanding of general survivorship in relation to early life stress experiences. Age at death can be a useful indicator of health under the hypothesis that healthier or more robust individuals will be able to survive the stresses of life longer and will therefore attain an older age at death. In the current study, all individuals considered were adults, and were therefore survivors of any childhood stress events. The factor which is then of interest is how long these individuals were able to survive childhood. To gain insight into these questions, it is ideally necessary to go further and to identify as closely as possible how old each individual was when they died.

While age estimation in children can be relatively straightforward, with reasonably tight age ranges, age estimation in adults is far more problematic (Bocquet-Appel and Masset, 1982). Unlike children, where age estimation is based on set processes of growth and development, age estimation in adults depends on wear and tear factors (Bocquet-Appel and Masset, 1982). Since this will be heavily impacted by

individual circumstances, it can be highly variable, and over a lifetimes' accumulation this difficulty is only compounded. It may not be possible to assign an age to an individual which is more specific than 'above 30' or 'older adult', and indeed, being more cautious and acknowledging limitations is far preferable than to extending interpretation too far in an attempt to get more precise numbers.

Unfortunately, from a statistical standpoint, frequently point estimates of age are required, and so it is common practice to use the midpoint of this age range as an approximate age of the individual. As will be discussed shortly, statistical methodologies are being developed which will allow more informed point estimates to be calculated (Weise et al., 2009; Milner and Boldsen, 2012a), but fragmentary remains such as those used in this study frequently create a challenge even for these techniques. In the meantime, it is essential to acknowledge that point estimates drawn from age ranges may cloud results, particularly if an individuals' actual age was to one or the other extreme of the age range. Such a practice, while elucidating broad patterns, will not be able to provide extensive detailed insight, particularly when the point estimates are then used to divide individuals into pre-defined age categories. The details for age estimation employed in the current research are outlined in more detail as part of the methods section, but such a practice of deriving point estimates was necessarily employed for the statistical analysis in this thesis. Wherever possible, the full range should therefore be incorporated into analysis, and this is also done where possible when age is considered in the following analysis.

A further complication to age estimation involves the influence of 'age mimicry' in which there is the tendency for the target skeletal age distribution to mirror the age

distribution of the reference sample (Bocquet-Appel and Masset, 1982, 1985). Bocquet-Appel and Masset (1985, p 111) cautioned that "it is impossible to estimate skeletal age without bias, unless we know before hand the age structure at death of the population to which it belongs," and of course we do not know this for a mortuary collection. Ideally, then, caution is necessary when applying strict age estimation techniques which have been developed using another population to the one being studied, and it is far more advisable to establish individual ages based on their situation within the distribution pattern that characterizes the particular sample of which they form a part (Hoppa and Vaupel, 2002).

There have been some promising inroads into our ability to accurately reconstruct age profiles through collaborative work being conducted out of the Max Planck Institute for Demographic Research (Weise et al., 2009; Milner and Boldsen, 2012b). This method involves the development of an expert age estimate through the application of statistical calibration to skeletal age estimation techniques. This method incorporates level of uncertainty into the calculations and employs all available age indicators for the age assessment. This method is in the process of further development and awaits more extensive publication. Its emphasis on the use of a broad overview of age indicators, particularly for fragmentary skeletal remains, in addition to the statistical application, is encouraging. As will be described in the Materials and Methods chapter, this first element (expert inference) was incorporated into skeletal age assessment for the current study.

As can be seen, while age at death has the potential to provide insight into generalized adult health, its inference is complicated by a number of factors. Any

interpretation based on these inferences must be treated with caution. The same might be said for the other health indicators which have been discussed (stature and body proportions, and infectious disease indicators), although they all have their unique challenges. The identification of these parameters, however, forms the basis for all components of this investigation. These challenges are not insurmountable, but must temper interpretation. The consideration of childhood stress parameters such as dental enamel defects in association with these adult health indicators has tremendous potential to provide insight into how humans respond to stress during development on a longitudinal level. This insight can be further informed through a nuanced approach with incorporates a contextual understanding of the sociocultural and environmental factors at play in these populations. The consideration of changes over time and differences between the two samples in this study will provide a backdrop to aid in the interpretation of the results while at the same time acting as an investigation in its own right to contribute to our understanding of these past populations.

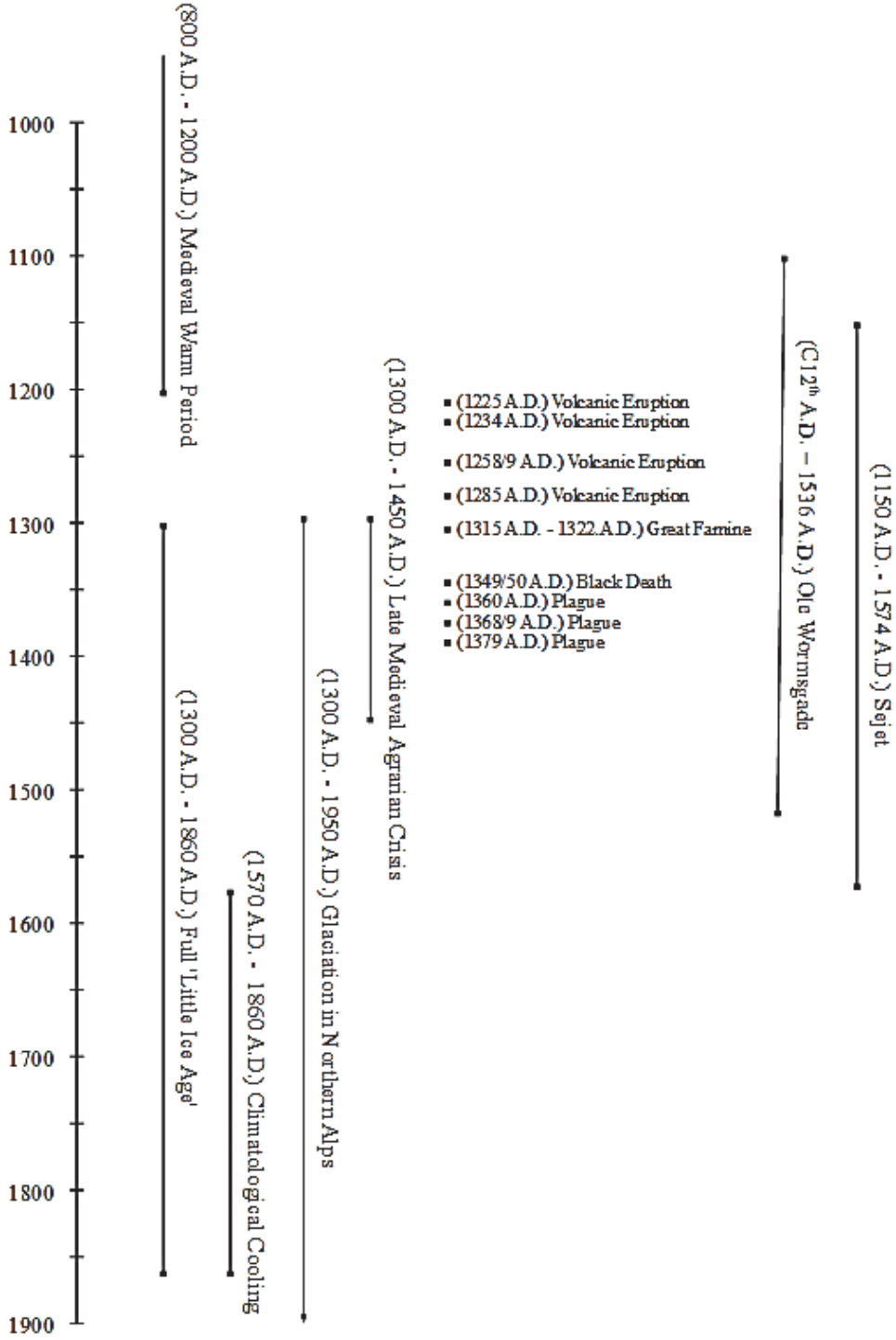
Danish medieval period and regional description

An important consideration for this research was the contextual background for the populations being studied. The extent to which health may or may not have changed over the course of the medieval period in Denmark in response to such events as periodic famine and diseases such as Black Death was of primary concern. Furthermore, as a gradual shift towards urbanisation occurred over this period, the health response of the respective urban and rural populations is of interest to this research. The following section will review the various forms of evidence which can provide contextual insight into these aspects of the investigation.

Temporal Patterns in Medieval Denmark

The medieval period in Denmark lasted from the end of the Viking period in 1050 A.D. to the beginning of the Reformation, in 1536 A.D. (Bennike, 1985; Boldsen, 1990b; Boldsen and Mollerup, 2006) (Figure 2-5). This broader medieval period can be roughly broken into two sub-periods, bordering the mid-14th century. The earlier medieval period has often been associated with the end of the Medieval Warm Period (800 A.D. -1200 A.D.), while the later period is marked by the beginning of what became known as the Little Ice Age (14th century A.D. to the mid-19th century A.D.) (Lamb, 1995; Fagan, 2000; Broecker, 2001; Grove, 2001, 2003). The period from the 14th to the mid-15th centuries has been associated with socioeconomic hardship, population decline, village desertion, and agricultural changes and has been formulated as the 'Late Medieval Agrarian Crisis' (Andrén, 1985; Kristensen, 1999; Orrman, 2003; Yoder, 2006; Antonson, 2009). This period can further be divided by the entrance of Black Death into Denmark in 1349 A.D. (Benedictow, 2004), and it is this which might be seen as more distinctly dividing the period into early and late medieval. More recent evidence is increasingly questioning the concept of a 'Medieval Warm Period' followed by a distinct 'Little Ice Age' commencing in the mid-14th century (Matthews and Briffa, 2005; Nesje et al., 2008). In order to better understand the circumstances which impacted the populations living over this time, it is therefore essential to review the literature surrounding these concepts.

Figure 2-5 Timeline for the medieval period showing the main date ranges (shown as lines) and key events occurring over the course of the period (shown as points). Information is cumulative from sources discussed in the associated text.



Lamb (1965) considered the historical evidence in conjunction with palaeoclimatic data (such as cyclonic activity). He concluded that a 'Medieval Warm Period' consisting of dry summers was followed by a 'Little Ice Age' characterized by dry winters. Historical evidence points to less sea ice in the arctic, allowing for safe and more open sea travel in the earlier period and Lamb (1965) notes the presence of Norse burials in southwest Greenland and plant roots in areas which are now dominated by permafrost (see also Grove, 2001, p 63). The presence of vineyards in England is also cited as evidence in support of warmer temperatures during the first few centuries of the second millennium. The records also suggest the increase in sea ice blocking shipping routes and harsh conditions contributing to the demise of the Greenland settlement. A rapid retreat of populations from marginal lands from the 14th century onwards and a loss of northern vineyards is also connected with increasingly harsh conditions (Lamb 1965). It is in this evidential background that the concepts of the 'Medieval Warm Period' and a 'Little Ice Age' dated to the mid-14th century onwards are founded, and it was this early work of Lamb which first brought the concept of a 'Medieval Warm Period' into the literature.

The concepts of a 'Medieval Warm Period' and a 'Little Ice Age' have been broadly incorporated into the literature surrounding studies of this period. However, a deeper consideration of climatic patterns suggests that the actual situation was far more complicated. In fact, identifying the specific temporal spans of both the 'Medieval Warm Period' and the 'Little Ice Age' proves to be highly problematic (Grove, 2001, 2003; Ogilvie and Jónsson, 2001). The end of the cooling phase associated with the 'Little Ice Age' can more accurately be climatologically situated quite significantly after the medieval period, towards the latter half of the 19th century (Ogilvie and Jónsson, 2001),

but its beginning is more commonly attributed to the mid-16th century, if established at all (Grove, 2001; Ogilvie and Jónsson, 2001). This is thus quite at odds from the attribution of the 'Little Ice Age' to the medieval period. The confusion caused by this, and the loose application of the broad concept to the historical record can be seen in the literature with fluctuating dates applied to both the beginning and the end of the 'Little Ice Age' (and the 'Medieval Warm Period'). The historical record documenting cold years in the earlier centuries (13th and 14th centuries) has been used to suggest the earlier onset, as per Lamb's work (1965).

The definition of these proposed historical phases is complicated by a number of factors. Perhaps the most critical of these are the difficulties caused by regional variation and the interplay between two phenomena– those of climate and glaciation (Matthews and Briffa, 2005; Nesje et al., 2008). The term 'Little Ice Age' was first applied by Matthes (1939, p 520) with regards to Holocene glacial advance in Nevada, California. The confusion in defining the period stems from a conflation of the two factors, with the pattern of glaciation being defined for the Northern Alps region as beginning in 1300 A.D. and ending in 1950 A.D., while the climatic cooling trend is dated to between 1570 A.D. and 1900 A.D (Matthews and Briffa, 2005). This is in itself consistent with the historical record which recounts changes in sea ice margins, for example in Iceland (Grove, 2003). Difficulties with defining the climatic trends are tied to greater fluctuations which tend to be seen in climate both on regional and temporal levels and which complicate our ability to chart them in the past with any definition higher than twenty year intervals (Matthews and Briffa, 2005).

Some sources of evidence, however, can be utilized to gain more insight. Matthews and Briffa (2005, p 30), summarize the evidence drawn from annual

fluctuations in tree rings, noting the appearance of "persistently cool summers over extratropical northern lands throughout much of the thirteenth century … and seen right across northern Europe and Western Siberia."[5] Axford et al. (2009) also cite evidence drawn from Iceland that while the first millennium A.D. was relatively warm overall (see also Briffa, 2000), a gradual cooling trend commenced in the twelfth century (Briffa et al., 2004). While the climatic cooling trend was relatively gradual until at least the mid-16th century, Bradley et al. (2003, p 405) note that temperatures in the Northern Hemisphere "from 1000 to 1200 A.D. (or 1100 to 1200 A.D.) were almost the same (or 0.03˚C cooler) as from 1901 to 1970 A.D … but [that] the latter period was on average ~0.35˚C cooler than the last 30 years of the 20th century." Büntgen et al., (2005) also note warmer temperatures during the first three centuries of the last millennium, followed by a cooling trend as indicated by dendrochronological data from Alpine regions in Switzerland and Austria.

Grove (2003:372-373) refers to the cooling impact of volcanic eruptions, and cites eruptions at approximately 1225 A.D. and in 1258 A.D (the later one registering as being more severe). Dybdahl (2012, p 1164) also comments on the severity of the 1258 A.D. eruption, which is indicated by both tree ring data and ice core data from the Arctic and Antarctica. It is further noted that the period from 1100 to 1250 A.D was particularly quiet while the years between 1250 and 1500 AD were extremely volatile in terms of volcanic activity which might therefore have contributed to cooler temperatures. In particular, five volcanic eruptions have now been confirmed over the course of the 13th century alone in multiple studies, all of which emitted significant aerosols into the

[5] Conclusions drawn from Briffa (2000), Esper et al. (2002), Grudd et al. (2002), Helama et al. (2002), Naurzbaev et al. (2002), Snowball et al. (2004), Luckman and Wilson (2005)

atmosphere (Langway et al., 1995; Cole-Dai et al., 2000; Budner and Cole-Dai, 2003). Three of these (1234 A.D., 1259 A.D., and 1285 A.D.) are evident in the ice core records for both the Northern and the Southern record (Langway et al., 1995; Budner and Cole-Dai, 2003). Until recently, all of these events are from unidentified volcanic eruptions (Budner and Cole-Dai, 2003). However, the eruption which has been documented as the 1258 A.D. /1259 A.D.[6] event may have been linked to the A.D. 1257 eruption of the Samalas volcano in the Rinjani Volcanic Complex of Indonesia, which resulted in "the largest volcanic sulfur release to the stratosphere of the past 7,000 y)" (Lavigne et al., 2013, p 16742). Placed in context, this eruption has been identified as one of the most significant volcanic events to occur over the course of the last 10,000 years, surmounting Krakatau (1883 A.D.) by 8 times and being twice as severe as Tabora (1815 A.D) in its resulting stratospheric sulfate load (Lavigne et al., 2013).

While climatic impact of even the most severe volcanic eruption has traditionally been thought to only last a maximum of five years (Budner and Cole-Dai, 2003), Budner and Cole-Dai (2003) note that this has recently come into question, and that the impact may be of longer duration. They observe that

> …even when the contribution of Event 21 (ca. 1276), a possible high latitude eruption that may not have impacted the climate system, is excluded, the cumulative volcanic flux … for the 13th century is still 6 times the average century volcanic flux … for the rest of the 904 – 1865 A.D. period. This century of frequent large volcanic eruptions coincides with the period of transition from the 'Medieval Warm Period' to the 'Little Ice Age' (Budner and Cole-Dai, 2003, p 174).

Thus, while major climatic cooling is not clear in climatological and dendrochronological records until the mid-16th century, the evidence does point to some level of cooling

[6] These two dates represent the same eruption in the literature, with the discrepancy in date being connected to the extent of the events' atmospheric impact.

occurring from at least the 13th century. This may further have been punctuated by short-term cooler years, especially over the course of the 13th century. The frequency of these may also have factored into a generally more difficult century with potential hold-over into the following century (Budner and Cole-Dai, 2003). It is further suggested that such cumulative events had the potential to impact the climate extensively enough to contribute significantly to the onset of a 'Little Ice Age' (Robock, 2000; Budner and Cole-Dai, 2003).

Regardless of whether or not a 'Little Ice Age' can be defined as a distinct event, it is necessary to understand what was happening to the populations living over this time. This must further be considered in the context of earlier periods which influenced the agricultural and settlement patterns seen in the later medieval period. Any cooling trends and harsh years from the mid-12th century onwards must be viewed in relation to the slightly warmer temperatures which populations had been accustomed to prior to 1000 A.D., and indeed up until the 12th century (Axford et al., 2009). If slight decreases in temperature (which can nevertheless impact vegetation) are further set against intermittent bad years causing crop failure, the hardships attested to by the historical documentation might be better understood. The weather does not have to have deteriorated suddenly for an impact to be felt. All that is necessary is that enough of a shift from previous warmer temperatures occurs, and if this is further punctuated by particularly bad years the effects on the population within any given lifetime can be severe.

In Denmark, as in other places around the Northern Hemisphere, including the United Kingdom, Norway, Iceland, and Greenland, both historical and archaeological

evidence has been cited for the abandonment of farms and villages in more marginal areas (Dyer et al., 1972; Antonson, 2009; Dybdahl, 2012; Hybel and Poulsen, 2013). Dybdahl (2012) reviews the evidence from Norway to show that this abandonment occurred prior to the onslaught of the Black Death and to tie it to deteriorating trends in crop production. Grove (2003) reviews the evidence from Iceland, and discusses in particular documentary evidence for poor crop years and failure in fisheries which led to extensive famine and the abandonment of settlements, particularly in more northern marginal areas. She notes more recent documented cases in Iceland in which poor agricultural and fishing years resulted in extreme famine despite more modern agricultural mechanisms. If such an impact was felt in these more recent situations with more advanced technology, it must have been even more severe for the medieval populations (Grove, 2003).

Difficulties are also reported on Greenland, and the populations' failure to adopt alternative modes of subsistence in the face of failing agriculture is noted (Grove, 2001, 2003). This is also reflected in Denmark where increased emphasis on cattle-based agriculture in association with the mid-14th century 'crises' occurred (Boldsen, 1997a, 2005a; Bøgh, 1999; Hybel and Poulsen, 2013). However, the extent to which these crises actually led to desertion prior to the mid-14th century in Denmark has been called into question based on a review of the historical literature (Hybel and Poulsen, 2013). It is likely that in this case the level of desertion was more moderate prior to the Black Death, but that population level of desertion peaked around 1400 AD and marked a combination of depopulation and re-organization (Hybel and Poulsen, 2013).

There are periods on record which may have contributed more to such desertions. While a number of difficult years are recorded in the historical record and substantiated by climatological and dendroclimatological data (Dybdahl, 2012), perhaps one of the

most significant periods was that occurring between 1315 A.D. and 1317 A.D. during which Campbell (2009) noted crop failure in England. The dendrochronological data from Norway also pointed to 1316 A.D. as being a bad year (Dybdahl, 2012) and it is this period which has historically been designated 'the Great Famine' (1315-1322) (Jordan, 1997). Dybdahl (2012) also notes bad periods in Norway between 1328 A.D. and 1330 A.D., between 1348 A.D. and 1351 A.D. and between 1369 A.D. and 1370 A.D. These latter two are also recorded for England (Dybdahl 2012; Campbell 2009), and of course the years surrounding 1350 A.D. were complicated further by Black Death.

Beyond the gradually cooling temperature punctuated by particularly bad years, various epidemiological circumstances factored into conditions faced by medieval populations. One disease which played a critical role in the health of the medieval Danish population was leprosy, which had been endemic to the Danish population prior to the mid-14th century (Boldsen, 2001, 2005b, 2008; Boldsen and Mollerup, 2006). Indeed, the prevalence of this disease during the earlier period has been the topic of a great deal of research (Boldsen, 1997a, 2001; Boldsen and Mollerup, 2006) and much of what we now know regarding the osteological diagnosis of this disease stems from early work on populations from the medieval period in Denmark (Møller-Christensen, 1951, 1952, 1953, 1958, 1978; Møller-Christensen and Faber, 1952). However, probably in large part in response to the intensive measures taken to isolate and eradicate the disease, it was in decline by the mid-14th century (Richards, 1960, 2000; Manchester, 1984, 1991), being virtually eradicated in Scandinavia by the middle of the 16th century (Boldsen, 2001).

At the same time, diseases such as tuberculosis were on the rise (Manchester, 1991; Boldsen and Mollerup, 2006), and the onslaught of the Black Death in the late 1349

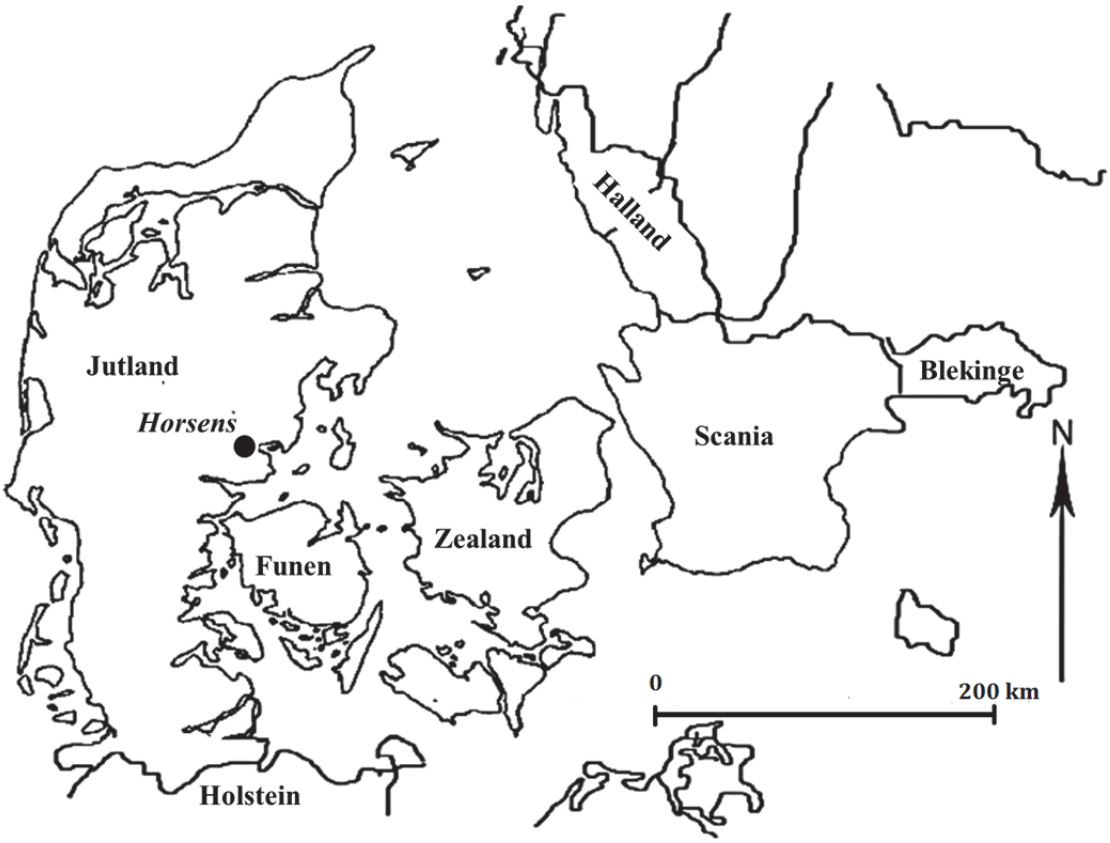
A.D. (Benedictow, 2004) had a significant demographic impact. It is estimated that by the end of 1350 A.D., as much as half of the population may have been wiped out by the Black Death (Bøgh, 1999; Benedictow, 2004). While hugely devastating, this did relieve the population pressure which had built up over the course of the earlier period (Bøgh, 1999; Hybel and Poulsen, 2013). The literature thus suggests that the population experienced extreme stress as a result of the combined 'crises' of famine and plague, but that there was a subsequent improvement in conditions in response to factors such as decreased population density (Bøgh, 1999; Yoder, 2006; Hybel and Poulsen, 2013). The bioarchaeological record, however, shows mixed results in terms of population health with research done by Yoder (2006, 2010) finding little support for these changing patterns of health over time. Yoder (2006, 2010) also found little evidence for change in diet over time, and these results are consistent with Turner's (2013) findings of a lack in temporal change in both stable isotope and dental microwear patterns (Turner, 2013). On the other hand, Boldsen (2005a) identifies increased dental attrition after 1300 A.D., which is interpreted as indicating a change in diet towards more heavy reliance on grain and a deterioration in living conditions over time. It is clear that further investigation is required to elucidate any patterns in health over the course of this period.

Urban and Rural Settings in Medieval Denmark

The consideration of such health patterns can also benefit from a regional perspective (Yoder, 2006). Different parts of Denmark appear to have been variably impacted by socioeconomic and environmental circumstances (Bøgh, 1999; Hybel and Poulsen, 2013), and since it might be expected that more densely populated areas (towns) will have different epidemiological profiles than less densely populated rural areas (Lewis et al., 1995; Steckel, 1995; Roberts and Manchester, 2005), a review of the evidence for

socio-environmental patterns in these settings will be beneficial. The layout of medieval Denmark with the town of Horsens, which is central to this thesis, can be seen in Figure 2-6.

Figure 2-6 Overview map of Medieval Denmark with the location of the town of Horsens marked. Based on Hybel and Poulsen (2013: Map 1, pp. xxiii) and Boldsen (1990b: Figure 11.1).



Hybel and Poulsen (2007) describe the rural component of the socioeconomic system as dominating the Danish medieval period. Most commonly, households were clustered into small settlements or villages rather than being isolated farms (although these did exist) (Hybel and Poulsen, 2013). Land was jurisdictionally divided into districts (earlier known as the *herred* and developing into administrative districts known as *len* (fiefs), ecclesiastically into parishes, and was further divided by military criteria (Hybel and Poulsen, 2013). There was also a tenurial system in place in which the village

acted as the basic unit (Hybel and Poulsen, 2013). The village was essentially "a community of free landowners and tenant farmers, along with their servants and farmhand slaves" (Hybel and Poulsen, 2013, p 144). A combination of common and privately owned land was in place and a system of common rule seems to have been fundamental in the government and management of much of village life (Hybel and Poulsen, 2013). There appear to have been boundaries around the common fields and separating the village from the fields, which were used both as pasture and for arable farming (Kristensen, 1999; Hybel and Poulsen, 2013).

The manorial system did not really begin to develop in Denmark until the 12th century (Bøgh, 1999; Hybel and Poulsen, 2013), and while it gradually became more dominant over the course of the medieval period it did not predominate until as late as the 16th and 17th centuries (Hybel and Poulsen, 2013). In fact, Bøgh (1999) writes of crown efforts to maintain communal access to 'forest' lands, suggesting that the importance of protecting peasant access against infringing estates was a central concern at least during the earlier period. In general, it appears that the dominant factor in rural life continued to be the agrarian activities with the village at the centre and ready access to communal land (Kristensen, 1999; Hybel and Poulsen, 2013). Grain production (rye, barley, and wheat) was central and the year was heavily scheduled around agrarian activities (Hybel and Poulsen, 2013). This remained a staple throughout the medieval period, and the diet was supplemented by gardening and by both gathering of wild resources and hunting and fishing (Hybel and Poulsen, 2013). There was a high level of regional diversity in agricultural developments, with the economy in some regions remaining largely grain based while in others such as Western Jutland it seems that cattle breeding gained

importance through medieval period. The towns and marketplaces became increasingly central and trade in cattle and oxen was tied in closely with the Hanseatic world[7] (Bøgh, 1999; Hybel and Poulsen, 2013).

While the Danish population remained a largely rural one until as recently as 1870 A.D. (with the rural population constituting at least 75% of the population) (Christensen and Mikkelsen, 2006), the growth of a market economy did increasingly bring people into towns from about the 11th century onward (Kristensen, 1999; Christensen and Mikkelsen, 2006; Hybel and Poulsen, 2013). Hybel and Poulsen (2013, p 227) define medieval Danish towns as "densely populated areas which functioned as centres of a hinterland and were also demarcated fiscally" (after Andrén, 1985). These were primarily centres of trade and administration, with the earliest being established in the 8th and 9th centuries, but becoming more prominent after 1000 A.D (Christensen and Mikkelsen, 2006; Hybel and Poulsen, 2013). Politically well positioned between the Baltic and European regions, Denmark became a focal point in trade both externally and regionally (between rural and urban markets) during the medieval period (Christensen and Mikkelsen, 2006; Hybel and Poulsen, 2013). The medieval town of Horsens, in which Ole Wormsgade is located, is identified as experiencing more intense settlement in the 11th century (Schørring 2000 as cited by Hybel and Poulsen 2007, 233; Kristensen, 1999). The period from the 11th through to the mid-14th century marked a period of increased urbanisation in Denmark, with more of the population beginning to live in towns, and with increased trade and craft specialization (Kristensen, 1999; Christensen and Mikkelsen, 2006; Hybel and Poulsen,

[7] The Hanseatic league was the German-controlled commercial and mercantile organization that spanned Northern Europe, connected via coastal networks of trade and defense and extension into interior cities via riverways. From the mid-13th through to the 16th centuries, this network extended from the Baltic through the North Sea (Liggio, 2007).

2013). By at least the late 13th century, there is evidence for regular weekly markets in towns which would have made movement between rural and urban settings a regular occurrence (Kristensen, 1999; Hybel and Poulsen, 2013). These markets played an important role in town planning, as has been shown at Horsens where a permanent centrally located market was established, with associated restructuring such as the development of parallel street organization (Kristensen, 1999; Hybel and Poulsen, 2013).

While medieval Danish towns were not as large as modern-day towns (Steckel (2004) writes of the generally small size of towns, particularly in Northern Europe), Kristensen (1999) paints a picture of crowding and dysentery in medieval Danish towns, in which historical records reveal measures put in place to deal with human waste. These included disposal outside of town and regulations regarding the location of toilets. Other points of interest which were of historic concern were the acquisition of clean water and the use of non-flammable building materials (Kristensen, 1999). Combined, we thus see a picture of towns characterized by relative rapid growth, high mobility both between town and countryside and on a more international scale, and a strong market economy with the growth of skilled trades and guilds in the period prior to Black Death (Andrén, 1989; Kristensen, 1999; Christensen and Mikkelsen, 2006; Hybel and Poulsen, 2013). This might be expected to create an atmosphere characterized by high levels of infectious disease (intestinal, viral, and bacterial) and by generally poorer health than in a rural setting (Steckel, 2004; Koepke and Baten, 2005).

Anthropological research does tend to reflect these patterns with health indicators consistent with generally poorer health in urban versus rural samples (Lewis et al., 1995; Lewis, 2002), although there are exceptions to this pattern (Kaupová et al., 2013). At the

same time, the picture in Denmark is complex with its more gradual move to urbanisation. The populations investigated in this thesis are located in relatively close proximity to each other, and it is possible that contact between the two communities may bring health profiles closer together. However, the distance was still great enough to potentially create a certain barrier at a time when travel would have been largely by foot. As seen in the next section, Yoder (2006) found little consistent difference in health indicators between rural and urban populations in medieval Denmark. At the same time, Boldsen (1990b, 1995) emphasizes the probable restriction of mobility during this period, relating shorter statures in the rural samples to population inbreeding. A great deal of research has already been done on health during the Danish medieval period, and it is to be hoped that this research will further add to our understanding of patterns of health during this period.

Health and previous research in Denmark

Bioachaeological research of the sort developed in this project is by necessity holistic in nature, pulling from a wide range of evidence. In any scientific inquiry, it is further necessary to build upon past research findings. As such, a brief background of past research as it informs the current investigation is necessary. This discussion incorporates both palaeopathological and palaeodemographic research as it comes together to inform our understanding of past population health in Denmark, as per Boldsen's (1997, p 230) description of palaeoepidemiology.

A great deal of palaeopathological work in Denmark has focused on the study of leprosy (Møller-Christensen, 1951, 1952, 1953, 1958, 1965, 1978; Møller-Christensen and Faber, 1952; Boldsen, 2001, 2005b, 2008, 2009; Segel, 2001; Boldsen and Freund,

2006; Boldsen and Mollerup, 2006). This research has contributed greatly to the identification of osteological changes in association with the disease (Boldsen and Fruend, 2006; Møller-Christensen, 1965, 1978, 1951, 1952, 1953, 1958; Møller-Christensen and Faber, 1952), to the statistical association of lesions to the disease itself (Boldsen, 2001, 2005c; Boldsen and Freund, 2006), and to the consideration of patterns in disease prevalence (Boldsen, 1997a, 2001, 2005b, 2008, 2009; Boldsen and Mollerup, 2006).

Dental enamel defects have been examined in Denmark both in relation to leprosy (Bennike et al., 2005; Boldsen, 2005b) and with respect to other aspects of health (Boldsen, 1997b, 1998; Thomas, 2003; Reid and Ferrell, 2006). Bennike et al. (2005) find that dental enamel hypoplasia (DEH) is less frequent in the nonleprosarium sample of Æbelholt than in the leprosarium sample from Næstved. Boldsen (1997 b, 1998) identified an association between DEH and body shape, but not size, supporting the position that stress resulting in the enamel defects had a greater impact on body proportions, which is not reflected in the final individual heights. A positive correlation is also observed between DEH and mortality rates in the Tirup sample (Boldsen, 2007).

All of these studies scored DEH based on macroscopic techniques. Similarly to the current study, however, Thomas (2003) thin sectioned a sample of 85 teeth from the rural village of Tirup in order to investigate the association between enamel defects and age at death. Using survival analysis, she found "that the severity of AS [accentuated striae] and the developmental timing of AS both contribute to a *variable* relationship between the presence of AS and risk of death" (Thomas, 2003, p 5). Reid and Dean (2006) have conducted microscopic analysis on teeth from 49 individuals from Tirup to

develop regression formulae which can be used for the estimation of periodicity in enamel formation and in the calculation of imbricational crown formation time. As will be seen, the standard which they derived in this way was utilized to estimate the age at occurrence for enamel defects in the current study.

Arcini (1999) conducted a bioarchaeological analysis of the medieval skeletal remains from the city of Lund. The sample was comprised of 3,305 individuals buried between 990 A.D. and 1536 A.D., and associated with three adjoining church cemeteries. The burials are divided into three periods, the first lasting from 990 A.D. to 1100 A.D., the second from 1100 A.D. to 1300 A.D., and the third from 1300 A.D. to 1537 A.D. (Cinthio, 1999). Arcini's (1999) conclusions are rather mixed. There is an apparent increase over time in subadult mortality, adult trauma, dental caries (in adults), and osteomyelitis (in males). This would be consistent with Qvist and Grøntved's (2001) findings in their study of chronic otitis media sequelae. At the same time, cribra orbitalia and periostitis decrease over time in both adults and subadults, and rates of dental enamel hypoplasia and stature measurements remain relatively constant (Arcini, 1999). It is thus concluded that health did not change considerably in Medieval Lund over the time period considered. This conclusion may be further substantiated by other bioarchaeological research, such as that conducted by Yoder (2006, 2010).

Yoder (2006, 2010) considered both rural and urban sites in Denmark. The urban sites were represented by the cemetery of St. Mikkel (which lies just outside of Viborg and dates to between the 12th and 16th centuries) and Ribe (dating to between 1250 A.D. and the early 15th century). The rural sample comes from Øm Kloster, a monastic site, and dates to between 1172 A.D. and the mid-16th century. This sample would have

included both monastic and lay individuals. Yoder considered the extent to which health changed over time, across sites, and according to both sex and social status. She did this through an examination of stable isotopes (both carbon and nitrogen), dental caries, cribra orbitalia and porotic hyperostosis, periosteal reactions, and femur length (as a proxy for stature). Her goal was to test whether the Medieval Agrarian Crisis which accompanied the Little Ice Age[8], and the Black Death epidemic, significantly impacted population health.

Yoder's (2006) bioarchaeological analysis does not find a significant or consistent change in health over time, possibly since the climate was highly variable and did not follow a straightforward cooling trend (discussed earlier). While there was minor patterning within different social classes and on both regional and temporal scales, these did not consistently point to wide scale deterioration in health followed by improvement after the demographic and epidemiological crises. Yoder's (2006) investigation did, however, find regional differences in peasant health and diet, as well as health differences between males and females. Yoder (2006, 2010) found differences in stable isotope values between urban and rural sites, and suggested that these findings either reflected differential access to or differential preference for food resources. This research suggested that there may have been a higher component of meat protein in the urban diet compared to a higher terrestrial plant based diet at the rural site.

[8] Yoder (2006) recognizes the more traditional historical definition of the 'Little Ice Age' as beginning in the late 13th century A.D., noting that this marks the beginning of a period of roughly five centuries of unstable weather. She acknowledged fluctuations in temperature and precipitation and pays particular definition to strains induced by poor growing seasons such as those associated with the 'Great Famine' of 1315 A.D. to 1322 A.D. (Jordan, 1997; Fagan, 2000; Yoder, 2006).

Yoder's (2006, 2010) results also showed higher frequencies of periosteal reaction at the rural site, and suggested that this might relate to higher rates of infection as a result of a nutritionally poor diet. In addition, however, she notes that the presence of a hospital at this site may have inflated the rates of infection visible in the skeletal remains. Higher rates of porotic hyperostosis at the urban poor parish site of S. Mikkel are interpreted as reflecting higher rates of parasitism in an urban environment, since the isotope signature points to a diet high in terrestrial and animal proteins which was unlikely to be iron deficient (Yoder, 2006, 2010).

The extensive study of leprosy in Denmark makes it a useful disease to consider in relation to different patterns of infection. However, the patterns of leprosy prevalence in urban versus rural settings is influenced not only by general health differences between the two populations but also by different patterns in segregation of people who suffered from the disease (Boldsen, 2009). In a multi-site study, Boldsen (2009) found higher prevalence rates of leprosy in rural as opposed to urban settings (particularly in rural populations which were more distant from urban centres), and later occurrence of the disease in rural settings. This is likely related to the establishment of leprosaria in medieval towns which would have led to the organised isolation of individuals with the disease from the general population and will have contributed largely to its eventual eradication (Boldsen 2009). The interaction between humans and animals in rural settings is in this case implicated for the demise of the disease in rural settings, as Boldsen (2009) attributes it to natural immunization conferred on the population from the related *M. bovis* (bovine tuberculosis, which is caused by a bacteria of the same genus as that which causes leprosy) (see also Manchester, 1984). Thus, while there was movement

of cattle through trade in both the urban and rural settings (particularly as this commodity became more important through the medieval period), it may be that in the absence of the segregating effects of the leprosarium in the rural setting the impact of bovine TB became a stronger selective factor in the demise of leprosy. In the context of this study, therefore, it may be expected that leprosy will be more common and of longer duration in the rural sample of Sejet.

Boldsen (1996) identified slightly higher mortality rates in the urban site of St. Mikkel, but suggested that these rates might be under-representative since much of the St. Mikkel sample was likely drawn from the peasant population, many of whom would have travelled in to the urban centre from rural settings. Boldsen (1984b, 1996) suggests that high rates of older subadult mortality imply that these urban centres were reliant on high levels of immigration to maintain population levels. Furthermore, it is suggested that the higher mortality in the urban population reflects higher epidemic disease loads in these urban centres. Boldsen (1984b) also notes the likelihood of urban centres to maintain endemic disease profiles while rural populations may have not provided the necessary population reservoir for such patterns. Boldsen's (1984b, 1996) results are also consistent with Yoder's (2006, 2010) findings of higher rates in periostitis at St. Mikkel. In this light, we might expect to see higher mortality rates and higher rates of infection from tuberculosis and treponematosis in the urban sample of Ole Wormsgade as part of the current study. The absence of any similar controlling mechanism to that of the leprosarium in the case of leprosy, combined with the larger population reservoir and higher population density, might be expected to lead to higher exposure and greater incidence of these diseases in comparison to the urban population at Sejet.

Given the observed changes over time in the historical literature, it would be expected that health will decline with the agrarian crisis, but might see an improvement with the relief in population pressure after the mid-14th century. We might expect to see more evidence for stress experiences in the early period, higher incidence of infection, and more growth stunting. However, the literature on climate change suggests that such a pattern might be overly simplistic, and certainly Yoder's (2006) results support the challenges in identifying broad-scale temporal changes. The actual situation may be one of variable conditions, with periods of very poor climate (associated with negative impacts on agricultural resources) occurring in the midst of gradual trends punctuated by better years. The particularly negative periods might not be captured within the resolution of the skeletal sample. Any broad scale negative temporal trend (in the form of a Little Ice Age) which may have made living more challenging may have also been counter-acted by reduced population pressure following the Black Death epidemic.

While the patterns of poor health followed by improved health might therefore be predicted based on broad patterns, both the climatological literature and the bioarchaeological investigations to date suggest that such patterns may be far more difficult to capture through bioarchaeological investigation, and that variable results may be anticipated. The historical literature furthermore points to growing urbanisation in Denmark over this period, and certainly the bioarchaeological literature indicates different health patterns being present in rural versus urban locations for a number of health parameters. Infectious disease rates might be particularly sensitive to this in relation to differential exposure, and it appears that the cultural response to diseases such as leprosy may have played a significant role in this regard. Such findings suggest that

differences will be found between the rural location of Sejet and the urban location of Ole Wormsgade.

Summary

The consideration of developmental enamel defects can pinpoint the occurrence of episodes of physiological perturbation (stress) sustained by an individual during the period covered by that enamel growth. It is possible that this, when considered in relation to adult health, will provide some insight into the question of individual frailty in that if there is an association between patterns of severity, frequency, or timing of the stress events and adult health parameters as identified from the adult skeletal remains, we may be seeing evidence of an impact of early life stress experiences on adult survivorship. Alternatively, however, the stress events sustained during childhood may be influenced in themselves by an individuals' ability to combat stress and so may be coloured by individual frailty at a young age. In this scenario, it is possible that any connection seen will reflect not an impact of stress on later life but rather a consistent pattern seen in an individual across the course of their lives. The question of where the threshold lies for an individual to be sufficiently 'stressed' as to experience a disruption in enamel development is, unfortunately, poorly understood. Thomas' (2003) results showing a variable relationship between accentuated striae of Retzius and mortality may speak to the importance of stress thresholds in the interpretation of stress-health relationships. The consideration of the relationship between developmental defects in enamel and adult health thus has the potential to inform our understanding of individual patterns of health and of possible connections between childhood health and adult health. This can not only provide insight into past health patterns, but has the potential to contribute another body of information to the study of such relationships amongst modern populations.

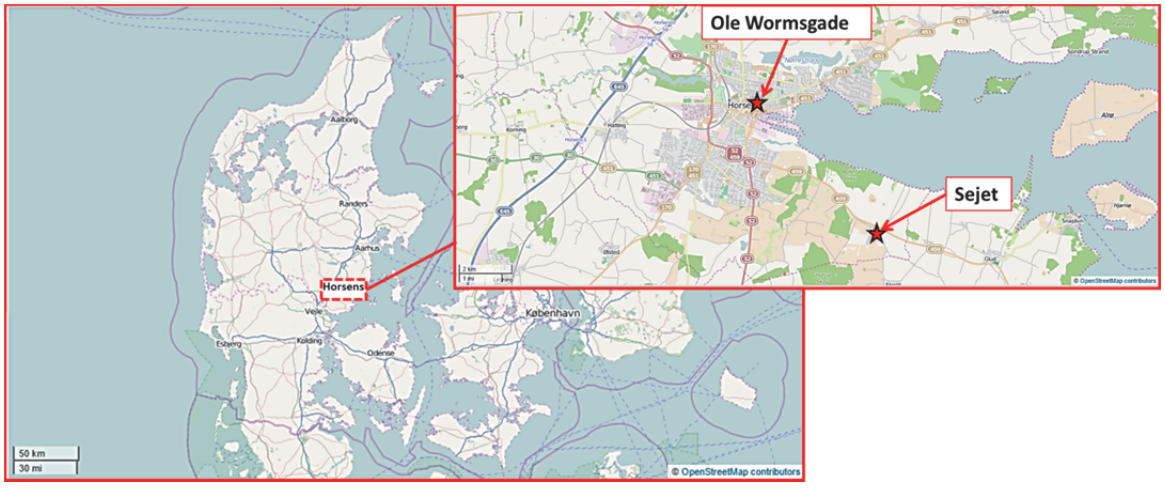
The contextual investigation of these patterns (within the temporal and regional framework) can further help to provide insight, as it can provide extensive background information on environmental factors which may have influenced such aspects as susceptibility and exposure to various stressors. The period in Denmark from the mid-12th to the mid-16th centuries marks of period of profound socioeconomic change. While care must be taken in the consideration of all aspects of health in past populations, the study of the skeletal remains of these peoples can provide tremendous insight into levels of stress, disease load, and mortality patterns. When considered from a bioarchaeological standpoint, integrating archaeological, historical, climatological, and economic sources (amongst others) it will be possible to build upon previous understandings and to develop a more complete understanding of the past. As will be seen in the next chapter, attempts to tease apart contextual influences (temporal, urban vs rural) in this research led to the consideration of subsamples from a rural and an urban cemetery which each span the period being considered. Such an approach is critical in developing a more nuanced understanding of the factors influencing environmental stressors experienced by the populations under study and in rebuilding the complex relationships between socioeconomic and environmental factors on the one hand and population health on the other.

Chapter 3 Materials and Methods

Samples

The skeletal material used in this project is currently curated at the Department of Anthropology, University of Southern Denmark (ADBOU). Both cemeteries are under the jurisdiction of Horsens Museum, and permission was acquired from the museum to work with the material. The two cemetery samples are those of Sejet and Ole Wormsgade (Figure 3-1). This section will provide a description of these sites, will highlight some of the specific strengths of these collections (along with site-specific difficulties encountered in dealing with these materials), and will outline the specific parameters of the acquired samples.

Figure 3-1 Map showing the location of Horsens, with the Ole Wormsgade and Sejet locations identified on the inset map. © OpenStreetMap contributors. Licensed under the Open Data Commons Open Database License (ODbL). Cartography licensed under the Creative Commons Attribution-ShareAlike 2.0 license (CC BY-SA). Copyright information can be found at http://www.openstreetmap.org/copyright



Individuals selected for this study had to be aged 18 years or older (based on epiphyseal fusion and third molar eruption), and have preserved anterior dentition which was not worn beyond the enamel portion which would have been developing at around 3 years of age (thus capturing the period from at least 3 years of age to approximately 6.2 years of age) (Reid and Dean, 2006). Teeth were compared against the charts developed from Northern European populations by Reid and Dean (2006) in order to estimate the age range represented by the preserved enamel. In the interests of maintaining a larger sample size, all anterior dentitions were reviewed for selection. Mandibular canines (which capture an age range of approximately 1.5 to 6.2 years) (Reid and Dean, 2006) were the tooth of preference, but maxillary canines (which span an age range from roughly 1.7-5.3 years of age) (Reid and Dean, 2006) were selected in the absence of mandibular canines or to complement worn mandibular canines. Maxillary first incisors (spanning an age range of approximately 1.1 – 5.0 years of age) (Reid and Dean, 2006) were also utilized on occasion if they were the only usable dentition (two individuals). In two circumstances, the maxillary second incisors (which capture a shorter but contemporary span from 1.8 to 5.1 years of age), were selected.

The above selection process, in addition to the other skeletal criteria outlined below, resulted in a sample of 160 individuals from whom only one tooth was selected and 7 individuals from whom two teeth were selected. As discussed previously, enamel defects can stem from systemic or localized causes. This research is focusing on systemic defects since the goal is to gain insight into the overall systemic condition of an individual over the course of their development, which is captured by the enamel. It is generally recommended that studies of this nature consider two teeth from each individual

in order to distinguish between systemic and localized defects (Hillson, 1992a, 1996, 2008). However, the literature also demonstrates a very low frequency of localized defects across populations (Goodman et al., 1980; Goodman and Rose, 1990). Given the precious nature of the material, the decision was therefore made not to sample across the complete dentition. High resolution macro images were taken, however, of all dentition so that visible defects could be observed.

Beyond the dental selection criteria, data which could provide insight into adult health had to be available for each individual. Such information included age at death, pathological changes, and stature dimensions such as long bone length and stature as measured in the grave (Boldsen, 1984a; Petersen, 2005). While not all individuals possessed a complete complement of the foregoing areas of adult health information, only those individuals possessing one or more sources of information, as well as satisfying the dental criteria, were selected for this study.

Sejet

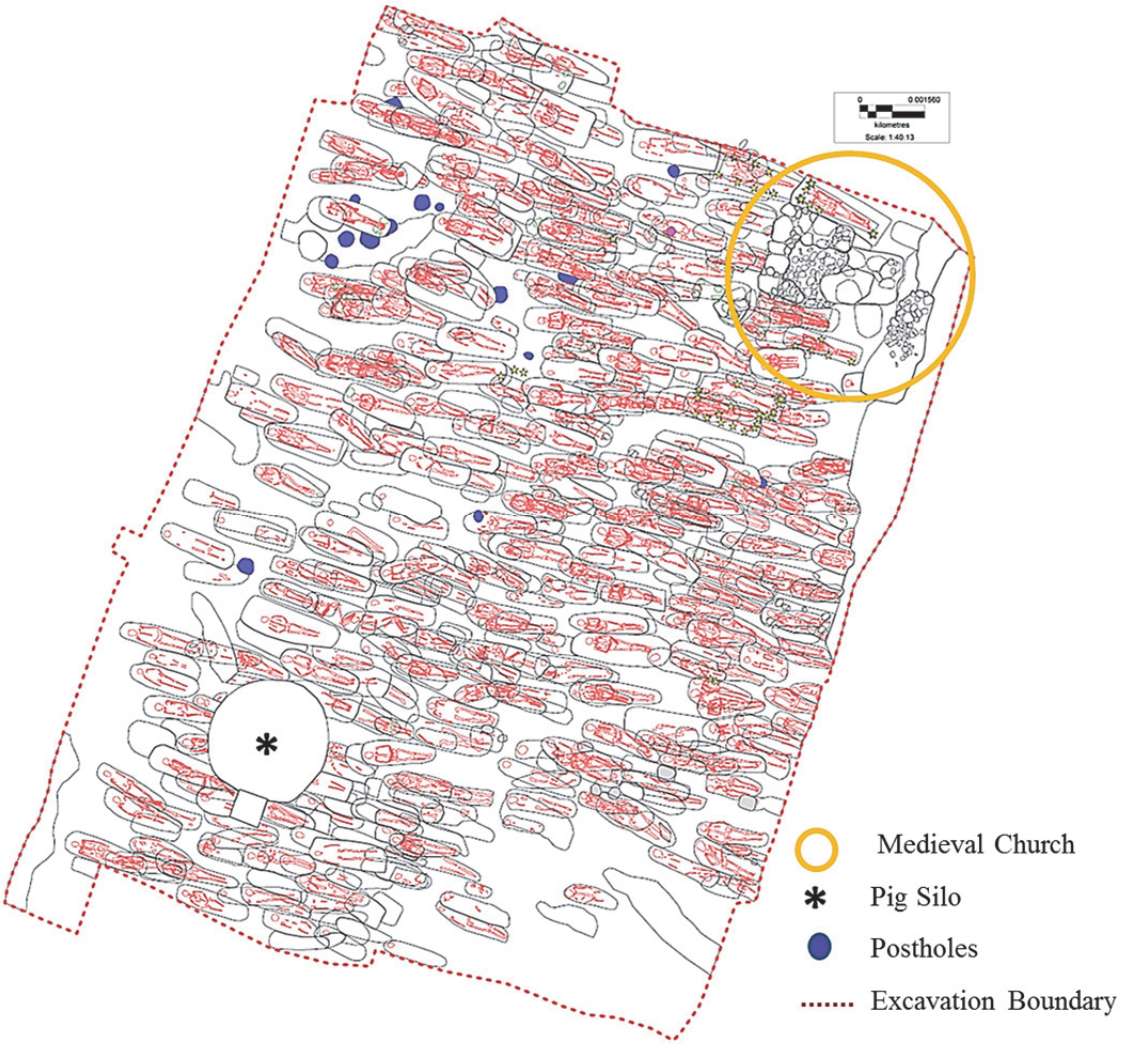
The cemetery of Sejet (HOM 1046) lies just outside of the town of Horsens and would have acted as a rural village cemetery. No historical record is preserved about the town or cemetery, but it is assumed that a cross section of the population would have been buried in the cemetery as this was the parish cemetery for the town. It was in use from A.D. 1150 to A.D. 1574, when the associated church was demolished. The cemetery was excavated in 2006 (Figure 3-2), and this led to the recovery of 435 *in situ* individuals, along with 194 individuals from disturbed contexts (i.e. loose finds) and 3 additional individuals whose context is no longer known (Pedersen and Boldsen, 2008). During the course of these excavations, four coins were found, all with preliminary dates

of between 1400 A.D. and 1500 A.D., offering further support for the cemeteries' use during this period (Kjærgård, 2006). None of the individuals with associated coins could be included in this study with two individuals being juvenile and two (one of whom had two associated coins) having insufficient preservation for this project. Excavation took place in advance of development on the site, and due to previous development the excavation was unable to recover the full limits of the cemetery.

From the recovered sample, 95 individuals were selected for this study. Prior to excavation, the land had been part of a pig farm, and preservation of the skeletal material was directly related to its proximity to the pig silo (being particularly poor closer to the silo due to acidic conditions in the soil) (Pedersen and Boldsen, 2008). The remains from this area tended to be highly fragmentary, with severe exfoliation of the periosteal surfaces. Enamel was also found to be extremely friable in many of the individuals from Sejet, and so 42 of the 137 individuals who were initially selected based on excavation records were eliminated from this study[9]. In the total cemetery sample, only 10% of the individuals at Sejet were well preserved, while 63% were registered as poorly preserved (Pedersen and Boldsen, 2008).

[9] This initial selection identified all adults who were recorded as having both cranial and postcranial material preserved.

Figure 3-2 Excavation map of Sejet. Excavation map of Sejet. The feature marked by the black asterisk is the later pig silo; features marked in blue are more recent postholes. The stone scatter marked by the yellow circle likely represents the remnants of the medieval church's west and south walls. The excavation boundary is marked by the dotted red line, but this does not represent the original extent of the cemetery. Individuals are represented by sketches in red, with grave cuts marked by the surrounding black lines (which are also used to represent other feature cuts). Map courtesy of Annemette Kjærgård at the Horsens Museum, © Horsens Museum.



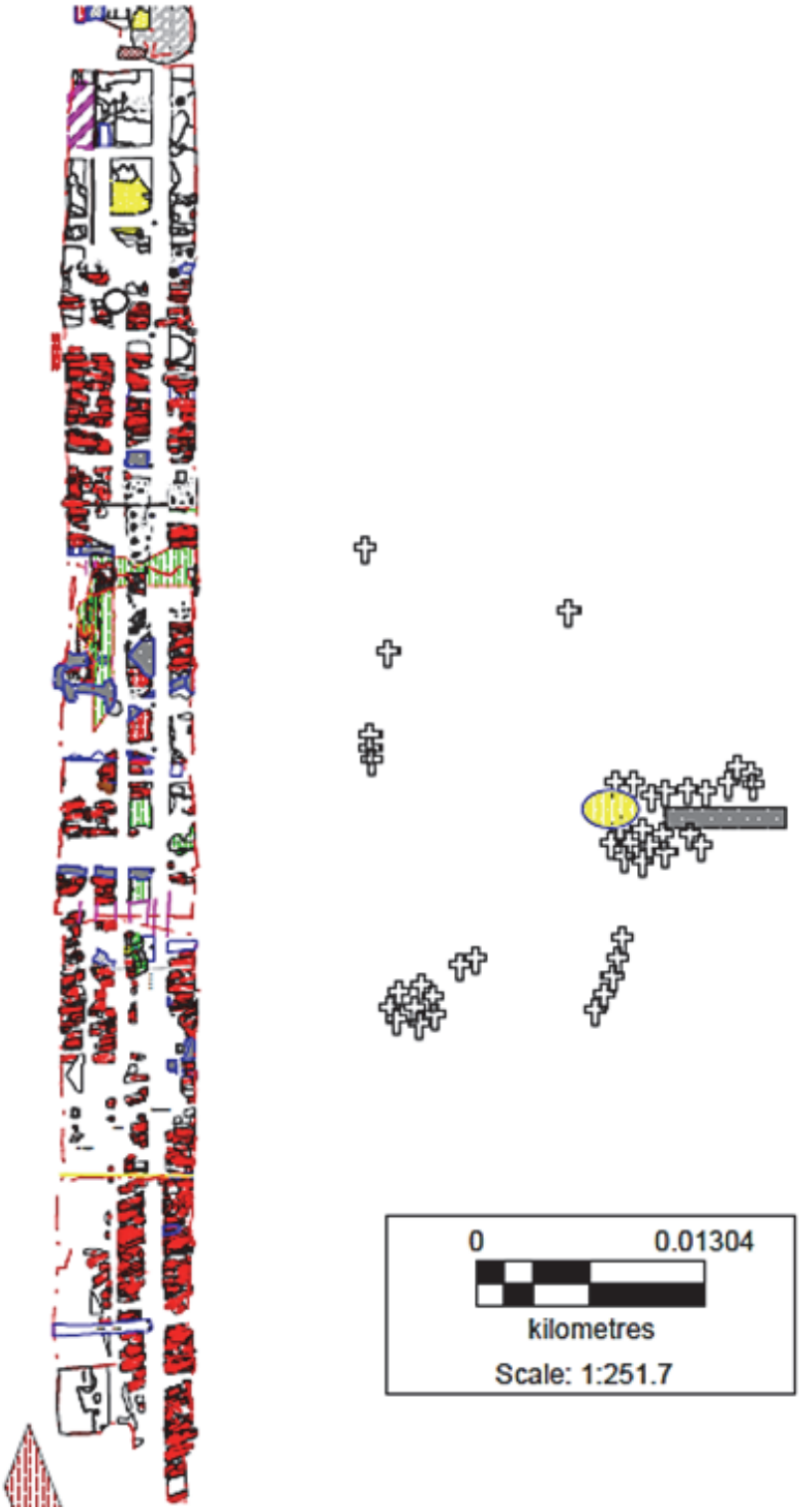
Ole Wormsgade

Ole Wormsgade (HOM 1649) was the urban parish cemetery of Horsens, associated with the Church of Our Lady (Vor Frue Kirke) (Birgitte et al., 2004) (Figure 3-3). Once again, records for this cemetery are sparse, but as a parish cemetery it also likely captured a cross section of the population, this time representing a more urban subset. Dating for Ole Wormsgade is extremely problematic, as there are very few records linked with the cemetery. It was most likely used from some time in the 12th century through to at least the Reformation of A.D. 1536 (Birgitte et al., 2004; Petersen pers. comm. 2011). The cemetery was excavated during the 2008 and 2009 field season in association with road works. The limits of this cemetery are, like Sejet, unknown, but may have covered an area of at least 9400 m^2 of which approximately 450 m^2 was excavated. Much of the material has been lost to previous development over the course of urban development.

The cemetery excavation led to the recovery of 401 *in situ* individuals, along with 510 loose grave finds (i.e. the minimum number of individuals recovered from grave fills but not part of the primary burial) and 76 loose finds from other contexts (i.e. the minimum number of individuals from non-grave contexts, such as surface finds which had been disturbed through previous construction processes). While most of the Ole Wormsgade sample was selected from *in situ* individuals, 10 individuals included in the present analysis were from loose finds. Non-*in situ* remains were typically not complete enough to satisfy the selection criteria. In most such cases, different skeletal elements could not be linked to a single individual with enough certainty to be used in the analysis. These 10 cases represent exceptions to this pattern by being complete enough to preserve both dental material and enough information for age estimation.

The Ole Wormsgade collection yielded a sample of 72 individuals for this study. The smaller size of the sample from this cemetery was largely due to truncation of many of the graves since the excavation took place along the course of a road. Frequently individuals were found with their upper bodies intact, but with their lower bodies missing, or vice versa. Similarly, there were many cases in which only the cranium or only the postcranium were present. Since both health-related skeletal information and dental material was required for this research, such truncation significantly reduced the usable sample. On a more positive note, however, the state of preservation of the Ole Wormsgade material was in general better than that of Sejet, and the condition of both the teeth and bone promoted the recovery of usable information.

Figure 3-3 Cemetery map of Ole Wormsgade. Burials are marked in solid red. As this cemetery was excavated in an urban setting along the course of roadwork, numerous incursions from later developments are visible surrounding these burials, with too many such features for individual description. The crosses and structural inclusions to the right of the main excavation area indicate burials from the same site but recovered in association with earlier excavation work and some recovered masonry from the original monastery walls (©Horsens Museum, used with permission).



Technical points on processing and analysis

Description of data collection. Sampling procedure

The sample was selected based on the availability of relatively unworn teeth (not worn past the first two deciles as determined from comparison with the growth charts developed by Reid and Dean (2006) (see also Figure 3-14) and availability of minimal sex and age information. Other health information, including stature (both as measured from the grave and collected as direct measurements from long bones), and details of pathology, was also available in many cases. It is unlikely that this methodology introduced any further bias than that which is innately part of such samples, but nevertheless possible sources of bias, including age, sex, preservation, and socio-economic biases, will be reviewed in the discussion (Chapter 5)

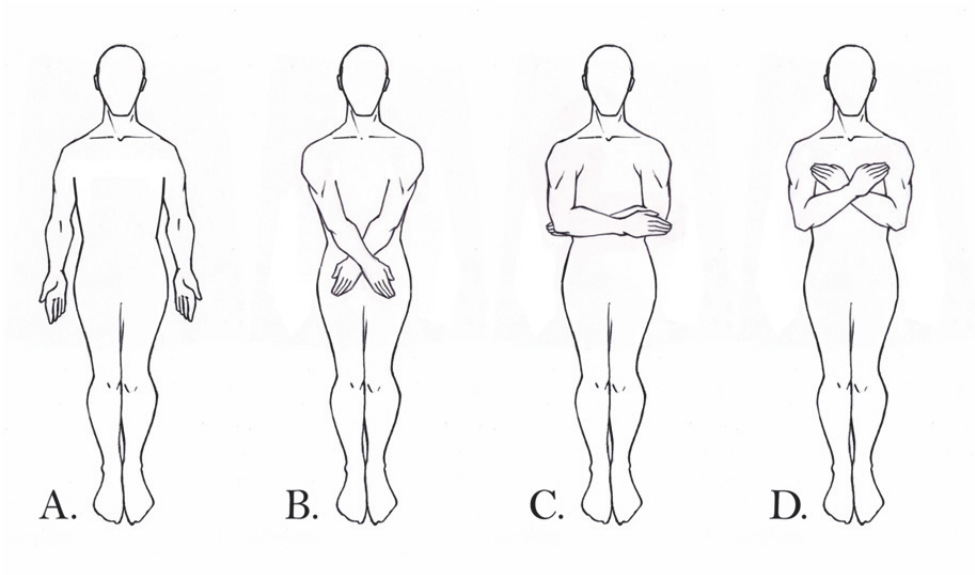
Chronological Distribution

A part of this research is concerned with the temporal changes in health in medieval Denmark, since over the course of the period being investigated Denmark underwent significant socioeconomic changes. In order to address this question, however, the temporal distribution of individuals within the cemeteries needs to be established. This will be done through the application of an arm position technique which has been heavily applied for the medieval period in Denmark. This is based on a relatively consistent chronological change in arm position which has been demonstrated in medieval Danish burial practices (Redin, 1976; Kieffer-Olsen, 1993; Jantzen et al., 1994). Early observations of apparent temporal patterning in arm positions at Æbelholt were made by Møller-Christensen (1958). Redin (1976) was later responsible for engaging in a careful consideration arm positions in relation to stratigraphy and grave groupings, defining four main arm positions (A, B, C, and D). Kieffer-Olsen (1993)

conducted an in depth study of eight cemeteries in which stratigraphy and grave groupings, burial style (for example coffin type), and absolute dating techniques such as dendrochronology and radiocarbon dating were integrated in an analysis of the temporal association with arm positions. This was further explored by Jantzen et al. (1994).

While the arm position categories have been found to be broadly consistent, and are commonly used to identify temporal distribution in Danish cemeteries (Redin, 1976; Boldsen et al., 2002; Thomas, 2003; Yoder, 2006, 2010; Weise et al., 2009; DeWitte and Hughes-Morey, 2012), their temporal boundaries have been found to be blurred in some cases and slightly variable across cemeteries (Redin, 1976; Kieffer-Olsen, 1993; Jantzen et al., 1994). The arm positions broadly reflect a progression from straight down along the sides of the body (A arm position) to crossed across the chest with the hands at the shoulders (D arm position) (Figure 3-4). In between, A arm positions are gradually replaced by B arm positions, with the arms crossed across the pelvis and this is followed by C arm positions, with the hands crossed straight across the abdomen (Redin, 1976; Kieffer-Olsen, 1993; Jantzen et al., 1994). The A arm position is most commonly used around 1050 A.D., although B arm position is found in a small percentage of cases (approximately 20%) (Jantzen et al., 1994). There is a transition to more heavy reliance on the B arm position by the late 13th century and until around 1400 A.D, when the C arm position becomes more popular (Jantzen et al., 1994) The D arm position becomes predominant after the late 15th century, and is used throughout the rest of the medieval period (Jantzen et al., 1994).

Figure 3-4 Arm positions used in chronological dating. The development of this system is based on research by Redin (1976), Kieffer-Olsen (1993), and Jantzen (1994). Line drawings © Janet LaFrance 2013 (used with permission). A arm position represents the period from around 1050 AD to the late 13th century AD. The B arm position is dominant from the late 13th century through to around 1400 AD. The C arm position is found most commonly from around 1400 A.D. while the D arm position seems to take over by the late 1400's AD (Jantzen 1994)



In some cases, a mixed arm position is found. This has been explained in relation to patterns of natural movement with bodily decomposition (Kieffer-Olsen, 1993). This has led to the designation where if A is combined with another arm position, the other arm position takes precedence. If C is combined with any other arm position, C takes precedence. If D is combined with either A or B, then D takes precedence (Kieffer-Olsen, 1993; Weise, 2009).

The subsample used in this research represents a relatively small proportion of the individuals who were datable from arm positions in the complete cemetery samples (Table 3-2). A total of 79 individuals were available from Sejet and only 47 individuals were available from the Ole Wormsgade sample for consideration of changing health and stress patterns over time. This can be compared with the number of individuals available with each arm position in the total cemetery sample in Table 3-1. Nevertheless, the

pattern between the complete sample and the subsample used in this study consistently demonstrates a higher proportion of individuals in the earlier periods, with very few in the later periods, and with the peak in Ole Wormsgade being slightly later than that in Sejet (Table 3-2, Figure 3-5, Figure 3-6).

Table 3-1: Arm position distribution within the broader cemetery samples showing the number of individuals with each arm position and their percentage in relation to the total cemetery samples (including individuals for whom arm position could not be established). MNI for Sejet is 632 individuals and for Ole Wormsgade the MNI is 987.[10] This includes both *in situ* and *ex situ* remains.

Cemetery		Frequency	Percent
Sejet	A	*138*	*21.8%*
	B	*77*	*12.2%*
	C	*30*	*4.7%*
	D	*4*	*.6%*
	Total	*249*	*39.4%*
Ole Wormsgade	A	*41*	*4.2%*
	B	*53*	*5.4%*
	C	*27*	*2.7%*
	D	*5*	*.5%*
	Total	*128*	*16.6%*

[10] It is worth noting here that the Ole Wormsgade material was particularly dissociated. Only 401 individuals were found *in situ* and these were frequently truncated. As noted above, this number also includes a MNI 510 individuals recovered as loose grave finds (fragmentary remains found in grave fill) and 76 individuals represented as loose finds. Ongoing work is attemting to re-associate some of these remains.

Table 3-2: Arm position distributions for the complete cemetery sample and the subsample used in this study. The percentage is in relation to the number of individuals from each sample (complete and subsample) who were sufficiently preserved for arm position to be determined. Known arm position representation reflects the extent to which known arm position individuals were sampled in this study (i.e., individuals with arm positions available from this study calculated in relation to the total number of individuals with that arm position available from the complete cemetery sample).

Cemetery		Complete Sample		Subsample		Known Arm Position Representation
		Frequency	Percent	Frequency	Percent	
Sejet	A	*138*	*55.4%*	*41*	*51.9%*	*29.7%*
	B	*77*	*30.9%*	*21*	*26.6%*	*27.3%*
	C	*30*	*12.0%*	*17*	*21.5%*	*56.7%*
	D	*4*	*1.6%*	*0*	*0%*	*0%*
	Total	*249*	*100%*	*79*	*100%*	*31.7%*
Ole Wormsgade	A	*41*	*32%*	*14*	*29.8%*	*34.1%*
	B	*53*	*41.4%*	*17*	*36.2%*	*32.1%*
	C	*27*	*21.1%*	*14*	*29.8%*	*51.9%*
	D	*5*	*3.9%*	*2*	*4.3%*	*40%*
	Total	*128*	*100%*	*47*	*100%*	*36.7%*

Figure 3-5 Number of individuals with each arm position within the total cemetery sample

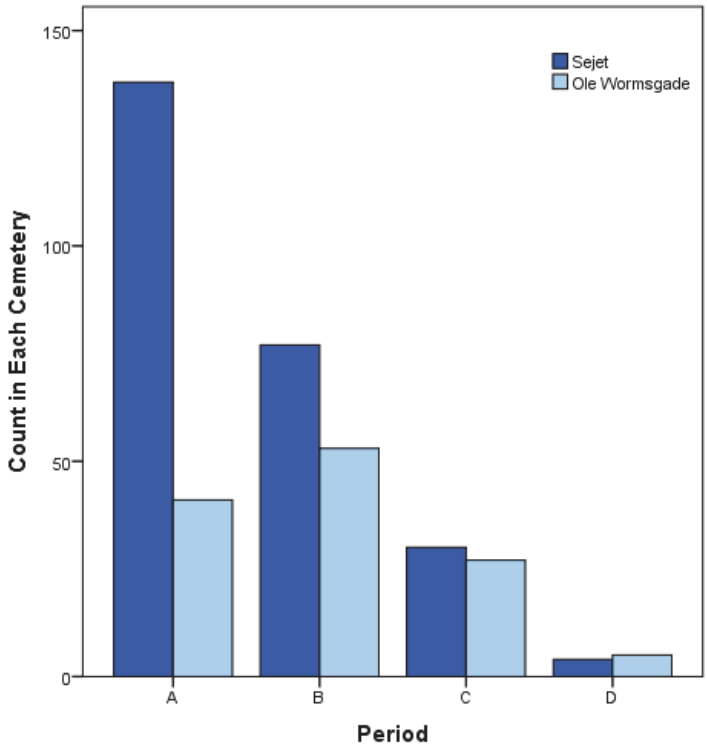
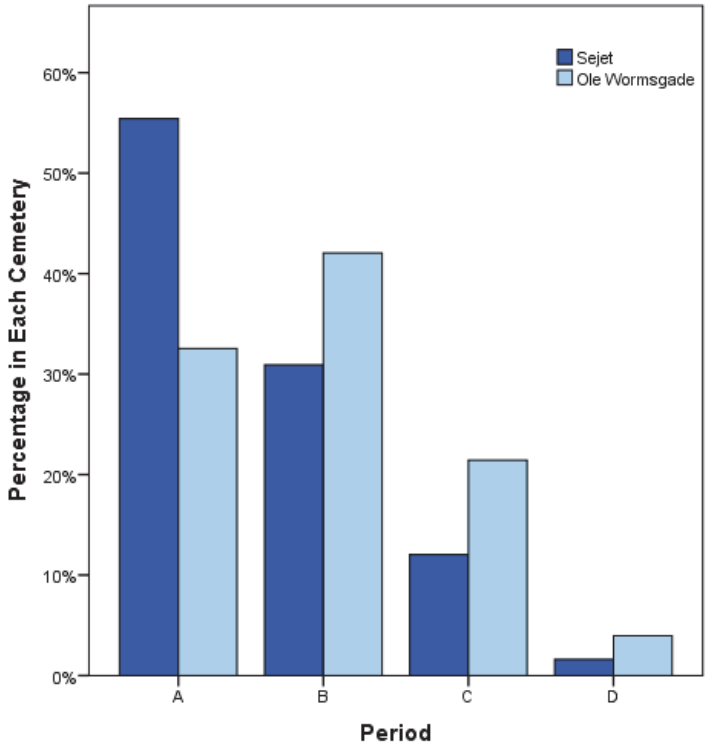


Figure 3-6 Percentage of individuals with each arm position within the complete cemetery with the number in each period expressed as a percentage of the total number of individuals from each cemetery with known arm positions



Sex estimation

Sex estimation was conducted using standard osteological procedures incorporating both cranial and postcranial features. Final counts for these can be seen in Figure 3-7 and Table 3-3. Both morphometric and visual techniques were applied, and all available features were scored. Metrics were taken of the pelvis and femur and computed using the equations provided by Albanese (2003, 2008) in an attempt to incorporate a metric component to sex estimation, but the material was so fragmentary that this information was of little assistance. Sex estimation was performed on all individuals and recorded in the database next to the sex on record at ADBOU. The previously recorded sex was not viewed prior to performing sex estimation for each individual, but this methodology acted as an interobserver check. Through this approach, different sex estimations between the current analysis and that previously conducted was found in eleven cases for Sejet (12%) and in seven cases for Ole Wormsgade (10%) (Appendix 1).

This difference between the two cemeteries was likely due to observer variability in the original sex estimation held on record at ADBOU. The Sejet material was originally scored by a single observer, Dorthe Pedersen. Multiple observers, on the other hand, were involved in the initial skeletal analysis of the Ole Wormsgade material, with segments of the sample being sexed by different observers. This could have introduced variability to the original estimates. In order to maintain consistent scoring to the greatest extent possible, all final sex scores were derived from my assessment. While inter-observer testing, in which all individuals were scored by a few observers, would have made it possible to assess the extent to which variability in scoring might have impacted the initial scores, the mix of scores by different observers without a clear record of which individuals were scored by whom made any assessment of this within the course of this

thesis impossible. The original Sejet scores, which were scored by a single observer, were acknowledged by that observer to possibly be affected by a learning curve (Pedersen 2010, pers. comm.). Through uniform scoring practices applied to the entire sample used in this study, it is suggested that a higher level of consistency has been achieved.

Figure 3-7 Sample distribution by sex

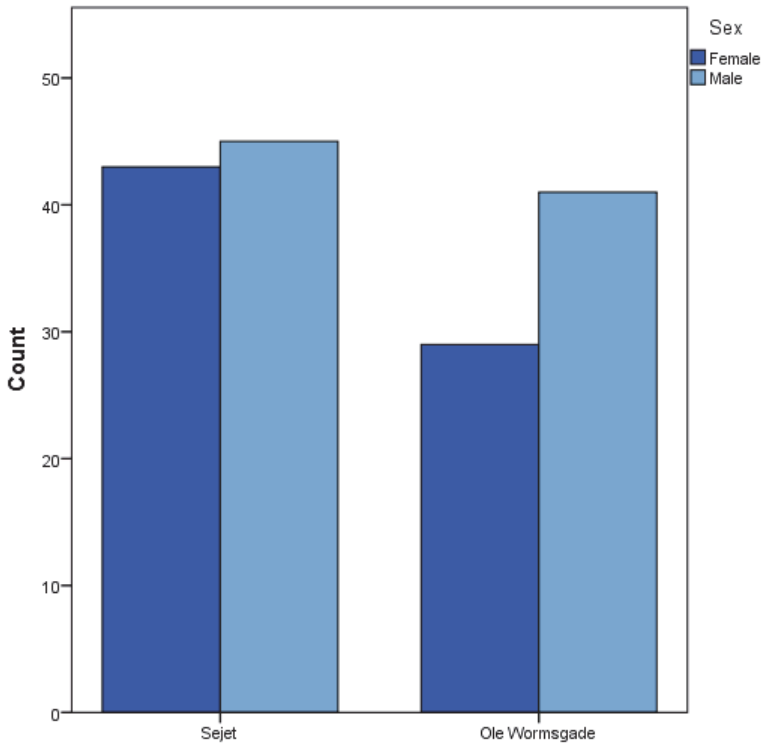


Table 3-3 Sample distribution by sex with percentages calculated in relation to the total number of individuals for whom sex could be estimated from each cemetery sample used in this study. The 'total' columns reflect the numbers and percentages by sex in relation to the combined cemetery subsamples used in this study.

		Study Cemetery Samples				Total	
		Sejet		Ole Wormsgade			
		N	Percent	N	Percent	N	Percent
Sex	Female	*43*	*45.3%*	*29*	*40.3%*	*72*	*43.1%*
	Male	*45*	*47.4%*	*41*	*56.9%*	*86*	*51.5%*
	Unknown	*7*	*7.4%*	*2*	*2.8%*	*167*	*4.2%*
Total		*95*	*100%*	*70*	*100%*	*158*	*100%*

Age Estimation

Age estimation was conducted using a multifactorial approach incorporating traditional morphological techniques, and complemented using a system being developed at ADBOU, known as expert inference (Tarp, 2009; Weise et al., 2009, 2012; Milner and Boldsen, 2012a) . Continuing work is being conducted with this method as part of a collaborative effort (Weise et al., 2009, 2012)[11]. The morphological techniques applied were pubic symphysis (Todd, 1920) and auricular surface (Lovejoy et al., 1985) morphology, dental wear (Lovejoy, 1985), and cranial suture closure (Meindl and Lovejoy, 1985). All of the above features were scored separately, with the expert inference (Tarp, 2009; Weise et al., 2009, 2012; Milner and Boldsen, 2012a) being conducted as a final stage. The expert inference involved the assessment of discrete features (the linea aspera, femoral caput fovea, femoral fossa trochanteria, tibial intercondylar eminence, and acetabular margin [Tarp, 2009]) combined with inference based on overall skeletal appearance. The statistical calibration of all of these features into a single age-related score is still under development and so was not integrated into the current analysis. As such, the application of this approach can only be termed expert inference and the full calibrated expert inference methodology (CEI) (Weise et al., 2009, 2012) could not be applied.

A final age range was derived based on these cumulative scores, and the median of this range was used as a proxy for age in the statistical analyses) with mean ages for the subsamples calculated from these median ages. Further examination of the age distribution can be found in the overall sample review of the results chapter. It is important to emphasize here that the scores used for the age of individuals *were* median

[11] See Literature Review for further details

scores based on age ranges acquired through the multi-factorial osteological analysis. A more sophisticated approach to age determination was not undertaken, partly due to issues of preservation which meant that in many cases adequate elements for the application of a statistical approach such as transition analysis (Boldsen et al., 2002; Boldsen, 2008; Milner and Boldsen, 2012b) were not available.

Stature estimation

Two methods for stature estimation were utilized. The first of these was stature as measured in the grave, which is used as a proxy for anatomical height (Boldsen, 1984a; Petersen, 2005, 2011) (Figure 3-8, Table 3-4). The stature in the grave value is based on a strict *in situ* measurement of the prone individual from the most distal point of the talus to the most superior point on the cranium, and has been shown to accurately represent individual stature at death (Boldsen, 1984a; Petersen, 2005, 2011). These measurements were recorded from the original excavation sheets as per availability. They were particularly uncommon for Ole Wormsgade since many of the burials were truncated. Petersen's (2005) correction of adding 2.5 cm on to the skeletal length in the grave to attain anatomical stature determinations was further applied to the measurements taken from the field notes.

No correction for age at death was integrated, as stature in the grave measurements are typically taken as accurate representations of the anatomical height of individuals at death (Boldsen, 1984a; Petersen, 2005, 2011). While it is possible that reduced stature with age could confound results (counter-acting any increased stature with predicted to be associated with increased survivorship for example), a correction factor was not applied as it was seen as more preferential to maintain consistency with the

published literature. Furthermore, the introduction of any such age-based correction factor could be confounded by variability in age-related height reduction across individuals and by inaccuracies in age estimation techniques. Such an approach would require further assumptions which might unnecessarily introduce further inaccuracies. As such, stature in the grave measurements were used directly as proxies for anatomical height.

The other stature estimates were derived from standard measurements using an osteometric board from all sufficiently preserved tibiae and femora (Figure 3-8, Table 3-4). Average estimates were taken from the left and right sides in the interests of maximizing sample size, and these measurements were used directly as proxies for stature throughout the analysis (Boldsen, 1984a). Femoral measurements proved to be available for more individuals at Ole Wormsgade than stature in the grave measurements and so both these and stature in the grave were employed in the analysis to consider patterns in health as they relate to height and body proportions.

Beyond the raw consideration of femoral lengths and stature in the grave, a consideration of body proportions was of interest in light of findings which suggest that body proportion but not size is impacted by childhood stress events (Boldsen, 1998). In order to consider this relationship, a long bone measurement (in this case femoral length, which was the most commonly available element) needs to be considered next to stature (in this case stature as measured in the grave). The approach taken in this research was to calculate a predicted height from femoral lengths and to compare this to stature in the grave measurements (used as a proxy for actual height). In order to acquire a predicted stature from femoral lengths, sex-specific regression formulae previously developed from

the nearby Tirup population were calculated. It was hoped that this would elucidate any divergence of predicted height (from femora) from actual height (from stature in the grave). The regression formulae applied to this sample (Boldsen, 1990a) are as follows:

$$\text{Female Stature}_{(\text{from femoral lengths})} = 2.318 * \text{femoral length} + 54.51$$

$$\text{Male Stature}_{(\text{from femoral lengths})} = 2.318 * \text{femoral length} + 58.16$$

While the development of sample specific regression formulae was considered, such an approach, given the small sample sizes, would have necessarily included all individuals in the sample. To then use the resulting height measurement to compare again to stature as measured in the grave would have been inappropriate. The inclusion of tibial measurements was also considered with the aim of increasing the sample size. However, consideration of the sample distribution showed that this would have only increased sample size by four individuals and all individuals were unsexed individuals from Ole Wormsgade, with no associated date. Especially given the already particularly small size of the Ole Wormsgade sample, such an approach would have potentially introduced severe bias into the analysis and thus tibial lengths were not integrated. Finally, the calculation of predicted stature for individuals for whom sex could not be determined was conducted by calculating stature using the formula for each sex and then averaging these two results. Any insight possible through a consideration of residual height will be briefly examined over the course of this thesis.

Figure 3-8 Sample sizes for different stature metrics. Through this it is apparent that femoral measurements are available for slightly more individuals than the next most common measurement (stature as measured in the grave). This is due to slightly more individuals having complete femora available from Ole Wormsgade in relation to the number of individuals from this cemetery for whom stature in the grave was available. The lower number of individuals available for each measurement at Ole Wormsgade reflects the nature of this cemetery with frequently truncated burials.

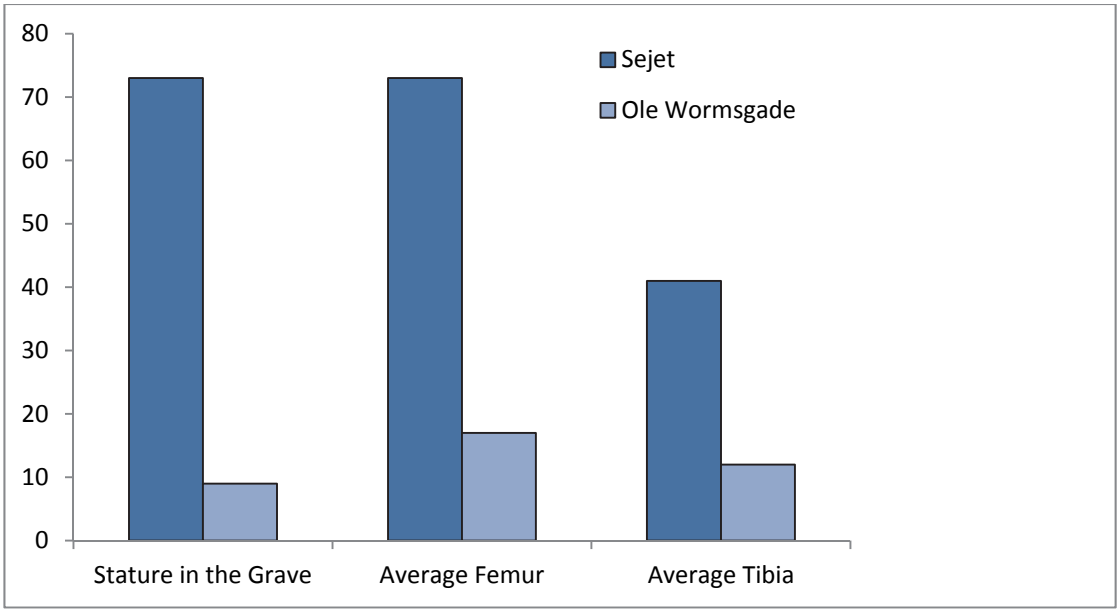


Table 3-4 Sample sizes for different stature metrics, with each then expressed as a percentage of the total sample for each category (combined cemetery samples used in this study, Sejet subsample, and Ole Wormsgade subsample respectively)

Cemetery		N	Percent
Total	Stature in the Grave	*82*	*49.1%*
	Average Femur	*90*	*53.9%*
	Average Tibia	*53*	*31.7%*
Sejet	Stature in the Grave	*73*	*76.8%*
	Average Femur	*73*	*76.8%*
	Average Tibia	*41*	*43.1%*
Ole Wormsgade	Stature in the Grave	*9*	*12.5%*
	Average Femur	*17*	*23.6%*
	Average Tibia	*12*	*16.7%*

Infectious Disease

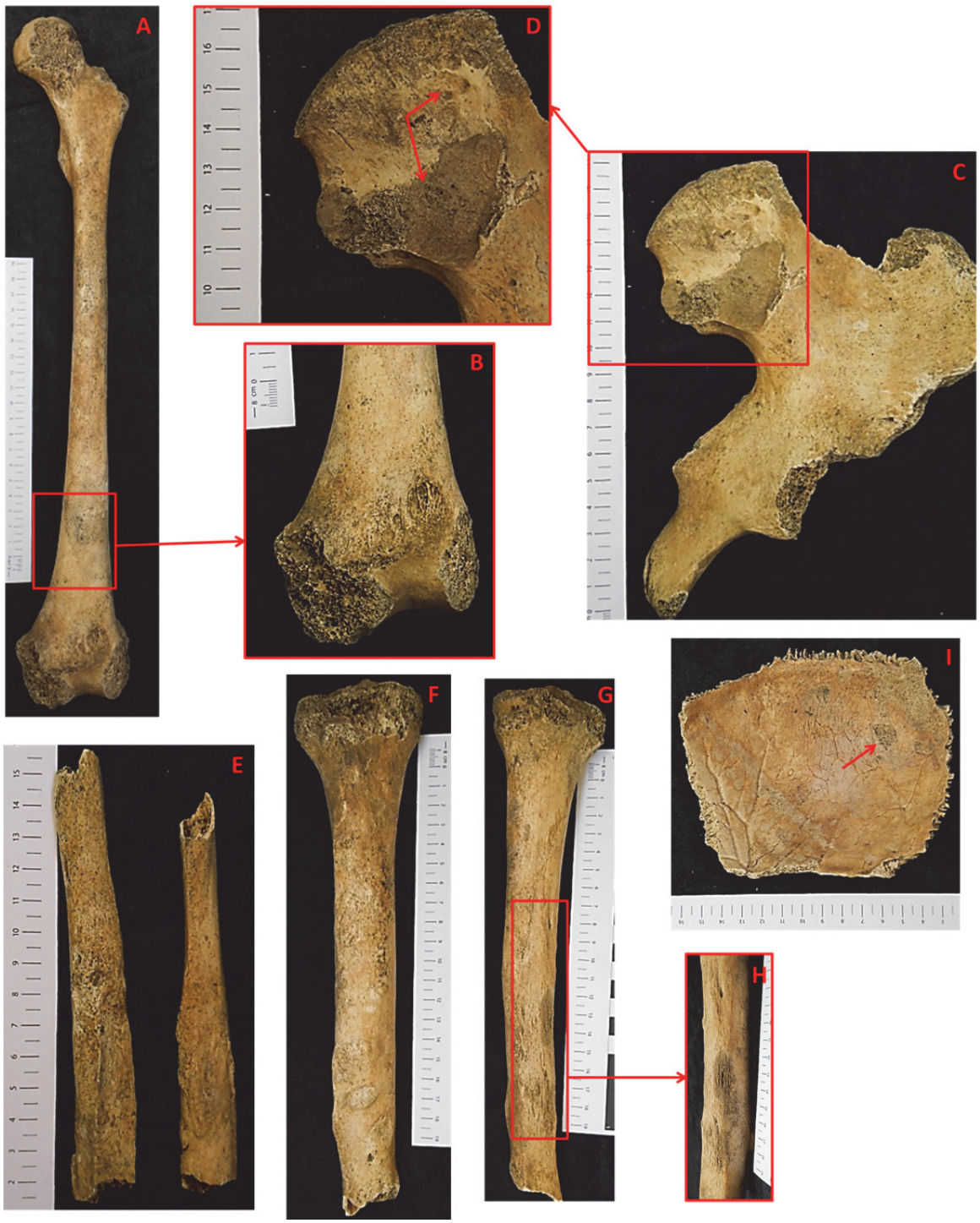
Three primary infectious diseases were diagnosed in this sample. These were tuberculosis, leprosy, and treponematosis. Differential diagnosis was performed for each disease, and the diagnoses were made based on the expression of the lesions (lytic, sclerotic, etc.) and their pattern of distribution across the skeleton. Since differential diagnosis can be problematic in skeletal remains, particularly when dealing with fragmentary material, individuals were classified as probable cases (if characteristic indicators were visible), possible cases (when lesions suggested a possible diagnosis but where this diagnosis was still uncertain due to missing or poorly preserved elements), or as no disease (in cases with sufficient preservation of elements but with elements exhibiting no evidence for a diseased state). Scoring for these diseases was based on standard osteological indicators for the diseases (Aufderheide and Rodriguez-Martin, 1998; Waldron, 2009; ADBOU, 2010). These cases are clearly distinguished throughout the analysis, with stronger emphasis placed on probable cases.

Treponemal disease was considered in a broad sense for this population. No attempt was made to identify the different forms of treponemal infection. Diagnostic criteria for treponemal disease used in this research were developed from the ADBOU Chronic Diseases manual (2010) and included (Figure 3-9):

- Necrotic changes to the frontal, parietal, and / or occipital bones
- Swelling of the periosteum in the clavicle, humerus, and / or femur (posterior distal femur)
- Periosteal swelling of the proximal ulna with pitting possibly occurring on the olecranon process

- Periosteal swelling in the tibia, with the extreme form of this manifesting in the characteristic ‘saber shin’ appearance

Figure 3-9 Treponemal changes to femur with pitting and bone remodelling particularly at the distal end (A and B – inset); ilium with pitting and bone remodelling on and around the auricular surface (C and D – inset); fibula with periosteal swelling and exostoses (E); tibia with sclerotic lesions, periosteal swelling, and the beginnings of saber shin formation (C and H inset); and parietal with lytic lesions (I). HOM 1046 A179 X179



The diagnosis of tuberculosis was far more problematic, as changes to the vertebral and joint surfaces (particularly load bearing joints) may be difficult to distinguish from osteoarthritic changes, as well as from other conditions of both infectious (brucellosis) and traumatic (anterior disc herniation) etiology (Mays, 2007). While only a small proportion of tuberculoid cases will experience skeletal involvement (Steinbock, 1976; Aufderheide and Rodriguez-Martin, 1998; Ortner, 2003; Roberts and Buikstra, 2003), tuberculosis can cause pathological changes particularly to the thoracic and lumbar regions of the spine and to weight bearing joints (Aufderheide and Rodriguez-Martin, 1998; Ortner, 2003; Roberts and Buikstra, 2003; Steinbock, 1976; Tuli, 2004). The suite of diagnostic changes for tuberculosis used here included (Figure 3-10, Figure 3-11, Figure 3-12):

- Inflammatory changes with porotic development and lytic lesions on the vertebral bodies (particularly towards the anterior margin), and expanded nutrient foramina on the vertebral bodies.

AND

- Inflammatory changes to the auricular surface of the ilia, with pitting, erosive change, and reactive bone growth.
- Similar inflammatory and erosive changes to the distal femora and proximal tibia (i.e. to the knee joint).

Detailed descriptions of these scoring systems and accompanying images can be found in the ADBOU Human Osteological Methods manual for chronic diseases (ADBOU, 2010).

Figure 3-10 Probable tuberculoid lesions showing lytic development in the auricular surface of the ilium (A with B inset); lytic development in the acetabulum (C); porosity on the femoral head (E with D inset and F); anterior destructive lesions on a lumbar vertebra (G); and expanded nutrient foramina in the vertebral column (H). HOM 1046 A11 X11.

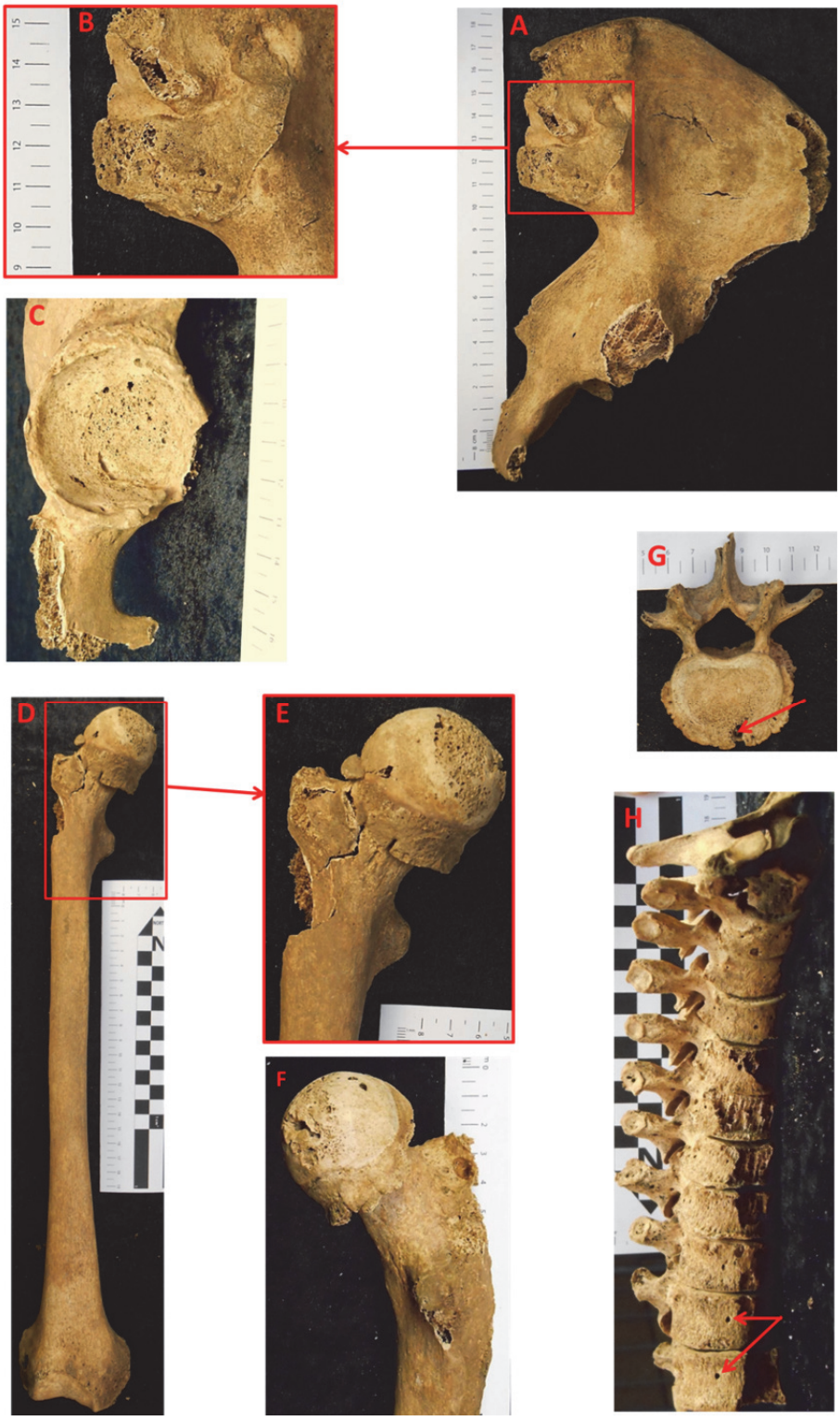


Figure 3-11 Possible tuberculoid changes to the vertebra showing porotic development (A) and expanded nutrient foramina (B) and to the auricular surface of the pelvis showing disruption of the joint surface with porotic development (C). HOM 1649 A1543 X1543

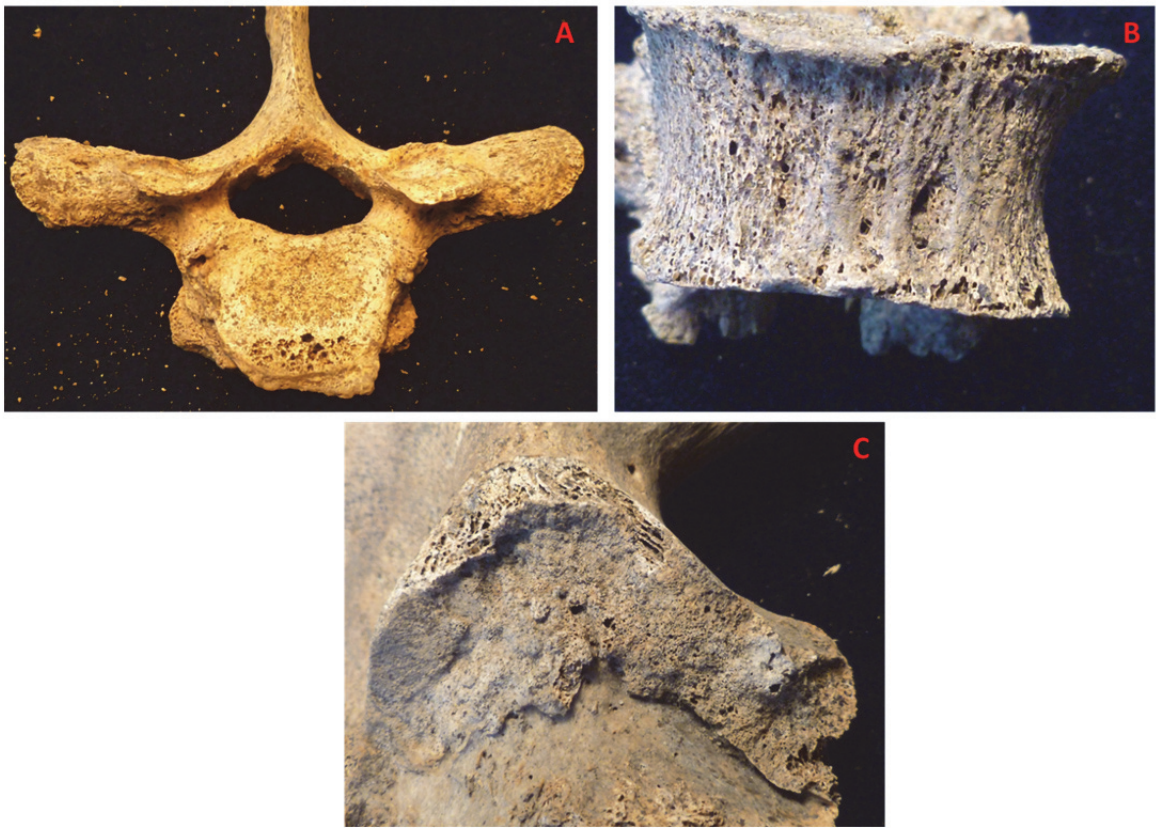
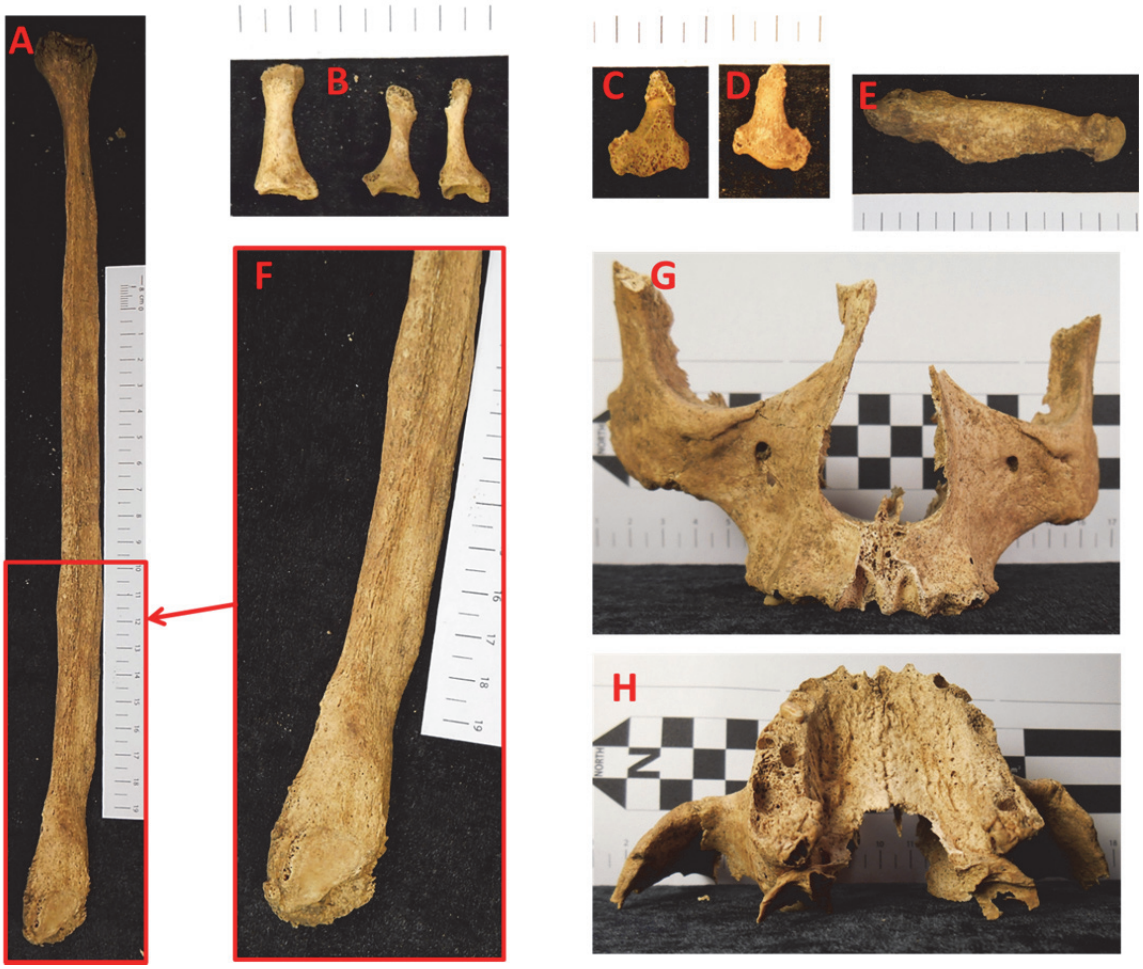


Figure 3-12 Possible tuberculoid changes to the vertebra showing anterior lytic lesions to the vertebral body (A) and expanded nutrient foramina (B), disruption of the acromial joint surface with some lytic development in the claviculae (C) and auricular surface of the pelvis with increased porosity to the joint surface (D). HOM 1649 A1519 X1519

The lesions associated with leprosy have been thoroughly investigated in the anthropological literature. The criteria are available in well published standards developed from Danish medieval material as described by Boldsen and Freund (2006). The pathological changes observed in the diagnosis of leprosy were (Figure 3-13):

- Remodelling along the edge of the nasal aperture
- Nasal spine remodelling/rounding /disappearance
- Destruction of the alveolar process, particularly over the maxillary I^1's
- Pitting or perforation of the maxillary palatine process
- Subperiosteal exostoses on the fibula
- Fibular hypertrophy
- Bony changes to the 5^{th} metatarsal
- Phalangeal fusion (in at least one joint resulting in a hook-like position)

Figure 3-13 Probable leprous changes to the (A) the fibula involving subperiosteal exostoses and hypertrophy; (B-D) the phalanges involving atrophy with 'pencilling in' appearance; (E) bony changes expressed on the 5th metatarsal (G) the maxilla involving resorption of the nasal aperture with possible resorption along the anterior alveolar process; and (F) the maxilla with destruction of the hard palate.



Unfortunately, later consideration of the material revealed issues with the scoring method initially applied, since missing material was not adequately integrated into the scores. This resulted in the retrospective re-scoring of lesions based on laboratory records (visual records, laboratory notes, and database records for individual elements) and in further review of individuals at ADBOU with a focus on scores for treponema and tuberculosis. Leprosy diagnosis was facilitated by the availability of the lambda scoring system produced through ADBOU (Boldsen, 2001, 2005c, 2008; Boldsen and Freund,

2006). This system is the product of many years of work to calculate the specificity and sensitivity of leprosy-related lesions, to test conditional independence for these lesions, and to integrate all of this information as well as missing element information into a statistical program which will calculate lambda scores for leprosy likelihood and prevalence. The reader is once again referred to the published work on this research for the standards of lesion identification and for the statistical frameworks behind the calculations (Boldsen, 2001, 2005c, 2008; Boldsen and Freund, 2006).

Despite the fact that these leprosy scores were generated retrospectively and not directly from the skeletal remains (but rather being record based), it is likely that through the integration of the lambda scoring system and as a result of the detailed records available from the initial skeletal scoring in Denmark, the leprosy scores more adequately reflect the true pathology prevalence rates in these samples. Any unobservable or missing features were scored as missing in the program, and so the scores do likely under-reflect the scores which might be taken directly from the skeletal remains.

As a result of this process, the scores for probable versus possible cases of leprosy used in this thesis are more strongly quantifiable and are slightly different from those used for tuberculosis and treponematosis, for which no such system of calculation was available. In the case of leprosy diagnosis, any individuals who had λ of between -1 and 1 were classed as possible cases, while individuals with scores equal to or greater than 1 were classed as probable cases (Boldsen, 2008). Individuals below -1 were categorized as unlikely to have suffered from leprosy (Boldsen, 2008). Due to these differences, it is not possible to directly compare the different diseases, but individual disease patterns can be considered.

It should be noted that prevalence rates of these diseases may be impacted in these subsamples by the selection criteria for individuals with relatively unworn teeth. Tuberculosis, for example shows skeletal involvement most frequently in individuals within the first three decades of life (Roberts and Buikstra, 2003). Leprosy, on the other hand, having a very different disease progression than tuberculosis, also shows a different relationship to mortality. Leprosy does not directly impact mortality in the same way as tuberculosis, rather having a severe impact on morbidity and quality of life. The disease develops relatively slowly, and the negative health impact is more heavily connected to disability and infection. In this vein, analysis of the nearby medieval Danish site of Tirup has shown that individual with signs of leprosy show a similar mortality profile to those who do not show signs of the disease (Boldsen, 2005b). As such, it is suggested that a similar mortality bias will not be seen with leprosy cases as it may in tuberculosis cases. Treponema shows different patterns depending on mode of contraction and form of the disease (Rothschild and Rothschild, 1995). Yaws and bejel are more commonly contracted in subadults. Syphilis on the other hand is sexually transmitted in reproductively mature individuals, although it may also be transmitted congenitally (Harper and Knauf, 2013). There may be an extended incubation period going into multiple decades, and this will complicate osteological diagnosis (Harper and Knauf, 2013). While contraction of the disease may occur prior to the age of 18 (particularly in a society where marriage often occurs prior to this age), we would expect appearance of osteological changes to occur from early adulthood onwards, consistent with age captured in this study.

Preparation for microscopy

Surface Microscopy

Casting

In preparation for the surface examination of the dental microstructures, and as part of the procedure for preserving a record of these teeth, high resolution casts were taken of all the teeth. These were done using silicon casting materials and epoxy resin. The teeth were first thoroughly cleaned, as described below for the thin sectioning methodology. Coltene President Regular Body Impression Material Plus Regular Body Surface Activated Compound (blue colour) was used to create the moulds (Thomas, 2003; Galbany et al., 2006). This is a polyvinylsiloxane impression material which is recommended over latex for its flexibility and gentleness on fragile materials (Hillson, 1992b). A Coltene applicator gun was used to inject the impression material into 1" diameter plastic tubing (with ¼" thick walls) which had been cut into sections which were 1 ½" in length. Impression material was injected into these moulds and the tooth crown pressed down into this arrangement. The impression material cures in 1 to 2 minutes, and since it begins to cure as soon as it is mixed, it cures in the Coltene mixing tip as well as in the mould. Once cured in the tip, the tip has to be discarded. Furthermore, the teeth had to be left long enough for the impression material to cure enough to maintain integrity, but if they were left too long it was difficult to get them out of the negative impressions safely. As a result, teeth were laid out ready for the negative impressions to be taken. The segments of plastic tubing were labelled with the individual number and set out by the teeth. The impression material was injected into the plastic tubing with a one minute interval in between injections and the tooth crown was immediately pushed down into the impression material. The one minute interval was to ensure enough

spacing between injections to allow time for each tooth to be removed safely before the next one was ready. It also meant that the mixed impression material could be used quickly enough not to plug the tip during a single session[12].

Once the negative impressions had been produced, they were inspected using the Olympus SZX12 Research Stereoscope for any signs of bubbles or missed areas. If the mould was satisfactory, the casting stage could begin. The same epoxy resin and hardener (EpoThin® Epoxy Resin was mixed with EpoThin® Epoxy Hardener) that was used to embedding the teeth prior to thin sectioning (see below) was used at this stage[13]. The resin mix was placed in a vacuum chamber to evacuate bubbles before being poured carefully into the negative impressions. These were then placed in plastic zip lock bags (3 to a bag) to account for any spills. They were evacuated once again and then placed in a ThermoFisher Scientific Sorvall ST16 centrifuge at 2500 rpms for 3 minutes to ensure that all crevices were filled with resin and to separate any residual air pockets out of the mould (Galbany et al., 2004). The resin was then left to cure overnight before extracting the positive casts.

[12] Before the tooth is removed, the impression material is checked to make sure it has cured enough. It should no longer be tacky, but should still be quite flexible, ie. Just past the 'tacky' stage.
[13] See the next section on thin sectioning for details on mixing this epoxy.

Internal Microscopy

Thin section procedure

A sample of teeth from 79 individuals was thin sectioned in order to gain further insight into growth and growth arrest during development in the individuals being studied (Table 3-5). These individuals were selected based on further review of the teeth once back in the Bioanthropology Digital Image Analysis Laboratory (BDIAL) at the University of Manitoba. Upon this re-evaluation, it was determined that some of the teeth initially selected were too worn for this stage of the analysis, with most of the zone of apposition (in the cuspal portion in which striae of Retzius do not come to the surface) worn away. In these cases, since striae of Retzius do come to the surface as perikymata in the imbricational enamel which tended to be preserved beyond the wear, it was determined that thin sectioning was not warranted. Sectioning would reveal individual cross-striation counts, but would tell not more about patterns in the long period markers than could be gained through surface microscopy. Furthermore, once greater familiarity with this sample was gained, it became clear that certain patterns in surface colouration were consistently tied to loss of integrity in the enamel microstructures. A few patterns were observed, one showing black inclusions and the other apparent as a white opacity often encompassing much of the enamel. After a few attempts to view microstructure in such teeth, further teeth showing these patterns were eliminated from the thin sectioned sample.

While thin sectioning is a destructive procedure, its potential to provide information over a greater age range in well-preserved teeth justified its application. This procedure also made it possible to consider enamel microstructures which were hidden on the surface by calculus which could not be removed in cleaning without damaging the

enamel. All processing took place in the (BDIAL). In order to maintain a record of the teeth being destroyed, measurements were taken, the 3D imaging was conducted, and high resolution casts were taken prior to destructive analysis. The thin sectioning procedures used were adapted from those used in the *Anthropology Hard Tissue and Light Microscopy Laboratory* at McMaster University (FitzGerald and Saunders, 2006) and in the *Reid Laboratory* at the University of Newcastle upon Tyne (England) (as outlined by Thomas, 2003: 74-75). Reference was also made to Hillson (1996) in relation to cutting, lapping and polishing procedures[14].

Table 3-5 Number of teeth by tooth type used for the internal analysis (with each tooth representing one individual).

Tooth Type	Number of Individuals / Teeth
Mandibular Canine	75
Maxillary Canine	2
Maxillary I1	1
Maxillary I2	1

All teeth were cleaned first using ethanol to remove most matrix and then with acetone to remove any residual deposits, including some organic deposits. A soft-wood pick was used under the Olympus SZX12 Stereomicroscope to remove additional deposits from the enamel surfaces. While some methodologies recommend the use of a weak soap solution (Thomas, 2003; FitzGerald and Saunders, 2006), Hillson (1996) cautions that the application of water can lead to cracking, particularly with fragile specimens. As such, Hillson's suggestion of using alcohol and acetone applied with

[14] The method of cutting here, in which the initial cut is made, the cut edge lapped and polished and then affixed to a slide, the slide fixed to the arm of the saw and then cut down to a section which is then lapped and polished again to finish, follows the guidelines outlined by Hillson (1996).

cotton swabs was adopted. Prior to embedding the teeth in epoxy resin, the section plane, which is the midline axial plane in anterior dentition, is marked using a graphite pencil (Antoine, 2000). Effectively a line is drawn on the buccal surface of the tooth from the lowest point on the cervical margin to the tip of the cusp, and then extended down through the tooth (FitzGerald and Saunders 2006).

FitzGerald and Saunders (2006) used cyanoacrylate to infiltrate and stabilize the enamel during a cut, but caution against its use in some archaeological teeth which might have fragile enamel. As many of the teeth from these samples were quite fragile, it was decided to proceed with the more secure (but time consuming) method of fully embedding the teeth for this stage of the project. The embedding agents used for this research were those recommended by FitzGerald and Saunders (2006) and used previously in BDIAL. This method employs EpoThin® Epoxy Resin mixed with EpoThin® Epoxy Hardener in a 5:1.95 ratio. Measurement of these is done by weight using an electronic scale under a fume hood. It is extremely critical that these measurements are as exact as possible, to avoid poorly cured resin. All moulds were pre-prepared by first applying epoxy release agent to the inside of the moulds and then mixing the epoxy compound and pouring a thin layer (approximately 1/8") into the bottoms of the moulds to create a buffer[15]. This was allowed to cure for approximately 5 hours until it was stiff but still tacky. The buffer had to be firm enough to support the tooth but not too firm for the tooth to be positioned. Once this stage had been reached, the teeth were positioned in the moulds so that they lay on their sides and the midline lay flat in the mould. Another batch of resin was mixed and the bubbles evacuated while

[15] Plastic disposable cups were used for the moulds. Two teeth could be positioned in these side by side and they were easily cracked to release the resin puck with embedded teeth afterwards.

teeth were being positioned. This was then poured over the teeth to just cover the teeth in the mould. These were then placed back in the vacuum chamber with the pressure brought down to 20 millibars and left overnight.

Once the moulds were fully cured, the epoxy puck containing the embedded tooth was removed from the mould and mounted on the chuck. For this project, thin sectioning made use of the Buehler IsoMet® 1000 low speed saw for initial cutting. The resin block was affixed to the IsoMet® 1000 saw arm using the clamp chuck provided by Buehler. The smallest blade which allowed minimum clearance was selected to reduce wedging (this was size 5, with a flange of 2.95 and 1.03" clearance). A minimum amount of weight (26 lbs) was used and the speed was set at 100 RPM, in order to produce the cleanest cut possible. Occasionally, it was necessary to shave excess resin off before performing the first cut. This allowed for more precise positioning for this cut. In these cases, more weight and speed was used since the specimen was not involved in the cut.

The main cut had to run directly down from the tip of the dentin horn, and so accuracy was paramount[16] for any teeth in which cuspal enamel was preserved. As such, care was taken to position the sample so that, taking into account the blade width of 400 µm, which would be lost in the cut, the edge of the section would follow the correct line. Once this was established, the micrometer on the IsoMet ® 1000 was set to zero, and the saw arm was lowered onto the running blade to make the first cut.

[16] As many of the teeth in this sample were worn beyond the cuspal enamel, finding the precise midline was not always as critical as it was for teeth with intact cuspal enamel. When this occurred, the cut was positioned to run perpendicular to the buccal perikymata and to maximize the amount of enamel exposed in the cut.

Upon completion of the first cut, the chuck was removed from the arm (taking care not to change the arm position). The specimen number was marked on a slide and the slide was heated on a hot plate so that a thin layer of dental sticky wax could be applied to the portion of its surface which would be mounted to the section. The cut surface of the section was then pressed down onto the slide (with the chuck still in place, taking care not to allow the resin block to move in the chuck). The resin block with the slide attached was then removed from the hot plate and allowed to cool until the sticky wax had set (this only takes approximately 30 seconds). A slight amount of pressure is applied at this stage to ensure as close adhesion as possible. The chuck was then re-attached to the saw arm and the arm was adjusted to produce a 400 μm section (once again taking into account the saw blade thickness). In order to do this, the micrometer is zeroed, and then moved over between 600 and 700 microns to produce a final section which was approximately 450 microns thick. If at all possible given tooth size and condition, a second section was also removed by once again adhering a slide and adjusting the arm position. This was done as a safeguard in case issues arose with later steps in thin section preparation on the first section. Once the block is removed from the chuck, or the arm re-positioned, the ability to attain a relatively precise estimate of slice thickness is eliminated, and so the second section was ideally taken at this stage.

Once the section(s) had been produced, the process of lapping and polishing could begin. The main objectives of this stage were to eliminate the saw marks and to attain as even a thickness across the section as possible. The extent to which the first side of the section was polished depended on the nature of the cut. If the exposed surface lay closer to the midline of the tooth than the other side of the section, a minimal amount of

polishing was done. If the midline of the tooth lay closest to the second surface (the one affixed at this point to the slide), then the section was ground down to around 250 μm at this stage.

While initially this procedure used the slide chuck associated with the MiniMet® 1000 to help with holding the slides during grinding, a target holder was attained from Buehler to help ensure even and faster polishing for approximately three quarters of the sample. Each slide was mounted into the target holder and ground according to the above criteria using 600 grit lapping paper (Hillson, 1996). Once the slide was close to the desired thickness, polishing was done using progressively smaller grits of Aluminum Oxide Abrasive Powders, beginning with of 9.5 μm (FitzGerald and Saunders 2006) and then proceeding through to 5 μm and 3 μm powders. At each stage, the surface was washed with distilled water and carefully observed for scratches. Only once scratches had been just eliminated was the slide washed in an ultrasonic bath with a small amount of soap to remove any residual grit and the next stage commenced. Care was taken to apply these steps as conservatively as possible to achieve the desired end of eliminating scratches. The section thickness was constantly monitored using a micrometer, with the goal of not polishing beyond 200 microns. This was, however, assessed on a per tooth basis to attain a final section as close to the ideal plane as possible (Antoine, 2000).

Once it had been put through the final cleaning, the polished side of the section was affixed to a clean slide[17] using Logitech (Struers) OCON-186 UV resin. This is a single part urethane acrylic bonding medium that cures under long wavelength UV light. Under the fume hood, a thin layer of the resin was spread on the sectioned surface and

[17] The slides were all cleaned beforehand with 99% ethanol to sterilize their surfaces.

this was then affixed to a cleaned and labelled slide to create a slide 'sandwich', which was clamped together and cured for 1 to 2 minutes under a Black Light UV lamp (FitzGerald and Saunders 2006). Once the second slide had been attached to the section, a hot plate set on low heat was used to melt the sticky wax on the first slide. This slide was then removed and any excess wax wiped from the section surface using KimWipe®. The slide was then rinsed with distilled water followed by ethanol, and then soaked in Xylene for five minutes before being wiped clean again of wax and unpolymerized resin (FitzGerald and Saunders 2006).

Lapping and polishing of the second surface proceeded in a similar fashion to the first surface, with the objective being to attain a polished section of 100 µm thickness. This process was constantly monitored using a digital micrometer caliper to check thickness along six points according to FitzGerald and Saunder's standards. The process was started with the 9.5µm aluminum oxide powder, and preceded with this grade until the sample had been evenly lapped down to between 120 and 140 µm. At this stage, each slide was carefully monitored and some were left thicker than others depending on their structural integrity. In some cases, 9.5 µm powder was used more extensively to reach the final satisfactory thickness. Once this thickness was attained, the process of progressively finer polishing outlined for the first side was followed to remove all scratches.

Analysis of Dental Thin Sections

Image Capture

Dental thin sections were analysed using an OLYMPUS BX51 microscope. Image capture was performed using an OLYMPUS DP72 digital camera in conjunction with OLYMPUS Stream Motion software (version 1.6.1), which was also used for image analysis. This system made it possible to perform automated photomontaging though the use of a motorized stage and the tiling functions of the software. A complete image of the dental thin section could therefore be captured and the crown considered along its entirety.

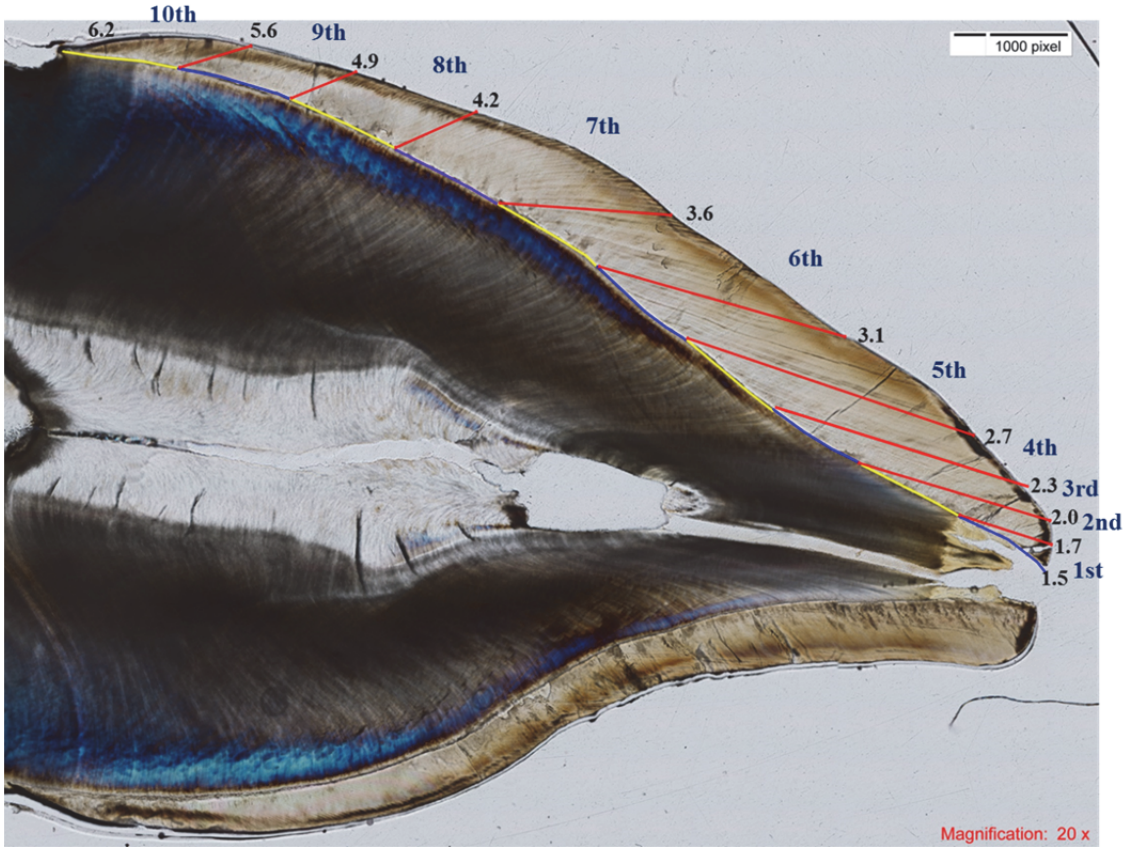
The initial stage to image capture was to establish an overview area using OLYMPUS Stream. The capture coordinates could then be easily established. A magnification of 10x was sufficient to capture most striae of Retzius during final image capture. On occasion, 20x magnification was utilized to increase clarity in some more problematic teeth. The focus map function on Olympus STREAM was utilized to attain the highest level of focus across the image as possible, since despite careful grinding and polishing, there was still minor fluctuation in section thickness (on the scale of 20 microns in some cases). The focus map involved the manual adjustment of focus to the optimum point at three places across the scan area. In the case of the dental thin sections, these three points were established along the labial surface of the crowns, one towards the cervical area, one at the mid-crown point, and one towards the cusp.

Image Analysis

Each crown was divided into deciles which were attributed to age ranges based on those developed by Reid and Dean (2006) (Figure 3-14). The sample used for these age ranges were Northern European in origin. Specifically, they were from the medieval

Danish town of Tirup (Reid and Dean, 2006; Reid and Ferrell, 2006), which was contemporary and close geographically to the two sites used for this study. As such, these ages for enamel development might be considered compatible for the current study populations. Following Thomas' (2003) approach, each decile was also corrected for the duration of growth in that decile by dividing the decile count by the amount of time spent during that growth interval (as estimated by Reid and Dean [2006]).

Figure 3-14 Approximate ages for crown divided into deciles. The ages at each decile mark are given in black numbers. The decile numbers as referred to in this dissertation are labelled in black from the 1st to the 10th decile. All ages at decile marks have been taken from Reid and Dean (2006). (Ole Wormsgade X1019)



Since these teeth were drawn from adult individuals, difficulty was encountered with dental wear. This meant that in some cases as much as the earliest two deciles of

enamel had been worn off. However, the dentin horn was far more complete than the enamel cusp and as such could be utilized to estimate the complete crown height. As a result, a measurement was taken from the CEJ to the dentin horn along the border of the dentin and the enamel. In some cases, the tip of the dentin horn had also been affected by some wear. In these cases, it was necessary to estimate the position of the dentin horn. This measurement was then divided into ten regions which were marked by measurement lines in the Olympus STREAM software. Upon completion of these subdivisions, points were placed on each identified accentuated striae.

The issues surrounding the identification of AS have been discussed in the literature review. For the purposes of this thesis, only those striae which stood out in relation to other striae in the surrounding region of the crown (divided according to cuspal, mid, and cervical thirds) were counted. These may or may not have been associated with surface defects. The number of AS in each decile was then recorded in a Microsoft Excel database which could be transferred into SPSS.

All teeth were scored five times using this methodology. The first scoring was extremely liberal in what was counted as an AS and what was not. During this trial, scoring was conducted with Goodman and Rose's (1990) definition of striae extending at least three quarters of the way to the enamel surface was employed. The second scoring led to some development of this method, with scoring being more conservative and only counting those AS which stood out particularly as strong striae in relation to the average striae in their close vicinity. In addition, the first scoring would have been influenced by the learning curve required to gain a better understanding of these structures. Finally, three more trials were conducted on different days to further account for any learning

curve and in an attempt to test consistency in results across trials.

Surface Analysis

While initially, some casts were imaged using a SEM at the University of Manitoba Department of Engineering, the SEM did not capture surface information in a measureable form and so was not particularly useful for an attempt to objectively identify surface microdefects. While the SEM did capture excellent images of the enamel surfaces, no profile information of the crown surface which would allow for the quantification of defect depth and width or distance between perikymata could be captured. All cast teeth and a portion of original teeth which had not been sectioned were therefore analysed for surface defects using an Olympus LEXT 3D Laser Measuring Microscope OLS40000, courtesy of Dr. Brooke Milne (Department of Anthropology, University of Manitoba). This microscope made it possible to capture high resolution surface images with associated profile information on surface relief. However, only preliminary results were possible from this portion of the analysis as further methods development is require to fully integrate this system. The LEXT has not been used for this type of analysis previously, and so this analysis presents the preliminary exploration of its utility. Further development to maximize magnification level while balancing the z-plane focal measurements and minimizing the signal to noise ratio is required.

For the purposes of this research, teeth were scanned using the 10x magnification lens, which reduced the signal to noise ratio while permitting the capture of a full longitudinal strip of the tooth crowns. This produced images of the teeth which could be rendered in 2D or 3D. The 2D images were sufficient for this preliminary analysis (Figure 3-15), but the images captured both the z and the x – y plane information and can

consequently be rendered in 3D if desired. Figure 3-16 illustrates the z-plane dimension, in this case reflected by a profile line drawn longitudinally from the CEJ to the cusp.

Figure 3-15 LEXT Surface Images showing an image based on a cast (top) and an image based on the original tooth (bottom). The red arrows mark the same defect as visible in each image. (Sejet X72)

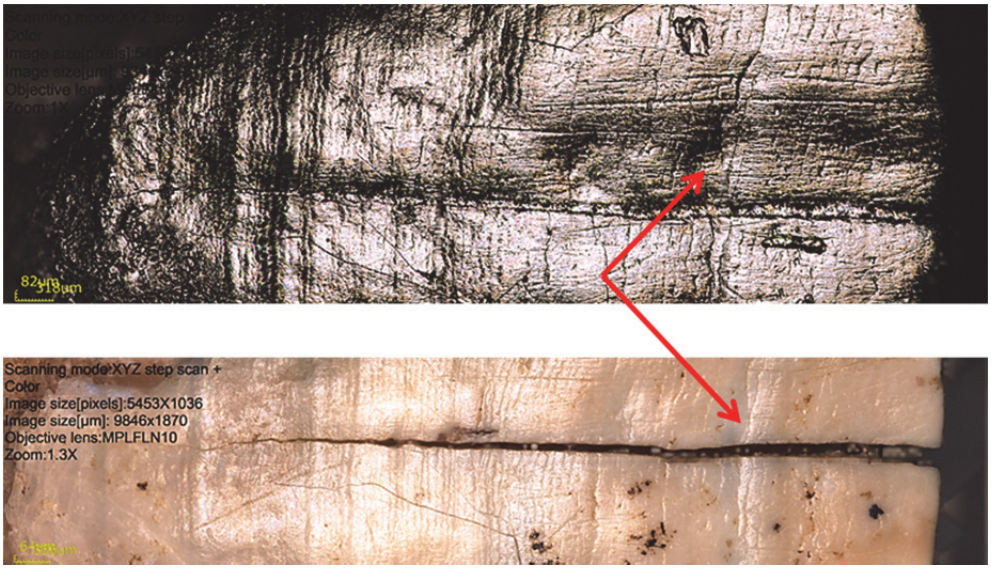
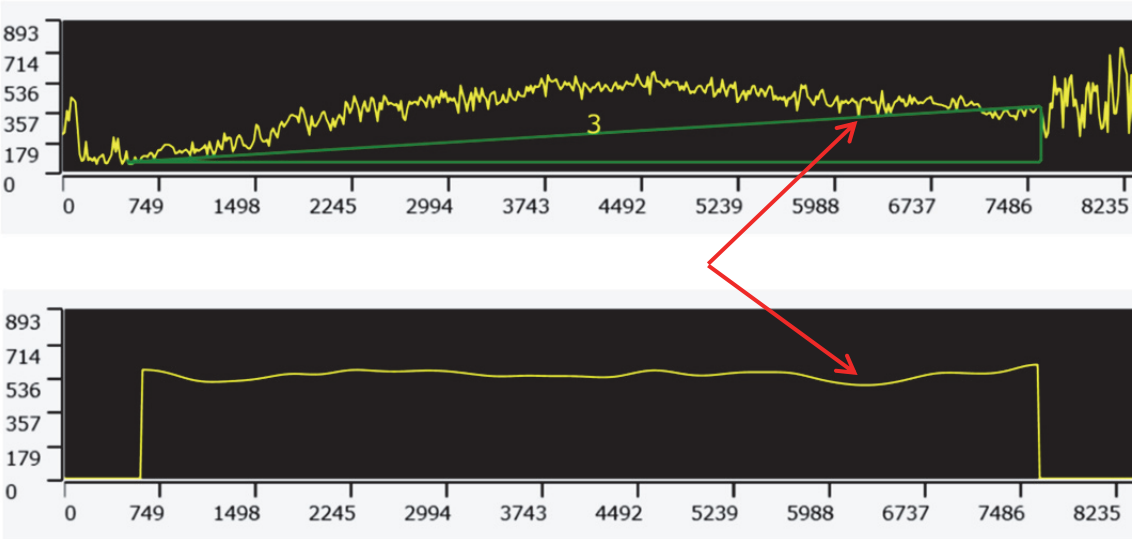


Figure 3-16 Images from the LEXT showing an uncorrected profile line (top) and a curve corrected waveform profile line (bottom) capturing the z-plane. The red arrows mark the same defect as visible in each image as well as that marked in Figure 3.11. (Sejet X72)



Raw and wave-form profile lines were generated along a longitudinal section of each tooth (Figure 3-16). These were produced in unadjusted and also in curvature corrected forms. The curvature correction made it possible to view overall changes independently of tooth curvature. The wave-form profile lines depicted simple gross curvature, and were useful aids in identifying hypoplastic events independently of any noise or perikymata. Assessment of surface defects for this thesis was done through the visual consideration of the LEXT images in association with the profile lines for added information.

In total, 123 individuals could be assessed for surface analysis (each represented by one tooth) (Table 3-6). The raw profile images may be useful in gaining a more detailed perspective of perikymata involvement and spacing, but time limitations made this beyond the scope of the current project.

Table 3-6 Number of teeth by tooth type used for the surface analysis (with each tooth representing one individual).

Tooth Type	Number of Individuals / Teeth
Mandibular Canine	111
Maxillary Canine	9
Maxillary I1	2
Maxillary I2	1

Statistical Analysis

The Microsoft Excel database was imported into IBM SPSS 20 statistical analysis software. Males and females were considered separately during this entire analysis. Scatter plots were generated to visually examine whether there was an association between age at death, stature in the grave, and each of the ten deciles (representing stress events occurring at a particular age in development) as well as overall enamel defects (both surface and internal). Pearson correlation coefficient was used to identify the direction and the strength of the correlation between these variables. Kaplan-Meier survival analysis was also conducted to consider whether the number of stress events indicated by DED's may have impacted survivorship and the likelihood of suffering from leprosy. T-tests and ANOVA were used to consider whether there was variation in stress events over time or across the two populations. Pearson's chi-square and Fisher's exact tests were used to assess population differences and changes from the early to later periods in infectious disease rates and Odds Ratios were used to assess the extent of any differences. In cases where variables were not normally distributed, a Kruskal-Wallis test was performed to test whether significant differences were found.

The diagnosis of leprosy prevalence rates was conducted according to slightly different methods, and employed theory and calculations developed by Boldsen (2005b; c, 2008, 2009), Boldsen and Freund (2006) and Boldsen and colleagues (2013). A full examination of this methodology can be reviewed in the published literature, but a brief overview is included here in the interests of clarity. Seven osteological features as described above[18] were scored for all individuals as present (1), absent (0), or missing /

[18] These being the edge of the nasal aperture, anterior nasal spine remodelling / reounding / resorption, destruction of the alveolar process, pitting or perforation of the hard palate, fibular subperiosteal exostoses,

unobservable (/). Boldsen (2001) calculated specificity and sensitivity of each of these features in relation to leprosy and in a later study demonstrated the conditional independence of these features (Boldsen, 2005c). The probability of each individual having suffered from leprosy was calculated by integrated each feature score with the specificity and sensitivity values for that feature, as defined by Boldsen (2005b). A Windows executable Delphi program for which the theory is further described in Boldsen (2008) was used for these calculations and can be found on the ADBOU website at http://adbou.dk/index.php?id=97. This generated the lambda (λ) value for each individual. This process was used for the consideration of sex differences, temporal differences, and site differences in leprosy prevalence rates.

fibular hypertrophy, and bony changes to the 5th metatarsal. All features are carefully described and established in Boldsen and Freund (2006)

Chapter 4 Results

Introduction

Over the course of this chapter, intraobserver error in AS observations will be examined and then the subsamples used in this study will be reviewed in relation to the overall cemetery samples in order to consider sample representativeness. As the selection criteria for adequate teeth may have affected the ages of individuals selected, a consideration of age distribution will be of fundamental importance. Age distribution will therefore first be considered in order to assess whether the sampled material adequately mirrors the broader samples. The implications of this for the results of this study will be briefly noted, and will be discussed more fully in the discussion chapter. A consideration of the overall subsample statistics will then be presented in order to establish the foundation for this investigation. This overview will also consider the relationship between males and females in the subsamples, as this will be critical to later statistical analysis which considers lumped versus sexually divided samples. The chapter will then move into the consideration of the relationship between childhood stress and adult health before moving into the historical analysis of health over time and between the urban and rural sites. Since the consideration of childhood stress and adult health is focused on the investigation of biological patterns, in stress and stress response, the cemetery samples are combined for this portion of the investigation.

Intraobserver Error in AS Count Trials

A key component of this thesis is the identification of accentuated striate of Retzius (AS). As the accuracy of these results is critical to a number of sections in this thesis, intraobserver error in counting the number of AS will be reviewed first before this chapter proceeds with a review of sample representativeness. Accentuated striae of Retzius were counted five times in the thin sectioned dental sample, constituting five 'trials'. A comparison of correlation between different trials shows a clear trend towards increasing correlation in the AS counts between consecutive trials. Early trials (1 and 2) show only a strong but significant ($r = 0.53$, $n = 75$, $p < 0.0005$) correlation (Figure 4-1) between each other. However, the last two trials show the strongest significant correlation ($r = 0.877$, $n = 79$, $p < 0.0005$) in between counts in the two trials (Figure 4-2). Trials were conducted over a six month period with a minimum of a week between each. This trend would seem to indicate the effect of a learning curve. The cohesion of total counts for accentuated striae in the last two trials suggests a high level of agreement over the identification of striae.

Figure 4-1 Correlation in accentuated striae of Retzius scores between the first and second trials

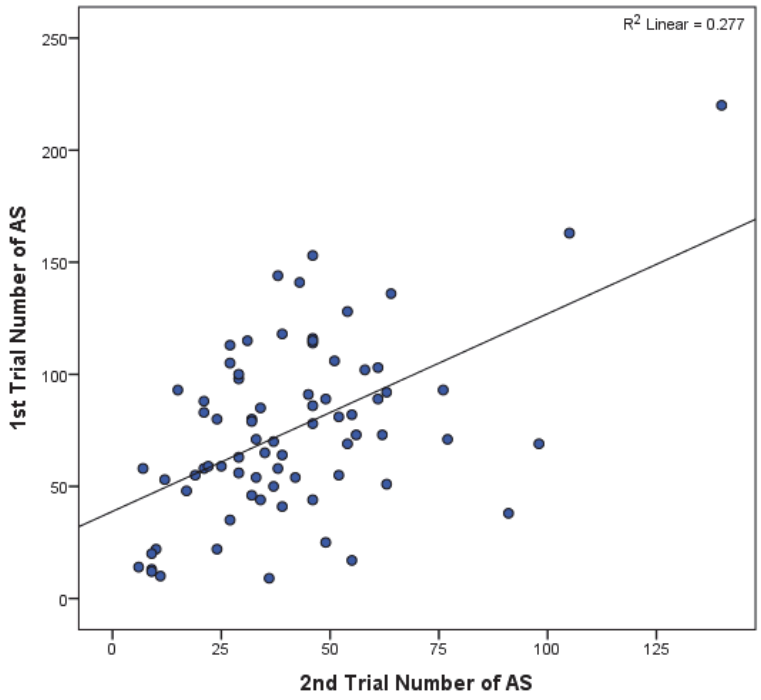
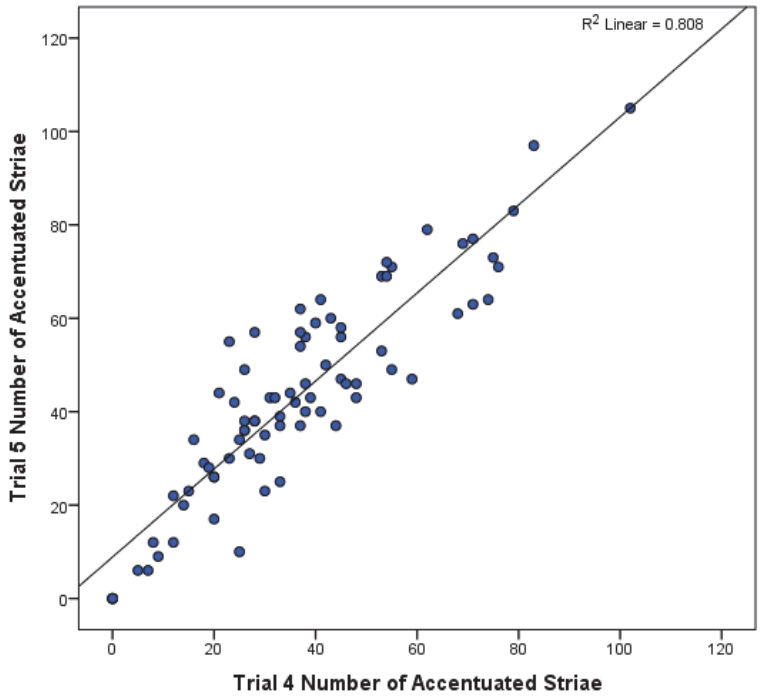
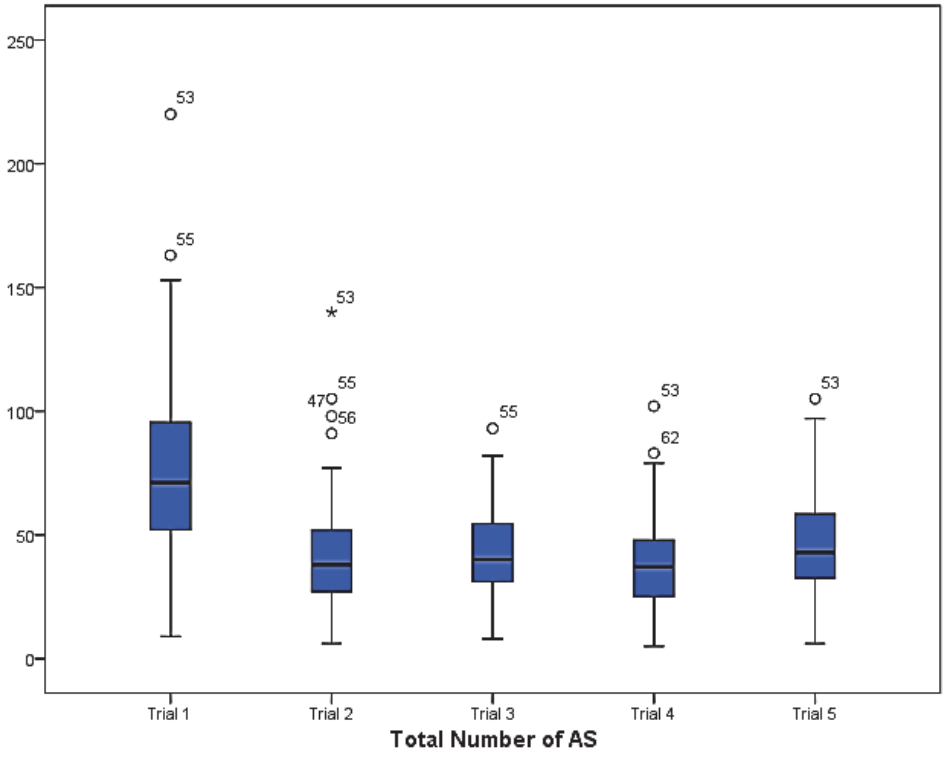


Figure 4-2 Correlation in accentuated striae of Retzius scores between the fourth and fifth trials



A consideration of the different trials showed increasing cohesion over the first three trials signifying the development of the scoring method and a learning curve pattern (Figure 4-3). The first trial was highly inclusive, as indicated by the wide range in counts. The second trial was aimed to be extremely conservative in scoring, but clearly still reflects the inclusion of more striae of Retzius as AS in the counting system. By the third trial, a levelling off is seen, and particularly the individual marked #53 can be seen to indicate greater cohesion in identification. This pattern justified the use of the fifth trial as the most accurate reflection of AS numbers.

Figure 4-3 Total accentuated striae of Retzius counts between all five trials



Different deciles exhibit differing degrees of cohesion between trials. This suggests a connection to ease of scoring in different regions of the tooth. The first decile, being right at the cusp, displays a high level of variability, but was also only scored for twenty-six individuals due to wear. The second decile was available for slightly more individuals (n =61) and was reasonably cohesive (r = 0.877) (Figure 4-4 and Figure 4-5). This correlation was also statistically significant (p = 0.001). The higher correlation may reflect the ease of scoring AS in this region, with relatively clear, thin, linear structures. However, more individuals would be required with this decile preserved to gain further insight into observer error as it might relate to this region of the crown.

Figure 4-4 Accentuated striae of Retzius counts for the second decile between all five trials

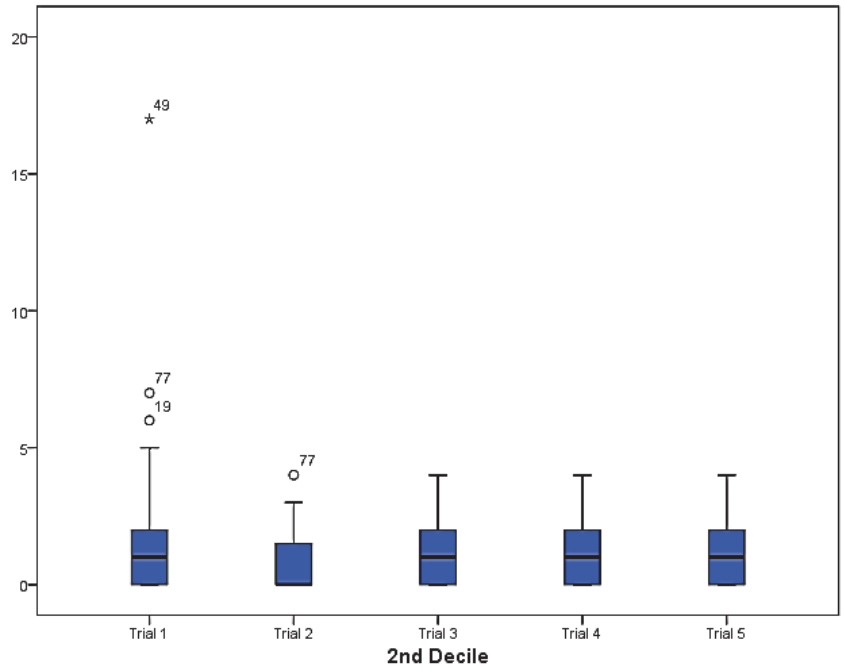
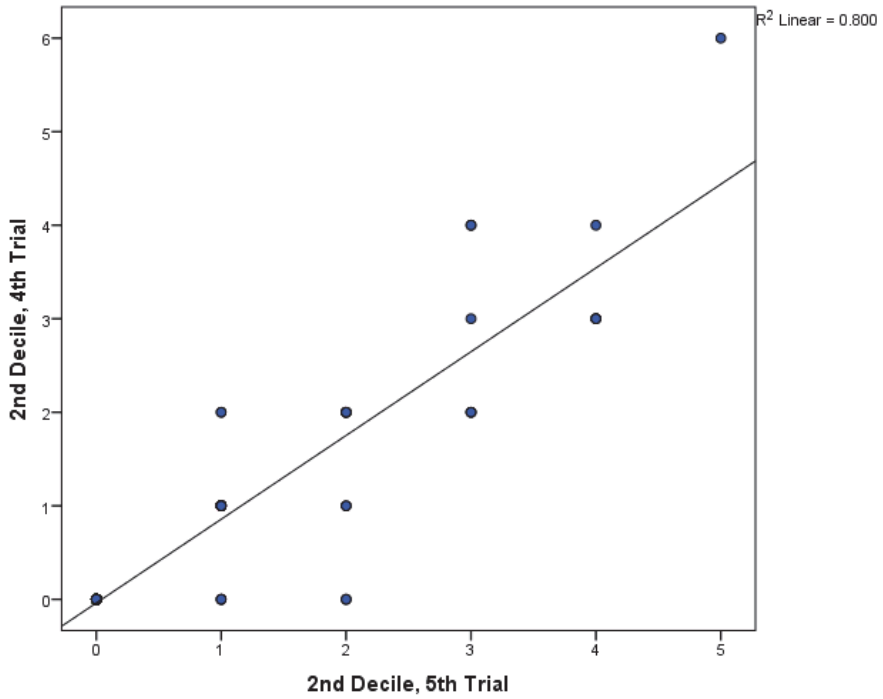


Figure 4-5 Correlation of accentuated striae of Retzius counts for the second decile between the fourth and fifth trials



The mid-crown deciles, from the 3rd to the 5th decile showed a variability in correlation between the last two trials ranging from $R^2 = 0.701$ ($r = 0.773$, $n = 75$, $p < 0.0005$) to $R^2 = 0.832$ ($r = 0.912$, $n = 76$, $p < 0.0005$) in the 5th and the 4th deciles respectively (Figure 4-6, Figure 4-7). While all correlations were statistically significant, there was no consistent patterning of increasing or decreasing correlation across these deciles. The mid-crown region can have quite clear structures, but it can also be difficult in this area to distinguish between those structures which are accentuated and those which are not. This is likely to result in a certain degree of error.

Figure 4-6 Accentuated striae of Retzius counts for the fourth decile between all five trials

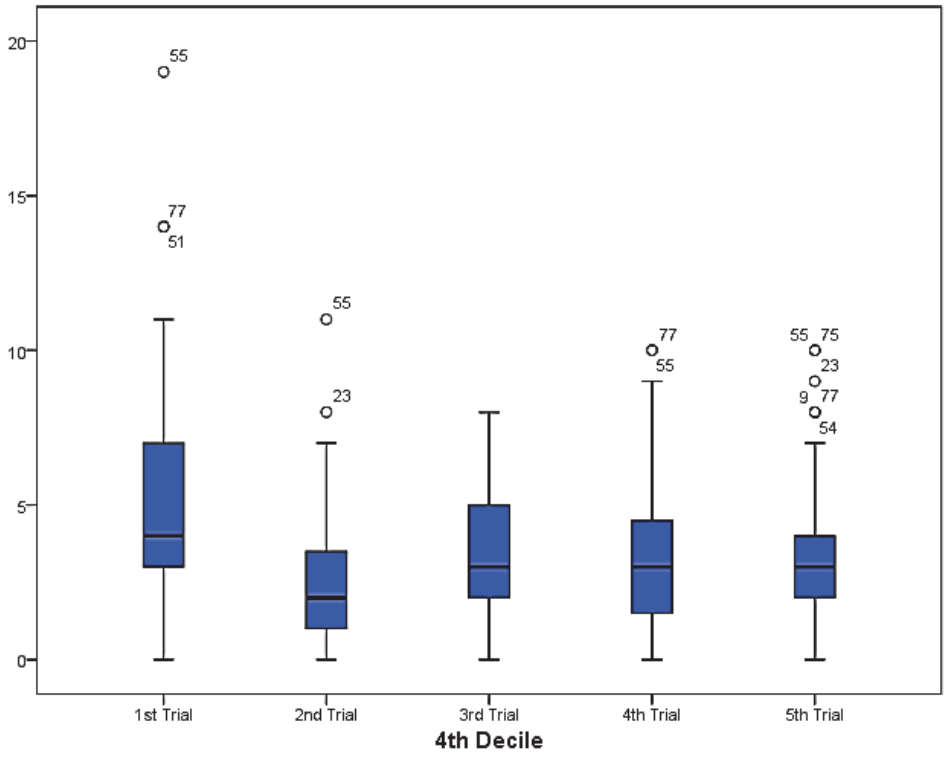
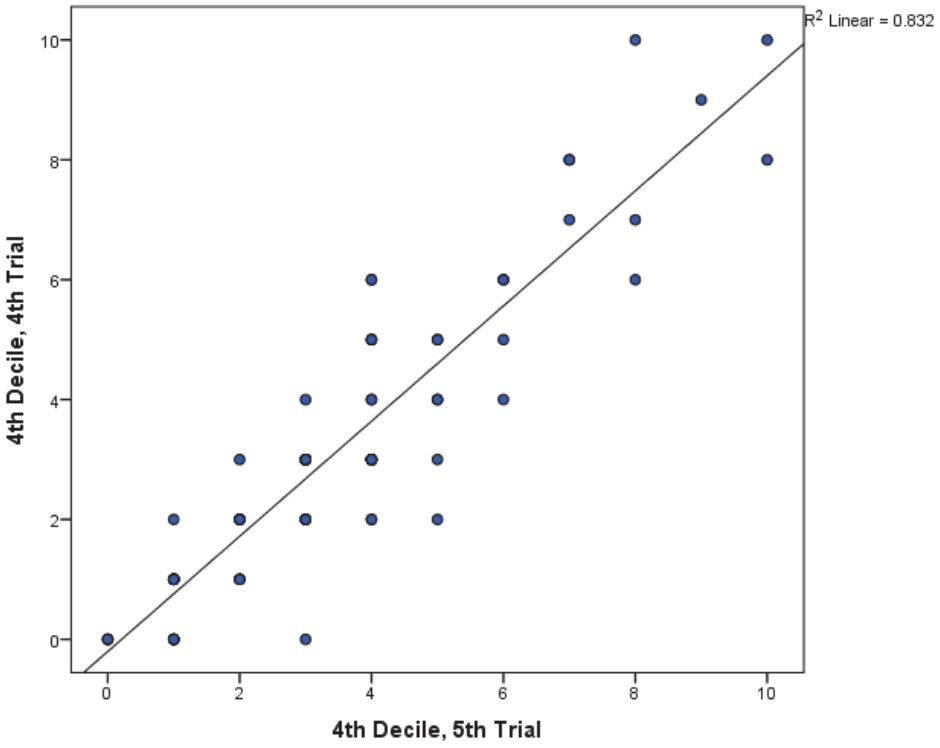


Figure 4-7 Correlation of the fourth decile accentuated striae of Retzius counts between the fourth and the fifth trials



Finally, the last three deciles showed relatively low levels of correlation from R^2 = 0.557 (r = 0.760, n =67, $p < 0.0005$) in the 9th decile to R^2 = 0.695 (r = 0.819, n =72, p < 0.0005) in the 8th decile, although these correlations were still statistically significant (p = 0.001) (Figure 4-8 and Figure 4-9). This is consistent with difficulties in scoring cervical enamel with striae of Retzius being close together and AS often faint and difficult to distinguish in relation to the normal striae.

Figure 4-8 Accentuated striae of Retzius counts for the tenth decile between all five trials

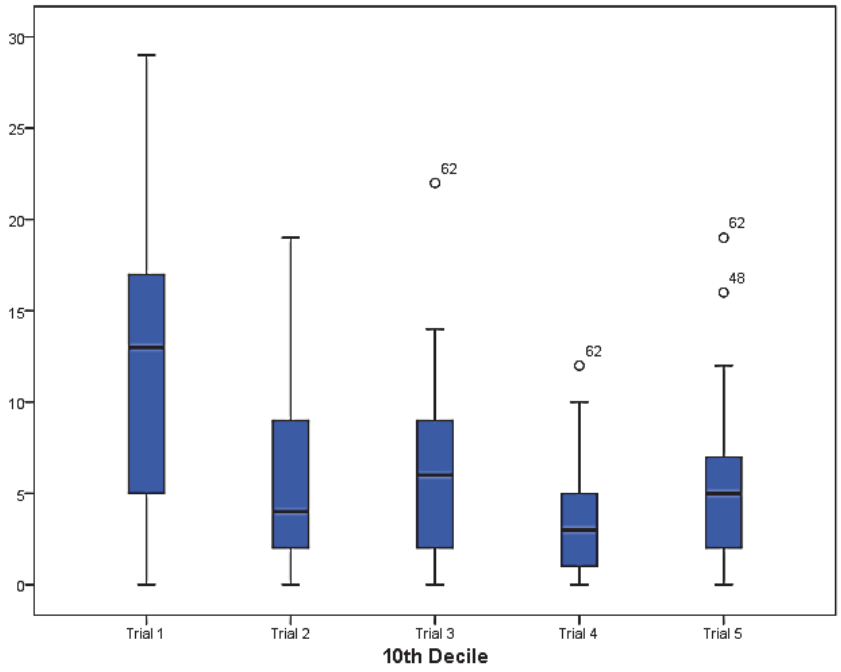
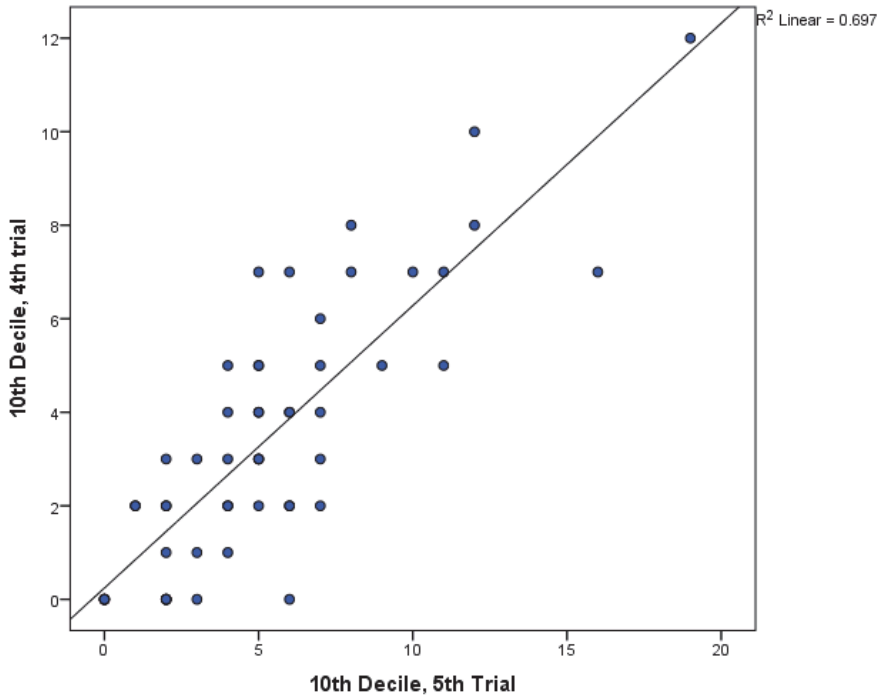


Figure 4-9 Correlation of the tenth decile accentuated striae of Retzius counts between the fourth and the fifth trials



Sample Overview

The purpose of this section is to provide a foundation upon which the main hypotheses in this investigation can be built. Broad patterns of health will be considered in terms of overall sample patterns and in terms of demographic breakdown by sex. The section will progress from considering the main patterns in age at death within the combined subsamples, and will progress through an overview of stature, stress (as indicated by accentuated striae of Retzius and dental enamel hypoplasia), and disease prevalence rates. Such a practice will provide a basis, for example, upon which a decision can be made about whether the combined sex sample should be used for statistical analysis with each parameter. In the case of a statistically significant difference being apparent between the sexes, a split analysis might be preferable. This chapter will furthermore present possible sample bias which could impact the results.

Age at Death

A total of 165 individuals were included in investigations of mortality as it related to health. Two males were not included in these analyses as their cause of death was identifiable as severe cuts to the cervical and cranial areas, likely resulting from beheading. These males were both from Sejet and were aged 38-42 and 35-42 respectively at their time of death. The mean age at death for adults in the complete cemetery samples was 37.49 (Figure 4-10). This was found to be significantly different (95% *clip*) from the mean age at death for adults in the subsample used for this study, which was 36.40 years of age (Figure 4-11)

Figure 4-10 Age distribution for the complete combined cemetery samples using the median age at death from the age range identified for each individual in this subsample of adults

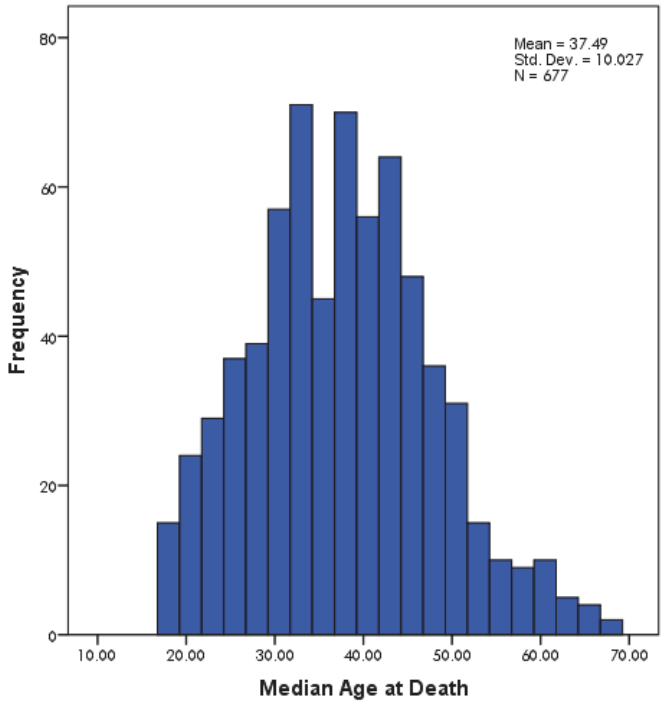
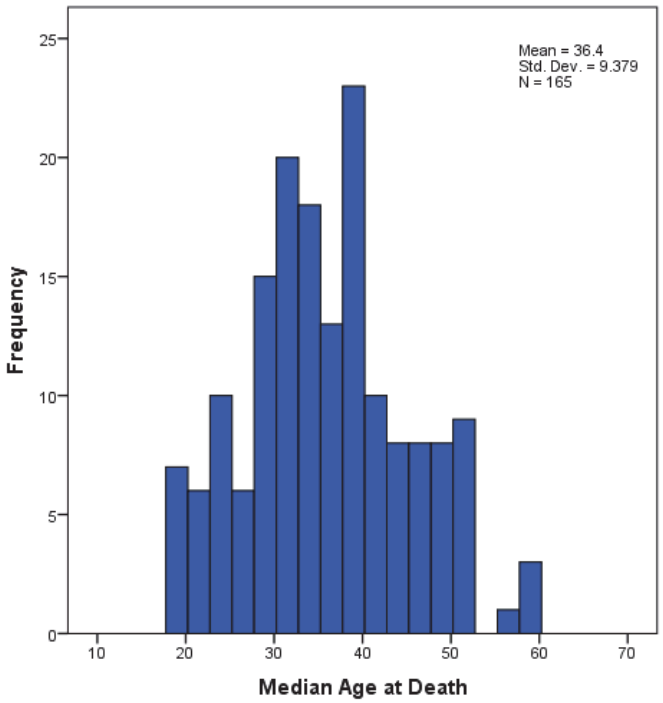
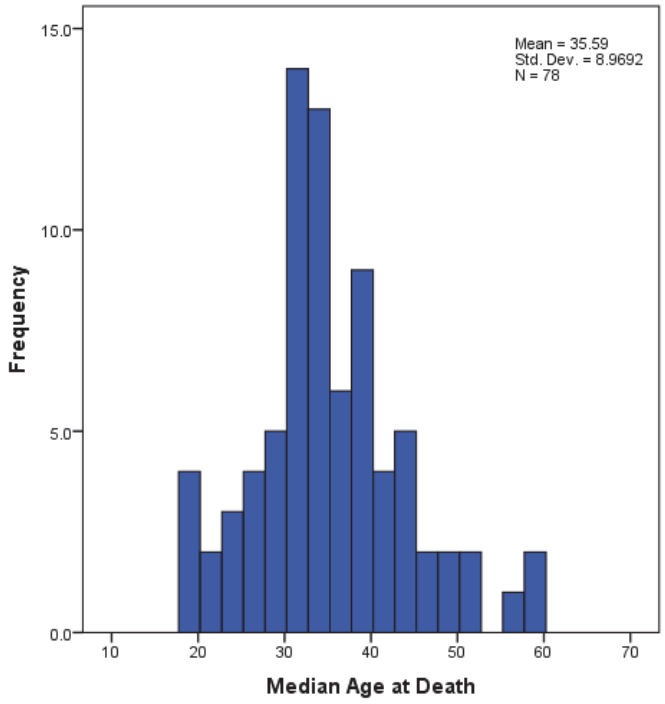


Figure 4-11 Age distribution for the combined cemetery subsamples using the median age at death from the age range identified for each individual in this subsample of adults



A further decrease in the median age at death to 35.59 year of age (Figure 4-12) is seen with the sample of 78 individuals who were selected for thin sectioning.[19] This overview suggests that there was little innate bias in the sampling process towards younger individuals with less worn teeth. Despite this, it is also clear from a consideration of the frequency distribution that the selection for AS did not further eliminate the oldest individuals from the subsample initially selected for this project, as individuals in the 50 to 60 years of age range are still included in this component of the analysis. Further consideration of the potential impacts of these any selection biases will be made in the section on site comparisons.

Figure 4-12 Age distribution for the combined cemetery thin-sectioned samples (the AS sample) using the median age at death from the age range identified for each individual in this subsample of adults



[19] While 79 individuals were available for AS counts, one of these was one of the males who was not included for male analysis due to a clear terminal trauma (also see above explanation).

Of the 165 individuals in the sample selected for this study, 84 were male and 72 were female. For nine individuals sex could not be reliably determined, though three of these are possible females and six are possible males. These individuals were not included in the sex-based analysis. Adult males had a mean age at death of 37.71 years, with the adult female mean age at death slightly lower at 35.12 years (Table 4-1). The difference in means was not statistically significant (t=1.708; df=154; p=0.090). The distribution of ages by sex can be seen in (Figure 4-13). Further testing using Kaplan-Meier survival analysis shows that while males generally trend towards surviving longer (Figure 4-14), this difference is not statistically significant ($\chi^2 = 3.056$, df = 1, $p = 0.08$).

Figure 4-13 Frequency distribution by sex of adult median age at death in the cemetery subsamples

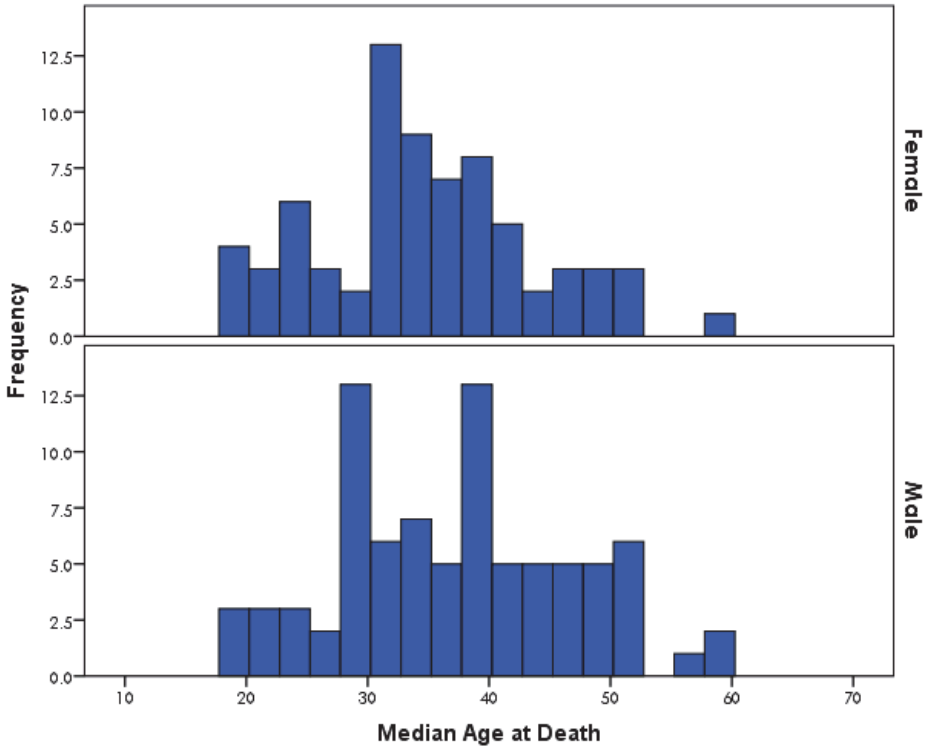
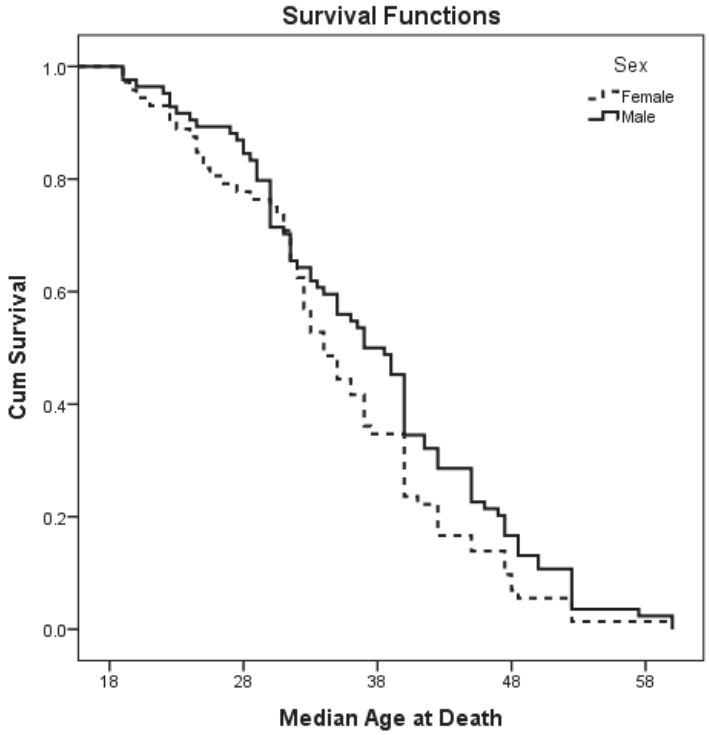


Figure 4-14 Kaplan-Meier survival curve showing the difference in median age at death between males and females for the cemetery subsamples



When the complete samples are considered using only those individuals whose sex could be identified (Figure 4-15, Table 4-1), the pattern of age seen in the subsamples is paralleled, but in this case the difference is significant (Table 4-2). It is critical to note with this comparison, however, that the statistics for all broader cemetery comparisons are drawn from the ADBOU cemetery databases, and thus the osteological data were not collected directly as part of this project. Further comparison will be conducted as part of the results section exploring site differences.

Figure 4-15 Frequency distribution by sex of adult median age at death in the complete cemetery samples

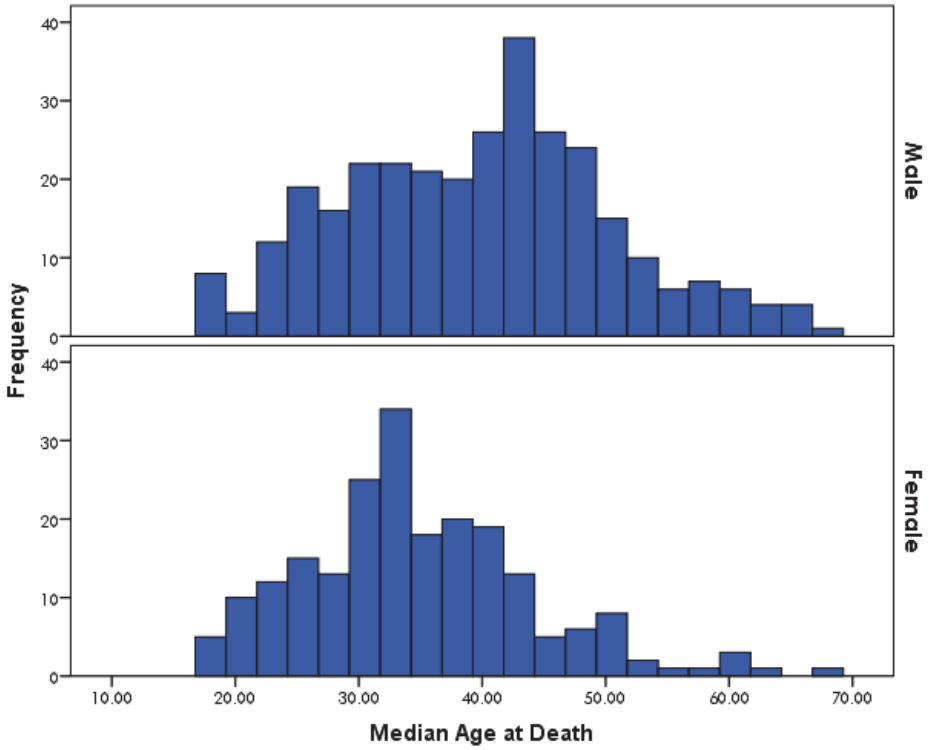


Table 4-1 Descriptive statistics for age at death by sex for the combined sites comparing the complete cemeteries with the cemetery subsamples used for this research.

	Complete Cemeteries			Cemetery Subsamples		
Sex	**N**	**Mean**	**Std. Deviation**	**N**	**Mean**	**Std. Deviation**
Male	***310***	***39.4532***	***10.59***	***84***	***37.71***	9.81
Female	***212***	***34.6769***	***9.17***	***72***	***35.12***	9.05

Table 4-2 Independent samples t-test for the difference in median age at death between sexes in the complete cemetery sample

	Levene's Test for Equality of Variances		t-test for Equality of Means						
								95% Confidence Interval of the Difference	
	F	Sig.	t	df	Sig. (2-tailed)	Mean Difference	Std. Error Difference	Lower	Upper
Equal variances not assumed	*7.773*	*0.005*	*5.486*	*492.04*	*.000*	*4.776*	*.871*	*3.066*	*6.487*

Stature

Femur length was available for 83 adults in this sample. This made it the most commonly available indicator of stature with stature in the grave being the next most common measurement (n =77). The decision was made to use femur length as a proxy for stature alongside stature in the grave measurements in order to maximize sample sizes. Population-specific regression formulae were developed in relation to stature in the grave measurements in the hopes of being able to consider stature from the long bones. However, largely due to smaller sample sizes these formulae showed high standard errors and so were not used for this investigation. Another point of interest was how height as predicted from long bone length (predicted height) might compare to stature as measured in the grave (actual height). Unfortunately, due to the relatively small sample sizes, the development of population-specific regression formulae involved all individuals in the sample, making the subsequent use of the derived stature estimates in relation to stature in the grave (to better understand residual height) unacceptably circular. As such, use regression formulae from closely related samples would have been preferable for this analysis, and so regression formulae developed for Tirup were applied, as has been done

previously for the larger Sejet anthropological report (Pedersen and Boldsen, 2008). Correlation was moderate to high between predicted and actual heights both in the complete subsample ($r = 0.82$, $n = 70$, $p < 0.0005$) (Figure 4-16) and for each sex ($r = 0.55$, $n = 32$, $p = 0.001$ for females and $r = 0.42$, $n = 34$, $p = 0.014$ for males) (Figure 4-17), but the patterns revealed through this approach did not provide more insight than that gained through the use of strict femoral length or stature as measured in the grave. This was particularly apparent for temporal and site comparisons, in which the late period and Ole Wormsgade respectively had severely reduced sample sizes. It is consequently not extensively reported in this thesis.

Figure 4-16 Correlation between actual stature (as measured from stature in the grave) and predicted stature (being height predicted from femoral length)

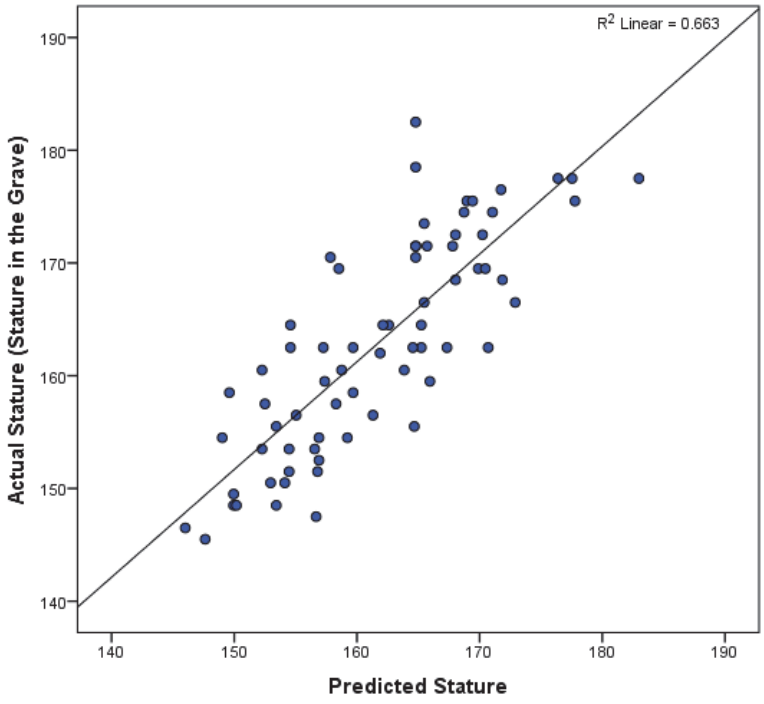
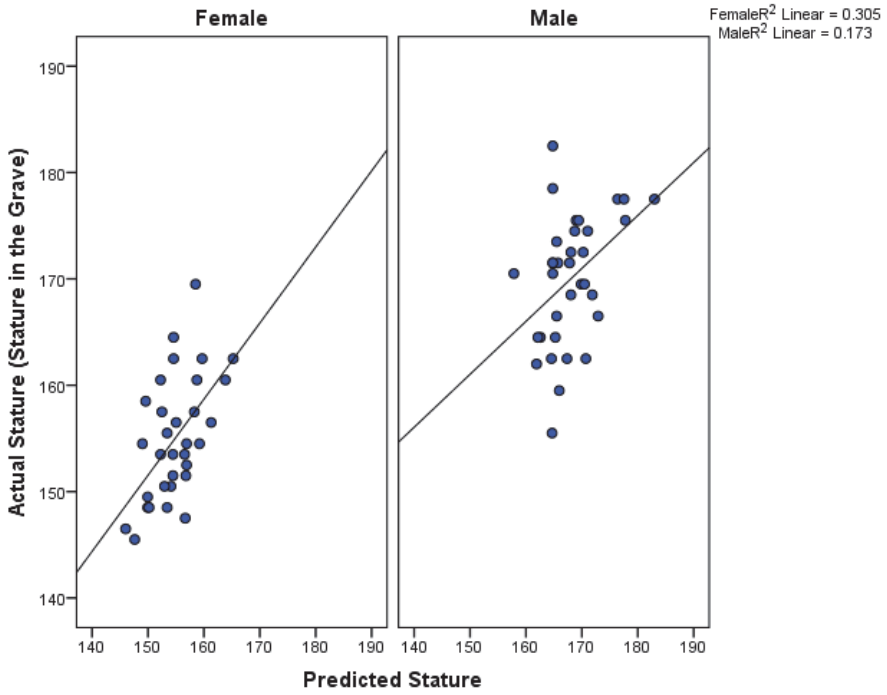


Figure 4-17 Correlation between actual stature (as measured from stature in the grave) and predicted stature (being height predicted from femoral length) in females and males.



Consideration of the difference in femur length between the complete sample available for this study and the broader cemetery samples using an Independent samples t-test (95% *c.i.*) revealed no significant differences. The frequency distributions for the respective samples also appear to be consistent with each other (Figure 4-18, Figure 4-19). Femur length as captured by the study sample therefore appears to be an unbiased representation of overall cemetery distribution.

Figure 4-18 Frequency distribution of average femur length for the complete combined cemetery sample

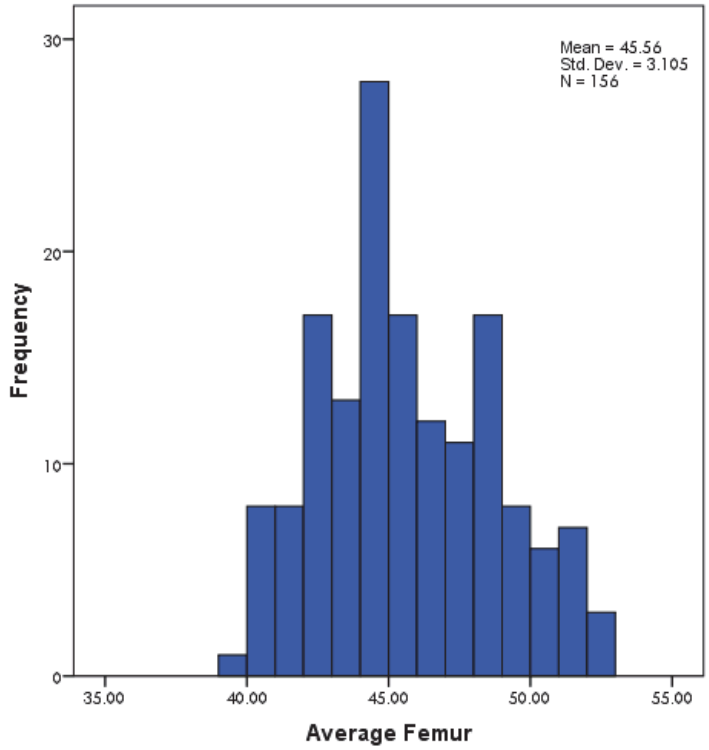
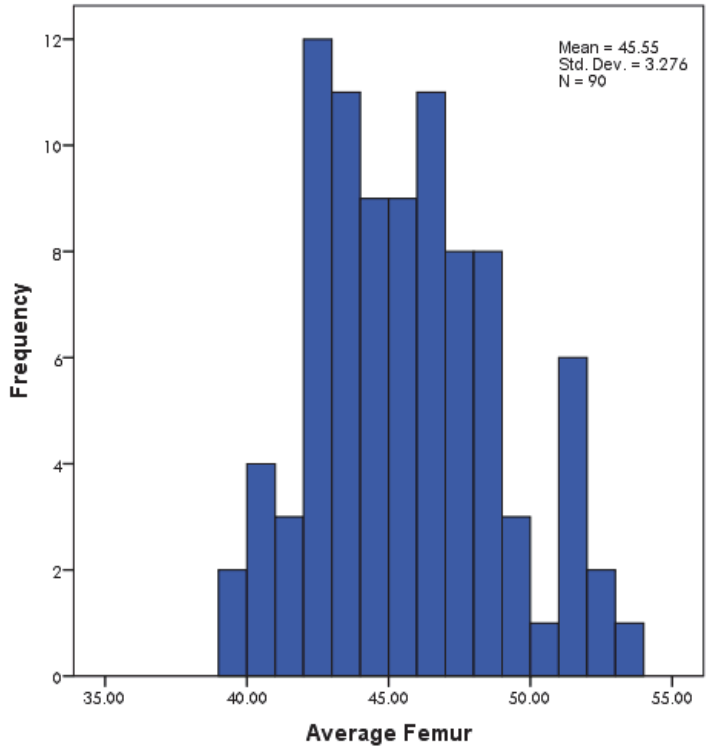


Figure 4-19 Frequency distribution of average femur length for the combined cemetery sample subsamples



Stature in the grave was a mean of 170.16 cm (n =37, sd = 6.86) for males and a mean of 155.48 (n =40, sd = 6.49) for females (Figure 4-20). Males had a mean femur length of 47.95 cm (n =44, sd = 2.42), with the female femur length slightly lower at 43.12 cm (n =39, sd = 1.96) (Figure 4-21). These sex differences were, unsurprisingly, found to be statistically significant for stature as measured in the grave ($t = 9.653$, $df = 75$, $p = 0.0001$) and for femur length ($t = 9.896$, $df = 81$, $p = 0.0001$).

Figure 4-20 Frequency distribution by sex of stature as measured in the grave for the combined cemetery subsamples used in this study

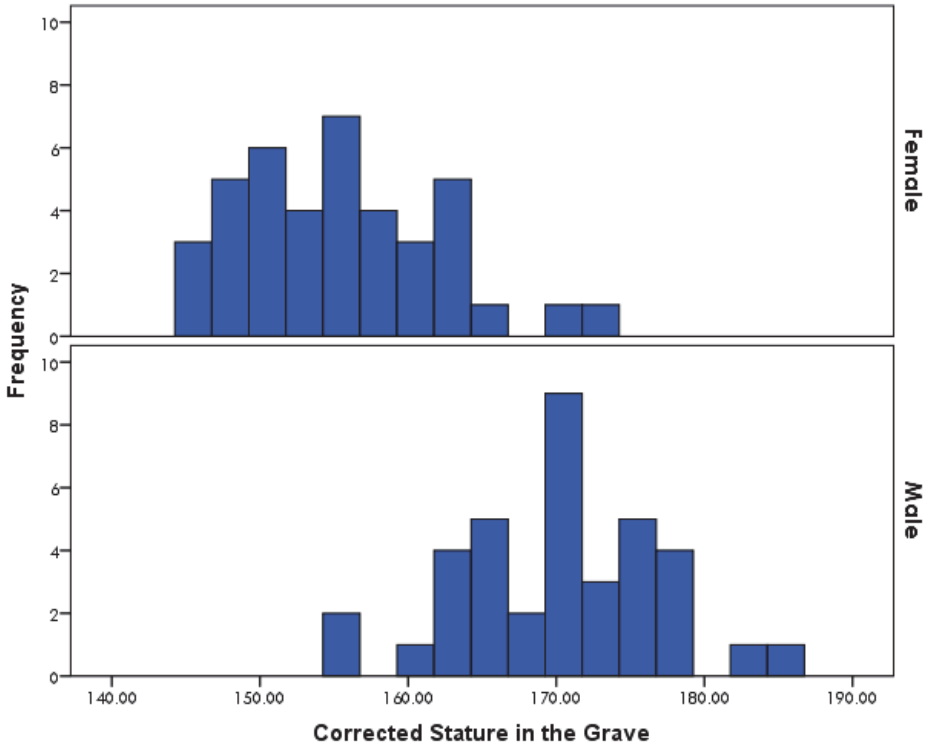
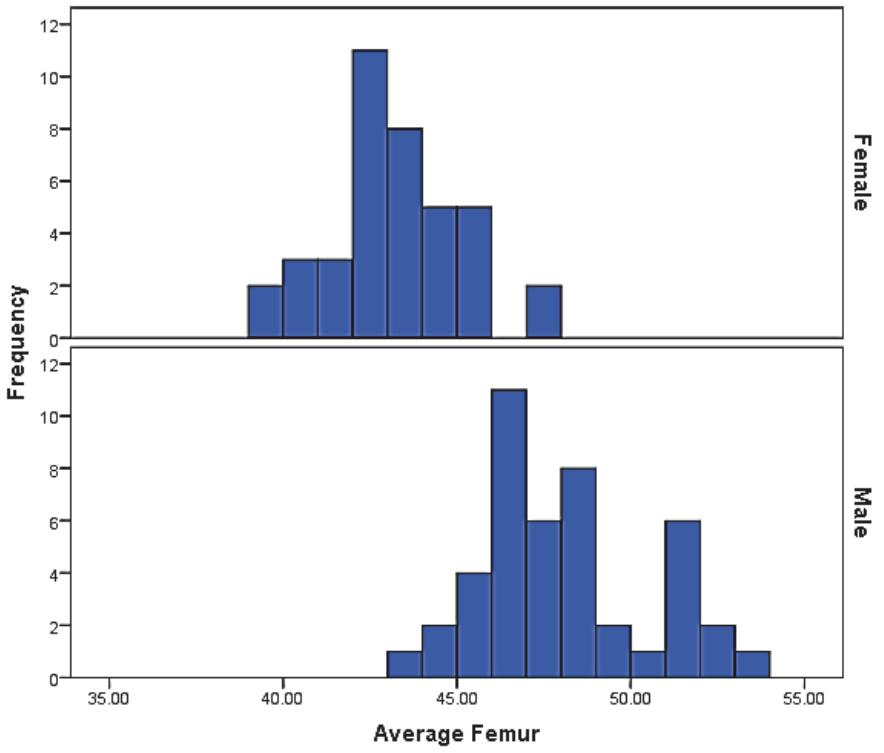


Figure 4-21 Frequency distribution by sex of femur length for the combined cemetery subsamples used in this study



Overall Stress Patterns

Levels of stress could not be compared between the broader cemetery samples and the sample used in this study, as neither accentuated striae of Retzius nor surface hypoplasia could be recorded microscopically for the broader sample. Any information on macroscopic hypoplasia available from the cemetery records is considered in the discussion section, but is not compatible enough to determine whether the sampling for this investigation was representative.

A subsample of 79 individuals was thin sectioned to identify accentuated striae of Retzius (AS) in the dentition. This consisted of 37 males and 39 females and three individuals whose sex could not be identified. As noted above, one male was not used for the age analysis as injuries clearly indicated traumatic death. While the mean age at death for this subsample was lower than for the larger (complete) cemetery samples, it is also clear from the frequency distribution (Figure 4-15) that older individuals were captured in this sample. As such, despite selection for sufficiently preserved enamel for thin sectioning, this sample does seem to have captured at least some individuals who might, due to their age, be expected to get selected out of this analysis. Overall the sample had a mean of 43.66 AS (sd=22.01), with males having a significantly higher mean number of AS ($\bar{x}$ = 50.30 AS) than females ($\bar{x}$ = 37.59 AS) (Table 4-3, Table 4-4, Figure 4-23). While there were males with as few AS as females, more males seem to have experienced a higher number of stress incidents than females (Figure 4-22). This difference exhibits a medium (r = 0.29 to strong d = 0.59) effect size.

Figure 4-22 Frequency Distribution by Sex of Accentuated Striae of Retzius

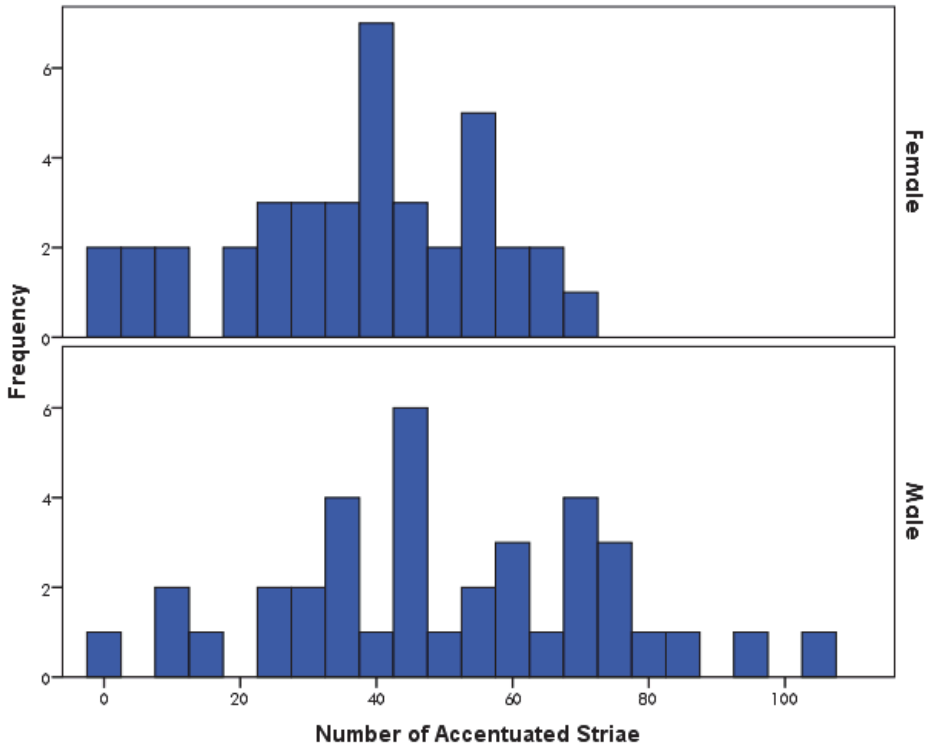


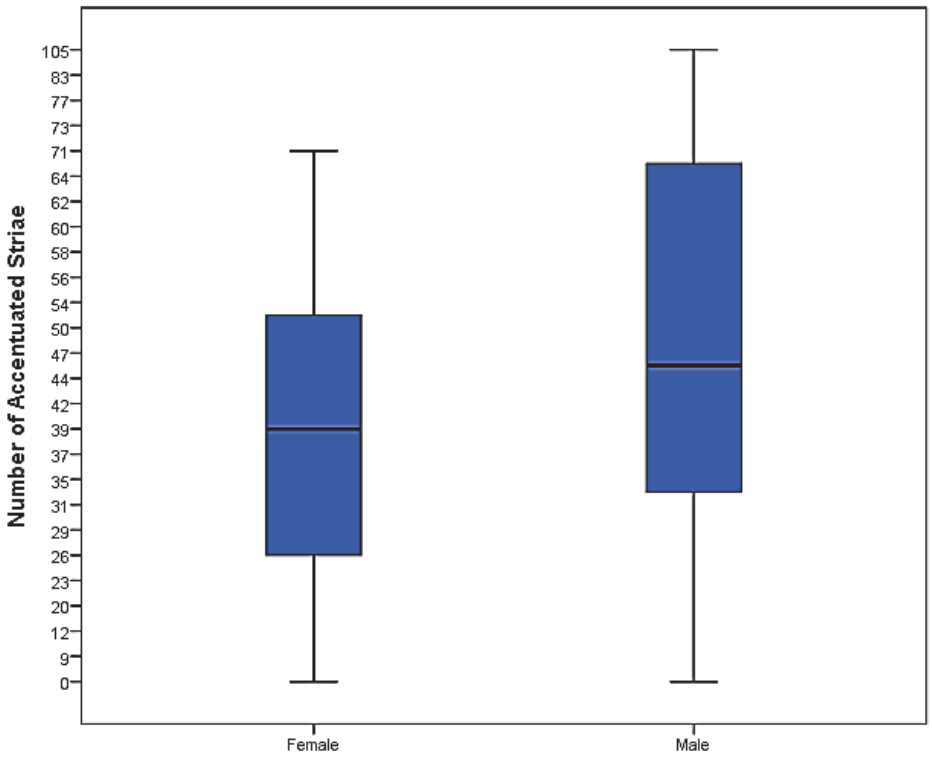
Table 4-3 Group statistics for accentuated striae of Retzius in males and females

	Sex	N	Mean	Std. Deviation	Std. Error Mean
Number of Accentuated Striae	Male	*37*	*50.30*	*24.469*	*4.023*
	Female	*39*	*37.59*	*18.572*	*2.974*

Table 4-4 Independent samples t-test for the difference in number of accentuated striae of Retzius between sexes

	Levene's Test for Equality of Variances		t-test for Equality of Means						
								95% Confidence Interval of the Difference	
	F	Sig.	t	df	Sig. (2-tailed)	Mean Difference	Std. Error Difference	Lower	Upper
Equal variances assumed	*3.287*	*.074*	*2.558*	*74*	*.013*	*12.71*	*4.967*	*2.811*	*22.604*

Figure 4-23 Sex differences in accentuated striae of Retzius



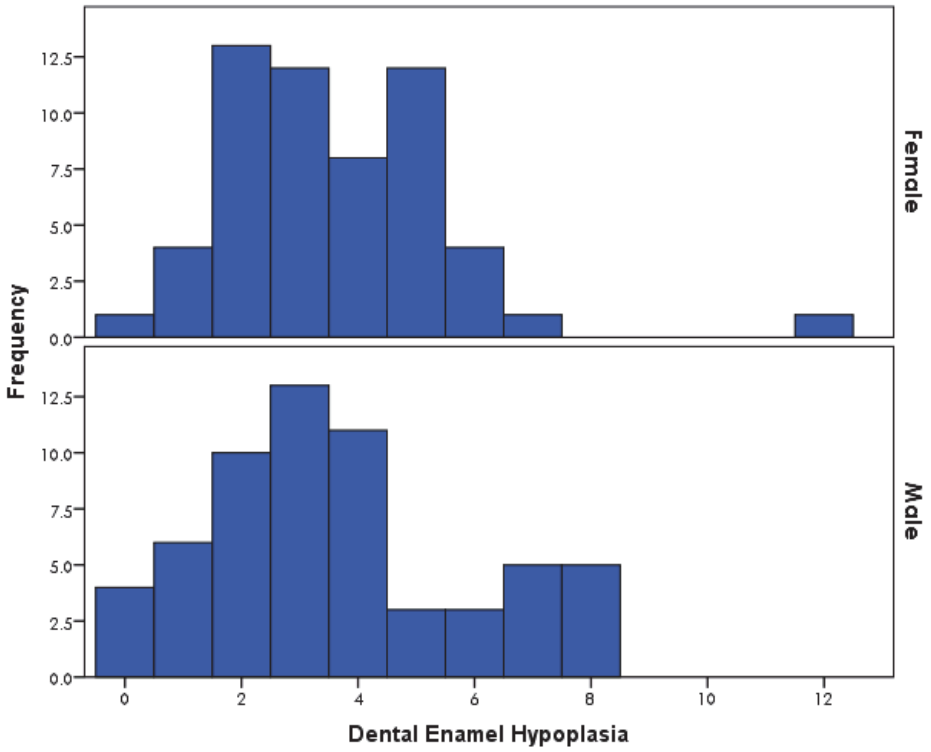
A breakdown by decile illustrates patterns of wear and visibility of enamel microstructure across the crown, with scoring being possible in fewer individuals for the first and tenth deciles (Table 4-5). This pattern is most pronounced for the first decile near the cusp, with only 26 individuals being scorable. When corrected for average period of growth encompassed within each decile, as per Reid and Dean's (2006) specifications, it is clear that the rate of AS per decile gradually increases across the crown, peaking in the mid-crown region. The peak occurs in the 5th decile, from approximately 2.7 to 3.1 years of age, with an average of 15.47 AS occurring across 75 individuals. This may be due to a real increase in AS during this period in the sampled population. Alternatively, it may partly reflect higher visibility of defects in this region of the crown.

Table 4-5 Descriptive statistics for AS in each decile, depicting raw counts with number of individuals with preserved material for each decile and the mean rate of defects based on correction for average decile duration (as based on Reid and Dean 2006).

Variable	N	Mean	Std. Deviation	Variable	Mean	Std. Deviation
1st Decile	*26*	*0.88*	*0.816*	1st Decile Rate	*5.19*	*5.74*
2nd Decile	*61*	*1.2*	*1.327*	2nd Decile Rate	*3.99*	*4.5*
3rd Decile	*74*	*2.2*	*1.923*	3rd Decile Rate	*7.39*	*6.42*
4th Decile	*76*	*3.55*	*2.306*	4th Decile Rate	*9.12*	*6*
5th Decile	*75*	*6.04*	*3.121*	5th Decile Rate	*15.47*	*8.153*
6th Decile	*76*	*6.96*	*4.107*	6th Decile Rate	*14.06*	*8.27*
7th Decile	*76*	*7.61*	*4.243*	7th Decile Rate	*13.03*	*7.59*
8th Decile	*72*	*8.29*	*5.014*	8th Decile Rate	*12.18*	*7.4*
9th Decile	*67*	*6.99*	*5.23*	9th Decile Rate	*10.18*	*7.65*
10th Decile	*56*	*5.27*	*3.85*	10th Decile Rate	*8.86*	*6.5*

A subsample of 123 individuals could be scored for DEH using the Olympus LEXT 3D Laser Measuring Microscope. This sample size was reduced from the total number of individuals used in this study due to surface wear and calculus obliterating surface structures. This consisted of 60 males and 56 females and 7 individuals who could not be sexed. Overall the sample had a mean of 3.65 defects (sd=2.072), with no significant difference in the mean number of hypoplastic defects between males and females. Overall, more males once again had higher defect counts, but there was one female outlier who had the most defects in the sample at twelve defects (Figure 4-24).

Figure 4-24 Frequency distribution by sex of dental enamel hypoplasia



Infectious Disease

A number of infectious diseases were identified in this sample, and these were consistent with those identified more broadly from the medieval period in Denmark. The main diseases which were apparent were leprosy, treponematosis, and tuberculosis. Other non-specific indicators of infection were also visible but were not analysed further. Tuberculosis and treponematosis were diagnosed based on an assessment of visible lesions and lesion distribution, while leprosy diagnosis made use of the Lambda program developed at ADBOU (Boldsen and Freund, 2006; Boldsen, 2008). As a result of the different scoring systems used, the rates for leprosy in these results are not comparable in any way to those of the other diseases. A cross-comparison of relative disease frequencies in these populations is therefore impossible, although it is possible to gain some independent insight into patterns of each disease (restricted as this was by the

limitations implicit in skeletal remains) (Ortner, 1991). This section will engage in an overview of disease prevalence rates in the subsamples used here and will furthermore break this analysis down by sex to establish baselines for this investigation.

Identifying potential bias in this subsample was also problematic. While some cross comparison of disease with the broader cemetery samples is examined in the discussion, the rates of pathology were recorded differently in this study than in the broader cemetery samples, having been recorded by different researchers and for different purposes. The examination of patterns in the discussion only involves broad observations. Since more detailed direct comparisons were not possible, a more detailed examination of bias is not engaged in during this section. A few broad observations are pertinent, however. The first is that these samples were not selected for pathology, but rather for adequate preservation. Preservation is the most likely factor to introduce bias into this component of the investigation. This is discussed in relation to leprosy, in particular, for which scores may be impacted by the lack of postcranial preservation at Ole Wormsgade (particularly with individuals in this subsample being truncated at the legs). Furthermore, selection for adequately preserved dentition may be the factor which significantly reduced mean age at death for Sejet in this subsample in relation to the overall cemetery sample. If the Sejet sample is biased towards younger individuals, diseases which factor out young individuals in the population (such as tuberculosis, which impacts individuals most commonly in the first three decades of life) may be slightly over-represented in this subsample. However, this might be expected to be counteracted to a certain extend by the exclusion of the younger age group (individuals who were under 18 years of age at the time of death) from the sampling process.

Tuberculosis

Lesions suggestive of tuberculosis were commonly found in this sample. Differential diagnosis was found to be complicated by issues of preservation and includes possible alternatives such as osteoarthritic changes, brucellosis, and traumatic anterior disk herniation. There were examples with clear and pronounced lytic changes to the vertebral bodies. In some cases, other joint surfaces were also affected, leading to a more positive diagnosis of tuberculosis. A slightly higher percentage of females were diagnosed with tuberculosis than males (Table 4-6, Figure 4-25). An odds ratio (0.714) showed that females were very slightly more likely to be diagnosed with probable tuberculosis[20] (Table 4-7) but this was not statistically significant using a chi-square test (Table 4-8).

Table 4-6 Tuberculosis in the complete subsample used in this study and by sex. These numbers are based on only those individuals who could be scored for tuberculosis.

		Number	Percent
Total Sample	**No TB**	***38***	***22.6***
	Possible TB	***42***	***25.0***
	Probable TB	***11***	***6.5***
Females	**No TB**	***16***	***22.2***
	Possible TB	***16***	***22.2***
	Probable TB	***6***	***8.3***
Males	**No TB**	***21***	***24.4***
	Possible TB	***22***	***25.6***
	Probable TB	***5***	***5.8***

[20] Once again, a case was determined as probable if the characteristic indicators were visible. A possible diagnosis was given in cases where lesions existed that were consistent with the disease but where a higher level of uncertainty existed due to missing or poorly preserved elements. No disease refers to cases with sufficient preservation of elements but with elements exhibiting no evidence for a diseased state.

Figure 4-25 Sex distribution of tuberculosis

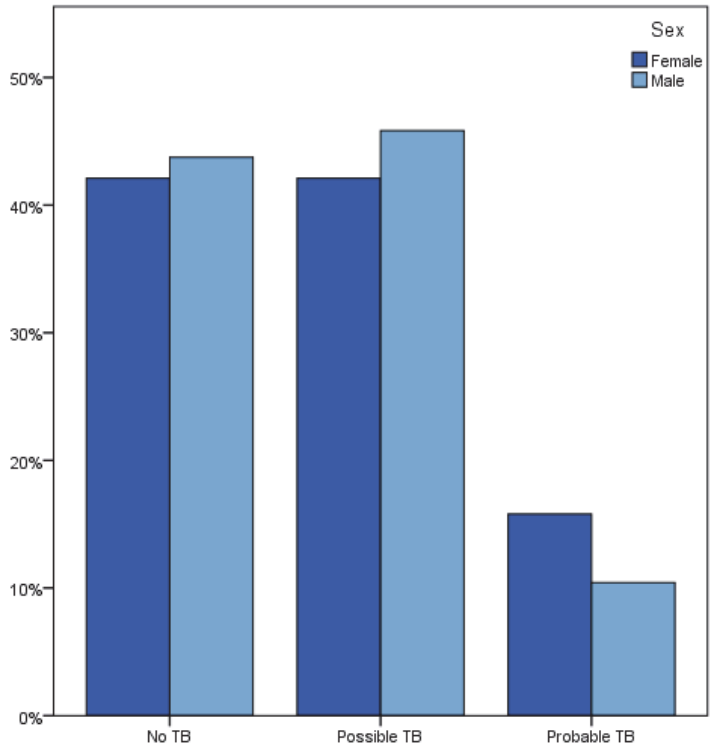


Table 4-7 Odds ratio reflecting rates of tuberculosis between the sexes

	Value	95% Confidence Interval	
		Lower	Upper
Odds Ratio for NewTB_OddsRatio (No Definite TB / Probable TB)	***.714***	***.210***	***2.431***
For cohort Sex = Female	***.833***	***.445***	***1.562***
For cohort Sex = Male	***1.167***	***.641***	***2.123***
N of Valid Cases	***84***		

Table 4-8 Chi-Square test reflecting the tuberculosis rates between the sexes

	Value	df	Asymp. Sig. (2-sided)	Exact Sig. (2-sided)	Exact Sig. (1-sided)
Pearson Chi-Square	*.292*[a]	*1*	*.589*		
Continuity Correction[b]	*.051*	*1*	*.822*		
Likelihood Ratio	*.289*	*1*	*.591*		
Fisher's Exact Test				*.754*	*.407*
Linear-by-Linear Association	*.288*	*1*	*.591*		
N of Valid Cases	*84*				

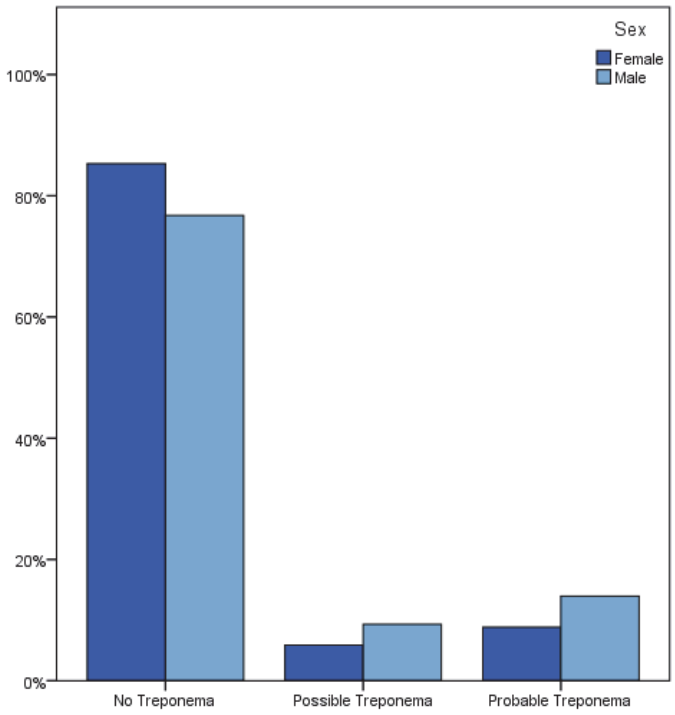
Treponematosis

Treponemal infection was apparent in this sample through diffuse lesions affecting both the cranium and the postcranium. Visible changes occurred in the form of periosteal thickening and erosive lesions. Males were more likely to be diagnosed with probable treponemal changes than females (odds ratio = 1.128) (Table 4-9, Table 4-10, Figure 4-26), but this was not found to be statistically significant using a chi-square test (Table 4-11).

Table 4-9 Treponematosis in the complete subsample used in this study and by sex. These numbers are based on only those individuals who could be scored for treponematosis.[21]

		Frequency	Percent
Total Sample	No Treponema	67	39.9
	Possible Treponema	6	3.6
	Probable Treponema	11	6.5
Females	No Treponema	29	40.3
	Possible Treponema	2	2.8
	Probable Treponema	3	4.2
Males	No Treponema	33	38.4
	Possible Treponema	4	4.7
	Probable Treponema	6	7.0

Figure 4-26 Sex distribution of treponematosis



[21] Once again, a case was determined as probable if the characteristic indicators were visible. A possible diagnosis was given in cases where lesions existed that were consistent with the disease but where a higher level of uncertainty existed due to missing or poorly preserved elements. No disease refers to cases with sufficient preservation of elements but with elements exhibiting no evidence for a diseased state.

Table 4-10 Odds ratio reflecting rates of treponematosis between the sexes

	Value	95% Confidence Interval	
		Lower	Upper
Odds Ratio for NewTrep_OddsRatio (No Definite Treponema / Probable Treponema)	*1.128*	*.235*	*5.407*
For cohort Sex = Female	*1.069*	*.439*	*2.608*
For cohort Sex = Male	*.948*	*.482*	*1.863*
N of Valid Cases	*79*		

Table 4-11 Chi-Square test reflecting the treponematosis rates between the sexes

	Value	df	Asymp. Sig. (2-sided)	Exact Sig. (2-sided)	Exact Sig. (1-sided)
Pearson Chi-Square	*.023*[a]	*1*	*.880*		
Continuity Correction[b]	*.000*	*1*	*1.000*		
Likelihood Ratio	*.023*	*1*	*.880*		
Fisher's Exact Test				*1.000*	*.600*
Linear-by-Linear Association	*.022*	*1*	*.881*		
N of Valid Cases	*79*				

Leprosy

Leprosy has been demonstrated to be a common disease in medieval Denmark (Boldsen, 2005b, 2009; Boldsen and Mollerup, 2006). The λ program is designed to estimate leprosy prevalence in a population based on seven lesions (Table 4-12) which are clearly distinguishable and which have been linked with leprosy (with their specificity, sensitivity and conditional independence tested) (Boldsen, 2001, 2005b; c, 2008; Boldsen and Freund, 2006). It also permits the statistical inclusion of missing features and so takes account of fragmentary remains.

Table 4-12 Scoring for osteological changes used for lambda scores, cumulative for Sejet and Ole Wormsgade.

	Unobservable	Positive	Negative
Nasal Edge	98	12	57
Nasal Spine	104	5	58
Alveolar Process	78	10	79
Palate	52	70	45
Fibular Subperiosteal Exostoses	107	34	26
Hypertrophy of the Fibula	121	16	30
5th Metatarsal	146	15	6

Following from Boldsen (2008), individuals who were unlikely to have leprosy were those who scored $\lambda = -1$; those who possibly had leprosy were those with λ scores of between -1 and 1; those who likely had leprosy had λ of ≥ 1. A consideration of these raw leprosy scores showed higher levels of possible leprosy in females, but higher levels of probable leprosy in males (Table 4-13, Figure 4-27). The odds ratio shows that probable cases of leprosy were diagnosed almost twice as often in females than in males (odds ratio = 1.972) (Table 4-14), but this was not statistically significant (Table 4-15).

Table 4-13 Leprosy in the complete subsample used in this study and by sex. These numbers are based on only those individuals who could be scored for leprosy

		Frequency	Percent
Total Sample	**No Leprosy**	***60***	***43.5***
	Possible Leprosy	***34***	***24.6***
	Probable Leprosy	***44***	***31.9***
Females	**No Leprosy**	***26***	***44.1***
	Possible Leprosy	***19***	***32.2***
	Probable Leprosy	***14***	***23.7***
Males	**No Leprosy**	***33***	***46.5***
	Possible Leprosy	***11***	***15.5***
	Probable Leprosy	***27***	***38.0***

Figure 4-27 Sex distribution of leprosy

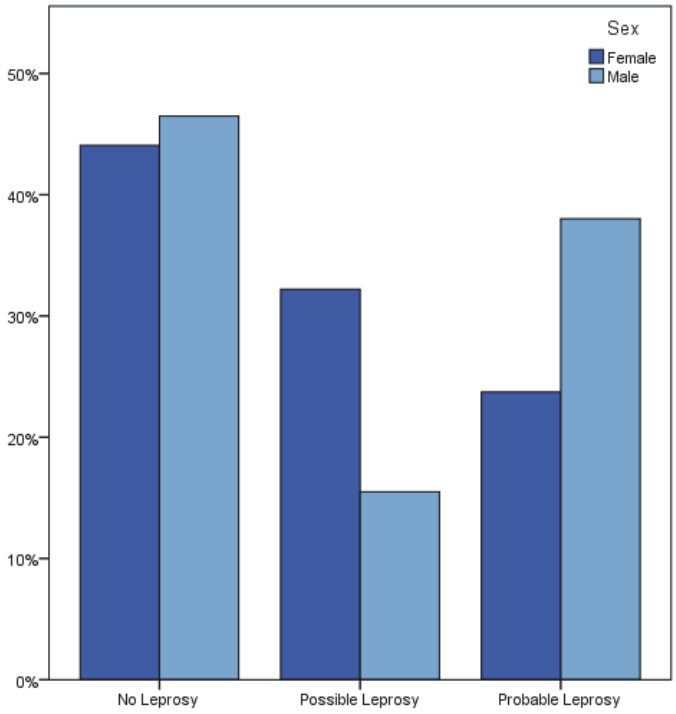


Table 4-14 Odds ratio reflecting rates of leprosy between the sexes

	Value	95% Confidence Interval	
		Lower	Upper
Odds Ratio for NewLep_OddsRatio (No Definite Leprosy / Probable Leprosy)	*1.972*	*.915*	*4.250*
For cohort Sex = Female	*1.481*	*.924*	*2.374*
For cohort Sex = Male	*.751*	*.554*	*1.018*
N of Valid Cases	*130*		

Table 4-15 Chi-Square test reflecting the leprosy rates between the sexes

	Value	df	Asymp. Sig. (2-sided)	Exact Sig. (2-sided)	Exact Sig. (1-sided)
Pearson Chi-Square	*3.051*[a]	*1*	*.081*		
Continuity Correction[b]	*2.425*	*1*	*.119*		
Likelihood Ratio	*3.096*	*1*	*.078*		
Fisher's Exact Test				*.091*	*.059*
Linear-by-Linear Association	*3.028*	*1*	*.082*		
N of Valid Cases	*130*				

Finally, the relationship between leprosy (λ) and median age at death for the adult sample was considered using a Kaplan-Meier survival analysis, according to the method developed by Boldsen (2008). As noted earlier in the methods section on age estimation (page 90), the approach used in this research made use of the median ages derived from the individual age ranges. These median estimates cannot be considered the equivalent of exact individual ages due to limitations in the osteological techniques.

The Kaplan-Meier survival analysis also showed no significant relationship between adult mean age at death and λ either for the entire sample ($\bar{x}$ = 0.605, df = 2, p = 0.739) (Table 4-16, Figure 4-28) or for females ($\bar{x}$ = 1.632, df = 2, p = 0.442) (Table 4-17, Figure 4-29) or males ($\bar{x}$ = 1.422, df = 2, p = 0.491) (Table 4-17, Figure 4-30) respectively. This is consistent with Boldsen's (2008) findings for the Lauchheim cemetery and for much of the Schleswig material (Boldsen et al., 2013).

Table 4-16 Mantel-Cox survival analysis testing the relationship between leprosy diagnosis and median age at death for the complete sample

	Chi-Square	df	Sig.
Log Rank (Mantel-Cox)	*.605*	*2*	*.739*
Test of equality of survival distributions for the different levels of Leprosy.			

Figure 4-28 Kaplan-Meier survival curve for the complete sample representing the relationship between median age at death and leprosy scores

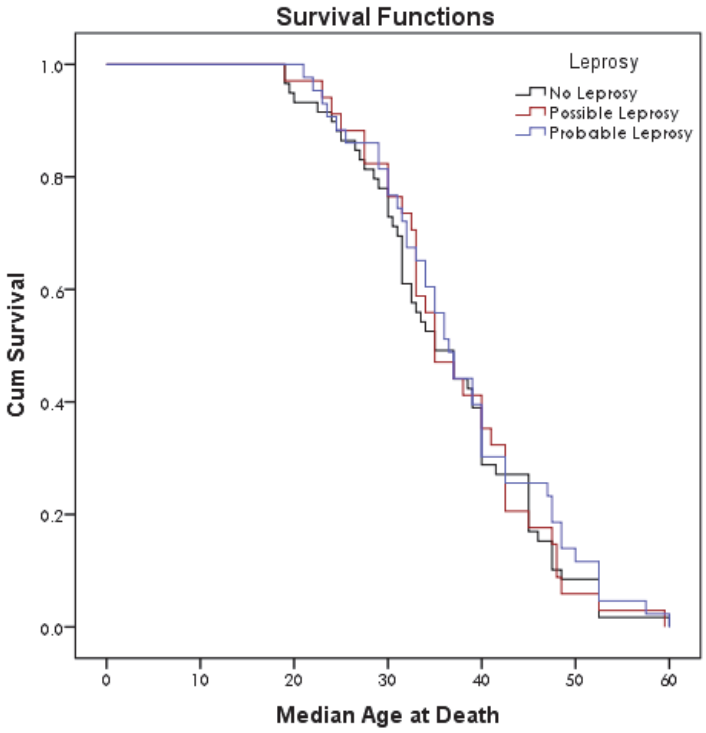


Table 4-17 Mantel-Cox survival analysis testing the relationship between leprosy diagnosis and median age at death by sex

Sex		Chi-Square	df	Sig.
Female	Log Rank (Mantel-Cox)	*1.632*	*2*	*.442*
Male	Log Rank (Mantel-Cox)	*1.422*	*2*	*.491*
Test of equality of survival distributions for the different levels of Leprosy.				

Figure 4-29 Kaplan-Meier survival curve for females representing the relationship between median age at death and leprosy scores

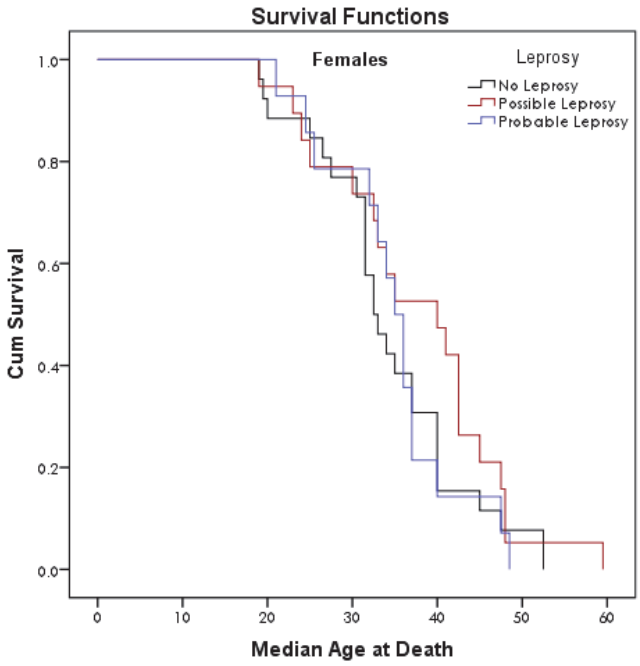
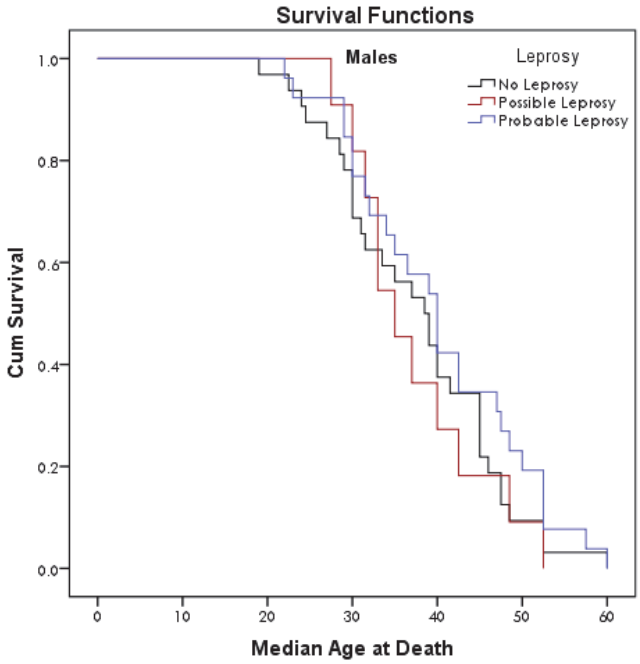


Figure 4-30 Kaplan-Meier survival curve for males representing the relationship between median age at death and leprosy scores



Temporal changes in health in medieval Denmark

Age at Death

When the samples were broadly broken down into two chronological phases, divided by the mid-14th century, a total of 95 individuals were available for the earlier period and 35 individuals for the later period. Not all individuals initially selected had information about arm position, and so these could not be considered as part of the temporal analysis. The imbalance in sample size from the two periods is consistent across many sites, with fewer burials in general dating to the period after 1350 A.D. It is possible that this reflects the general decline in population size following the Black Death epidemic (Bøgh, 1999; Benedictow, 2004). This imbalance needs to be kept in mind when considering all results.

Since the adult mean age at death is not significantly different between males and females, the combined sex samples for each time period were considered initially. The subsamples by date satisfied Levene's test for equality of variance (F = 0.097, p = 0.756) (Table 4-19), and showed a normal distribution with no significant skewness or kurtosis (Figure 4-31) and no significant divergence from normality indicated by a Shapiro-Wilk test (Table 4-18), which is used preferentially over the Kolmogorov-Smirnov test in response to small sample sizes and since this test has been shown to be the most powerful (Dyer, 1974; Steinskog et al., 2007; Razali and Wah, 2011). Adults in the earlier period showed a slightly lower mean age at death ($\bar{x}$ = 35.96, sd = 9.476, SE = 0.972 compared to $\bar{x}$ = 37.91, sd = 9.803, SE = 1.65 for the later period) (Figure 4-31). Independent samples t-tests revealed that the difference was not statistically significant (t = -1.035, df = 128, p = 0.303) (Table 4-19).

Figure 4-31 Frequency distribution of median age at death for the early and late periods in the combined sex sample

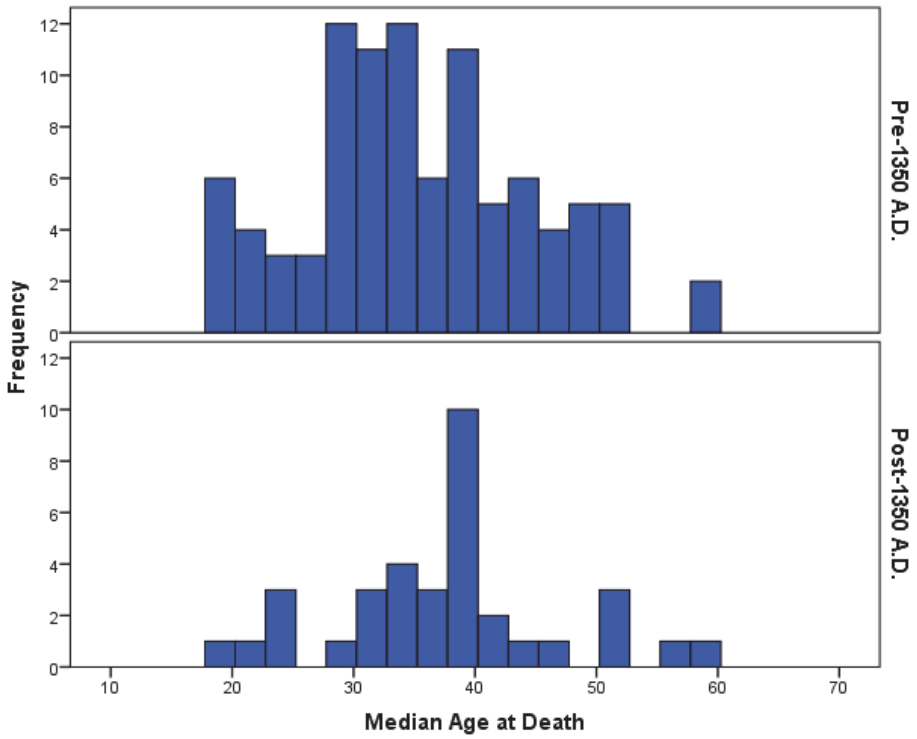


Table 4-18 Test for normality for median ages at death between the early and later periods

		Shapiro-Wilk		
	EarlyLate	Statistic	df	Sig.
Median Age at Death	Pre-1350 A.D.	*.979*	*95*	*.125*
	Post-1350 A.D.	*.965*	*35*	*.313*

Table 4-19 Independent samples t-test considering median age at death by early and late periods

	Levene's Test for Equality of Variances		t-test for Equality of Means						
								95% Confidence Interval of the Difference	
	F	Sig.	t	df	Sig. (2-tailed)	Mean Difference	Std. Error Difference	Lower	Upper
Equal variances assumed	*.097*	*.756*	*-1.035*	*128*	*.303*	*-1.956*	*1.891*	*-5.698*	*1.786*
Equal variances not assumed			*-1.018*	*58.916*	*.313*	*-1.956*	*1.921*	*-5.801*	*1.888*

Figure 4-32 Median age at death between in the early and late periods

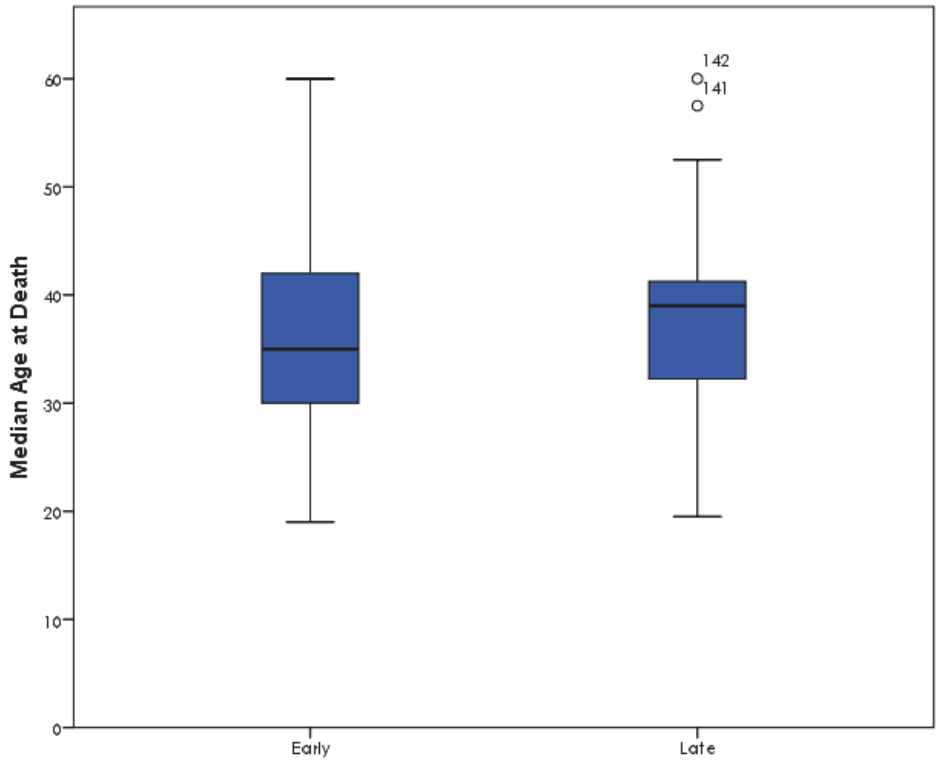
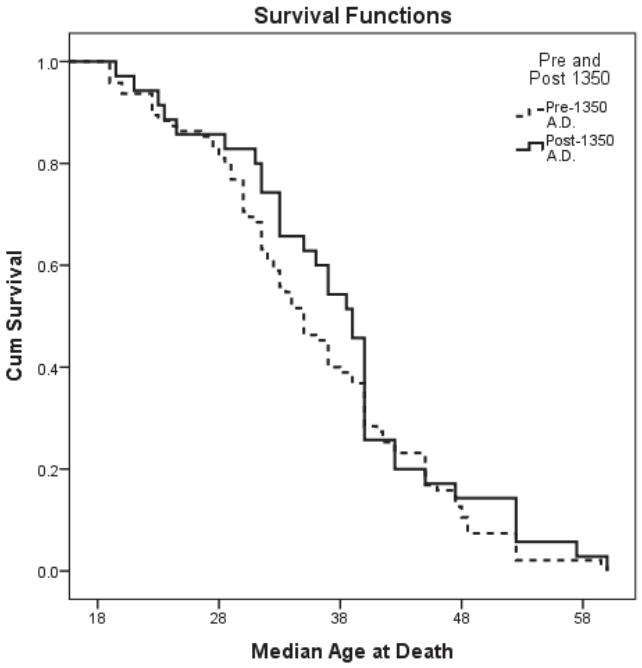


Figure 4-33 Kaplan-Meier survival curve showing the difference in median age at death in the early and late periods



When broken down further by sex, a Shapiro-Wilk test revealed that males violated the assumption of normality for the later period (Table 4-20). A Kruskal-Wallis test showed that there was a significant change in the mean age at death for males in the study sample, increasing from a mean of 36.42 years of age prior to 1350 A.D. to a mean of 43.53 years of age after 1350 A.D. (Table 4-21). However, the small number in the later sample limits any inference from this result (Table 4-20). A reverse change is seen in females, with a decrease in mean age at death from 35.05 in the early period to a mean of 33.72 in the late period. Once again, sample sizes in the later period are much smaller and this temporal change is not statistically significant for females in the subsample. These patterns with each sex can further be observed for the median age at death and range in Figure 4-34 but the contrasting patterns between the sexes combined with smaller sample sizes for the later period cautions against drawing conclusions about these patterns.

Figure 4-34 Median age at death for males and females in the early and late periods

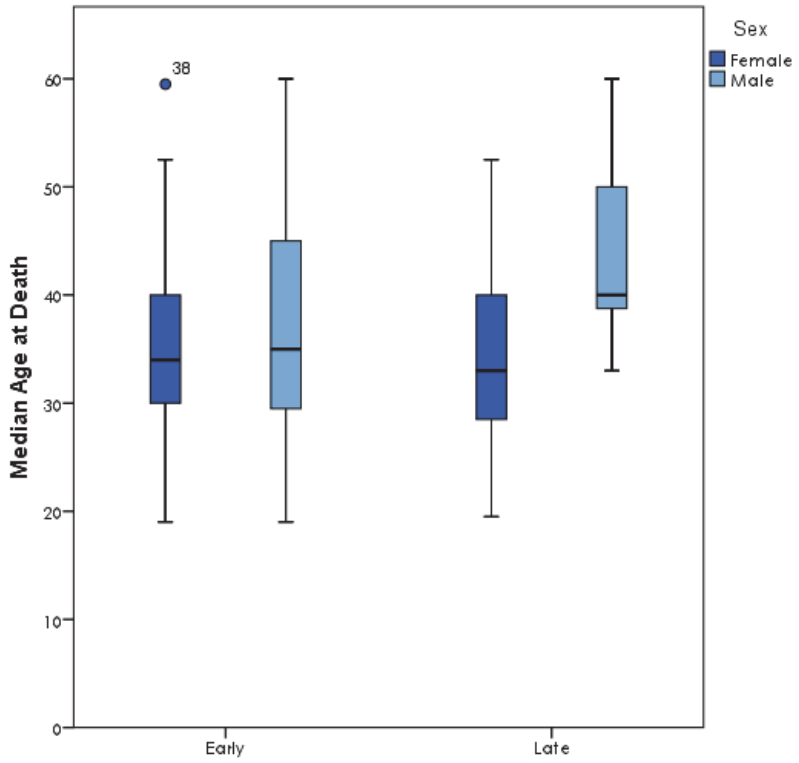


Table 4-20 Descriptive statistics and test for normality for median ages at death for males and females between the early and late periods

Sex		Pre and Post 1350	Descriptive Statistics			Shapiro-Wilk		
			N	Mean	Std. Dev.	Statistic	df	Sig.
Female	Median Age at Death	Pre-1350 A.D.	*38*	*35.05*	*9.595*	*.975*	*38*	*.552*
		Post-1350 A.D.	*18*	*33.72*	*8.620*	*.970*	*18*	*.792*
Male	Median Age at Death	Pre-1350 A.D.	*52*	*36.42*	*9.790*	*.972*	*52*	*.247*
		Post-1350 A.D.	*16*	*43.53*	*8.063*	*.887*	*16*	*.049*

Table 4-21 Kruskal-Wallis test for median age at death between the early and late periods for males and females

Sex		Median Age at Death
Female	Chi-Square	*.171*
	df	*1*
	Asymp. Sig.	*.680*
Male	Chi-Square	*6.131*
	df	*1*
	Asymp. Sig.	*.013*

Further breakdown of the sample showed that while there was a slight increase in the adult mean age at death over time this change was not statistically significant for any of the periods with adequate sample sizes (Table 4-22). While there was a significant difference detected using a One-Way ANOVA ($F = 3.272$, $df = 3$, $p=0.023$), this is only significant between the latest period group represented by D arm position (those buried after the mid-15th century) and all earlier groups (LSD and Bonferonni post-hoc tests). This group is only represented by 2 individuals, and so this statistic cannot be considered representative. Hochberg's GT2 posthoc test, which controls for very different sample sizes still found significant differences, but the Tamhane's and Dunnett T3 post-hoc tests which are more conservative and control for Type I errors respectively (Field, 2009) showed no statistical significance between the categories, which is probably the most accurate reflection of these data based on this small sample size (i.e., a much larger samples in each temporal category would be necessary for any conclusions to be made).

Table 4-22 Descriptive statistics for median age at death across four periods

	N	Mean	Std. Deviation	Std. Error	95% Confidence Interval for Mean		Minimum	Maximum
					Lower Bound	Upper Bound		
Pre-1250 A.D.	*57*	*35.42*	*8.699*	*1.152*	*33.11*	*37.73*	*19*	*53*
1250 -1350 A.D.	*39*	*36.67*	*10.483*	*1.679*	*33.27*	*40.06*	*19*	*60*
1350 A.D. to mid-15th century A.D.	*32*	*36.92*	*9.000*	*1.591*	*33.68*	*40.17*	*20*	*58*
Post mid-15th century A.D.	*2*	*56.25*	*5.303*	*3.750*	*8.60*	*103.90*	*53*	*60*
Total	*130*	*36.48*	*9.567*	*.839*	*34.82*	*38.14*	*19*	*60*

While there were no significant changes in adult mean age at death for both samples combined, an ANOVA did show a significant change in mean age at death over time for males, but not for females ($F = 4.351$, $df = 3$, $p=0.007$) (Table 4-24). When considered further, it was clear that the largest contributing factor to this was the difference between the latest period individuals and those from the earlier periods which has already been discussed (Table 4-23, Figure 4-35). However, there was also a statistically significant difference apparent between individuals from the pre-1250 A.D. subsample and the individuals from the 1350 A.D. to mid-15th century subsample ($p = 0.029$)

This difference in means was significant with the LSD posthoc test ($p=0.029$) but was not when using the more conservative Tamhane ($p=0.059$) and Dunnett T3 ($p=0.057$) posthoc tests. The Bonferroni and Hochberg tests controlling for type I errors and different sample sizes also did not detect statistically significant differences, and so while these results are tantalizing, and while the incease in the adult mean age at death would be

consistent with the hypothesis predicting an improvement in health after the relief of pressure following the events of the mid-14th century, these results must be treated with caution.

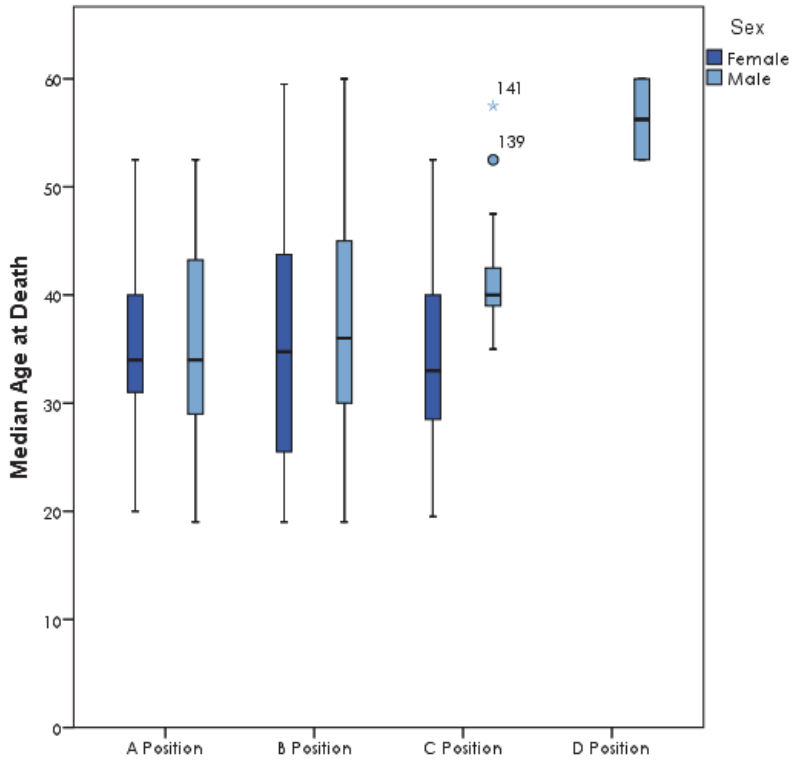
Table 4-23 Descriptive statistics for median age at death across four periods for males and females

Sex		N	Mean	Std. Deviation	Std. Error	95% Confidence Interval for Mean		Minimum	Maximum
						Lower Bound	Upper Bound		
Female	Pre-1250 A.D.	22	34.66	8.107	1.728	31.06	38.25	20	53
	1250 -1350 A.D.	16	35.59	11.599	2.900	29.41	41.77	19	60
	1350 A.D. to mid-15th century A.D.	18	33.72	8.620	2.032	29.44	38.01	20	53
	Total	56	34.63	9.235	1.234	32.15	37.10	19	60
Male	Pre-1250 A.D.	31	35.63	9.545	1.714	32.13	39.13	19	53
	1250 -1350 A.D.	22	37.39	10.063	2.145	32.92	41.85	19	60
	1350 A.D. to mid-15th century A.D.	13	42.38	6.426	1.782	38.50	46.27	35	58
	Post mid-15th century A.D.	2	56.25	5.303	3.750	8.60	103.90	53	60
	Total	68	38.10	9.835	1.193	35.71	40.48	19	60

Table 4-24 One Way ANOVA considering the difference in median age at death between the sexes

Sex		Sum of Squares	df	Mean Square	F	Sig.
Female	Between Groups	29.711	2	14.856	.169	.845
	Within Groups	4661.414	53	87.951		
	Total	4691.125	55			
Male	Between Groups	1097.977	3	365.992	4.351	.007
	Within Groups	5383.152	64	84.112		
	Total	6481.129	67			

Figure 4-35 Male and female age at death over four periods



Stature

A statistically significant decrease in mean stature as measured in the grave from 165.06 (N=57, sd=9.302) to 157.03 (N=18, sd=9.156) was seen over time using an independent samples t-test (t = 3.235, df = 28.5, p = 0.003) (Figure 4-36). A statistically significant decrease from 157.5 cm (N=23, sd=6.78233) to 151.58 cm (N=12, sd=4.18783) is also apparent for females over time (Figure 4-37, Table 4-25, Table 4-26). The effect size for this difference is found to be high (d = 0.96, r = 0.43). While there is a decrease in the mean stature for males from the early to the later period from $\bar{x}$ = 171.07 cm (n =30, sd = 6.65) to $\bar{x}$ = 167.92 cm (n =6, sd = 5.75) (in contrast to the increase in mean age at death for this group) (Table 4-25, Figure 4-37), this difference is not

statistically significant (Table 4-26). The issues of small sample size can be seen in Table 4-25.

Interestingly, there was no statistically significant difference in femur length for either males or females between the two periods. The particularly small sample size for the later period (n =14 females and n =9 males when femur length is used, n =12 females and n =6 males for stature in the grave), however, makes these patterns highly unlikely to be representative.

Figure 4-36 Frequency distribution of stature in the grave for the early period combined sample

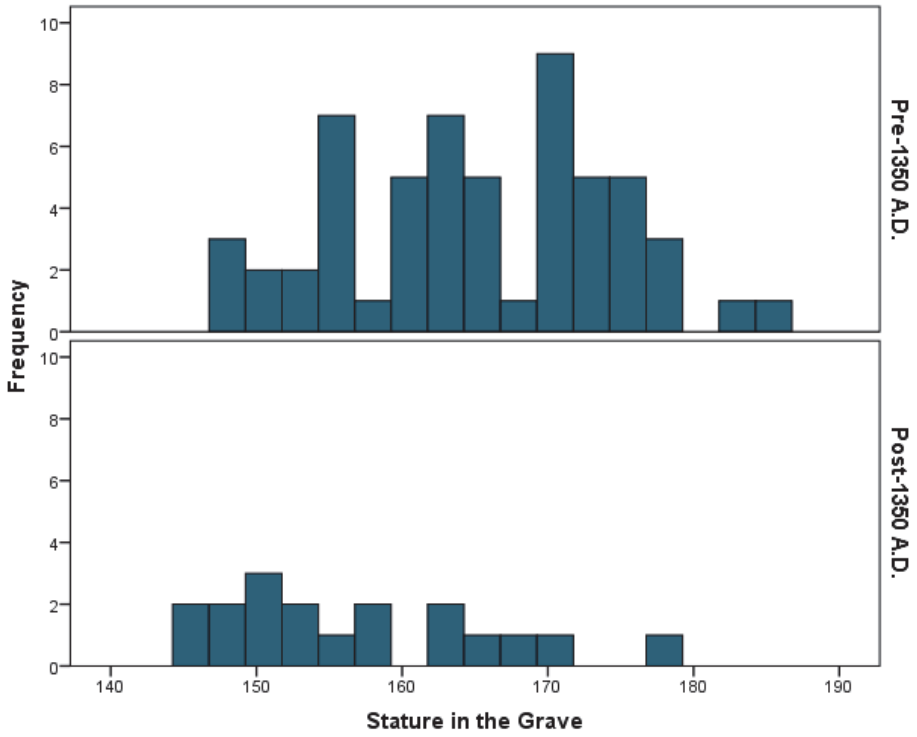


Table 4-25 Descriptive statistics for stature from early to late periods by sex

Sex		Pre and Post 1350	N	Mean	Std. Deviation	Std. Error Mean
Female	Stature	Pre-1350 A.D.	*23*	*157.5000*	*6.78233*	*1.41421*
		Post-1350 A.D.	*12*	*151.5833*	*4.18783*	*1.20892*
Male	Stature	Pre-1350 A.D.	*30*	*171.0667*	*6.65755*	*1.21550*
		Post-1350 A.D.	*6*	*167.9167*	*5.74819*	*2.34669*

Figure 4-37 Stature in the Grave for males and females in the early and late periods

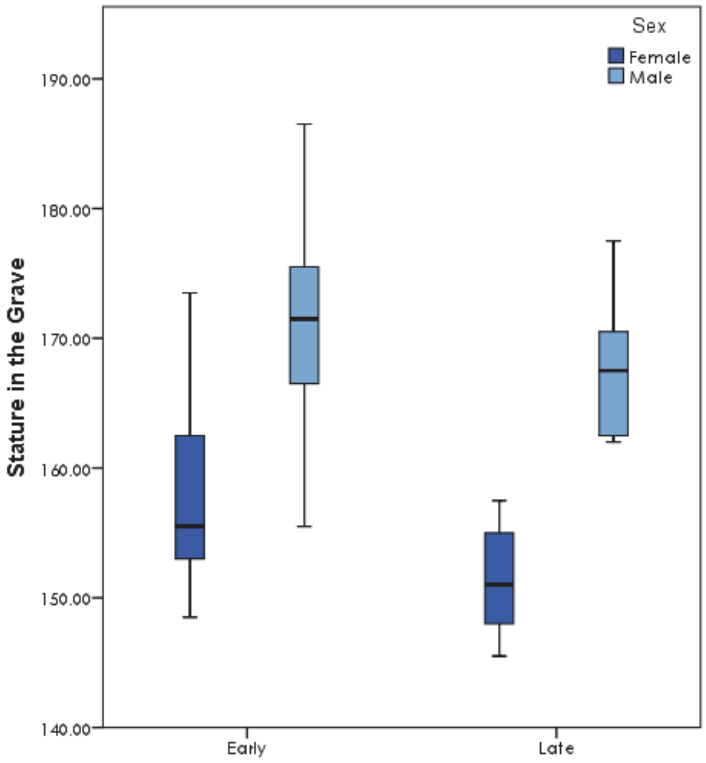


Table 4-26 Independent samples t-test for mean stature between the early and late periods for males and females

Sex		Levene's Test for Equality of Variances		t-test for Equality of Means						
									95% Confidence Interval of the Difference	
		F	Sig.	t	df	Sig. (2-tailed)	Mean Difference	Std. Error Difference	Lower	Upper
Female Stature	Equal variances assumed	*3.905*	*.057*	*2.750*	*33*	*.010*	*5.91667*	*2.15180*	*1.53881*	*10.29453*
	Equal variances not assumed			*3.180*	*31.877*	*.003*	*5.91667*	*1.86051*	*2.12632*	*9.70702*
Male Stature	Equal variances assumed	*.155*	*.697*	*1.078*	*34*	*.288*	*3.15000*	*2.92109*	*-2.78638*	*9.08638*
	Equal variances not assumed			*1.192*	*7.94*	*.268*	*3.15000*	*2.64280*	*-2.95177*	*9.25177*

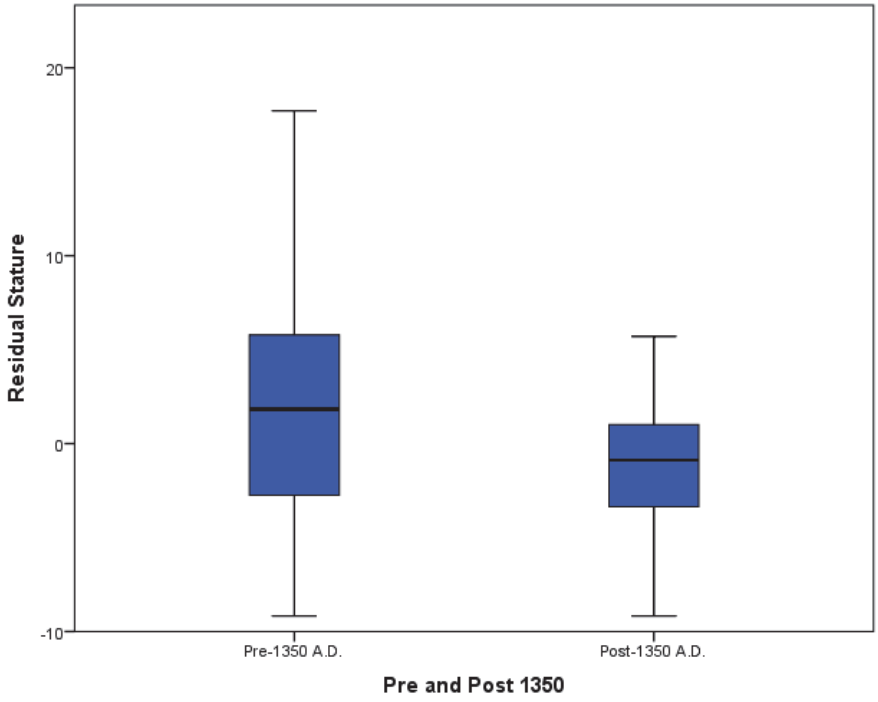
A consideration of residual height[22] showed a significant decrease in the mean from the early to the late period ($t = 2.083$, $df = 66$, $p = 0.039$) (Table 4-27). This suggests that, using this subsample, femoral height increased in relation to sitting height from the earlier to the late periods. This result, does, however, need to be treated with extreme caution as only 18 individuals were available in the late period. Overlap in the range is clear between the periods, but the later period lacks the higher residuals (which would have indicate shorter femoral lengths than expected given height in the grave) (Figure 4-38).

[22] Residual height is calculated by determining the predicted height using the Tirup regression formulae and by subtracting this height from the height derived from stature in the grave.

Table 4-27 Descriptive statistics showing residual height from the early to the late periods

Sex	Pre and Post 1350	N	Mean	Std. Deviation	Std. Error Mean
Residual Stature	Pre-1350 A.D.	*50*	*1.8150*	*5.80799*	*.82137*
	Post-1350 A.D.	*18*	*-1.2602*	*3.85478*	*.90858*

Figure 4-38 Box plot showing the difference in residual height between the early and late periods



Further temporal breakdown (Figure 4-39) shows a significant difference in females (Table 4-28) between both of the earlier periods and the period just after 1350 A.D. (1350 A.D. to the mid15th century) (p=0.031 and p=0.018, respectively using LSD posthoc test). Only the difference between the period right before and right after 1350 A.D. was significant using more robust posthoc tests (Tamhane and Dunnett T3) (Table 4-29). The small sample size of nine individuals from the period lasting from the mid-13th to the mid-14th centuries is problematic in this respect (Table 4-30).

Figure 4-39 Stature in the Grave for males and females over four periods

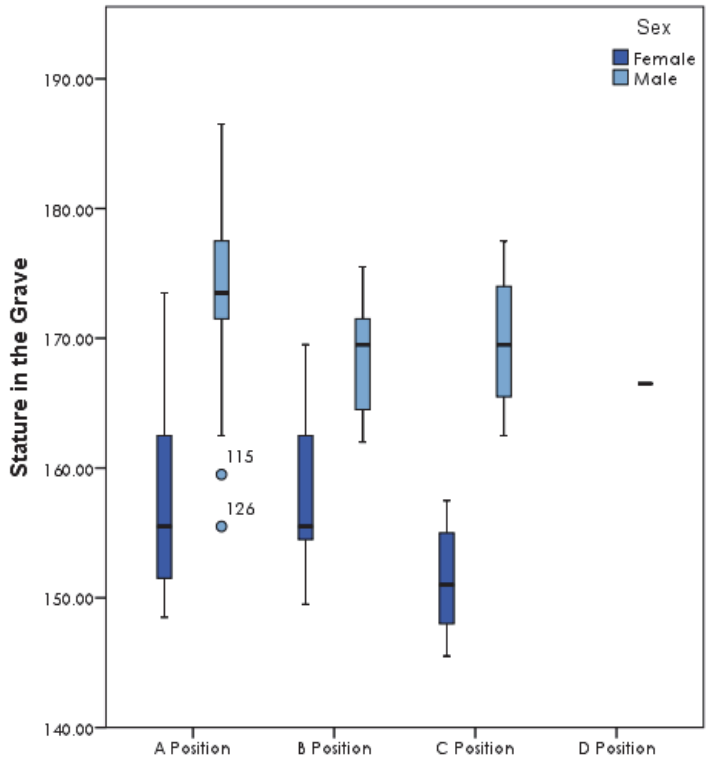


Table 4-28 ANOVA for stature as measured in the grave over four periods for males and females

Sex		Sum of Squares	df	Mean Square	F	Sig.
Female		*284.999*	*2*	*142.500*	*3.813*	*.033*
		1195.972	*32*	*37.374*		
		1480.971	*34*			
Male		*117.869*	*3*	*39.290*	*.910*	*.447*
		1382.318	*32*	*43.197*		
		1500.187	*35*			

Table 4-29 Tamhane and Dunnett T3 post-hoc tests for stature as measured in the grave by four periods for females

Sex	(I) Arm Position Dates	(J) Arm Position Dates	Mean Difference (I-J)	Std. Error	Sig.	95% Confidence Interval	
						Lower Bound	Upper Bound
Tamhane	Pre-1250 A.D.	1250 -1350 A.D.	-1.27778	2.86833	.961	-8.7915	6.2359
		1350 A.D. to mid-15th century A.D.	5.41667	2.27926	.079	-.4874	11.3207
	1250 -1350 A.D.	Pre-1250 A.D.	1.27778	2.86833	.961	-6.2359	8.7915
		1350 A.D. to mid-15th century A.D.	6.69444*	2.44035	.049	.0185	13.3704
	1350 A.D. to mid-15th century A.D.	Pre-1250 A.D.	-5.41667	2.27926	.079	-11.3207	.4874
		1250 -1350 A.D.	-6.69444*	2.44035	.049	-13.3704	-.0185
Dunnett T3	Pre-1250 A.D.	1250 -1350 A.D.	-1.27778	2.86833	.958	-8.7583	6.2028
		1350 A.D. to mid-15th century A.D.	5.41667	2.27926	.077	-.4653	11.2986
	1250 -1350 A.D.	Pre-1250 A.D.	1.27778	2.86833	.958	-6.2028	8.7583
		1350 A.D. to mid-15th century A.D.	6.69444*	2.44035	.047	.0677	13.3212
	1350 A.D. to mid-15th century A.D.	Pre-1250 A.D.	-5.41667	2.27926	.077	-11.2986	.4653
		1250 -1350 A.D.	-6.69444*	2.44035	.047	-13.3212	-.0677

*. The mean difference is significant at the 0.05 level.

Table 4-30 Descriptive statistics for mean stature as measured in the grave across four periods for males and females

Sex		N	Mean	Std. Deviation	Std. Error	95% Confidence Interval for Mean		Minimum	Maximum
						Lower Bound	Upper Bound		
Female	Pre-1250 A.D.	14	157	7.23	1.93	152.83	161.17	148.50	173.50
	1250 -1350 A.D.	9	158.28	6.36	2.12	153.39	163.17	149.50	169.50
	1350 A.D. to mid-15th century A.D.	12	151.58	4.19	1.21	148.92	154.24	145.50	157.50
	Total	35	155.47	6.60	1.12	153.20	157.74	145.50	173.50
Male	Pre-1250 A.D.	17	172.38	8.02	1.95	168.26	176.51	155.50	186.50
	1250 -1350 A.D.	14	168.82	4.28	1.15	166.35	171.30	162.00	175.50
	1350 A.D. to mid-15th century A.D.	4	169.75	6.18	3.09	159.91	179.59	162.50	177.50
	Post mid-15th century A.D.	1	166.50	.	.	.	.	166.50	166.50
	Total	36	170.54	6.55	1.09	168.33	172.76	155.50	186.50

Stress

The consideration of the frequency of stress events showed no significant change from the earlier to the later period for either males or females. Once again, the later period is poorly represented by a small sample size, but the distribution of AS frequencies for both sexes does not appear different based on these limited samples. This is true for both the broad date division (Figure 4-40, Figure 4-41, Table 4-32, Figure 4-42, Figure 4-43) and for the finer division by arm position (Figure 4-44) where there are unfortunately particularly small sample sizes.

Figure 4-40 Frequency distribution for AS in the early and late periods

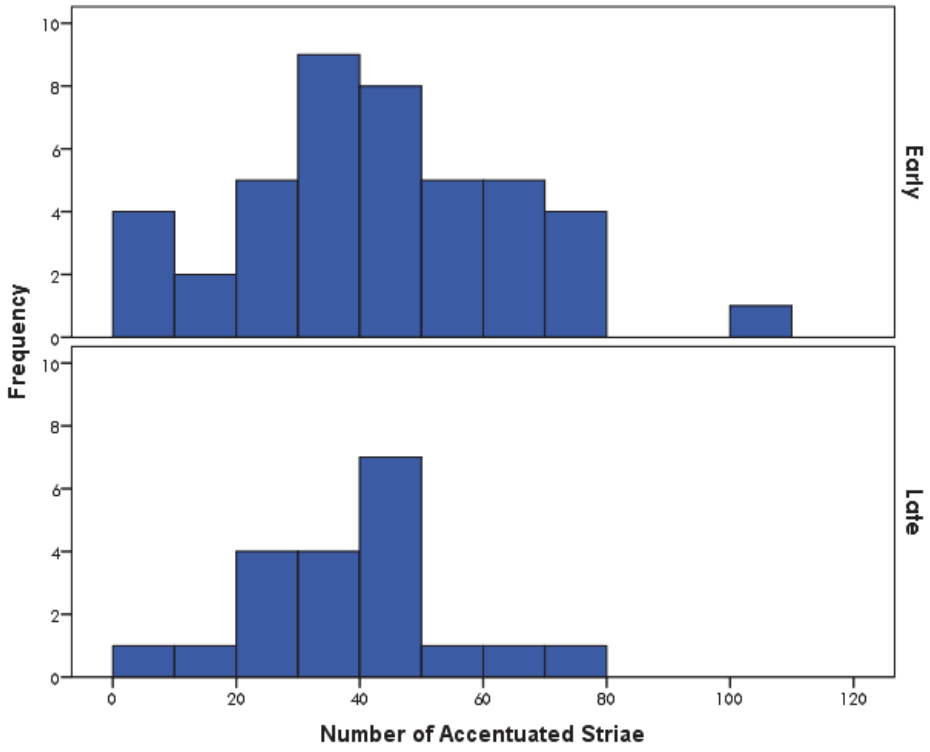


Table 4-31 Descriptive statistics showing the mean number of AS in the early and the late period

	Period	N	Mean	Std. Deviation	Std. Error Mean
Number of Accentuated Striae	Early	*43*	*42.77*	*22.576*	*3.443*
	Late	*20*	*38.10*	*17.131*	*3.831*

Figure 4-41 Box plot showing the median number of AS and the range of AS expression for the early and the late samples

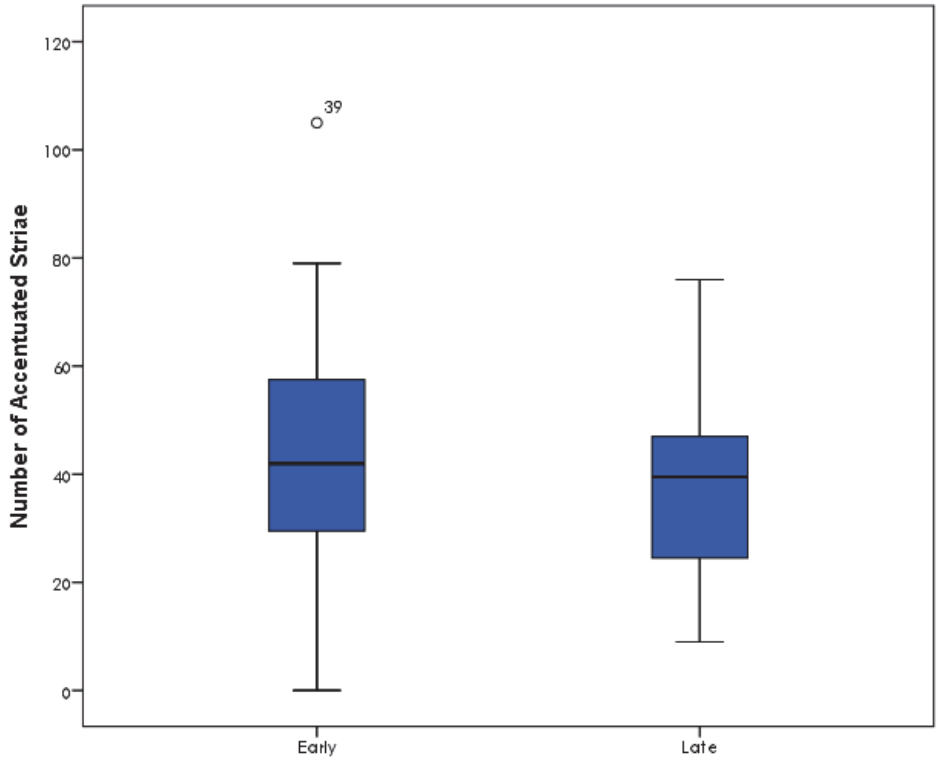


Table 4-32 Descriptive statistics showing the mean number of AS in the early and the late period by sex

Sex		Pre and Post 1350	N	Mean	Std. Deviation	Std. Error Mean
Female	Number of Accentuated Striae	Early	*17*	*33.82*	*20.203*	*4.900*
		Late	*12*	*37.33*	*11.602*	*3.349*
Male	Number of Accentuated Striae	Early	*24*	*49.38*	*23.239*	*4.744*
		Late	*8*	*39.25*	*24.135*	*8.533*

Figure 4-42 Frequency distribution for accentuated striae of Retzius by early and late period for males and females

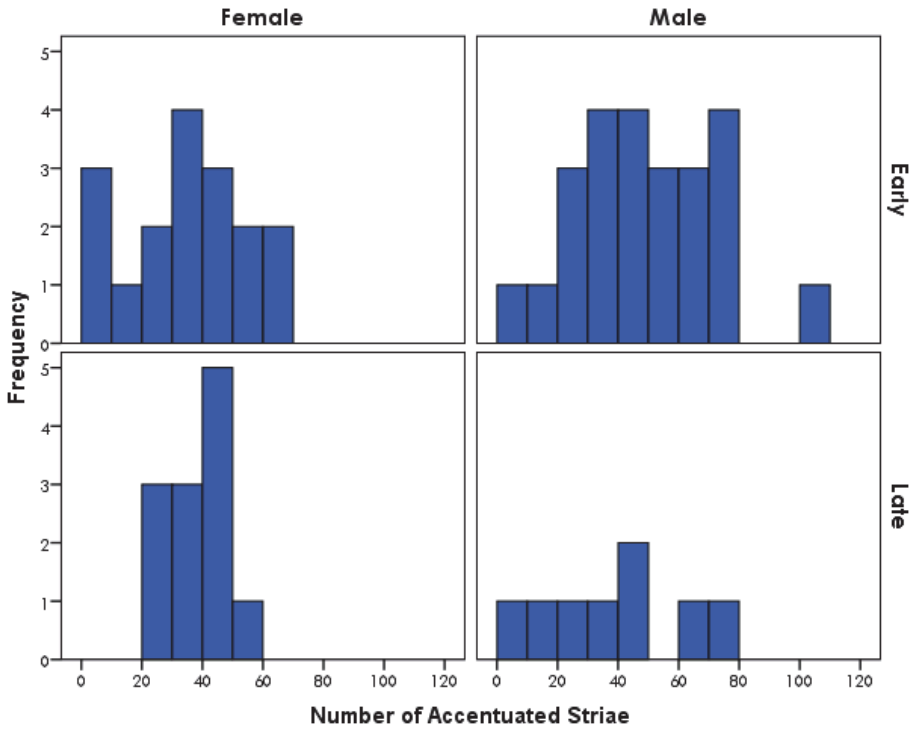


Figure 4-43 Bar graph showing the median number of AS and the range of AS expression for the early and the late samples of males and females

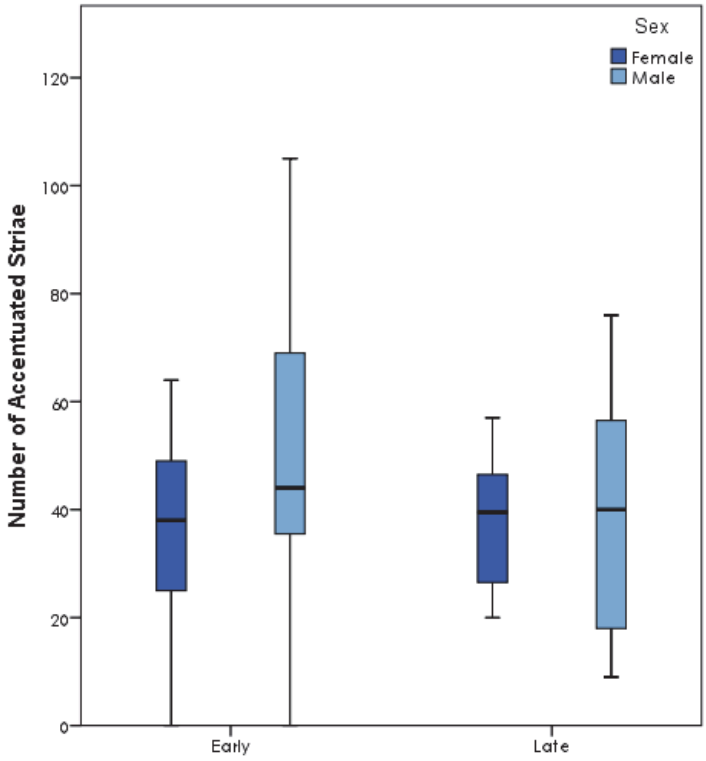
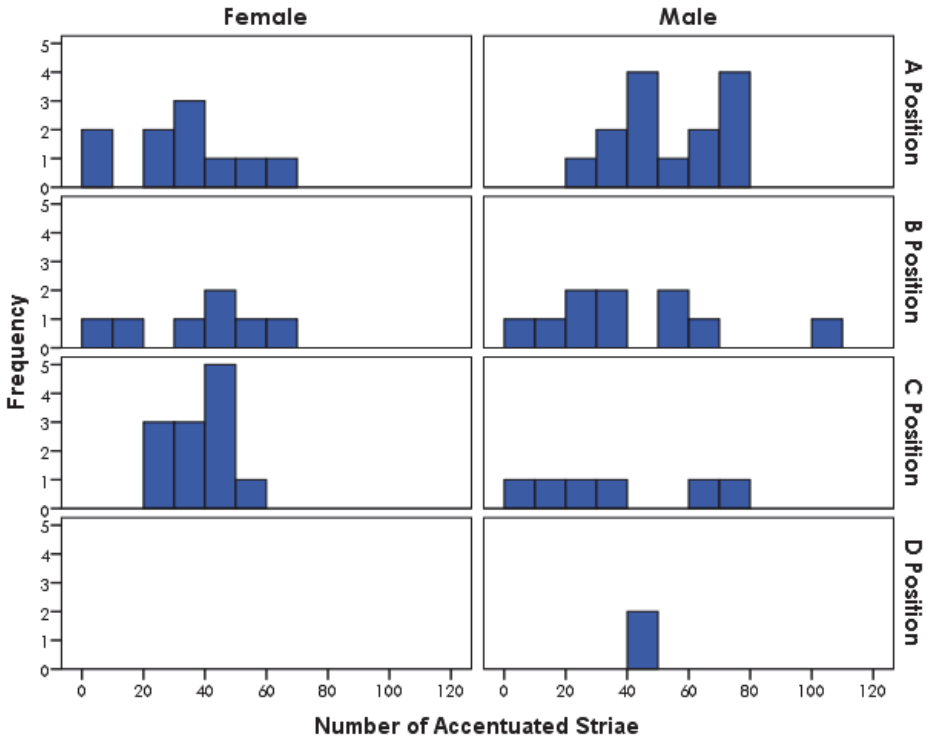


Figure 4-44 Frequency distributions of accentuated striae of Retzius by four periods for males and females



While sample sizes were slightly larger for the consideration of surface defects, the early and late periods were still badly biased (with 75 individuals from the earlier sample and 25 from the later sample) (Figure 4-65, Figure 4-45, Table 4-33). The consideration of stress on this level also showed no significant change in the mean number of stress events from the early to late periods with the complete sample (Figure 4-46) or with the sexes considered separately (Figure 4-47, Figure 4-48). This was consistent when the sample was broken down further by sex for the four-period temporal scale (Figure 4-49), but once again this further compounds the sample size issues and cannot be taken as representative.

Table 4-33 Descriptive statistics of DEH for the early and late periods

	Pre and Post 1350	N	Mean	Std. Deviation	Std. Error Mean
Dental Enamel Hypoplasia	Early	*75*	*3.49*	*1.955*	*.226*
	Late	*25*	*3.44*	*1.635*	*.327*

Figure 4-45 Frequency distribution of DEH in the early and late periods

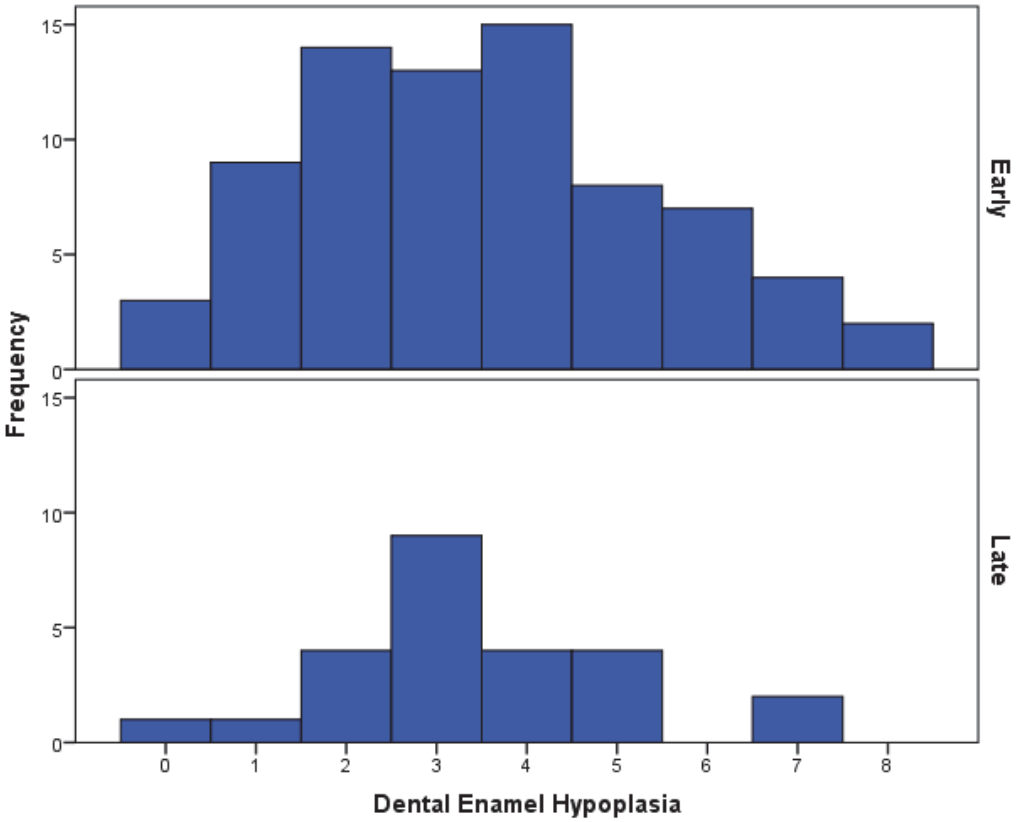


Figure 4-46 Box plots showing the median and range for DEH in the early and late periods

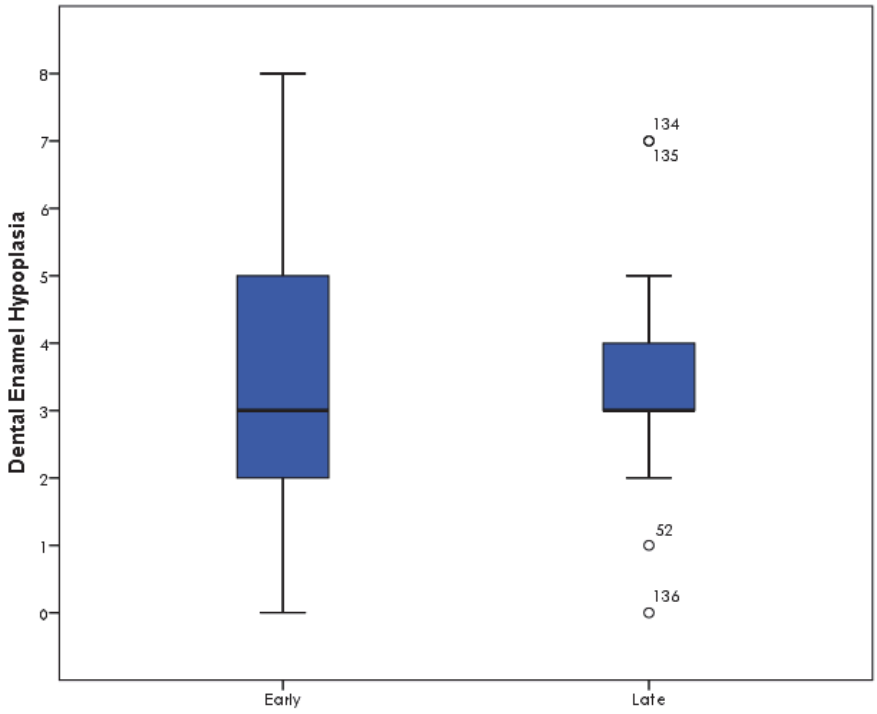


Figure 4-47 Frequency distribution for dental enamel hypoplasia by early and late periods for males and females

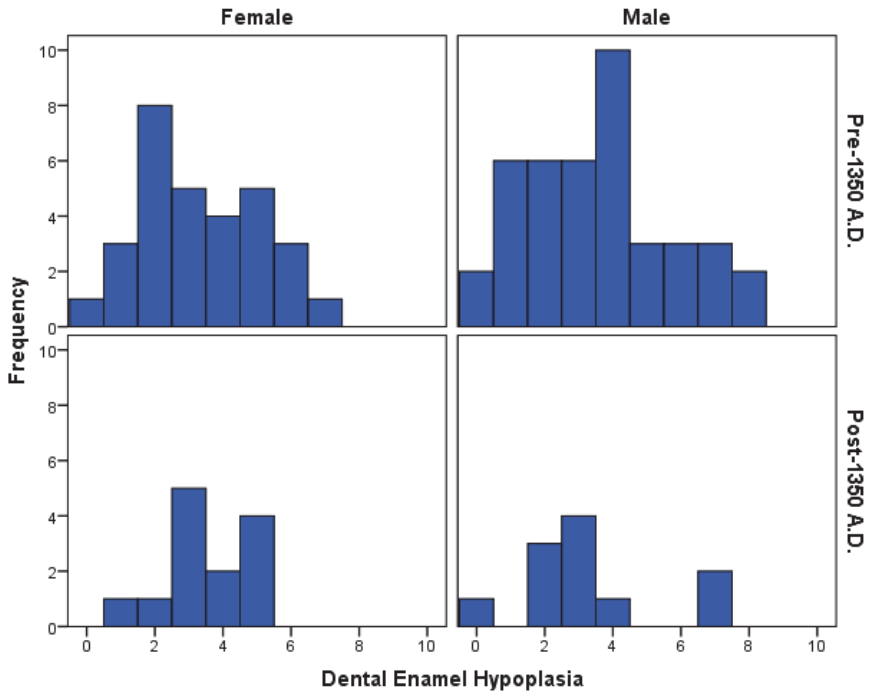


Figure 4-48 Boxplot showing the median and range for DEH for males and females in the early and late periods

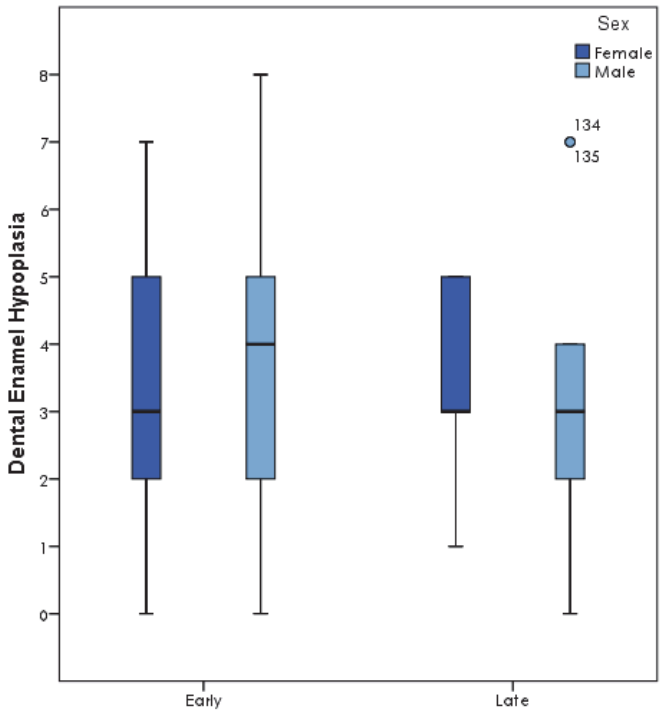
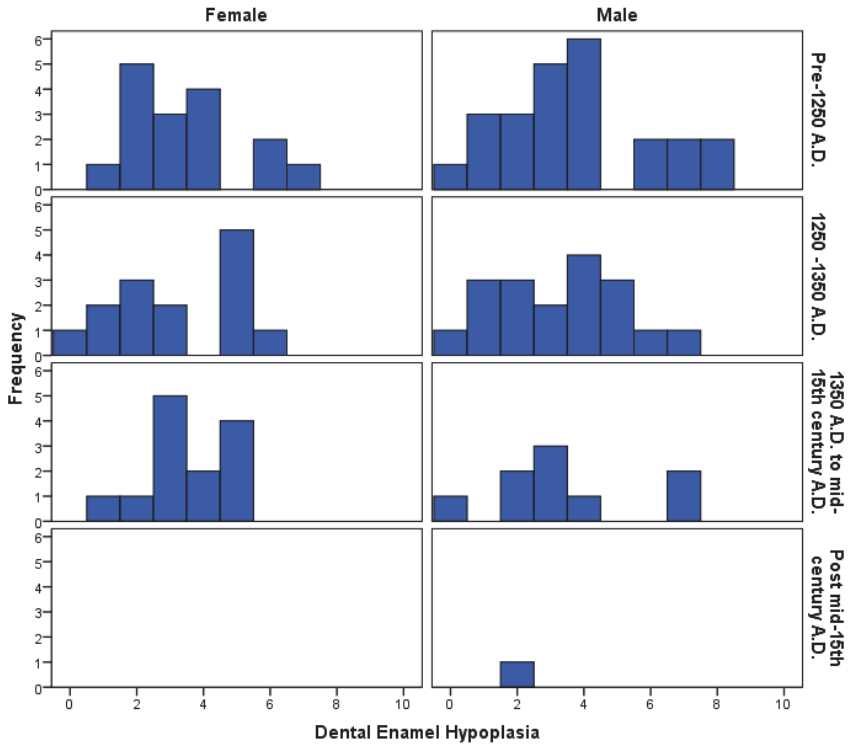


Figure 4-49 Frequency distributions of DEH by four periods for males and females



Disease

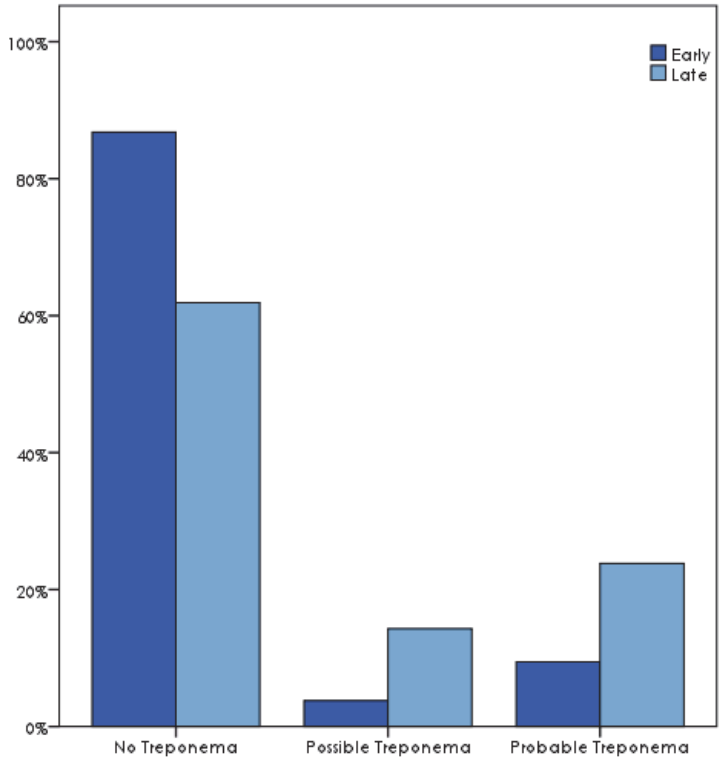
Treponema

Probable treponema was found more frequently in the later period sample (23.81%, n =5) than in the earlier period sample (9.43%, n =5). Despite there being a larger sample for the earlier period (53 individuals who could be scored) than in the later period (n =21), more cases were found in the later period sample (Table 4-34, Figure 4-50). This pattern in prevalence rates held when individuals with possible treponema were included in the 'treponemal' category as well (with n=7 individuals showing pathological changes in each sample, the early sample had a frequency of 13.2% and the later sample had a frequency of 33%). Due to a particularly small number of probable treponemal cases in each period, a Fisher's Exact Test was carried out and this temporal change was not found to be statistically significant (Table 4-35).

Table 4-34 Frequency of probable and possible treponemal cases[23] for the early and late samples, combined with percent of cases as a factor of total number of individuals who could be scored for treponema in each period

	Early		Late		
	Frequency	Percentage	Frequency	Percentage	Total
No Treponema	*48*	*90.57*	*16*	*76.19*	*64*
Possible Treponema	*2*	*3.77*	*3*	*14.29*	*5*
Probable Treponema	*5*	*9.43*	*5*	*23.81*	*10*
Total	*53*	*71.62*	*21*	*28.38*	*74*

Figure 4-50 Diagnosis of treponema for the early and late subsamples expressed as a percentage of total number of individuals who could be scored for treponema in each period



[23] Once again, a case was determined as probable if the characteristic indicators were visible. A possible diagnosis was given in cases where lesions existed that were consistent with the disease but where a higher level of uncertainty existed due to missing or poorly preserved elements. No disease refers to cases with sufficient preservation of elements but with elements exhibiting no evidence for a diseased state.

Table 4-35 Chi-Square test and Fisher's Exact test for probable treponema rates in the early and late periods

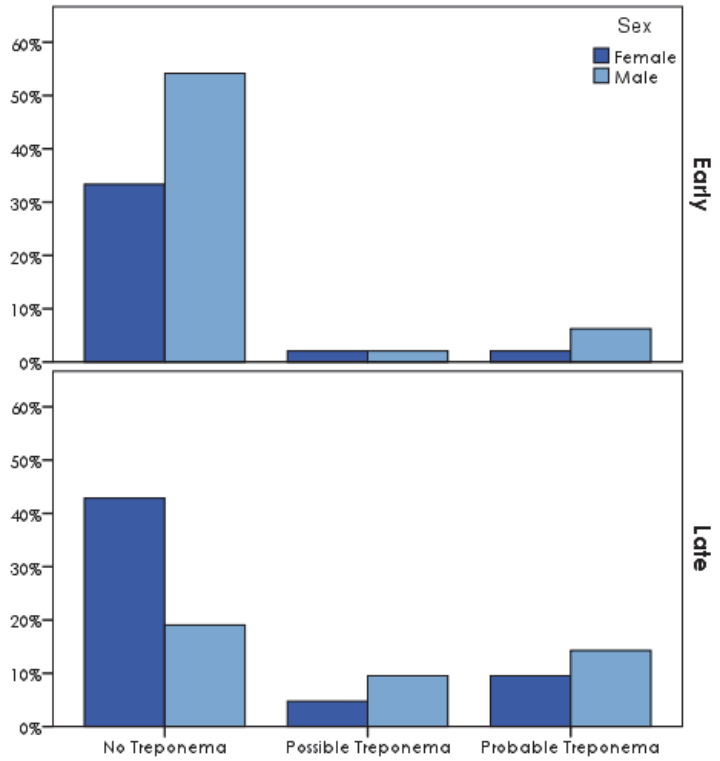
	Value	df	Asymp. Sig. (2-sided)	Exact Sig. (2-sided)	Exact Sig. (1-sided)
Pearson Chi-Square	*2.659*[a]	*1*	*.103*		
Continuity Correction[b]	*1.572*	*1*	*.210*		
Likelihood Ratio	*2.439*	*1*	*.118*		
Fisher's Exact Test				*.135*	*.108*
Linear-by-Linear Association	*2.624*	*1*	*.105*		
N of Valid Cases	*74*				

A consideration of the sex distribution for treponema in these two samples shows a difference once again with it being slightly more commonly found in males (14.29% of males, n =3) than in females (9.52% of females, n =2) for the later period (Table 4-36, Figure 4-51). Fisher's Exact Test showed the change in frequency of probable and possible cases combined to be significant in males (p = 0.018), but not in females (p = 0.531) where there were particularly small sample sizes (Table 4-37). No significant temporal difference was found in the occurrence of strictly probable cases of treponema in either males or females.

Table 4-36 Frequency of treponematosis by sex from the early to late periods and percentage expressed in relation to total number of individuals who could be scored for treponema in each period[24]

		Early		Late	
		Frequency	Percentage	Frequency	Percentage
Female	No Treponema	16	*33.3*	9	*42.86*
	Possible Treponema	1	*2.08*	1	*4.76*
	Probable Treponema	1	*2.08*	2	*9.52*
	Total	18	*37.5*	12	*57.14*
Male	No Treponema	26	*54.17*	4	*19.05*
	Possible Treponema	1	*2.08*	2	*9.52*
	Probable Treponema	3	*6.25*	3	*14.29*
	Total	30	*62.5*	9	*42.9*

Figure 4-51 Diagnosis of treponema in males and females for the early and late subsamples expressed as a percentage of total number of individuals who could be scored for treponema in each period



[24] Once again, a case was determined as probable if the characteristic indicators were visible. A possible diagnosis was given in cases where lesions existed that were consistent with the disease but where a higher level of uncertainty existed due to missing or poorly preserved elements. No disease refers to cases with sufficient preservation of elements but with elements exhibiting no evidence for a diseased state.

Table 4-37 Chi-Square test and Fisher's Exact test for treponema rates using combined probable and possible cases in the early and later periods by sex

Sex		Value	df	Asymp. Sig. (2-sided)	Exact Sig. (2-sided)	Exact Sig. (1-sided)
Female	Pearson Chi-Square	1.000[a]	1	.317		
	Continuity Correction[b]	.250	1	.617		
	Likelihood Ratio	.980	1	.322		
	Fisher's Exact Test				.364	.304
	Linear-by-Linear Association	.967	1	.326		
	N of Valid Cases	30				
Male	Pearson Chi-Square	6.953[c]	1	.008		
	Continuity Correction[b]	4.778	1	.029		
	Likelihood Ratio	6.210	1	.013		
	Fisher's Exact Test				.018	.018
	Linear-by-Linear Association	6.774	1	.009		
	N of Valid Cases	39				

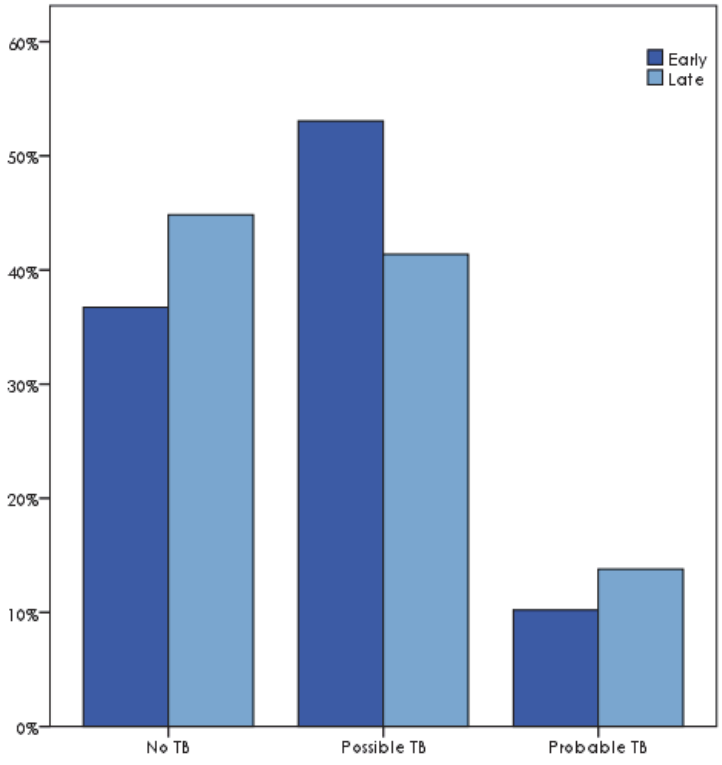
Tuberculosis

The frequency of probable tuberculosis shows no significant change over time using a Fisher's Exact Test (p=0.446) (Table 4-29). The prevalence rates go from 6.41% (n =5) of individuals in the early sample having probable cases of tuberculosis to 13.33% (N=4) of individuals in the later sample (Table 4-38, Figure 4-52), with an odds ratio of 1.408 (Table 4-40).

Table 4-38 Frequency of probable and possible tuberculosis cases for the early and late samples, combined with percent of cases as a factor of total number of individuals who could be scored for tuberculosis in each period[25]

	Early		Late		
	Frequency	Percentage	Frequency	Percentage	Total
No TB	18	***23.08***	13	***43.33***	***31***
Possible TB	26	***33.33***	12	***40.00***	***38***
Probable TB	5	***6.41***	4	***13.33***	***9***
Total	78	***72.22***	30	***27.78***	***108***

Figure 4-52 Diagnosis of tuberculosis for the early and late subsamples expressed as a percentage of total number of individuals who could be scored for tuberculosis in each period



[25] Once again, a case was determined as probable if the characteristic indicators were visible. A possible diagnosis was given in cases where lesions existed that were consistent with the disease but where a higher level of uncertainty existed due to missing or poorly preserved elements. No disease refers to cases with sufficient preservation of elements but with elements exhibiting no evidence for a diseased state.

Table 4-39 Chi-Square test for tuberculosis rates in the early and late periods

	Value	df	Asymp. Sig. (2-sided)	Exact Sig. (2-sided)	Exact Sig. (1-sided)
Pearson Chi-Square	.230[a]	1	.632		
Continuity Correction[b]	.013	1	.910		
Likelihood Ratio	.226	1	.635		
Fisher's Exact Test				.720	.446
Linear-by-Linear Association	.227	1	.634		
N of Valid Cases	78				

Table 4-40 Risk estimate for tuberculosis during the early and late periods showing the odds ratio

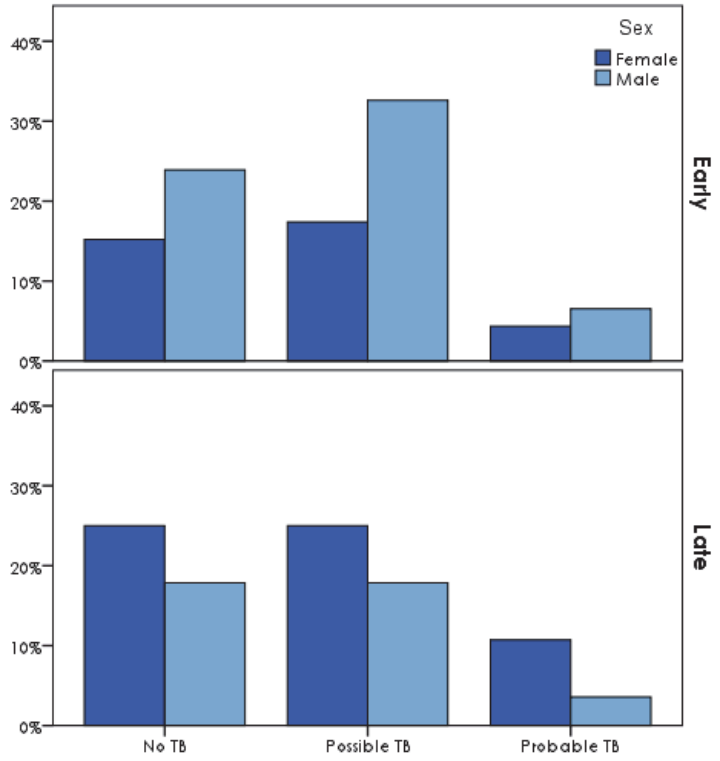
	Value	95% Confidence Interval	
		Lower	Upper
Odds Ratio for Pre and Post 1350 (Early / Late)	1.408	.346	5.729
For cohort NewTB_OddsRatio = No Definite TB	1.042	.876	1.239
For cohort NewTB_OddsRatio = Probable TB	.740	.216	2.536
N of Valid Cases	78		

When broken down by sex, this trend stayed consistent with no statistically significant change over time. Fewer males were diagnosed with probable tuberculosis in the later period (N=1 as opposed to N=3), but the small number of individuals in the later sample (N=11 males as opposed to N=29 males in the earlier sample) cautions against drawing conclusions from this pattern (Table 4-41). This trend was not found to be statistically significant using a Fisher's Exact Test ($p = 0.7$). Females exhibit slightly higher prevalence rates in the later period (odds ratio = 1.607) but this is not statistically significant ($p = 0.5$ using a Fisher's Exact Test). These trends can be seen in Figure 4-53. A consideration of probable and possible cases together once again revealed no significant change over time in either of the sexes.

Table 4-41 Frequency of tuberculosis by sex from the early to late periods and percentage expressed in relation to total number of individuals who could be scored for tuberculosis in each period[26]

		Early		Late	
		Frequency	Percentage	Frequency	Percentage
Female	No TB	7	*15.22*	7	*25.00*
	Possible TB	8	*17.39*	7	*25.00*
	Probable TB	2	*4.35*	3	*10.71*
	Total	17	*36.96*	17	*60.71*
Male	No TB	11	*23.91*	5	*17.86*
	Possible TB	15	*32.61*	5	*17.86*
	Probable TB	3	*6.52*	1	*3.57*
	Total	29	*63.04*	11	*39.29*

Figure 4-53 Diagnosis of tuberculosis by sex for the early and late subsamples expressed as a percentage of total number of individuals who could be scored for tuberculosis in each period



[26] Once again, a case was determined as probable if the characteristic indicators were visible. A possible diagnosis was given in cases where lesions existed that were consistent with the disease but where a higher level of uncertainty existed due to missing or poorly preserved elements. No disease refers to cases with sufficient preservation of elements but with elements exhibiting no evidence for a diseased state.

Leprosy

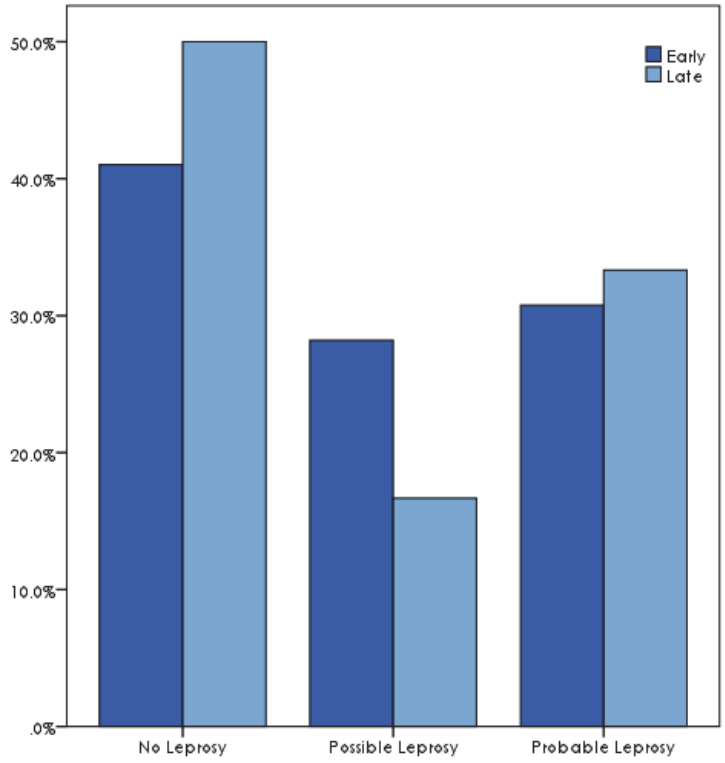
The raw frequency of probable cases of leprosy was slightly higher in the later period (n =34 at 33.33%) than in the earlier period (n =24 at 30.77%) (Table 4-42, Figure 4-54). However, these raw scores suggest that this difference was minimal and a Mantel-Cox Log Rank test showed that the observed leprosy (lambda) scores were not significantly different between the two periods (χ^2. = 0.552, df = 1, p = 0.457).

Table 4-42 Frequency of leprosy for each period and percentage expressed in relation to total number of individuals who could be scored for leprosy in each period. These counts are based on the raw lambda scores, as opposed to the derived statistics[27]

	Early		Late		
	Frequency	Percentage	Frequency	Percentage	Total
No Leprosy	32	***41.03***	15	***50.00***	***47***
Possible Leprosy	22	***28.21***	5	***16.67***	***27***
Probable Leprosy	24	***30.77***	10	***33.33***	***34***
Total	78	***72.22***	30	***27.78***	***108***

[27] Once again, in the case of leprosy diagnosis, any individuals who had λ of between -1 and 1 were classed as possible cases, while individuals with scores equal to or greater than 1 were classed as probable cases (Boldsen, 2008). Individuals below -1 were categorized as unlikely to have suffered from leprosy (Boldsen, 2008).

Figure 4-54 Diagnosis of leprosy for the early and late subsamples expressed as a percentage total number of individuals who could be scored for leprosy in each period (based on lambda scores)

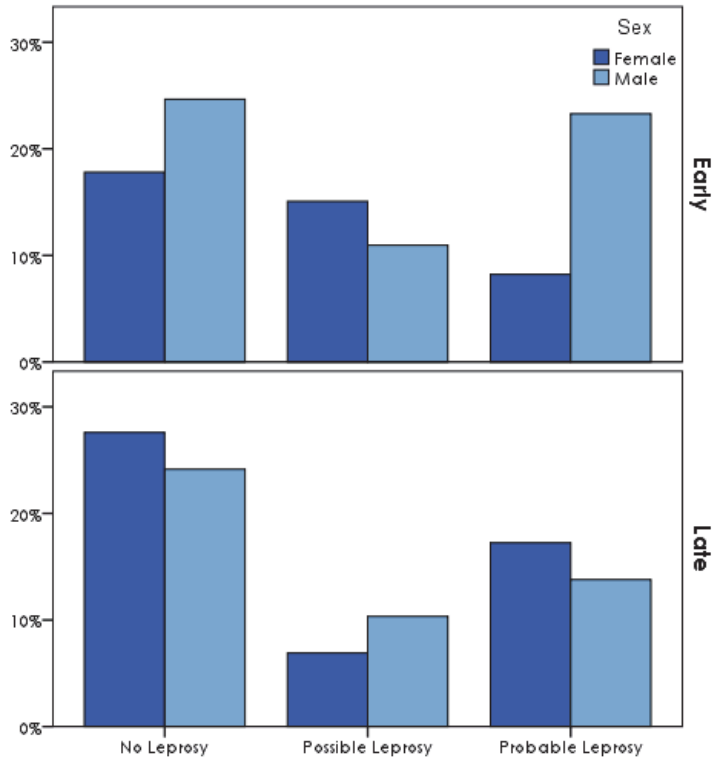


A consideration of raw leprosy scores by sex once again in which lambda scores are used to diagnose probable leprosy cases shows a decrease in leprosy cases for males over time from 23.29% (N =17) to 13.79% (N =4) whereas probable cases in females seem to become twice as common in the later period 8.22% (N =6) to 17.24% (N =5) (Figure 4-55, Table 4-43), with an odds ratio = 2.0. However, neither of these changes is shown to be statistically significant using a Fisher's Exact Test (p = 0.266 for females and p = 0.343 for males). It is interesting that this change would seem to suggest a pronounced change in the sex distribution in the people who likely had leprosy from the early to the late period, but the lack of statistical significance using these raw score limits any interpretation based on these patterns.

Table 4-43 Frequency of leprosy by sex from the early and late periods and percentage expressed in relation to total number of individuals who could be scored for leprosy in each period. These counts are based on the raw lambda scores, as opposed to the derived statistics[28]

		Early		Late	
		Frequency	Percentage	Frequency	Percentage
Female	No Leprosy	13	*17.81*	8	*27.59*
	Possible Leprosy	11	*15.07*	2	*6.90*
	Probable Leprosy	6	*8.22*	5	*17.24*
	Total	30	*41.10*	15	*51.72*
Male	No Leprosy	18	*24.66*	7	*24.14*
	Possible Leprosy	8	*10.96*	3	*10.34*
	Probable Leprosy	17	*23.29*	4	*13.79*
	Total	43	*58.90*	14	*48.28*

Figure 4-55 Leprosy by sex from the early to late periods and percentage expressed in relation to total number of individuals who could be scored for leprosy in each period (based on lambda scores)



[28] Once again, in the case of leprosy diagnosis, any individuals who had λ of between -1 and 1 were classed as possible cases, while individuals with scores equal to or greater than 1 were classed as probable cases (Boldsen, 2008). Individuals below -1 were categorized as unlikely to have suffered from leprosy (Boldsen, 2008).

Site Differences

Age at Death

In order to appropriately contextualize the discussion of site differences in longevity, a brief examination of the subsamples in relation to the broader cemetery samples will be conducted. It is important to note here once again that the broader cemetery statistics were not collected by the author, but rather are derived from the cemetery databases available through ADBOU.

A consideration of the broader cemetery distribution (i.e., from the complete cemetery samples) shows no significant difference in mean age at death between Sejet and Ole Wormsgade. On the other hand, the cemetery subsamples used in this study do show some differences in mean age at death when considered by cemetery, with Ole Wormsgade having a slightly higher mean age at death than Sejet (Table 4-44, Figure 4-57, and Figure 4-59). The difference in mean age at death between the complete cemetery samples and the subsamples used in this study is shown to be statistically significant for Sejet (with an independent samples t-test showing $p = 0.006$, $df = 147$, $t = 2.783$). The frequency distributions for these cemeteries clearly depict this trend, with the Sejet subsample not capturing the oldest individuals. While not as extreme, there is also a visible difference for Ole Wormsgade, although here the mean age at death increases in the subsample. In this case, it is clear from the frequency distribution that there are fewer individuals in the older age category for the subsample, with none positioned above 60 years of age. However, a slight skew can also been seen in the subsample, with the peak falling closer to 40 than to 30 as it does in the complete sample. Overall, the smaller sample seems to have eliminated the oldest, but also to have reduced sample size overall in such a way as to skew the distribution enough to increase the mean age at death.

Table 4-44 Descriptive statistics comparing mean age at death in the complete cemetery samples to that in the subsamples used for this study

Complete Cemetery Samples	N	Mean	Std. Deviation
Sejet	*383*	*37.51*	*9.53*
Ole Wormsgade	*295*	*37.46*	*10.62*
Total	*678*	*37.49*	*10.01*
Cemetery Subsamples			
Sejet	*93*	*34.8*	*8.93*
Ole Wormsgade	*72*	*38.47*	*9.60*
	165	*36.40*	*9.38*

Figure 4-56 Frequency distribution of median age at death for the complete adult sample from Sejet

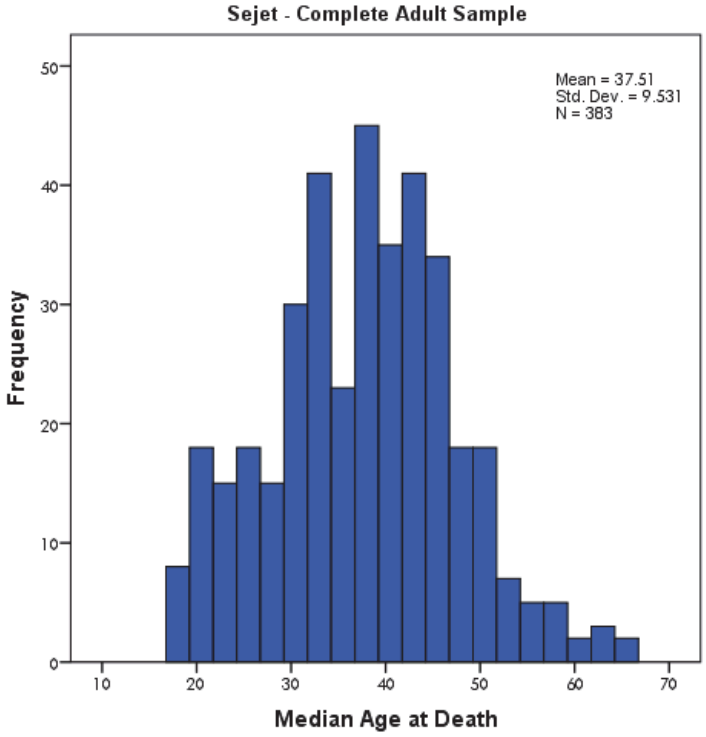


Figure 4-57 Frequency distribution of median age at death for the study subsample from Sejet

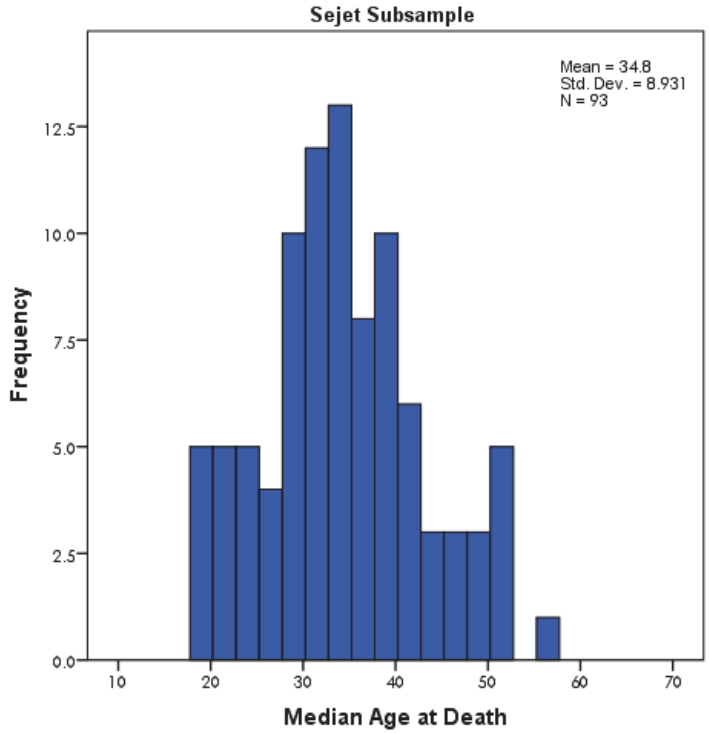


Figure 4-58 Frequency distribution of median age at death for the complete adult sample from Ole Wormsgade

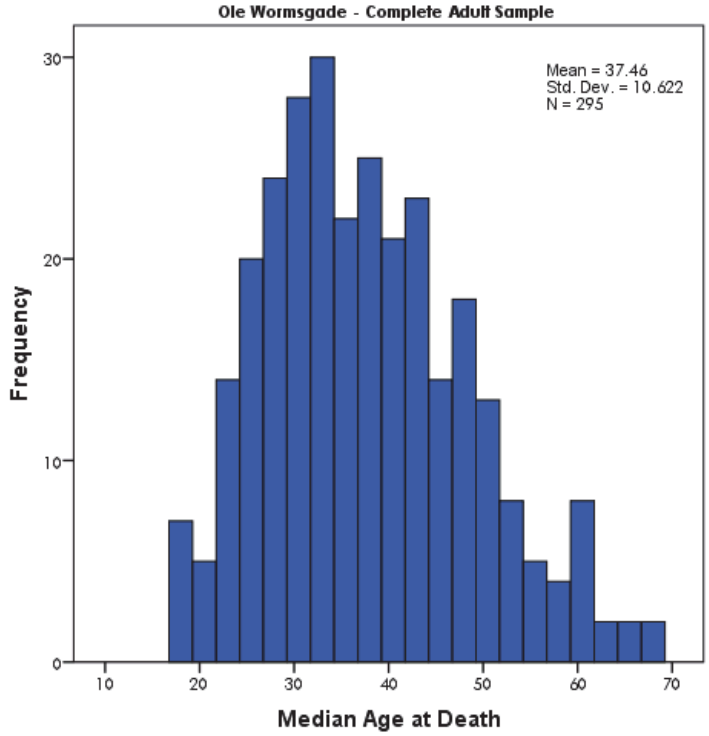
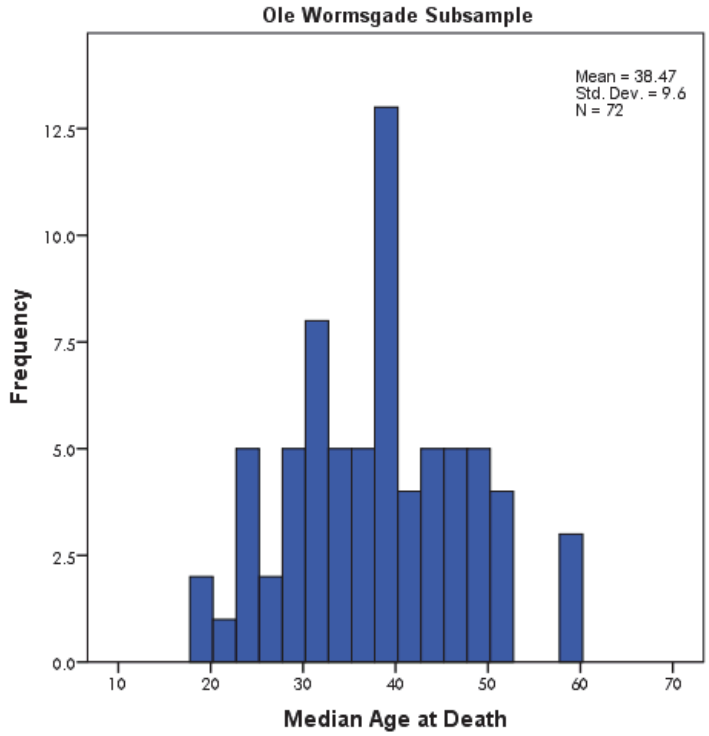


Figure 4-59 Frequency distribution of median age at death for the study subsample from Ole Wormsgade



Further consideration by sex shows that this reduced age in the subsample is present for both males and females at Sejet (Table 4-45). The slightly higher mean age at death in the subsample for Ole Wormsgade as opposed to the Sejet subsample is seen through this breakdown to result from a higher mean age at death in the female component, which is a particularly small subsample (N =29) (Table 4-45). Comparison of the frequency distributions, however, also shows that while the oldest ages are missing from the subsamples across the board, the individuals in these subsamples do represent the full spectrum of ages in other respects (Figure 4-60, Figure 4-61).

Table 4-45 Descriptive statistics detailing mean age at death for both sites in the complete cemeteries and cemetery subsamples used for the current study

Cemetery	Sex	Complete Cemeteries			Cemetery Subsamples		
		N	Mean	Std. Deviation	N	Mean	Std. Deviation
Sejet	Male	*139*	*39.99*	*10.47*	*43*	*36.05*	*10.06*
	Female	*105*	*34.23*	*8.32*	*43*	*33.29*	*8.06*
Ole Wormsgade	Male	*171*	*39.02*	*10.70*	*41*	*39.46*	*9.34*
	Female	*107*	*35.12*	*9.95*	*29*	*37.83*	*9.87*

Figure 4-60 Frequency distribution of median age at death for the complete cemetery samples broken down by cemetery and sex

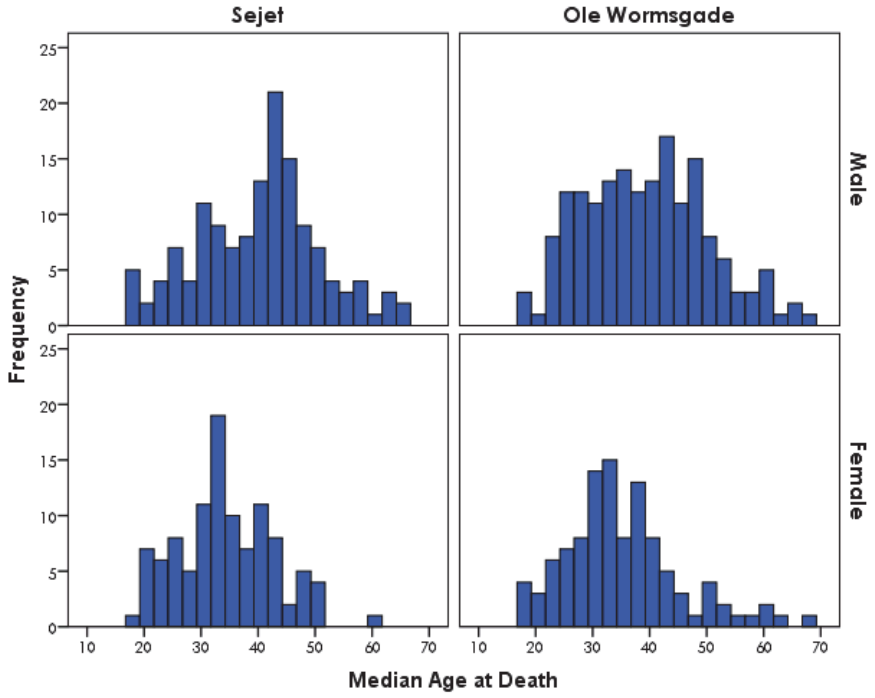
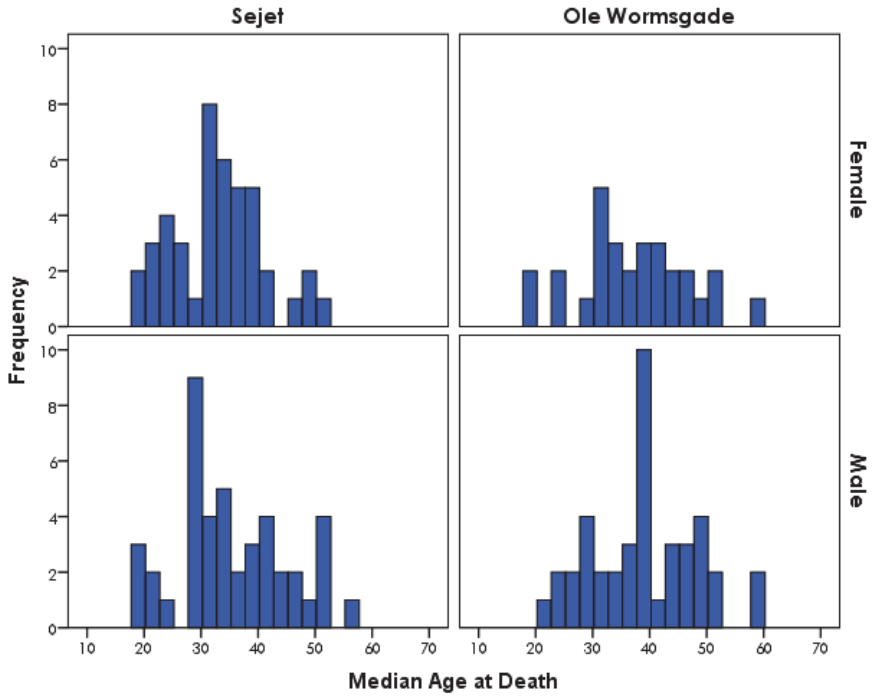


Figure 4-61 Frequency distribution of median age at death for the cemetery subsamples broken down by cemetery and sex



In sum, both subsamples show underrepresentation of the older individuals, but this is most pronounced at Sejet, leading to a statistically significant difference between the subsample used in this study and the larger adult cemetery sample. This makes further intersite comparison using the cemetery subsample problematic. The broader sample suggests that there is no statistically significant difference between sites for this health parameter, but as the data for the broader cemetery samples were not collected as part of this study, the scoring may not be entirely compatible.

Despite the limitations indicated from the above consideration, it is pertinent to engage in a consideration of age differences in these subsamples. Such an investigation will help to inform our understanding of health patterns within the subsample of individuals captured by this study, but caution must be exercised in extending these

results beyond the subsample. Adults in the Sejet subsample did show a significantly lower mean age at death ($\bar{x}$ = 34.80, N =93, sd = 8.931) than those from the Ole Wormsgade subsample ($\bar{x}$ = 38.47, N =72, sd = 9.6) (Table 4-46), with a small to medium effect size to this difference (d = 0.40, r = 0.19).

Table 4-46 Independent sample t-test for site differences in mean age at death (with ages at death for each individual based on the median for each age range)

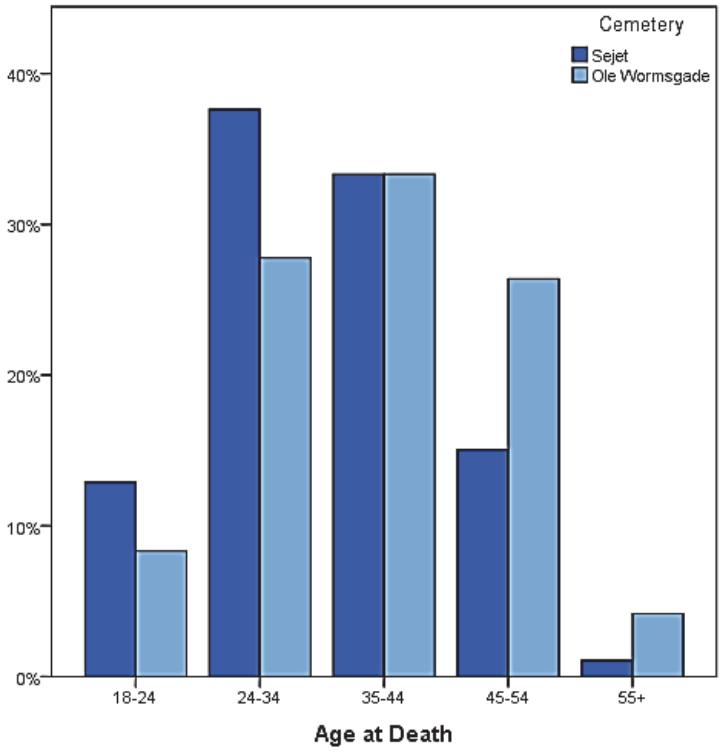
	Levene's Test for Equality of Variances		t-test for Equality of Means						
								95% Confidence Interval of the Difference	
Median Age at Death	F	Sig.	t	df	Sig. (2-tailed)	Mean Difference	Std. Error Difference	Lower	Upper
Equal variances assumed	*.563*	*.454*	*-2.529*	*163*	*.012*	*-3.664*	*1.449*	*-6.525*	*-.804*

When considered according to the separate age categories, the slight bias towards the younger age categories at Sejet can be further observed (Table 4-47, Figure 4-62). The highest percentage of the total sample captured at this site was in the 18 – 34 combined age groups. The proportion fell in the later categories, with particularly low representation in the oldest age group (at 5.9%). The Ole Wormsgade subsample shows a different pattern, being generally more evenly representative. The lowest percentage captured is once again the oldest, 55+ category at 15.2% of the complete sample. However, the second oldest category (45-54 years of age) is most strongly represented by this subsample (at 35.8% of the total sample).

Table 4-47 Comparison of age distribution by age category in the complete cemetery samples and in the cemetery subsamples included in this study

		Complete Samples		Subsamples		Percent of
Cemetery		Frequency	Percent	Frequency	Percent	Total
Sejet	18-24	43	6.8	12	12.9	27.9
	24-34	102	16.1	35	37.6	34.3
	35-44	143	22.6	31	33.3	21.7
	45-54	77	12.2	14	15.1	18.2
	55+	17	2.7	1	1.1	5.9
	Total	632	100	93	100.0	14.7
Ole Wormsgade	18-24	30	6.3	6	8.3	20.0
	24-34	99	20.9	20	27.8	20.2
	35-44	90	19.0	24	33.3	26.7
	45-54	53	11.2	19	26.4	35.8
	55+	23	4.9	3	4.2	13.0
	Total	474	100	72	100.0	15.2

Figure 4-62 Age distribution between sites

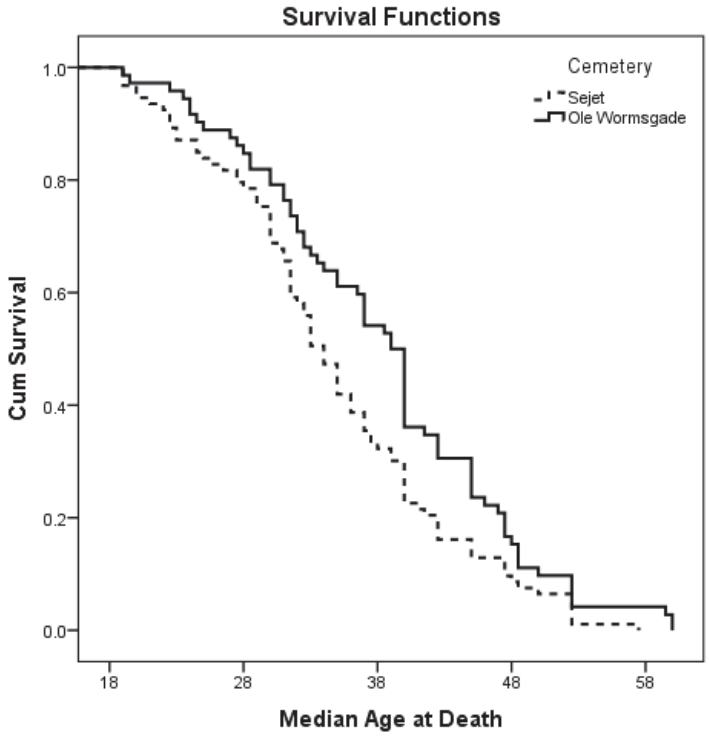


Kaplan-Meier survival analysis further illustrates this difference between the two site subsamples, with individuals from the Ole Wormsgade subsample surviving longer (Figure 4-63). This difference is once again seen to be statistically significant using a Mantel-Cox Log Rank test (Table 4-48).

Table 4-48 Mantel-Cox survival analysis testing the difference in median ages of death for individuals from the Sejet and Ole Wormsgade subsamples

	Chi-Square	df	Sig.
Log Rank (Mantel-Cox)	***5.821***	***1***	***.016***

Figure 4-63 Kaplan-Meier survival curve illustrating the difference in median ages of death for individuals from the Sejet and Ole Wormsgade subsamples

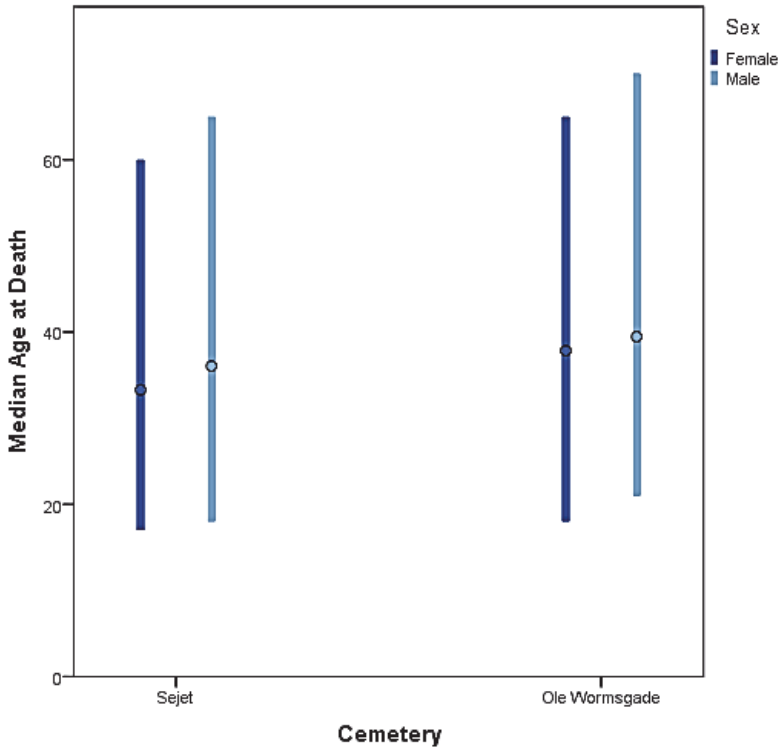


Further consideration shows this difference to be significant with females with a mean age at death of 33.29 years (N=43, sd=8.059) for Sejet and one of 37.83 years (N=29, sd=9.865) for those at Ole Wormsgade, and with a small effect size (d = 0.50, r = 0.24) (Table 4-49, Figure 4-64).

Table 4-49 Independent sample t-test for male and female site differences in mean age at death (with ages at death for each individual based on the median for each age range)

		Levene's Test for Equality of Variances		t-test for Equality of Means						
									95% Confidence Interval of the Difference	
		F	Sig.	t	df	Sig. (2-tailed)	Mean Difference	Std. Error Difference	Lower	Upper
Female	Equal variances assumed	*1.73*	*.19*	*-2.14*	*70*	*.04*	*-4.54*	*2.12*	*-8.77*	*-.31*
Male	Equal variances assumed	*.70*	*.41*	*-1.61*	*82*	*.11*	*-3.42*	*2.12*	*-7.64*	*.80*

Figure 4-64 High-low chart showing age ranges for males and females by site



Stature

Unfortunately, due to the nature of Ole Wormsgade cemetery, being along the course of roadwork where many of the burials were truncated, stature in the grave was unavailable for most of the individuals. This made sample sizes for this variable extremely small and limited any capacity to consider site differences (Table 4-51). Slightly more individuals had femur length available from Ole Wormsgade, but the sample sizes were still small (Table 4-52). In total, 64.7% of the individuals for whom femoral lengths could be measured in the broader cemetery were sampled for this investigation. This consisted of 81.1% of the available individuals from Sejet but only 34.7% of the available individuals from Ole Wormsgade (Table 4-50). A consideration of mean subsample femoral length in relation to the overall cemetery sample values

derived from the ADBOU databases showed no statistically significant differences. This suggests that the subsamples are representative, at least of the broader cemetery samples.

Table 4-50 Descriptive statistics comparing femoral lengths in the complete cemetery samples to that in the subsamples used for this study

Complete Cemetery Samples	N	Mean	Std. Deviation
Sejet	*90*	*45.29*	*3.02*
Ole Wormsgade	*49*	*45.87*	*3.14*
Total	*139*	*45.5*	*3.07*
Cemetery Subsamples			
Sejet	*73*	*45.38*	*3.22*
Ole Wormsgade	*17*	*46.27*	*3.52*
Total	*90*	*45.55*	*3.28*

Neither stature in the grave nor femoral length metrics indicated any significant difference between the two sites with the subsamples for either sex using an independent samples t-test, although the effect size between sites for males was strong ($d = 0.93$ and $r = 0.42$ for stature in the grave and $d = 0.42$ and $r = 0.21$ for femur length). Residual stature was uninformative due to the particularly small sample from Ole Wormsgade. The distribution of stature in the grave on one hand and femoral length on the other between both sites can be observed in Figure 4-65 and Figure 4-66.

Table 4-51 Descriptive statistics for male and female stature in the grave measurements by site

Sex		Cemetery	N	Mean	Std. Deviation	Std. Error Mean
Female	Stature in the Grave	Sejet	*37*	*155.4730*	*6.47854*	*1.06507*
		Ole Wormsgade	*3*	*155.5000*	*8.18535*	*4.72582*
Male	Stature in the Grave	Sejet	*31*	*171.0968*	*6.75329*	*1.21293*
		Ole Wormsgade	*6*	*165.3333*	*5.60060*	*2.28643*

Table 4-52 Descriptive statistics for male and female femoral length measurements by site

Sex		Cemetery	N	Mean	Std. Deviation	Std. Error Mean
Female	Average Femur	Sejet	32	43.0703	1.91281	.33814
		Ole Wormsgade	7	43.3643	2.33501	.88255
Male	Average Femur	Sejet	35	47.7443	2.41894	.40888
		Ole Wormsgade	9	48.7667	2.39505	.79835

Figure 4-65 Boxplot showing stature in the grave between sites for males and females

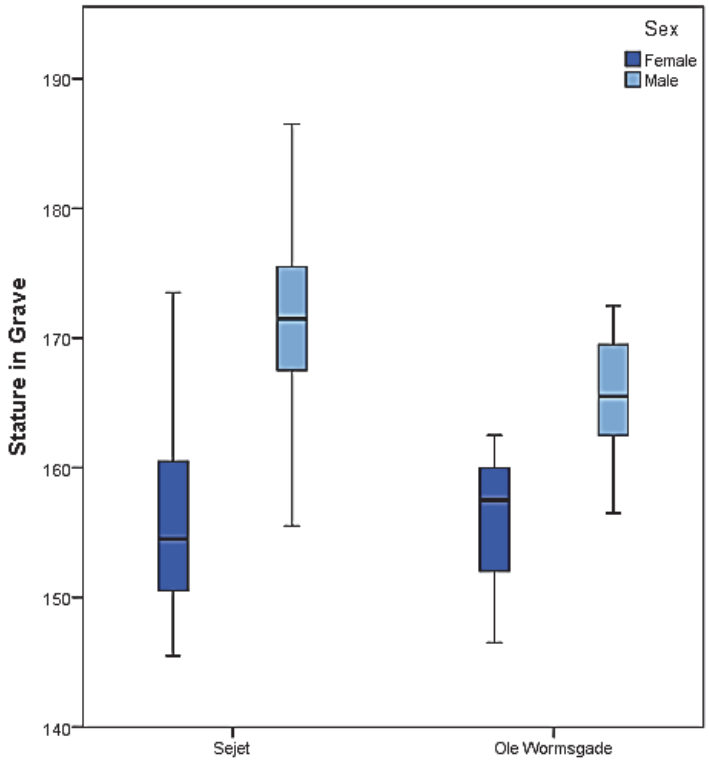
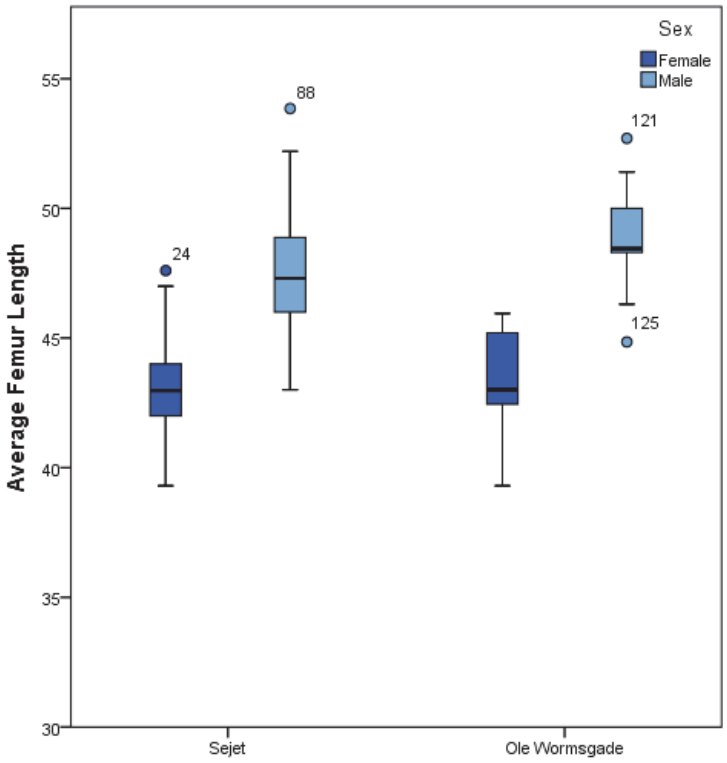


Figure 4-66 Boxplot showing femur length between sites for males and females



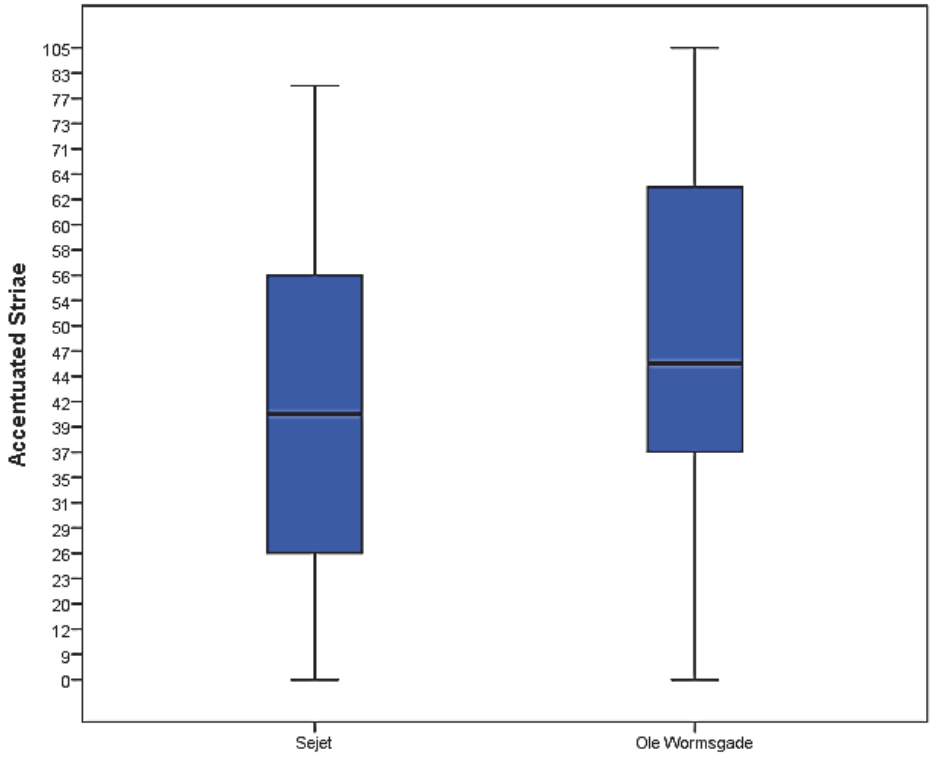
Stress

The sample from Ole Wormsgade had a higher mean number of AS ($\bar{x}$ = 48.49, N =37, sd = 23.224) than the Sejet sample ($\bar{x}$ = 39.4, N =42, sd = 20.24) (Table 4-53, Figure 4-67), but this difference was not found to be statistically significant using an independent samples t-test (F = 0.354, df = 77, p = 0.07) (although the effect size was large with d = 0.81 and r = 0.37).

Table 4-53 Descriptive statistics comparing mean number of AS between the two subsamples

	N	Mean	Std. Deviation
Sejet	*42*	*39.4*	*20.24*
Ole Wormsgade	*37*	*48.49*	*23.224*

Figure 4-67 Boxplot showing the median and range for AS counts between sites

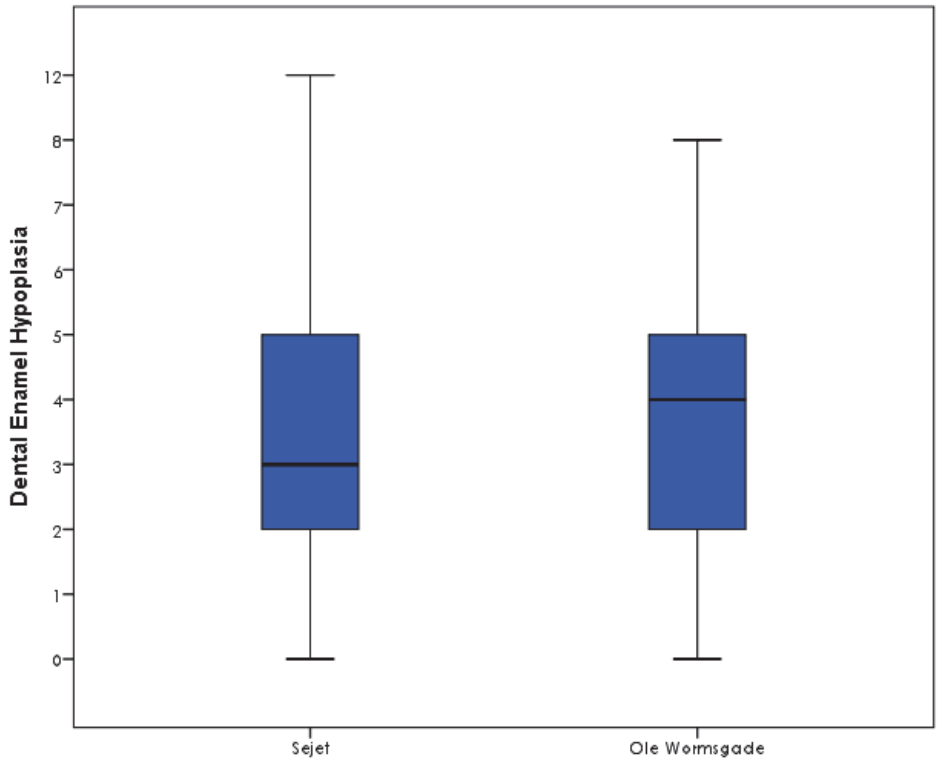


Further consideration of AS frequency in males and females considered separately also showed no statistically significant difference between those at Sejet and those at Ole Wormsgade. Similarly, the consideration of surface defects (Table 4-54, Figure 4-68) did not show any statistically significant difference between sites ($F = 0.202$, $df = 121$, $p = 0.862$). However, there were fewer individuals from Ole Wormsgade (N =46) than from Sejet (N =77) who could be scored for surface defects, and so any results should be treated with caution. No direct comparison could be made with the broader cemetery samples, as no compatible stress scores were available for these larger samples.

Table 4-54 Descriptive statistics comparing mean number of DEH between the two subsamples

	N	Mean	Std. Deviation
Sejet	*77*	*3.68*	*2.118*
Ole Wormsgade	*46*	*3.61*	*2.016*

Figure 4-68 Boxplot showing the median and range for DEH counts between sites



Disease

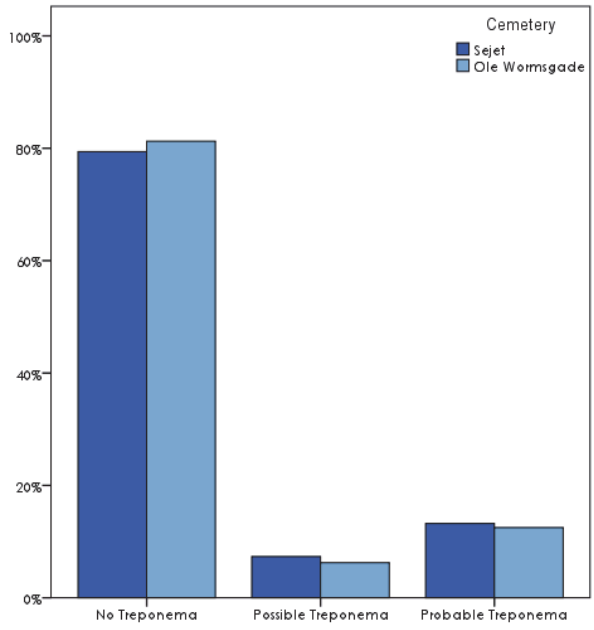
Treponema

Overall, individuals in the subsample from Sejet (13.24%, N =9) were diagnosed slightly more frequently with treponema than those from Ole Wormsgade (12.5%, N =2) (Table 4-55, Figure 4-69). This difference was not significant using Fisher's Exact test (p=0.651, odds ratio=0.937).

Table 4-55 Frequency of probable and possible treponemal cases for Sejet and Ole Wormsgade, combined with percent of cases as a factor of total scorable for treponema at each site[29]

	Sejet		Ole Wormsgade		
	Frequency	Percentage	Frequency	Percentage	Total
No Treponema	54	***79.41***	13	***81.25***	***47***
Possible Treponema	6	***8.82***	1	***6.25***	***27***
Probable Treponema	9	***13.24***	2	***12.50***	***34***
Total	68	***80.95***	16	***19.05***	***108***

Figure 4-69 Treponema rates by site



[29] Once again, a case was determined as probable if the characteristic indicators were visible. A possible diagnosis was given in cases where lesions existed that were consistent with the disease but where a higher level of uncertainty existed due to missing or poorly preserved elements. No disease refers to cases with sufficient preservation of elements but with elements exhibiting no evidence for a diseased state.

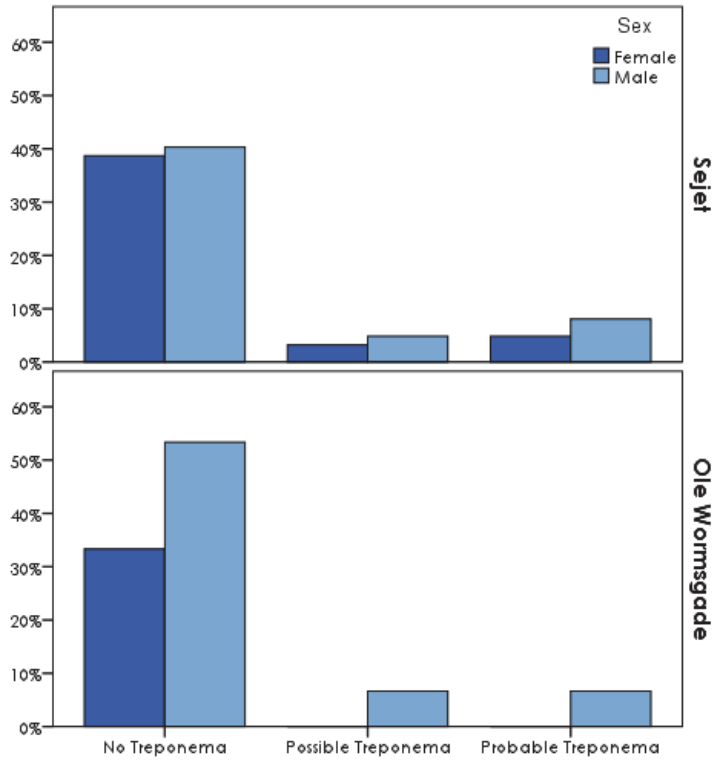
When the sexes were considered separately, cases of probable treponematosis were higher at Sejet for both males and females (Table 4-56, Figure 4-70). This difference was not statistically significant using a Fisher's Exact test ($p = 0.611$ for females and $p = 0.571$ for male), but once again small sample sizes impede the extent to which conclusions can be drawn.

Table 4-56 Frequency of treponematosis by sex from Sejet and Ole Wormsgade and percentage expressed in relation to total scorable individuals from each site[30]

		Sejet		Ole Wormsgade	
		Frequency	Percentage	Frequency	Percentage
Female	No Treponema	24	*38.71*	5	*33.33*
	Possible Treponema	2	*3.23*	0	*0*
	Probable Treponema	3	*4.84*	0	*0*
	Total	29	*46.77*	5	*33.33*
Male	No Treponema	25	*40.32*	8	*53.33*
	Possible Treponema	3	*4.84*	1	*6.67*
	Probable Treponema	5	*8.06*	1	*6.67*
	Total	33	*53.23*	10	*66.67*

[30] Once again, a case was determined as probable if the characteristic indicators were visible. A possible diagnosis was given in cases where lesions existed that were consistent with the disease but where a higher level of uncertainty existed due to missing or poorly preserved elements. No disease refers to cases with sufficient preservation of elements but with elements exhibiting no evidence for a diseased state.

Figure 4-70 Treponematosis by sex from Sejet and Ole Wormsgade and percentage expressed in relation to total individuals who could be scored for treponema from each site



A few notes on differential site preservation which could impact scores for treponematosis are pertinent to any interpretation of these results. Specifically, the truncation of many of the Ole Wormsgade burials could restrict ability to capture some of the characteristic changes of treponema which typically occur in the lower limbs (ex. saber shin). The selection of this sample for teeth meant that individuals who were truncated at the lower limbs (leaving the cranium intact) tended to be selected. Individuals at Ole Wormsgade were typically truncated so that either the upper portion or the lower portions of their bodies were preserved, but not the corresponding half. While overall quality of preservation at Ole Wormsgade was higher, this could have skewed results towards artificially higher prevalence rates at Sejet. As the results indicate, the

occurrence of treponema was slightly lower at Ole Wormsgade. This could mean that the urban sample did indeed suffer slightly less from this disease (although the differences are negligible). It could also mean that the scores for Ole Wormsgade were artificially supressed due to the particular conditions of preservation at this site.

Tuberculosis

The rates of both possible and probable tuberculosis were also higher at Sejet (N=23, 52.27% and N=9, 20.45% respectively) than at Ole Wormsgade (N=19, 40.43% and N=2, 4.46% respectively) (Figure 4-71, Table 4-57, Table 4-59) and this difference was found to be statistically significant for probable cases using a Fisher's Exact test (p=0.019) (Table 4-58).

Table 4-57 Frequency of probable and possible tuberculosis cases for Sejet and Ole Wormsgade, combined with percent of cases as a factor of total scorable for treponema at each site[31]

	Sejet		Ole Wormsgade		
	Frequency	Percentage	Frequency	Percentage	Total
No TB	12	***27.27***	26	***55.32***	***38***
Possible TB	23	***52.27***	19	***40.43***	***42***
Probable TB	9	***20.45***	2	***4.26***	***11***
Total	44	***48.35***	47	***51.65***	***91***

[31] Once again, a case was determined as probable if the characteristic indicators were visible. A possible diagnosis was given in cases where lesions existed that were consistent with the disease but where a higher level of uncertainty existed due to missing or poorly preserved elements. No disease refers to cases with sufficient preservation of elements but with elements exhibiting no evidence for a diseased state.

Figure 4-71 Tuberculosis rates by site

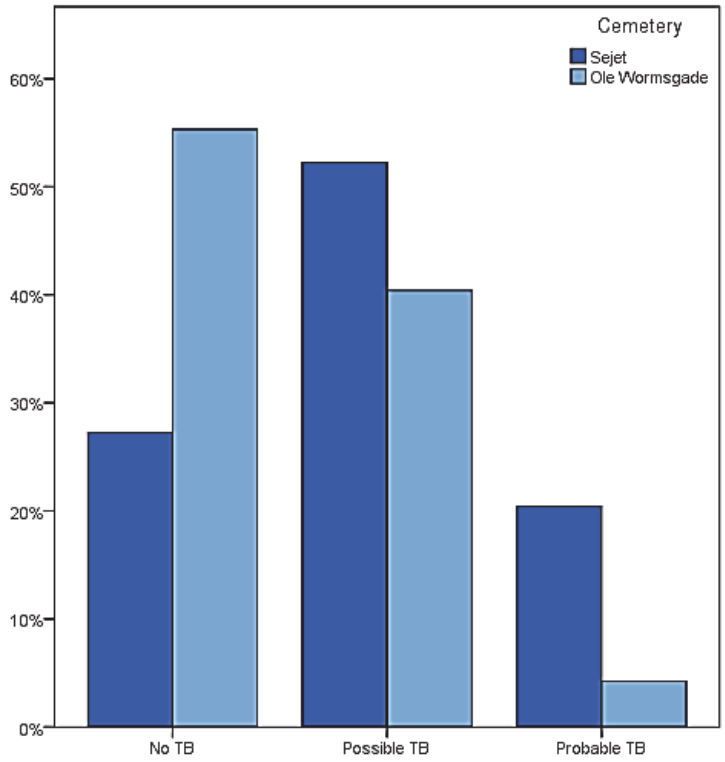


Table 4-58 Chi-Square test for probable tuberculosis rates for Sejet and Ole Wormsgade

	Value	df	Asymp. Sig. (2-sided)	Exact Sig. (2-sided)	Exact Sig. (1-sided)
Pearson Chi-Square	***5.612***[a]	***1***	***.018***		
Continuity Correction[b]	***4.191***	***1***	***.041***		
Likelihood Ratio	***5.973***	***1***	***.015***		
Fisher's Exact Test				***.024***	***.019***
Linear-by-Linear Association	***5.550***	***1***	***.018***		
N of Valid Cases	***91***				

Table 4-59 Risk estimate for probable tuberculosis between sites showing the odds ratio

	Value	95% Confidence Interval	
		Lower	Upper
Odds Ratio for Cemetery (Sejet / Ole Wormsgade)	***.173***	***.035***	***.851***
For cohort NewTB_OddsRatio = No Definite TB	***.831***	***.707***	***.976***
For cohort NewTB_OddsRatio = Probable TB	***4.807***	***1.099***	***21.031***
N of Valid Cases	***91***		

When the samples were considered seperately by sex, the same pattern persisted, being particularly pronounced in females (Figure 4-72). Females at Sejet appeared to have been almost three times more likely to show bony changes consistent with tuberculosis (Table 4-61). Once again, these differences were statistically significant (p=0.014) (Table 4-62).

Table 4-60 Frequency of tuberculosis by sex from Sejet and Ole Wormsgade and percentage expressed in relation to total individuals who could be scored for tuberculosis from each site[32]

		Sejet		Ole Wormsgade	
		Frequency	Percentage	Frequency	Percentage
Female	**No TB**	5	***12.5***	11	***23.91***
	Possible TB	9	***22.5***	7	***15.22***
	Probable TB	6	***15.0***	0	***0***
	Total	20	***50.0***	18	***39.13***
Male	**No TB**	7	***17.5***	14	***30.43***
	Possible TB	10	***25.0***	12	***26.09***
	Probable TB	3	***7.5***	2	***4.35***
	Total	20	***50.0***	28	***60.87***

[32] Once again, a case was determined as probable if the characteristic indicators were visible. A possible diagnosis was given in cases where lesions existed that were consistent with the disease but where a higher level of uncertainty existed due to missing or poorly preserved elements. No disease refers to cases with sufficient preservation of elements but with elements exhibiting no evidence for a diseased state.

Figure 4-72 Tuberculosis by sex from Sejet and Ole Wormsgade and percentage expressed in relation to total individuals who could be scored for tuberculosis from each site

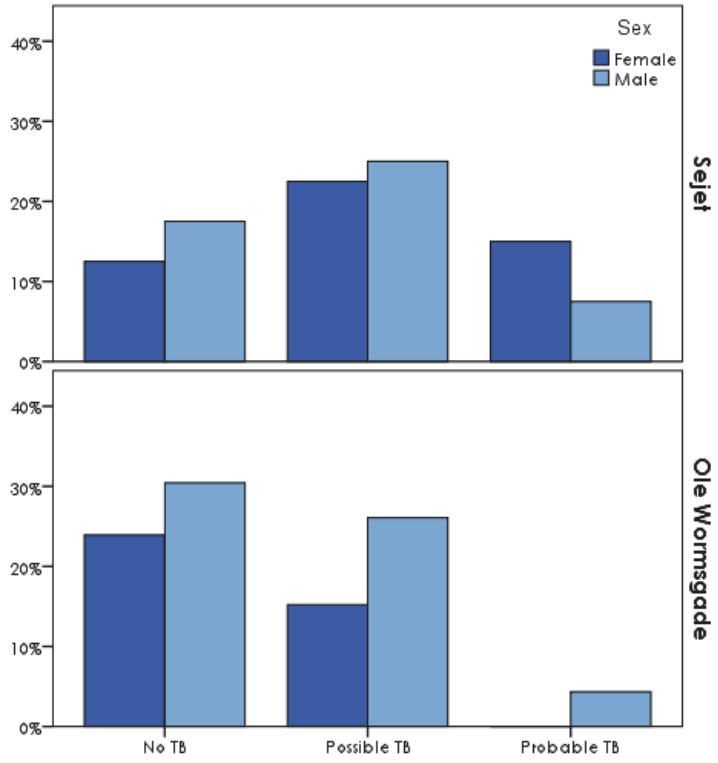


Table 4-61 Risk estimate for tuberculosis by sex between sites showing the odds ratio

Sex		Value	95% Confidence Interval	
			Lower	Upper
Female	For cohort NewTB_OddsRatio = No Definite TB	*.700*	*.525*	*.933*
	N of Valid Cases	*38*		
Male	Odds Ratio for Cemetery (Sejet / Ole Wormsgade)	*.436*	*.066*	*2.888*
	For cohort NewTB_OddsRatio = No Definite TB	*.915*	*.741*	*1.130*
	For cohort NewTB_OddsRatio = Probable TB	*2.100*	*.386*	*11.434*
	N of Valid Cases	*48*		
a. No statistics are computed because NewTB_OddsRatio is a constant.				

Table 4-62 Chi-Square test for tuberculosis rates by sex for Sejet and Ole Wormsgade

Sex		Value	df	Asymp. Sig. (2-sided)	Exact Sig. (2-sided)	Exact Sig. (1-sided)
Female	Pearson Chi-Square	6.413[a]	1	.011		
	Continuity Correction[b]	4.355	1	.037		
	Likelihood Ratio	8.714	1	.003		
	Fisher's Exact Test				.021	.014
	Linear-by-Linear Association	6.244	1	.012		
	N of Valid Cases	38				
Male	Pearson Chi-Square	.772[c]	1	.380		
	Continuity Correction[b]	.159	1	.690		
	Likelihood Ratio	.760	1	.383		
	Fisher's Exact Test				.636	.340
	Linear-by-Linear Association	.756	1	.385		
	N of Valid Cases	48				

These differences in prevalence rates are interesting, particularly in light of the fact that the quality of preservation of such elements as the vertebrae was very poor at Sejet. At Ole Wormsgade, however, truncation may have resulted in underscoring with fewer postcranial elements being captured in this study. While the number of probable cases was still small, the site differences are robust enough to suggest that there may have been some real difference in prevalence rates for this disease. However, it should be noted once again that diagnosis of tuberculosis can be highly problematic, with few cases actually manifesting in bony changes, and with other conditions expressing similar bony changes (Steinbock, 1976; Aufderheide and Rodriguez-Martin, 1998; Ortner, 2003; Roberts and Buikstra, 2003; Mays, 2007; Mutolo et al., 2012). It is likely that rates of tuberculosis were higher at both sites. Due to differential patterns and quality of preservation between the two sites, the results should be treated with caution. However, given the statistically significant higher rates at Sejet despite poor quality of preservation,

it may be that tuberculosis was more common, or more likely to manifest skeletally amongst individuals of this population.

Leprosy

As has already been noted, the analysis of leprosy prevalence rates was conducted in quite a different fashion, using lambda calculations based on the work of Boldsen et al. (2013) and Boldsen and Freund (2006). The scores for this disease are thus not directly comparable to those from the other disease, and probably more accurately capture disease prevalence rates in these populations. For comparative purposes, the basic descriptive statistics derived from the raw lambda scores can be used to show that rates of probable cases at Sejet were higher than those at Ole Wormsgade (Table 4-63, Figure 4-73). A Fisher's Exact test shows this to be significant for both sexes combined (N =138, df = 1, p = 0.007) (Table 4-64).

Table 4-63 Frequency of leprosy at Sejet and Ole Wormsgade and percentage expressed in relation to total scorable individuals from each site. These counts are based on the raw lambda scores which are calculated from the direct feature scores (and which integrate specificity and sensitivity, as opposed to the derived lambda statistics which integrate variance and mean lambda scores to derive a population distribution. This process is described further in the methods chapter under the statistical analysis section.[33]

	Sejet		Ole Wormsgade		
	Frequency	Percentage	Frequency	Percentage	Total
No Leprosy	22	***29.33***	38	***60.32***	***60***
Possible Leprosy	22	***29.33***	12	***19.05***	***34***
Probable Leprosy	31	***41.33***	13	***20.63***	***44***
Total	75	***61.48***	63	***45.65***	***138***

[33] Once again, in the case of leprosy diagnosis, any individuals who had λ of between -1 and 1 were classed as possible cases, while individuals with scores equal to or greater than 1 were classed as probable cases (Boldsen, 2008). Individuals below -1 were categorized as unlikely to have suffered from leprosy (Boldsen, 2008).

Figure 4-73 Leprosy by sex from Sejet and Ole Wormsgade and percentage expressed in relation to total individuals who could be scored for leprosy from each site. Percentages are based on the raw lambda scores, as opposed to the derived lambda statistics.

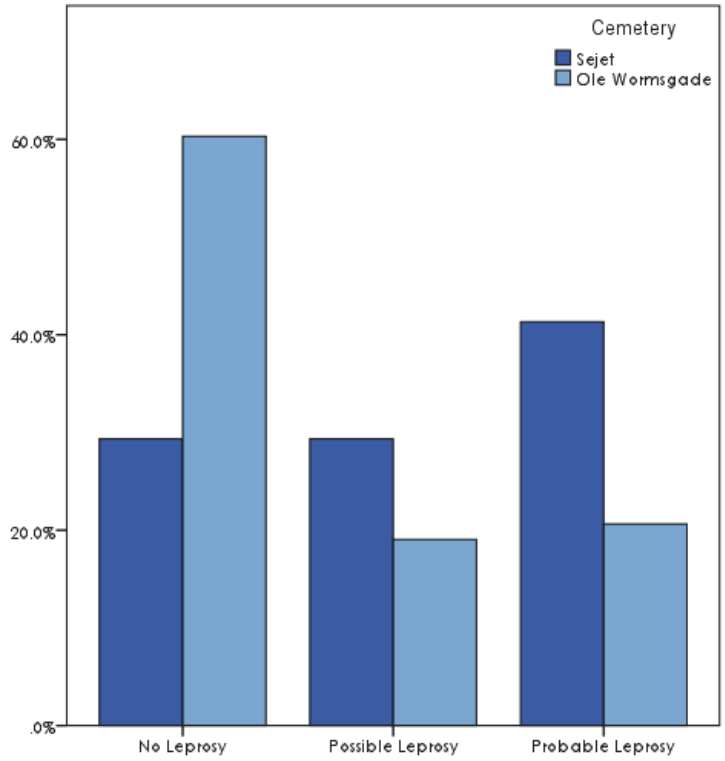


Table 4-64 Chi-Square test for probable leprosy rates for Sejet and Ole Wormsgade

	Value	df	Asymp. Sig. (2-sided)	Exact Sig. (2-sided)	Exact Sig. (1-sided)
Pearson Chi-Square	*6.754*[a]	*1*	*.009*		
Continuity Correction[b]	*5.835*	*1*	*.016*		
Likelihood Ratio	*6.923*	*1*	*.009*		
Fisher's Exact Test				*.011*	*.007*
Linear-by-Linear Association	*6.705*	*1*	*.010*		
N of Valid Cases	*138*				

When broken down by sex, the descriptive statistics in which probable and possible cases were identified from lambda scores indicate that the pattern of higher rates of leprosy in the Sejet subsample than in the Ole Wormsgade subsample is consistent for both males and females (Table 4-65, Figure 4-74). A Fisher's Exact test shows that these differences are significant in both cases (Table 4-66).

Table 4-65 Frequency of leprosy by sex from Sejet and Ole Wormsgade and percentage expressed in relation to total scorable individuals from each site. These counts are based on the raw lambda scores, as opposed to the derived statistics[34]

		Sejet		Ole Wormsgade	
		Frequency	Percentage	Frequency	Percentage
Female	No Leprosy	11	*12.5*	14	*20.0*
	Possible Leprosy	21	*23.86*	12	*17.14*
	Probable Leprosy	11	*12.5*	3	*4.29*
	Total	43	*48.86*	29	*41.43*
Male	No Leprosy	10	*11.36*	23	*32.86*
	Possible Leprosy	16	*18.18*	10	*14.29*
	Probable Leprosy	19	*21.59*	8	*11.43*
	Total	45	*51.14*	41	*58.57*

[34] Once again, in the case of leprosy diagnosis, any individuals who had λ of between -1 and 1 were classed as possible cases, while individuals with scores equal to or greater than 1 were classed as probable cases (Boldsen, 2008). Individuals below -1 were categorized as unlikely to have suffered from leprosy (Boldsen, 2008).

Figure 4-74 Leprosy by sex from Sejet and Ole Wormsgade and percentage expressed in relation to total scorable individuals from each site (based on raw lambda scores)

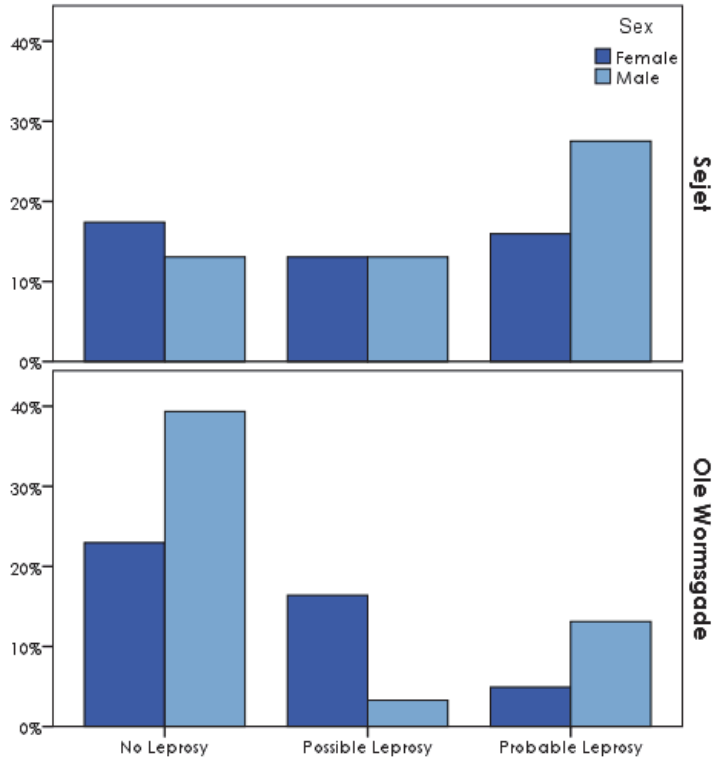


Table 4-66 Chi-Square test for probable leprosy rates for males and females at Sejet and Ole Wormsgade

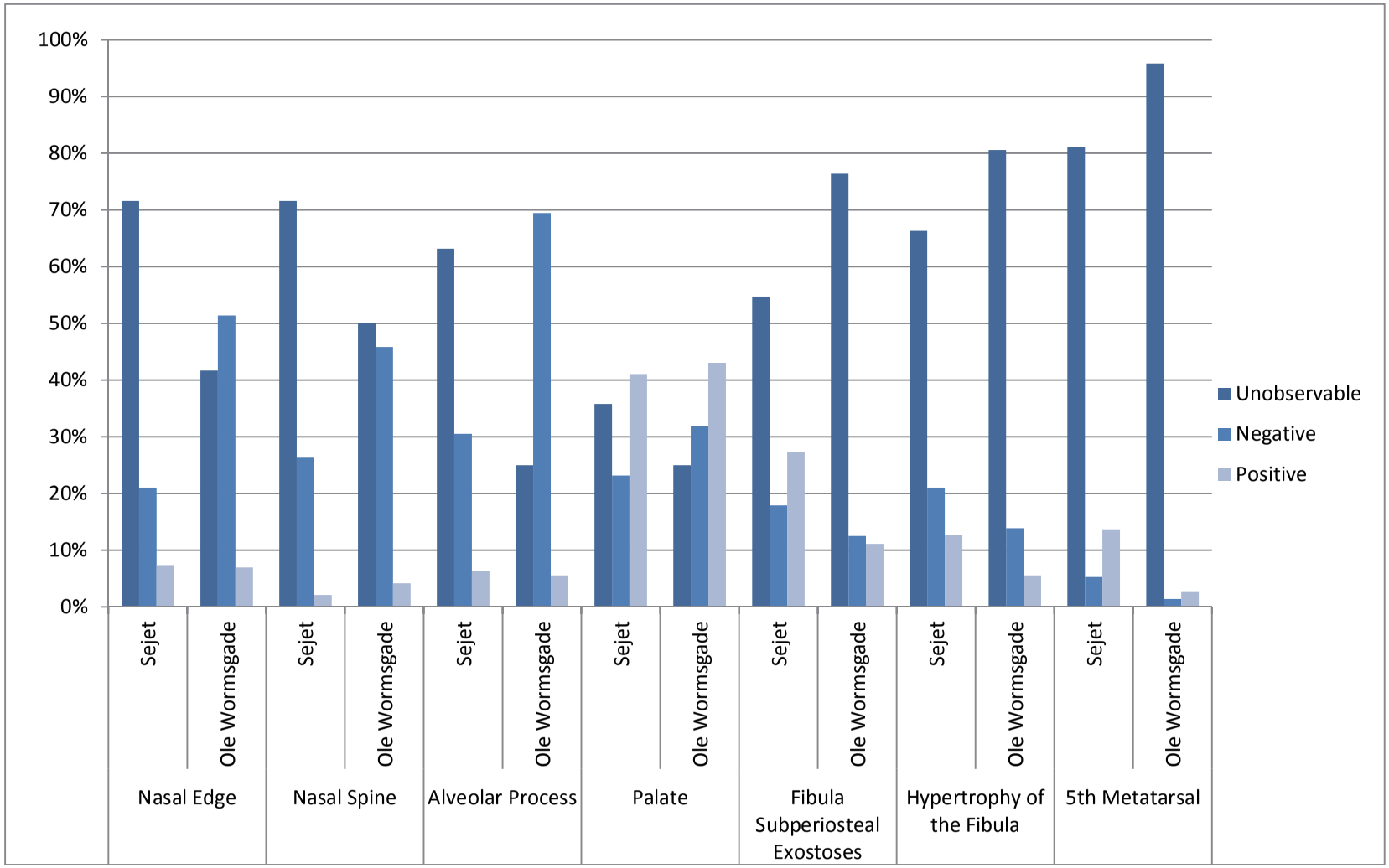
Sex		Value	df	Asymp. Sig. (2-sided)	Exact Sig. (2-sided)	Exact Sig. (1-sided)
Female	Pearson Chi-Square	4.379[a]	1	.036		
	Continuity Correction[b]	3.188	1	.074		
	Likelihood Ratio	4.636	1	.031		
	Fisher's Exact Test				.063	.035
	Linear-by-Linear Association	4.305	1	.038		
	N of Valid Cases	59				
Male	Pearson Chi-Square	5.820[c]	1	.016		
	Continuity Correction[b]	4.699	1	.030		
	Likelihood Ratio	5.950	1	.015		
	Fisher's Exact Test				.027	.015
	Linear-by-Linear Association	5.738	1	.017		
	N of Valid Cases	71				

A cautionary note with these results is once again necessary, as these scores may be somewhat problematic due to the differential preservation at both sites. Since many of the Ole Wormsgade burials were truncated, and since this study required cranial material to be present for the dental component, many individuals in the Ole Wormsgade sample did not have their lower limbs preserved, and so scores for the fibula and metatarsals were not available. The result of this differential preservation can be seen in Table 4-67 and Figure 4-75 with lower scores for postcranial material for Ole Wormsgade. The lower λ for Ole Wormsgade may be partly reflecting this differential preservation, with scores being lower simply because the postcranial elements were not available. Sejet also shows high scores for palate changes and fibula exostoses. This pattern requires further exploration with the larger cemetery sample to gain further insight into its implications.

Table 4-67 Lesion scores of lambda statistic between sites

Lesion	Site	Unobservable	Negative	Positive	Total
Nasal Edge	Sejet	68	20	7	95
	Ole Wormsgade	30	37	5	72
Nasal Spine	Sejet	68	25	2	95
	Ole Wormsgade	37	30	5	72
Alveolar Process	Sejet	60	29	6	95
	Ole Wormsgade	18	50	4	72
Palate	Sejet	34	22	39	95
	Ole Wormsgade	18	23	31	72
Fibula Subperiosteal Exostoses	Sejet	52	17	26	95
	Ole Wormsgade	55	9	8	72
Hypertrophy of the Fibula	Sejet	63	20	12	95
	Ole Wormsgade	58	10	4	72
5^{th} Metatarsal	Sejet	77	5	13	95
	Ole Wormsgade	69	1	2	72

Figure 4-75 Distribution of lesion scores of lamba statistic between sites



Childhood stress incidence and adult health

The consideration of childhood stress incidence in relation to adult health parameters incorporates the complete subsample initially selected for this research and does not engage in any subsampling by site and time. As no statistically significant difference in stress was observed either in the temporal comparison or in the consideration of site differences, the lumping of this sample was justified. Furthermore, this consideration is focused on considering the biological association between stress and health. It is proposed that there will be a pattern in biological human response to stress experiences during development. The purpose of this section will be to gain insight into this pattern.

Enamel defects and longevity

No association was found between adult mean age at death and the number of stress events as represented by accentuated striae (AS) in the overall sample. When sexes were considered separately, however, a slight difference in this association was apparent between males and females. Figure 4-76 and Figure 4-77 show the correlation of these two factors. The scatter-plots used median age at death (as the median for the age range determined for each individual), while the high-low graphs were utilized to take the age ranges into account. These plots indicated a weak but nevertheless statistically significant positive correlation ($r = 0.357$, N =39, $p = 0.026$) in females between age at death and the number of AS, suggesting that females with more stress events early in life seemed to have lived longer (i.e. had a higher mean age at death). This same pattern was not visible for the males in the sample. While the individuals who had the highest age range in the male sample had low to moderate levels of stress and did not experience high levels of

stress, this association (which would be the opposite from that seen in females) showed extremely weak correlation which was not statistically significant (r = -0.209, N =35, p = 0.228). The effect size for both male (r = 0.21) and female (r = 0.36) correlations was, however, strong. Furthermore, the male trend seems to fluctuate and this may indicate a more complex relationship between AS and mortality. A consideration of median age at death in relation to AS counts in the different deciles showed no correlation between the two variables at different points in enamel development.

Figure 4-76 High-low graph showing relationship between AS and age range

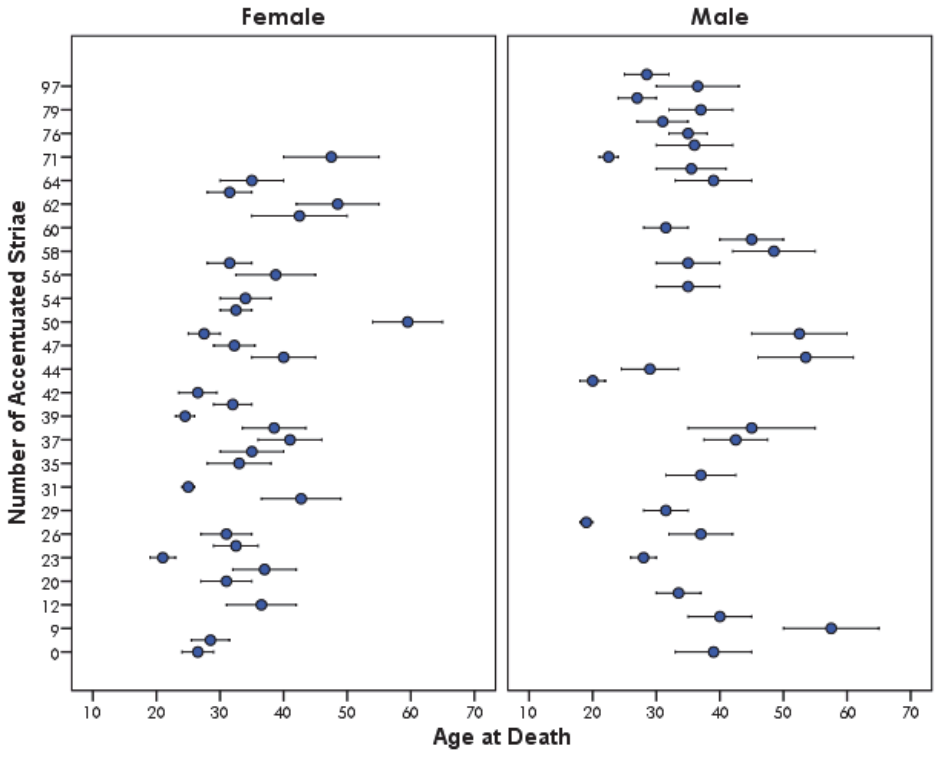
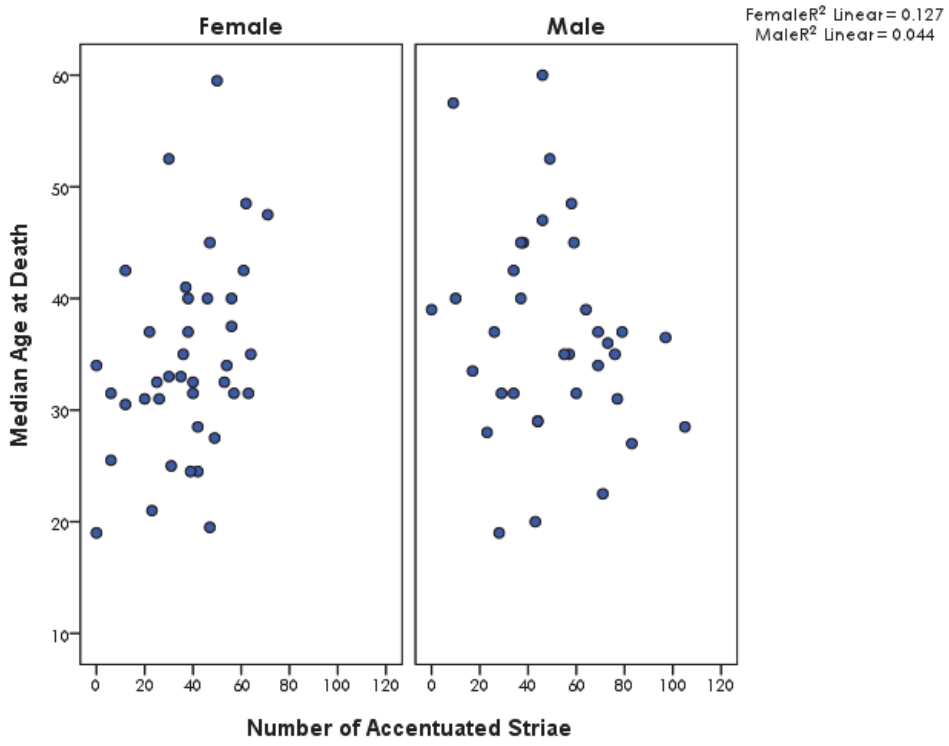


Figure 4-77 Scatter plot showing relationship between AS and mean ages at death

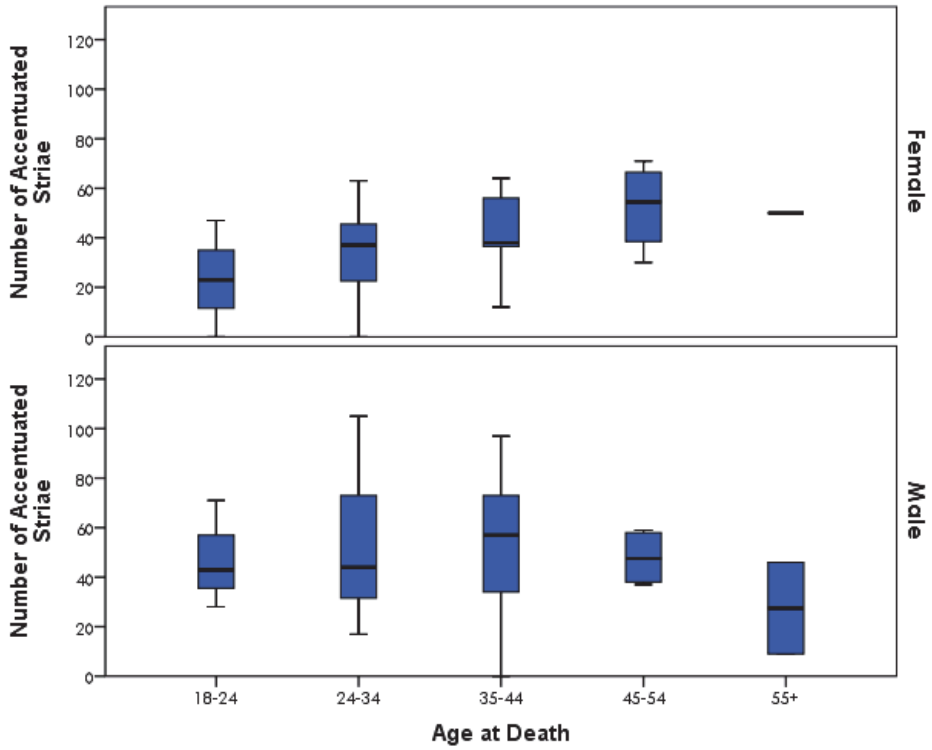


In order to test these patterns further, a one-way ANOVA was conducted both for the broader sample and for males and females seperately. For this, individuals were broken into four age categories based on their median age at death (18-24, 25-34, 35-44, 45-50, and 55+ years) (Table 4-68, Figure 4-78). No significant association was found between any of the adult age categories and the number of accentuated striae for either the combined sample or when separated by sex.

Table 4-68 Descriptive statistics highlighting sample size for thin sectioned material in each age group and the percentage of individuals captured by thin sectioning in relation to total subsample used for this study

Age Category	N	% Thin Sectioned	Mean	Std. Deviation
18-24	*6*	*3.6*	*35.33*	*24.17*
25-34	*33*	*19.8*	*40.42*	*22.85*
35-44	*25*	*15.0*	*47.44*	*23.42*
45-54	*10*	*6.0*	*49.70*	*12.77*
55+	*3*	*1.8*	*35.00*	*22.61*
Total	*77*	*46.1*	*43.30*	*22.06*

Figure 4-78 Accentuated striae of Retzius distribution by age group



Finally, in order to consider whether the number of AS related to survival to a certain age, Kaplan-Meier survival analysis was used to plot severity of AS against adult median age at death. For this, the samples were divided into three groups based on the number of accentuated striae they possessed (minimal = 0-30, moderate = 31-58, and severe = 59+). Categories were defined based on the interquartile distribution of AS in the sample (Table 4-69).

Table 4-69 Descriptive Statistics for AS Categories. Percentage within each sex reflects the percentage of individuals in each category in relation to the total number of individuals from the respective sex with AS counts available. Cumulative percentage is calculated in relation to the total number of individuals available for AS (combined sex).

Sex		Frequency	Percent (Within Each Sex)	Cumulative Percent
Female	0-30	*13*	*33.3*	*17.1*
	31-58	*21*	*53.8*	*27.6*
	59+	*5*	*12.8*	*6.6*
	Total	*39*	*100.0*	*51.3*
Male	0-30	*8*	*21.6*	*10.5*
	31-58	*15*	*40.5*	*19.7*
	59+	*14*	*37.8*	*18.4*
	Total	*37*	*100.0*	*48.7*

No clear pattern or statistically significant correlation was visible between the number of stress events and median age at death for the complete sample. The results of the log rank tests showed a slightly different relationship between males and females, with more stress in females being correlated with a slightly higher median age at death, while the correlation with males was neutral to negative. Neither of these patterns were statistically significant (Table 4-70).

Table 4-70 Mantel-Cox survival analysis testing the relationship between the frequency of accentuated striae of Retzius and median age at death by sex

Sex		Chi-Square	df	Sig.
Female	Log Rank (Mantel-Cox)	*2.396*	*2*	*.302*
Male	Log Rank (Mantel-Cox)	*4.780*	*2*	*.092*
Test of equality of survival distributions for the different levels of Level of Stress2.				

Figure 4-79 Kaplan-Meier survival curve representing the relationship between median age at death and the frequency of accentuated striae of Retzius for females

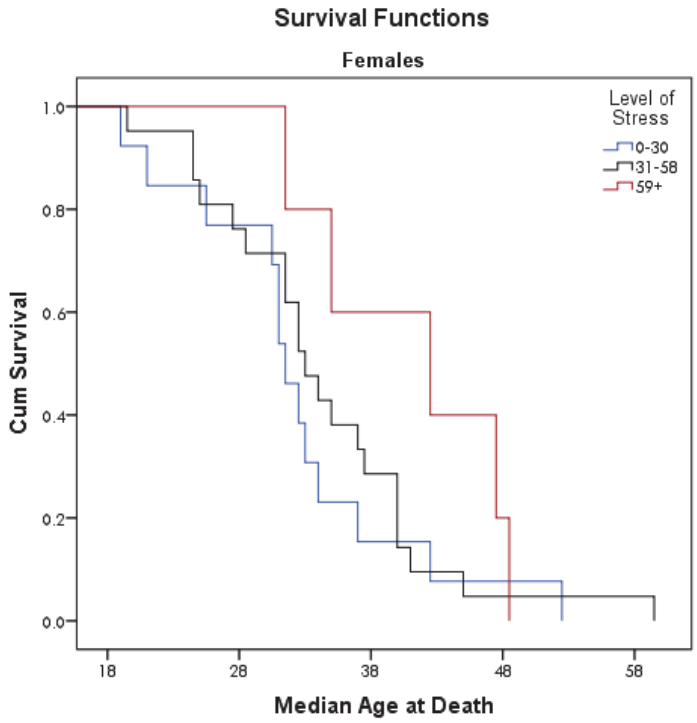
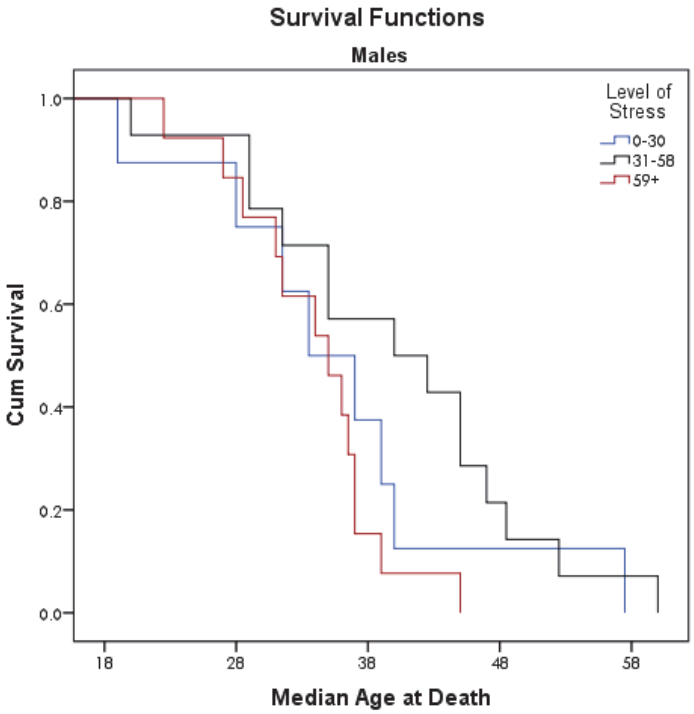


Figure 4-80 Kaplan-Meier survival curve representing the relationship between median age at death and the frequency of accentuated striae of Retzius for males



Sample sizes were too small for extensive consideration by decile, but AS in each decile were considered in relation to median age at death. The age ranges attributed to each decile can be reviewed in Table 4-71, as derived from Reid and Dean (2006). The way in which AS were counted in each decile can also been seen in Figure 3-14. Each decile received its own unique level attribution of minimal or severe AS based on the frequency distribution of AS rates for that decile. Individuals in the upper quartile of the frequency distribution for each decile were assigned to the severe stress category (Table 4-72). No decile showed significant differences in survivorship between individuals scored with severe versus minimal AS in the overall subsample.

Table 4-71 Age ranges for each decile, based on Tirup sample as investigated by Reid and Dean (2006)

Decile Number	Minimum Age	Maximum Age
1	*1.5*	*1.7*
2	*1.7*	*2.0*
3	*2.0*	*2.3*
4	*2.3*	*2.7*
5	*2.7*	*3.1*
6	*3.1*	*3.6*
7	*3.6*	*4.2*
8	*4.2*	*4.9*
9	*4.9*	*5.6*
10	*5.6*	*6.2*

Table 4-72 Frequencies and interquartile ranges for AS rates in each decile. The frequency column reflects the number of individuals for which counts were available in each decile. The mean column reflects the mean number of AS counts across the sample for each decile and the standard deviation column reflects the standard deviation of counts in each decile. The percentile columns reflect the frequency distribution of the AS scores, showing the cut-off points / thresholds used to determine levels of stress for each individual. Individuals with low stress had AS counts ranging from the 25th percentile value to the 50th percentile value. Individuals with moderate stress had AS counts covering the second quartile (50%) through to the upper quartile (75%). Individuals with severe stress had AS counts in the upper quartile range for the frequency distribution (above 75%).

				Percentiles		
Decile	Frequency	Mean	s.d.	25	50	75
1st	*25*	*5.19*	*5.74*	*0*	*5*	*6.25*
2nd	*61*	*3.99*	*4.50*	*0*	*3.33*	*6.67*
3rd	*74*	*7.38*	*6.41*	*3.33*	*6.67*	*10*
4th	*76*	*9.11*	*6.00*	*5*	*7.5*	*12.5*
5th	*75*	*15.47*	*8.14*	*10*	*15*	*17.5*
6th	*76*	*14.06*	*8.27*	*8*	*13*	*20*
7th	*76*	*13.03*	*7.59*	*8.33*	*11.67*	*17.92*
8th	*72*	*12.18*	*7.40*	*7.14*	*11.43*	*17.14*
9th	*67*	*10.18*	*7.65*	*4.29*	*8.57*	*14.29*
10th	*56*	*8.86*	*6.50*	*3.33*	*8.33*	*11.67*

A consideration of sex differences in stress per decile revealed some interesting patterns. While females had more stress than males in the first decile, otherwise males consistently exhibited higher rates of stress (Table 4-73). Only the 6^{th} ($t = 2.307$, $df = 62.8$, $p = 0.024$) and 10^{th} ($t = 2.509$, $df = 49.4$, $p = 0.015$) deciles showed significant differences in mean AS rates (Table 4-73). It appears from these results that over the course of childhood, males suffered from higher rates of stress than females, with the exception to this being in the early infancy period (from approximately 1.5 to 1.7 years of age (Reid and Dean 2006).

Table 4-73 Frequencies and rates of stress per decile by sex

Variable	Sex	N	Mean	Std. Deviation	Variable	Mean	Std. Deviation
1st Decile	Male	13	0.92	0.76	1st Decile Rate	4.62	3.80
	Female	12	0.92	0.90		6.25	7.42
2nd Decile	Male	29	1.24	1.41	2nd Decile Rate	4.03	4.74
	Female	30	1.00	1.14		3.44	3.96
3rd Decile	Male	35	2.31	2.18	3rd Decile Rate	7.71	7.27
	Female	36	2.11	1.74		7.13	5.81
4th Decile	Male	36	4.00	2.65	4th Decile Rate	10.14	6.79
	Female	37	3.05	1.91		7.97	5.20
5th Decile	Male	36	6.78	3.52	5th Decile Rate	17.08	8.81
	Female	36	5.31	2.62		13.89	7.47
6th Decile	Male	36	8.08	4.63	6th Decile Rate*	16.28	9.28
	Female	37	5.86	3.26		11.91	6.65
7th Decile	Male	36	8.28	4.52	7th Decile Rate	14.10	8.06
	Female	37	7.08	4.08		12.23	7.37
8th Decile	Male	35	8.69	5.64	8th Decile Rate	12.60	8.26
	Female	34	8.09	4.49		12.07	6.73
9th Decile	Male	33	8.24	5.15	9th Decile Rate	12.00	7.68
	Female	31	5.90	5.26		8.64	7.54
10th Decile	Male	26	6.88	4.26	10th Decile Rate*	11.60	7.23
	Female	28	3.86	2.95		6.46	4.91

Kaplan-Meier survivor analysis was once again used to plot the relationship between the rates of AS in each decile and median age at death for males and females separately (Figure 4-81, Figure 4-82). This showed a broad pattern of females with more stress events per decile appearing to survive longer in many of the deciles (1st to 4th deciles, and the 7th and 8th deciles). Other deciles showed a less clear distinction between individuals with minimal versus severe AS. If a distinction was visible in the male subsample within the difference deciles, males with more stress tended not to survive as

long (Figure 4-82). This pattern is significant in males for the 8th decile (Table 4-74, Figure 4-81, and Figure 4-82). In females, a significant difference was seen in the 3rd decile, with the 7th decile also approaching significance (Table 4-75, Figure 4-83, and Figure 4-84). Further sampling would be required to gain greater insight into the extent to which this pattern reflects actual impacts of early life stress on mean age at death during different points in development.

Table 4-74 Mantel-Cox log rank test showing the relationship between the frequency of accentuated striae of Retzius in the 8th decile and median age at death by sex

Sex		Chi-Square	df	Sig.
Female	Log Rank (Mantel-Cox)	*3.475*	*1*	*.062*
Male	Log Rank (Mantel-Cox)	*6.025*	*1*	*.014*

Figure 4-81 Kaplan-Meier survival curve representing the relationship between median age at death and the frequency of accentuated striae of Retzius in the 8th decile for females

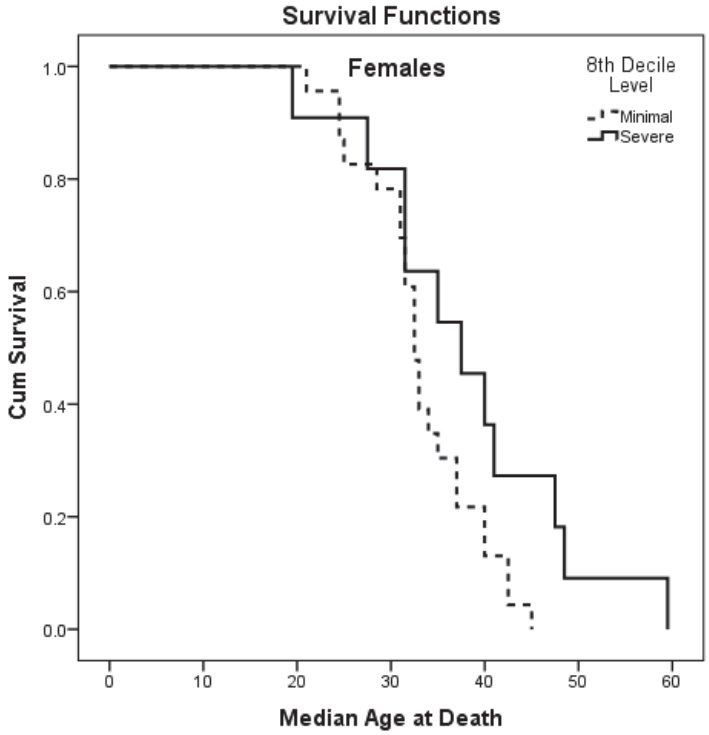


Figure 4-82 Kaplan-Meier survival curve representing the relationship between median age at death and the frequency of accentuated striae of Retzius in the 8th decile for males

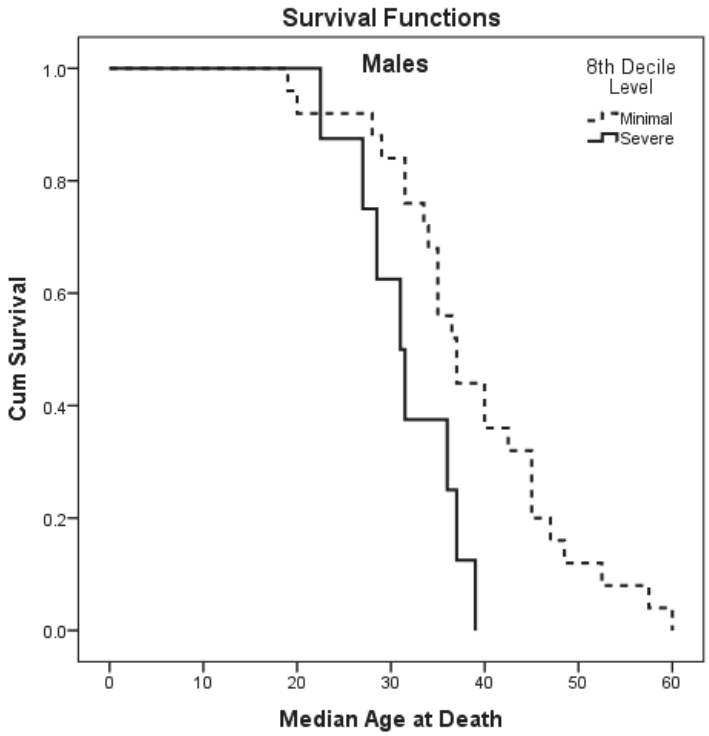


Table 4-75 Mantel-Cox log rank test showing the relationship between the frequency of accentuated striae of Retzius in the 3rd decile and median age at death by sex

Sex	Chi-Square	df	Sig.
Female	*5.232*	*1*	*.022*
Male	*.808*	*1*	*.369*

Figure 4-83 Kaplan-Meier survival curve representing the relationship between median age at death and the frequency of accentuated striae of Retzius in the 3rd decile for females

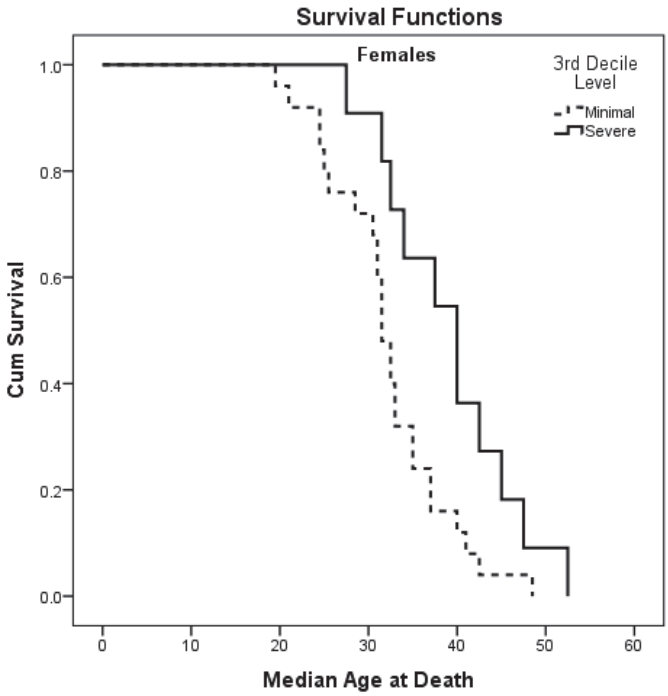
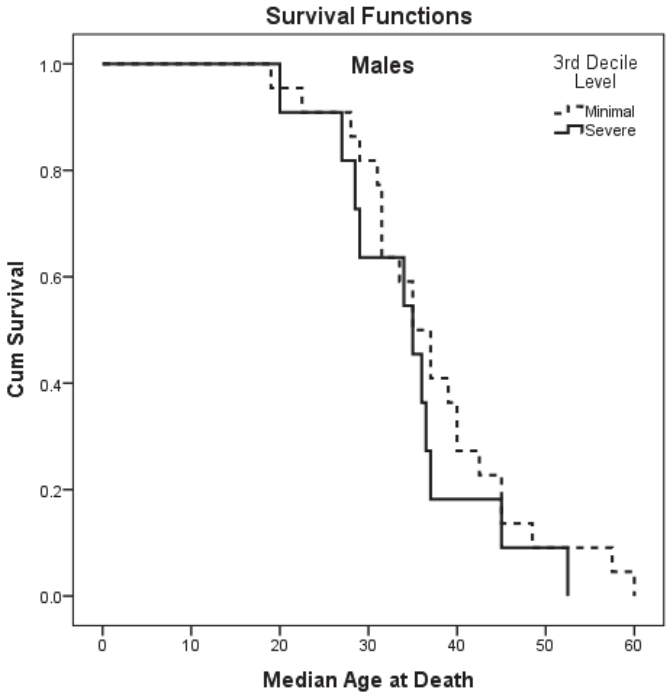


Figure 4-84 Kaplan-Meier survival curve representing the relationship between median age at death and the frequency of accentuated striae of Retzius in the 3rd decile for males

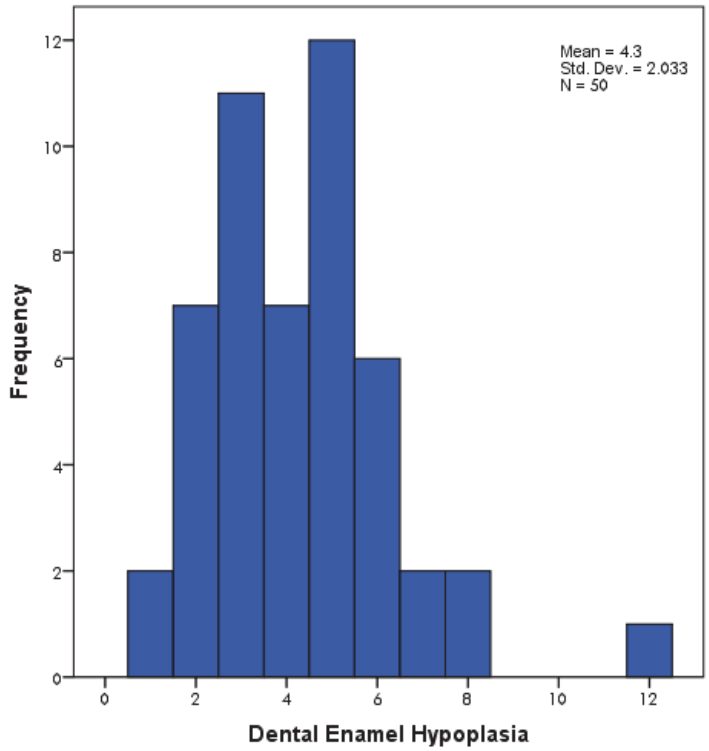


Further testing was conducted using hypoplastic counts based on visual assessment of the LEXT surface images and the profile lines. Individuals were divided into two levels based on the interquartile distribution of DEH (Table 4-76). Individuals with between zero and four surface defects were counted as having low stress and those with between five and twelve defects were counted as having high stress.

Table 4-76 Descriptive statistics of DEH categories

Dental Enamel Hypoplasia Level	N	Mean	Std. Deviation
Low	***20***	*2.45*	***.686***
High	***30***	*5.53*	*1.655*

Figure 4-85 Overall frequency distribution of DEH

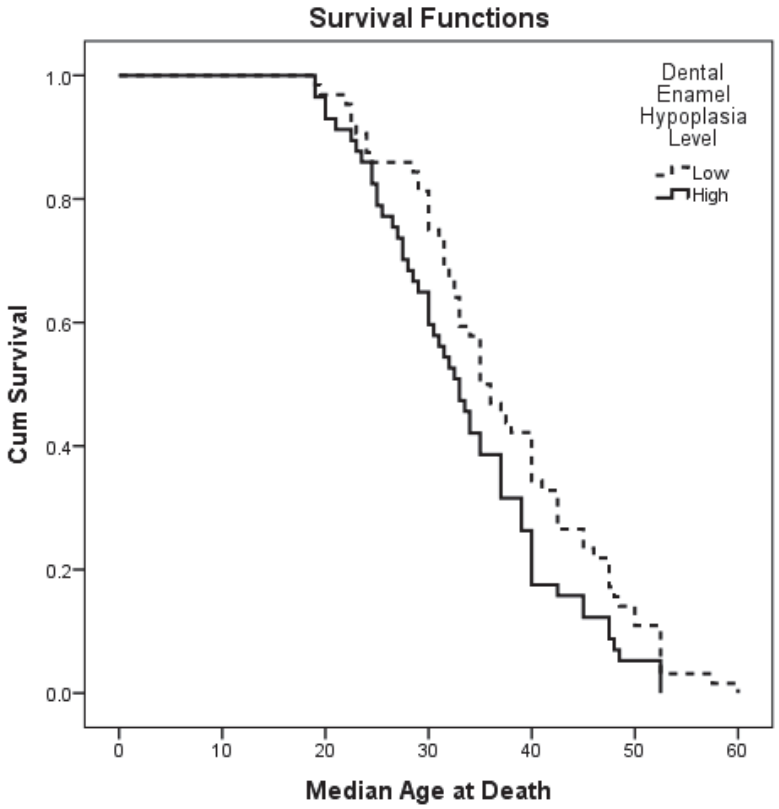


Kaplan-Meier survival analysis was conducted to consider whether any relationship was shown between these more severe defects and survival. The overall analysis with combined sexes did show a statistically significant relationship between individuals with more hypoplastic defects and decreased survivorship (Table 4-77, Figure 4-86).

Table 4-77 Mantel-Cox survival analysis testing the relationship between the frequency of dental enamel hypoplasia and median age at death

	Chi-Square	df	Sig.
Log Rank (Mantel-Cox)	***4.354***	***1***	***.037***
Test of equality of survival distributions for the different levels of Dental Enamel Hypoplasia Level.			

Figure 4-86 Kaplan-Meier survival curve representing the relationship between median age at death and the frequency of dental enamel hypoplasia



These results were further broken down by sex, and this showed that the negative correlation was significant for males, but that there was no significant relationship between DEH and survivorship for females (Table 4-78, Figure 4-87, and Figure 4-88).

Table 4-78 Mantel-Cox survival analysis testing the relationship between the frequency of dental enamel hypoplasia and median age at death by sex

Overall Comparisons				
Sex		**Chi-Square**	**df**	**Sig.**
Female	**Log Rank (Mantel-Cox)**	***.005***	***1***	***.946***
Male	**Log Rank (Mantel-Cox)**	***5.937***	***1***	***.015***
Test of equality of survival distributions for the different levels of Dental Enamel Hypoplasia Level.				

Figure 4-87 Kaplan-Meier survival curve representing the relationship between median age at death and the frequency of dental enamel hypoplasia for females

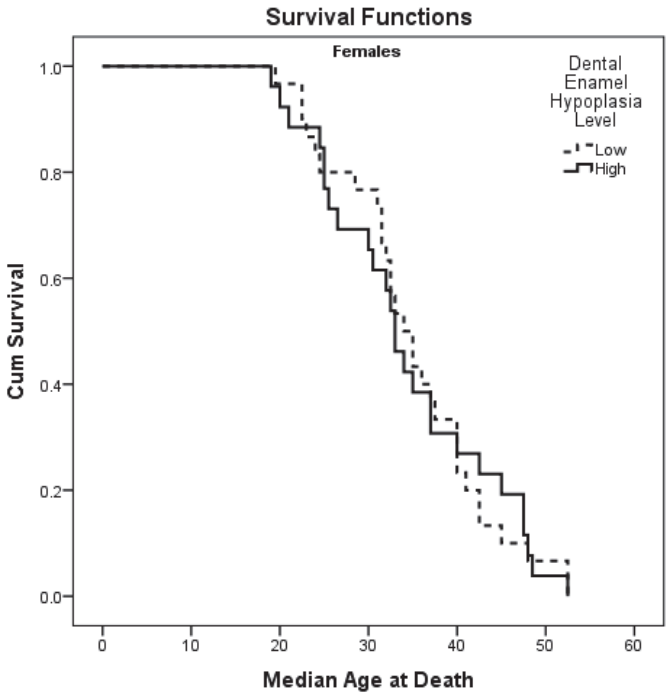
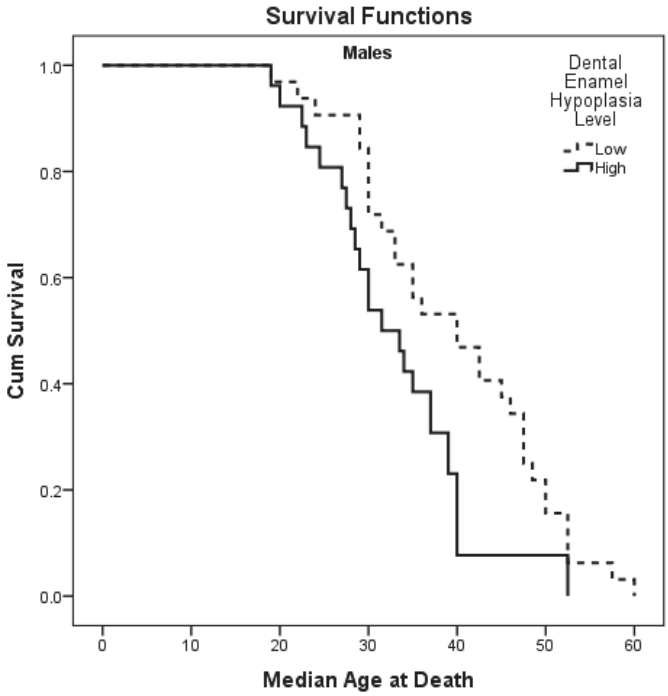


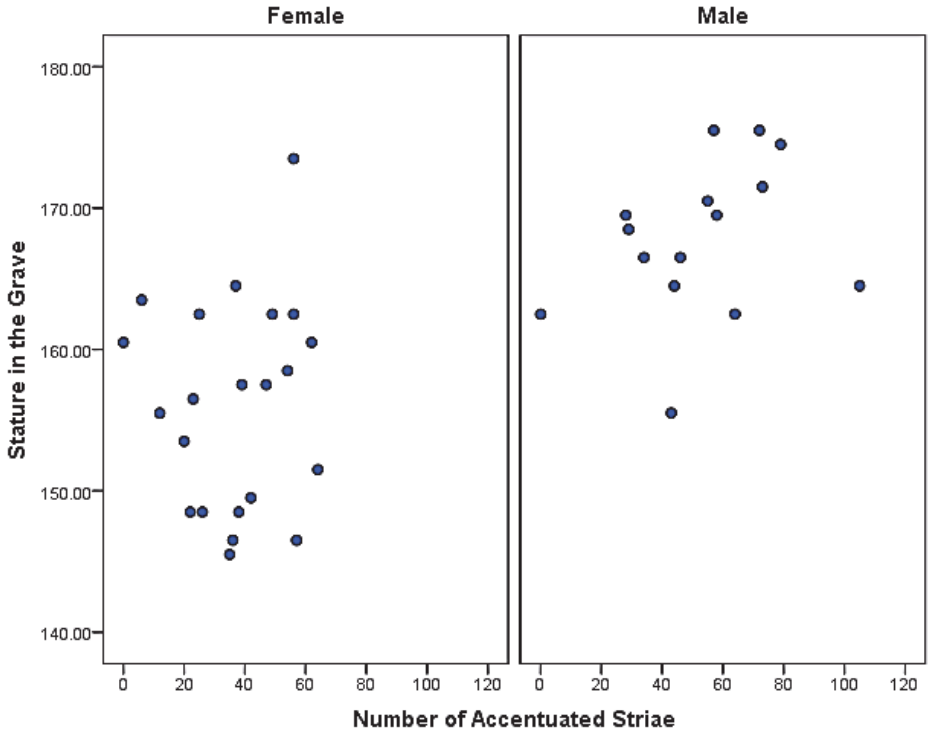
Figure 4-88 Kaplan-Meier survival curve representing the relationship between median age at death and the frequency of dental enamel hypoplasia for males



Enamel defects and stature / growth arrest

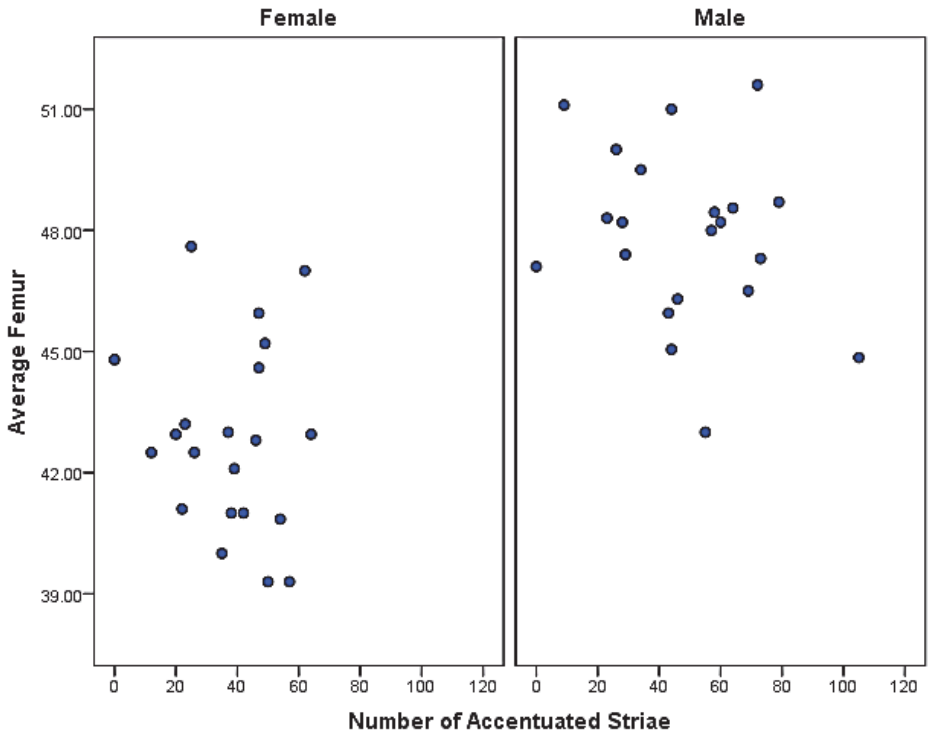
A consideration of the relationship between stress as indicated by the number of AS and achieved adult stature showed virtually no association for females (r = 0.054, N =22, p = 0.811) and only a very weak correlation for males (r = 0.318, N =15, p = 0.248) (Figure 4-89). Although the effect size for males was strong, this correlation was not statistically significant. Similarly, virtually no correlation was apparent for either sex when the DEH counts were plotted against stature in the grave. This suggests that the number of stress events early in life had little to no impact on adult stature.

Figure 4-89 Scatter plot showing relationship between accentuated striae of Retzius and stature in the grave



When femur length is used, a different trend emerges. While still minimal, we see a negative correlation between femur length and the number of AS (r = -0.092, N =21, p = 0.693 for females and r = -0.249, N =21, p = 0.277 for males) (Figure 4-90). The small sample sizes for both of these combinations, and the minimal correlation makes these trends inconclusive, but the effect size for males is strong and that for females medium. It is possible that these different trends are a by-product of catch-up growth, but further examination is required to learn more in this respect. DEH frequencies did not show any appreciable correlation with femur length (r = -0.129, N =29, p = 0.514 for females and r = -0.055, N =36, p = 0.752 for males).

Figure 4-90 Scatter plot showing relationship between accentuated striae of Retzius and femur length



Residual height (predicted height based on femoral length subtracted from stature in the grave) was considered in relation to AS to see if this approach could further elucidate any patterns. In the combined sex sample, these results showed a weak (r = 0.167, n = 35; r = 0.149, n = 57) and insignificant (p = 0.338; p = 0.149) correlation for AS and DEH respectively (Table 4-79). When broken down by sex, similarly low and insignificant correlations were apparent. A slightly higher correlation was apparent for females with DEH, where the correlation was moderate (r = 0.370, n = 24), but this correlation was still not significant (p = 0.076).

Table 4-79 Pearson's correlation and significance of correlation for males and females with AS counts and DEH in relation to residual height.

AS	N	p	r
Overall	***35***	***0.338***	***0.167***
Female	***18***	***0.911***	***0.028***
Male	***15***	***0.246***	***0.319***
DEH			
Overall	***57***	***0.149***	***0.268***
Female	***32***	***0.076***	***0.370***
Male	***29***	***0.869***	***-0.032***

The comparison of decile counts of AS to stature showed mixed results. The trends viewed in this respect echoed those seen for the overall accounts, suggesting a cumulative effect. When considered in relationship to stature in the grave, the decile correlation for AS tended to be weakly positive, with the highest correlation occurring in the 10^{th} decile with a strong effect size of $r = 0.29$ for females, and a stronger positive trend visible for males (with $r = 0.71$) which was statistically significant (p = 0.022) (Figure 4-91). When considered in relation to femur length, however, the trend reversed

and tended to be weakly negative. This was significant in the case of males for the 6th decile ($p = 0.047$, $r = 0.45$) but the same was not the case with females ($r = 0$ for females) (Figure 4-92).

Figure 4-91 Scatter plot showing relationship between accentuated striae of Retzius for the 10th decile and stature in the grave

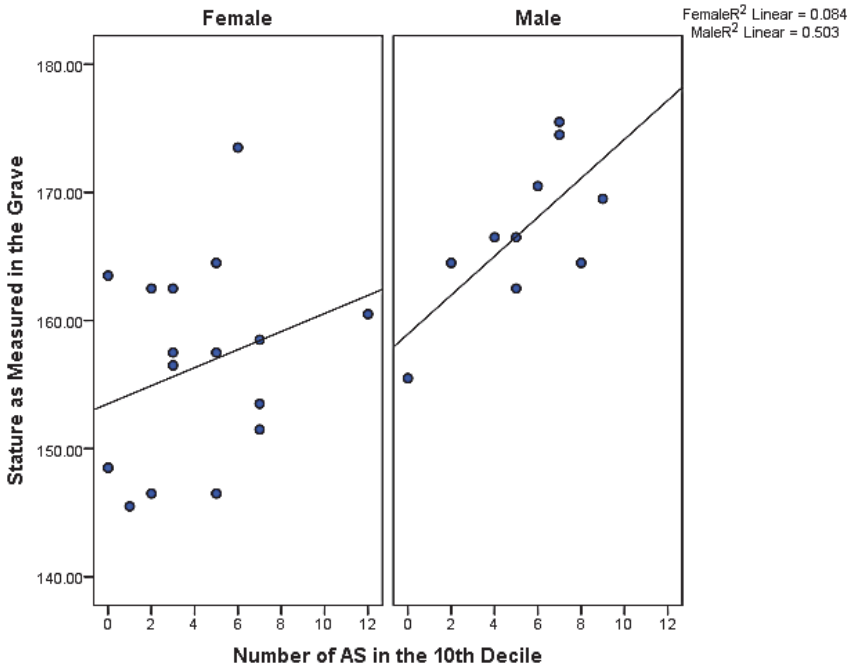
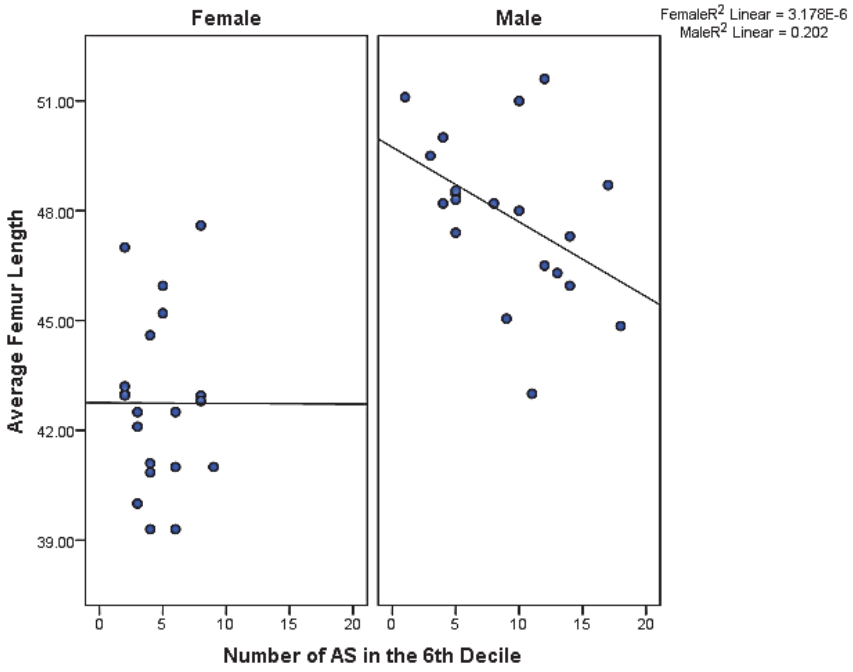


Figure 4-92 Scatter plot showing relationship between accentuated striae of Retzius for the 6th decile and femur length



Furthermore, while these patterns for all stature metrics are largely followed in all deciles that show any trend and while effect sizes are medium to large (Table 4-80), there is a high level of variability, with little to no correlation in some deciles and even the occasional reversal of these trends (Figure 4-91, Figure 4-92, Table 4-80). In general, very low to virtually no correlations are apparent across the deciles, suggesting that if there is a relationship much larger sample sizes are required to gain further insight.

Table 4-80 Effect sizes for correlations between the number of accentuated striae of Retzius and both femur length and stature in the grave measurements. Significant correlations are marked in bold.

Femur Length to Number of AS				**Stature in the Grave to Number of AS**		
Decile #	***Females***	***Males***		***Decile #***	***Females***	***Males***
1	-	-		**1**	-	-
2	0.367423	0.207364		**2**	0.094868	0.401248
3	0.044721	0.134164		**3**	0.264575	0.07746
4	0.181659	0.044721		**4**	0.202485	0.230217
5	0.164317	0.114018		**5**	0.063246	0.230217
6	-	**0.449444**		**6**	-	-
7	0.134164	0.2		**7**	0.254951	0.357771
8	0.07746	0.070711		**8**	0.07746	-
9	0.104881	0.294958		**9**	0.130384	0.083666
10	0.41833	0.126491		**10**	0.289828	**0.709225**

Table 4-81 Correlation coefficients for femur length and stature in the grave by decile. Significant correlations are marked in bold.

Femur Length to Number of AS				Stature in the Grave to Number of AS		
Decile #	***Females***	***Males***		***Decile #***	***Females***	***Males***
1	-	-		**1**	-	-
2	-0.135	+0.043		**2**	+0.009	+0.161
3	-0.002	-0.018		**3**	+0.070	+0.006
4	-0.033	-0.002		**4**	+0.041	+0.053
5	-0.027	-0.013		**5**	-0.004	+0.053
6	-	**-0.202**		**6**	-	-
7	-0.018	-0.040		**7**	+0.065	+0.128
8	+0.006	-0.005		**8**	+0.006	-
9	+0.011	-0.087		**9**	-0.017	+0.007
10	+0.175	+0.016		**10**	+0.084	**+0.503**

The record of AS by deciles had the potential to provide the opportunity to consider the impact of timing of stress events on growth arrest (Floyd and Littleton, 2006). Specifically, the difference between femoral correlations and stature in the grave correlations presents an interesting pattern, and the ability to test timing of stress events in relation to differential growth would have been valuable. In an attempt to develop this further, residual height was once again considered, this time in relation to each decile (Table 4-82, Table 4-83). These results yielded little in the way of correlation between residual height and AS rates in each decile. There was, however, a significant positive correlation between AS rate and residual height in the 4^{th} decile ($n = 33$, $r = 0.413$, $p = 0.017$). Further breakdown showed this to be significant positive for females but not for males (Figure 4-93). This decile reflects growth from approximately 2.3 to 2.7 years of age according to Reid and Dean's calculations (2006). This particular correlation might suggest that females developing during this period had reduced femoral length in relation

to final stature with more stress. Applying these techniques to a larger sample, however, is necessary provide useful insight into this in the future.

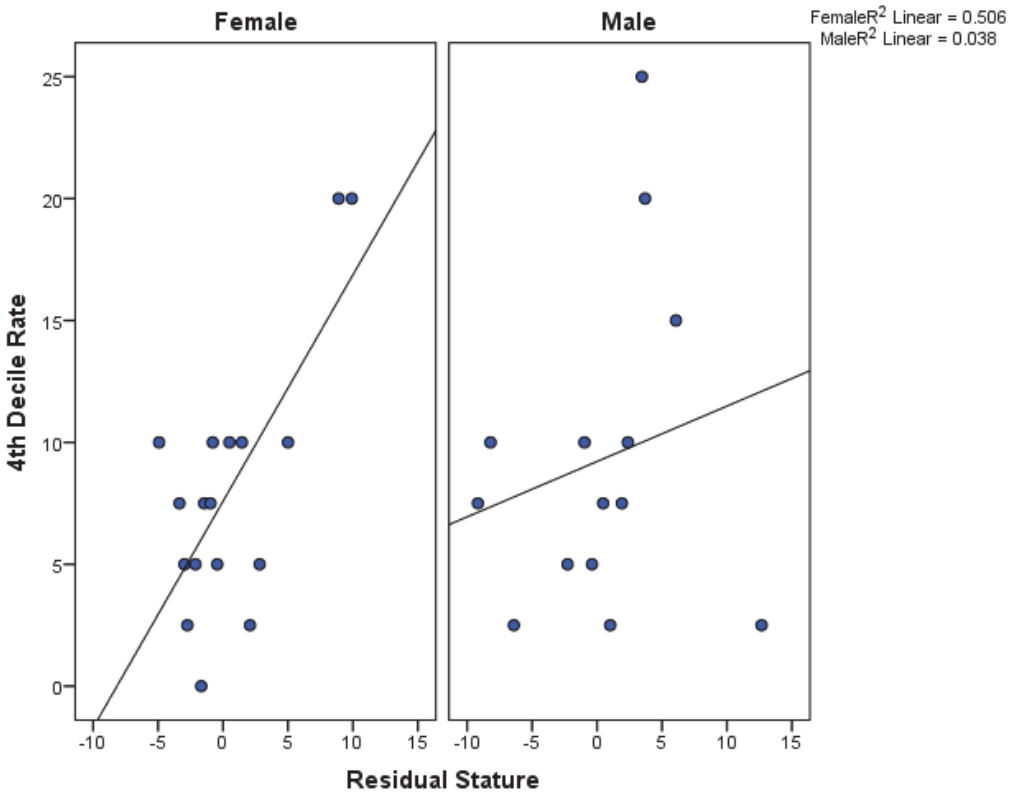
Table 4-82 Pearson's correlation and significance of correlation for males and females in AS counts by decile. Significant correlations are marked with an asterisk.

Females – AS Count	N	p	r	Males – AS Count	N	p	r
1st	*8*	*0.128*	*0.584*	1st	*5*	*0.137*	*0.759*
2nd	*14*	*0.369*	*0.260*	2nd	*12*	*0.760*	*0.099*
3rd	*17*	*0.261*	*0.289*	3rd	*14*	*0.497*	*0.198*
4th	*17*	*0.025**	*0.541*	4th	*14*	*0.502*	*0.196*
5th	*16*	*0.212*	*0.330*	5th	*14*	*0.438*	*0.226*
6th	*17*	*0.111*	*-0.400*	6th	*14*	*0.239*	*0.337*
7th	*17*	*0.891*	*0.036*	7th	*14*	*0.238*	*0.337*
8th	*16*	*0.979*	*0.007*	8th	*14*	*0.506*	*-0.194*
9th	*14*	*0.322*	*-0.286*	9th	*12*	*0.557*	*0.189*
10th	*13*	*0.825*	*0.068*	10th	*10*	*0.234*	*0.414*

Table 4-83 Pearson's correlation and significance of correlation for males and females in AS rates by decile. Significant correlations are marked with an asterisk.

Females – AS Rate	N	p	r	Males – AS Rate	N	p	r
1st	*32*	*0.579*	*0.233*	1st	*5*	*0.137*	*0.759*
2nd	*14*	*0.464*	*0.213*	2nd	*12*	*0.761*	*0.099*
3rd	*17*	*0.261*	*0.289*	3rd	*14*	*0.497*	*0.198*
4th	*17*	*0.001**	*0.711*	4th	*14*	*0.502*	*0.196*
5th	*16*	*0.489*	*0.187*	5th	*14*	*0.438*	*0.226*
6th	*17*	*0.098*	*-0.415*	6th	*14*	*0.239*	*0.337*
7th	*17*	*0.818*	*0.060*	7th	*14*	*0.238*	*0.337*
8th	*16*	*0.715*	*0.099*	8th	*14*	*0.506*	*-0.194*
9th	*14*	*0.443*	*-0.223*	9th	*12*	*0.557*	*0.189*
10th	*13*	*0.825*	*0.068*	10th	*10*	*0.234*	*0.414*

Figure 4-93 Scatterplot showing correlation between residual height and decile rate for males and females in the 4th decile of growth



Enamel defects and infection / disease

Consideration of whether the number of stress events experienced during childhood impacted susceptibility to leprosy was done by plotting λ scores in relation to the number of AS. This analysis showed little correlation between these two variables with the combined sample (r = -0.133, N =65, p = 0.290). When further broken down, little correlation is seen with either of the sexes (r = -0.182, N =31, p = 0.328 for females, r = -0.113, N =31, p = 0.545 for males).

One-way ANOVA was also performed to test whether any of the diseases considered here related to differing mean in AS and to DEH. The AS and DEH counts were compared between individuals who did not have indicators of each disease, those who were possible sufferers, and those who were probable sufferers. No significant differences were found in the mean frequency of either defect score between the different groups for treponematosis (Table 4-86, Figure 4-96), tuberculosis (Table 4-84, Figure 4-94), or leprosy (Table 4-85, Figure 4-95).

Figure 4-94 Boxplot illustrating the median and ranges for AS scores in relation to tuberculosis diagnosis

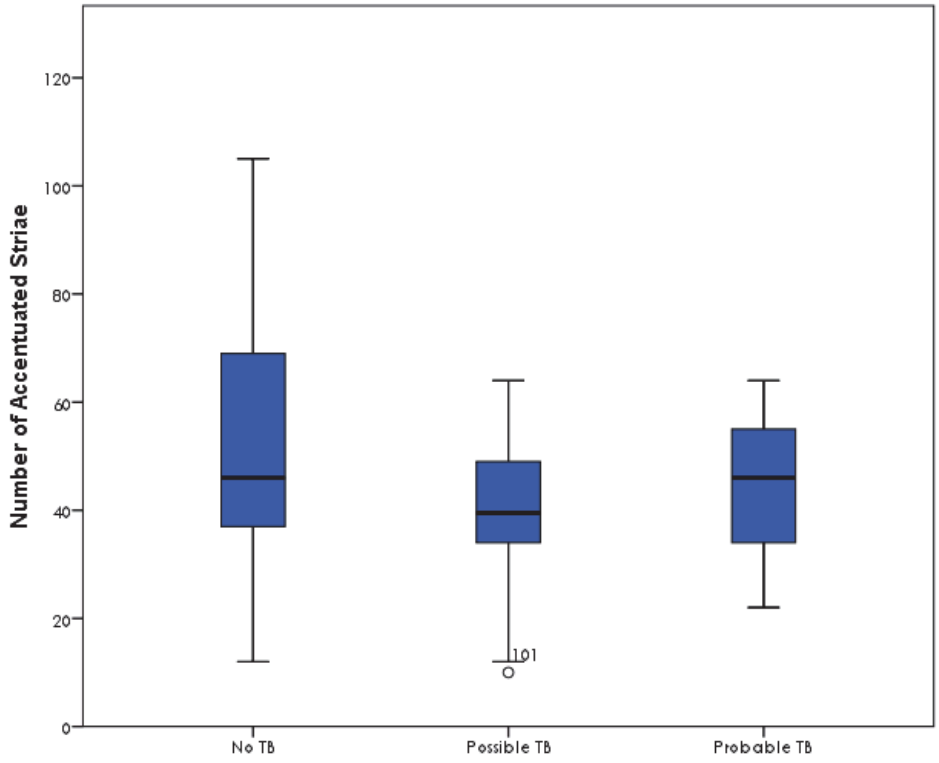


Table 4-84 Descriptive statistics detailing the difference in AS counts and DEH between tuberculosis diagnostic categories

		No TB	Possible TB	Probable TB
Total AS	**N**	21	22	3
	Mean	50.76	39.41	44.00
	Std. Deviation	25.35	14.16	21.07
DEH	**N**	***29***	***30***	***7***
	Mean	***3.69***	***3.17***	***2.43***
	Std. Deviation	***1.63***	***1.76***	***1.27***

Figure 4-95 Boxplot illustrating the median and ranges for AS scores in relation to leprosy diagnosis

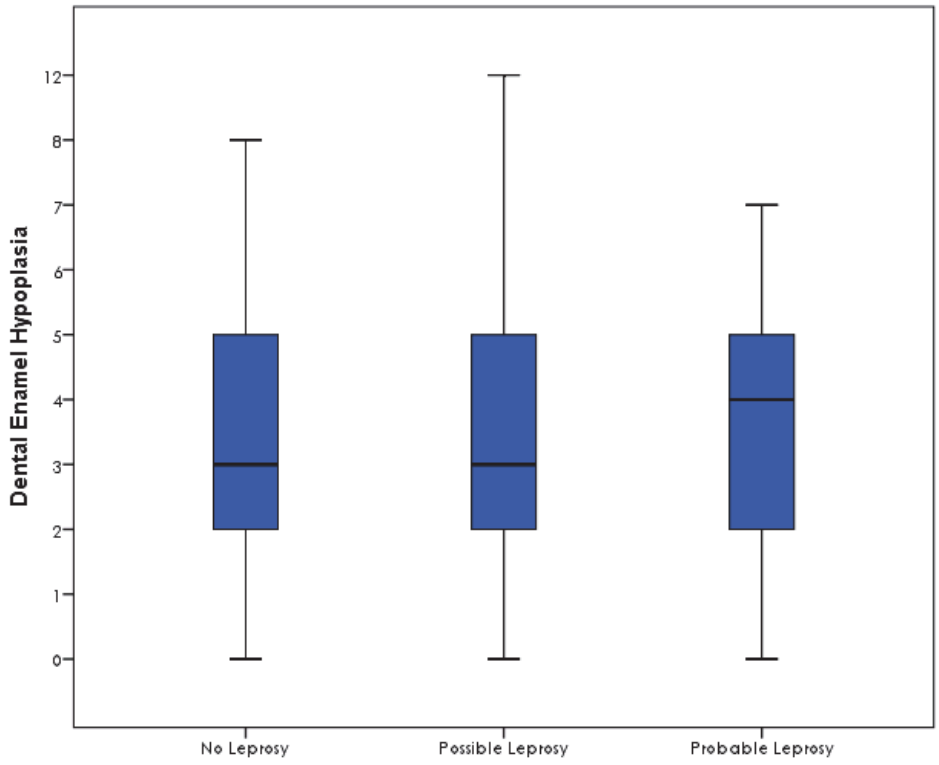


Table 4-85 Descriptive statistics detailing the difference in AS counts and DEH between diagnostic categories for leprosy

		No Leprosy	Possible Leprosy	Probable Leprosy
Total AS	N	*31*	*15*	*19*
	Mean	*47.71*	*42.27*	*39.47*
	Std. Deviation	*11.01*	*20.03*	*26.71*
DEH	N	*44*	*29*	*33*
	Mean	*3.59*	*3.76*	*3.52*
	Std. Deviation	*2.14*	*2.28*	*1.81*

The difference in mean AS between categories of treponema did approach significance (Figure 4-96, Table 4-86, Table 4-86), but small sample sizes of diagnosed treponemal cases restrict interpretation of this result. If possible cases are dropped from the analysis and an t-test performed to consider the difference in mean AS between individuals with probably treponema and those who did not show any signs of treponema, the difference is significant ($F = 2.077$, $df = 39$, $p = 0.029$). In this case, the individuals with treponema ($n = 4$) had a higher mean number of AS ($\overline{x} = 63$, $sd = 31.633$) than individual with no bony changes indicating treponema ($\overline{x} = 38.78$, $n = 37$, $sd = 19.112$). Once again larger sample sizes are required to draw conclusions from this, and it is clear that there is an overlap in AS range between the two disease categories, but this is suggestive of a possible connection between the number of stress events during growth and development and response to treponema later in life.

Figure 4-96 Boxplot illustrating the median and ranges for AS scores in relation to treponemal diagnosis

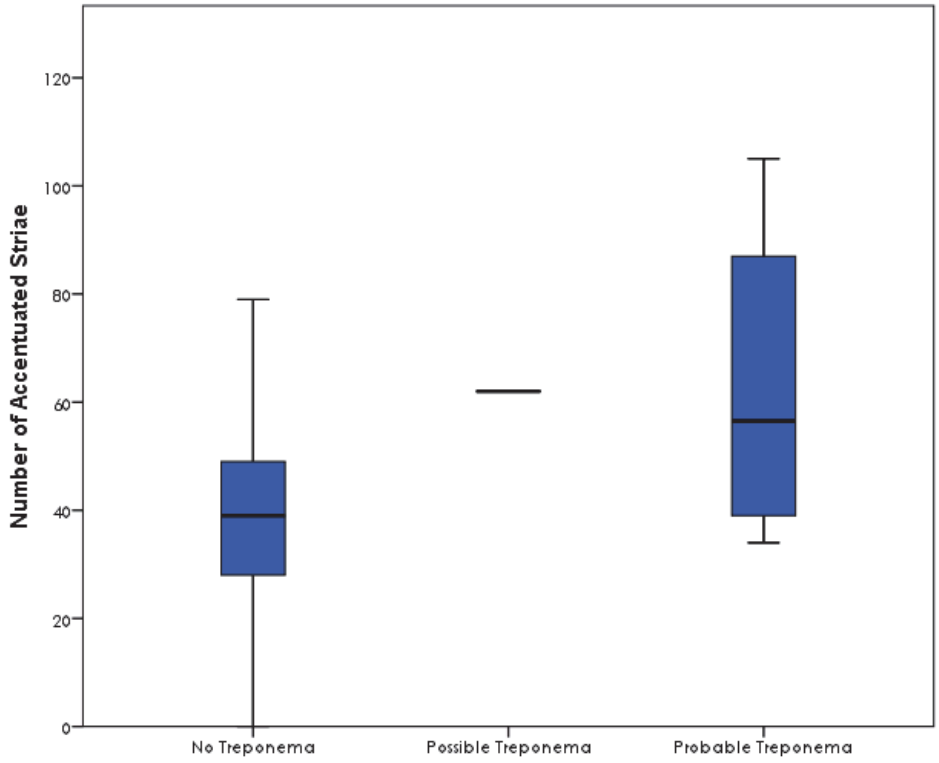


Table 4-86 Descriptive statistics detailing the difference in AS counts and DEH between treponemal diagnostic categories

		No Treponema	Possible Treponema	Probable Treponema
Total AS	**N**	37	1	4
	Mean	38.78	62.00	63.00
	Std. Deviation	19.11	62.00	31.63
DEH	**N**	***51***	***6***	***9***
	Mean	***3.55***	***3.67***	***3.33***
	Std. Deviation	***2.12***	***1.37***	***1.23***

Table 4-87 Independent samples t-test comparing AS mean between probable cases of treponema and cases with no sign of treponemal change

		Levene's Test for Equality of Variances		t-test for Equality of Means						
									95% Confidence Interval of the Difference	
		F	Sig.	t	df	Sig. (2-tailed)	Mean Difference	Std. Error Difference	Lower	Upper
Number of Accentuated Striae	Equal variances assumed	*2.077*	*.158*	*-2.261*	*39*	*.029*	*-24.216*	*10.711*	*-45.882*	*-2.550*
	Equal variances not assumed			*-1.502*	*3.241*	*.224*	*-24.216*	*16.126*	*-73.442*	*25.010*

Decile counts for AS did not show a consistently significant difference in mean stress scores between disease diagnostic categories for each disease type. The descriptive statistics for deciles with each disease can be seen in Table 4-88. Only the first decile showed a significant result for tuberculosis ($F = 13.781$, $df = 2$, $p = 0.002$) and this score was restricted by only being scored with one case of probable tuberculosis.

Treponemal scores showed far more significant differences in mean AS across the deciles, with significant differences apparent for the 6th and 9th, and 10th deciles (Table 4-89). However, with only four individuals scored as probable cases of treponema and one scored as a possible case (Table 4-88), sample sizes are insufficient to draw any conclusions from these results.

Table 4-88 AS descriptive statistics by disease categories for each decile

Decile		No TB	Possible TB	Probable TB	No Leprosy	Possible Leprosy	Probable Leprosy	No Treponema	Possible Treponema	Probable Treponema
1st	N	6	5	1	11	4	6	12	0	1
	Mean	1.67	6.00	25.00	6.82	2.50	5.00	6.67		0.00
	s.d	2.58	5.48	.	7.83	2.89	3.16	7.18		.
2nd	N	16	17	1	23	12	14	26	1	3
	Mean	2.31	3.92	10.00	2.33	6.39	3.57	4.36	6.67	0.00
	s.d	3.97	4.29	.	2.91	5.59	4.23	4.97	.	0.00
3rd	N	21	21	2	31	13	16	34	1	4
	Mean	7.94	4.60	3.33	7.10	9.49	6.25	6.86	3.33	10.83
	s.d	6.19	4.77	4.71	6.60	7.43	5.56	6.25	.	9.95
4th	N	21	22	3	31	14	17	35	1	4
	Mean	10.95	7.61	7.50	8.55	11.79	7.94	8.57	7.50	10.63
	s.d	5.84	4.60	9.01	6.51	6.23	5.54	5.86	.	4.73
5th	N	20	22	3	31	14	17	34	1	4
	Mean	17.13	12.73	20.00	15.16	16.43	15.59	15.00	15.00	23.13
	s.d	9.57	4.81	11.46	8.56	9.39	7.78	7.83	.	14.20
6th	N	21	22	3	31	14	17	35	1	4
	Mean	17.14	11.27	13.33	15.23	13.57	12.12	12.76	4.00	23.00
	s.d	10.46	7.03	3.06	8.48	8.56	8.85	8.00	.	9.59
7th	N	21	22	3	31	14	17	35	1	4
	Mean	15.28	11.82	13.33	14.70	11.55	11.47	12.12	11.67	15.83
	s.d	8.98	5.86	7.26	7.74	6.01	6.87	6.63	.	12.21
8th	N	20	22	3	29	14	15	33	1	4
	Mean	11.41	12.44	15.12	13.92	10.06	12.00	11.87	17.14	16.43
	s.d	7.40	6.77	5.49	8.74	5.41	6.52	7.25	.	6.65
9th	N	20	19	2	27	13	15	30	1	4
	Mean	10.73	8.45	13.57	11.49	8.62	10.67	8.50	24.29	12.14
	s.d	7.51	7.51	9.09	8.46	6.62	8.65	6.39	.	7.05
10th	N	17	15	2	23	10	13	25	1	4
	Mean	11.27	6.67	10.00	8.70	9.33	10.38	7.37	20.00	8.75
	s.d	8.89	3.33	2.36	7.30	3.16	8.48	4.55	.	4.17

Table 4-89 ANOVA test results for AS scores in each decile by treponemal categories

		Sum of Squares	df	Mean Square	F	Sig.
1st Decile Rate	Between Groups	*41.03*	*1*	*41.03*	*.80*	*.39*
	Within Groups	*566.67*	*11*	*51.52*		
	Total	*607.69*	*12*			
2nd Decile Rate	Between Groups	*58.46*	*2*	*29.23*	*1.28*	*.30*
	Within Groups	*617.09*	*27*	*22.86*		
	Total	*675.56*	*29*			
3rd Decile Rate	Between Groups	*71.61*	*2*	*35.80*	*.81*	*.45*
	Within Groups	*1584.80*	*36*	*44.02*		
	Total	*1656.41*	*38*			
4th Decile Rate	Between Groups	*16.74*	*2*	*8.37*	*.25*	*.78*
	Within Groups	*1233.26*	*37*	*33.33*		
	Total	*1250.00*	*39*			
5th Decile Rate	Between Groups	*236.98*	*2*	*118.49*	*1.62*	*.21*
	Within Groups	*2629.69*	*36*	*73.05*		
	Total	*2866.67*	*38*			
6th Decile Rate	Between Groups	*470.41*	*2*	*235.20*	*3.55*	*.04*
	Within Groups	*2454.19*	*37*	*66.33*		
	Total	*2924.59*	*39*			
7th Decile Rate	Between Groups	*50.20*	*2*	*25.10*	*.48*	*.62*
	Within Groups	*1943.53*	*37*	*52.53*		
	Total	*1993.73*	*39*			
8th Decile Rate	Between Groups	*96.47*	*2*	*48.23*	*.93*	*.40*
	Within Groups	*1814.37*	*35*	*51.84*		
	Total	*1910.84*	*37*			
9th Decile Rate	Between Groups	*276.19*	*2*	*138.09*	*3.32*	*.05*
	Within Groups	*1331.99*	*32*	*41.63*		
	Total	*1608.18*	*34*			
10th Decile Rate	Between Groups	*156.06*	*2*	*78.03*	*3.84*	*.03*
	Within Groups	*548.37*	*27*	*20.31*		
	Total	*704.43*	*29*			

The subdivision of the sample by sex showed no difference in means using a one-way ANOVA between either overall DEH or AS and any of the diseases diagnosed for this study. There were some significant differences in means for some of the deciles with tuberculosis and treponema. In the case of tuberculosis, females showed a difference in mean stress markers amongst individuals who were diagnosed with tuberculosis and those who were not for the first ($F = 9.829$, $df = 2$, $p = 0.029$) and second deciles ($F = 5.769$, $df = 2$, $p = 0.016$). However, only one individual was diagnosed with probable TB in each case, and so once again these results cannot be seen as representative of a broader pattern. Amongst males, the 4th decile showed a significant difference in means between TB classifications ($F = 4.542$, $df = 2$, $p = 0.024$), but a same sample size issue prevails. In the case of treponema, females show significant differences in means for the 9th ($F = 5.424$, $df = 1$, $p = 0.035$) and 10th decile ($F = 8.861$, $df = 1$, $p = 0.01$), but no significant results were shown for males. The case with these results is similarly extremely problematic, with only one possible case of treponema being present for this analysis, and with no individuals being diagnosed as probable cases.

The potential for the 'healthy' component of this sample to have undetected illness does need to be considered with these results, but given that the pattern persists through different registry systems through diseases, this would seem to strongly indicate the absence of a connection between AS and illness in these individuals.

Summary

The results from the adult sample used for this study show sex-based differences on many levels. Females overall had slightly lower mean ages of death, but this was not significant. Males, on the other hand, had significantly higher numbers of AS, but this was not visible for DEH. All three diseases that were scored showed higher rates in males than in females, but this was not statistically significant.

These differences are further manifest in the consideration of the connection between early life stress events and later life health. Overall, the results suggest that females may respond better to stress than males. Each variable considered showed that females with more stress tended to live longer than males with more stress. In the case of males, a neutral or negative relationship was observed, and this was significant in some instances. These patterns were only significant for some deciles and for DEH patterns in females.

The consideration of stature, on the other hand, produces a mixed picture which is impeded by relatively small sample sizes. This is characterized by stronger (although still very weak) correlations overall for males whether stature as measured in the grave or femur length is used. While the trend seems to be positive for stature in the grave, however, it appears to be negative in the case of femur length. A consideration using residual height highlighted this relationship, with males with more AS exhibiting shorter femoral lengths than expected given stature in the grave. These results are, interestingly, consistent with those found in the Tirup sample by Iregren and Boldsen (1990). The patterns seen could have to do with timing of catch-up growth, with femoral length

capturing a briefer period of growth as opposed to stature in the grave which will reflect cumulative growth. However, larger sample sizes are required to gain further insight into the extent to which these trends reflect real relationships. In particular, the consideration of components of growth in relation to developmental periods (using the decile system) with larger samples would be informative. No correlation was visible between early life stress and any of the three diseases considered here. While this may reflect the lack of a connection, further research applying more developed methods for scoring these diseases (particularly tuberculosis and treponema) is required.

Site differences were apparent on a few levels, but many of these are problematic due to small subsample sizes. Potential bias as a result of sampling sizes and possibly as a result of the sampling procedure requiring less worn teeth can be observed in the comparison with the broader cemetery samples. Within the subsample used for this study, adults at Sejet had a statistically significant lower mean age at death than those at Ole Wormsgade, and this was further found to be statistically significant for adult females. There was no statistically significant difference in stature between the sites, but this is impeded by the lower sample sizes from Ole Wormsgade. Adults from the Ole Wormsgade subsample had a slightly higher frequency of AS, but this was not statistically significant and was not visible for DEH. All three diseases showed higher frequencies at Sejet, but this was only significant in the cases of leprosy and tuberculosis. When broken down by sex, it was apparent that treponemal frequencies were lower for both sexes at Ole Wormsgade, but these results were not significant and small sample sizes impeded any interpretation of these results. Tuberculosis showed a sex difference,

being significantly more common in females at Sejet but not in males. Both sexes showed significantly higher frequencies of leprosy at Sejet, but the results for males should be treated with caution and so it can only be said that females from these subsamples show significantly higher frequencies at Sejet. As such, the site analysis shows significantly lower adult mean age at death and higher rates of pathology in Sejet subsample, but extending these results beyond the current study should be treated with caution.

Temporal differences were also problematic to consider due to the low numbers of individuals from the later period. Overall, while there was a slightly lower mean age at death during the earlier period, this was not statistically significant. Males showed a significantly lower mean age at death during the early period, but this was problematic due to the drastically smaller later period sample sizes. Females showed non-significant decrease over time. When stature is considered, however, there is a significant decrease in mean stature for females from the early to late period, but for males a similar decrease is not significant. The use of femur length, with small sample sizes, shows no significant change for either sex. Similarly, the number of stress events, whether internal or surface, showed no significant change either for the complete sample or for the sex-divided sample. On the other hand, there are possible changes in disease frequencies from the early to late periods, with treponemal cases showing a slight increase (which was not statistically significant and so needs to be treated with caution due to small sample sizes). Tuberculosis frequencies show no a statistically significant change over time, either with the complete sample or when the sexes are considered separately. Leprosy frequencies appear to decline from the early to the late period, but not significantly. A slight reversal

in relative frequency of diagnosed leprosy cases is also observable between the sexes. While more common in males during the early period, slightly more females appear to be affected during the later period. This temporal change is significant in males, but not in females. Thus, while these results hint at possible changes in health patterns over time, the smaller sample sizes in the later period caution against any conclusive interpretation of temporal change in health.

Chapter 5 Discussion

Introduction

The purpose of this research was threefold. Ultimately, it sought to gain a better understanding of the health of the populations being studied. The study sample drew from a broad span of time, from approximately the mid-12th to the mid-16th centuries and encompassing a series of climatic and epidemiological events. One objective was therefore to examine whether the population and socioeconomic changes recorded historically had an impact on health. The study also included populations from two sites that are located in close proximity to each other, but while one was rural, the other was urban. A second objective was therefore to test health differences between the two sites. Since these populations lived over the course of a particularly tumultuous period in history (Benedictow, 2004, 2012; Hybel and Poulsen, 2013), any impact on health of changing socio-environmental factors was of interest. The final objective was to gain insight into the relationship between evidence of childhood stress and adult health by looking at specific (nonspecific) childhood stress events recorded in dental enamel and comparing these to evidence for adult health.

While the information collected about the relationship between early life stress incidents (in the form of accentuated striae of Retzius and dental enamel hypoplasias) and later life health was informative and points towards interesting new avenues for exploration, the study of temporal and site differences was problematic on a number of levels. Information on the broader cemeteries was acquired through the cemetery databases produced by ADBOU. Comparison of the statistics from the subsamples used in this research with statistics from these broader cemetery sample databases showed

inconsistencies which bring into question the representativeness of the subsamples in relation to the broader picture. Namely, some of the patterns which appeared through the current analysis were not maintained when the larger cemetery statistics were considered. In order to gain better insight into these issues and into the site patterns, the broader site statistics will therefore be considered as much as possible for inter-site comparisons. Unfortunately, temporal changes could not be considered on the larger cemetery sample level, as this information is not available at the point of examination. A further issue arose in the consideration of temporal changes. To compound the possible poor representativeness of the subsamples, sample sizes for the later period (post-1350 A.D.) were much smaller and so the extent to which conclusions could be drawn given available information was unfortunately limited.

Temporal Changes in Health

One of the biggest challenges faced with the temporal consideration of medieval sites seems to be a general paucity of material from the period after the mid-14th century. This is visible in the distribution of individuals by time for both Ole Wormsgade and Sejet (Figure 3-5, Figure 3-6). Some sites, such as Tirup, had gone out of use by the mid14th century (Kieffer-Olsen, 1993). Others which do bridge this margin have, like Ole Wormsgade and Sejet, fewer burials after approximately 1350 A.D. This is seen in Yoder's (2006) sample distribution for all of her sites (Øm Kloster, Lille Sct. Mikkelsgade, and Ribe). There are some cemeteries which represent largely late-period cemeteries, and one of these was used by Arcini (1999) in her examination of health in medieval Lund (Trinitatis parish church dated to A.D. 1300 to A.D. 1536).

This common imbalance of material from the later period makes it challenging to compare health patterns over time and to gain insight into the impacts of factors such as environmental stressors in the form of, for example, crop failure and the decline of marginal zones. In itself, however, this trend in burials is interesting as it likely speaks to population patterns over these periods. The lower number of burials after the mid-14th century likely in itself speaks to the massive depopulation following the famine years and the onslaught of Black Death in 1350 A.D. (as well as to its recurrence in 1360 A.D., 1368-1369 A.D., and 1379 A.D.) (Bøgh, 1999). This pattern complements the historical literature which estimates that as much as 50% of the population was wiped out by the plague (Bøgh, 1999; Benedictow, 2004), and only began to recover around 1450 A.D (Bøgh, 1999). We may have fewer people because there were fewer people around, and this in itself is interesting.

It has been proposed that the relief of pre-plague population pressure[35] may have resulted in healthier conditions in the following period (Benedictow, 2004, 2012). Certainly historical records suggest that after the Black Death, workers were at a premium for landowners, who lowered taxes in an attempt to attract workers (Bøgh, 1999; Benedictow, 2004). Wages went up, both rent and land prices decreased, and it is likely that there was a marked improvement in living conditions with lower food prices (in particular grain) (Bøgh, 1999; Benedictow, 2004). Benedictow (2004, p 390) notes that

[35] It is estimated that the population tripled from approximately ½ a million in A.D. 1100 to approximately 1.5 million by A.D. 1250 (Bøgh, 1999, p 25). While this estimate of growth may be on the extreme end, Hybel and Poulsen (2013) also paint a picture of population growth over this period, and possibly extending back into the preceding Viking period. They do not suggest an approximate population at the beginning of the period, but they do propose gradual expansion punctuated by periods of slower growth culminating in a population of approximately 1,326,000 by the first half of the 13th century (Hybel and Poulsen, 2013, p 128)

> … the pattern of demand and consumption changed. After having satisfied a basic demand for consumption of energy in the form of grain-based or bean-based, cheap, calorie-rich foods, people still had an economic surplus that they could use to increase their consumption of other foodstuffs that tasted better … In short, people ate more meat and butter, [and] drank more beer and wine …

This period also saw increasing emphasis on cattle rearing, since cattle are less labour intensive and can be traded to bring revenue into the country (Poulsen, 1997; Bøgh, 1999; Hybel and Poulsen, 2013). Indeed, Denmark became a major centre for oxen trade during this later period (they became the main export), and market towns (such as Horsens) grew and became more important to these trade networks (Andrén, 1989; Poulsen, 1997; Hybel and Poulsen, 2013). It is during this time that we also see the rise of organized guilds and the importance of skilled trades increasing, with the possibility of peasants acting as middlemen in trade networks (Andrén, 1989; Bøgh, 1999). In this context, there is greater potential for the average worker to own land. There is also an influx of people into more urban settings, and a change in emphasis in agriculture which could have resulted in dietary shifts and in different pathogen exposures (e.g. to bovine TB) (Boldsen and Mollerup, 2006; Boldsen, 2009). A similar situation has been proposed for England (Manchester, 1984). These changes impacted people at all levels of society. The question, then, is to what extent and in what way did these changes affect population health?

While it might be expected that the relief in population pressure and subsequent improvement in living conditions may have resulted in better overall health, neither Yoder (2006) nor Arcini (1999) identified significant consistent changes in health over time in their samples. Benedictow (2012) paints a picture from the earlier period of

severe malnutrition with corresponding susceptibility to infection, high mortality, and the beginnings of declining population – or at least a stationary one. At the same time, she notes that the following period had high rates of fertility with women marrying early and with high numbers of marriages. Infectious diseases still ran rampant with high rates of mortality. Given this description, it is possible that while there were significant changes, rates of disease and mortality may have still remained high (especially for women) (Benedictow, 2012).

Overall, the lack of a statistically significant temporal change in adult mean age at death seen here was consistent with Benedictow's (2012) comments. The slightly lower mean age at death in the subsample used here from the earlier period would be consistent with the stressful situation at that time. However, given small sample sizes, the lack of statistical significance, and the issues with sample representativeness, this can only be suggestive of a possible trend. This is also in line with both Yoder's (2006) and Arcini's (1999) findings of a lack of consistent change in health over time.

Interestingly, however, stature (as measured in the grave) did show a statistically significant decrease over time for females and a slight (but not statistically significant) decrease for males. The pattern was further visible when residual height was considered, with a significant decrease possibly suggesting that predicted height (as measured by femoral length) was closer to actual height (as measured from stature in the grave). Whether this indicates reduced growth arrest during the period involved in femoral growth is a question which requires further investigation. While the small number of individuals, particularly for the later period samples, necessitates caution about

extrapolating extensively from these results, the effect size for the differences in stature in the grave was large ($d = 0.957$, $r = 0.432$) and the difference in residual height was significant using an independent samples t-test.

Pedersen and Boldsen (2008) note in the Sejet Anthropological Report that the overall sample at Sejet had a lower mean stature than that detected at the nearby rural site of Tirup. They propose that this may be reflective of the slightly later use of Sejet, since Tirup went out of use by the mid-14th century. Boldsen and Søgaard (1998) also detected an insignificant decrease in mean stature of approximately 2 cm overtime at Tirup using stature as measured in the grave. The decrease in stature in the grave over time seen in the current study would be consistent with these broader results at both Sejet and Tirup, and it is important to note in this respect that the results from the current study reflect largely the Sejet sample, as only 7 individuals were available from Ole Wormsgade as opposed to the 63 from Sejet. If the trend of a decrease in mean 'actual' stature is compared with the decreased residual height (effectively reflecting the lack of a significant change in femoral length), we would appear to be a change in proportions over time, with the overall stature decrease potentially reflecting a change in exposure to stress over the course of development or a population change towards people with shorter stature but with more concentration of stature in the limbs. Further examination of these patterns for both Sejet and Ole Wormsgade using the larger cemetery samples would be informative, but was not possible within the scope of this current project.

On the other hand, Arcini (1999) finds no evidence for consistent stature change over time in medieval Lund, but this is based on reconstruction of stature from long bones

using the generic regression formulae developed by Sjøvold (1990). This also consistent with Gustaffson et al. (2007), who found no change in stature or in sexual dimorphism from the 10th through to the 20th century in Swedish populations. Once again, Gustaffson et al. (2007) used the Sjøvold (1990) regression formulae applied to femoral lengths. Furthermore, the use of a composite sample of measured heights from more recent populations and stature computed from femoral lengths for earlier periods is problematic with this study. A further study by Boldsen (1993) using multiple sites from 1100 A.D. onwards and measuring both femur length as a proxy for height and more recent 19th and 20th century census data found no temporal changes, which would also be consistent with the findings based on femoral length for the current study. In further support of this pattern, Yoder's (2006) multi-site analysis using femur length, found no significant changes over time at any of her sites, for either males or females.

Farther afield, Steckel (2004) also finds a decrease in stature from 173.4 cm in the early medieval period to approximately 167 cm quite a bit later in the seventeenth and eighteenth centuries in northern Europe. He suggests that this substantial decrease might be due to deteriorating climate, growing urbanization, and an increase in trade economy with its facilitation of the spread of infectious disease. In Denmark, the shift towards urbanisation was likely slower than it was in England (Hybel and Poulsen, 2013). Furthermore, while some diseases such as leprosy appear to have declined in Denmark over the medieval period (Boldsen, 2001), other diseases such as tuberculosis likely became more common (Boldsen and Mollerup, 2006). Boldsen and Søgaard (1998) have further emphasised the potential contribution of such factors as inbreeding to patterns in

adult height, and it is likely that increased urbanisation over time would have led to different patterns of population movement and contact.

Ultimately, larger sample sizes for the later period are required in order to draw any firm conclusions about stature change over time. It is possible that the differing results across studies are reflective more of these issues than of real situations. However, the consistent decrease in stature when measurements are based on in grave measurements, suggests that this may be the dominant pattern. Furthermore, if the differing trends seen in the current study between stature in the grave and femur length represent real differences in how the two variables change over time, this could be influencing the findings from other studies and needs to be taken into consideration.

The results from this study found no significant change in AS over time, suggesting that the number of stressors facing children growing up did not change substantially. This is once again consistent with Arcini's (1999) findings for DEH which showed no substantial change over time. However, the results from disease prevalence rates suggest that infectious disease did on some levels exhibit temporal change. None of the diseases scored showed statistically significant changes over time, but non-significant fluctuation was visible. Treponema was slightly higher in the later period, with this difference being significant in males but not females. There was no statistically significant change in tuberculosis prevalence over time. The analysis of tuberculosis also needs to be treated with caution, however, due to the uncertainties in TB differential diagnosis. In the absence of vertebral changes characteristic of Pott's disease, diagnosis was made based on the presence of lytic lesions in the vertebral bodies with little to no

new bone formation and the presence of lytic lesions affecting other joints (also with little remodelling) (Aufderheide and Rodriguez-Martin, 1998; Jørgensen, 2009; Waldron, 2009; ADBOU, 2010). However, the difficulties surrounding diagnosis combined with the extremely low frequency of osteological involvement characteristic of tuberculosis cases (Aufderheide and Rodriguez-Martin, 1998; Jørgensen, 2009; Waldron, 2009) means that its frequencies in this analysis will almost certainly be underestimated for both periods. It is therefore possible that these numbers do not capture the full scope of patterns in tuberculosis, and thus cannot be considered conclusive. It is furthermore possible that some of the cases diagnosed as tuberculosis may in fact be indicative of another pathological condition such as brucellosis (Aufderheide and Rodriguez-Martin, 1998; Ortner, 2003; Mays, 2007).

An increase in rates of treponema for the later period would be consistent with the literature which suggests that syphilis at least was a New World introduction and so did not appear in the Old World until after the return of Columbus at the end of the fifteenth century (Armelagos et al., 2012). However, it is unclear exactly what strain of treponemal infection is present in these samples (Pedersen and Boldsen, 2008) and so while these frequencies certainly are compatible with a New World origin hypothesis, there is still uncertainty in this interpretation. The lack of statistical significance with small sample sizes restricts any interpretation of these results. Successful isolation of microbial DNA from these skeletal remains would be advantageous in answering these questions, but this has proven challenging in the past (Bouwman and Brown, 2005; von Hunnius et al., 2007).

The patterns seen with leprosy rates are consistent with what has been found elsewhere (Boldsen and Mollerup, 2006; Boldsen, 2009). These results indicate that there was a higher prevalence rate of the disease in the earlier burials, although this was once again not statistically significant. We also see a different trend in sex distribution from the earlier to the later period, with it being more common in males during the earlier period, but being diagnosed as probable amongst females slightly more frequently during the later period. A pattern of higher prevalence for the earlier period and a decline after A.D. 1400 is consistent across Denmark (Boldsen and Mollerup, 2006; Boldsen, 2009). It is suggested that this decline, particularly in urban centres, was due to the introduction of leprosaria in the 13th century and to the consequent increased segregation of individuals with the disease (Boldsen and Mollerup, 2006; Boldsen, 2009). The disease appears to have been virtually eradicated in the main population by the 16th century (Boldsen and Mollerup, 2006; Boldsen, 2009).

Consistent with Yoder's (2006) and Arcini's (1999) findings, the results for changes in health over time are fairly mixed. Given these results, it is not possible to reject the null hypothesis that there was no change in health over time in these samples. We see a possible decrease in stature, which might be indicative of stress early in life with consequent growth stunting. However, this is not substantiated by any change in the number of early life stress events recorded as accentuated striae of Retzius. The lack of change in mean age at death also suggests that there was no significant change in population health as it related to longevity. Finally, the evidence for changes in infectious disease prevalence is mixed. The treponemal and leprosy results for an increase during

the later period and a decrease over time respectively are consistent with historical evidence and with what is known from other sites (Boldsen and Mollerup, 2006; Boldsen, 2009; Harper et al., 2011). The lack of a significant change in rates of tuberculosis over time may on the surface suggest that this disease was relatively constant in the population, but further analysis using larger sample sizes, and possible supplementing with molecular techniques would be necessary to gain more conclusive insight.

While Yoder (2006) found extremely mixed results for patterns health over time, Quist and Grøntved (2001) found evidence for deteriorating health from the 11th to the 14th centuries in an increased prevalence for chronic otitis media sequelae. While Arcini's (1999) results for medieval Lund were overall quite variable, she did observe higher rates of subadults in the later period, which she took as an indication of higher rates of infection leading to higher child mortality (although this could also be indicative of higher fertility rates as suggested by Benedictow [2012]). Arcini (1999) also found a decrease in periostitis over time, which would suggest a decline in infectious disease.

These mixed results speak as a cautionary note for the interpretation of change over time. The low number of individuals representing the later period marks a severe imbalance in the samples, and the later period sample cannot be considered representative of the population as a whole, particularly not the living population. Difficulties in comparing disease prevalence rates across different samples has been highlighted by Wood et al. (1992) and this further makes the consideration of evidence from different studies problematic. As will be discussed further in the next chapter, there are many ways in which our interpretation of these patterns is restricted and many areas which

require development to gain further insight into patterns of health in these past populations.

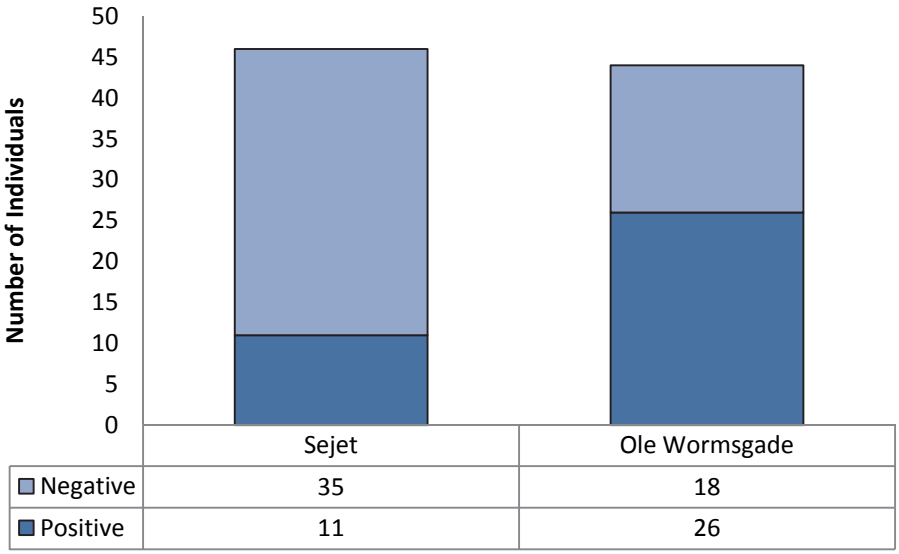
Site Differences

The results from this study show that while females had a slightly lower mean age at death than males, this difference was not statistically significant. The difference is consistent with that seen in the larger cemetery populations from both Sejet and Ole Wormsgade. Differences are apparent in the adult mean ages of death between these subsamples and the larger cemetery populations. At Sejet, both sexes in the subsamples have lower mean ages of death than in the broader cemetery samples, and the difference between the cemetery subsample and the broader cemetery mean age at death was statistically significant for males. Both males and females in the Ole Wormsgade subsample on the other hand have higher mean ages at death than those from the larger adult sample and this difference was negligible in the case of males but was statistically significant for females.

These differences may be a product of the smaller sample sizes, and may also have been influenced by the selection criteria factoring out the most worn teeth (and thus the older individuals) (in the case of Sejet at least). A further influential factor is the fact that these individuals had their age estimated again as part of this research. This resulted in different ages for individuals in some cases, and this was particularly the case for a slightly higher percentage of individuals from the Ole Wormsgade sample, which had previously been assessed by multiple observers with different levels of training (see Appendix 2 Individuals with Age Re-Assigned). This resulted in more individuals being

aged lower at Sejet and higher at Ole Wormsgade (Figure 5-1), which is consistent with the discrepancies seen with the mean age at death scores.

Figure 5-1 Bars depicting the directional change in age for individuals in the Sejet and Ole Wormsgade subsamples. A negative change signifies the use of a lower age in the current study than was originally recorded and a positive change signifies an older age estimation. See also Appendix 2 Individuals with Age Re-Assigned.



The scoring used in the present study made use of standard osteological age estimation features in associated with expert inference techniques (Weise et al., 2009, 2012; Milner and Boldsen, 2012a; b). As such, it is as accurate as possible given current age determination techniques. Transition analysis could not be employed for either sample due to preservation issues which prevented the assessment of essential skeletal features (Pedersen and Boldsen, 2008, 2010). However, Boldsen (1988:340) has noted that "regardless of the power of the statistical techniques to control for bias, the ultimate accuracy of the mortality profile is limited by the power of the age techniques initially employed." These age estimation techniques, regardless of how comprehensive and

inclusive of information, are necessarily restricted by the very nature of adult skeletal material. The most productive approach is to conduct the age assessment using all available information (Milner and Boldsen, 2012a), and to be familiar enough with the sample to establish age based on the range of changes and patterns seen within that particular sample. This is the approach which was taken during the current analysis.

The discrepancy in ages, however, highlights the innate difficulties with skeletal age determination, with inter-observer differences being apparent in the final results. The extent to which this can influence our interpretation of health in past populations based on age at death statistics is a pronounced difficulty which must be born in mind with any interpretation. However, the fact that females consistently had a lower mean age at death in both samples and across different age determinations would seem to isolate a very real phenomenon in the health of these populations.

The lower mean age at death in females is influenced by fewer females living into the later ages (i.e., past 50 years of age). A good deal of the pattern seen in women is likely influenced by childbirth complications. A similar demographic pattern has been observed at the nearby rural site of Tirup (Boldsen, 1998; Usher, 2000) where Boldsen (1997a) notes that the different mortality profiles are focused around more women dying towards the end of their reproductive age. The cemetery of Viborg Sct. Morten also had a similar demographic profile, with the mean age for males being 2.2 years older than that for females (Tarp, 2011). Arcini (1999), however, finds no sex specific difference in mortality for medieval Lund. Many (74%) of the individuals in Arcini's sample had only sufficient preservation for age estimation based on suture closure and / or tooth wear. As

the strength of both of these indicators for age estimation is limited (Perizonius, 1984; Boldsen, 1991, 2005a; Lynnerup and Jacobsen, 2003; Milner and Boldsen, 2012a), the ability of these estimates to inform interpretation beyond very broad patterns must necessarily be considered with caution.

Yoder (2006) identifies either no sex difference (at St. Mikkel and Ribe) or a higher mean age at death in females (at Øm Kloster) in her study. Boldsen (1984b), on the other hand, does see a difference with the St. Mikkelsgade sample with women having higher mortality rates during their childbearing years. This study made use of a proportional method and maximum likelihood principles (Boldsen, 1984b, 1988). Further advances to statistical techniques for reconstructing mortality profiles (Boldsen et al., 2002) could well impact the mortality reconstruction of this sample. Yoder (2006) employs the development of these statistical techniques in the form of transition analysis (Boldsen et al., 2002) as part of her age assessment for her samples, and this may explain the discrepancy. Of her sites, the Øm Kloster population might be expected to be most similar to Sejet, since the lay component of this monastic site might approximate a rural sample. It is also the closest in proximity to Horsens. The pattern seen at Øm Kloster is, however, different on a number of levels from that at Sejet. It is males at Øm Kloster who have the lower mean age at death (33.31 years of age as opposed to the female 35.16 years of age), and the mean ages at death are lower than those found at either Sejet or Ole Wormsgade. The lay population at Øm Kloster appear to have lived highly active lives compatible with patterns which might be expected for a population regularly involved in farming activities and are thus likely largely representative of a rural population

(Campbell, 2007). At the same time, the cemetery was associated with a rural monastery (Boldsen, 1990b) and thus likely includes a range of people from different social strata with slightly different patterns of population movement than a typical rural village. Lille Sct. Mikkelsgade, being a parish cemetery located just outside of Viborg and the urban cemetery of Ribe perhaps more closely approximates the type of population seen at Ole Wormsgade. No significant difference in mean age at death is visible for either males or females, but the mean age at death for individuals is closest at Ribe (37.57 years of age for the overall sample) (Yoder, 2006).

These are basic comparisons based on mean age at death calculations and do not involve more complex comparison of mortality profiles across Denmark. However, they do serve to highlight possible variability in the sex distribution of mortality. While individuals at Ole Wormsgade, Sejet, and Tirup followed a similar pattern, other sites such as Øm Kloster showed females with higher mean ages of death and still other sites (St. Mikkel and Ribe) show no sex specific differences or variable results (with St. Mikkel being slightly problematic due to results differing by study). This variability in patterning clearly could benefit from still further exploration on a wider scale. The overall adult mean ages of death for both Sejet and Ole Wormsgade fall within the range of other sites.

Despite the different patterning due to the re-assessment of individual ages for this study and the resulting differences in mean age at death for males and females, the patterns observed in the present study are consistent for females with that seen in the larger sample, namely that they lived slightly longer at Ole Wormsgade. With the

subsample selected for this study, males also lived longer at Ole Wormsgade. However, for the wider samples we do not see this pattern. Namely, males at the two sites have very little difference in mean age at death, and the combined sexes also see a minimal difference in adult mean age at death between the two sites. Given these results, it cannot be concluded that there was any appreciable difference between the rural and urban sites, at least with this variable.

The height difference between males and females is unsurprisingly consistent regardless of whether the entire sites or the subsamples from this study are utilized. There was a variability in the final mean statures however for Sejet, with individuals (and particular males) having lower mean statures for the complete samples. Stature for the subsamples used here therefore appears to be representative at least of the broader cemetery samples.

Stature was available for significantly more individuals at Sejet than at Ole Wormsgade for the subsamples due to the truncation of many burials at Ole Wormsgade. While stature in the grave is the most accurate estimation for anatomical height (Boldsen, 1984a; Petersen, 2005, 2011), slightly more individuals were available from Ole Wormsgade if the femur was used. However, even with the femur the sample numbers in the subsamples are vastly unbalanced and so any inter-site patterns based on these comparisons are problematic. A similar issue arises if residual height is considered, as the sample sizes in this case are limited by the number of individuals with stature in the grave measurements from Ole Wormsgade ($n = 7$). The individuals from these subsamples showed the mean for Ole Wormsgade being lower for males ($p = 0.050$), but

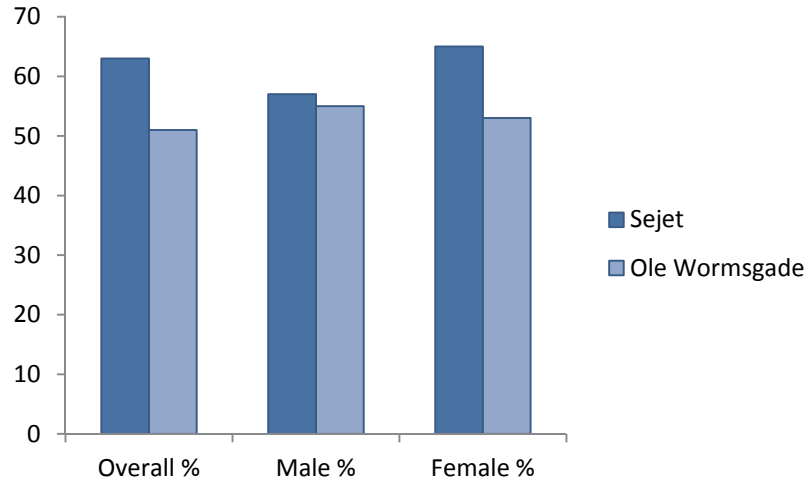
roughly the same for females when femoral length is used. There was no statistically significant difference in residual height between the two sites.

As there are far more individuals in the complete cemetery populations and as there is a more equal distribution between sites, it will be useful to consider the samples for the inter-site comparison. When the information from the complete cemetery samples was considered, the mean statures for the two sites were not appreciably different (N =90 for Sejet; N =49 for Ole Wormsgade) (Table 4-50). While there is an imbalance in sample size based on these data (derived from the ADBOU databases), the lack of a statistically significant difference with these larger samples suggests that this is accurately reflecting a clear consistency between the two sites.

These results are not consistent with other research which has found a difference between rural and urban stature in Denmark, with rural stature estimated to have been as much as 8 cm shorter than urban stature (Boldsen, 1990b). These findings are further identified using a multi-site comparison across Eastern and Western Denmark (Boldsen, 1993). Boldsen (1990) attributes the difference in heights between Tirup and Lille Sct. Mikkelsgade to the likelihood of more inbreeding in the rural population of Tirup compared to the more diverse urban centre at Lille Sct. Mikkelsgade. The lack of a difference between Sejet and Ole Wormsgade may indicate that there was greater population movement between the rural and urban centre in this case, making for a more uniform population between the two.

The consideration of differences in childhood stress showed no significant site differences for either the combined subsample or for males and females considered separately. Individuals at Ole Wormsgade did have a higher mean number of AS, but this was not statistically significant and individuals at Sejet showed a slightly (not significantly) higher number of DEH on average. It was not possible to directly compare the number of AS with the larger sample, since AS cannot be scored for more individuals than were selected for the current subsample. Frequencies of DEH for these samples though, as provided in the skeletal reports (Pedersen and Boldsen, 2008, 2010), suggest that (consistent with this subsample) more individuals suffered from DEH at Sejet than at Ole Wormsgade (Figure 5-2). As will be discussed in the next section on the relationship between AS and health from these sites, DEH may be most compatible with strong / severe AS as defined by Thomas (2003). This is consistent with the observation made early on by Rose (1977) when he noted that DEH was caused by the convergence at the enamel surface of accentuated striae of Retzius (see also Goodman and Rose, 1990). However, as formal testing of the direct relationship between these two features in the Danish populations proved to be beyond the scope of this thesis, further analysis is required before this link can be drawn more conclusively. It will be useful to re-evaluate this situation once a scoring system more similar to the one used by Thomas (2003) has been developed. This system should adjust for differential expression of defects across the crown, and incorporate a structural understanding of AS formation and expression such as that presented by Witzel and colleagues (2008) (see also Chapter 6, Future Directions).

Figure 5-2 Overall sample dental enamel hypoplasia prevalence. Data acquired for Sejet from Pedersen and Boldsen (2008) (unpublished anthropological report) and for Ole Wormsgade from Pedersen and Boldsen (2010) (unpublished anthropological report).



The interpretation of overall patterns of disease in these subsamples needs to be treated cautiously due to data limitations. Based on the estimates of disease prevalence from these subsamples, some site differences were apparent. Both tuberculosis and leprosy were significantly more prevalent at the rural site of Sejet than at the urban site of Ole Wormsgade. Treponema was also very slightly more common at Sejet, but this was not statistically significant. An examination by sex shows that females had higher rates of tuberculosis than males at both Sejet and Ole Wormsgade, while males show higher rates of leprosy and treponema at both sites. The complete cemetery samples, based on the values provided in the cemetery reports show a number of clear differences from those patterns visible in the subsamples used in this study (Table 5-1) (Pedersen and Boldsen, 2010). Unlike the subsample, it is the Ole Wormsgade population which appears to have higher prevalence rates of all three diseases (Pedersen and Boldsen,

2008). The sex differential also varies, with females at Sejet but males at Ole Wormsgade showing higher rates of treponema (although similar to the subsample pattern, these differences are not drastic for this disease). Both males and females show higher rates of tuberculosis at Ole Wormsgade in the larger cemetery samples, while this is only the case for females in the smaller cemetery sample. Finally, while males at both sites in the larger cemetery sample do show the higher rates of leprosy, consistent with the subsample pattern, there appears to be a complete reversal with the balance shifted over to Ole Wormsgade in terms of site dominance for both sexes with this disease (i.e., the disease is more common in both sexes at Ole Wormsgade rather than Sejet).

Table 5-1 Broader cemetery infectious disease prevalence rates integrating information from the complete cemetery populations. Data for Sejet acquired from Pedersen and Boldsen (2008) (unpublished anthropological report). Data for Ole Wormsgade acquired from Pedersen and Boldsen (2010) (unpublished anthropological report).

	Sejet				**Ole Wormsgade**			
	Males		***Females***		***Males***		***Females***	
Treponema	7%	N =5	8%	N =5	23%	N =29	20%	N =23
Tuberculosis	4%	N =3	7%	N =4	44%	N =63	33%	N =30
Leprosy	21%	N =13	11%	N =6	54%	N =60	53%	N =41

It is thus clear that the patterns seen with the current subsamples are not entirely consistent with the larger cemetery populations and so the patterns in terms of sex distribution in the infectious diseases covered by the subsamples cannot be extrapolated into broader interpretations of sex differences in susceptibility. Tuberculosis, rather than being consistently higher in females (at both sites), shows a more variable sex distribution

in the broader sample. Treponema in the larger sample is no longer consistently higher in males at both sites, but rather is higher in females at Sejet but in males at Ole Wormsgade. Leprosy, on the other hand, does show consistently higher rates in males whether the subsample or larger cemetery sample is considered. Overall, there is a shift in balance from the subsample to the greater cemetery samples from higher rates of infectious disease at Sejet to higher rates at Ole Wormsgade. It is possible that this reflects the differential site preservation in combination with the selection criteria. Individuals were selected for cranial material, and thus individuals at Ole Wormsgade who were truncated from the head down rather than from the postcranium up tended to be selected (individuals with lower extremities missing were still included if there was dental material preserved, but not the other way around). This may have resulted in underscoring of disease prevalence rates for Ole Wormsgade, shifting the balance over to Sejet.

The site comparison of health thus provides rather mixed results. While individuals in the subsamples showed a significantly lower mean age at death at Sejet, this may be a result of sampling bias and the overall samples show no significant difference in this variable between sites. There was furthermore no significant difference in stature when the overall samples were considered. We do see higher rates of infection in the urban centre of Ole Wormsgade in the larger sample, but while there was a slightly higher frequency of AS at Ole Wormsgade, this was not statistically significant and the overall sample suggests very slightly higher levels of stress at Sejet. As such, there do not seem to be significant inter-site differences in health except for the higher incidents of

infectious disease in the urban centre. This is compounded by the broad span of time represented by these subsamples. In particular, the Ole Wormsgade sample is skewed towards a slightly later timeframe, and so this later period may have a stronger influence on this sample. However, with both cemeteries the later period sample is so small that it is suggested to have limited influence. Larger sample sized would be necessary to gain further more conclusive insight into the relationship between these parameters (time and place).

Childhood Stress and Adult Health

The overall higher prevalence of disease in males at Ole Wormsgade, and the higher prevalence of leprosy in males at Sejet, combined with higher frequencies of AS might suggest that overall males were more stressed than females in these samples. When the complete cemetery populations are considered, males are also diagnosed with all three diseases at both sites more often than females (with the exception being treponema which is 1% more common in females at Sejet). There thus appears, from these results to be some difference in health between males and females in these populations. There are a few possibilities for interpretation of these patterns. Both will be considered over the course of this discussion. In the first case, it is possible that cultural factors played a role in different approaches to childrearing between boys and girls. The alternative interpretation lies in a biological difference to stress response between boys and girls.

The higher rates of disease in general combined with higher counts of AS in males might be interpreted as males being less buffered culturally from stress and illness or as

males suffering from more stress than females at these two sites (and especially at the urban site of Ole Wormsgade). Alternatively, in line with the points made by the *Osteological Paradox* (Wood et al., 1992) it is necessary to consider the hypothesis that males were actually more robust and therefore more likely to survive childhood stress incidents (and to survive diseases long enough to sustain osteological changes). However, the consistently more negative correlation between the number of stress events indicated in the dental enamel and age at death amongst the males in this study (as opposed to the females) may argue against the second interpretation. The relationship between childhood stress and adult health is extremely complicated, and is influenced by a variety of factors. A review of this literature in relation to the results is therefore essential to this discussion.

The work which has been done with modern populations suggests that early life experiences of stress can lead to permanent phenotypic changes in individuals (Barker et al., 2002; De Boo and Harding, 2006; Devaskar and Raychaudhuri, 2007; Jirtle and Skinner, 2007). These relationships have been identified between a number of early childhood stressors and later life health problems (Barker et al., 1989; Eriksson et al., 1999; Gern et al., 1999; Bo et al., 2000; Lemanske, 2002; Hales and Ozanne, 2003; Burdge and Lillycrop, 2010). Limitations with both cross-sectional and longitudinal studies of modern populations, however, highlight potential contributions which can be made by the study of past populations. By identifying specific indicators of (nonspecific) childhood stress in association with health information from the same individuals on large population scales, studies of past populations can help to inform this study both for

modern and past populations. It has been noted previously that dental enamel hypoplasia in the first 18 months of life are most likely to be associated with final height for age measurements, suggesting a particularly strong impact of these early life stress events (Floyd and Littleton, 2006). This is in line with the vast literature surrounding the fetal and early life origins of health and disease (Barker et al., 1993, 2002; Barker, 1995, 2001; Desai and Hales, 1997; Eriksson et al., 2000; Hales and Ozanne, 2003) and is consistent with the rapid development taking place in infants during this period (Bogin, 1999).

This thesis captures a slightly later period of childhood development in the dental enamel than many of these studies. However, the results do identify certain patterns which may be related to such relationships. As discussed in the literature review, there are still a number of systems developing over this period (Bogin, 1999; Chamley et al., 2005; MacGregor, 2008), and a stressor which stimulates a stress response may impact these developing systems. Goodman and Rose (1990) discuss considered the importance of the closely interrelated factors of stress severity, duration, and timing working together with individual frailty and stress response in the formation of dental enamel defects. It was predicted in this thesis that stress events as indicated by dental enamel disruption may also be sufficient to impact a concurrently developing systems, thus resulting in a correlation between the enamel defects and adult health. While correlations were apparent from the results in some cases, it is also apparent from this study that the situation is complex and that further research using larger samples and standardized methodology is required.

One particular aspect of consideration is that the stress thresholds used in this research were assigned solely based on the frequency distribution of stress markers, and thus may inadequately capture stress thresholds as they relate to individual health and frailty. Currently, our understanding of such stress thresholds as they relate to severity, frequency, and timing of stress events to health outcomes is poor. Other arbitrary thresholds were tested over the course of this research, and some suggested stronger and even statistically significant correlations between the frequency of AS and age at death. It may be that a better understanding of where these thresholds should be established will aid in the development of a better identification of any patterns. In the absence of such an understanding, the frequency distribution was used to establish thresholds. However, though not necessarily having a strong biological basis but rather a statistical one, it is possible that this division might cloud some of the biological patterns.

Despite these limitations, the results from this research do show correlations between dental enamel defects and adult health indicators which are largely consistent with previous findings. Past investigations of DEH in association with adult mortality risk have found that the presence of DEH has a negative association with age at death in males, but not in females (Boldsen, 2007; DeWitte, 2010). This is consistent with the survival analysis of DEH and with patterns in AS. The broadly positive or neutral trend seen in females in relation to overall enamel defects is thus interesting and seems counterintuitive to what might be predicted by epigenetic theory (Devaskar and Raychaudhuri, 2007) and the developmental origins of health and disease theory (Barker, 2001, 2004a, 2007; Barker et al., 2002; De Boo and Harding, 2006). In these situations,

more stress is seen to have an adverse impact on health (De Boo and Harding, 2006; Boldsen, 2007) and as will be discussed shortly, a particularly adverse impact on males (Stini, 1969; Stinson, 1985; DeWitte, 2010; Bale, 2011). Thus, the overall patterning between males and females for this study is consistent with that which has been observed previously. However, the positive trend for females in relation to overall AS frequency, while not statistically significant, requires further investigation as it would seem to suggest that in some circumstances at least, more stress incidents in females may actually be connected in some way to increased survival.

There is a precedent for the pattern seen with the present results in the anthropological literature. Thomas (2003) found a complex relationship between AS and mortality which seemed to be dependent on the severity of AS. Thomas (2003) distinguished between 'weak' and 'strong' AS in her analysis and found that more AS were associated with a greater risk of dying before the age of 30. However, weak AS occurring before the age of seven were associated with a reduced risk of dying between the ages of seven and thirty. She suggested that a possible explanation for this pattern was that individuals who exhibited weak AS and those who exhibited strong AS for the same age range were likely subjected to similar illnesses but that the more robust individuals were not as severely impacted and so only developed weak AS. This would be consistent with those individuals then being more likely to live longer. Alternatively, it is possible that the weak AS mark less severe stress episodes which served to strengthen an individuals' immune response rather than to weaken their ability to deal with conditions through life.

Unfortunately, Thomas (2003) did not consider sex differences in her sample (partly because it seems that relatively few individuals in her sample could have sex determined), and so it is not clear to what extent this contributed to the mortality patterns. If females in her sample expressed more weak striae, it may well be that these two factors (i.e., weak AS being more common in females and reflecting a different stress response) are connected in the positive relationship she observed. This will be discussed further later in this chapter in the discussion of differential expression of defects and the nature of stress response. The fact that females at Sejet and Ole Wormsgade exhibited fewer AS in general might be suggestive of fewer strong defects to count in females. If DEH can be considered the surface manifestation of severe AS (Goodman and Rose, 1990), then the tendency of females generally to have fewer DEH's in the current investigation (apart from the one outlier) suggests that it might be weak AS which are influencing the current positive findings. Further testing of the Tirup sample would be informative in this respect, as would the application of a strength-based system to the current sample. However, there are likely confounding factors implicit in any methodology which divides AS based on how prominent the structure appears (along with benefits to such an approach). In order to better understand the similarities and differences between these two sets of results, a brief review of Thomas' methodology will be useful.

There are a couple of key differences between Thomas' research and the current study beyond the absence of sex differentiation in the Tirup sample. The first of these is in the definition of AS and the second lies in the nature of the sample mortality profiles. While striae of Retzius are recognized as histological structures which have a regular time

periodicity associated with a particular individuals' circaseptan rhythm (Risnes, 1986; Antoine, 2000; Reid and Ferrell, 2006; Smith, 2006; Antoine et al., 2009), and while accentuated striae of Retzius or Wilson bands (Goodman et al., 1992) and the associated surface changes (such as dental enamel hypoplasia) have been directly linked to the occurrence of 'stress' early in life (Mellanby, 1929, 1930, 1934; Suckling, 1980; Suckling and Purdell-Lewis, 1982; Suckling et al., 1983, 1986; Suckling and Pearce, 1984; Goodman et al., 1991; Schwartz et al., 2006), the identification of these accentuated structures is highly problematic (Goodman and Rose, 1990; Thomas, 2003). In reality, the appearance of accentuated striae will depend upon their location in the crown (Hillson, 1996; Hillson and Bond, 1997) and on the location of the histological section in relation to the tip of the dentin horn (Antoine, 2000; Antoine et al., 2009). This means that the extent to which an AS might appear 'weak' or 'strong' will to a certain extent at least depend upon factors which are independent of individual frailty and stress parameters (timing, duration, severity). However, since part of this variability is dictated by the location of the defect and since location is related to age at occurrence, an interpretation of AS patterns which seeks to gain insight into individual frailty will necessarily be confounded by the fact that individual frailty may well change in association with immune development through childhood (and by extension frailty may be variable across crown development).

Thomas' (2003) research sought to deal with some of the difficulties in defining AS by distinguishing between those which are clearly identifiable AS which extend at least three-quarters of the way from the dentin-enamel junction to the surface of the

enamel (Goodman and Rose, 1990; Goodman et al., 1992) and those which are less clear but which nevertheless seem to represent some level of accentuation. It is these latter structures which are the most problematic from an identification standpoint. Perhaps the most beneficial aspect of Thomas' (2003) approach is effectively the establishment of a higher threshold for clear defects while still incorporating less clear defects. The difficulty still exists, however, when it comes to attempting to define both the lower and upper thresholds for weak AS (and the lower threshold for strong AS). Through her approach, Thomas (2003) adopted Goodman and Rose's (1990) strict definition with her strong AS designation, but also extended her analysis to include other less clearly defined defects. While Goodman and Rose (1990) maintained that defects would either be present or absent depending on whether a stress threshold was breached, the different patterns observed by Thomas (2003) suggest that the different expressions of AS may have some connection to the true individual-stress interaction.

However, the potentially confounding factor of appearance varying based on location in the crown is essential to consider. An evaluation of the rates of weak AS in Thomas' (2003) results show that they are most common early on in development (from birth to six months of age) and gradually decline in frequency. This would be consistent with the overall appearance of striae over the course of crown development. The cuspal area tends to have clean lines of AS which can appear quite faded. By the time the mid-crown is reached, lines are far darker and closer together overall and lines towards the cervical area are extremely difficult to identify unless they are quite accentuated. The fact that weak AS at Tirup were associated with a positive influence on health may

therefore also have to do with the fact that they are most likely to be counted early on in life when infants are still buffered by the protective factors which are so important in breast milk (Labbok et al., 2004; Field, 2005; Jackson and Nazar, 2006).

While both the current study and that conducted by Thomas (2003) looked at the full crown development and tried to consider possible changing patterns with age at occurrence of stress events, the cuspal enamel is often lost due to wear in older individuals. This led to a further difference in approach between the two studies. Studies of AS typically choose teeth which are unworn so as to preserve the entire duration of development, and this was the approach taken by Thomas (2003). In this way, she was able to include a larger sample with the early stages of development captured (0 – 2 years of age). She also sampled M_1 to M_3 to help capture the entire span of development represented by dental enamel. This also made it possible for her to identify AS across multiple teeth to ensure the systemic nature of the defects. This is an ideal approach in many respects, but it does have some issues – both practically and theoretically. On the practical level, it involves destroying multiple teeth from each individual which is not ideal for archaeological samples. On the theoretical level, by restricting the sampling to individuals with minimally worn teeth, there was an upper age restriction – the oldest individuals in the sample were estimated to be 32 and 33 years of age, with all other individuals aged below 30 years. This means that while survivors of childhood stress experiences were sampled as well as non-survivors, no individuals who successfully survived to older age were sampled. With this type of study, unfortunately, either the

earliest stress events or the oldest individuals will be underrepresented depending on which sampling direction is taken.

The current study, by choosing to allow for loss of the earliest life stress information in order to incorporate older individuals, captures a slightly different age demographic than Thomas' study. Understanding the implications of the results from these different approaches is far from straightforward. Thomas' findings highlight the possibility that AS can be related to positive, as well as negative health outcomes. The current results point to the sex-specificity of some of these relationships. It is still unclear, however, why a positive outcome was seen with females in the present study. To gain further insight into this finding, a review of what is known about sex differences in stress responses and health will be beneficial.

Unfortunately, some of the mechanisms behind sex differences in health are still poorly understood, and there is a gap in the literature covering differences in developmental timing between the sexes with systems early on in development. In fact, studies are only starting to look at the sex differences to stress response (Weinstock, 2007; Buss et al., 2009). This is an area which is beginning to receive attention, however, and light is starting to be shed on the different responses to early life experiences by males and females (Bale, 2009, 2011). It is likely that hypothalamic-pituitary hormones and sex steroids have an impact on systems from a very early stage and are influential in system development. Increasingly, sex (chromosomal makeup) and sex hormones are being associated with differing responses to environmental stressors in varying forms in both humans and non-human animal models (Ahmed et al., 1985; Klein, 2000; Verthelyi,

2001; Beagley and Gockel, 2003; Lang, 2004; Bouman et al., 2005; Wang et al., 2006; Carey et al., 2007). Fairly early on, Stini (1969) noted a stronger ability in Colombian girls to adjust to malnutrition and to maintain stable health while more pronounced long term effects of protein deficiency were visible in males. Hormonal influence was cited as a possible influential factor to these differing responses.

Women generally mount a stronger humeral and cell-mediated immune response (Verthelyi, 2001; Shames, 2002) and are better able to combat infection from a number of viral, bacterial, and parasitic infections (Stinson, 1985; Roberts et al., 1996; Beagley and Gockel, 2003) than men. At the same time, the complex and still not fully understood interaction between hormones and immune mechanisms means that autoimmune disorders and allergic diseases preferentially affect women (Ahmed et al., 1985; Stinson, 1985; Shames, 2002; Gleicher and Barad, 2007; Fairweather et al., 2008). Furthermore, while women respond to stress in the form of a variety of infections and trauma by increasing antibody production, the inflammatory response is higher in males and this can lead to higher mortality (Klein, 2000; Lang, 2004; Wang et al., 2006; Fairweather et al., 2008). Beyond differences caused by differing immune function, Weinstock (2007) also discusses varying behavioural and neurological disorders between men and women, linking this to a sexually differentiated foetal response to increases in corticosterone levels caused by maternal stress. Thus it is clear that on many fundamental levels males and females have very different health outcomes and responses to stress in varying forms. This would appear to begin early on in development, and the sexual dimorphism of these patterns at these early stages is still poorly understood (Bale, 2011).

Sex differences in response to early life (both prenatal and postnatal) environmental stressors have been documented in both humans (Bouman et al., 2005) and non-humans (Dominguez et al., 2003; Domínguez et al., 2003; Carey et al., 2007), but Bale (2011) notes that sexual differentiation (along with gonad and brain development) occurs after early pregnancy, and so many of these systems would not be impacted by particularly early fetal experiences. It is possible that early epigenetic modification of the Y chromosome SRY promotor region could have an effect on sexual differentiation which occurs slightly later (Bale, 2011) and that a sex-specific stress response could be tied into this early development. Thus, the possibility of sex-differentiated early prenatal influences is very real, as are possible impacts at later points in development both once sexual differentiation begins to take place and during the post-natal and the pubertal periods (Bale, 2011).

There are therefore a number of points at which a sex-specific impact could take place. The nature of the impact will depend on what is happening at that point in development, on individual genetic makeup (including sex) and on the nature of the stressor. Differing responses to stress by the two sexes are complexly tied to differential developmental patterns, and it is clear that the rates and timings are very different in males and females for a number of systems (Taylor, 1969; Buss et al., 2009). Males have been shown to have a slower rate of cortical development than females (Taylor, 1969; Weinstock, 2007). Buss et al. (2009) also observed different rates in maturation of fetal heart rate startle response with males showing larger responses and taking longer to mature (six weeks longer). Uekert et al. (2006) noticed sex-specific immune responses in

children from a birth cohort study between the ages of one and three years, indicating possible sex-specific schedules for immune development. Unfortunately, since the relationship between sex, hormones, and immune maturation is still not understood (Shames, 2002), it is impossible to fully understand where many of the sex differences begin.

Attaining insight into differential rates of development of early physiological systems is fundamental to understanding sexually dimorphic health patterns (Selevan et al., 2000; Cohen Hubal et al., 2008; Bale, 2011). External stressors will have the most impact on those systems which are developing when they occur, and if systems are developing at different points for males and females, the sexes will be impacted in a variable manner. If this continues into early childhood, then these systems would still be impacted at their unique point in development, resulting in a sex-differentiated response. Based on what we know of other systems, it seems that females in general develop at a faster pace than males, at least up until the juvenile growth spurt. It is likely that the immune system follows this trend (as is further supported by Uekert et al.'s [2006] findings). This has two primary implications. The first is that slower developing systems have more time to sustain stress (Weinstock, 2007) and the second is that more developed systems may be better able to cope with stressors. In this light, Buss et al. (2009, pp 633–634) note that

> … there is substantial evidence from animal and human studies that suggests there are different developmental consequences resulting from a variety of prenatal environmental exposures based on the offspring's sex [e.g. 32-34]. This may be either a reflection of sex-specific susceptibility to environmental insults or it may reflect different neurodevelopmental trajectories for males and females that result

> in sex-specific developmental intervals of maximum susceptibility to environmental exposures.

In their study of fetal startle responses, Buss et al. (2009) suggest that they have the first evidence for potential sex-specific developmental windows. This emphasizes the importance of more study considering differential developmental timings in males and females.

While correlation does not necessarily reflect a causal relationship, the results from the AS counts in association with mortality profiles for the Sejet and Ole Wormsgade samples would be consistent with these broad patterns of sex differences. The results may therefore suggest that males were more inclined to experience stress events on a sufficient level to produce dental enamel defects. Furthermore, we see that male mortality appears to have a negative connection to the frequency of DEH (which might be seen to represent severe manifestations of the internal stress markers) and that females not only had evidence for fewer stress events, but the overall frequency of internal stress events in females was positively correlated with a later age at death.

While these patterns are interesting and might be interpreted with respect to cultural parameters, they are consistent with expectations based on current understandings of biological sex differences (Carey et al., 2007; Bale, 2009) in health and so this seems the most parsimonious explanation for the patterns observed. If cultural buffering were responsible for the pattern observed between mortality and dental enamel defects, it would be expected that this would also be seen in females experiencing greater longevity. A consideration of non-survivors in this respect would be useful, but was not possible in

this study and would be complicated by the challenges faced in establishing sex for juveniles (see upcoming discussion). The current sample, representing only the survivors of childhood stress events, is confounded by the impact of childbirth on early adulthood for women. Higher mortality rates are apparent across the female sample, however, which would seem to run counter to the correlation seen between the frequency of childhood stress events and survivorship.

The historical literature available for the period does not speak to any differential treatment of males and females during childhood. Neither a survey of the literature nor consultation with experts in this field revealed anything that could speak directly to such patterns. While absence of evidence is not necessarily evidence of absence, there is currently seems to be nothing to suggest differential treatment of children. Nevertheless, it is possible to discuss a few scenarios for differential treatment of males and females which might have influenced their exposure to and experience of disease and stressful situations. It is possible that girls were raised in a more insular environment, staying closer to their home and to their local community. There is mention in the *Gesta Normannorum* (History of the Normans), dating to a slightly early point in time (11th century) of boys, once old enough, being forced to leave their homes to seek employment and land elsewhere (Christiansen, 1998, pp 15–16 as cited by Hybel and Poulsen, 2013, 111). This could speak to a broader differences in perception and treatment of boys and girls, which might have led to higher levels of exposure and stress in boys than in girls, along with sex-based differences in treatment of illness. While this reference precedes the period covered by the populations in this study, and therefore cannot be considered

directly relevant, it is possible that it speaks to perceptions maintained into a later period. A further important point to consider with this source is that it was written as a Norman commission and therefore may present a somewhat biased perspective of the 'Nordic people' it is describing.

Quite probably the greatest differential health impact between the sexes was the pattern of fertility and associated risks in pregnancy, childbirth, and lactation, and through caregiving activities with exposure to infection (Benedictow, 2012). Marriage may well have occurred at an early age during particularly the later medieval period, with girls being married between the ages of 14 and 20 (Benedictow 2012). Indeed, (Benedictow, 2012, p 31) notes that this period reflected

> a demographic regime characterized by early age at marriage with consequent exposure to sexual intercourse, high levels of fertility and corresponding maternal reproduction-related super-mortality and also [that] particular work-related exposure to epidemic disease [would] impl[y] a higher mortality-rate for women relative to men not only in those of reproductive age but also in preceding and subsequent working ages. This suggests that, on average, the females of premodern Europe would have had a generally lower life-expectancy than men, although this was particularly pronounced in those of reproductive age.

Unfortunately, the lack of direct evidence for differential treatment of boys and girls during development precludes any conclusions in this regard, but certainly the possibility of differential treatment of boys and girls over the course of their growth and development needs to be maintained as a possible interpretation.

An alternative interpretation of the patterns observed lies in a consideration of biological differences between males and females. A possible interpretation of the positive trend seen in females in relation to overall AS lies in the different ability of

women to deal with stress from a variety of pathogens. This research highlighted prevalence rates of three infectious diseases and found higher rates in females for only one of these (tuberculosis), and this was not a statistically significant difference. If anything, the results show that males were more likely to suffer from the only infectious disease which showed a statistically significant difference between the sexes. These results may speak to differential exposure to disease and risk of infection, to differential frailty (and also subsequent risk of health outcomes), or to sample bias due to insufficient sample sizes. However, it does not speak to higher levels of female exposure or infection. In a society faced with a high pathogen load, the ability to survive environmental stressors such as infectious diseases may have gone farther than simply being able to get past that particular barrier. Consistent with the old adage that 'if it doesn't kill you it will make you stronger,' it is possible that the girls who survived more stress events were able to build up a stronger immunity to the pathogens in their environment and to deal with them later in life, thus conferring an advantage which would actually aid in survival.

In this situation, the difference seen between males and females would lie in the stronger ability of females to deal with such pathogens in the first place, but also in the possibility of differing developmental schedules which has been outlined above. While neonates do have an active immune system, it is not fully mature at birth and so infants are more susceptible to infection than adults (West, 2002; Burns-Naas et al., 2008; Ygberg and Nilsson, 2012). If the female immune system, consistent with other systems, develops faster than the male immune system, it may be better able (at an earlier age) to mount an active response and to start building up a strong immunity to pathogens in the

environment. If this is the case, then while males will be more reliant on the various protective factors conferred by breastmilk and will be less able to actively build their own resistance until a slightly later age, females who become infected will be better able to deal with that infection and to in effect benefit from it in the long run if they are strong enough to survive. Beyond the stronger ability of females to combat infection (which is connected to a variety of factors, including hormones), such an acquired immunity might once again help to explain the lower prevalence of leprosy (the main disease we know to have been endemic in medieval Denmark) (Boldsen, 2001, 2005b, 2008) in females, since the pathogens involved in these diseases would have been present in the environment throughout their lives.

This interpretation would further be consistent with the findings for DEH and mortality in this study. It is possible that what we are seeing here is different patterning depending on the severity of the stress impact. Since the AS count includes less pronounced defects than those expressed as DEH's, it is possible that we are seeing more immune priming in females whereas more severe stress incidents may have been too much for the infant immune system to cope with, thus resulting in detrimental health impacts. This would further be consistent with Thomas's (2003) findings between weak and strong AS. In this case, the above discussion of immune development and differing patterns between the sexes might be expected to result in exactly the differing patterns observed.

Unfortunately, the other variables considered for adult health are not able to provide us with much more information as to these scenarios. There was only a weak

positive correlation between overall stature and AS for males, virtually no correlation with females, and nothing of any statistical significance. This is consistent with Boldsen's (1997b, 1998) findings with stature. Interestingly, the pattern appeared slightly different when femur length was considered, with a very slightly negative correlation for males (and an even weaker negative correlation for females). However, these correlations were low and not statistically significant and the sample sizes were too small for any conclusions to be made. It is interesting to observe these relationships when Boldsen (1998) also found no link with stature, but a negative relationship between stress and body proportions. The consideration of residual height by decile did some a significant correlation for females with the 4th decile, but the strength of this given that no further significant correlations were visible in the other deciles is somewhat suspect. The absence of a significant correlation with leprosy prevalence[36] suggests that the number of stress events early in life did not significantly impact an individuals' likelihood to develop osteological changes consistent with leprosy later in life. While the finding of no association between leprosy prevalence and mortality profile was consistent with Boldsen's (2005b) findings for Tirup, Boldsen did find that individuals with DEH were less likely to have high leprosy scores.

[36] Since tuberculosis and treponema were not scored with as sophisticated a system (Boldsen, 2005b; c, 2008; Boldsen and Freund, 2006), and since the sex distribution of these diseases is different between the subsamples used in this study and the scores recorded for the broader samples (Pedersen and Boldsen, 2008, 2010), leprosy is the best documented to consider here (i.e. the scores for leprosy likely most closely reflect the likelihood of an individual suffering from a disease). Furthermore, while we know that leprosy was endemic in medieval Denmark (Boldsen, 2005b) we have less information about prevalence rates for tuberculosis and treponematosis. It has been suggested that tuberculosis became more common with the agricultural transition as cattle rearing became practiced more and as leprosy began to decline (Boldsen, 2009). The situation of treponema in pre-Columbian times is still highly contentious and far from clear (Harper et al., 2011).

It is possible that there simply was no association between AS at Sejet and Ole Wormsgade and the likelihood of developing osteological lesions characteristic of leprosy. However, with the present results, such a conclusion might be premature. It is impossible to directly compare macroscopic DEH scores with AS, or even with the microscopic scoring of DEH used during the course of this investigation. AS and DEH are scored on entirely different levels, capturing different sorts of information. DEH scores effectively capture the most severe manifestations of AS, roughly equivalent to Thomas's (2003) most 'strong' AS scores. The microscopic DEH scores used here are more compatible, and the fact that they showed no statistically significant association with disease is interesting. However, the fact that even they are scored differently than macroscopic defects makes cross-comparison problematic.

There is still a poor understanding of what influences the manifestation of a stress event in the enamel, and it is likely a combination of stress severity, duration, timing (as it relates to location on the crown), and individual frailty (which once again may be variable over the course of crown development (Goodman and Rose, 1990; Neiburger, 1990; FitzGerald et al., 2006; Witzel et al., 2008). Due to the lack of clarity in the etiology of different AS, the current study tried to identify AS as those striae which were more accentuated in relation to the striae in their surrounding crown region. In doing this, however, it did not separate the particularly strong AS from those which were weaker but which nevertheless appeared accentuated. This may have obscured a distinction between more severe and less severe manifestations in the consideration of AS. If, as Thomas (2003) suggests, weak and strong AS have a different relationship to health, it is possible

that the inclusion of both into the same count system in the current study effectively caused one to cancel out the other. The fact that DEH scores for this study did show a negative correlation to adult mortality, similar to Thomas' findings for strong AS, would seem to support this suggestion. However, more detailed scoring of DEH microscopically is required to attain better definition than was achievable within the scope of this study. This is a point which will require further exploration, ideally incorporating a larger sample (and perhaps involving a re-assessment of the Tirup material).

The appearance of stress clearly requires a nuanced approach. The results from this study, when considered in conjunction with Thomas' (2003) findings, clearly highlight the fact that dental enamel defect formation is highly complex, and that any interpretation of these structures requires a deep understanding of the basis for their formation. Goodman and Rose (1990) have made inroads in this direction with their proposal of a threshold model incorporating various risk factors which will factor into a stress response surpassing a necessary threshold for permanent enamel response. Witzel et al. (2008) have contributed to our understanding through their analysis of ameloblast changes in relation to defect manifestations. Ultimately, all factors involved in differential expression must be fully understood, and this understanding is still limited. However, contrary to Goodman and Rose's (1990) position, the pattern of results seen in this and Thomas' (2003) research suggest that any threshold for ameloblast disruption may better be understood in terms of a sliding threshold, which is dependent upon the highly complex and changeable nature of individual frailty. This is also more consistent

with FitzGerald and Saunders (2005) position on the variable nature of expression of defects, in which they assert that all stress events have the potential to cause defects and that no minimal defect expression will be identifiable (i.e., the suggestion that no minimal threshold exists).

The very fact that these stress markers are non-specific means that they could have been caused by anything that would sufficiently activate the neuroendocrine system, autonomic nervous system (ANS) and / or the immune system so as to elicit a stress 'response'. It is likely that we will never be able to determine what stressor caused a particular defect, since the range of possible stressors which can illicit the same response is extensive (Neiburger, 1990). This can be highlighted by the point made by (McEwen, 2002, p 922) who notes that

> … contrary to the late Hans Selye, who emphasized physical stressors [174] psychological and experiential factors are among the most powerful stressors, e.g. novelty, withholding of reward, and anticipation of punishment rather than the punishment itself are among the most potent activators of HPA [hypothalamic-pituitary-adrenal axis] and ANS activity [114, 116].

Unfortunately for interpretive purposes perhaps, the etiology of these structures can therefore not be reduced to nutritional and infectious grounds (Neiburger, 1990). Only through a bioarchaeological approach which establishes a clear context could we hope to offer some level of clarity on this situation, and only then if there is a marked heightening in prevalence of severe enamel defects which can be matched with specific environmental stressors. Such a situation is unfortunately highly unlikely, and even should it occur any interpretations would need to be treated with extreme caution (Neiburger, 1990).

What we can gain further insight into, however, are the biological relationships between stress and health. If we can attain a better understanding of the connection between the physical manifestation of these structures and the etiological frameworks (in terms of the balance between individual frailty and severity of the stress event), we can gain tremendous insight into the long-term impacts of these stress events. Identifying the specific cause of the defect is perhaps not as critical as being able to understand these biological relationships. However, it is imperative that we know exactly what we are looking at first, and what leads to the variability in how these structures are manifest in the crown in far more detail, before we can gain such insight.

Summary

The results from this research on all levels point to the need for further study. The consideration of temporal patterns is restricted by small sample sizes for the later period. The patterns observed in relation to both temporal changes and site differences are variable, pointing to neither improvement nor deterioration of health over time or clear differences between the two sites. The patterns expected based on the historical record and the climatic record are therefore not necessarily borne out. We do not see particularly consistent negative health in the early period with subsequent improvement, for example. Sex differences are apparent on a number of levels. The interpretation of these sex differences is informed by differential stress responses between the sexes and the incorporation of early life stress information within the context of the adult health parameters proves invaluable in this respect. These sex-related patterns which emerge in

relation to stress and health strongly argue for the necessity of taking sex differences into account in all future studies of this nature.

Chapter 7 Conclusions and Future Directions

In relation to the three objectives of this study (to identify temporal changes, to identify site differences, and to examine the relationship between childhood stress and adult health), this research found that:

1) There were no consistent changes in health over time, and thus the null hypothesis that health did not change over time could not be disproven.
2) There were no consistent patterns in health between Sejet and Ole Wormsgade, and thus the null hypothesis that there were no differences in health between the rural and urban site could not be disproven.
3) There was a statistically significant relationship between early life stress incidents and adult mortality. This showed variable expression between males and females, with males generally experiencing more negative mortality impacts in relation to childhood stress events. These patterns were not clearly manifest in relation to childhood stress and either stature or disease prevalence.

This study has in a sense left more questions than answers about both patterns in medieval Denmark and the relationship between dental enamel defects and health. The differences between sites are mixed, but primarily show higher rates of infection at the rural site of Sejet while at the same time showing virtually no difference in stature for either males or females at this site. Consistent, perhaps, with the higher incidence of disease at Sejet, individuals there also seemed to have a lower mean age at death. The historical literature suggests that leprosy persists longer in rural areas since segregation was less well organised than in urban areas (resulting in broader population exposure in

these rural communities) (Boldsen, 2009). In the case of tuberculosis, in its bovine form at least (and also in the case of the possible differential diagnosis of brucellosis), a potential connection to cattle-based agriculture might be expressed. Close contact with cattle in a rural area could have influenced exposure to the disease (Boldsen and Mollerup, 2006).

Any interpretation of these results should be treated with caution on a number of levels, however. First, in line with the Osteological Paradox (Wood et al. 1992), the expression of pathological changes could actually mean that individuals were healthier and more likely to survive longer with the disease. Furthermore, the results for age at death are problematic for these subsamples since the consideration of overall cemetery patterns does not show the same patterns. In fact, the results from this investigation suggest no difference in age at death between the two populations. We are therefore left with what appear to be differences in disease expression, but without any further support for site differences. The results from disease could be interpreted as reflecting different exposure in the two communities, different levels of frailty, or indeed they could be confounded by differential preservation.

There were few statistically significant changes over time, with small sample sizes in the later period being particularly problematic in this respect. While there were changes in infectious disease rates, these results are plagued by the challenges faced in the osteological identification of specific diseases (Ortner, 1991; Wood et al., 1992). Only leprosy showed a statistically significant decrease over time, consistent with expected trends based on the literature review. The slight increases in both tuberculosis and

treponematosis is also consistent with expected trends, but as neither change is statistically significant these results cannot be heavily interpreted. In all cases, prevalence rates are almost certainly under-identified across both samples and in both time periods. While a decrease in stature was visible for females over time, the small sample sizes in the later period and the fact that this change is not visible in femur length, makes the interpretation of this finding problematic.

Perhaps the most interesting results from this research lie in pervasive differences between males and females. In relation to overall health parameters, males lived longer (a factor possibly connected to female mortality during childbirth), but showed a higher frequency of AS and generally higher rates of the only statistically different infectious disease (leprosy). DeWitte (2010) in exploring sex differentials in frailty in the East Smithfield Black Death cemetery in London also found lower rates of stress markers amongst females, suggesting that in this population either earlier stress incidence increased risk of mortality in males over females or women were less heavily impacted by the Black Death epidemic irrespective of previous health impacts.

The different patterns observed between the sexes in this thesis highlight the need for any health-related studies to consider males and females separately. While the possibility of culturally-induced health differences between males and females should be considered, there is currently no known evidence from the medieval Danish literature to suggest that there may have been differences in treatment or in exposure of males and females during this period. This is not to say that such cultural differences did not exist,

but there is no evidence for them at present. As discussed below, it is possible that further developments in osteology will enable further insight to be gained in this respect.

In the interests of parsimony, a biological explanation for these different patterns, which is supported by biological understandings of male and female differences, is favoured at the current time. The clinical and experimental research on humans and non-human animals suggests that there are very real differences in health patterns which exist between the sexes (La Salle et al., 2004; Bouman et al., 2005; Carey et al., 2007; Bale, 2009, 2011; Gabory et al., 2013). The general trends from any anthropological studies which have considered specific sex differences with respect to individual health parameters (i.e., the direct relationship between individual stress and health) show that when these patterns are considered, males are more negatively impacted than females by stress (Boldsen, 2007; DeWitte, 2010). The results from the current study were consistent with these patterns with regards to mortality. However, while one decile did show a negative relationship with males between AS frequency and mortality, overall AS counts showed a positive relationship for females and a neutral one for males. While the overall trend of females responding better to stress in relation to males is consistent, the positive trend for the overall pattern is interesting and requires further investigation on a larger scale.

These patterns show that there does seem to be a relationship between childhood stress as expressed by the frequency of dental enamel defects and adult mortality, but since this study only captured the survivors of childhood stress events it is missing part of the broader picture. The incorporation of subadults into such an investigation would

therefore be necessary to gain crucial insight into the differences between those who survived and those who did not. Such an investigation may also shed further light on any patterns in child rearing which may not be recorded in the historical record. In such an investigation, it would be imperative to consider the sample from a sex-specific standpoint. Such an approach might indeed be used to clarify from an osteological source whether there were any differences in stress experiences between male and female children during growth and development. However, the osteological data alone would not resolve the question of differential exposure versus differential response. All that is visible is the evidence of a non-specific stress event occurring which was sufficient to disrupt enamel development in that individual. For this marker to appear, the individual must have been exposed to something which stimulated a stress response. However, a person who does not show the stress marker could equally have been exposed to the same stimulant, but have responded differently to this stressor in such a way as to not have their enamel growth disrupted.

By studying subadults, insight might be gained on growth patterns, diet, and trauma for example. The identification of sex in subadults would allow us to consider sex differences in stress markers. The interpretation of these pattern, however, will need to be nuanced and to acknowledge the limitations of the evidence. Furthermore, determining sex is far more complicated for subadult remains since primary sex-specific characteristics do not begin to develop before puberty (Scheuer and Black, 2000). Despite this, there have been some advances in this area and there are future directions

which might prove useful in improving our ability to determine sex from juvenile remains.

Recent research by Osipov et al. (2013) on the bony labyrinth suggests that over 80% accuracy in sex estimation can be achieved from this element on subadult remains. The early completion in development of this element, combined with its likelihood to survive due to its location in the petrous portion of the temporal bone makes it a possible useful addition to the osteologists' repertoire (Osipov et al., 2013). Hassett (2012) has also found that the cervical diameter of the permanent canine can produce sex estimates on known-sex individuals with 93.8% accuracy. The compilation of such traits which complete development early and which express sexual dimorphism may help with future sex estimation for subadult remains.

A further important area to investigate is whether or not there is sexual-dimorphism in circaseptan rhythm as expressed through enamel cross-striations. The regular time periodicity of cross striations has been established and this has been observed to range from 7 to 12 days between long period markers (striae of Retzius) (FitzGerald, 1998; Reid and Ferrell, 2006). However, other than individual variability, we have no sense of what causes this range in humans, although we have some sense of the factors which lead to the development of cross-striations in the first place. FitzGerald (1998) provides a comprehensive review of research into these factors, which largely comes down to variation in carbon dioxide levels in the blood which is strongly associated with sleep patterns.

There has been little study on sexual dimorphism in circaseptan rhythms in the literature, and those studies that have looked at sex-specific patterns show mixed results. Smith et al. (2007) did find that females in known-sex human samples had a significantly higher periodicity than males in dental enamel. If sexual dimorphism can be securely established in the periodicity of dental enamel, its addition to any research involving internal analysis of dental enamel in juvenile remains (such as a study considering the nonsurvivor component) could be useful in sex estimation for both adults and subadults. Finally, ancient DNA could provide fundamental and secure sex determination for juvenile remains (Stone et al., 1996; Faerman et al., 1998; Mays and Faerman, 2001). Any study which involves destructive processing of juvenile material should therefore seek to integrate such a component into the protocol.

There is a great deal to be gained by continued investigation of the relationship between childhood stress and adult health from a sex-specific standpoint. By building a methodology which can adequately assess the relationship between childhood stress and adult health from skeletal remains, the doors open to potentially very large samples which consider health on a longitudinal level. However, there are many avenues which require development in order to satisfactorily pursue this line of investigation. These can be broken down into two main categories – we need to be able to be able to 'read' both adult health and childhood stress better from skeletal remains.

Future Directions in Adult Health

The consideration of adult health is in particular plagued by the difficulties in estimating age at death. Since adult age is defined not by the biological processes of growth and maturation but rather by factors of wear and tear which are variable depending on individual and cultural factors (activity, diet, etc.), age ranges are wide (particularly for the older age categories) (Bocquet-Appel and Masset, 1982; Buikstra and Konigsberg, 1985; Milner and Boldsen, 2012a). This pattern results in a challenging situation from a computational standpoint, since simply utilizing the midpoint for the age range does not integrate error ranges from the different age estimation parameters used and does not take account of differences in likelihood for an individual being any given age within that range (Boldsen, 1988).

These difficulties are unlikely to be easily resolved, but inroads are being made on a number of fronts. The development of statistical methods such as transition analysis (Boldsen, 1996; Boldsen et al., 2002; Milner and Boldsen, 2012b) to deal with issues such as age mimicry (Bocquet-Appel and Masset, 1982; Mensforth, 1990) and which incorporate information such as the likelihood of a particular feature representing a particular age (Milner and Boldsen, 2012a), have made tremendous advances in approaching the issues with adult age estimation. However, the strength of these methods is still limited by the accuracy of the techniques used for estimating age in the first place (Boldsen et al., 2002; Milner and Boldsen, 2012a) .

Advances in the direct interpretation of this material in terms of age parameters are being made with methodologies which employ a multifactorial approach (Boldsen et

al., 2002; Weise et al., 2009, 2012; Milner and Boldsen, 2012a). The approach being advocated by Milner and colleagues (Milner et al., 2008; Milner and Boldsen, 2012a) makes use of elements which have hitherto been largely ignored, but which can nevertheless provide insight by establishing particular cut-off points for age in specific elements. In this approach, rather than restricting the assessment of age to features which show a progression of changes, incorporating binary features which can contribute to information of specific transitions (from young to old) can help to narrow down age categories (Milner et al., 2008; Milner and Boldsen, 2012a). The continued development of more features which can be integrated into a multifactorial approach, can gradually contribute to the refinement of age estimation techniques (Milner and Boldsen, 2012a).

One technique which shows some potential in this area is the use of cementum annulations for age estimation. There is a great deal to be done yet to establish the accuracy of this methodology, and the results thus far have been variable likely in part due to unstandardized techniques and processing. Jankauskas et al. (2001) produced transverse sections and found a mean error of 6.46 years from known age samples (with the error in some cases exceeding 10 years) and Joshi et al. (2010) estimated errors of up to 11 years using longitudinal sections. Kasetty et al. (2010) also obtained low levels of accurate aging when considering longitudinal sections. Finally, Roksandic et al. (2009) obtained levels of accuracy that were roughly compatible with standard macroscopic osteological techniques and also emphasized issues with diagenesis for these techniques. On the other hand, some results suggest that this method can be used to age adults at least as accurately as other techniques, and perhaps with a higher degree of accuracy. Wittwer-

Backofen et al. (2004) found a standard error of 2.5 years using a large sample (N =363) of known age individuals. Kaur et al. (2011) also found high levels of correlation (with no more than 1.5 year error) between cementum annulation ages and real age in modern teeth. While these latter studies are tantalizing, it is clear from the former poorer results that extreme caution and extensive further research is required before these techniques are fully accepted. Indeed, more accurate results appear to currently be the exception rather than the rule. The destructive nature the process required to count cementum annulations should caution against any widescale use of these techniques until greater consistency in identification and more extensive knowledge of the formation processes involved is attained.

In general, transverse sections seem to perform better than longitudinal sections and the location of the section along the tooth root is of fundamental importance (Naji et al., 2013). A further source of error can result from the need to add the cementum annulation counts to tooth eruption time, which inherently integrates an error range from variable eruption schedules. If histological sectioning is already taking place, the incorporation of such techniques as cementum annulation counts into the broader age estimation protocols such as transition analysis (Boldsen et al., 2002; Milner and Boldsen, 2012b) and calibrated expert inference (Weise et al., 2009, 2012; Milner and Boldsen, 2012a) could be advantageous. In the case of the samples used for the present analysis, enough material is still intact for transverse sections to be produced from many of the teeth in order to consider cementum annulation counts for refinement of the age estimations. However, as Roksandic et al. (2009) caution, careful thought must be given

to destructive analysis and more development of these techniques is warranted prior to such destructive analysis for the sole purpose of cementum annulation work.

Gaining a more accurate and precise sense of age at death is essential to acquiring a better understanding of health on both the individual and population levels, and is a necessary next step to the current research. Another aspect of health that could benefit the current research but which requires further development in techniques and methodology is the recognition and registration of pathological conditions. Osteological remains are limited in their capacity to record diseases, having low sensitivity and low specificity (Eisenberg, 1992; Hutchinson, 1992; Wood et al., 1992; Wood and Milner, 1994; Milner et al., 2008). Bone responds slowly to disease stimulus and so only a limited number of chronic conditions will leave their mark on the bone (Ortner, 1991; Wood et al., 1992). Bone also responds in only a limited number of ways, and so many conditions lead to similar bony changes (Ortner, 1991; Stuart-Macadam, 1991; Wood et al., 1992). Through a better understanding of the nature of lesions and of their distribution, a great deal of progress has been made in disease diagnosis (Wright and Yoder, 2003). However, the need to integrate missing elements, to understand lesion specificity and sensitivity, and to incorporate all possible diseases into a differential diagnosis prevails. This amount of information is difficult to account for and traditionally osteologists have relied on individual assessment based on lesion characteristics and patterning. Without being able to incorporate all parameters of disease patterning into diagnostics, the consideration of disease prevalence in a population and across populations is highly problematic.

There are a few ways forward as we seek to develop parameters and diagnoses which can be accurately applied on a population and cross-population level. The development of the lambda program (Boldsen and Freund, 2006; Boldsen, 2008) for estimating the likelihood of an individual having suffered from leprosy and population prevalence of leprosy has built on decades of research into the characteristic bony changes associated with leprosy (Møller-Christensen and Faber, 1952; Møller-Christensen, 1965, 1978; Boldsen, 2001, 2005c, 2008; Boldsen and Freund, 2006; Boldsen and Mollerup, 2006). Through its integration of detailed background work into the specificity and sensitivity of core lesions and its incorporation of missing elements into its calculations, this program captures a level of detail which produces a far more objective standardised assessment of disease prevalence rates. The development of such programs for other diseases which might be found osteologically would be a significant advance in this area.

While developing a similar approach for other diseases would be useful, osteologists would still need to identify possible diseases to consider first and would be restricted to those in the osteological repertoire. Furthermore, until such programs are developed, we must still rely on collections which in most cases include individuals with varying levels of preservation. This is difficult to integrate into a broad scope skeletal analysis. One way around this is to focus on a specific lesion at one locus, such as tibial periostitis (DeWitte and Wood, 2008). By considering tibial periostitis (along the medial and anterior surfaces by the diaphysis), DeWitte and Wood (2008) were able to consider a single lesion which relates to non-specific infection or trauma. This lesion was used as a

proxy for individual frailty, and did seem to relate to mortality (DeWitte and Wood, 2008; DeWitte, 2010). Such an approach facilitates the ability to account for missing elements, and since no final diagnosis is sought the lesion only needs to be representative of general health. However, this of course does not answer questions about specific disease prevalence rates.

Increasingly, advances are being made in the identification of pathogens from ancient DNA analysis (Nuorala, 2004; Wiechmann and Grupe, 2005; Gardner et al., 2010; Bos et al., 2011; Schuenemann et al., 2011). Diseases such as leprosy (Nuorala, 2004; Donoghue et al., 2005; Taylor et al., 2006; Stone et al., 2009), tuberculosis (Nuorala, 2004; Donoghue et al., 2005; Donoghue, 2008; Stone et al., 2009), and *Yersinia pestis* (Raoult et al., 2000; Wiechmann and Grupe, 2005; Haensch et al., 2010; Bos et al., 2011, 2012; Schuenemann et al., 2011; Tran et al., 2011; Cui et al., 2012) (a candidate for the causative agent of Black Death) are amongst those which have been successfully identified from osteological remains.

The development of new techniques such as targeted enrichment strategies and high-throughput DNA sequencing make it possible to reconstruct longer lengths of aDNA while avoiding the amplification issues involved in older PCR protocols (Schuenemann et al., 2011). Libraries such as Illumina sequencing libraries can be generated to preserve the enriched extracts for future use (Bos et al., 2011; Schuenemann et al., 2011). From this, it is possible to conduct MDA (microbial detection array) (Gardner et al., 2010) to survey for a broad spectrum pathogens, as well as to search for specific pathogens. As such, it is possible to run unbiased investigations to detect any diseases which might be

present, regardless of whether or not these are expected. In this way, an unbiased approach to possible diseases in a sample may be conducted and a great deal can be learned about the disease load in a population as well as about specific diseases which might be of interest. The ability to build Illumina sequencing libraries (Gardner et al., 2010; Bos et al., 2011) also makes it possible to preserve all extracts for future studies.

Ancient pathogen DNA has been successfully isolated from dental pulp (La et al., 2008; Nguyen-Hieu et al., 2011) and from dental calculus (Preus et al., 2011), opening doors to a better understanding of any disease which might have been systemic (even those which had not yet or which do not cause osteological changes) and to microbes which might have existed in the oral cavity. While the identification of aDNA is restricted by conditions of preservation, the potential to gain insight into a broader scope of diseases and to gain specific diagnoses of diseases which are of particular interest is enormous.

Future Directions in Childhood Stress

The incremental nature of enamel structures and their association with non-specific stress places them in a position to provide insight into early life experiences which can be directly linked to time of occurrence. However, it is still clear that a great deal of work is necessary to learn more about how a stress experience translates into defect expression and therefore about how a defect should be interpreted (Guatelli-Steinberg and Lukacs, 1999). Unfortunately, the identification of specific stress events is far from straightforward (Goodman and Rose, 1990; Thomas, 2003; FitzGerald and Saunders, 2005). While there are enamel defects which are quite clear, their appearance

is highly variable and it is difficult in many cases to determine where to draw the line between normal and accentuated structures (FitzGerald and Saunders, 2005). Goodman and Rose (1990) maintained that there could be no differentiation in different grades of AS, but rather that something either was or was not a defect. They suggested that any variability would be due to differential susceptibility of particular ameloblasts but that a threshold existed based on whether an ameloblast was sufficiently stressed for activity to be disrupted. FitzGerald and Saunders (2005) on the other hand maintained that these structures exist along a continuum of expression from normal through to extreme pathological, with no set thresholds.

If we are going to use these structures in the future, parameters do need to be established which can be consistently followed by all researchers. It is necessary to build on work such as that conducted by Thomas (2003) who distinguished between 'weak' and 'strong' AS and Witzel (2008) who attempted to define expression and thresholds based on stage of ameloblast disruption (as earlier discussed by Goodman and Rose [1990]). The appearance of a differential relationship between dental enamel defects and mortality in both Thomas' (2003) research and in the current results emphasizes the importance of fully understanding the basis behind defect variability. If some defects reflect beneficial stress response, while others relate to stress responses having detrimental health impacts, combining the two could cloud the results. Care is therefore necessary so as not to assign random thresholds with no etiological foundation, and thus overlook key information.

Generally, we need to move away from attempts to link AS and dental enamel defects with specific causes of stress (Neiburger, 1990). While dental enamel defects have

been linked with particular stressors such as nutritional deficiency (Mellanby, 1929, 1930, 1934; Sweeney et al., 1969, 1971; Pindborg, 1982; Goodman and Rose, 1988; Goodman et al., 1991) and infection (Sarnat and Schour, 1941; Sweeney et al., 1969; Pindborg, 1982; Suckling et al., 1983, 1986; Suckling and Pearce, 1984; Goodman and Rose, 1990), there are many causative agents and the relationship between these causes and defect formation is variable and poorly understood (Neiburger, 1990). Attributing a dental enamel defect to a particular causative agent (typically nutritional deficiency or infection in anthropological studies) is overly simplistic (Neiburger, 1990) and limiting. All systemic enamel defects are formed in relation to physiological stress responses which can have an impact on other body systems in the individual. The specific cause of the stressor is likely to never be identified in archaeological samples, but it is important to be able to recognise that a stress event occurred and to be able to gain insight from this into overall patterns of stress and their broader impact on health. Thus, instead of seeking to associate specific stressors with defects on a population level, we should focus future research on three key areas. Essentially, we need to understand stress and stress response better on a biological level and as it relates to sexual-dimorphism and to individual patterns in frailty, we need to understand how dental enamel defects form in relation to this understanding of stress, and we need to build a protocol based on this integrated understanding of stress and defect development. Techniques such as laser ablation and potentially in the future synchrotron radiation present the potential for conducting trace element analysis in conjunction with microscopic imaging of dental enamel. Such

approaches have tremendous potential to further elucidate patterns in tooth formation and to identify fundamental differences between normally forming and pathological enamel.

The development of a better understanding in this area will take years of research and it will need to incorporate information from clinical and experimental findings. It will be essential to approach this from a sex-specific standpoint and to draw from clinical and experimental research which is considering the impact of developmental timing and critical windows of individual health (and ultimately frailty) (Dietert et al., 2000; Selevan et al., 2000; West, 2002; La Salle et al., 2004; Carey et al., 2007; Buss et al., 2009). Such a foundation is imperative if the field is to move forward in gaining a comprehensive perspective of the etiology behind variable defect formation. Only within this context will it be possible to build a protocol for classifying dental defects which is informed by the many factors influencing their formation. In this, it will be necessary to take the individual's sex and the timing of enamel development into consideration. Both aspects contribute to individual frailty and will impact the response seen in dental enamel development.

In order to develop such an understanding, it is necessary to consider stress response, how the severity of a stress response is determined, and what physiological changes might occur in relation to differing levels of stress response. Allostasis involves, effectively, the maintenance of homeostasis, which is the system of coordinated physiological processes responsible for keeping normally operating systems in a state of functional balance (McEwen, 2008). Building on previous models of allostasis (McEwen, 1998, 2002, 2004, 2008; Wingfield, 2003, 2005; Goymann and Wingfield,

2004; Korte et al., 2005), Romero et al. (2009) proposed a reactive scope model for understanding differing reactions to a stressor. Such models as this might be helpful in understanding enamel defects.

Within the context of this model, four levels of activity in relation to stress are outlined. The first two of these exist within the normal range, with the first (predictive homeostasis) having to do with normal functioning in relation to typical fluctuations in the body and surrounding environment (i.e., circadian rhythm), while the second (reactive homeostasis) relates to the necessary reaction to abnormal environmental stressors (Romero et al., 2009). The next two states are beyond the normal range and are classed as pathological (reactive homeostatic states). Homeostatic overload has been proposed as the range of allostatic response which is either inordinately high or of too long a duration in relation to that required to return to a state of homeostasis (Romero et al., 2009). Homeostatic failure occurs when a response is insufficient (Romero et al. 2009).

Romero et al.'s (2009) model is useful in its ability to reconcile concepts of stress, homeostasis, and the more recent move towards allostasis. It is also less restrictive than other models of allostasis which deal entirely with a concept of stress based around energy input and output (McEwen, 2002; McEwen and Wingfield, 2003, 2010). Another helpful point in this model is the integration of a concept of a sliding threshold, in which "the normal reactive scope, can differ between individuals and within a single individual in response to certain stimuli" (Romero et al., 2009, p 380). It is clear that all individuals will have different levels of frailty and that this level of frailty will not remain constant over the course of an individuals' life but will be impacted by a number of factors

(genetic, epigenetic, and environmental). For example, Goodman and Rose (1990) highlight such components in their consideration of enamel defect formation.

Through this model, it might be predicted that a mismatch between the requisite and ultimate stress response will result in more severe changes, such as hypoplastic defects and 'strong' AS. So long as an individuals' stress response is adequate, but not beyond that necessitated by the specific stressor, no defect should be formed. Thus, a pathological enamel defect is expected to appear when individuals enter Romero et al's (1990) reactive states (homeostatic overload or homeostatic failure). It will not, however, be possible to distinguish between the two responses in the dental enamel. The ultimate impact on the enamel and on other systems will furthermore be dependent on the developmental stage of the individual at that point in time.

Such a sliding threshold may also be relevant in considering the sex-based differences observed in this study. Differential developmental timings and biological differences which have been observed between males and females in modern populations would suggest that different thresholds may be relevant to each sex respectively. It is imperative that we understand the conditions under which enamel defects are formed and by integrating concepts of stress thresholds we may begin to move forward with this understanding. The consideration of AS in relation to later life health indicators brings an interesting dimension to the study of health in the past and can make useful contributions to our understanding of health in modern populations. The current results do suggest sex-related differences in responses to stress, with females appearing to manage childhood stress better than males. This is consistent with clinical and experimental data which

have identified sexual dimorphism in health outcomes and in responses to early life stress (Stinson, 1985; Roberts et al., 1996; Klein, 2000; McEwen, 2002; Uekert et al., 2006; Fairweather et al., 2008; Buss et al., 2009; Bale, 2011). Approaching health questions such as these with osteological remains takes anthropological analysis of dental defects beyond the difficulties entailed by the lack of etiological specificity which are characteristic of such structures (Neiburger, 1990), since regardless of this etiological background, the defect itself indicates that that particular individual sustained a level of systemic stress.

At the centre of this is being able to integrate an understanding of differential frailty (Wood et al., 1992; DeWitte, 2010). Beyond the clinical and experimental fields into individual stress response, there are other fields which we can make use of to contribute to this broader field using skeletal remains. Human dental material is a reservoir of information. Not only does it preserve a chronological record of development, but it can preserve genetic information on both humans (Green et al., 2008; Adler et al., 2011; Der Sarkissian et al., 2013) and pathogens (Papagrigorakis et al., 2006; La et al., 2008; Nguyen-Hieu et al., 2011; Preus et al., 2011). The study of pathogen aDNA has already been discussed, and the recovery of such information can give us a sense of the types of stressors which were in the environment and experienced by the individual.

There is, however, a vast field that is only beginning to be explored and which can hugely contribute to our understanding of direct impacts of environmental stressors on the people being studied. The field of epigenetics is growing and is increasingly identifying

specific genetic changes which can be linked with environmental stressors and health outcomes (Jaenisch and Bird, 2003; Devaskar and Raychaudhuri, 2007; Dolinoy et al., 2007; Jirtle and Skinner, 2007; Meaney, 2010). The human DNA extracted from dental (and skeletal) remains can similarly be used to gain insight into the specific changes which will have influenced an individuals' frailty. The human DNA itself can be used to tell us about the health of individuals. This can be considered on a general front, but also in relation to such events as epidemics like the Black Death to gain insight into co-evolutionary factors which shape human health in a broader sphere.

Conclusion

There is still a great deal to be discovered, and this field is still relatively new, but by applying the increasing scope of epigenetic knowledge and our growing understanding of the developmental impacts of stress to past populations, future research might be able to answer some of the questions about individual frailty far more directly. If considered within this broader context of increased understanding of individual frailty and stress response, it may be possible to take the study of dental enamel defects a step farther, to move beyond simply trying to associate a cause to a non-specific indicator, and to gain a far more detailed understanding of early life stress experiences and later life health. From this, we can integrate threshold models such as those which are being developed in relation to allostatic load (McEwen, 1998; McEwen and Wingfield, 2003; Romero et al., 2009) to gain a better understanding of the formation of dental microstructures and to build a protocol for their registration which is soundly based on their physiological basis.

The investigation of health in past populations is frequently challenged by technical and methodological issues. In order to learn more about patterns in medieval Denmark, continued research including larger sample sizes is critical. This is particularly the case for the later period for which there are fewer human remains. However, beyond this, continued methodological developments in age estimation and in the identification and recording of infectious disease patterns is critical. The extensive work which has taken place in Denmark thus far on leprosy diagnosis and prevalence calculation (Boldsen, 2001, 2005b, 2008, 2009; Boldsen and Freund, 2006; Boldsen and Mollerup, 2006; Boldsen et al., 2013) is a positive indication of what can be obtained through the careful examination of lesion specificity and sensitivity and through integration into a system which incorporates this information along with missing element information.

Chapter 8 References Cited

ADBOU. 2010. Human Osteological Methods: Chronic Diseases, Unpublished registration manual. Odense, Denmark: ADBOU, Syddansk Universitet. Available from: http://adbou.dk/fileadmin/manualer/kroniskesygdomme221111_vandmaerke.pdf

Addyman PV. 1989. The archaeology of public health at York, England. World Archaeology 21(2):244–264.

Adler CJ, Haak W, Donlon D, Cooper A. 2011. Survival and recovery of DNA from ancient teeth and bones. Journal of Archaeological Science 38(5):956–964.

Ahmed SA, Penhale WJ, Talal N. 1985. Sex hormones, immune responses, and autoimmune diseases. Mechanisms of sex hormone action. The American Journal of Pathology 121(3):531-551.

Albanese J, Eklics G, Tuck A. 2008. A Metric Method for Sex Determination using the Proximal Femur and Fragmentary Hipbone. Journal of Forensic Sciences 53(6):1283–1288.

Albanese J. 2003. A metric method for sex determination using the hipbone and the femur. Journal of Forensic Sciences 48(2):263-273.

Andrén A. 1985. Den urbana scenen: städer och samhälle i det medeltida Danmark. Acta Archaeologica Lundensia 8(13), CWK: Malmö.

Andrén A. 1989. State and towns in the Middle Ages: The Scandinavian experience. Theory and Society 18:585–609.

Antoine D. 2000. Evaluating the periodicity of incremental structures in dental enamel as a means of studying growth in children from past human populations. Unpublished PhD Thesis. London: Institute of Archaeology, University College London. Available from: http://ethos.bl.uk/OrderDetails.do?uin=uk.bl.ethos.394732

Antoine D, Hillson S, Dean MC. 2009. The developmental clock of dental enamel: a test for the periodicity of prism cross-striations in modern humans and an evaluation of the most likely sources of error in histological studies of this kind. Journal of Anatomy 214(1):45–55.

Antonson H. 2009. The extent of farm desertion in central Sweden during the late medieval agrarian crisis: landscape as a source. Journal of Historical Geography 35(4):619–641.

Arcini C. 1999. Health and Disease in Early Lund: Osteo-pathologic studies of 3,305 individuals buried in the cemetery area of Lund 990-1536. Unpublished PhD Thesis. Lund: Division of Social Medicine and Global Health, Kulturen.

Armelagos GJ, Goodman AH, Harper KN, Blakey ML. 2009. Enamel hypoplasia and early mortality: Bioarcheological support for the Barker hypothesis. Evolutionary Anthropology: Issues, News, and Reviews 18(6):261–271.

Armelagos GJ, Zuckerman MK, Harper KN. 2012. The Science Behind Pre-Columbian Evidence of Syphilis in Europe: Research by Documentary. Evolutionary Anthropology: Issues, News, and Reviews 21(2):50–57.

Auerbach BM, Ruff CB. 2010. Stature estimation formulae for indigenous North American populations. American Journal of Physical Anthropology 141(2):190–207.

Aufderheide AC, Rodriguez-Martin C. 1998. The Cambridge Encyclopedia of Human Paleopathology, 1st edition. Cambridge: Cambridge University Press.

Axford Y, Geirsdóttir Á, Miller GH, Langdon PG. 2009. Climate of the Little Ice Age and the past 2000 years in northeast Iceland inferred from chironomids and other lake sediment proxies. Journal of Paleolimnology 41(1):7–24.

Bach H. 1965. Zur Berechnung der Körperhöhe aus den langen Gliedmaßenknochen weiblicher Skelette. Anthropologischer Anzeiger 29:12–21.

Bale TL. 2009. Neuroendocrine and Immune Influences on the CNS: It's a Matter of Sex. Neuron 64(1):13–16.

Bale TL. 2011. Sex differences in prenatal epigenetic programing of stress pathways. Stress 14(4):348-356.

Barker DJ. 1995. Fetal origins of coronary heart disease. British Medical Journal 311:171–174.

Barker DJP. 2001. Fetal and infant origins of adult disease. Monatsschrift Kinderheilkunde 149(1):S2–S6.

Barker DJP. 2004a. The developmental origins of adult disease. Journal of the American College of Nutrition 23(suppl. 6):588S–595S.

Barker DJP. 2004b. The developmental origins of well-being. Philosophical Transactions of the Royal Society B: Biological Sciences 359(1449):1359–1366.

Barker DJP. 2007. The origins of the developmental origins theory. Journal of Internal Medicine 261(5):412–417.

Barker DJP, Osmond C. 1986. Infant mortality, childhood nutrition, and ischaemic heart disease in England and Wales. The Lancet 327(8489):1077–1081.

Barker DJP, Osmond C, Winter PD, Margetts B, Simmonds SJ. 1989. Weight in infancy and death from ischaemic heart disease. The Lancet 334(8663):577–580.

Barker DJ, Godfrey KM, Gluckman PD, Harding JE, Owens JA, Robinson JS. 1993. Fetal nutrition and cardiovascular disease in adult life. The Lancet 341(8850):938–941.

Barker DJP, Eriksson JG, Forsén T, Osmond C. 2002. Fetal origins of adult disease: strength of effects and biological basis. International Journal of Epidemiology 31(6):1235–1239.

Beagley KW, Gockel CM. 2003. Regulation of innate and adaptive immunity by the female sex hormones oestradiol and progesterone. FEMS Immunology & Medical Microbiology 38(1):13–22.

Beaton GH. 1989. Small but healthy? Are we asking the right question? Human Organization 48(1):30–39.

Bee H, Boyd D. 2010. The developing child. 12th ed.. Boston: Allyn & Bacon.

Béguelin M. 2011. Stature estimation in a central Patagonian prehispanic population: development of new models considering specific body proportions. International Journal of Osteoarchaeology 21(2):150–158.

Benedictow OJ. 2004. The Black Death, 1346-1353: The Complete History. Woodbridge: The Boydell Press.

Benedictow OJ. 2012. New Perspectives in Medieval Demography: The Medieval Demographic System. In: Town and Countryside in the Age of the Black Death: Essays in Honour of John Hatcher. The Medieval Countryside. Turnhout: Brepols Publishers. p 3–42.

Bennike P. 1985. Palaeopathology of Danish Skeletons: A Comparative Study of Demography, Disease and Injury. Copenhagen: Akademisk Forlag.

Bennike P, Lewis ME, Schutkowski H, Valentin F. 2005. Comparison of Child Morbidity in Two Contrasting Cemeteries from Denmark. American Journal of Physical Anthropology 128(4):734–746.

Berti PR, Mahaney MC. 1995. Conservative Scoring and Exclusion of the Phenomenon of Interest in Linear Enamel Hypoplasia Studies. American Journal of Human Biology 7(3):313–320.

Birgitte J, Johannsen H, Kristiansen I. 2004. Danmarks Kirker: Århus: Nationalmuseet.

Blackwell DL, Hayward MD, Crimmins EM. 2001. Does childhood health affect chronic morbidity in later life? Social Science & Medicine 52(8):1269–1284.

Bocquet-Appel J-P, Masset C. 1982. Farewell to paleodemography. Journal of Human Evolution 11(4):321–333.

Bocquet-Appel J-P, Masset C. 1985. Matters of moment. Journal of Human Evolution 14(2):107–111.

Bøgh A. 1999. Samfundet. In: Daglivliv i Danmarks middelalder – en arkæologisk kulturhistorie. 1st ed. Copenhagen: Nordisk Forlag A/S. p 24–53.

Bogin B. 1995. Plasticity in the growth of Mayan refugee children living in the United States. In: Human variability and plasticity. Cambridge: Cambridge University Press. p 46–74.

Bogin B. 1999. Patterns of Human Growth. Cambridge: Cambridge University Press.

Bogin B, Silva MIV, Rios L. 2007. Life history trade-offs in human growth: Adaptation or pathology? American Journal of Human Biology 19(5):631–642.

Bogin B, Smith P, Orden AB, Varela Silva MI, Loucky J. 2002. Rapid change in height and body proportions of Maya American children. American Journal of Human Biology 14(6):753–761.

Bogin B, Varela-Silva MI. 2010. Leg length, body proportion, and health: a review with a note on beauty. International Journal of Environmental Research and Public Health 7(3):1047–1075.

Boldsen JL. 1984a. A Statistical Evaluation of the Basis for Predicting Stature from Lengths of Long Bones in European Populations. American Journal of Physical Anthropology 65(3):305–311.

Boldsen JL. 1984b. Palaeodemography of two southern Scandinavian Medieval Communities. Meddl. Lunds universitets historiska museum 5:107–115.

Boldsen JL. 1988. Two methods for reconstructing the empirical mortality profiles. Human evolution 3(5):335–342.

Boldsen JL. 1990a. Body Proportions, Population Structure, and Height Prediction. Journal of Forensic Medicine Instanbul 6:157–165.

Boldsen JL. 1990b. Height variation in the light of social and regional differences in medieval Denmark. In: From the Baltic to the Black Sea: Studies in Medieval Archaeology. Boston: Unwin Hyman. p 181–187.

Boldsen JL. 1991. Ageing and Dental Attrition in a Medieval Rural Danish Population. International Journal of Anthropology 6(4):217–224.

Boldsen JL. 1993. Height variation in Denmark A.D. 1100-1988. In: Populations of the Nordic countries: Human population biology from the rpesent to the Mesolithic. Proceedings of the Second Seminar of Nordic Physical Anthropology. Lund: Institute of Archaeology. p 52–60.

Boldsen JL. 1995. The place of plasticity in the study of the secular trend for male stature: an analysis of Danish biological population history. Human variability and plasticity Cambridge: Cambridge University Press p:75–90.

Boldsen JL. 1996. Patterns of Childhood Mortality in Medieval Denmark. Rivista di Antropologia (Roma) 74:147–159.

Boldsen JL. 1997a. Estimating Patterns of Disease and Mortality in a Medieval Danish Village. In: Integrating Archaeological Demography: Multidisciplinary Approaches to Prehistoric Population. Vol. 24. Occasional Paper. Southern Illinois University at Carbondale: Center for Archaeological Investigations. p 229–241.

Boldsen JL. 1997b. Lasting consequences of episodes of ill health in early childhood- a study of a medieval village community. In DF Roberts, P Rudan, T Skarić-Hurić (eds.) Growth and Development in the changing world. Zagreb: Croatian Anthropological Society. p 167–193.

Boldsen JL. 1998. Body proportions in a medieval village population: effects of early childhood episodes of ill health. Annals of Human Biology 25(4):309–317.

Boldsen JL. 2001. Epidemiological Approach to the Paleopathological Diagnosis of Leprosy. American Journal of Physical Anthropology 115(4):380–387.

Boldsen JL. 2005a. Analysis of Dental Attrition and Mortality in the Medieval Village of Tirup, Denmark. American Journal of Physical Anthropology 126(2):169–176.

Boldsen JL. 2005b. Leprosy and mortality in the Medieval Danish village of Tirup. American Journal of Physical Anthropology 126(2):159–168.

Boldsen JL. 2005c. Testing Conditional Independence in Diagnostic Palaeoepidemiology. American Journal of Physical Anthropology 128(3):586–592.

Boldsen JL. 2007. Early Childhood Stress and Adult Age Mortality - A Study of Dental Enamel Hypoplasia in the Medieval Danish Village of Tirup. American Journal of Physical Anthropology 132(1):59–66.

Boldsen JL. 2008. Leprosy in the early medieval Lauchheim community. American Journal of Physical Anthropology 135(3):301–310.

Boldsen JL. 2009. Leprosy in Medieval Denmark Osteological and epidemiological analyses. Anthropologischer Anzeiger 67(4):407–425.

Boldsen JL, Freund UH. 2006. Osteological leprosy: Epidemiology and diagnosis. Scandinavian Journal of Forensic Science 2:67–72.

Boldsen J, Iregren E. 1990. The reflection of childhood growth episodes in adult mortality. In: Populations of the Nordic countries: Human population biology from the present to the Mesolithic. Proceedings of the Second Seminar of Nordic Physical Anthropology. Lund: Institute of Archaeology. p 105–112.

Boldsen JL, Milner GR, Konigsberg LW, Wood JW. 2002. Transition analysis: a new method for estimating age from skeletons. In Hoppa RD and Vaupel JW (eds.) Paleodemography: Age Distributions from Skeletal Samples. Cambridge: Cambridge University Press, p. 73–106.

Boldsen JL, Mollerup L. 2006. Outside St. Jorgen: Leprosy in the Medieval Danish City of Odense. American Journal of Physical Anthropology 130(3):344–351.

Boldsen JL, Rasmussen KL, Riis T, Dittmar M, Weise S. 2013. Schleswig: Medieval leprosy on the boundary between Germany and Denmark. Anthropologischer Anzeiger 70(3):273–287.

Boldsen JL, Søgaard J. 1998. A History of Height in Denmark. In J Komlos and J Baten (eds.) The Biological Standard of Living in Comparative Perspective: Contributions to the Conference Held in Munich, January 18-22, 1997, for the XIIth Congress of the International Economic History Association. Franz Steiner Verlag: Stuttgart.

Bo S, Cavallo-Perin P, Scaglione L, Ciccone G, Pagano G. 2000. Low birthweight and metabolic abnormalities in twins with increased susceptibility to Type 2 diabetes mellitus. Diabetic Medicine 17(5):365–370.

Bos KI, Schuenemann VJ, Golding GB, Burbano HA, Waglechner N, Coombes BK, McPhee JB, DeWitte SN, Meyer M, Schmedes S, Wood J, Earn DJD, Herring DA, Bauer

P, Poinar HN, Krause J. 2011. A draft genome of Yersinia pestis from victims of the Black Death. Nature 478(7370):506–510.

Bos KI, Stevens P, Nieselt K, Poinar HN, DeWitte SN, Krause J. 2012. Yersinia pestis: New Evidence for an Old Infection. PLoS ONE 7(11):e49803.

Bouman A, Heineman MJ, Faas MM. 2005. Sex hormones and the immune response in humans. Human Reproduction Update 11(4):411–423.

Bouwman AS, Brown TA. 2005. The limits of biomolecular palaeopathology: ancient DNA cannot be used to study venereal syphilis. Journal of Archaeological Science 32(5):703–713.

Bradley RS, Hughes MK, Diaz HF. 2003. Climate in Medieval Time. Science 302(5644):404–405.

Breitinger E. 1937. Zur Berechnung der Körperhöhe aus den langen Gliedmaßenknochen. Anthropologischer Anzeiger 14:249–274.

Briffa KR. 2000. Annual climate variability in the Holocene: interpreting the message of ancient trees. Quaternary Science Reviews 19(1):87–105.

Briffa KR, Osborn TJ, Schweingruber FH. 2004. Large-scale temperature inferences from tree rings: a review. Global and planetary change 40(1):11–26.

Broecker WS. 2001. Paleoclimate: Was the Medieval Warm Period Global? Science 291(5508):1497 –1499.

Bromage TG, Dean MC. 1985. Re-evaluation of the age at death of immature fossil hominoids. Nature 317(6037):525–527.

Brook AH, Fearne JM, Smith JM. 1997. Environmental causes of enamel defects. In DJ Chadwick and G Cardew (eds) Dental Enamel, CIBA Foundation Symposium. Chichester: John Wiley & Sons, Ltd. p 212–225.

Brudevold F, Söremark R. 1967. Chemistry of the mineral phase of enamel. In A Mills (ed) Structural and Chemical Organization of Teeth. Vol. 2. New York: Academic Press. p 247–277.

Budner D, Cole-Dai J. 2003. The number and magnitude of large explosive volcanic eruptions between 904 and 1865 AD: Quantitative evidence from a new South Pole ice core. Geophysical Monograph Series 139:165–176.

Buikstra JE, Konigsberg LW. 1985. Paleodemography: Critiques and Controversies. American Anthropologist 87(2):316–333.

Büntgen U. 2009. Environmental drivers of Yersinia pestis-a holistic perspective on Medieval Europe. In 9th EMS Annual Meeting, 9th European Conference on Applications of Meteorology (ECAM) Abstracts, held Sept. 28-Oct. 2, 2009 in Toulouse, France. http://meetings. copernicus. org/ems2009/, id. EMS2009-307. Vol. 1, p. 307.

Büntgen U, Esper J, Frank DC, Nicolussi K, Schmidhalter M. 2005. A 1052-year tree-ring proxy for Alpine summer temperatures. Climate Dynamics 25(2-3):141–153.

Büntgen U, Tegel W, Nicolussi K, McCormick M, Frank D, Trouet V, Kaplan JO, Herzig F, Heussner K-U, Wanner H, Luterbacher J, Esper J. 2011. 2500 Years of European Climate Variability and Human Susceptibility. Science 331(6017):578–582.

Burdge GC, Lillycrop KA. 2010. Nutrition, epigenetics, and developmental plasticity: implications for understanding human disease. Annual Review of Nutrition 30:315–339.

Burns-Naas LA, Hastings KL, Ladics GS, Makris SL, Parker GA, Holsapple MP. 2008. What's So Special about the Developing Immune System? International Journal of Toxicology 27(2):223–254.

Buss C, Davis EP, Class QA, Gierczak M, Pattillo C, Glynn LM, Sandman CA. 2009. Maturation of the human fetal startle response: Evidence for sex-specific maturation of the human fetus. Early Human Development 85(10):633–638.

Campbell B. 2009. Four famines and a pestilence: harvest, price, and wage variations in England, 13th to 19th centuries. Stockholm: KSLAB.

Campbell ML. 2007. Biomechanical Modifications of a Danish Medieval Population. Unpublished Masters Thesis, University of Manitoba (Canada).

Cardoso HFV, Gomes JEA. 2009. Trends in adult stature of peoples who inhabited the modern Portuguese territory from the Mesolithic to the late 20th century. International Journal of Osteoarchaeology 19(6):711–725.

Carey MA, Card JW, Voltz JW, Germolec DR, Korach KS, Zeldin DC. 2007. The impact of sex and sex hormones on lung physiology and disease: lessons from animal studies. American Journal of Physiology - Lung Cellular and Molecular Physiology 293(2):L272–L278.

Chamley CA, Carson P, Randall D, Sandwell M, and Randall D. 2005. Developmental Anatomy and Physiology of Children: A Practical Approach, 1st edition. London: Churchill Livingstone, Elsevier Ltd.

Christensen SB, Mikkelsen J. 2006. The Danish urban system pre-1800: a survey of recent research results. Urban History 33(3):484.

Christiansen E. 1998. Dudo of St Quentin: History of the Normans. Woodbridge: Boydell Press.

Cinthio M. 1999. The archaeological context. In: Health and Disease in Early Lund: Osteo-Pathological Studies of 3,305 Individuals Buried in the First Cemetery Area of Lund 900-1536. Sweden: Department of Community Health Sciences, Medical Faculty: Lund University. p 18–46.

Cohen Hubal EA, Moya J, Selevan SG. 2008. A lifestage approach to assessing children's exposure. Birth Defects Research Part B: Developmental and Reproductive Toxicology 83(6):522–529.

Cole-Dai J, Mosley-Thompson E, Wight SP, Thompson LG. 2000. A 4100-year record of explosive volcanism from an East Antarctica ice core. Journal of Geophysical Research: Atmospheres 105(D19):24431–24441.

Condon K, Rose JR. 1992. Intertooth and Intratooth variability in the occurrence of developmental enamel defects. Journal of Paleopathology, Monographic Publications 2:61–77.

Cook DC, Buikstra JE. 1979. Health and differential survival in prehistoric populations: prenatal dental defects. American Journal of Physical Anthropology 51(4):649–664.

Cucina A, Işcan MY. 1997. Assessment of Enamel Hypoplasia in a High Status Burial Site. American Journal of Physical Anthropology 9:213–222.

Cui Y, Yu C, Yan Y, Li D, Li Y, Jombart T, Weinert LA, Wang Z, Guo Z, Xu L, Zhang Y, Zheng H, Qin N, Xiao X, Wu M, Wang X, Zhou D, Qi Z, Du Z, Wu H, Yang X, Cao H, Wang H, Wang J, Yao S, Rakin A, Li Y, Falush D, Balloux F, Achtman M, Song Y, Wang J, Yang R. 2012. Historical variations in mutation rate in an epidemic pathogen, Yersinia pestis. Proceedings of the National Academy of Sciences 110(2):577–582.

Dallman MF. 2003. Stress by any other name.....? Hormones and Behavior 43(1):18.

De Boo HA and Harding, JE. 2006. The developmental origins of adult disease (Barker) hypothesis. Australian and New Zealand Journal of Obstetrics and Gynaecology, 46(1), 4–14.

Desai M, Hales CN. 1997. Role of fetal and infant growth in programming metabolism in later life. Biological Reviews 72(2):329–348.

Devaskar SU, Raychaudhuri S. 2007. Epigenetics-A science of heritable biological adaptation. Pediatric Research 61(5, part 2):1R–4R.

DeWitte SN. 2010. Sex differentials in frailty in medieval England. American Journal of Physical Anthropology 143(2):285–297.

DeWitte SN, Hughes-Morey G. 2012. Stature and frailty during the Black Death: the effect of stature on risks of epidemic mortality in London, A.D. 1348–1350. Journal of Archaeological Science 39(5):1412–1419.

DeWitte SN, Wood JW. 2008. Selectivity of Black Death mortality with respect to preexisting health. PNAS 105(5):1436–1441.

DeWitte S, Slavin P. 2013. Between Famine and Death: England on the Eve of the Black Death—Evidence from Paleoepidemiology and Manorial Accounts. Journal of Interdisciplinary History 44(1):37–60.

Dietert RR, Etzel RA, Chen D, Halonen M, Holladay SD, Jarabek AM, Landreth K, Peden DB, Pinkerton K, Smialowicz RJ, Zoetis T. 2000. Workshop to identify critical windows of exposure for children's health: immune and respiratory systems work group summary. Environmental Health Perspectives 108(Suppl 3):483.

Dolinoy DC, Weidman JR, Jirtle RL. 2007. Epigenetic gene regulation: linking early developmental environment to adult disease. Reproductive Toxicology 23(3):297–307.

Dominguez R, Cruz-Morales SE, Carvalho MC, Xavier M, Brandao ML. 2003. Effect of steroid injection to newborn rats on serotonin activity in frontal cortex and raphe. Neuroreport 14(4):597–599.

Domínguez R, Cruz-Morales SE, Carvalho MC, Xavier M, Brandao ML. 2003. Sex differences in serotonergic activity in dorsal and median raphe nucleus. Physiology & Behavior 80(2):203–210.

Donoghue HD. 2008. Palaeomicrobiology of tuberculosis. Paleomicrobiology: Past Human Infections Springer-Verlag, Heidelberg:75–97.

Donoghue HD, Marcsik A, Matheson C, Vernon K, Nuorala E, Molto JE, Greenblatt CL, Spigelman M. 2005. Co-infection of Mycobacterium tuberculosis and Mycobacterium leprae in human archaeological samples: a possible explanation for the historical decline of leprosy. ProcBiolSci 272(1561):389–394.

Dupertuis CW, Hadden JA. 1951. On the reconstruction of stature from long bones. American Journal of Physical Anthropology 9(1):15–54.

Dybdahl A. 2012. Climate and demographic crises in Norway in medieval and early modern times. The Holocene 22(10):1159–1167.

Dyer AR. 1974. Comparisons of Tests for Normality with a Cautionary Note. Biometrika 61(1):185–189.

Dyer C, Styles P, Minchinton WE, Chaloner WH, Sutherland G, Church R. 1972. Deserted Medieval Villages. Midland History 1(4):55–62.

Eisenberg LE. 1992. Comment to the Osteological Paradox. Current Anthropology 33(4):359–360.

Elo IT, Preston SH. 1992. Effects of Early-Life Conditions on Adult Mortality: A Review. Population Index 58(2):186–212.

Eriksson J, Forsén T, Tuomilehto J, Osmond C, Barker D. 2000. Fetal and childhood growth and hypertension in adult life. Hypertension 36(5):790–794.

Eriksson JG, Forsén T, Tuomilehto J, Winter PD, Osmond C, Barker DJP. 1999. Catch-up growth in childhood and death from coronary heart disease: longitudinal study. Bmj 318:427–431.

Esper J, Cook ER, Schweingruber FH. 2002. Low-frequency signals in long tree-ring chronologies for reconstructing past temperature variability. Science 295(5563):2250–2253.

Eveleth PB, Tanner JM. 1976. Worldwide variation in human growth. Cambridge: Cambridge University Press.

Faerman M, Bar-Gal GK, Filon D, Greenblatt CL, Stager L, Oppenheim A, Smith P. 1998. Determining the sex of infanticide victims from the late Roman era through ancient DNA analysis. Journal of Archaeological Science 25(9):861–865.

Fagan BM. 2000. The Little Ice Age: how climate made history, 1300-1850. New York: Basic Books.

Fairweather D, Frisancho-Kiss S, Rose NR. 2008. Sex Differences in Autoimmune Disease from a Pathological Perspective. The American Journal of Pathology 173(3):600–609.

Field A. 2009. Discovering Statistics Using SPSS. 3rd ed. London: Sage Publications Limited.

Field CJ. 2005. The Immunological Components of Human Milk and Their Effect on Immune Development in Infants. J Nutr 135(1):1–4.

FitzGerald CM. 1998. Do enamel microstructures have regular time dependency? Conclusions from the literature and a large-scale study. Journal of Human Evolution 35(4-5):371–386.

FitzGerald CM, Rose JC. 2008. Reading Between the Lines: Dental Development and Subadult Age Assessment Using the Microstructural Growth Markers of Teeth. In MA Katzenberg and SR Saunders (eds.) Biological Anthropology of the Human Skeleton. Vol. 2nd. Hoboken: John Wiley & Sons, Inc. p 237–263.

FitzGerald CM, Saunders SR. 2005. Test of histological methods of determining chronology of accentuated striae in deciduous teeth. American Journal of Physical Anthropology 127(3):277–290.

FitzGerald C, Saunders S. 2006. Preparing Undecalcified Ground Tooth Sections. Lab Manual, Anthropology Hard Tissue and Light Microscopy Laboratory, McMaster University

FitzGerald C, Saunders S, Bondioli L, Macchiarelli R. 2006. Health of infants in an Imperial Roman skeletal sample: perspective from dental microstructure. American Journal of Physical Anthropology 130(2):179–189.

Floud R, Gregory A, Wachter K. 1990. Height, Health and History: Nutritional Status in the United Kingdom, 1750-1980. Cambridge: Cambridge University Press.

Floyd B, Littleton J. 2006. Linear enamel hypoplasia and growth in an Australian Aboriginal community: not so small, but not so healthy either. Annals of human biology 33(4):424–443.

Fogel RW, Engerman SL, Floud R, Friedman G, Margo RA, Sokoloff K, Steckel RH, Trussell TJ, Villaflor G, Wachter KW. 1983. Secular Changes in American and British Stature and Nutrition. The Journal of Interdisciplinary History 14(2):445–481.

Formicola V. 1993. Stature reconstruction from long bones in ancient population samples: An approach to the problem of its reliability. American Journal of Physical Anthropology 90(3):351–358.

Formicola V, Franceschi M. 1996. Regression equations for estimating stature from long bones of early Holocene European samples. American Journal of Physical Anthropology 100(1):83–88.

Fully G. 1956. Une nouvelle méthode de détermination de la taille. Annales de Médicine Légale 35:266–273.

Fully G, Pineau H. 1960. Détermination de la stature au moyen du squelette. Annales de Médicine Légale 40:145–154.

Gabory A, Roseboom TJ, Moore T, Moore LG, Junien C. 2013. Placental contribution to the origins of sexual dimorphism in health and diseases: sex chromosomes and epigenetics. Biology of Sex Differences 4(1):1–14.

Galbany J, Estebaranz F, Martínez LM, Romero A, De Juan J, Turbón D, Pérez-Pérez A. 2006. Comparative analysis of dental enamel polyvinylsiloxane impression and polyurethane casting methods for SEM research. Microscopy Research and Technique 69(4):246–252.

Galbany J, Martinez LM, Perez-Perez A. 2004. Tooth replication techniques, SEM imaging and microwear analysis in primates: methodological obstacles. Anthropologie 42(1):5–12.

Gardner S, Jaing C, McLoughlin K, Slezak T. 2010. A microbial detection array (MDA) for viral and bacterial detection. BMC Genomics 11(1):668.

Genovés S. 1967. Proportionality of the long bones and their relation to stature among Mesoamericans. American Journal of Physical Anthropology 26(1):67–77.

Gern JE, Lemanske RF, Busse WW. 1999. Early life origins of asthma. Journal of Clinical Investigation 104:837–843.

Giannecchini M, Moggi-Cecchi J. 2008. Stature in archeological samples from central Italy: Methodological issues and diachronic changes. American Journal of Physical Anthropology 135(3):284–292.

Gillman MW. 2005. Developmental origins of health and disease. New England Journal of Medicine 353(17):1848–1850.

Gleicher N, Barad DH. 2007. Gender as risk factor for autoimmune diseases. Journal of Autoimmunity 28(1):1–6.

Gluckman PD, Hanson MA. 2004a. Living with the past: evolution, development, and patterns of disease. Science 305(5691):1733–1736.

Gluckman PD, Hanson MA. 2004b. Developmental origins of disease paradigm: a mechanistic and evolutionary perspective. Pediatric Research 56(1):311–317.

Gluckman PD, Hanson MA, Pinal C. 2005. The developmental origins of adult disease. Maternal & Child Nutrition 1(3):130–141.

Gluckman P, Pinal C. 2001. Glucose tolerance in adults after prenatal exposure to famine. The Lancet 357(9270):1798.

Goodman AH. 1996. Early life stresses and adult health: insights from dental enamel development. In CJK Henry and SJ Ulijaszek (eds.) Long-term Consequences of Early Environment: Growth, development and the lifespan developmental perspective. Vol. 37. Society for the Study of Human Biology. Cambridge: Cambridge University Press. p 163–182.

Goodman AH, Armelagos GJ. 1988. Childhood stress and decreased longevity in a prehistoric population. American Anthropologist 90(4):936–944.

Goodman AH, Armelagos GJ, Rose JC. 1980. Enamel Hypoplasias as Indicators of Stress in Three Prehistoric Populations from Illinois. Human Biology 52(3):515–528.

Goodman AH, Martin DL, Klein CP, Peele MS, Cruse NA, McEwen LR, Saeed A, Robinson BM. 1992. Cluster Bands, Wilson Bands and Pit Patches: Histological and Enamel Surface Indicators of Stress in the Black Mesa Anasazi Population. Journal of Paleopathology, Monographic Publications 2:115–127.

Goodman AH, Martinez C, Chavez A. 1991. Nutritional supplementation and the development of linear enamel hypoplasias in children from Tezonteopan, Mexico. American Journal of Clinical Nutrition 53(3):773–781.

Goodman AH, Rose JC. 1988. Dental Enamel Hypoplasias as Indicators of Nutritional Status. In MA Kelley and CS Larsen (eds.) Advances in Dental Anthropology. New York: John Wiley & Sons, Inc. p 279–293.

Goodman AH, Rose JC. 1990. Assessment of Systemic Physiological Perturbations from Dental Enamel Hypoplasias and Associated Histological Structures. Yearbook of Physical Anthropology 33:59–110.

Goymann W, Wingfield JC. 2004. Allostatic load, social status and stress hormones: the costs of social status matter. Animal Behaviour 67(3):591–602.

Green RE, Malaspinas AS, Krause J, Briggs AW, Johnson PLF, Uhler C, Meyer M, Good JM, Maricic T, Stenzel U. 2008. A complete Neandertal mitochondrial genome sequence determined by high-throughput sequencing. Cell 134(3):416–426.

Grove JM. 2001. The Initiation of the "Little Ice Age" in Regions Round the North Atlantic. Climatic Change 48(1):53–82.

Grove JM. 2003. The Little Ice Age. New York: Routledge.

Grudd H, Briffa KR, Karlén W, Bartholin TS, Jones PD, Kromer B. 2002. A 7400-year tree-ring chronology in northern Swedish Lapland: natural climatic variability expressed on annual to millennial timescales. The Holocene 12(6):657–665.

Guatelli-Steinberg D, Larsen CS, Hutchinson DL. 2004. Prevalence and the duration of linear enamel hypoplasia - a comparative study of Neandertals and Inuit Foragers. Journal of Human Evolution 47:65–84.

Guatelli-Steinberg D, Lukacs JR. 1999. Interpreting sex differences in enamel hypoplasia in human and non-human primates: Developmental, environmental, and cultural considerations. American Journal of Physical Anthropology 110(S29):73–126.

Gunnell D, Rogers J, Dieppe P. 2001. Height and health: predicting longevity from bone length in archaeological remains. Journal of Epidemiology and Community Health 55(7):505–507.

Gustafsson A, Werdelin L, Tullberg BS, Lindenfors P. 2007. Stature and sexual stature dimorphism in Sweden, from the 10th to the end of the 20th century. American Journal of Human Biology 19(6):861–870.

Haensch S, Bianucci R, Signoli M, Rajerison M, Schultz M, Kacki S, Vermunt M, Weston DA, Hurst D. 2010. Distinct Clones of Yersinia pestis Caused the Black Death. PLoS Pathogens 6(10):e1001134.

Hales CN, Ozanne SE. 2003. Fetal and early postnatal growth restriction lead to diabetes, the metabolic syndrome and renal failure. Diabetologia 46(7):1013–1019.

Harper KN, Knauf S. 2013. Treponema pallidum infection in primates: clinical manifestations, epidemiology, and evolution of a stealthy pathogen. In JF Brinkworth and K Pechenkina (eds.) Primates, Pathogens, and Evolution. New York: Springer. p 189–219.

Harper KN, Zuckerman MK, Harper ML, Kingston JD, Armelagos GJ. 2011. The origin and antiquity of syphilis revisited: An Appraisal of Old World pre-Columbian evidence for treponemal infection. American Journal of Physical Anthropology 146(S53):99–133.

Hassett BR. 2012. Evaluating sources of variation in the identification of linear hypoplastic defects of enamel: a new quantified method. Journal of Archaeological Science 39:560–565.

Helama S, Lindholm M, Timonen M, Meriläinen J, Eronen M. 2002. The supra-long Scots pine tree-ring record for Finnish Lapland: Part 2, interannual to centennial variability in summer temperatures for 7500 years. The Holocene 12(6):681–687.

Hillson S. 1992a. Dental enamel growth, perikymata and hypoplasia in ancient tooth crowns. Journal of the Royal Society of Medicine 85:460–466.

Hillson S. 1992b. Impression and replica methods for studying hypoplasia and perikymata on human tooth crown surfaces from archaeological sites. International Journal of Osteoarchaeology 2(1):65–78.

Hillson S. 1996. Dental Anthropology. Cambridge: Cambridge University Press.

Hillson S. 2005. Teeth. Cambridge: Cambridge University Press.

Hillson S. 2008. Dental Pathology. In MA Katzenberg and SR Saunders (eds.) Biological Anthropology of the Human Skeleton. Vol. 2nd. Hoboken: John Wiley & Sons, Inc. p 301–340.

Hillson S, Antoine D, Dean C. 1999. A detailed developmental study of the defects of dental enamel in a group of post-medieval children from London. In: Dental Morphology'98: Proceedings of the 11th International Symposium on Dental Morphology. Oulu, Finland. p 102.

Hillson S, Bond S. 1997. Relationship of Enamel Hypoplasia to the Pattern of Tooth Crown Growth: A Discussion. American Journal of Physical Anthropology 104(1):89–103.

Hoppa RD, Fitzgerald CM. 1999. Human growth in the past: studies from bones and teeth. Cambridge: Cambridge University Press.

Hoppa RD, Vaupel JW. 2002. Paleodemography: Age distributions from skeletal samples. Cambridge: Cambridge University Press.

Von Hunnius TE, Yang D, Eng B, Waye JS, Saunders SR. 2007. Digging deeper into the limits of ancient DNA research on syphilis. Journal of Archaeological Science 34(12):2091–2100.

Hutchinson DL. 1992. Comment to the Osteological Paradox. Current Anthropology 33(4):360.

Hybel N, Poulsen B. 2013. The Danish Resources C. 1000 - 1550: Growth and Recession. Leiden: Brill.

Infante PF, Gillespie GM. 1974. An epidemiologic study of linear enamel hypoplasia of deciduous anterior teeth in Guatemalan children. Archives of Oral Biology 19(11):1055–1061.

Jackson KM, Nazar AM. 2006. Breastfeeding, the Immune Response, and Long-term Health. JAOA: Journal of the American Osteopathic Association 106(4):203–207.

Jaenisch R, Bird A. 2003. Epigenetic regulation of gene expression: how the genome integrates intrinsic and environmental signals. Nature Genetics 33:245–254.

Jankauskas R, Barakauskas S, Bojarun R. 2001. Incremental lines of dental cementum in biological age estimation. HOMO - Journal of Comparative Human Biology 52(1):59–71.

Jantzen C, Kieffer-Olsen J, Madsen PK. 1994. De små brødres hus i Ribe. Mark og Montre Aarborg for Kunst og Kulturhistoriske Museer 30:26–36.

Jantz LM, Jantz R l. 1999. Secular change in long bone length and proportion in the United States, 1800–1970. American Journal of Physical Anthropology 110(1):57–67.

Jatautis Š, Mitokaitė I, Jankauskas R. 2011. Analysis of cribra orbitalia in the earliest inhabitants of medieval Vilnius. Anthropological Review 74(1):57–68.

Jirtle RL, Skinner MK. 2007. Environmental epigenomics and disease susceptibility. Nature Reviews Genetics 8(4):253–262.

Jordan WC. 1997. The Great Famine: Northern Europe in the Early Fourteenth Century. Princeton: Princeton University Press.

Jørgensen L. 2009. *Tuberkulose i Middelalderen: Tublerculosis in medieval Denmark* (Masters). Syddansk Universitet (University of Southern Denmark), Odense, Denmark. Retrieved from http://adbou.dk/fileadmin/specialer/Tuberkulose_i_middelalderen.pdf

Joshi PS, Chougule MS, Agrawal GP. 2010. Comparison of polarizing & phase contrast microscopy for estimation of age based on cemental annulations. Indian Journal of

Forensic Odontology 3(3). Available from: http://rfppl.com/subscription/upload_pdf/Art%203_a63.pdf

Judes H, Eli I, Jaffe M, Attias D, Jagerman K. 1985. The histological examination of primary enamel as a possible diagnostic tool in developmental disturbances. Journal of Pedodontics 10(1):68–75.

Kaplan BA. 1954. Environment and human plasticity. American Anthropologist 56(5):780–800.

Kasetty S, Rammanohar M, Raju Ragavendra T. 2010. Dental Cementum in Age Estimation: A Polarized Light and Stereomicroscopic Study. Journal of Forensic Sciences 55(3):779–783.

Kaupová S, Brůžek J, Velemínský P, Černíková A. 2013. Urban-rural differences in stature in the population of medieval Bohemia. Anthropologischer Anzeiger 70(1):43–55.

Kaur I, Shetty D, Manchada A, Singh HP. 2011. Efficacy Of Age Estimation In Forensic Dentistry Using Cemental Annulations As A Criteria. The Internet Journal of Forensic Science 4(2): doi. 10.5580/1e57.

Kausrud KL, Begon M, Ari TB, Viljugrein H, Esper J, Büntgen U, Leirs H, Junge C, Yang B, Yang M, Xu L, Stenseth NC. 2010. Modeling the epidemiological history of plague in Central Asia: Palaeoclimatic forcing on a disease system over the past millennium. BMC Biology 8(1):112.

Kemkes-Grottenthaler A. 2005. The short die young: The interrelationship between stature and longevity—evidence from skeletal remains. American Journal of Physical Anthropology 128(2):340–347.

Kieffer-Olsen J. 1993. Grav og gravskik i det middelaldrige Danmark: otte kirkegårdsudgravninger. Doctoral Dissertation. University of Århus, Århus, Denmark.

King T, Hymphrey LT, Hillson S. 2005. Linear Enamel Hypoplasias as Indicators of Systemic Physiological Stress: Evidence From Two Known Age-at-Death and Sex Populations From Postmedieval London. American Journal of Physical Anthropology 128(3):547–559.

Kjærgård, A. 2006. *Udgravningsrapport: Undersøgelse af middelalderlig kirkegård, Sejet Ødekirkegård* (Excavation No. 2003-2123-0776) (pp. 1–202). Horsens, Denmark: Horsens Museum.

Klein H. 1931. Etiology of Enamel Hypoplasia in Rickets as Determined by Studies on Rats and Swine. Journal of the American Dental Association 18:866.

Klein SL. 2000. The effects of hormones on sex differences in infection: from genes to behavior. Neuroscience & Biobehavioral Reviews 24(6):627–638.

Koepke N, Baten J. 2005. The biological standard of living in Europe during the last two millennia. European Review of Economic History 9(1):61–95.

Komlos J. 1990. Height and Social Status in Eighteenth-Century Germany. The Journal of Interdisciplinary History 20(4):607–621.

Komlos J. 1993. The Secular Trend in the Biological Standard of Living in the United Kingdom, 1730-1860. The Economic History Review 46(1):115–144.

Komlos J, Baten J. 2004. Looking Backward and Looking Forward: Anthropometric Research and the Development of Social Science History. Social Science History 28(2):191–210.

Konigsberg LW, Hens SM, Jantz LM, Jungers WL. 1998. Stature estimation and calibration: Bayesian and maximum likelihood perspectives in physical anthropology. American Journal of Physical Anthropology 107(S27):65–92.

Korte SM, Koolhaas JM, Wingfield JC, McEwen BS. 2005. The Darwinian concept of stress: benefits of allostasis and costs of allostatic load and the trade-offs in health and disease. Neuroscience and Biobehavioral Reviews 29(1):3–38.

Kristensen H. 1999. Land, by og bygninger. In E Roesdahl (ed.) Daglivliv i Danmarks middelalder – en arkæologisk kulturhistorie. 1st ed. Copenhagen: Gyldendalske Boghandel, Nordisk Forlag A/S. p 54–81.

Kuzawa CW. 2005. Fetal origins of developmental plasticity: Are fetal cues reliable predictors of future nutritional environments? Am J Hum Biol 17(1):5–21.

Labbok MH, Clark D, Goldman AS. 2004. Breastfeeding: maintaining an irreplaceable immunological resource. Nature Reviews: Immunology 4(7):565–572.

Lamb HH. 1965. The early medieval warm epoch and its sequel. Palaeogeography, Palaeoclimatology, Palaeoecology 1:13–37.

Lamb HH. 1995. Climate, History and the Modern World. London, UK: Routledge.

Lang TJ. 2004. Estrogen as an immunomodulator. Clinical Immunology 113(3):224–230.

Langway CC, Osada K, Clausen HB, Hammer CU, Shoji H. 1995. A 10-century comparison of prominent bipolar volcanic events in ice cores. Journal of Geophysical Research: Atmospheres 100(D8):16241–16247.

Larsen CS. 1999. Bioarchaeology: Interpreting Behaviour from the Human Skeleton. Cambridge: Cambridge University Press.

La Salle S, Mertineit C, Taketo T, Moens PB, Bestor TH, and Trasler JM. 2004. Windows for sex-specific methylation marked by DNA methyltransferase expression profiles in mouse germ cells. Developmental Biology 268(2), 403–415.

Lasker GW. 1969. Human biological adaptability. The ecological approach in physical anthropology. Science (New York, NY) 166(3912):1480–1486.

La VD, Aboudharam G, Raoult D, Drancourt M. 2008. Dental Pulp as a Tool for the Retrospective Diagnosis of Infectious Diseases. In Raoult D and Drancourt M (eds.) Paleomicrobiology: Past Human Infections. Berlin Heidelberg: Springer-Verlag, p. 175-196.

Lavigne F, Degeai J-P, Komorowski J-C, Guillet S, Robert V, Lahitte P, Oppenheimer C, Stoffel M, Vidal CM, Pratomo I. 2013. Source of the great AD 1257 mystery eruption unveiled, Samalas volcano, Rinjani Volcanic Complex, Indonesia. Proceedings of the National Academy of Sciences 110(42):16742–16747.

Lemanske RF. 2002. The Childhood Origins of Asthma (COAST) study. Pediatric Allergy and Immunology 13(s15):38–43.

Lewis ME. 2002. Impact of industrialization: Comparative study of child health in four sites from medieval and postmedieval England (A.D. 850-1859). American Journal of Physical Anthropology 119(3):211–223.

Lewis ME, Roberts CA, Manchester K. 1995. Comparative study of the prevalence of maxillary sinusitis in later Medieval urban and rural populations in Northern England. American Journal of Physical Anthropology 98(4):497–506.

Liggio LP. 2007. The Hanseatic League and Freedom of Trade. Journal of Private Enterprise 23(1): 134-141.

Lovejoy CO. 1985. Dental wear in the Libben population: its functional pattern and role in the determination of adult skeletal age at death. American Journal of Physical Anthropology 68(1):47–56.

Lovejoy CO, Meindl RS, Pryzbeck TR, Mensforth RP. 1985. Chronological metamorphosis of the auricular surface of the ilium: A new method for the determination of adult skeletal age at death. American Journal of Physical Anthropology 68(1):15–28.

Lovejoy CO, Russell KF, Harrison ML. 1990. Long bone growth velocity in the Libben population. American Journal of Human Biology 2(5):533–541.

Luckman BH, Wilson RJS. 2005. Summer temperatures in the Canadian Rockies during the last millennium: a revised record. Climate Dynamics 24(2):131–144.

Lynnerup N, Jacobsen JCB. 2003. Brief Communication:Age and fractal dimensions of human sagittal and coronal sutures. American Journal of Physical Anthropology 121(4):332–336.

MacGregor J. 2008. Introduction to the Anatomy and Physiology of Children : A Guide for Students of Nursing, Child Care and Health. Florence, KY, USA: Routledge. Available from: http://site.ebrary.com/lib/alltitles/docDetail.action?docID=10228323

Maijanen H. 2009. Testing Anatomical Methods for Stature Estimation on Individuals from the W. M. Bass Donated Skeletal Collection. Journal of Forensic Sciences 54(4):746–752.

Maijanen H, Niskanen M. 2006. Comparing stature-estimation methods on Medieval inhabitants of Westerhus, Sweden. Fennoscandia Archaeologica 23:37–46.

Maijanen H, Niskanen M. 2010. New regression equations for stature estimation for medieval Scandinavians. International Journal of Osteoarchaeology 20(4):472–480.

Manchester K. 1984. Tuberculosis and leprosy in antiquity: an interpretation. Medical History 28(2):162–173.

Manchester K. 1991. Tuberculosis and leprosy: evidence for interaction of disease. In: DJ Ortner and AC Aufderheide (eds.)Human palaeopathology: Current syntheses and future options. Washington, DC: Smithsonian Institution Press. p 23–35.

Matthes FE. 1939. Report of Committee on Glaciers, April 1939. Transactions, American Geophysical Union 20(4):518.

Matthews JA, Briffa KR. 2005. The “little Ice Age”: Re-Evaluation of an Evolving Concept. Geografiska Annaler: Series A, Physical Geography 87(1):17–36.

Mays SA. 2007. Lysis at the anterior vertebral body margin: evidence for brucellar spondylitis? International Journal of Osteoarchaeology 17(2):107–118.

Mays S, Faerman M. 2001. Sex Identification in Some Putative Infanticide Victims from Roman Britain Using Ancient DNA. Journal of Archaeological Science 28(5):555–559.

McEwen BS. 1998. Stress, adaptation, and disease: Allostasis and allostatic load. Annals of the New York Academy of Sciences 840(1):33–44.

McEwen BS. 2002. Sex, stress and the hippocampus: allostasis, allostatic load and the aging process. Neurobiology of Aging 23(5):921–939.

McEwen BS. 2004. Protection and damage from acute and chronic stress: allostasis and allostatic overload and relevance to the pathophysiology of psychiatric disorders. Annals of the New York Academy of Sciences 1032(1):1–7.

McEwen BS. 2008. Central effects of stress hormones in health and disease: Understanding the protective and damaging effects of stress and stress mediators. European Journal of Pharmacology 583(2-3):174–185.

McEwen BS, Wingfield JC. 2003. The concept of allostasis in biology and biomedicine. Hormones and Behavior 43(1):2–15.

McEwen BS, Wingfield JC. 2010. What's in a name? Integrating homeostasis, allostasis and stress. Hormones and Behavior 57(2):105.

McMichael AJ. 2010. Paleoclimate and bubonic plague: a forewarning of future risk? BMC Biology 8(1):108.

Meaney MJ. 2010. Epigenetics and the biological definition of gene x environment interactions. Child Development 81(1):41–79.

Meiklejohn C, Babb J. 2011. Long Bone Length, Stature and Time in the European Late Pleistocene and Early Holocene. In R Pinhasi and JT Stock (eds.) Human Bioarchaeology of the Transition to Agriculture. Chichester: Wiley-Blackwell, p 151–175.

Meindl RS, Lovejoy CO. 1985. Ectocranial suture closure: A revised method for the determination of skeletal age at death based on the lateral-anterior sutures. American Journal of Physical Anthropology 68(1):57–66.

Mellanby M. 1929. Diet and teeth: an experimental study, part I: Dental structure in dogs. Medical Research Council Special Report 140, pp. 308. London: Medical Research Council.

Mellanby M. 1930. Diet and the Teeth. An Experimental Study. Part II. A. Diet and Dental Disease. B. Diet and Dental Structure in Mammals other than the Dog. Med Res Council Special Report 153, pp. 94. London: Medical Research Council

Mellanby M. 1934. Diet and the teeth. An experimental study. Part 3. The effect of diet on dental structure and disease in man. Med Res Council Special Report 191, pp.180. London: Medical Research Council.

Mensforth RP. 1985. Relative tibia long bone growth in the Libben and Bt-5 prehistoric skeletal populations. American Journal of Physical Anthropology 68(2):247–262.

Mensforth RP. 1990. Paleodemography of the Carlston Annis (Bt-5) Late Archaic skeletal population. American Journal of Physical Anthropology 82(1):81–99.

Milner GR, Boldsen JL. 2012a. Skeletal Age Estimation: Where We Are and Where We Should Go. A Companion to Forensic Anthropology:224–238.

Milner GR, Boldsen JL. 2012b. Transition analysis: A validation study with known-age modern American skeletons. American Journal of Physical Anthropology 148(1):98–110.

Milner GR, Wood JW, Boldsen JL. 2008. Advances in Paleodemography. In MA Katzenberg and SR Saunders (eds.) Biological Anthropology of the Human Skeleton. Vol. 2nd. Hoboken: John Wiley & Sons, Inc.p 561–600.

Moczek AP. 2010. Phenotypic plasticity and diversity in insects. Philosophical Transactions of the Royal Society B: Biological Sciences 365(2540):593–603.

Møller-Christensen V. 1951. Om Udgravningen af Næstved Sct. Jørgensgård i Aaderup. Medicinisk Forvm 4(9):289–304.

Møller-Christensen V. 1952. Case of leprosy from the middle ages of Denmark. Acta Medica Scandinavica 142(S266):101–108.

Møller-Christensen V. 1953. Ten lepers from Næstved in Denmark: A Study of Skeletons from a Medieval Danish Leper Hospital. Copenhagen: Danish Science Press.

Møller-Christensen V. 1958. Bogen om Æbelholt Kloster. Kobenhavn: Dansk Videnskabs Forlag.

Møller-Christensen V. 1965. New knowledge of Leprosy through Paleopathology. International Journal of Leprosy 33(3):S603–S610.

Møller-Christensen V. 1978. Leprosy Changes in the Skull. Odense: Odense University Press.

Møller-Christensen V, Faber B. 1952. Leprous Changes in a material of medieval skeletons from the St. Georges Court, Næstved. Acta Radiologica [Old Series] 37(3):308–317.

Mutolo MJ, Jenny LL, Buszek AR, Fenton TW, Foran DR. 2012. Osteological and molecular identification of brucellosis in ancient Butrint, Albania. American Journal of Physical Anthropology 147(2):254–263.

Naji S, Bertrand B, D'Incau E, Brandt E, Blondiaux. 2013. Cementochronology, to cut or not to cut? Poster presented at the 82nd Meeting of the American Association of Physical Anthropologists, Knoxville, Tennessee.

Naurzbaev MM, Vaganov EA, Sidorova OV, Schweingruber FH. 2002. Summer temperatures in eastern Taimyr inferred from a 2427-year late-Holocene tree-ring chronology and earlier floating series. The Holocene 12(6):727–736.

Neiburger EJ. 1990. Enamel hypoplasias: Poor indicators of dietary stress. American Journal of Physical Anthropology 82(2):231–232.

Nesje A, Dahl SO, Thun T, Nordli Ø. 2008. The "Little Ice Age" glacial expansion in western Scandinavia: summer temperature or winter precipitation? Climate Dynamics 30(7):789–801.

Nguyen-Hieu T, Aboudharam G, Drancourt M. 2011. Mini Review: Dental Pulp as a Source for Paleomicrobiology. Bulletin of the International Association for Paleodontology 5(1): 48-54.

Nuorala E. 2004. Molecular Palaeopathology : Ancient DNA analyses of the bacterial diseases tuberculosis and leprosy. Doctoral Thesis. Stockholm: Arkeologiska forskningslaboratoriet.

Ogilvie AEJ, Jónsson T. 2001. " Little Ice Age" Research: A Perspective from Iceland. Climatic Change 48(1):9–52.

Olivier G, Aaron C, Fully G, Tissier G. 1978. New estimations of stature and cranial capacity in modern man. Journal of Human Evolution 7(6):513–518.

Orrman E. 2003. The condition of the rural population. In: The Cambridge History of Scandinavia. Vol. 1. Cambridge: Cambridge University Press. p 581–610.

Ortner DJ. 1991. Theoretical and methodological issues in paleopathology. In DJ Ortner and AC Aufderheide (eds.) Human Paleopathology: Current Syntheses and Future Options. Washington: Smithsonian Institution Press. p 5–12.

Ortner DJ. 2003. Identification of Pathological Conditions in Human Skeletal Remains. San Diego: Academic Press.

Osipov B, Harvati K, Nathena D, Spanakis K, Karantanas A, Kranioti EF. 2013. Sexual dimorphism of the bony labyrinth: A new age-independent method. American Journal of Physical Anthropology 151(2):290–301.

Papagrigorakis MJ, Yapijakis C, Synodinos PN, Baziotopoulou-Valavani E. 2006. DNA examination of ancient dental pulp incriminates typhoid fever as a probable cause of the Plague of Athens. International Journal of Infectious Diseases 10(3):206–214.

Pearson K. 1899. Mathematical Contributions to the Theory of Evolution. V. On the Reconstruction of the Stature of Prehistoric Races. Philosophical Transactions of the Royal Society of London Series A, Containing Papers of a Mathematical or Physical Character 192:169–244.

Pedersen D, Boldsen J. 2008. Antropologisk Rapport, HOM 1046, Sejet Ødekirkegård. Odense, Denmark: Retsmedicinsk Institut, Antropologisk afdeling, ADBOU Syddansk Universitet.

Pedersen D, Boldsen J. 2010. Amtropologisk Rapport: HOM 1649, Ole Worms Gade. Odense, Denmark: Retsmedicinsk Institut, Antropologisk afdeling, ADBOU Syddansk Universitet.

Perizonius WRK. 1984. Closing and non-closing sutures in 256 crania of known age and sex from Amsterdam (a.d. 1883–1909). Journal of Human Evolution 13(2):201–216.

Petersen HC. 2005. On the Accuracy of Estimating Living Stature from Skeletal Length in the Grave and by Linear Regression. International Journal of Osteoarchaeology 15(2):106–114.

Petersen HC. 2011. Technical note: A re-evaluation of stature estimation from skeletal length in the grave. American Journal of Physical Anthropology 144(2):327–330.

Pindborg JJ. 1982. Aetiology of developmental enamel defects not related to fluorosis. International Dental Journal 32(2):123–134.

Poulsen B. 1997. Agricultural technology in medieval Denmark. In GG Astill and J Langdon (eds.) Medieval farming and technology: the impact of agricultural change in Northwest Europe. Leiden: Koninkijke Brill. p 115–145.

Preus HR, Marvik OJ, Selvig KA, Bennike P. 2011. Ancient bacterial DNA (aDNA) in dental calculus from archaeological human remains. Journal of Archaeological Science 38(8):1827–1831.

Qvist M, Grøntved AM. 2001. Chronic otitis media sequelae in Skeletal Material from Medieval Denmark. Laryngoscope 111(1):114–118.

Raoult D, Aboudharam G, Crubezy E, Larrouy G, Ludes B, Drancourt M. 2000. Molecular identification by "suicide PCR" of Yersinia pestis as the agent of Medieval Black Death. Proceedings of the National Academy of Sciences 97(23):12800–12803.

Raxter MH, Auerbach BM, Ruff CB. 2006. Revision of the Fully technique for estimating statures. American Journal of Physical Anthropology 130(3):374–384.

Raxter MH, Ruff CB, Auerbach BM. 2007. Technical Note: Revised Fully Stature Estimation. American Journal of Physical Anthropology 133:817–818.

Razali NM, Wah YB. 2011. Power comparisons of shapiro-wilk, kolmogorov-smirnov, lilliefors and anderson-darling tests. Journal of Statistical Modeling and Analytics 2(1):21–33.

Redin L. 1976. Lagmanshejdan: Ett gravfält som spegling av sociala strukturer i Skanör. Acta Archaeologica Lundensia 4(10):1–201. Bonn and Lund

Reid DJ, Dean MC. 2000. Brief Communication: The Timing of Linear Hypoplasias on Human Anterior Teeth. American Journal of Physical Anthropology 113(1):135–139.

Reid DJ, Dean MC. 2006. Variation in modern human enamel formation times. Journal of Human Evolution 50(3):329–346.

Reid DJ, Ferrell RJ. 2006. The relationship between number of striae of Retzius and their periodicity in imbricational enamel formation. Journal of Human Evolution 50(2):195–202.

Retzius A. 1837. Microscopic investigation of the structure of the teeth. Archives of Anatomical Physiology 8:486–519.

Richards P. 1960. Leprosy in Scandinavia. Centaurus 7(1):101–133.

Richards P. 2000. The medieval leper and his northern heirs. Woodbridge, Suffolk: Boydell & Brewer Ltd.

Risnes S. 1986. Enamel apposition rate and the prism periodicity in human teeth. Scandinavian Journal of Dental Research 94(5):394–404.

Risnes S. 1998. Growth tracks in dental enamel. Journal of Human Evolution 35(4-5):331–350.

Roberts CA, Buikstra JE. 2003. The Bioarchaeology of Tuberculosis: A Global View on a Reemerging Disease. Florida: University Press of Florida.

Roberts CA, Cox M. 2003. Health and Disease in Britain: From prehistory to the present day. Gloucester: Sutton Publishing.

Roberts C, Manchester K. 2005. The archaeology of disease, 3rd ed. Sparkford, UK: Sutton Publishing.

Roberts C., Satoskar A, Alexander J. 1996. Sex steroids, pregnancy-associated hormones and immunity to parasitic infection. Parasitology Today 12(10):382–388.

Roberts DF. 1995. The pervasiveness of plasticity. Human Variability and Plasticity:1–17.

Robock A. 2000. Volcanic eruptions and climate. Reviews of Geophysics 38(2):191–219.

Roksandic M, Vlak D, Schillaci MA, Voicu D. 2009. Technical note: Applicability of tooth cementum annulation to an archaeological population. Am J Phys Anthropol 140(3):583–588.

Romero LM, Dickens MJ, Cyr NE. 2009. The reactive scope model—a new model integrating homeostasis, allostasis, and stress. Hormones and Behavior 55(3):375–389.

Roseboom TJ, van der Meulen JHP, Ravelli ACJ, Osmond C, Barker DJP, Bleker OP. 2001. Effects of Prenatal Exposure to the Dutch Famine on Adult Disease in Later Life: An Overview. Twin Research 4(5):293–298.

Rose JC. 1977. Defective Enamel Histology of Prehistoric Teeth from Illinois. American Journal of Physical Anthropology 46(3):439–446.

Rose JC, Armelagos GJ, Lallo JW. 1978. Histological Enamel Indicator of Childhood Stress in Prehistoric Skeletal Samples. American Journal of Physical Anthropology 49(4):511–516.

Roth E. 1992. Comment to The Osteological Paradox. Current Anthropology 33(4):363–364.

Rothschild BM, Rothschild C. 1995. Treponemal Disease Revisited: Skeletal Discriminators for Yaws, Bejel, and Venereal Syphilis. Clinical Infectious Diseases 20(5):1402–1408.

Ruff C. 2002. Variation in Human Body Size and Shape. Annual Review of Anthropology 31:211–232.

La Salle S, Mertineit C, Taketo T, Moens PB, Bestor TH, Trasler JM. 2004. Windows for sex-specific methylation marked by DNA methyltransferase expression profiles in mouse germ cells. Developmental Biology 268(2):403–415.

Der Sarkissian C, Balanovsky O, Brandt G, Khartanovich V, Buzhilova A, Koshel S, Zaporozhchenko V, Gronenborn D, Moiseyev V, Kolpakov E, Shumkin V, Alt KW, Balanovska E, Cooper A, Haak W, the Genographic Consortium. 2013. Ancient DNA Reveals Prehistoric Gene-Flow from Siberia in the Complex Human Population History of North East Europe. PLoS Genet 9(2):e1003296.

Sarnat BG, Schour I. 1941. Enamel hypoplasias (chronic enamel aplasia) in relationship to systemic diseases: a chronological, morphological and etiological classification. Journal of the American Dental Association 28:1989–2000.

Saunders SR, Hoppa RD. 1993. Growth deficit in survivors and non-survivors: Biological mortality bias in subadult skeletal samples. American Journal of Physical Anthropology 36(S17):127–151.

Schell LM. 1995. Human biological adaptability with special emphasis on plasticity: history, development and problems for future research. In CGN Mascie-Taylor and B Bogin (eds.) Human variability and plasticity. Cambridge: Cambridge University Press. p 213–237.

Scheuer L, Black S. 2000. Developmental Juvenile Osteology. London: Academic Press.

Schuenemann VJ, Bos K, DeWitte S, Schmedes S, Jamieson J, Mittnik A, Forrest S, Coombes BK, Wood JW, Earn DJD, White W, Krause J, Poinar HN. 2011. Targeted enrichment of ancient pathogens yielding the pPCP1 plasmid of Yersinia pestis from victims of the Black Death. Proceedings of the National Academy of Sciences 108(38):E746–E752.

Schwartz GT, Reid DJ, Dean MC, Zihlman AL. 2006. A Faithful Record of Stressful Life Events Recorded in the Dental Developmental Record of a Juvenile Gorilla. Int J Primatol 27(4):1201–1219.

Sciulli PW, Giesen MJ. 1993. An update on stature estimation in prehistoric native Americans of Ohio. American Journal of Physical Anthropology 92(3):395–399.

Sciulli PW, Hetland BM. 2007. Stature Estimation for Prehistoric Ohio Valley Native American Populations Based on Revisions of the Fully Technique. Archaeology of Eastern North America 35:105–113.

Sciulli PW, Schneider KN, Mahaney MC. 1990. Stature estimation in prehistoric Native Americans of Ohio. American Journal of Physical Anthropology 83(3):275–280.

Seckler D. 1980. Small but Healthy: A Crucial Hypothesis in the Theory, Measurement and Policy of Malnutrition. mimeo.

Seckler D. 1982. Small but healthy? Some basic problems in the concept of malnutrition. Newer concepts in nutrition and their implications for policy Pune, India: Maharashtra Association for the Cultivation of Science p:139–148.

Seckler D. 1984. The "Small but Healthy?" Hypothesis: A Reply to Critics. Economic and Political Weekly 19(44):1886–1888.

Segel KL. 2001. Bioarchaeological analysis of St. Jørgensgård, a Medieval Leprosy Hospital in Odense, Denmark. Doctoral Dissertation. Chicago: The University of Chicago.

Selevan SG, Kimmel CA, Mendola P. 2000. Identifying critical windows of exposure for children's health. Environmental Health Perspectives 108 (Suppl. 3):451.

Seow WK. 1991. Enamel hypoplasia in the primary dentition: a review. Journal of Dentistry for Children 58(6):441–452.

Shames RS. 2002. Gender differences in the development and function of the immune system. Journal of Adolescent Health 30(4):59–70.

Simmons RA. 2009. Developmental origins of adult disease. The Pediatric Clinics of North America 56(3):449–466.

Sjøvold T. 1990. Estimation of stature from long bones utilizing the line of organic correlation. Human Evolution 5(5):431–447.

Smith TM. 2006. Experimental determination of the periodicity of incremental features in enamel. Journal of Anatomy 208(1):99–113.

Smith TM, Reid DJ, Dean MC, Olejniczak AJ, Professor RJFA, Martin LB. 2007. New perspectives on chimpanzee and human molar crown development. In: SE Bailey and J-J Hublin (eds.). Dental Perspectives on Human Evolution: State of the Art Research in

Dental Paleoanthropology. Vertebrate Paleobiology and Paleoanthropology. Dordrecht, Netherlands: Springer, p 177–192.

Snowball I, Korhola A, Briffa KR, Koç N. 2004. Holocene climate dynamics in Fennoscandia and the North Atlantic. In RW Battarbee, F Gasse, and CE Stickley (eds.) Past climate variability through Europe and Africa 6. Netherlands: Springer, pp. 465–494.

Steckel RH. 1995. Stature and the Standard of Living. Journal of Economic Literature 33(4):1903–1940.

Steckel RH. 2004. New Light on the "Dark Ages": The Remarkably Tall Stature of Northern European Men during the Medieval Era. Social Science History 28(2):211–229.

Steckel RH. 2005. Young adult mortality following severe physiological stress in childhood: Skeletal evidence. Economics & Human Biology 3(2):314–328.

Steckel RH, Rose JC. 2002. The backbone of history: health and nutrition in the Western Hemisphere. Cambridge: Cambridge University Press.

Steckel RH, Rose JC, Larsen CS, Walker PL. 2002. Skeletal health in the Western Hemisphere from 4000 B.C. to the present. Evolutionary Anthropology: Issues, News, and Reviews 11(4):142–155.

Steinbock RT. 1976. Paleopathological Diagnosis and Interpretation: Bone Diseases in Ancient Human Populations. Springfield, Il: Charles C Thomas Pub Limited.

Steinskog DJ, Tjøstheim DB, Kvamstø NG. 2007. A Cautionary Note on the Use of the Kolmogorov–Smirnov Test for Normality. Monthly Weather Review 135(3):1151–1157.

Stenseth NC, Atshabar BB, Begon M, Belmain SR, Bertherat E, Carniel E, Gage KL, Leirs H, Rahalison L. 2008. Plague: Past, Present, and Future. PLoS Med 5(1):e3.

Stenseth NC, Samia NI, Viljugrein H, Kausrud KL, Begon M, Davis S, Leirs H, Dubyanskiy VM, Esper J, Ageyev VS, Klassovskiy NL, Pole SB, Chan K-S. 2006. Plague dynamics are driven by climate variation. PNAS 103(35):13110–13115.

Stini WA. 1969. Nutritional stress and growth: Sex difference in adaptive response. American Journal of Physical Anthropology 31(3):417–426.

Stinson S. 1985. Sex differences in environmental sensitivity during growth and development. American Journal of Physical Anthropology 28(S6):123–147.

Stinson S. 1992. Nutritional Adaptation. Annual Review of Anthropology 21:143–170.

Stone AC, Wilbur AK, Buikstra JE, Roberts CA. 2009. Tuberculosis and leprosy in perspective. American Journal of Physical Anthropology 140(S49):66–94.

Stone A, Milner GR, Pääbo S, Stoneking M. 1996. Sex determination of ancient human skeletons using DNA. American Journal of Physical Anthropology 99:231–238.

Stothers RB. 1999. Volcanic Dry Fogs, Climate Cooling, and Plague Pandemics in Europe and the Middle East. Climatic Change 42(4):713–723.

Stuart-Macadam PL. 1991. Porotic hyperostosis: changing interpretations. In DJ Ortner and AC Aufderheide (eds.) Human Paleopathology: Current Syntheses and Future Options. Washington: Smithsonian Institution Press, pp. 36–39.

Suckling G. 1980. Defects of Enamel in Sheep Resulting from Trauma During Tooth Development. Journal of Dental Research 59(9):1541–1548.

Suckling G, Elliott DC, Thurley DC. 1983. The Production of Developmental Defects of Enamel in the Incisor Teeth of Penned Sheep Resulting from Induced Parasitism. Archives of Oral Biology 28(5):393–399.

Suckling G, Elliott DC, Thurley DC. 1986. The Macroscopic Appearance and Associated Histological Changes in the Enamel Organ of Hypoplastic Lesions of Sheep Incisor Teeth Resulting from Induced Parasitism. Archives of Oral Biology 31(7):427–439.

Suckling GW, Pearce EIF. 1984. Developmental defects of enamel in a group of New Zealand children: their prevalence and some associated etiological factors. Oral Epidemiology 12(3):177–184.

Suckling GW, Purdell-Lewis DJ. 1982. The Pattern of Mineralization of Traumatically-induced Developmental Defects of Sheep Enamel Assessed by Microhardness and Microradiography. Journal of Dental Research 61(10):1211–1216.

Sweeney EA, Cabrera J, Urrutia J, Mata L. 1969. Factors Associated with Linear Hypoplasia of Human Deciduous Incisors. Journal of Dental Research 48(6):1275–1279.

Sweeney EA, Saffir AJ, de Leon R. 1971. Linear hypoplasia of deciduous incisor teeth in malnourished children. The American Journal of Clinical Nutrition 24(1):29–31.

Tanner JM. 1963. The Regulation of Human Growth. Child Development 34(4):817–847.

Tanner JM. 1981a. Growth and Maturation during Adolescence. Nutrition Reviews 39(2):43–55.

Tanner JM. 1981b. A History of the Study of Human Growth. Cambridge: Cambridge University Press.

Tanner JM. 1981c. Catch-up Growth in Man. Br Med Bull 37(3):233–238.

Tarp, P. 2009. CEI-Analyse - Ny Metode til Aldersbestemmelse se ved Døden i Skeletsamlinger (CEI-Analysis - New Ways in Determining Age at Death in Skeletal Samples). Masters Dissertation. Syddansk Universitet (University of Southern Denmark), Odense.

Tarp P. 2011. Antropologisk Rapport: VSM 09.715 Viborg Sct. Morten. Odense, Denmark: Retsmedicinsk Institut, Antropologisk afdeling, ADBOU Syddansk Universitet.

Taylor D. 1969. Differential rates of cerebral maturation between sexes and between hemispheres: Evidence from epilepsy. The Lancet 294(7612):140–142.

Taylor GM, Watson CL, Bouwman AS, Lockwood DNJ, Mays SA. 2006. Variable nucleotide tandem repeat (VNTR) typing of two palaeopathological cases of lepromatous leprosy from Mediaeval England. Journal of Archaeological Science 33(11):1569–1579.

Telkkä; A. 1950. On the prediction of human stature from long bones. Cells Tissues Organs 9(1-2):103–117.

Thomas RF. 2003. Enamel defects, well-being and mortality in a medieval Danish Village. Unpublished Doctoral Dissertation. Pennsylvania State University.

Todd TW. 1920. Age changes in the pubic bone. I. The male white pubis. American Journal of Physical Anthropology 3(3):285–334.

Tran T-N-N, Forestier CL, Drancourt M, Raoult D, Aboudharam G. 2011. Brief communication: Co-detection of Bartonella quintana and Yersinia pestis in an 11th-15th burial site in Bondy, France. American Journal of Physical Anthropology 145(3):489–494.

Trotter M. 1970. Estimation of stature from intact long limb bones. In TD Stewart (ed) Personal identification in mass disasters. Washington, DC: National Museum of Natural History, pp. 71–83.

Trotter M, Gleser GC. 1951. Trends in stature of American whites and Negroes born between 1840 and 1924. American journal of physical anthropology 9(4):427.

Trotter M, Gleser GC. 1952. Estimation of stature from long bones of American Whites and Negroes. American Journal of Physical Anthropology 10(4):463–514.

Trotter M, Gleser GC. 1958. A re-evaluation of estimation of stature based on measurements of stature taken during life and of long bones after death. American Journal of Physical Anthropology 16(1):79.

Trotter M, Gleser GC. 1977. Corrigenda to "estimation of stature from long limb bones of American Whites and Negroes," American Journal Physical Anthropology (1952). American Journal of Physical Anthropology 47(2):355–356.

Tuli SM. 2004. Tuberculosis of the Skeletal System: Bones, Joints, Spine and Bursal Sheaths. New Delhi: Jaypee Brothers Publishers.

Turner S. 2013. Dietary Reconstruction in Medieval Holbæk, Denmark using Dental Microwear and Macrowear, Isotopic Analyses, and Dental Caries. Masters Thesis. Georgia: Georgia State University. http://digitalarchive.gsu.edu/anthro_theses/74

Uekert SJ, Akan G, Evans MD, Li Z, Roberg K, Tisler C, DaSilva D, Anderson E, Gangnon R, Allen DB, Gern JE, Lemanske Jr. RF. 2006. Sex-related differences in immune development and the expression of atopy in early childhood. Journal of Allergy and Clinical Immunology 118(6):1375–1381.

Ulijaszek SJ. 1996. Long-term consequences of early environments on human growth: a developmental perspective. In: Long-term consequences of early environment: Growth, development and the lifespan developmental perspective. Vol. 37. Society for the Study of Human Biology. Cambridge: Cambridge University Press.

Usher BM. 2000. A Multistate Model of Health and Mortality for Paleodemography: Tirup Cemetery. Doctoral Thesis. Pennsylvania: Pennsylvania State University

Verthelyi D. 2001. Sex hormones as immunomodulators in health and disease. International Immunopharmacology 1(6):983–993.

Waldron T. 2009. Palaeopathology: Cambridge Manuals in Archaeology. Cambridge: Cambridge University Press.

Walløe L. 2008. Medieval and Modern Bubonic Plague: Some Clinical Continuities. Medical History Supplement 27:59–73.

Wang M, Crisostomo P, Wairiuko GM, Meldrum DR. 2006. Estrogen receptor-α mediates acute myocardial protection in females. American Journal of Physiology - Heart and Circulatory Physiology 290(6):H2204–H2209.

Watts R. 2011. Non-specific indicators of stress and their association with age at death in Medieval York: Using stature and vertebral neural canal size to examine the effects of stress occurring during different periods of development. International Journal of Osteoarchaeology 21(5):568–576.

Weinstock M. 2007. Gender Differences in the Effects of Prenatal Stress on Brain Development and Behaviour. Neurochemical Research 32(10):1730–1740.

Weise S. 2009. The Mediaeval Cemetery St. Jörgen in Malmö: A Palaeodemographic Analysis. Doctoral Thesis. Odense, Denmark: University of Southern Denmark.

Weise S, Boldsen JL, Gampe J, Milner GR. 2009. Calibrated expert inference and the construction of unbiased paleodemographic mortality profiles. American Journal of Physical Anthropology Supplement 48:269.

Weise S, Boldsen JL, Gampe J, Milner GR. 2012. How old was "Geriatrix"? Estimating the age of old individuals with Calibrated Expert Inference. Presented at the The 81st Annual Meeting of the American Association of Physical Anthropologists, Portland, OR Available from: http://meeting.physanth.org/program/2012/session08/weise-2012-how-old-was-geriatrix-estimating-the-age-of-old-individuals-with-calibrated-expert-inference.html

Welford MR, Bossak BH. 2009. Validation of Inverse Seasonal Peak Mortality in Medieval Plagues, Including the Black Death, in Comparison to Modern Yersinia pestis-Variant Diseases. PLoS ONE 4(12):e8401.

West-Eberhard MJ. 1989. Phenotypic Plasticity and the Origins of Diversity. Annual Review of Ecology and Systematics 20:249–278.

West LJ. 2002. Defining critical windows in the development of the human immune system. Human & Experimental Toxicology 21(9-10):499–505.

White TD. 1978. Early Hominid Enamel Hypoplasia. American Journal of Physical Anthropology 49(1):79–84.

White TD, Black MT, Folkens PA. 2012. Human osteology. Burlington, MA: Elsevier: Academic Press Inc.

White TD, Folkens PA. 2005. The Human Bone Manual. Burlington, MA: Academic Press.

Wiechmann I, Grupe G. 2005. Detection of Yersinia pestis DNA in two early medieval skeletal finds from Aschheim (Upper Bavaria, 6th century A.D.). American Journal of Physical Anthropology 126(1):48–55.

Wingfield JC. 2003. Control of behavioural strategies for capricious environments. Animal Behaviour 66(5):807–816.

Wingfield JC. 2005. The concept of allostasis: coping with a capricious environment. Journal of Mammalogy 86(2):248–254.

Winter GB, Brook AH. 1975. Enamel Hypoplasia and Anomalies of the Enamel. Dental Clinics of North America 19(1):3–24.

Wittwer-Backofen U, Gampe J, Vaupel JW. 2004. Tooth cementum annulation for age estimation: results from a large known-age validation study. American Journal of Physical Anthropology 123(2):119–129.

Witzel C, Kierdorf U, Schultz M, Kierdorf H. 2008. Insights From the Inside: Histological Analysis of Abnormal Enamel Microstructure Associated With Hypoplastic Enamel Defects in Human Teeth. American Journal of Physical Anthropology 136(4):400–414.

Wood J, Milner G. 1994. Reply: The Osteological Paradox Reconsidered. Current Anthropology 35(5):631–637.

Wood JW, Milner GR, Harpending HC, Weiss KM. 1992. The Osteological Paradox: Problems of Inferring Prehistoric Health from Skeletal Samples. Current Anthropology 33(4):343.

Wright LE, Yoder CJ. 2003. Recent Progress in Bioarchaeology: Approaches to the Osteological Paradox. Journal of Archaeological Research 11(1):43–70.

Ygberg S, Nilsson A. 2012. The developing immune system – from foetus to toddler. Acta Paediatrica 101(2):120–127.

Yoder C. 2010. Diet in medieval Denmark: a regional and temporal comparison. Journal of Archaeological Science 37(9):2224–2236.

Yoder CJ. 2006. The late medieval agrarian crisis and Black Death plague epidemic in medieval Denmark: A paleopathological and paleodietary perspective. Doctoral Dissertation. Texas: Texas A & M University.

iduals with Sex Re-Assigned

Ventral Arc (Phenice 1969)	Sciatic Notch	Subpubic concavity (Phenice 1969)	Medial aspect ischiopubic ramus (Phenice 1969)	Preauricular sulcus	Acetabulum	Obturator Foramen	Nuchal Crest	Mastoid Process	Supraorbital Margin	Glabella	Mental Eminence	Notes
/	F	/	/	/	/	/	F	M?	M	M	M?	Sciatic notch wide, nuchal torus smooth with minimal protruberance, olecranon fossa, overall gracile individual.
/	/	/	/	/	M?	/	M	/	/	/	M?	Extremely fragmentary indivividual. Cranial features (nuchal torus and mental eminence) are more masculine, but this individual is too fragmentary for secure sex determination.
M	?	M	M	M	M?	/	?	M?	F?	F?	?	While this individual had largely masculine features (both pelvic and cranial), with severe pathological changes (likely treponematosis), a short stature, and gracile cervical vertebra and small humeral head, it was decided to keep this individual as inderterminate.

1046	204	?	M	/	?	/	/	?	M	/	?	M?	F?	F?	?	Very mixed features and quite fragmentary. Overall more gracile individual. But scores are contradictory and / or intermediate.
1046	291	M	/	/	/	/	/	/	/	/	M?	M	M	M	M	No pelvic features were available for this individual. All cranial features were robust and strongly masculine. Postcranial size and robusticity collaborated this assessment.
1046	386	F	?	/	F?	/	/	F	?	/	F	F	F	F?	/	The combined more feminine appearance of the scorable pelvic and cranial features, combine with pverall gracile postcranium, suggest that this individual was likely female.
1046	481	F	?	/	F	/	/	/	F	/	F	F	/	/	F	This individual was quite gracile, with a wide sciateic notch (F) and a feminine acetabulum (F). All scorable cranial features were also feminine.
1046	501	F	M	/	F	/	/	F?	F?	/	F?	F	?	/	F?	The attribution of feminine scores for both pelvic and cranial features supported a feminine designation for this individual
1046	541	F	?	/	/	/	/	/	/	/	F	F	/	/	F	No pelvic features were available for this individual. The nuchal torus, mastoid process, and mental eminence were all extremely feminine.
1046	542	F	?	F	F	F	F	F	F	/	F	F	F	F	F	Most pelvic features (except the obturator foramen) were available for this individual, as well as all cranial features. All features were strongly female.
1046	575	F	M	/	?	/	/	F	F	/	?	M	F	F	F	The only feature that was scored as M for this individual was the mastoid process. Overall, this individuals scores pointed towards a female designation.
1649	1105	F	M	/	F	/	/	F	F	/	M	F	M	M	?	While this individual did have a mix of features, all scorable pelvic features were scored as F. The cranium was more mixed but greater weight was given to the clear pelvic features for final scoring of this individual as female than to the mixed cranial features.
1649	1198	F	M	/	/	/	/	/	/	/	F?	?	F	F	F?	No pelvic features were available for this individual. All available cranial features were scored as probable female or as intermediate.

1649	1424	?	M	/	?	/	/	M	?	/	/	F	F	F	M	This was a younger individual who was nevertheless fairly robust and so may well be a young male. However, limited scoring was available for the pevis (the preauricular sulcus was scored as male) and the cranium was scored mostly as probable female with the exception of the mental eminence. Due to the mixed features, there was not enough confidence for a secure sex determination.
1649	1485	?	M	/	/	/	/	/	/	/	F	F	F?	F	F?	No pelvic features were available for this individual. All cranial features were scored as female or possible female. Postrcranium was gracile to intermediate. Overall, this individual therefore looked more female than male but without pelvic features it was decided to score this individual as indeterminate.
1649	1490	F	M	/	/	/	/	/	/	/	F	F	F	F	F	No pelvic features were available for this individual. All cranial features were strongly feminine and the postcranium was gracile. These characteristics were extreme enough to result in a positive score of this individual as female.
1649	1524	F	M	/	F	/	F?	F	F	/	F	F	/	F	F?	All pelvic features were scored as probable or possible female. Cranial features agreed with this with all scores being most consitent with the individual being female.
1649	1587	F	M	F?	F	F	F?	F	F?	?	F	F	F?	F	F	All scores, both pelvic and cranial were scored as probable or possible female for this individual, giving a secure female sex determination.

Appendix 2 Individuals with Age Re-Assigned

Individual	New Sex	New Age	Original Age	Pubic symphysis	Auricular Surface	Suture Closure Age	Dental Wear Age	CEI
11	F	30-42	30-45	49-50+	50-55	48, SD 10.5	40-45	42-54
16	F	32-42	38-48	/	30-40	/	30-55	35-42
28	F	42-55	35-50	/	/	/	45-55	42+
29	M	21-24	24-28	/	/	34 SD 7.8; 41.1 SD 19	16-20	21-24
33	F	42-54	50-70	/	60+	/	55+	35-42
77	F	25-30	27-37	/	20-30	/	/	25-30
95	M	18-20	17-20	/	20-25	/	30-35	18-20
109	M	40-55	55-70	/	/	51.5 SD12.6; 45.5 SD8.9	45-55	35-50
113	M	28-35	35-50	/	30-38	/	40-55	28-35
116	F	30-38	30-40	/	30-40	/	30-50	30-35
121	M	35-45	32-42	/	35-45	AT LEAST 39.4 SD 9.1	24-50	40-50
152	F	35-45	42-52	/	/	39.4 SD9.1	40-55	30-42
156	F	30-35	38-50	/	30-40	/	40-55	30-35
172	M	28-38	27-40	/	30-40	/	24-40	28-38
188	M	33-45	35-50	/	/	/	40-55	33-45
195	M	24-34	30-37	/	30-40	/	20-30	24-30
198	?	30-40	35-45	/	0-40	48.8 SD10.5; 36.2 SD 6.2	40-55	30-35
204	?	25-30	23-28	/	25-35	39.4 SD9.1	16-30	25-30
222	F	30-40	26-35	/	25-38	/	/	35-42
243	F	20-25	18-25	/	/	/	16-24	22-35
299	F	25-35	32-42	/	/	/	24-35	25-35
311	M	25-35	28-38	18-24	30-35	/	24-40	25-33
325	M	25-35	30-38	/	/	/	20-40	25-35
386	F	30-38	37-47	/	30-40	AT LEAST 34.7 SD 7.8	24-55	30-35
387	M	28-40	45-60	/	30-45	/	35-45	28-33
388	F	22-24	18-22	/	20-24	/	/	20-30
402	M	45-60	55-75	/	50-60	AT LEAST 45.2 SD12.6	40-55+	45-60
403	F	35-45	35-55	/	30-40	/	45-55	35-42
414	M	40-55	55-70	/	60+	/	/	42+

433	M	18-22	17-20	/	20-24	/	20-24	20-24
437	F	30-42	40-60	/	/	/	45-55	35-42
440	M	33-42	38-52	/	30-40	/	40-55	33-42
465	F	27-35	27-40	/	/	/	20-24	27-35
471	M	25-33	30-40	22-26	25-30	/	20-55	24-33
472	M	22-24	23-30	20-30	25-30	34.7 SD7.8	35-45	21-24
486	F	30-40	26-34	40-45	34-44	/	30-55	35-42
504	F	20-25	17-25	/	/	/	20-24	20-24
515	?	33-45	35-50	/	35-45	45.2 SD 12.6	40-55	40-55
526	M	18-20	19-21	/	20	/	20-30	20-30
527	F	35-42	30-50	/	/	/	24-30	24-30
533	M	25-35	35-50	/	35-45	/	24-35	24-35
539	M	28-35	35-60	/	/	/	24-30	24-30
542	F	35-45	32-42	35-45	35-40	35+	24-55	24-55
554	M	28-38	35-42	25-44	30-40	/	40-55	40-55
555	M	33-42	35-50	/	25-35	/	40-55	40-55
564	M	45-60	50-65	45+	60+	48.8 SD 10.5; 45.5 SD8.9	45-55	45-55
1006	M	42-50	30-43	35-45	35-45	AT LEAST 39.4 SD9.2	35-51	35-51
1024	F	35-45	25-35	25-35	35-45	/	40-55	40-55
1026	M	32-42	30-38	30-40	35-45	48.8 SD10.5; 45.5 SD8.9+	24-45	24-45
1027	F	30-40	25-40	/	/	39.4 SD 9.1; 41.1 SD10	20-40	20-40
1030	M	21-27	21-23	/	/	45.2 SD12.6	18-40	18-40
1064	M	40-60	35-60	/	/	51.1 SD12.6; 43.4 SD10.7	20-45	20-45
1067	M	35-45	30-45	/	/	48.8 SD10.5; 51.9 SD12.5	45-55	45-55
1074	M	35-45	40-55	19-46	35-45	39.4 SD9.1(+)	45-55	45-55
1075	M	25-35	22-30	/	/	45.2 SD12.6; 41.1 SD10	24-55	24-55
1076	M	32-42	38-48	30-40?	30-34	34.7 SD7.8; /	24-50	24-50
1088	M	25-30	23-27	22-35	30-38	39.4 SD9.1	20-35	20-35
1099	M	42-55	50-65	44-60	40-44	/	45-55	45-55
1105	F	30-35	28-38	/	25-35	39.4 SD9.1	20-35	20-35
1155	F	25-35	28-38	/	/	34.7 SD7.8	20-30	20-30
1180	F	42-54	25-35	35-50	40-50	39.4 SD9.1; 32 SD8.3	40-55	40-55
1187	M	42-52	Above 25	/	/	48.8 SD10.5; 41.1 SD10	40-55	40-55
1188	M	45-60	30-60	/	35+	/; 56.2 SD8.5	40-55	40-55
1247	M	25-32	23-26	30-40	40-45	34.7 SD7.8; 32 SD8.3	20-30	20-30
1262	M	35-45	45-60	/	/	AT LEAST 39.4 SD9.1; 36.2 SD6.2	40-55	40-55
1282	F	28-35	25-33	/	25-35	30.5 SD9.6; 32 SD8.3	20-40	20-40
1285	M	35-60	Over 25	/	/	39.4 SD9.1; 36.2 SD 6.2	40-55	40-55
1292	F	28-38	32-42	/	/	39.4 SD9.1; 36.2 SD 6.2	20-35	20-35

1295	F	40-55	40-50	/	/	45.2 SD12.6; /	30-55	30-55
1333	F	35-45	45-60	/	40-50	/	35-55	35-55
1350	M	30-40	25-32	/	/	At least 39.4 SD 9.1	20-45	20-45
1360	M	42-55	27-32	35-50	35-50	45.2 SD12.6; 41.1 SD10	40-55	40-55
1362	M	33-50	25-45	/	/	/	24-50	24-50
1376	F	54-65	50-65	/	55+	/	40-55	40-55
1401	M	35-45	30-45	35-45	40-60	/	40-55+	40-55+
1402	M	42-55	50-70	35-50	50-60	48.8 SD10.5; 32 SD8.3	30-55	30-55
1416	M	42-50	38-40	44-50	35-50	/; 56.2 SD8.5	24-35	24-35
1432	F	35-50	40-55	/	/	/	40-50	40-50
1436	F	40-55	27-40	/	/	51.5 SD12.6; 45.5 SD8.9	40-55	40-55
1480	M	35-45	25-38	/	/	/; 56.2 SD8.5	30-55	30-55
1485	?	27-35	25-30	/	/	45.2 SD12.6; 36.2 SD6.2	16-30	16-30
1501	F	42-54	35-50	/	/	/; 56.2 SD8.5	30-55	30-55
1519.2	F	35-50	35-55	/	35-40	39.4 SD9.1; /	35-55	35-55
1524	F	32-42	40-50	/	/	45.2 SD12.6; 41.1 SD10	35-55	35-55
1543	M	50-70	55-80	/	50-70	/	45-55+	45-55+
1547	F	30-38	30-45	/	30-45	34.7 SD7.8	30-55	30-55
1553	M	33-45	35-50	39-44	40-45	48.8 SD10.5; 41.1 SD10	40-55	40-55
1570	F	45-60	50-70	/	40-50	AT LEAST 48.8 SD 10.5	35-55+	35-55+
1587.1	F	18-21	18-19	18-19	20		16-24	16-24
1638	M	35-45	30-40	/	40-50	AT LEAST 39.4 SD9.1	24-55	24-55
1639	M	33-45	35-45	35-45	30-35	39.4 SD9.1	35-55	35-55