Was childhood health declining in the period leading to the Black Death?

A comparison between early and late pre-Black Death, Black Death, and post-Black Death periods

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To my family and baby tortoises who I love with all my heart.

Abstract

The Black Death was a pandemic that spread through Africa, Asia, and Europe. It was caused by a zoonotic disease known as the bubonic plague. This disease is transmitted by the bacterium *Yersinia pestis*. In England, the Black Death was an unprecedented catastrophic event dated between the years AD 1348-1350. There are multiple debates in relation to the exact mortality of the plague. However, it was estimated to have been between 30% and 60% of the population. Although traditionally it was considered that the plague had started a period of crisis, further studies indicate that the outbreak was part of a longer period of hardship that started in the 12th century. More recent studies have tackled analysing the period leading to the Black Death. However, these studies have focused exclusively on individuals living in London.

This investigation aimed to evaluate childhood health in the period leading to the Black Death and its possible influence on the outbreak within and outside London to assess whether population human health was failing before the pandemic. The study sample comprised primary and secondary data from individuals from five sites outside of London (n=1341) and five sites from the London region (n=724).

Individuals were examined for multiple indicators of physiological stress (cribra orbitalia, DEH), dietary indicators (vitamin C and D deficiency, folic acid), infections related to immunosuppression (tuberculosis), and estimates of disrupted growth and development (puberty stage estimation, VNC dimensions, and long bone osteometrics).

Results showed evidence of stress during the late pre-Black Death period. However, when compared to the early pre- and post-Black Death periods, few statistically significant differences were found. The London region showed a significant decline in health during the Black Death period which represented not only the catastrophic outbreak but also a decline in health in the period immediately before the pandemic. This decline was followed by an improvement in health, especially for men, which reflected an increase in access to resources and higher wages after the plague. Changes in the health of females were less clear. Comparisons between regions showed different patterns, especially between males and females. These differences show the variability existing in the extra-London region concerning how the plague developed and affected rural populations.

This study highlights the importance of studying health status not only after but also before a pandemic, as well as analysing regions outside London to create a more comprehensive image of health in medieval England.

Contents

List of Figures

Chapter 2: The Black Death and the medieval period **9**

List of Tables

Chapter 5: Results **110**

Declaration

I confirm that this is my own work and the use of all material from other sources has been properly and fully acknowledged.

FLORENCIA NATALIA BOTTA

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Disclaimer

As I am not a native English speaker, a proofreader (Dr Coco's Proofreading service - Cory Stade) was used to check some sections of this thesis for typos. Changes made by the proofreader were not final and were revised and modified by the author before the submission of this thesis. The proofreader did not contribute to any of the content of the writing in this thesis.

1 | Introduction

1.1 Background and rationale

The Black Death is the name of a pandemic that hit Africa, Asia and Europe during the 14th century. It was caused by a disease called bubonic plague. In Europe, it caused massive mortality, leading to the death of at least 30% of the population. Although there were smaller outbreaks of the disease throughout the late medieval period, the Black Death pandemic was the most significant for the region. Although this pandemic affected several countries, the focus of this investigation was on England where the outbreak hit between AD 1348 and 1350 (Benedictow, 2004).

The Black Death was not the first or last instance where the bubonic plague was identified. Outside smaller outbreaks, there have been several large epidemics and pandemics caused by the disease. The first one was identified in the Roman world and was named the Justinian plague. It spread through Europe starting in the years AD 541-544 up to 750 (Keller et al., 2019). The Black Death was the name given to the second pandemic previously explained. Afterwards, there were smaller epidemics like the Great London Plague, which affected London between AD 1665 and 1666 (Defoe, 2008), as well as modern epidemics outside Europe like the Indian plague which hit the country between 1886 and 1902-21 and resulted in the death of millions of people (Benedictow, 2004; Klein, 1902).

Although the Black Death is sometimes used to describe the bubonic plague disease, the term refers to a specific pandemic outbreak. However, in modernity, there have been discussions concerning the name of the pandemic. Recent investigations have started to use the expression "Black Death" to refer to a different and more lasting pandemic, the death of African, Asian, and Latin American individuals as a product of systemic racism, police violence, segregation policies, and institutional oppression (Canham, 2021; Driessen, 2005). This thesis uses the term Black Death when necessary to refer to the medieval pandemic. However, when possible, it is replaced by "plague" to avoid confusion and acknowledge the new, modern meaning of the expression Black Death.

There have been multiple studies about the impact of its spread. Although traditionally it was considered that the plague had started a period of crisis in England, further studies indicate that the outbreak was part of a larger crisis period which started in the 12th century (Dyer, 1994, 2002). This crisis was characterised by multiple social and environmental changes including retraction of settlement patterns from marginal lands, a decline in land productivity, more difficulties in accessing resources, as well as changes in legal regulations and relations between lords and peasants (Birrell, 2010; Briggs, 2010; Dyer, 2002; Masschaele, 2010). This culminated in several catastrophic events like the Great Famine (AD 1315-1317), the Great Bovine pestilence (AD 1319-1320) and the Black Death (DeWitte and Kowaleski, 2017).

As living conditions and diet deteriorated, it is likely that the population of England would have been more susceptible to pathologies in the period leading up to the Black Death (DeWitte and Wood, 2008). On the other hand, the period after the Black Death was likely very different. The high mortality and previous social and economic changes caused by escalating tensions between lords and peasants (Hatcher, 1977; Miller and Hatcher, 1978) led to a redistribution of wealth (Hatcher, 1977). High mortality allowed for population pressures to decrease, and brought more opportunities for resources and work for lower classes, which resulted in an improvement in their health and diet (Davis, 2012; Müller, 2010). However, the social, cultural and biological shock caused by the Black Death could have also had a negative impact on the subsequent health of its survivors and following generations (Dyer, 2002; Gowland, 2015).

1.1.1 Previous studies on the impact of the Black Death

Social historians have addressed the impact of the Black Death using multiple lines of evidence. Benedictow (2004, 2012) explored demographic changes, population dispersion through Europe, and the origin and mortality patterns of the Black Death using poll tax records, the death and replacement of priests, as well as requests for wills. Mengel (2011) mapped the geographical distribution of the outbreak. Other researchers have concentrated on the impact of the pandemic on specific regional areas or religious groups. Colet et al. (2016); Cohn Jr (2007) analysed documentary evidence of the escalation of violence against the Jewish population during the Black Death within the context of antisemitism in medieval Europe. Birrell (2010); Briggs (2010) reviewed court documents to evaluate changes in internal disputes in relation to demesne managers in small communities in England. Archaeologists such as Lewis (2016*a*) explored the impact of the Black Death in Europe by comparing relative frequencies of high medieval (early 12th-early 14th century) and late medieval (late 14th-late 16th century) pottery to estimate demographic changes after the Black Death. Lewis observed that the pottery-using population was 45% lower after the Black Death hinting at a decline in the population. Orton et al. (2014) used evidence for a decline of codfish skeletal remains during the 14th century as an indication that imports into London became difficult, a situation that only began to improve in the 15th century. Christakos et al. (2007) utilised multiple sources to create spatiotemporal stochastic models to map and compare epidemics like the Black Death and the Indian Plague. Other studies of the Black Death by Gowland and Chamberlain (2005); Margerison and Knüsel (2002) compare mortality patterns between plague and attritional sites. The former study analyses mortality profiles using Bayesian ageing methods to compare the previously mentioned Royal Mint site in London and the Blackgate site in Newcastle upon Tyne. Similarly, the latter compares the Royal Mint site and St. Helen on The Walls in York. The main objective of these studies is to use paleodemographic data to identify catastrophic assemblages, in this case, related to the Black Death, in situations where historical information is missing or incomplete. In more recent years, there has been an increase in biomolecular research into the pathogen and its evolution. Haensch et al. (2010) identified *Yersinia pestis* ancient DNA (aDNA) on skeletal evidence from plague pits in England, France, Germany, Italy, and the Netherlands to relate the bacterium to the Black Death outbreak. Similarly, Cesana et al. (2017) used a combination of bioarchaeological and molecular analysis to identify the first victims of the plague in medieval Italy. Raoult et al. (2000) uses "suicide PCR" protocols to identify the aDNA of the bacterium in teeth samples of non-adults found in a medieval grave from southern France. These studies aim to specifically identify the bacterium *Yersinia pestis* in skeletal remains to diagnose if it was related to the Black Death pandemic. The investigations also contribute to sequencing the genome of the disease around the world through history (Keller et al., 2019; Morelli et al., 2010; Spyrou et al., 2019). The project "After the Plague: Health and History in Medieval Cambridge" (2016-2020) from Cambridge University focused specifically on the analysis of aDNA to identify the *Yersinia pestis* genome related to the first pandemic (the Justinian Plague) and its relation to the Black Death (Keller et al., 2019). More direct evidence for the impact of the Black Death on human health comes from the analysis of skeletal remains, mostly from the large, well-dated cemeteries of London (DeWitte, 2012, 2014, 2017; DeWitte and Lewis, 2021; DeWitte and Wood, 2008; Kendall et al., 2013; Yaussy et al., 2016; Yaussy and DeWitte, 2018). More recently, scholars have begun to focus on areas outside London, for example, its impact on Cambridge (Giberti, 2019), but again the project only focused on a single region. There are multiple reasons why London is favoured for analysis of the Black Death in England. Firstly, the medieval post-Black Death period is very short (AD 1350-1550), lasting from the end of the Black Death to the end of the medieval period. Very few sites outside London have dating which allows the identification of this period. In many cases, these samples are mostly composed of post-medieval populations. London has many sites with more detailed stratigraphy which allows the identification of post-plague groups. Due to the large mortality caused by the plague, London also has sites like East Smithfield which were exclusively used during the Black Death period. Sites like this are not found outside London. Furthermore, sites like Spital Square (previously known as St Mary Spital) are dated with smaller subperiods, which allows the evaluation of multiple subperiods within the pre-Black Death period (DeWitte, 2014). Comparably, the Cambridge project centred on a large population recently found in the Cambridge area for which a similarly detailed stratigraphy was applicable. This allowed the comparison of multiple subperiods within the pre-Black Death period, as well as the identification of a post-plague group. However, these sites do not represent life in all of England. London was a highly urbanised area, unlike any other urban site in the country. It had a much higher population density, more migration, and a different population composition and working life than other regions in England (Rigby, 2010). This had an impact on the health of its inhabitants, both before, during, and after the plague (Lewis, 2016*b*). Although there are studies of areas outside London, like the Cambridge University project (Giberti, 2019) and analyses of plague victims at a mass graveyard in Thornton Abbey, Lincolnshire (Willmott et al., 2020), they concentrate on specific areas. There is no compilation of health status outside London before the plague. This investigation centres on the analysis of childhood and pubertal health. This is because the stress experienced during formative years, especially the first 1000 days of life, has an impact on subsequent adolescent and adult health (Barker, 2012; Gowland, 2015; Uauy et al., 2011). For this reason, childhood health status is a good indicator for analysing population health (Lewis, 2018).

1.2 Aims and objectives

This research aimed to evaluate whether the health of the English population was in decline prior to the outbreak of the pandemic of the 14th century by measuring childhood health in adults and children dating to before and after the Black Death. Given that the majority of studies have been undertaken on individuals living within the capital, a secondary aim was to assess whether circumstances were similar for people living outside the London region. For that purpose, individuals from multiple sites across England dated to the pre-Black Death (AD 900-1348) and the post-Black Death periods (AD 1350-1550) were analysed and compared. To further assess if health was declining throughout the period previous to the plague, the pre-Black Death period was divided into two subperiods: the early pre-Black

Death (AD 900-1100), and the late pre-Black Death (AD 1110-1348). This division was adapted from criteria used by DeWitte (2014); DeWitte and Lewis (2021) and based on available dating from each skeletal collection. The final analysis was done by integrating primary and secondary osteological analyses and archaeological and contextual evidence to create a more comprehensive image of childhood health in the past.

The main objectives of this thesis were to:

1. Compare evidence for physiological stress, malnutrition and growth disruption experienced during childhood in non-surviving non-adults and surviving adults aged under 35 years using primary and secondary data.
2. Evaluate differences between sexes and their relation to childhood stress and nutrition, emphasising differences between the sexes during developmental years.
3. Compare prevalence rates between groups from London and extra-London cemeteries to identify how the crisis period affected health in different living environments.
4. Contrast results from osteological analyses against historical conceptions of health and diet during the medieval period.
5. Integrate primary and secondary osteological analyses, and archaeological and contextual evidence to create a more comprehensive image of childhood health in the past.

1.3 Structure of the thesis

Chapter 1 is this introduction, which summarises the thesis.

Chapter 2 describes the archaeological and historical context of the Black Death period. This involved the crisis period leading to the Black Death, including social, economic, demographic, and cultural changes, as well as previous catastrophic events previous to the plague. The period of the Black Death itself is also described, explaining how the disease spread and its likely mortality based on recorded evidence. The post-Black Death period is then described, explaining the impact the Black Death had on the economy, migration, and health, among other things. Special attention has been placed on childhood and pubertal development as well as obstetric health. Previous studies covering medieval childhood and adolescent health, catastrophic events, and the Black Death are described to identify areas that have not been explored, on which this research will focus.

In chapter 3, childhood health and the adolescent growth spurt are explained, describing the relationship between a child and its natural and cultural environment, the impact of childhood stress on adolescent development and puberty, and adult health and its relation to obstetric health. Finally, the conditions, pathologies, and indicators analysed, as well as their relevance in this research, are also explained.

The first section of chapter 4 describes the material analysed in this thesis. This includes a description of the sites where the individuals analysed in this investigation were buried, and their location, archaeological context, and period. For secondary evidence, the data sources are cited.

The second section of chapter 4 describes the methods used in this investigation to evaluate childhood health and pubertal development. The indicators, conditions, and pathologies follow the ones described in chapter 3.

In chapter 5, results from the investigation are presented. The analysis follows the conditions described in chapters 3 and 4. This includes descriptions of the data, comparisons between pre-, post-, and Black Death periods, early and late pre-Black Death subperiods, extra-London and London regions, as well as female-male differences. All analyses are presented with statistical analyses when possible.

Chapter 6 discusses the results from chapter 5 focusing on the differences between periods, regions, and sexes. Results are contextualised utilising archaeological and historical evidence to describe childhood health and pubertal development changes concerning the Black Death.

Finally, chapter 7 summarises the results of this investigation and their interpretation. It briefly describes the limitations of the analysis and the measures taken to reduce them. It continues with the conclusions of the thesis and the lines of research that could be further explored.

2 The Black Death and the medieval period

2.1 Introduction

Children are among the most sensitive to environmental stressors. The immune system of children is not fully developed until the age of 7-8 years (Kloc et al., 2020). This makes them more vulnerable to new pathogens (Lewis, 2018). As the bones of children are still forming, stress can affect their development, leaving identifiable marks on the body. In some cases, such as the teeth, which do not remodel (Hillson, 2005), indicators of stress experienced during childhood can also be observable in adult skeletons (Lewis, 2007). This makes the study of childhood health fundamental to the understanding of population health (Aufderheide et al., 1998). Childhood nutrition and health status also have a significant influence on health in later adolescence and adulthood (Lewis, 2018). In the past decade, the Developmental Origins of Health and Disease (DOHaD) hypothesis has increasingly pointed to the significance of the initial 1000 days of life for subsequent health, including vulnerability to diseases and other environmental stressors in adulthood. Furthermore, it has signalled the health and nutrition of the mother as determining the long-term health of her infant, especially during pregnancy, breastfeeding and weaning periods (Barker et al., 2011; Gowland, 2015; Gluckman and Hanson, 2006; Uauy et al., 2011). To fully evaluate metabolic-nutritional stress experienced during childhood, it is fundamental not only to analyse skeletal markers of physical stress but also to examine the results in the context of the medieval period before and after the Black Death. In particular, to consider how social and economic changes may have affected maternal and infant diets, stress experienced by the mother, food availability, sanitation, potential pathologies the child may be exposed to, and cultural practices related to child-rearing (Gowland, 2015).

The medieval period (AD 410-1550) was a time of numerous natural, cultural, and social changes. Natural changes included multiple temperature fluctuations between warm (the Medieval Climate Anomaly: AD 950-1250, 1350-1450) and cooler sub-periods (The Little Ice Age: AD 1250-1350, and especially from AD 1450 onwards) (Bailey, 2021; Campbell, 2012), which affected the fertility and productivity of the land, as well as agricultural/pastoral capabilities (Campbell, 2012). The period also experienced social changes often interconnected within larger factors. During the 9th century, small scattered farms were replaced by nucleated villages (Hamerow, 2011). During the 10 and 11th centuries. agricultural production intensified, increasing the number of settlements and leading to population growth. This was accompanied by increased urbanisation observed in the creation of towns dependent on rural activity and marketplaces where surplus could be traded (Astill, 2010; Dyer, 1994). These changes led to more circulation of money and paid workers replacing serfs (Hatcher, 1977). Towards the 13th century, overexploitation of the soil and increased population pressure led to soil depletion and retraction of previously expanded settlements and hamlets in marginalised lands (Dyer, 2002). This culminated in a crisis period that peaked during the first half of the 14th century with the Great Famine (AD 1315-1322) (Schofield, 2013) and the Black Death (AD 1348-1350) (Benedictow, 2004). After the Black Death, the great mortality of the plague put the surviving workers in a position where their work was highly required Bailey (2021). This resulted in wages rising and a redistribution of wealth. This led to conflicts between peasants and lords which culminated in the peasant revolt of AD 1381 (Hatcher, 1977). All the numerous changes had an impact on the lifestyle, health, and nutrition of the population.

2.2 Pathogenesis of *Yersinia pestis*

The medieval pandemic of the 14th century, known as the Black Death, or "Great Pestilence", spread from Asia through Europe via established trade routes between AD 1346 and 1353, and was present in England in AD 1348-1350 (Benedictow, 2004). It is now widely accepted that the pandemic was caused by the gram-negative bacterium *Yersinia pestis*, a zoonotic disease (Barbieri et al., 2020). Although this was the most significant outbreak of the disease, it was not the only one. There were smaller instances of the epidemic, including one in AD 1361-1362 (Bailey, 2021) known as pestis secunda or the "second pestilence" (DeWitte and Kowaleski, 2017).

To comprehend its effects on the larger population, it is important to understand the transmission and pathogenesis of the disease. The acute disease that follows infection has three forms: bubonic, pneumonic, and septicaemic plague (Raoult et al., 2013) (Figure 2.1). It also has two main mechanisms for transmission: vector-borne (most common), and airborne (less common) (Benedictow, 2004; Kool and Weinstein, 2005). The risk of mortality of the disease without treatment can be from 80 to 100% depending on its manifestation (Kool and Weinstein, 2005; Robb et al., 2021).

The primary manifestation of the disease at the site of inoculation is bubonic plague. This form is vector-borne. It infects the lymphatic system creating inflammation in the lymph nodes (observed through the formation of a bubo) (Raoult et al., 2013). When the lymphatic system is overwhelmed, the disease infects the bloodstream as septicaemic plague. This is observed through the appearance of small petechial haemorrhages visible on the skin. The bubonic plague is the less virulent form of the disease, taking several days to kill its hosts and presenting a survival rate of 20% without treatment (Benedictow, 2004). The second manifestation (septicaemic plague) is rarer (Nelson et al., 2020) and more virulent, resulting in death as fast as 19 hours after the appearance of the first symptoms (Benedictow, 2004; Kiple, 2003; Manchester, 1992; World Health Organization, 2000). Septisemic plague is often secondary to bubonic (Sebbane et al., 2006), and sometimes pneumonic, plague (Jullien et al., 2021). However, there have been modern cases registered of primary septicaemic plague (Sebbane et al., 2006).

The third manifestation of the disease is pneumonic plague. This form occurs when the infection reaches the lungs. Primary pneumonic plague is caused by contagion through respiratory droplets from another infected individual, while secondary pneumonic plague is caused by untreated bubonic or septicaemic plague infecting the lungs (Benedictow, 2004). Similarly to septicaemic plague, pneumonic plague is more deadly than bubonic plague. It can develop from and devolve into septicaemic plague. Incubation of the disease takes approximately one day, and death can occur between one and three days after symptoms appear (Kool and Weinstein, 2005). If left untreated, this form of the disease is nearly 100% deadly (Benedictow, 2004).

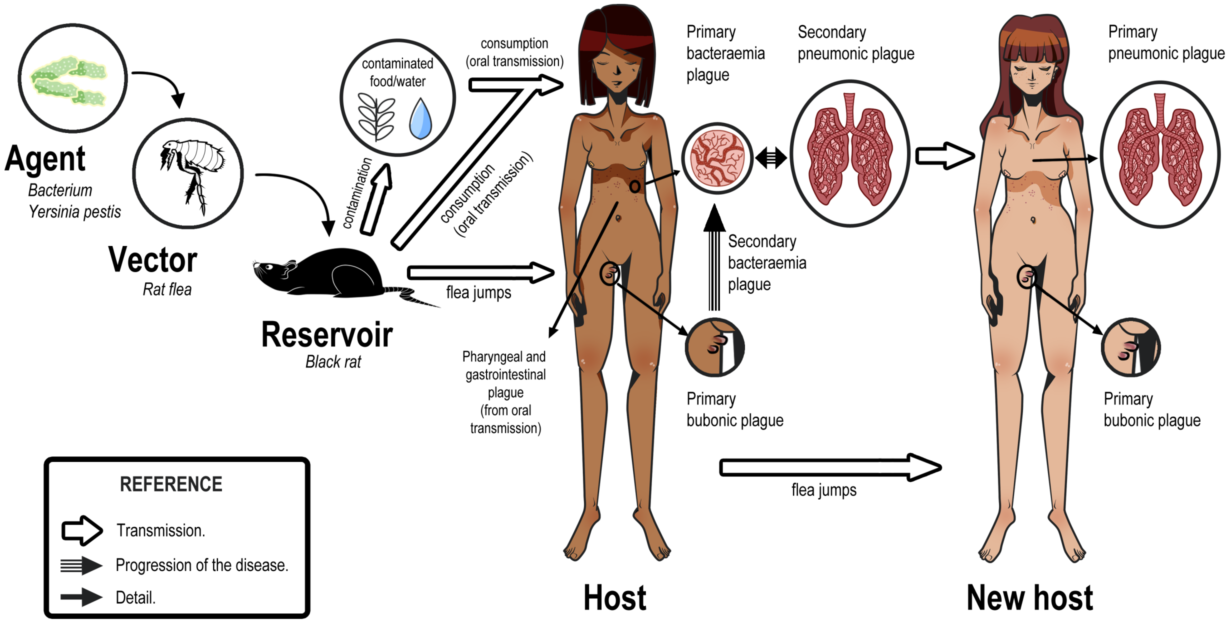
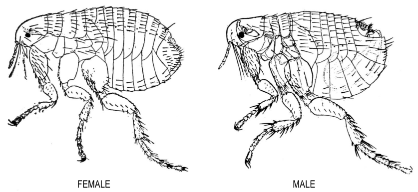


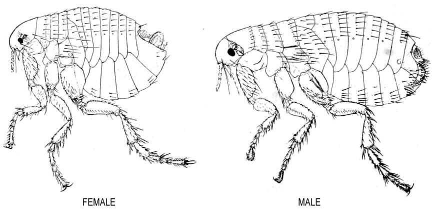
Figure 2.1: Plague transmission based on descriptions by Benedictow (2004) and Barbieri et al. (2020)

The distribution of most epidemic diseases is not uniform across different contexts but depends on many factors that may increase or diminish the possibilities of contact. In many cases such as tuberculosis or other airborne diseases, the probabilities of contagion increase in more densely populated areas (de Abreu e Silva et al., 2016). This relation can also be observed with oral-faecal transmitted diseases where higher density may result in poorer sanitation (Stinson, 2012). However, in the case of the Black Death, the situation was different as it had multiple ways of spreading which altered the impact of the disease on different populations.

There are two main forms of plague transmission; by a vector (vector-borne) or by respiratory (airborne) droplets. The first is the form of transmission of the bubonic plague. It involves three factors: the agent, the vector, and the reservoir. The agent is the bacterium *Yersinia pestis*, the vector is the flea and the reservoir is the rat (Raoult et al., 2013). The vector that carries the bubonic plague is the rat flea (*Xenopsylla cheopis*). More recently, investigations have also highlighted that the human flea (*Pulex irritans*) (Figure 2.2) could also have acted as a vector for the disease (Ratovonjato et al., 2014). Laboratory experiments have shown that the *Pulex irritans* was less effective as a vector than the *Xenopsylla cheopis*, especially after being fed human bacteremic blood (Miarinjara et al., 2021).



(a.) Xenopsylla cheopis

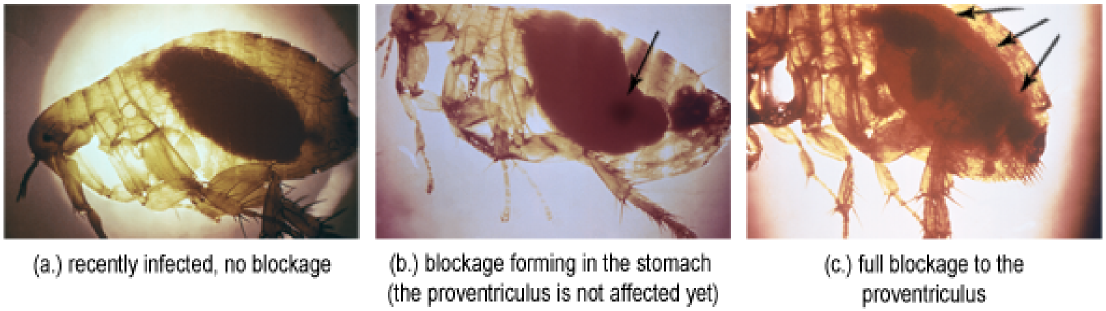


(b.) Pulex irritans

Figure 2.2: *Diagrams of rat fleas Xenopsylla cheopis and human fleas (Pulex irritans) (Herms, 1915). Public Domain Mark.*

There is a broad spectrum of animals that can act as bubonic plague reservoirs. These extend from the most commonly known, like black and brown rats, to other rodents and even larger animals like camels and pigs (Barbieri et al., 2020). In medieval England, the most common reservoir for the bubonic plague was the black rat (*Rattus rattus*). The brown rat (*rattus norvegicus*) was introduced later during the 18th century (Guiry and Gaulton, 2016). The black rat is not native to the UK but its presence has been recorded in several archaeological sites across England since the Roman period, declining towards the 4th to 7th century and increasing again from the 8th century (See figure B.1). This suggests that the animal may have been reintroduced to England through Viking trading ships. Its presence in London increased towards the 13th and 14th centuries, likely due to deficient living conditions which involved poor waste management and close proximity to domestic animals (Rielly, 2010). The frequency of rat remains in England can also be correlated to the first and second pandemics of plague (Justinian plague and Black Death) (Orton et al., 2023).

Similarly to other vector-borne diseases such as Chagas, the bite from a rat flea alone is not enough for transmission to occur (Centers for Disease Control and Prevention, 2017). For transmission to occur, bacteria must concentrate and cluster to cause a digestive blockage in the gut of the flea (Benedictow, 2004; Yue et al., 2017). Transmission occurs only when a flea with a blocked gut bites a new host (in this case either a black rat or human) and regurgitates the blockade into the initial bite wound (Eisen and Gage, 2009) (Figure 2.3). The rat then dies and the flea searches for a new host. This increases the number of fleas in the remaining rats and affects humans who become new hosts for the flea. In medieval England, overcrowding and the build-up of food waste attracted the black rat, and its fleas, to domestic areas allowing the disease to spread rapidly (Benedictow, 2004).


(a.) recently infected, no blockage

(b.) blockage forming in the stomach (the proventriculus is not affected yet)

(c.) full blockage to the proventriculus

Figure 2.3: *Series of photos showing the progression of the disease affecting a rat flea (Xenopsylla cheopis) infected with yersinia pestis modified from Public Health Image Library, Center for Disease Control (Images nº 14283-14285) (Public domain).*

The other form of transmission is through respiratory droplets. This form involves transmission primarily from human to human, results in pneumonic plague, and is less effective than when the disease is transmitted by the vector. The reason is that the size of the bacteria requires large droplets, which results in extremely close contact being required for transmission (Butler, 2009). This has been observed in more modern plague epidemics. One example is the Manchurian epidemics in China, which hit the region in two waves (1910-1911 and 1920-1921) and affected marmot hunters. During these epidemics, it was observed that pneumonic plague was not highly contagious among people (Kool and Weinstein, 2005). This was also registered in Uganda, where the disease is endemic (Kugeler et al., 2017). Differently from other respiratory diseases responsible for pandemics like COVID-19, where individuals can become contagious while asymptomatic (Gallo et al., 2022), individuals with pneumonic plague are not contagious during the first 24 hrs. Only when they become symptomatic do they turn contagious and only significantly in the last stage of the disease. From the Manchurian experience, researchers observed that for contagion to occur, close contact was required. Simple mask-wearing and sometimes even just turning the head to avoid coughing on another person could aid in preventing spread (Kool and Weinstein, 2005). A similar phenomenon was observed during the Indian epidemic, which hit the country in 1896 and lasted until approximately 1914 (Evans, 2018), where mortality was proportionally higher in areas with less population density. This was the result of the main transmission being through a vector and the risk of contagion being more related to the number of rodents than the number of humans (Benedictow, 2004). Benedictow (2004) proposes that this could be applied to England. They hypothesise that in England, although black rat colonies were attracted to places with food, and were used to human presence, the ratio between rats and humans was variable. In scarcely populated rural areas, a rat colony likely lived within a household; however, in urban areas, it was probable that the same rat colony lived in the proximity of several households. When considering this factor, and the low transmissibility of pneumonic plague, it resulted in a higher chance of individuals in rural areas being bitten by fleas from depleted rat colonies than people who lived in cities. In this case, the higher density not only would not increase the chances of contracting the disease but diminished it (Benedictow, 2004). This does not mean that the disease was not airborne or that the pneumonic plague played no part in transmission. The existence of the East Smithfield cemetery shows the high mortality of the disease in London. There is evidence that non-ventilated areas increased the risk of transmission (Kool and Weinstein, 2005). However, the potential disparity in transmission effectiveness must be considered when analysing the plague in England, particularly when comparing the effects of the plague between London and areas outside London with less population density (Benedictow, 2004). Although large urban centres likely had a higher total number of deaths caused by the plague, small rural areas were possibly more vulnerable to proportionally higher mortality resulting in the area being completely devastated.

Although the traditional vector and airborne types are the most studied forms of transmission of the disease, it is important to mention that other variants must also be considered. In modern outbreaks of the bubonic plague, oral transmission of the plague caused by consumption of undercooked/raw meat/liver of a diseased animal led to less frequent manifestations of the disease like pharyngeal and gastrointestinal plague. This variation of the plague has been found to be 100% deadly if not treated (Barbieri et al., 2020). Although it is unlikely that there was consumption of black rats during the medieval period in England, there is evidence of consumption of unusual foodstuffs such as horses, dogs, cats and mice during the famine in other European countries, such as during the 16th-century famine in Leiden, the Netherlands (Pollman, 2022). Furthermore, a more likely form of contracting the disease was through the contamination of food or water sources with infected rat urine (Barbieri et al., 2020). This is a possible situation in England, particularly in places with poor sanitation. More recently, studies like Jullien et al. (2021) proposed that plague could also be transmitted by coming into contact with bodily fluids or infected corpses or carcasses. A similar situation has been observed in modern cases of plague, where individuals contracted the disease through aerosol released during the skinning of hunted infected animals (World Health Organization, 2008). This has important implications as, excepting mass graves like the East Smithfield cemetery in London, most plague victims were subjected to similar funerary rites as non-plague individuals (Kacki et al., 2011). it is important to mention that the cycle of the plague does not always appear as an epidemic (World Health Organization, 2010). The disease spreads from an enzootic to an epizootic cycle when it spreads through animals (Goldberg et al., 2022), to then pass to an endemic and epidemic phase among humans. Historic and epidemiological approaches to the understanding of the disease have used this information to estimate the progress of the epidemic and locate its origin, as well as in more modern settings limit its dispersion (Benedictow, 2004; Andrianaivoarimanana et al., 2019). In this thesis, these studies are used to estimate the impact of the disease in various contexts.

2.2.1 Mortality

It is difficult to estimate plague mortality. The first estimates for the Black Death were made using historical records. This created a bias towards localities with better-kept administrative records (Izdebski et al., 2022). These records were in many cases administrative documents related to tax collection and therefore were biased towards males over the age of 12 years (Benedictow, 2004; Izdebski et al., 2022). Various authors have estimated mortality rates based on multiple sources of evidence. Benedictow (2004) used historical records such as the demographic system of the late medieval period and information obtained from chronicles from manorial courts, bishops, monastic and court records (Benedictow, 2004). These records were relevant as they described the reaction the population had to the Black Death. Priests were, in some cases, exposed to the disease by their work administering the last funerary rites. Furthermore, records such as tax and rent collection as well as requests for creating and changing wills also indirectly show population mortality. Benedictow (2004) has concluded that mortality was between 30% and 60% in England. Archaeological approaches such as Lewis (2016*a*) focused on pottery, recording a decline of 45% of the pottery-using population in Eastern England after the Black Death. DeWitte and Kowaleski (2017) combined historical and bioarchaeological data to estimate mortality in England. They estimated mortality between 40% and 60%, with an estimation of 50% mortality among adult males in the countryside. This novel approach is significant as it not only estimates mortality but also delves into how the plague affected different sectors of the population. Although the Black Death caused unprecedented mortality in England, and Europe in general, analysing its true impact on the population is difficult. Furthermore, its effects cannot be assessed in a vacuum, but as part of a larger process which facilitated the spread of the disease and intensified its influence. Izdebski et al. (2022) proposed to analyse mortality using "big paleoecology". This approach used palynological data to estimate field cultivation as a proxy to infer survival in rural places. They estimated that mortality around Europe was highly variable. Regretfully, their analysis did not take any samples from England, only looking at Wales and Ireland. Many of the social and economic changes observed after the plague had started before its outbreak. To understand this context, historians such as Postan (1899-1981) created a general model which links the process of economic expansion with subsequent retraction and catastrophic episodes (Dyer, 2002). Bailey (2021) and Izdebski et al. (2022) show evidence of lower temperatures and higher rainfall affecting Europe and specifically England before the plague. This likely had a negative effect on crops. For this reason, it is important to contextualise the plague within the medieval period to fully understand how it developed, and how its aftermath affected the population (Dyer, 2002).

2.3 The medieval period before the Black Death (AD 900-1348)

2.3.1 Socio-cultural context: expansion and crisis

Throughout the medieval period, multiple social, demographic and economic changes affected the lives of its inhabitants. These changes were not homogeneous and had different ramifications according to the region and specific context. Furthermore, changes took multiple directions, affecting different sections of the population (e.g., lords and peasants) differently. Nevertheless, in general terms, there was a process of economic growth and expansion from AD 1100 to AD 1290, followed by a period of retraction and crisis (AD 1290-1350) (Dyer, 2002).

The transition between the 8th and 14th centuries included a multiplicity of changes, which included the Norman Conquest (AD 1066) and its impact on the social, economic, and political landscape of Britain, as well as a transition from a majority rural population to an increasingly urban one (Dyer, 1994). Towards the 8th century, the settlement pattern suffered a change. Small and scattered settlements were replaced with nucleated villages (Dyer, 2002; Hamerow, 2011). Subsequently, during the 9th century, the "field system" of primarily agricultural production by peasants in seigneurial lands started to form. During this time land exploitation was preserved within a careful balance. In this context, crops were cultivated according to the possibilities of the land, complementing agricultural usage with pastoral activities to maintain soil fertility. Special attention was placed on maintaining an ecological balance. However, towards the 10th and 11th centuries, the field system was expanded and intensified (Dyer, 1994). This expansion was observed in an increase in settlements as well as an expansion of arable lands in woodlands previously used for pastoral activities. This was reflected in tax records which show that some manors had occupied land normally used for sheep farming with crops (Dyer, 2002). The intensification of production generated population growth that instigated the fragmentation and creation of hamlets and new settlements in marginal lands. This continued during the 12th century. An increase in production led to the creation of marketplaces in villages for the exchange of surplus and other goods during the 12th and 13th century (Astill, 2010; Dyer, 1994), and the expansion of urban centres. This increased the circulation and value of money. Social relations between Lords and peasants changed. From an economic perspective, many servants became paid workers, who additionally had to pay rent to the lord in cash rather than in production. This made them more vulnerable to the effects of inflation and changes in the market. Concerning prices, agricultural commodities had high values, and the high offer of labour made wages low. In some places like Corfe Castle in Dorset, work accounts at the end of the 13th century describe an increase in demand for labourers for the harvest period and difficulties procuring them, which led to a temporary increase in wages for male workers. There are also accounts for the same period detailing that the higher demand for labourers led to hiring more women and children in places like Rockingham, Northamptonshire, and Woodstock, Oxfordshire. Although these accounts show periods of wage increase, the overall trend before the Black Death was of sustained low wages (Langdon, 2010). From a social perspective, although during this period there was economic and commercial expansion, the field system and relationship between peasants and lords resulted in a very unequal distribution of wealth (Hatcher, 1977). Additionally, during this time, the regulation of production by demesnes became stricter. The field system was maintained by a regulatory system where managers like Reeves and bailiffs acted as intermediaries between the lord and peasants (Briggs, 2010). This was also further regulated by a system of courts which operated at different levels and ensured law and order. This system allowed the control of the lands by the lord, as well as the participation of the peasants in its management. Additionally, as the courts were located in towns, this participation also allowed the further development of markets surrounding the courts (Briggs, 2010; Masschaele, 2010). The dominance of the landlord's power was further strengthened with regulatory reforms that employed debt collectors (e.g., reeves and bailiffs) to act as intermediaries between the lord and peasants (Briggs, 2010; Masschaele, 2010). Others have argued that changes in employment rules improved the lives of the peasants who had greater autonomy reflected in disputes between the peasants and landlords, increased wages and the acquisition of small parcels of land by some peasants (Dyer, 1994, 2002; Tompkins, 2010).

The period of economic and commercial expansion came to an end in the second half of the 13th century, beginning a period of stagnation and crisis brought about by environmental, economic and social changes (Campbell, 2012; Dyer, 1994). Firstly, the intensification of farming resulted in over-exploitation of the land and soil depletion. This can be observed in estate surveys which mentioned soil depletion and some accounts from manorial officials which described a decrease in agricultural yield. This eventually led to the eventual abandonment of marginal hamlets (i.e., deserted medieval villages) and the retraction of well-established settlements. An example of this retreat can be observed in the abandonment of hamlets and farmsteads on Dartmoor, Devon and Snowdonia, northwestern Wales (Dyer, 2002). It has been argued that reduced food production, in addition to harsh labour laws and overcrowding, left the lower-status medieval population vulnerable during the Black Death outbreak (Langdon, 2010). Diet was not only influenced by the availability of food sources, but also by religious and social norms that dictated which foods were consumed by whom, when, and which foods were to be avoided (Ashby, 2002).

One of the most significant variables affecting diet during the medieval period was socioeconomic class. Diet during the medieval period was mostly composed of grains, lentils, peas, and beans. Higher classes had more access to meat, which was less accessible to lower classes who depended on living cattle for agricultural production (Müller, 2010). However, this does not mean that meat was absent from the peasant diet. Stable isotopes from poor rural sites like Wharram Percy, Yorkshire indicated that a mixed diet with a component of animal and plant protein (Richards et al., 2002). Consumption of fish also varied amongst the population based on multiple elements. Similarly to meat, fish was mostly consumed by higher classes (Burt, 2013). Furthermore, location also influences its consumption. Stable isotope analysis of individuals from the coastal site of Stoke Quay, Ipswich presented evidence of marine consumption (Farber et al., 2017; Farber, 2020) while studies from other poor rural populations

Wharram Percy exhibited little dietary input from marine sources (Richards et al., 2002).

However, marine consumption was not the same throughout the medieval period. This change is referred to as the "Fish Event Horizon", which is described as a change in fishing practices from freshwater species to marine fishes like cod and herring. This is observed in multiple lines of evidence. Zooarchaeological evidence from sites like Blue Bridge Lane and Fishergate York in Yorkshire, and Stoke Quay in Ipswich show high frequencies of freshwater such as eel dated ton approximately between the 7th and early 9th century (Farber, 2020). Between the late 10th and late 11th century there is evidence of an increase in cod and herring production observed in multiple sites in York, London, Northampton, Southampton, and Norwich (Barrett et al., 2004; Orton et al., 2014). Cod was treated for short preservation through salting and drying, while herring was brined for long-term preservation (Barrett and Orton, 2016; Harland et al., 2016). This does not mean that other fishes were not exploited. After the 12th century, freshwater fish like pike, cyprinids, and perch, may have been produced from ponds belonging to higher classes (Harland et al., 2016). Barrett et al. (2004) describes two sites: Bow Street, Langport, Somerset dated to the 13-14th century and Stert Street, Abingdon, Oxfordshire dated to the 15-16th century where high frequencies of eel were found. These sites acted likely as speciality stores and were not common. This indicates that marine consumption increased through the medieval period.

2.3.2 The Great Famine

In AD 1314, the English population experienced a period of low temperatures and high precipitation causing crop failures and diseases in cattle, events referred to as the Great Famine (AD 1315-1322) (Schofield, 2013) and the Great Bovine Pestilence (AD 1319-1320) (Slavin, 2012). The loss of cattle not only affected access to fresh meat and dairy but also hindered the ploughing of fields and the transportation of grain (Slavin, 2012). This crisis affected both the lords and peasants, although to different extents. Peasants had a reduced capacity to produce crops, while landlords saw diminished returns on their property. In response to the crisis, several manors employed fewer people and raised the price of grain. These changes affected peasants the most, through both lost income and the rising price of their dietary staples (Dyer, 2002).

The effects of famine on mortality are indirect. People rarely die of starvation but are more susceptible to disease as the result of malnutrition and immunosuppression (Geber, 2014). In England, the limited access to foodstuff, particularly high sources of protein like meat and dairy had a negative impact on population health (Jordan, 2010; Dyer, 2002). The Great Famine did not only affect the health of peasants but also had an impact on the marketplace and economy. The decrease in production impacted trade. Lower classes, particularly in urban areas, resorted to selling household items to purchase food. Higher classes resorted to leasing part of their property and reducing the number of servant under their employment resulting in further difficulties for lower classes Jordan (2010). Although these difficulties did not start with the Great Famine, they were worsened by the famine and would be further exacerbated by the Black Death (Dyer, 2002; DeWitte, 2017).

2.4 The Black Death (AD 1348-1350) and the period after (AD 1350-1550)

The high mortality caused by the Black Death had short and long-lasting consequences beyond the immediate loss of life. The 14th century was characterised by a combination of serfdom work and paid labour (Dyer, 2002; Bailey, 2021). Due to the high mortality of the Black Death, the number of available workers was reduced. This created struggles in finding workers, which led to higher wages. Many rural workers chose to migrate in search of work, which resulted in a further loss for the region (Bailey, 2021). The high demand to secure workers also led to the incorporation of more women into the workforce (Campbell, 2012; Lewis, 2016*b*). The loss of peasants led to a drop in revenue for lords from rent as tenants died, reducing their income. These struggles led to the creation of measurements to ensure workers remained in the rural areas. The most important was the AD 1349 "Ordinance of Labourers", reinforced in AD 1351 by the "Statute of Labourers" which attempted to control the market by limiting wages and demanding compulsory work from people deemed "idle" (Bailey, 2021; Bennett, 2010). This work applied to both serfs and free workers (Bennett, 2010). Later ordinances would also seek to restrict migration, demanding permits for rural workers to move to prevent losing more workers. Although rural areas are normally less documented than urban centres (Izdebski et al., 2022), the documentary evidence of these restrictions shows more evidence for rural areas than for urban centres. Bailey (2021) argues that in the immediate aftermath of the Black Death, the need for workers was so great that many of these restrictions were often ignored in practice in favour of retaining workers. However, Bennett (2010) mentions that these restrictions were imposed but were not always properly recorded, particularly for women who may have wanted to avoid formal procedures. Increased attempts to control peasants and workers led to rising resentment among workers, which culminated in the peasant revolt of AD 1381 (Bennett, 2010; Hatcher, 1977).

The low number of workers also affected the market. Bailey (2021) mentions that in the immediate aftermath of the Black Death, there was an increase in inflation due to increases in cost and difficulties in the supply of fuel, salt, and grain. This lasted until AD 1353 when a better harvest aided in controlling the immediate crisis. Changes in agricultural activities were accompanied by changes in the market and social regulations, which led to the increased usage of towns as centres for commerce and social exchange which further attracted migrant rural workers (Lewis, 2016*b*; Masschaele, 2010). One of the most significant changes observed after the Black Death is stagnation in population growth, due both to high mortality and reduced fertility (Benedictow, 2004; Hatcher, 1977). Demographic models show that the age of first marriage during the period was between 24 and 26.5 years (Benedictow, 2012) which also contributed to population stagnation (Hatcher, 1977). The drop in fertility is a topic of much debate by social historians, as it goes against what is expected after such a population crisis (Benedictow, 2012; Campbell, 2012; Dyer, 1994, 2002; Hatcher, 1977). For example, the aftermath of the Second World War saw a "baby boom" (Phillipson, 2007). One possible explanation for this stagnation is related to the changing role of women in society. Authors like Goldberg (1986) state that after the Black Death, many women migrated from rural areas to urban towns to work as servants. This provided them with better resources and allowed them to delay marriage until possibly age 25 years (Goldberg, 1986). However, these examples only apply to urban York and decreased at the beginning of the 15th century. Bennett (2010) stated that in rural areas women were frequently in more vulnerable positions, even after the Black Death. The author explains that women, especially single women, were often excluded from records and more vulnerable to being forced into compulsory labour. This may indicate an incentive to marry younger. However, the evidence does not indicate that was the case. There is evidence that social pressures often drove women to migrate to urban areas in search of better opportunities (Bennett, 2010; Lewis, 2016*b*). Müller (2010) describes how after the Black Death, the rise in wages led to changes in the food production system and diet. From a production standpoint, agricultural activity became very expensive to maintain. Soil fertility was low, and the price of waged labour was high. Additionally, more wealthy peasants were able to afford more expensive foods such as meat, previously reserved for high-status individuals, increasing the demand for cattle raising and increased pastoralism (Izdebski et al., 2022). Ale became widely produced and consumed as a source of fluids and carbohydrates (Bailey, 2021). The upper classes found new ways to differentiate themselves by adopting the consumption of game and other luxurious items and tightening control over hunting grounds (Lamb et al., 2014; Müller, 2010). It could be argued that as a result of changes in food production that followed the Black Death, all classes experienced a rise in quality of life, which resulted in better health. This was not equal for all groups in society. The Black Death served as an accelerator to previous circumstances, but it did not affect all regions equally (Bailey, 2021). This resulted in differential mortality among regions (Izdebski et al., 2022), social classes, and men and women (DeWitte, 2012, 2017; DeWitte and Lewis, 2021). Similarly, the aftermath of the plague was not equal across England. Bailey (2021) described that areas with a low proportion of smallholders and landless experienced more difficulties reoccupying the land as they could not fill their holdings they had to attract new tenants from outside. This resulted in a slower recuperation. Furthermore, the wealth of the community, areas that had been more affected by poor weather and failing crops before the Black Death had more difficulties recuperating (Bailey, 2021; DeWitte and Kowaleski, 2017).

It is important then to consider not just the overall impact of the Black Death, but how it impacted the health of different sections of society. Health inequality is defined as systematic differences that cause a disparity in the health of people occupying unequal positions in society (Kapilashrami et al., 2015). The poorest in society live shorter lives and suffer more illness than the richest. For this reason, socioeconomic status is a strong predictor of health and life expectancy due to multiple disadvantages, including psychological stress, poor childhood and maternal health, poverty and social exclusion, unemployment, addiction, a poor diet, and lack of exercise (Brunner and Marmot, 2006; Marmot, 2010). Intersectionality theory was proposed by Crenshaw (1989) to explain the complex and interrelated systems of oppression that affect Black women and has become more widely adopted to explain the complexities of health inequality. Intersectionality theory is based on three precepts. Firstly, people and social groups have multiple overlapping identities that are shaped by systems of oppression. Secondly, intersecting identities, for example, ethnic group and gender, can be multiplicative in their effect. This results in one identity enhancing the disadvantage caused by the other. Lastly, social inequalities are caused and shaped by multiple intersecting systems of oppression, for instance, sexism, classicism, and racism (DeWitte and Yaussy, 2020). Intersectionality is fundamental in following movements like third (Evans, 2015) and forth (Soucie et al., 2018) feminism and queer theory (Rahman, 2010). It is important to utilise an intersectional approach to analyse how different groups mediated by class, sex and age were impacted by the plague. In terms of social class, although the high mortality of the Black Death led to the belief that it killed indiscriminately, wealthier classes had lower mortality (DeWitte and Kowaleski, 2017). This may be related to better living conditions that protect them from being exposed to the disease. The wealthier classes also had the means to avoid potential risks. For example, higher-ranking priests could use their power to avoid having to administer the last sacrament to plague victims, sending lower-ranking subordinates instead

(Benedictow, 2004). There is much discussion about the differential impact of the plague on women and men. Paleodemographic analyses of skeletons from medieval cemeteries in London showed a higher vulnerability of males to the plague (DeWitte, 2012; DeWitte and Kowaleski, 2017). This agrees with historical records which highlight higher male mortality (Benedictow, 2004). However, this study focuses solely on London, which does not represent the variability in England (Bailey, 2021). Women were often under-recorded, which could explain their absence from historical documents (Bennett, 2010; Goldberg, 1986). Furthermore, women spent more time in domestic areas, likely in proximity to black rats and their fleas, and were in charge of taking care of the ill (Benedictow, 2004). Close contact with rat corpses who died from the plague and infected victims may have made women more vulnerable to both pneumonic (Kool and Weinstein, 2005; Jullien et al., 2021) and bubonic plague (Benedictow, 2004). Furthermore, mortuary practices during the medieval period required the body to be washed by the women of the household Gilchrist (2012). This close contact with the corpses of plague victims and potentially their bodily fluids would have also made them more vulnerable to contracting pneumonic plague (Jullien et al., 2021). Before the Black Death, women earned lower wages than men, had a harder time finding work, were often not considered in the records, and were punished more severely for any trespass (Bennett, 2010). Women also had limited access to capital and were perceived as needing to be "supervised" by a male relative. This pressure was observed in the bioarchaeological record. DeWitte and Lewis (2021) analyse changes in the age of menarche before and after the plague at the London site of Spital Square (previously known as St Mary Spital). Although the authors cannot estimate the exact age of menarche, estimates of pre- and post-menarche ages showed that women living in the period leading to the Black Death experienced delayed menarche, reflecting metabolic nutritional stress. After the Black Death, the mean age of pre- and post-menarche was reduced again (DeWitte and Lewis, 2021). This shows an improvement in the quality of life of females. However, the improvement in females was not as clear as the one observed in males (DeWitte, 2017). There is evidence that women experienced an improvement in their quality of life. This was observed by more access to positions in urban areas and opportunities for independent development in a variety of areas of labour and particularly in the victualling trade (Goldberg, 1986). However, this was not equal for all women. Bennett (2010) mentions how single women were disproportionately targeted for compulsive service under the "Stature of labourers" in rural areas. This was observed in different regions through regional orders like the Coventry Leet, ordering women under 50 years of age to go into service (Bennett, 2010; Goldberg, 2001). Goldberg (1986) also noted that although many women were able to access work as servants in urban areas, there were still many women who struggled with poverty and had, in many cases, to turn to prostitution. The author also mentions that the initial increase in wages and opportunities decreased significantly by the beginning of the 15th century. The models used by DeWitte and Wood (2008) to estimate the frailty of women and men in relation to the Black Death provide a novel approach to understanding the disease. However, it does not fully consider the implications of women's social disadvantages before the plague. Furthermore, its focus on the London population is not representative. This is particularly relevant in the aftermath of the Black Death where the differential impact of the outbreak resulted in great variability in economic and social changes all across England. DeWitte and Lewis (2021) also propose that the improved health of women in London after the plague was due to migration from rural areas. However, many women migrated from rural to urban areas seeking better life opportunities (Goldberg, 1986), which may imply that, in many cases, they were coming from more precarious social and economic situations. This highlights the importance of studying the health of men and women in areas outside London.

Childhood health during the medieval period is not commonly referred to in historical accounts. Therefore, many historians theorise how the Black Death may have affected child care and infant mortality through the interrogation of parish records and demographic models (Benedictow, 2004; Hatcher, 1977). Childhood health is affected by a multitude of factors including the health of the mother, as well as genetic, epigenetic and congenital factors, which overlap with elements from the child's immediate and general environment, such as diet, family, housing, contact with pathogens and pollutants, medical knowledge, climate, and war (Lewis, 2018) (Figure 2.4). For this reason, children could have been affected by changes in their social environment, and changes in the health, social and economic possibilities of their parents (Benedictow, 2004; Lewis, 2018). It is important to consider all these areas when interpreting the impact of the Black Death on children.

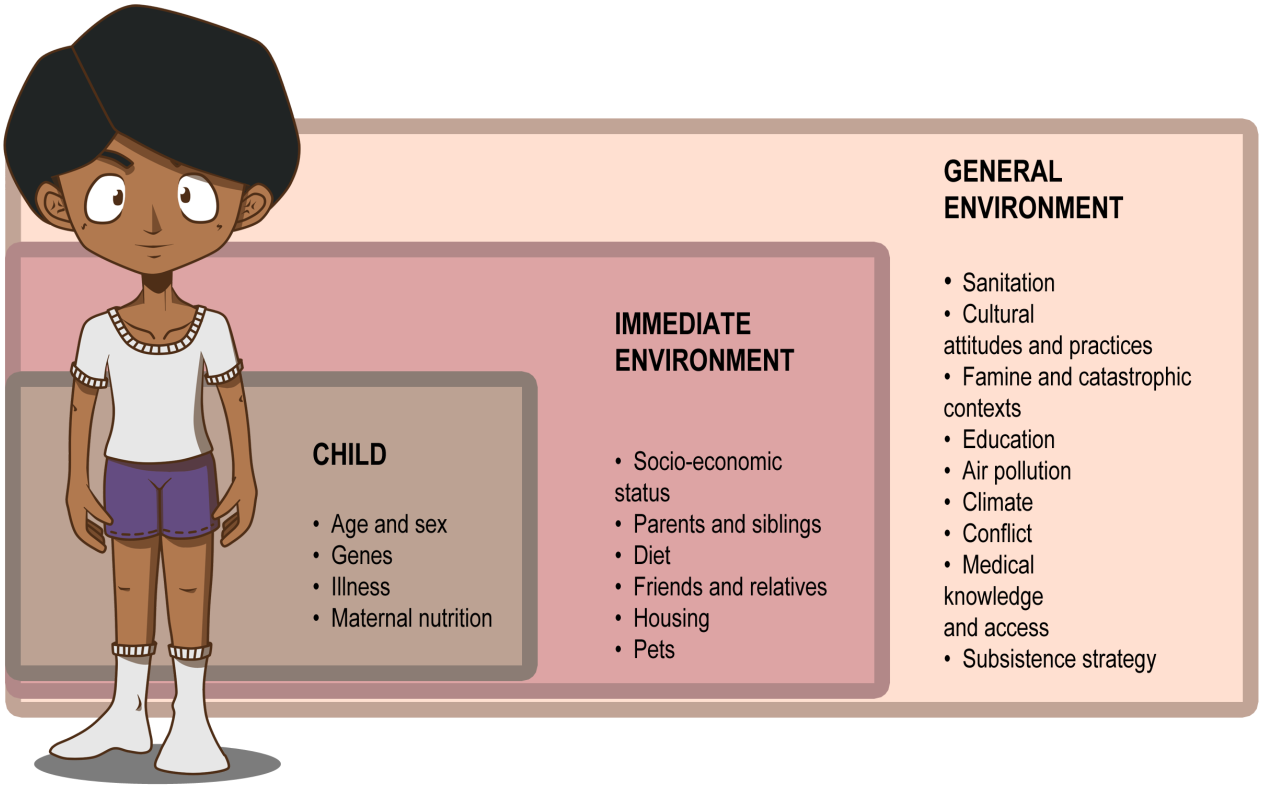


Figure 2.4: *Elements affecting childhood health adapted from Lewis (2018, p. 2)*

During a catastrophe, children are in a particularly vulnerable position (Geber, 2014), as their support system becomes affected. To maintain body growth, children have greater nutritional requirements (Fleischer Michaelsen et al., 2003). They also have developing immune systems which make them more vulnerable to pathogens. This results in higher vulnerability to malnutrition and diseases caused by poor environmental conditions (Gwela et al., 2019; Fleischer Michaelsen et al., 2003). During the first years of life, children are also highly dependent on their parents. Through breastfeeding, they receive vital nutrition and immunity from the person who breastfeeds them (Fleischer Michaelsen et al., 2003). This means that if the parent is immunocompromised or experiences nutritional stress, they will be negatively affected (Dórea, 2000; Fleischer Michaelsen et al., 2003; Pilz et al., 2018; Yang et al., 2013). Similarly, if the parent lacks access to food resources to complement breastfeeding during the weaning period, or lacks proper housing to avoid contamination of the food, the child may experience vitamin, protein, or iron deficiencies (Lewis, 2018). Before the Black Death, stress experienced by pregnant and breastfeeding women would have affected not only their health but also the health of their children. Additionally, during catastrophic situations like the Great Famine and the Black Death, many children may have been abandoned or neglected due to their immediate family dying (Benedictow, 2004). This also extends to psychosocial aspects. Extreme crises like famines deeply affect familial relationships. This was observed in documented experiences during the Irish Famine where parents were seen fighting their own children to obtain food (Geber, 2014).

Wet nurses were women who were employed to breastfeed children other than their own in cases of maternal illness, death or abandonment Fildes (1988); Phillips (2022). Wet nursing was also usually restricted to higher classes hiring people from comparatively lower classes to nurse their children (Phillips, 2022). Wet nurses were carefully selected based on their moral standing and required to follow a strict set of rules controlling their diet, behaviour, and sexuality (Soranus of Ephesus, 1991) This was done as it was believed that women could "pass on" bad traits to the child through their breast milk (Phillips, 2022). However, this does not mean that the child was completely protected. Human milk does not have the same composition throughout the breastfeeding period. In the first days after the birth, the milk produced is called colostrum, which is rich in immunologic components such as secretory immunoglobulin (Ig)A, lactoferrin, leukocytes and developmental factors like epidermal growth factor (EGF) (Ballard and Morrow, 2013). This type of milk is followed by transitional milk, which lasts between the fourth day and the 2nd week after birth. This milk is rich in nutrients like potassium and calcium. After the 4th to 6th week after birth, the milk is considered mature. This milk does not share its immunological or nutritional properties as the colostrum or transitional milk (Ballard and Morrow, 2013; Fleischer Michaelsen et al., 2003). This meant that in some cases, as the breastfeeding was done by a person who had given birth and had breastfed prior to the birth of the child in need, the milk was likely mature and did not contain the same type of nutrients such as vitamins, and more importantly, antibodies (Fleischer Michaelsen et al., 2003). For lower classes, acquiring carefully selected wet nurses when a parent had died or could not otherwise breastfeed or take care of the infant was not possible. This could have led to replacing breast milk with cow's milk, which could have resulted in additional difficulties. Firstly, the child would not receive immunological protection from the colostrum (Ballard and Morrow, 2013) and would be exposed to pathogens earlier (Lewis, 2018). Furthermore, ingestion of cow's milk causes infants to lose occult blood from their gastrointestinal tract. This could lead to cases of iron-deficiency anaemia (Ziegler et al., 1990).

Additionally, times of crisis may have also led to more severe forms of birth control such as intentional infanticide. Due to the social bias of the period which favoured first-born males (Bennett, 2010; Dyer, 2002; Goldberg, 1986), this could have led to female infanticide (Benedictow, 2004).

After the plague, children likely experienced changes in their lifestyles similar to the ones experienced by adults. The high mortality may have led to many orphans seeking work at younger ages. Although there were restrictions in movement to prevent further loss of rural workers (Bailey, 2021; Lewis, 2016*b*), the lack of a workforce opened the job market for younger children and adolescents to be able to migrate to urban centres for jobs (Lewis, 2016*b*). Urban areas offered multiple forms of work which included training in trades under apprenticeships. Apprenticeships refers to a contract, usually for a term of seven years, in which young men and women were taught a trade in exchange for their services (Dunlop and Denman, 1912). Apprenticeships were regulated under a gild which prevented competition with cheaper labour and guaranteed that the apprentice would be well trained (Dunlop and Denman, 1912). The first recorded apprenticeship was found in an ordinance of London Lorimers in AD 1261. By the 15th century, the system was employed by most guilds and towns (Dunlop and Denman, 1912; Lewis, 2016*b*). Apprenticeships were more common among men than women (Goldberg, 1986). These types of contracts were not available to all the population. To pursue an apprenticeship, it was necessary to pay an admission fee (Dunlop and Denman, 1912). This requirement made the position unavailable for poorer children who may have opted for less secure employment as menial workers or domestic servants (Lewis, 2016*b*). Apprenticeships may have extended to 25 years of age, which also socially extended the period of adolescence as apprentices were not considered fully adults (Lewis, 2016*b*). This was similarly observed in female domestic workers who due to their position may have delayed marriage until after 25 years (Goldberg, 1986; **?**).

The job market after the plague affected migration. While men under apprenticeships were under seven-year contacts, women were able to move yearly, engaging in shorter-term migrations for causal labour or working as domestic servants (Goldberg, 1986; Lewis, 2016*b*).

There is much discussion about the health consequences of these changes. Lewis (2016*b*) evaluates spinal pathologies related to strain from physical activities, trauma and infectious diseases in children and adolescents between the ages of 6.6 and 25 years of age. Results show that although there was an improvement in health after the Black Death in London, observed in an earlier age of menarche in females and higher stature in males (DeWitte and Lewis, 2021), urban areas showed more evidence of stress than rural environments. This was reflected in spinal pathologies which were a product of labour-intensive tasks, trauma caused by interpersonal fighting among males and higher frequencies of infectious diseases like tuberculosis in females (Lewis, 2016*b*). This shows that although there was more work available, the living conditions in urban areas and the type of labour may have been unfavourable, particularly for females (Bennett, 2010; Goldberg, 1986; Lewis, 2016*a*). This shows the variability in responses of different groups of the population in the aftermath of the Black Death (Bailey, 2021). It also highlights the significance of examining places outside London and the need to expand the analysis to make an overall analysis of health before and after the Black Death.

3 | Indicators of childhood health in bioarchaeology

The analysis of childhood health is of great significance for understanding overall population health. This is because children are among the most sensitive to environmental stressors (Aufderheide et al., 1998; Lewis, 2018). In the past decade, the developmental origins of health and disease (DOHaD) hypothesis has increasingly pointed to the significance of the initial 1000 days of life for subsequent health, including vulnerability to diseases and other environmental stressors in old age (Barker, 2012; Uauy et al., 2011). The human body is an open system in constant communication with its natural and cultural environment (Goodman et al., 1988). This means that the analysis of childhood health must consider not only the biological factors intrinsic to the body such as illness, sex and age, but also elements from the individual's immediate and general environment. This includes diet, stress experienced by the mother, food availability, sanitation, exposure to pathogens, and cultural practices concerning child rearing (Gowland, 2015; Lewis, 2018) (Figure 2.4).

Health analysis in the osteoarchaeological record poses numerous difficulties that must be addressed. Firstly the concept of health is difficult to define. The World Health Organization defines good health as: "a state of complete physical, mental and social well-being and not merely the absence of disease or infirmity" (World Health Organization, 2014*a*, p. 1). However, this modern definition is not necessarily applicable to all circumstances. Temple and Goodman (2014) describe health as part of an individual's overall well-being and "associated with individual perception of well-being, physiological well-being, and mortality" (Temple and Goodman, 2014, p. 190), which could vary in relation to changing concepts of health and disease. They argue that the concept of health includes a broad spectrum of areas and greatly exceeds the capabilities of bioarchaeological analysis (Temple and Goodman, 2014).

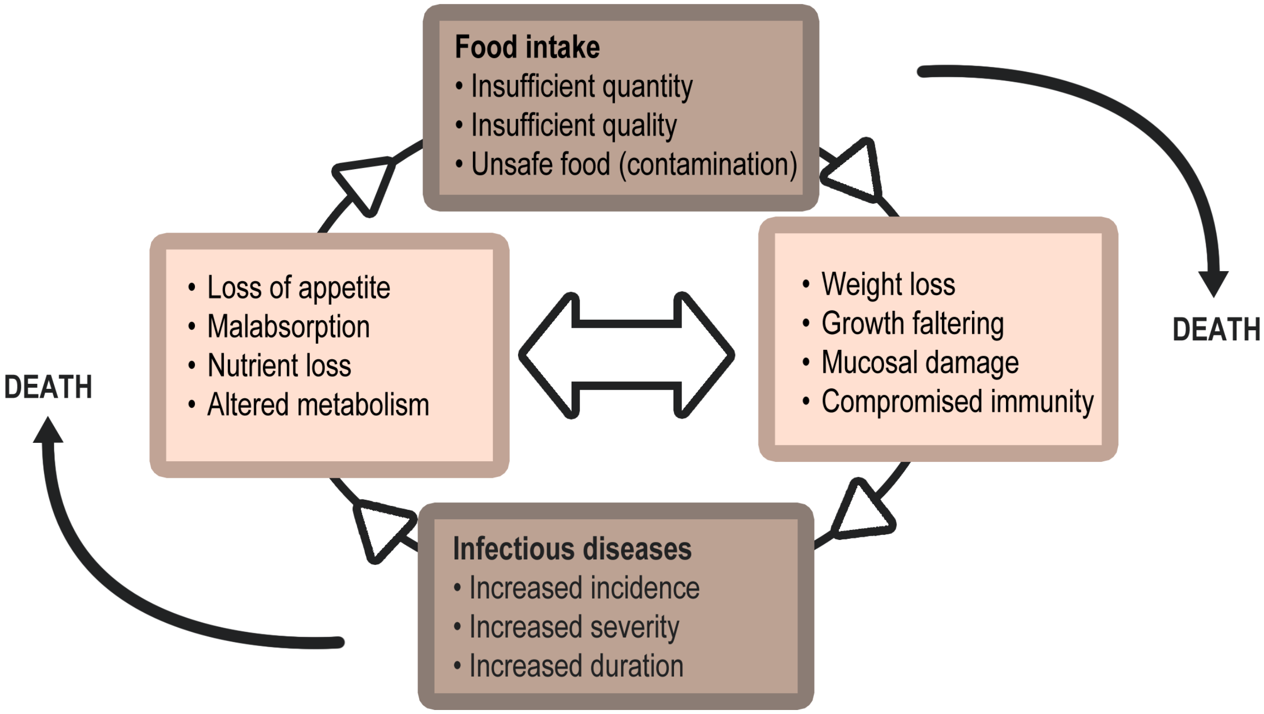


Figure 3.1: *The cycle of infection-undernutrition adapted from Shetty (2010, p. 59)*

The identification and evaluation of stress experiences suffer similar difficulties. Stress, as well as its relationship to health, can include multiple aspects from biological, cultural, social and even psychological perspectives. Many of these stressors are not directly observable in the skeletal record, and for this reason, the current research focuses on nutritional stress. This stress is related to a lack of intake of nutrients. Although the primary cause of this might be a lack of direct access to food, it can also be affected by indirect factors. These include poor sanitation and pollution which affects nutritional absorption, for example, parasitosis (Byers et al., 2001), or lack of exposure to sunlight (Brickley and Ives, 2008*a*). Poor nutrition can have a direct impact on the body's immune system, which instead makes undernourished individuals more susceptible to opportunistic infections such as tuberculosis (Taghizade Moghaddam et al., 2016), which in turn affects an individual's nutritional status by limiting the body's ability to absorb nutrients (Shetty, 2010). This creates a vicious cycle between lack of nutrition and infection in which both elements influence and worsen the other (Figure 3.1).

3.1 Childhood health analysis

In order to evaluate childhood health, it is fundamental to estimate age and sex, as males and females, as well as different age cohorts, will have different lived experiences. However, age and sex estimations present several challenges that vary in relation to the available osteological record. For this reason, the methods to estimate age and sex in bioarchaeology have been the subject of much debate described in the following sections.

3.1.1 Age estimation

Age estimation in non-adults is primarily based on tooth formation. This is because teeth do not remodel and, compared to skeletal growth, their development is not as affected by environmental changes (Cunningham et al., 2016). This does not mean that tooth formation and eruption are completely unaffected by environmental changes. Pathologies like rickets and genetic disorders like osteogenesis imperfecta can affect dental mineralisation (Huber, 2007; Davit-Béal et al., 2014). Furthermore, genetic conditions like Down's syndrome as well as congenital diseases like congenital syphilis affect tooth formation and eruption (Hillson et al., 1998; Moraes et al., 2007). However, when compared to the impact of environmental stressors on skeletal development, weight, or height, dentition is considered less affected and more reliable (Smith, 1991).

Many studies focus on the estimation of age based on dentition. Some investigations focus on creating charts for age estimation. These include studies by Schour and Massler (1940*a*,*b*). These charts have been criticised for not describing the analysed sample, nor explaining the method applied (AlQahtani et al., 2014). These charts have been adapted by Ubelaker (1989), who added a Native American sample to the analysis. Other charts include Moorrees et al. (1963*a*,*b*) and Demirjian and Levesque (1980) based on radiological observation and econometrics of large populations. These charts not only show dental eruption but also include stages for dental formation and mineralisation by age. The first method is based on radiographic imaging taken from longitudinal studies of child health and development conducted at the School of Public Health at Harvard University and the Fels Research Institute in Yellow Springs, Ohio. The resulting charts showing development stages for permanent dentition were later modified by Smith (1991). The second was based on radiological observations of mandibular permanent teeth in 1446 boys and 1482 girls aged 2-20 years of French-Canadian origin. Both methods were applied to multiple populations and although they showed an inter-population error when tested in South African groups (Phillips and Kotze, 2009), the first one was proven to be more reliable when estimating ages for 19th-century Canadian groups (Saunders et al., 1993) as well as current populations from London (Liversidge, 2015). Furthermore, Moorrees et al. (1963*a*,*b*) showed that there was little difference in accuracy when looking at sex-specific and sex-pooled charts (Saunders et al., 1993). This is particularly relevant when applying the method to archaeological collections where sex estimation may not be possible at younger ages.

AlQahtani et al. (2010) developed an atlas based on radiographs of known-age individuals between 28 weeks *in utero* and 23 years of age from collections held at the Royal College of Surgeons of the Natural History Museum, and known age-at-death skeletal remains from Christ Church Spitalfields, in London, and Moorrees et al. (1963*a*,*b*)'s stages of development. AlQahtani et al. (2014) compared both methods, finding the latter more accurate, particularly for ages 1-18 years.

3.1.1.1 Infant and child mortality

Among non-adults, infants and children are among the most vulnerable to suffering stress and dying. Robb et al. (2021) identifies child mortality as one of the most significant causes of death during the medieval period. Infant mortality is defined as the death of individuals under the age of one year, while child mortality includes deaths under the age of five years (Centers for Disease Control and Prevention, 2022; UNICEF, 2021). The analysis of infant and child mortality can provide valuable information about maternal and population health (Gowland, 2015; Lewis, 2018). In this investigation, special attention was placed, when possible, on individuals under five years of age.

It is important to consider that although the method developed by AlQahtani et al. (2010) for age is the most accurate, results are always mean ages with standard errors (AlQahtani et al., 2010). This must be considered when interpreting results.

3.1.2 Sex estimation

3.1.2.1 Sex and gender in bioarchaeology

When estimating sex, it is important to understand the concepts utilised and their implications in health analysis. Sex as a category is based on biological traits. There is no unique criterion to define biological sex. It can be based on multiple traits such as chromosomes, hormone levels, and secondary sexual characteristics, among others (Krieger, 2003). There is much discussion concerning how gender is defined. Different hypotheses relate it to psychological identity, social constructs, and performativity (Zosuls et al., 2009; Butler, 2020). Neither sex nor gender is binary. They both are defined within a spectrum which includes not only females and males but also intersex and non-binary individuals respectively. However, in many instances, social constrictions rooted in racism and colonialism reduce existing variability to binary categories defined within "bionormality" (DuBois and Shattuck-Heidorn, 2021).

In bioarchaeological analysis, sex is estimated based on morphological traits (Buikstra and Ubelaker, 1994; Schutkowski, 1993) and sometimes DNA analysis in non-adults (Lugli et al., 2020; Parker et al., 2019). Sex is estimated within a spectrum between female and male (Buikstra and Ubelaker, 1994). However, these methods have limitations. Although the spectrum acknowledges individuals in the middle whose sex is unknown, this does not refer to inter-sex people. There have been some instances where aDNA analysis has allowed identifying inter-sex people, for example, an individual with sex-chromosomal aneuploidy XXY was identified at the grave found at Suontaka Vesitorninmäki, Hattula, Finland (Moilanen et al., 2022). However, due to limitations in methodology and preservation, they remain mostly invisible in the bioarchaeological record. The archaeological context can provide, in some instances, information to estimate gender. For example, early medieval burials presented grave goods associated with different genders. Dresses and jewellery are linked with female adult graves, while weapons, tools, and drinking vessels may be present at the burials of males (Härke, 2014). Nevertheless, gender identity remains invisible in the bioarchaeological record. Sex and gender have different definitions, but they are often interrelated. Colonialism and racism have affected how sex and gender are defined utilising white European populations as the ideal. This was further exacerbated by the applications of statistical norms in the 19th century which were used to define "normal" and "healthy", pathologising (DuBois and Shattuck-Heidorn, 2021) and othering groups who differed (Johnson et al., 2004).

In this investigation, sex estimation was evaluated based on a spectrum. This means that when analysing morphological traits, each category was estimated within a spectrum that includes, depending on the trait, three to five categories female, probable female, underdetermined, likely male, and male. However, in order to statistically analyse the available data, and more importantly, contextualise it and compare it to similar previous investigations, it was necessary to use the categories of female, male, and unknown sex. The interpretation of the analysis discusses the limitations of only utilising sex estimation based on morphological traits and their relation to gender roles.

3.1.2.2 Sex estimation in non-adults

Sex estimation in non-adults is more complicated than in adults as secondary sexual characters have not developed yet, resulting in reduced sexual dimorphism (Schutkowski, 1993). There have been several approaches to determining sex in non-adults. Nevertheless, their accuracy is not as high as studies involving adults (Campo et al., 2018). As the development between males and females significantly changes after the onset of puberty, it becomes fundamental to include sex estimation in the analysis of pubertal development. More recent studies concentrate on odontometrics of both deciduous and permanent teeth. Okazaki (2005), and Żądzińska et al. (2008) used odontometrics of the first molar for Yayoi, Muromachi and modern Japanese and medieval Polish populations respectively. Investigations like Viciano et al. (2013) used measurements from both deciduous and permanent dentition to estimate sex. More recent studies like Aris et al. (2018) use 10 measurements of the first permanent molar, which include crown, cervical and cusp measurements to estimate sex in non-adults from British populations. The sample was composed of 37 adults and 22 non-adults with recorded age and sex from the crypt of Christ Church, Spitalfields, London dated to the 17th–19th century. The other sample comes from Black Gate cemetery in Newcastle-upon-Tyne. Individuals from this site were dated to the c. 8th–12th century and their sex was estimated based on morphological characteristics of the skull and pelvis. When looking at the Christ Church, Spitalfields sample, 90.9% of all immature individuals were correctly classified. However, when applying the same method to other English samples like skeletons from Black Gate cemetery in Newcastle-upon-Tyne, accuracy dropped to 83.3% (96.7% for males and 65.2% for females). The disadvantages of odontometric approaches are several. Firstly, these methods are population-specific, and to be able to use them in new populations, a prior reference from known individuals must be made. Aris et al. (2018) observed that when it is applied to populations different from the one the original study was made for, accuracy drops significantly. Furthermore, the study does not completely address the extent to which oral pathologies and dental wear may affect measurements taken from teeth. Lastly, for the analysis to be accurate, multiple measurements from multiple teeth must be taken from children as well as from adults for reference. This process is very time-consuming and might not be feasible for large populations. Other researchers have analysed sex estimation through osteometrics of diaphyseal dimensions. Stull et al. (2017) estimated sex in modern South African children aged 0 to 12 years based on 18 post-cranial measurements. Results showed an accuracy of 70% to 93%. Although this method has shown higher accuracy, similar to previous odontometric approaches, it depends on the completeness of the osteological record. The method requires all diaphyseal lengths as well as proximal and distal breadth measurements to be taken, which may not be available in the archaeological record. Morphological approaches to sex estimation include the observation of bones such as the ilium, humerus, and mandible. Weaver (1980) developed a method based on metric and non-metric indicators in the ilium. Results from his investigation showed only one of the non-metric indicators as significant for the estimation of sex in non-adults: the auricular surface. However, accuracy was higher for males (73.1%-91.7%) than for females (43.5%-75%). This shows that this method cannot be used in isolation. Following this line of investigation, Schutkowski (1993) took morphometric traits found in the ilium, along with traits found in the mandible, to assess sexual dimorphism in non-adults. This method was criticised for being population-dependent (Rösing et al., 2007), having a high rate of intra- and inter-observer error, and should not be used solely within forensic contexts. However, Irurita Olivares and Alemán Aguilera (2016) tested Schutkowski's method and found statistically significant ($p < 0.05$) differences between males and females over three months for: a) the angle of the greater sciatic notch (positive predictive values of 0.730 and 0.592 for males and females respectively), b) the depth of the greater sciatic notch (positive predictive values of 0.797 and 0.681 for males and females respectively) and protrusion of the chin region (positive predictive values of 0.665 and 0.688 for males and females respectively). Another method for sex estimation in non-adults was developed by Luna et al. (2017) based on observations of the auricular surface of the ilium. For this method, they analysed a sample of 34 individuals (21 females and 13 males) from the Coimbra identified skeletons collection located at the University of Coimbra, Portugal who died between 1887 and 1934. The methodology included the evaluation of metric and non-metric variables. From all the metric variables, two ratios related to the shape of the anterior area of the auricular surface showed significant differences between males and females. Similarly, from all the non-metric variables, the overall morphology and the apex morphology showed significant differences between males and females. The sexual allocation probabilities were 0.85 for the metric variables and 0.76 for the non-metric variables. Although this method showed higher accuracy, it was developed based on a small sample from a modern population and has not been further tested in other groups. The distal humerus has been used for the estimation of sex in adolescents and adults. The method was initially developed for adults by (Rogers, 1999) based on 20 humeri from the Grant Skeletal Collection of the Department of Anthropology of the University of Toronto and later tested by the same author in 35 individuals with known sex from The University of New Mexico Documented Collection and 93 individuals from the William M. Bass Donated Skeletal Collection. the method was based on the observation of four features of the distal humerus: trochlear constriction, trochlear symmetry, olecranon fossa shape, and angle of the medial epicondyle. These features contribute to the carrying angle of the arm, which differs between males (10-15 degrees) and females (20-25 degrees) (Rogers, 1999, 2009). The method was then tested by (Falys et al., 2005) in a collection of 351 adult humeri (184 male and 167 female) from St Bride's Church, Fleet Street, London dated to the 17th to the early 19th centuries. The study found the method to be 79.1% accurate for estimating sex, with the olecranon fossa shape being the most accurate (84.6%) (Falys et al., 2005). The method then was tested in 42 adolescents between the ages of 11 and 20 years from two collections: the Christ Church Spitalfields Collection housed at the British Museum of Natural History, London, England and the Luis Lopes skeletal collection from the Museu Bocage in Lisbon, Portugal. The method was 80% accurate in estimating sex in males and 82% accurate in evaluating females (Rogers, 2009). However, it is important to consider that the sample for this investigation was small and the accuracy of the method when only considering the Christ Church Spitalfields Collection was 71.4%. The accuracy of these methods does not rise to the standards of forensic practice and should not be used in such contexts. It is also considerably less accurate than similar methods for sex estimation in adults (Schutkowski, 1993). In bioarchaeological contexts, these methods should not be used in isolation. However, when combined, they could provide information valuable for the analysis of childhood and adolescent health. Accuracy levels in the aforementioned methods have shown higher levels for males, which should also be considered when interpreting results. In recent years, molecular approaches to sex estimation have provided more accuracy than morphological indicators. Studies like Lugli et al. (2020) and Parker et al. (2019) focus on the analysis of tooth enamel proteome. This includes amelogenin proteins AMELX and AMELY, as well as enamelin (ENAM), ameloblastin (AMBN), amelotin (AMTN), tuftelin (TUFT1), matrix metalloproteinase 20 (MMP20) and kallikrein 4 (KLK4). These analyses have been previously applied to the study of samples from ancient hominins (*Homo erectus*(Cappellini et al., 2019) as well as modern humans from medieval Ireland (Tierney and Bird, 2015). The amelogenin protein AMELY is linked to the Y chromosome and the male sex, while AMELX is linked to the X chromosome and the female sex. The analysis of peptides related to either AMELY or AMELX aids in discriminating males from females. Results have shown high accuracy. However, this method has shown inter-population differences leading to variable levels of accuracy (Lugli et al., 2020). Furthermore, to obtain the information required for these molecular analyses, a small part of the bone tissue needs to be extracted from the skeleton (e.g. a part of even an entire tooth). This results in the destruction of the tissue. In this investigation permission to study the remains was granted solely for non-invasive analyses for which this technique does not qualify. These restrictions added to the limitations due to the cost and quality of the sample making it unsuitable for this investigation.

It is important to mention the biases and influences of racism and colonialism in how sex and gender studies extend to non-adults and their development (DuBois and Shattuck-Heidorn, 2021). This issue has become a subject of much controversy in Europe and particularly the UK (Pearce et al., 2020; Tudor, 2021) and affects the area of biological and forensic Anthropology (Tallman et al., 2021). In this investigation, sex estimation of non-adults was focused on the study of adolescence: This period was defined from 10 to 25 years of age. This range was based on two studies of modern Dutch children and medieval adolescents which found the onset of puberty between the ages of 10 and 12 (Lewis et al., 2016) until the age in which the brain matures completely (Blum et al., 2012; Rogol et al., 2002). Using the same range as previous studies like (DeWitte and Lewis, 2021) also allowed comparing results.

3.1.3 Measuring physiological stress

3.1.3.1 Growth delay

Growth delay has been recognised as an indicator of nutritional stress during childhood and adolescence (Bogin, 1999).

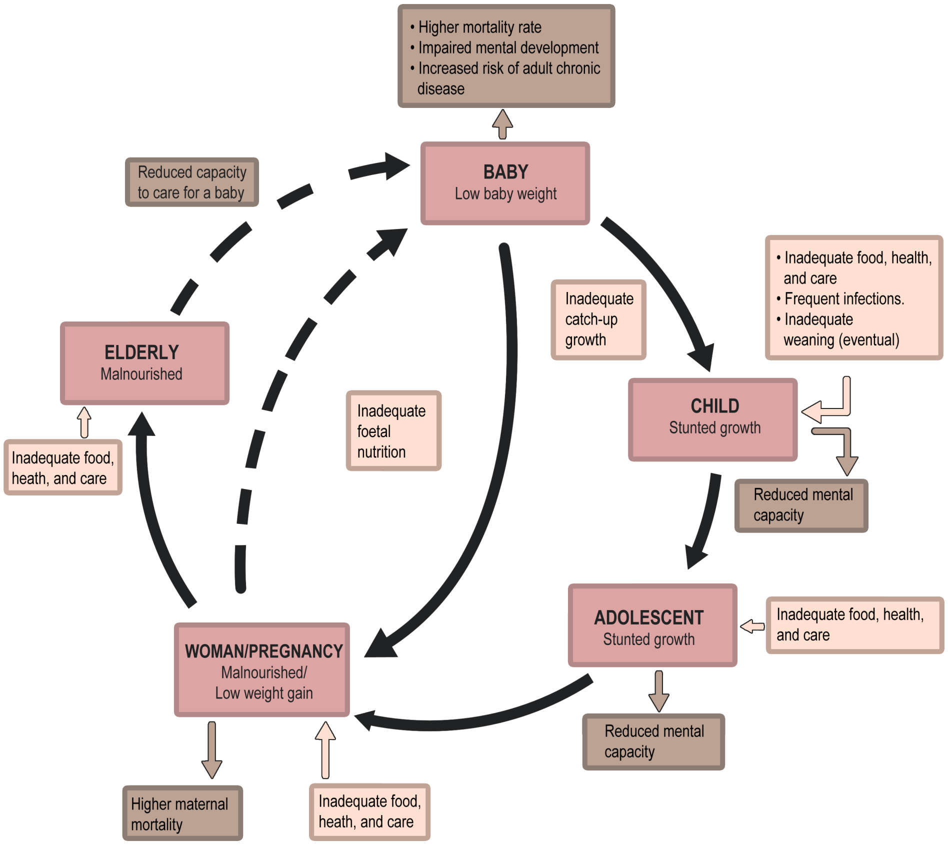


Figure 3.2: *Stress in different periods of life and its impact on health adapted from Shetty (2010, p. 145)*

Metabolic-nutritional stress experienced during childhood can alter normal growth and development. This can be observed in multiple variables during childhood and adolescence (Bogin, 1999) (Figure 3.2). The World Health Organization defined growth retardation as when the proportion of weight for age and height for age falls -2 and -3 standard deviations (SDs) (Z-scores) (De Onis and Blossner, 1997) below the median expected for a specific age cohort according to the WHO Child Growth Standards (WHO Multicentre Growth Reference Study Group, 2006*a*). Although the relationship between stature and health is not necessarily direct (Temple, 2008), previous researchers have tried to estimate stature to assess growth delay and its relationship with health. This was observed through the development of regression equations based on osteometrics, which used long bone length (Trotter, 1970), or sacral and coccygeal vertebrae (Pelin et al., 2005) to calculate height. However, they also presented difficulties as growth does not often progress at a linear pace, and sometimes a single bone fails to properly reflect the growth of the entire body. Additionally, they were also proven to be population-specific (White and Folkens, 2005). Trotter (1970) was based on remains from soldiers from the US who had died during the Vietnam War (1955-1975), while Pelin et al. (2005) was based on a sample from modern hospital patients from Turkey. For this reason, some studies have directly used bone dimensions to assess growth. Geber (2014) analysed 230 children with estimated ages between 6 months to 12 years buried at the workhouse in the city of Kilkenny, Ireland between 1842 and 1851. They estimated growth delay by comparing femoral lengths to a modern reference sample. WHO has used weight and height to evaluate nutritional status in breastfed children around the world (WHO Multicentre Growth Reference Study Group, 2006*b*). For this purpose the organisation developed the Growth Reference Study Group that studied groups from Brazil (South America), Ghana (Africa), India (Asia), Norway (Europe), Oman (Middle East) and the USA (North America) to construct growth charts (WHO Multicentre Growth Reference Study Group, 2006*a*). They did not take measurements of bones in their Growth Reference Study Group, and for this reason, the sample cannot be used as a reference. Geber (2014) instead uses the Denver Growth Study. This sample is composed of healthy children of European descent in the United States between the ages of 6 months and 12 years. This sample was chosen due to having comparable radiological evidence (Maresh, 1970) and being considered representative of normal child growth by the World Health Organization (WHO Multicentre Growth Reference Study Group, 2006*b*). This approach has the benefit of evaluating the existence of growth delay against a fixed reference point. However, when using archaeological references, it is difficult to assess how relevant references from current populations are. It is preferable to directly compare bone lengths between either analysed groups or between the group and a referential sample. Additionally, limiting the investigation to femoral lengths might restrict the samples due to preservation issues, as well as overlook evidence of growth delay present in other bones and possible evidence of allometry (Vercellotti et al., 2011).

3.1.3.2 Vertebral development and neural canal size

In recent years, researchers have focused on the analysis of vertebral growth and the development of the neural vertebral canal to assess growth delay (Newman and Gowland, 2015; Watts, 2011). This approach is chosen because the vertebral neural canal develops early in life and it can be negatively affected by adverse conditions during formative years. Furthermore, the development of the vertebral neural canal, estimated by measuring the antero-posterior (AP) and transverse (TR) diameters (Watts, 2013), is not related to the size of the vertebral body, which can be affected by geographical origins or sex (Newman and Gowland, 2015; Watts, 2015). The vertebrae develop *in utero* and the spine continues to grow and almost completely fuse by 20-25 years of age (Cunningham et al., 2016). The neural arches fuse between the ages of 1 and 2 years, starting with the lower thoracic and lumbar vertebrae and ending with the cervical arches. Similarly, neurocentral fusion is experienced firstly on the lumbar vertebrae and later on the cervical and lastly the thoracic vertebrae at the age of 5-6 years. TR dimensions may still be affected by growth disruptions until 12-14 years of age for modern populations (Rajwani et al., 2002; Watts, 2013). Measuring the vertebral neural canal allows for identifying stress at specific ages during childhood and adolescence that may have led to growth delay. However, when interpreting results from this analysis, it is important to consider that the relatively long development of the vertebral neural canal may allow the individual to recover before the canal finishes developing resulting in no significant reduction in VNC dimensions (Amoroso and Garcia, 2018).

3.1.3.3 Cribra orbitalia

Cribra orbitalia is a broad stress indicator that can be observed as porosity on the roof of the orbits. There have been multiple discussions concerning the specific aetiology of cribra orbitalia and its relation to stress and pathology. Cribra orbitalia is observed as porous lesions on the anterolateral roof of the orbit (Offenbecker and Case, 2016). General consensus has linked this condition to diploic hyperplasia caused by anaemia (Stuart-Macadam, 1991; Oxenham and Cavill, 2010; Offenbecker and Case, 2016). Due to the fragility of the orbital bone, the level of expansion required to perforate the bone is smaller than the rest of the cranial vault (Brickley, 2018). Anaemia is defined by the WHO as:

> ... a disorder characterised by a blood haemoglobin concentration lower than the defined normal level and it is usually associated with a decrease in the circulating mass of red blood cells. This may result from decreased generation of blood cells, or their premature destruction, or from loss through chronic blood loss or haemorrhage. (World Health Organization, 1992, p. 2)

The standards for normal iron levels are established based on nutritional requirements and differentiated by age and sex (Ramakrishnan, 2001) (see , table B.3 for further details) . There are numerous ways to classify different types of anaemia based on diverse criteria such as origin, cause, cell size, and morphology, among others. The totality of these classifications vastly exceeds the scope of this investigation. For this investigation, the types of anaemia more commonly related to nutritional deficiencies and immunosuppression are:

- **Iron deficiency anaemia:** This is anaemia caused by an iron deficiency. The WHO has classified it as the most common type of anaemia worldwide (World Health Organization, 1992; Ramakrishnan, 2001). It is defined by the reduction of iron storage, low serum iron, transferrin saturation and low haemoglobin concentrations (Williams et al., 1990, ch. 48). Haemoglobin is an important component of the red blood cell. This metalloprotein is rich in iron, and for that reason, its decrease affects the morphology and general aspect of red blood cells. The reduction of haemoglobin causes a diminution in their size (microcytosis) and colour (hypochromia). As with all anaemia, the degree of intensity can vary.

- **Megaloblastic anaemia:** These types of disorders are characterised by impaired DNA synthesis (Williams et al., 1990, ch. 47). Similarly to iron deficiency, this disorder affects the morphology of the cell. The inability to properly undergo the process of mitosis (cell division) makes the cell grow without being able to divide. This division results in large, immature red blood cells (macrocytosis) with low functionality. These cells can be identified by having more cytoplasm in relation to their nucleus. Megaloblastic anaemia has a lot of causes, but they are mainly related to vitamin B12 and folic acid deficiency (Djuric et al., 2008; Ramakrishnan, 2001).

Acquired anaemia can be caused by a multitude of factors as well, which include:

- **Poor nutrition:** This includes substandard intake of iron, vitamin B12, and folic acid which can lead to iron deficiency and megaloblastic anaemia respectively. However, these nutritional deficits rarely occur in isolation. Often the lack of other micronutrients such as retinol, riboflavin, and vitamin A have been acknowledged as related to anaemia as well (Ramakrishnan, 2001). A lack of other nutrients like magnesium has also been linked to cribra orbitalia. This is because magnesium is a co-factor in the process of bone mineralisation (Djuric et al., 2008; Ravaglioni et al., 1996).
- **Blood loss/haemorrhage:** Blood loss can contribute to the development of anaemia, particularly iron deficiency anaemia. This condition can present in several ways. In women, it can be caused by menstrual blood loss or blood loss during childbirth (Ramakrishnan, 2001). In men and older women, it can be related to gastrointestinal bleeding related to ulcers, hiatal hernia, gastritis, haemorrhoids, vascular anomalies

and neoplasms (Williams et al., 1990), or trauma (Morris et al., 1992). In very young infants, gastric blood loss can be related to the introduction of cow's milk into the diet during lactation (Jiang et al., 2000).

- **Malabsorption/parasite infection:** Although sometimes the nutritional intake is adequate to the body's requirements, malabsorption results in the inability to properly synthesise consumed food. Malabsorption can be caused by many factors. In this case, the most relevant include parasite infections, which cause upper-track chronic diarrhoea such as *Giardia lamblia*, *Diphyllobothrium*, some forms of hookworm and roundworm (Djuric et al., 2008; Ramakrishnan, 2001), tapeworms, and liver flukes (Godde and Hens, 2021). Some types of foods that contain high levels of phytates like flax and maize can interfere with normal iron absorption, although fermentation can help reduce this effect (Brune et al., 1992).
- **Hormonal conditions and pregnancy:** Females can be more susceptible to some forms of anaemia such as iron deficiency and megaloblastic anaemia due to blood loss during menstruation and higher iron and vitamin requirements during adolescents and pregnancy (Allen, 2000; Ramakrishnan, 2001).

These circumstances are not mutually exclusive and can occur simultaneously. Although cribra orbitalia has been related to non-specific metabolic stress caused by anaemia, there is a great debate concerning its specific aetiology, whether it is primarily due to iron deficiency (Oxenham and Cavill, 2010), megaloblastic anaemia, nutritional deficiencies or due to parasitosis and/or malabsorptive diseases (Walker et al., 2009). Additionally, although all anaemia has a multitude of clinical symptoms, not all cases are severe enough to cause marrow hyperplasia and skeletal changes. For this reason, only the most severe cases are observable in the skeletal record (Wapler et al., 2004). The age of the individual experiencing stress has profound significance on the occurrence of cribra orbitalia. This is because porosity caused by marrow hyperplasia can only happen in areas with red or mixed marrow (Brickley, 2018). Children are born with red (hematopoietic) marrow. This marrow is responsible for erythrocyte production, and it is distributed all over the skeleton. As the individual grows up, red marrow gradually converts to yellow (adipocyte) marrow. This marrow is characterised by minimal blood cell production and it is mostly composed of fatty cells (Brickley and Ives, 2008*a*; Herrmann et al., 2012). During childhood, red marrow is distributed all over the body. As they grow up this marrow becomes yellow, full of inactive fatty cells gradually over the body. In the orbits, after the age of 10, red marrow has converted to mixed marrow (a combination of red and yellow marrow), and by the age of 40, red marrow is almost exclusively found in vertebrae. If the stress experienced is severe enough, lesions from cribra orbitalia may reactivate at older ages. However, due to the nature of marrow conversion, it is highly unlikely for this to happen after the age of 30 years (Brickley, 2018). Cribra orbitalia is not the only pathology characterised by the porosity of the orbital roof. Other nutritional deficiencies that show similar skeletal markers are scurvy and rickets. It is very difficult to differentiate between lesions caused by marrow hyperplasia and lesions caused by subperiosteal haemorrhage related to scurvy or suboptimal mineralisation caused by rickets (Klaus, 2017). To be able to distinguish these pathologies, it is important to do a careful structural analysis of the observed porosity (Brickley, 2018). Lesions caused by cribra orbitalia are characterised by pitting in an outward direction caused by marrow expansion. This pitting is often crescent-shaped, symmetrical, and does not project above the lamellar surface. Alternatively, scurvy lesions present porosity of small size, are asymmetrically distributed and favour the area immediately posterior to the orbital margin (Klaus, 2017). New bone formation related to bleeding in the orbits is observed. The "pitting" of the porosity is found penetrating the surface of the cortex (Brickley and Ives, 2006; Klaus, 2017). Cribra orbitalia may be found along other cranial lesions like porotic hyperostosis. Porotic hyperostosis is a broad stress indicator that can be observed as pitting and porosity on the external surface of the cranial vault (Walker et al., 2009), which results from a thinning of the outer table of the bones and a thickening of the diploe (spongy) bone located in the middle table (Stuart-Macadam, 1992) Similarly, to cribra orbitalia, its specific aetiology has been argued. However, consensus has related the lesions to anaemia (Oxenham and Cavill, 2010; Walker et al., 2009). Cribra orbitalia may also be recorded along other cribrous lesions like cribra humeralis and femoralis (Djuric et al., 2008). Alternatively, scurvy presents porosity in other sectors of the cranium and post-cranium, including the scapula, long bones, maxilla and the greater wings of the sphenoid bone (Brickley and Ives, 2006; Klaus, 2017). The latter is identified as pathognomonic by Ortner et al. (1999). Similarly, the porosity characteristic of rickets is larger and more irregular than in cribra orbitalia and scurvy (Ortner and Mays, 1998). Additionally, evidence of rickets is often found in correlation with the deformation (bending) of long bones (Brickley and Ives, 2008*a*). Although there are specific criteria to distinguish these lesions, it is important to consider health as a complex and multivariable concept. Nutritional deficiencies seldomly happen in isolation and multiple lesions can be present in a bone (Lewis, 2018). In the past few years, there have been new discussions concerning the aetiology of cribra orbitalia, its relationship with stress and pathology, as well as its use in paleopathology (Zdilla et al., 2022). New investigations combine bioarchaeological, forensic, and clinical data to further contextualise the causes of cribra orbitalia. Researchers like O'Donnell et al. (2020) highlight the possible relation between cribra orbitalia and cardiovascular conditions and respiratory infections. Similarly, Gomes et al. (2022) look at the co-occurrence between cribra orbitalia and cribra humeralis and femoralis as well as its relationship with pulmonary infections, especially tuberculosis. For this purpose, the authors evaluate the presence of cribra orbitalia on forensic individuals with known causes of death from New Mexico, the United States and Coimbra, Portugal respectively. Although these conditions could be linked to other factors unrelated to diet (Zdilla et al., 2022), there is a strong link between infection, particularly respiratory and malnutrition, and especially in children (Cunha, 2000). In their investigation, O'Donnell et al. (2020) does not analyse the presence of anaemia, so their investigation does not disprove possible links between both conditions. A more innovative perspective on the causes of cribra orbitalia considers that the condition bears little relationship with pathology. Zdilla et al. (2022) propose that cribra orbitalia has a relation to the vascular anatomy of the sphenoid and frontal bones rather than reflecting a pathological condition. The analysis concludes that cribra orbitalia is more likely to appear in individuals with meningo-orbital foramen than in people without. This casts doubts on interpretations of cribra orbitalia prevalence, as it may be related to vascular anatomy rather than reflecting any form of stress. There are arguments against this hypothesis. The authors discuss that the rate of cribra orbitalia across populations is very similar. Although in some cases this is true for overall scores, when looking at distributions within each investigation cited, there is much more variation in rates. Furthermore, results from many of the investigations used as examples for similarities have not been analysed for meningo-orbital foramen presence, so the connection cannot be observed. The investigation completely discards the possible relation between pathology and cribra orbitalia. However, bioarchaeological studies like Yaussy and DeWitte (2018) described higher frequencies of cribra orbitalia in famine contexts, while investigations focusing on modern populations like O'Donnell et al. (2020) showed that individuals who had violent causes of death (not chronic conditions) showed lower rates of cribra orbitalia than the ones who had. It is possible that even if there was a likeness of developing cribra orbitalia primarily related to the meningo-orbital foramen, the condition was developed by individuals who were experiencing stress. The difficulties in interpreting the aetiology of cribra orbitalia and its possible link to normal development highlight the weakness of its use as an indicator of metabolic nutritional stress. It is important to consider its prevalence within the context of multiple indicators and the wider context of the analysed population.

cribra orbitalia has been evaluated in populations both ancient and contemporaneous around the world. Multiple studies such as for example Mays (2010); Mittler and Van Gerven (1994); Peck (2013); Suby (2014); Walker (1986) have recorded cribra orbitalia to analyse population health. In medieval England this condition has been recorded in several investigations. Researchers like Lewis (2002*a*) evaluated 482 non-adults with observable orbits from four medieval and post-medieval sites in England. Results showed frequencies of cribra orbitalia 55 (Raunds Furnells), 56% (St. Helen-on-the-Walls), 56% (Wharram Percy) and 57% (Christ Church). Yaussy and DeWitte (2018) examined 305 non-adult individuals from Spital Square in London, finding 48.1% 35.8% of cribra orbitalia for the famine and attritional context respectively. Other studies focused on adult populations. Sullivan (2005) analysed 147 adult skeletons with at least one observable orbit dated to the medieval period from the Gilbertine Priory of St. Andrew, Fishergate, York, finding 37.4% of the population with evidence of cribra orbitalia. Analysis of adult populations in London included works from DeWitte (2010) and Yaussy et al. (2016). The first analysed 299 adults from the East Smithfield Black Death cemetery and the latter studied 1031 adult individuals from Spital Square, London, finding 35% of individuals with evidence of cribra orbitalia.

3.1.3.4 Dental enamel hypoplasia

Dental enamel defects are caused by the premature cessation of enamel secretion, which results in reduced enamel thickness and reflects a period of metabolic-nutritional stress experienced during tooth formation (Hillson, 2014). There are multiple factors linked to the development of enamel hypoplasia, including hereditary anomalies and localised trauma. However, it has been most frequently related to periods of systemic metabolic stress, which leads to a disruption in normal enamel formation (Goodman and Rose, 1990; Hillson, 2014). For example, nutritional deprivation such as deficiencies in vitamin A; systemic and infectious diseases like osteomyelitis or neonatal disturbances which include high fevers experienced by the mother during pregnancy can result in the defects. Other factors like vitamin D deficiency and lack of fluorine in water can also disrupt enamel formation. Defects can be linear, plane form, pitted or localised (Towle and Irish, 2020). For that reason, similarly to cribra orbitalia, this condition is considered a non-specific indicator of physiological stress (Hillson, 2005). The diverse nature of defects has been discussed in recent investigations. Researchers like Towle and Irish (2020) have proposed that different types of enamel hypoplasia could have different aetiologies. Linear enamel hypoplasia (LEH) is the most common type observed and reported and is generally related to metabolic-nutritional stress (Guatelli-Steinberg et al., 2012). Plane enamel hypoplasia has been related to severe linear defects, in the most severe cases, with life-threatening events (Towle et al., 2018) like severe malnutrition or infection (Ogden et al., 2007). Pitting may be related to periods of stress or crown and tooth position, and localised defects could result from trauma during development, have genetic causes, or be related to the morphology of the tooth (Towle and Irish, 2020). Similarly to cribra orbitalia, the broad spectrum of possible causes of dental enamel hypoplasia (DEH) makes it fundamental to contextualise the analysis within multifactorial studies to better assess potential periods of metabolic-nutritional stress.

There is much discussion concerning what is the most accurate method to identify the presence and the age-of-formation of enamel hypoplasia, particularly linear enamel hypoplasia. Previous studies have shown that the most accurate methods to identify linear enamel hypoplasia involve destructive techniques that allow histological observations of each tooth (Witzel et al., 2008). Other less invasive microscopic techniques involve taking impressions, creating replicas from the crown surface of the selected teeth and analysing them through a microscope (Spectra Services, Rochester, New York, United States) and Vision Gauge Software (Vision X, Inc., Point Claire, Quebec, Canada) (Temple, 2014). Scanning Electron microscopy (SEM) can be used to estimate the spacing between adjacent perikymata to identify the presence of different types of enamel hypoplasia, like for example, cuspal (Ogden et al., 2007) to identify linear enamel hypoplasia and estimate its age at formation (Ritzman et al., 2008). It can also be used together with histological analysis to analyse how the enamel defect formed (Witzel et al., 2008). However, these techniques are very time-consuming and expensive and do not allow for the observation of large collections. Furthermore, it can lead to the over-recording of defects (Hassett, 2014).

When assessing the age-at-formation of defects, similarly to its identification, destructive histological techniques permit observing “hidden” defects that are not observable after the age of 2, and smaller defects are generated after small periods of stress (less than 5 days) (Hillson, 2005, 2014). Among non-destructive techniques, there are two main methods to estimate age-at-formation. The first involved regression equations based on mean crown height (CH) per tooth type and the distance between the defect and the cement-enamel junction. The most widely used of these equations was designed by Goodman and Rose (1990). This formula has subsequently been adapted to other populations with different mean crown heights (Martin et al., 2008). The problem with this method is that it assumes a linear tooth formation, which is not the case. It also overlooks hidden cuspal enamel which is related to tooth formation before the second year of life (Primeau et al., 2015). The second technique addressed many biases and critiques which exist in the previous one. Reid and Dean (2000, 2006) and Holt et al. (2012) created a decile chart based on histological analysis of teeth from multiple populations. This analysis includes hidden cuspal enamel and reduced inter-population error. Comparisons among the two methods showed higher accuracy for the latter (Martin et al., 2008). However, the technique does not address all limitations. If the method is used for macroscopic observations of linear enamel hypoplasia, although the estimates of age-at-formation are likely to be more accurate, the limitations in the identification of defects due to hidden cuspal enamel will make for estimations before 2 years of age or defects caused by periods of stress under 5 days unlikely to be detected and therefore not included in the analysis.

3.1.4 Specific vitamin deficiencies

3.1.4.1 Vitamin C deficiency

Vitamin C (ascorbic acid) plays a key role in blood formation, folic acid metabolism, and bleeding, as well as collagen synthesis including connectivity for skin, cartilage and bone (World Health Organization, 1999). Differently from other mammals who synthesise vitamin C from glucose in their livers, humans, along with non-human primates and guinea pigs, can only incorporate ascorbate exogenously (Padayatty et al., 2003) and it is not stored within the body (Kothari et al., 2020). Vitamin C is found in a variety of food but more importantly vegetables and fresh fruits, especially citruses (Halcrow et al., 2014).

Scurvy is a pathology caused by vitamin C deficiency. Similarly to anaemia, this deficiency can be caused by several reasons, such as 1) limited intake, 2) increased requirements, 3) malabsorption of the vitamin (Halcrow et al., 2014), or 4) due to genetic predisposition (Delanghe et al., 2007). It can affect adults (Brickley et al., 2016) and non-adults (Brickley and Ives, 2006; Lewis, 2018; Schattmann et al., 2016). In clinical settings, scurvy can be identified after sustained vitamin C deficiency lasting between 1 and 3 months (Golriz et al., 2017). Recuperation from scurvy is much more rapid, showing clinical improvement two days after vitamin C is reintroduced to the diet (Greenfield, 1990). The disease presents multiple clinical symptoms. The most commonly associated are perifollicular haemorrhage, loss of secretion of salivary and lacrimal glands, swelling of parotid and submaxillary glands, loss of dental fillings, loss of hair and dryness and itching of the skin (Sjögren's syndrome), femoral neuropathy resulting from haemorrhage into the femoral sheaths, oliguria with oedema (especially of the lower extremities), psychological disturbances followed by decrements in psychomotor performance, impaired vascular reactivity (for example, poor responses to stimuli that normally activate vasomotor adaptive mechanisms), and scorbutic arthritis (Hodges, 1971). Although in the past it was considered a rare symptom, ocular haemorrhages, especially in the bulbar conjunctivas, have been observed in patients with scurvy (Hood and Hodges, 1969; Hodges et al., 1969; Hodges, 1971), as well as in the bioarchaeological record (Brickley and Ives, 2008*a*; Geber and Murphy, 2012). Haemorrhages from ruptured blood vessels can also be manifested through bleeding gums in teething children and adults (Brickley and Ives, 2006; Halcrow et al., 2014; Planerova et al., 2017).

In infants, this disease was initially named Moeller-Barrow disease (Lewis, 2018). Although infantile scurvy can be detected in individuals as young as 5 days old, it is normally registered in infants aged between 5 and 24 months (Brickley and Ives, 2006). This disease can be caused by changes in the diet of the infant. This can be due to the introduction of animal milk during the weaning diet or to replace breast milk (Brickley and Ives, 2008*a*). Vitamin C is sensitive to heat, so boiling milk can also limit vitamin C intake (Brickley and Ives, 2008*a*; Saavedra et al., 2018). Breast milk is generally rich in vitamin C. However, malnourished lactating mothers can also result in a reduced intake of vitamin C in infants (Ahmed et al., 2004). Other reasons that can lead to the development of scurvy in infants and children are cultural practices, abuse and neglect (Halcrow et al., 2014). In modern clinical cases, scurvy has been observed in children with autism spectrum disorder (ASD) with very restricted diets (Kothari et al., 2020; Planerova et al., 2017; Saavedra et al., 2018).

Although scurvy in adults develops after months of deficient vitamin C intake, in children, due to higher nutritional requirements, this period can be much shorter (Brickley and Ives, 2006). Its symptoms include irritability, tenderness of the legs, pseudoparalysis (usually involving the lower extremities), costochondral beading and bleeding around erupting teeth. If scurvy is treated, the individual can recuperate. However, if left untreated, it can lead to death (Kothari et al., 2020; Padayatty et al., 2003).

In the bioarchaeological record, vitamin C deficiency is considered identifiable in the skeleton only after 6 months of deficiency. However, in cases of infantile scurvy due to higher nutritional requirements, this period can be shorter (Brickley and Ives, 2006). The identification of scurvy in non-adults has been detailed by Ortner and Ericksen (1997), Ortner et al. (1999), and Brickley and Ives (2008*a*). Lesions are characterised by scorbutic lesions related to haemorrhage, which leads to bone deposits and porosity. Multiple bones of the body are affected. However, the greater wings of the sphenoid bone have received more attention due to being considered pathognomonic of the condition (Ortner et al., 1999; Geber and Murphy, 2012).

Scurvy rarely occurs in isolation and is often related to other deficiencies. As scurvy has an influence on iron absorption it is common to observe it simultaneously with anaemia (Halcrow et al., 2014; Ortner et al., 1999). This is also observed in the skeleton where orbital roof lesions caused by scurvy can be mixed with lesions related to anaemia such as cribra orbitalia (Brickley, 2018). Scurvy can also present with other pathologies such as rickets (Schattmann et al., 2016). Researchers like Lewis (2010) described findings of 364 non-adults under the age of 17 years presenting rickets and/or scurvy in 11.2% of cases, and cribra orbitala in 38.5% of cases in skeletons from the Romano-British Poundbury Camp in Dorset. Similarly, an analysis of 545 non-adults and 425 adults from the Kilkenny Union Workhouse in Ireland during the Irish Famine showed co-occurrence of scurvy and rickets and scurvy and tuberculosis in 14 and 3 cases for juveniles respectively, and scurvy and residual rickets and scurvy and tuberculosis in 9 and 1 case in adults respectively. Schattmann et al. (2016) identified 12 possible cases of co-occurrence between scurvy and rickets in 48 individuals

(25%) under the age of 5 years buried at the Saint-Amé collegiate church in Douai, France and dated between the 16th and 18th century.

3.1.4.2 Vitamin D deficiency

Vitamin D is a steroid pro-hormone usually associated with regulating the amount of phosphate and calcium in the body (Bell et al., 2010), particularly affecting the skeleton. However, it also has other functions including immunoregulation, blood pressure homeostasis, cell division and programmed cell death (Snoddy et al., 2016). Its deficiency can have multiple consequences in the body such as decreased intestinal calcium absorption, secondary hyperparathyroidism, hypophosphatemia, increased bone turnover and lower bone mineral density (Brickley and Ives, 2008*a*). Although this deficiency can be related in a smaller proportion to a lack of dietary intake, as well as genetic factors, it is mostly related to a lack of exposure to UV-B irradiation (sunlight). For this reason, when analysing possible causes for rickets it is important to consider not only diet but also climate and exposure to sunlight (Brickley and Ives, 2008*a*; Snoddy et al., 2016), as well as cultural practices that could affect sunlight exposure such as swaddling in children (Rohnbogner and Lewis, 2017). In children, deficiencies in calcium and bone mineralisation impact both intermembrane (osteomalacia) and epiphyseal growth (rickets). Once the epiphyses have fused, only intramembranous bone turnover is affected (osteomalacia) (Lewis, 2018). Hence, rickets is exclusively experienced during developmental years. In modern cases, it most commonly occurs between 6 and 18 months. Clinical symptoms of rickets include apnoea, convulsions or spasms, particularly in the face, hands and feet (tetany), and muscle weakness (Brickley and Ives, 2008*a*). Similarly to vitamin C deficiency, skeletal changes resulting from rickets require a more severe deficiency. This is unlikely to be caused by seasonal changes but by a more sustained deprivation. In the skeletal record, initial changes include fraying and flaring of the growth plate margins and metaphyseal junction as the cartilage structure starts to lose the organised vertical arrangement. When the disease is more advanced it is possible to see long bone bending deformities and metaphyseal swelling, particularly in the tibiae (Brickley and Ives, 2008*a*). These changes can also be observed radiologically through trabecular coarsening or thinning, loss of cortico-medullary distinction, cortical tunnelling, and biomechanical alternations (Mays et al., 2006). The identification of rickets in the bioarchaeological register has been systematised by studies such as those by Ortner and Ericksen (1997); Ortner and Mays (1998), and later Brickley and Ives (2008*a*). These studies were based on macroscopic observation as well as radiological analyses. As rickets affects the chondrocytes and growth plate, it cannot be observed in adults. Nevertheless, long bone bowing in adults may indicate the presence of residual rickets in adults who suffered the condition during their development (Brickley et al., 2010).

Osteomalacia is defined as the inadequate or delayed mineralisation of osteoid in cortical and spongy bone. Although this condition is caused by vitamin D deficiency, other factors such as genetics and neoplastic processes might also have an impact on the development of the conditions (Brickley and Ives, 2008*a*). Although osteomalacia is usually related to adults, it can also be suffered by no-adults (Lewis, 2007). Skeletal markers of osteomalacia show incomplete mineralisation of bone on pre-existing surfaces. However, endochondral defects are not visible as the epiphyses are fused (Brickley and Ives, 2008*a*). The identification of osteomalacia is more complicated than rickets as the demineralisation characteristic of the disease can be confused with others such as osteoporosis, particularly in older ages (Brickley et al., 2005; Mays and Brickley, 2018). However, Looser's zones (pseudofractures) are considered pathognomonic of the condition. These pseudofractures occur in poorly mineralised regions of the bone. They often appear symmetrically at locations such as the superior and inferior pubic rami, the medial femoral neck and medial sub-trochanteric region, throughout the ribs, and on the lateral border of the scapula (Brickley et al., 2005).

3.1.5 Specific infectious diseases

Infectious diseases significantly affected the population health during the medieval period. Although the Black Death caused unprecedented mortality, it was not the greatest health issue during the medieval period (Robb et al., 2021). Infectious diseases like tuberculosis, leprosy, dysentery, and cholera, among others, had a greater longer-term impact on the health of medieval populations (Robb et al., 2021). The spread of infectious diseases is related to climate conditions (Lafferty, 2009), living conditions (McNicholas et al., 2000), and population pre-existent health (Narasimhan et al., 2013). Poor sanitation conditions and overcrowding can lead to the spread of faecal-oral infections like cholera and airborne infections like tuberculosis (Lewis, 2018; McNicholas et al., 2000). People who are immunosuppressed due to poor nutrition are more susceptible to contracting diseases like tuberculosis and leprosy (Narasimhan et al., 2013). Due to their underdeveloped immune system, children are more vulnerable to pathologies like tuberculosis or cholera (Basu Roy et al., 2019; Leung et al., 2012; Lewis, 2018), which can negatively affect their development or cause death (Fox et al., 2016; Lewis, 2018). These pathologies also weaken the body, increasing the risk of contracting other diseases. In the past, tuberculosis has been identified in co-occurrence with pathologies related to metabolic-nutritional scurvy by researchers such as Geber and Murphy (2012) in both adults and children from the Kilkenny Union Workhouse during the Irish Famine. For this investigation, the presence of pathologies in the period previous to the Black Death would have contributed to the frailty of the population and their vulnerability to the Black Death (DeWitte and Wood, 2008).

3.1.5.1 Tuberculosis

Tuberculosis is a chronic infectious disease. It has several forms, but the two most common ones are human tuberculosis (caused by the *Mycobacterium tuberculosis* (*M. tuberculosis*) and bovine tuberculosis (caused by the *Mycobacterium bovis* (*M.Bovis*)) (Ortner, 2003; Roberts, 2020). This organism is one of the most common causes of granulomatous infection (Zumla and James, 1996). The likelihood of contracting the disease is related to intrinsic and extrinsic factors. Intrinsic factors such as immunosuppression and malnutrition can increase the chances of being infected (Narasimhan et al., 2013; Nelson and Wells, 2004). There is also a debate suggesting that there could be a genetic predisposition to contracting the disease (Lewis, 2018), as well as population density, air pollution, and the median age of the infected individuals. In the case of children, parental health status also plays an important role (Nelson and Wells, 2004). Although in modern times, new risk factors such as HIV infections are added to the list of risk factors for the disease (Pawlowski et al., 2012), in past populations tuberculosis was related mostly to urbanisation, poverty and overcrowding (Lewis, 2018). For this reason, it has been widely recorded after the post-medieval period (Roberts, 2020; Roberts et al., 2016). Nevertheless, there is much evidence that tuberculosis also affected populations during the medieval period, particularly the late medieval period. Tuberculosis has been identified in past medieval populations in numerous studies. Dawson and Robson Brown (2012) identified a case of tuberculosis in a non-adult skeleton buried at the cemetery from the priory of St Peter and St Paul, Taunton, Somerset dated to the late medieval period. Walter and DeWitte (2017) evaluated tuberculosis in an adult sample (n=485) from Barton-Upon-Humber and Spital Square as a part of a survival and mortality analysis to assess the differences between urban and rural settings. Robb et al. (2021) analysed the burden of disease during the medieval period and established that tuberculosis and pulmonary infections had the highest prevalence (30%) recorded. The likelihood of contracting tuberculosis varies according to health status, age, and living conditions (Narasimhan et al., 2013). To be infected with tuberculosis it is necessary not only to come in contact with an infected person but also to be susceptible to the disease (Fox et al., 2016). Infants, followed by children, are the most susceptible to this disease. Most of the elements that facilitated tuberculosis infections were related to poverty and urbanisation (Lewis, 2018; Newman, 2015). In children, there were several ways to contract the disease. Due to their living conditions, children were likely to contract the disease from adults (Lewis, 2018). This makes the analysis of tuberculosis in children relevant to population health as they can be taken as a proxy for the adult population. Children could contract the disease from close contact with infected family members (Lewis, 2018; Mittal et al., 2014). Vertical transmission through the placenta is also possible *in utero* or intrapartum resulting in infection in the lungs and gut (Mittal et al., 2014). Although breast milk does not transmit tuberculosis, there is limited evidence suggesting breastfeeding may increase the risk of infection (Mittal et al., 2014). Children (and adults) can also contract human and bovine tuberculosis (*Mycobacterium bovis* through consuming infected meat and milk (Currier and Widness, 2018; Fox et al., 2016; Waddington, 2003). In children, cow milk can be used to replace breast milk and meat can be included in weaning diets.

Children under the age of 5 are the most susceptible (Lewis, 2018). In modern populations, there is a period towards the middle of childhood referred to as the "golden years" where this susceptibility is reduced and then it increases again in adolescence (Gabrovska et al., 2022).

The disease can present itself in several variations:

- **Pulmonary tuberculosis (TB)** is the most common form of the disease. Common clinical symptoms of active lung tuberculosis are a cough with sputum and blood at times, chest pains, weakness, weight loss, fever and night sweats (Taghizade Moghaddam et al., 2016). In the past, infants were the most susceptible to this manifestation of the disease (Lewis, 2018).
- **Extrapulmonary tuberculosis** includes tuberculous lymphadenitis (affecting the lymphatic nodes), pleural tuberculosis, tuberculous emphysema, genitourinary tuberculosis, abdominal tuberculosis, ocular tuberculosis, laryngeal tuberculosis, musculoskeletal tuberculosis, spinal tuberculosis (Pott's disease), extra-axial musculoskeletal tuberculosis (affecting the joints), pericardial tuberculosis (Kumar et al., 2010), and gastrointestinal tuberculosis affecting the gastric system. The last form can appear both as a primary (Amarapurkar et al., 2003) and secondary infection (Ma et al., 2019).

  In children, primary gastrointestinal tuberculosis is caused by *M. bovis* by consuming infected cow milk. Oral transmission results in these infections having a higher number of extra-pulmonary granulomas (Fox et al., 2016).

Tuberculosis infections are also classified as:

- **Primary:** caused by a primary infection through inhalation of infected droplets coughed or sneezed into the air by individuals with tuberculosis (Kumar et al., 2010). Infants and children are more likely to suffer this form of the disease (Veedu et al., 2013).

- **Secondary:** caused by the reactivation of a latent infection (Geng et al., 2005). As the primary infection is most common in infants and children, reactivation of tuberculosis can appear in children and adolescents (Lewis, 2018) (Gabrovska et al., 2022).
- **Miliary:** similarly to secondary plague infection, it is caused by the spread of tuberculosis to extra-pulmonary tissue (Lewis, 2011). This complication is most common in children, particularly under the age of 3 years (Lewis, 2018).

Tuberculosis can affect multiple systems in the body, but only a few expressions of the disease involve osteological lesions. This is because treatment or death might prevent further development of the disease (Santos and Roberts, 2001). For example, in modern cases, musculoskeletal tuberculosis appears in 1% to 3% of infected individuals (Kumar et al., 2010), although it has been reported that the incidence could be between 3 and 5% (Lewis, 2018). Although this may have been higher in past populations due to the lack of chemotherapy, it still represents a small sample of the total affected individuals. This means that only a reduced number of cases of infected individuals are observable in the bioarchaeological record. For this reason, it is expected that the actual frequency of tuberculosis is under-represented in this investigation.

There are no pathognomonic bone lesions to identify tuberculosis. Similarly to the bubonic plague, the only way to definitively identify the disease in skeletal records is through the identification of the DNA of the tubercle bacilli (Mays et al., 2001; Wilbur et al., 2009). This makes the macroscopic identification of the disease more complicated but not impossible. Differently from the bubonic plague, the chronic condition of tuberculosis results in bone lesions that can be used for evaluating the presence of tuberculosis (Dawson and Robson Brown, 2012; Lewis, 2011, 2018; Ortner, 2003). Although multiple bones in the body can present lesions due to tuberculosis, the spine is involved in up to 50% of skeletal tuberculosis cases (Teo and Peh, 2004). Spinal lesions affect at least two vertebrae and particularly involve the lower spine (Ortner, 2003). These lesions are characterised by osteopenia and bone loss. The distribution of lesions is not homogeneous in all ages. These spinal lesions are likely to appear mostly in individuals over the age of 10 years (Lewis, 2018). The tubercle bacilli are mostly found in areas rich in red marrow. For this reason, as the distribution of red marrow changes with age, so do the bones showing lesions of tuberculosis. Although all individuals are likely to show lesions on the spine and ribs, children are likely to exhibit the disease in the cranium, mandible, scapula (Lewis, 2018), tubular bones, hands, and feet (Ortner, 2003).

There has been much discussion on how to identify tuberculosis in the skeletal record. For non-adult analyses, studies such as Rohnbogner and Lewis (2017) have pointed out the significance of the identification of conditions like Pott's disease (spinal collapse). In adults, skeletal tuberculosis lesions have been found in ribs and tubular bones (Assis et al., 2011). However, it is important to mention that these lesions may not be present in infected individuals. Mays et al. (2001) identified the bacterium *M.tuberculosis* in nine individuals with osteological lesions compatible with tuberculosis. From that sample, five did not show lesions on the ribs. This must be considered when interpreting results from the osteological analysis of the disease.

Due to its relationship to immunosuppression, tuberculosis can act as an opportunistic infection experienced during the course of other conditions. For example, individuals who experience deficiencies in vitamin D have been linked to tuberculosis and other upper respiratory infections. This is because vitamin D plays an important role in immunity, particularly phagocytosis and pathogen digestion (Snoddy et al., 2016). Lewis (2018) also reports cases of reactivation of tuberculosis due to whooping cough and measles. For this reason, it is important to evaluate this pathology in correlation with other deficiencies, as well as growth delay. In past populations, tuberculosis was related to poor environmental conditions, especially air pollution caused by urbanisation. As not all populations had the same exposure to pollutants, this disease did not affect everyone the same way. The World Health Organization states that tuberculosis is more prevalent in males (World Health Organization, 2018*a*). However, this is based on current adult populations and is not necessarily reflected in medieval childhood cases. Previous studies have found, due to working conditions, adolescent women were more affected by tuberculosis (Lewis et al., 2015; Lewis, 2016*b*).

3.1.5.2 Plague (Black Death)

There is no pathognomonic skeletal lesion to indicate plague infection. In some reduced individual cases, the *Yersinia pestis* bacterium has been identified through DNA analysis (Cesana et al., 2017; Haensch et al., 2010; Keller et al., 2019; Raoult et al., 2000; Willmott et al., 2020). However, this has not been done at a population level. In this investigation, plague victims were identified by contextual mortuary information, as victims of this disease were buried in cemeteries constructed for that purpose (e.g. East Midfield Black Death Cemetery) (Grainger et al., 2008). Through archaeological evidence, historical records and paleodemographic analysis (Gowland and Chamberlain, 2005), as well as radiocarbon dating it may be possible to identify catastrophic contexts in cemeteries and mass burials (e.g. Thornton Abbey) (Willmott et al., 2020). However, in many cases, victims of the plague were buried by the general population (Giberti, 2019). This resulted in many victims of the Black Death, particularly in rural areas, as well as smaller outbreaks of plague, not being identifiable in the osteological record. This creates a bias that must be addressed when interpreting osteological results from non-plague cemeteries.

3.1.6 Childhood and obstetric stress

The Developmental Origins of Health and Disease (DOHaD) hypothesis has signalled the health and nutrition of the mother as determining the long-term health of her infant, especially during pregnancy, breastfeeding and weaning periods (Barker et al., 2011; Barker, 2012; Gluckman and Hanson, 2006; Uauy et al., 2011) (Figure 3.2). Studies like Barker (2012) explore the connection between poor foetal nutrition, birth weight, and vulnerability to chronic disease. Therefore, in order to fully evaluate nutritional stress experienced during childhood, it is fundamental to consider maternal health in its analysis. In the bioarchaeological record, maternal health analysis presents multiple difficulties. Firstly, it is very difficult to identify women who gave birth or breastfed from skeletal markers or mortuary items (Han et al., 2019). Similarly, although it might be possible to identify individual circumstances where a pregnant woman was buried, it is very difficult to identify stress experienced during pregnancy in skeletal remains. For that reason, the analysis of maternal stress in bioarchaeology has been limited (Lieverse et al., 2015; Viva et al., 2020). Gowland (2015) proposed to expand the clinical analysis related to the DOHaD hypothesis explored by Barker (2012) to bioarchaeology, using the child (under 3 years–1000 days) as a proxy for generational health. The rationale behind this is that, as during pregnancy and breastfeeding the body provides nutrients and immune protection to the fetus and baby, stress experienced during intrauterine life and/or the breastfeeding period reflects stress experienced by the mother. Markers that can indicate maternal stress in the skeleton are included below.

3.1.6.1 Deciduous dental enamel hypoplasia

As it was explained previously, dental enamel hypoplasia is considered a broad, non-specific stress indicator (Hillson, 2005). Enamel hypoplasia affects both deciduous (Infante and Gillespie, 1974; Ogden et al., 2007; Kanasawa et al., 2014) and permanent dentition (Hillson, 2005; Ritzman et al., 2008). Although this period varies among populations, generally enamel hypoplasia found in deciduous dentition reflects stress experienced between the second trimester and the first year of life (AlQahtani et al., 2010). This implies that analysis of deciduous dentition can be used as a proxy to evaluate potential maternal stress (Gowland, 2015), while defects found in permanent dentition reflect stress experienced until the 6th year of life (Reid and Dean, 2006). Differently from other tissues in the body, enamel does not remodel, so enamel defects are observable in the adult dental record. This broadens the available sample. The analysis of DEH allows for potentially identifying stress experienced by an individual during pregnancy in skeletal remains of children that still have deciduous dentition, reflecting stress experienced during pregnancy. Similarly, DEH identified in permanent dentition can potentially identify stress experienced during formative years, including for earlier ages, stress experienced during the breastfeeding period. Both of these markers can be used to identify maternal stress.

3.1.6.2 Spina bifida

Congenital conditions refer to anomalies and pathological changes in the normal development during intra-uterine life or birth (Sarry El-Din and El Banna, 2006). An example of this is how conditions related to neural tube defects are recorded. Neural tube defects occur *in utero*, when the folding of the neural plate does not occur by the 28th day after conception. This can result in damage or even death of the foetus (Wang et al., 2019). These defects can be caused by a combination of genetic and environmental factors. However, folic acid deficiency has been identified as playing a key role in their occurrence (López-Camelo et al., 2005). Many of these conditions like anencephaly are extremely rare and very unlikely to be identified in the osteological record. However, others like spina bifida (Silva-Pinto et al., 2010) are more common.

Spina bifida is neural tube defect. It is generally related to deficiencies in folic acid during pregnancy (Northrup and Volcik, 2000). Its analysis provides information not only about intra-uterine stress, but can also be used as proxy to identify maternal stress (Gowland, 2015). Spina bifida occurs when the vertebral laminae do not fuse correctly, leaving the spinal cord partially or completely exposed. The severity of the symptoms depends on the level of the defect and the nerves involved (Northrup and Volcik, 2000). However, from a clinical perspective expressions of this condition can be grouped into two classifications (Singh, 2013) that are distinguishable in the archaeological record (Lewis, 2018). These are cystica or aperta and occulta. The first one is the most severe form of the condition and without treatment leads to death in early infancy, due to the sacral bones not being fused. This manifestation is often not observable in the osteological record (Lewis, 2018). The second one includes occulta and lypomyelomeningocele. These manifestations are less severe than aperta (Singh, 2013). Depending on the size of the lipoma putting more or less pressure on the spinal cord, lypomyelomeningocele can present clinical symptoms. Spina bifida occulta generally presents no symptomatology and can remain unnoticed. Individuals with these less severe conditions usually survive until adulthood. In the osteological record, these conditions are identified as a partial exposure of the neural canal between S1 and S5 (Singh, 2013).

While spina bifida has been linked to deficiencies of folic acid in the diet of the gestating person (López-Camelo et al., 2005), this is not the only reason to be taken into consideration. Previous investigations, both clinical (Wang et al., 2019) and archaeological (Silva-Pinto et al., 2010), have also linked the condition to arsenic poisoning. Arsenic may have been used in combination with mercury to treat syphilis. However, this treatment started during the post-medieval period (Zuckerman, 2016). Due to its link to the diet of the pregnant person, identifying spina bifida provides the opportunity to identify obstetric stress.

3.1.6.3 Perinatal growth delay

Growth delay or disruption of perinates and infants from what is estimated as "normal" can take different expressions with more or less severity. These expressions are intrauterine growth disruption (IUGR), being small for gestational age (SGA), and low birth weight (LBW). These three conditions refer to different processes, although they often overlap (Monk and Moore, 2004). Intrauterine growth restriction refers to the failure of a fetus to grow along a consistent centile (Monk and Moore, 2004). Being small for gestational age is defined as infants with dimensions smaller than average and birth weight under the 10th percentile (Longo et al., 2014; Monk and Moore, 2004). A fetus who has IUGR also experiences SGA, but SGA can occur without IUGR (Longo et al., 2014).

Multiple issues can lead to growth delay in a fetus. Poor nutrition of the pregnant person results in direct nutritional stress for the child. As explained before, this can be linked to congenital conditions like spina bifida (Northrup and Volcik, 2000). Intrauterine growth restriction has been linked to perinatal morbidity and mortality (Resnik, 2002), premature birth and increased risk for re-natal congenital infections caused by agents such as cytomegalovirus (CVM), herpes simplex virus (HSV), *toxoplasma gondii*, treponema, and varicella-zoster virus (VZV) (Longo et al., 2014). Environmental stressors such as pollutants that may reduce the influx of oxygen to the fetus may also contribute to delaying their developing condition which reduces the oxygen available to the fetus (Rich et al., 2009). Furthermore, SGA can increase the risk of post-natal infections (Longo et al., 2014).

In bioarchaeology, the identification of perinatal growth disruption is observed through measurements of long-bone diaphyseal length. This can be compared alongside metaphyseal width (Hodson, 2017), or compared to dental age assessment (Hodson, 2018; Hodson and Gowland, 2020). This analysis can only be done in non-survivors, which means only the most severe cases are observed. This means that results will probably underrepresent existing cases, as many less severe ones did not lead to the death of the foetus/baby.

To reduce bias caused by analysing only non-survivors (Wilson, 2014), this analysis needs to be part of a larger, multifactorial approach. This approach should include conditions reflecting perinatal stress observable in survivors like spina bifida and deciduous dental enamel hypoplasia.

3.1.6.4 Other conditions

Diseases related to vitamin deficiencies like rickets experienced in perinates or lactating children can reflect vitamin deficiencies in the mother (Schoenmakers et al., 2016). During pregnancy the body experiences major changes in vitamin D metabolism and becomes important for immunomodulation of the maternal-fetal interface (Pilz et al., 2018). The effects of vitamin D deficiency can be observed in multiple instances. This includes pregnancy, childbirth, and lactation. In the first, vitamin D deficiency can lead to adverse gestational outcomes such as pre-eclampsia and gestational diabetes (Palacios et al., 2016; Pilz et al., 2018). During childbirth, the effects of severe osteomalacia in the pelvis may cause deformations affecting the pubic symphyses, pubic rami, ilium and sacrum which may result in complications during vaginal birth (Brickley et al., 2005; Brickley and Ives, 2008*a*). Finally, vitamin D deficiency in lactation can affect both mother and baby. The vitamin concentration in breast milk is approximately 20%, making it very difficult for a child to receive the required units from it alone (Pilz et al., 2018). However, clinical studies have shown that women who received vitamin D supplements during pregnancy had babies with increased mean birth weight and reduced the risk of SGA age as well as reduced risk of asthma or recurrent/persistent wheeze for the first three years of the life of the baby (Roth et al., 2017). This shows that although breast milk is not the only way for infants to receive vitamin D, a deficiency in vitamin D during the lactation period can have negative consequences on the child, making it more likely for the child to develop rickets (Schoenmakers et al., 2016). This is particularly relevant for archaeological contexts, where access to vitamin supplements was not possible.

3.2 Stress during adolescent growth

Adolescence is defined by the WHO as between the ages 10 and 19 years and refers to the period of transition between childhood and adulthood (World Health Organization, 2014*b*). This overlaps with the category of youth of the United Nations defined as between the ages of 15 and 24 years by the United Nations Secretary General (1981). The beginning of adolescence is marked by the start of the onset of puberty, while the ending of youth is defined by the full maturation of the brain (Blum et al., 2012; Rogol et al., 2002). This period of transition is not limited to biological changes but also includes psychological, social and cultural changes in relation to the role of the individual in society, their activities and relationships (Patton and Viner, 2007; Resnick et al., 2012). For this reason, many societies have different rites of passage and criteria to define when someone reaches adulthood (Alsaker, 1996). From a biological perspective, adolescence begins with the onset of puberty (Bogin, 2015). This is defined as a dynamic period of development marked by brain maturation, rapid changes in body size, shape, and composition (Patton and Viner, 2007; Rogol et al., 2002). These changes are characterised by the development of secondary sexual characteristics and sexual maturity and therefore are different for men and women (Abbassi, 1998).

Growth experienced during puberty is described as one of the most rapid phases of human development. During this time, average weight doubles and height increases by more than 15%. This growth, referred to in many cases as a “growth spurt”, increases nutritional requirements for both men and women. This makes them more susceptible to nutritional deficiencies, which subsequently impacts their development (Shetty, 2010; World Health Organization, 1993). Additionally, during this period the immune system is still developing, which makes adolescents more susceptible to opportunistic infections and other pathologies related to immunosuppression. Stress can also be exacerbated by changes in lifestyle related to the inclusion of the young person into new environments such as the workforce, or becoming pregnant (Lewis et al., 2016; Shetty, 2010).

Stress, chronic illness, and undernutrition due to adverse dietary and environmental conditions are known to cause pubertal delay (Patton and Viner, 2007). This is observed as stunting when the growth spurt is delayed or stopped. In women, it is associated with delayed menarche (Belachew et al., 2011; Galler and Barrett, 2001; Lewis et al., 2015). Similarly to childhood stress, pubertal delay must not be considered in isolation, but related to other stages of development. Maternal and childhood stress can affect pubertal development, which also has an impact on adulthood (Gowland, 2015; Lewis et al., 2015) (Figure 3.2). It is important to identify stress experienced during these critical periods of development in order to fully understand their impact on population health. Furthermore, the environment in which a person inhabits, both natural and cultural, is fundamental to properly understand pubertal development and delay. This is especially important when looking at past populations. Pubertal development has been addressed by multiple studies from diverse disciplines. Currently, the World Health Organization has an ongoing global project called “Health for the World’s Adolescents. A second chance in the second decade” based on reports from the past two decades (World Health Organization, 2014*b*). However, when looking at past populations it is important not to make direct analogies between past and present groups. Environmental stressors, standard ages for the onset of puberty, and cultural norms and expectations experience changes through time and space (Patton and Viner, 2007).

In the medieval period, adolescence was also defined from a biological, social and legal perspective. The category most widely used during the medieval period to describe adolescence was described by Isidore de Sevilla (c.560-636) who divided the life cycle into five categories that were communicated to the population through religious sermons, art and architecture (Augenti and Gilchrist, 2011). The category of *adolescentia* was defined between the ages of 14 and 28 years of age. However, it is important to consider this category did not necessarily reflect social practices (Goldberg, 2006; Mawhinney, 2015) and may vary through the medieval period. For example, poll tax records only accounted for people over the age of 12-14 years (Benedictow, 2004; Lewis et al., 2016), defining individuals under that age as children. However, after the Black Death children may have started working as young as 6 years old (Lewis et al., 2016).

Bioarchaeological studies of populations from London during the post-medieval period showed a relationship between environmental stressors and delayed pubertal development (Lewis et al., 2015). According to the written record, the age of the onset of puberty during the medieval period was around 13 years of age, with women having menarche around 14 years of age (Soranus of Ephesus, 1991). However, it is possible that in periods of crisis, especially the period leading to the Black Death, the age of onset of puberty was different. This was observed by DeWitte and Lewis (2021) in London. The authors looked at 74 individuals dated before and after the Black Death and observed a rise in the mean age of post-menarche age during the period leading to the Black Death (AD 1200-1250).

The end of adolescence and the start of adulthood were also not constant during the medieval period, as they varied according to class, profession and gender. From a legal perspective, adulthood could be reached between the ages of 21 and 24. However, apprenticeships could extend this period until 25 years of age (Lewis, 2016*b*). From a cultural perspective, the age for different stages of life was also variable. For example, the age of marriage similarly changed in relation to period, system of subsistence, gender and socioeconomic position. These changes both reflected and had an impact on fertility, demography and population health. There is much discussion about the age of marriage during the medieval period. Initially, it was thought that marriage was common for females after menarche (14 years) (Soranus of Ephesus, 1991). However, subsequent studies have estimated the age of marriage to have been later. Although high socioeconomic classes might have married before the age of 17 years, to maximise the amount of children peasants were likely to marry at around 24-28 years (Benedictow, 2012). The age of marriage for peasants in pre-industrial societies was defined by the death of the landholder and the ability to acquire land (Bailey, 1996). For this reason, it is to be expected for the average age of marriage to have risen in the wake of the Black Death, as most people struggled to make a living (Benedictow, 2012).

3.3 Oral health

Dental caries is one of the most common forms of infection (Hicks et al., 2003). It refers to a disease process characterised by the focal demineralisation of the enamel, dentine and cement by organic acids (Larsen, 1997). It is considered a multifactorial disease (Turner, 1979; Ferraro and Vieira, 2010), and can be related to age, oral hygiene (Hillson, 2005), food preparation (Molleson et al., 1993), and immunity (Leone and Oppenheim, 2001), as well as other oral pathologies like periodontitis (Otani et al., 2008), antemortem tooth loss (Cucina and Tiesler, 2003), and abscesses (Hillson, 2005).

Even though there are many indicators of oral health, dental caries have the strongest and most direct relationship with diet (Larsen, 1995; Turner, 1979). Foods that are most cariogenic are the ones with high sugar. High carbohydrate foodstuffs containing, for example, starch, have also been linked to the formation of caries (Hillson, 2005; Tayles et al., 2000). In terms of texture of the food, the consumption of soft edibles can result in less salivation during mastication which can lead to the formation of dental caries (Tayles et al., 2000). During the medieval period, there is evidence that that access to food resources and diet changed, particularly after the Black Death (Bailey, 2021; Müller, 2010). Frequencies of dental caries can provide information about changes in diet after the Black Death such as a the incorporation of more cariogenic foods like breads made from refined flour, and foods with a higher content of sugar (e.g. ale), as well as differences between females and males.

Poor oral health can also have an impact on health. In adults, evidence of tooth decay has been linked to higher frequencies of cardiovascular disease (Joshy et al., 2016). Studies of past populations like DeWitte and Bekvalac (2010) found that individuals from St Mary Graces, London with poor oral health had higher risks of mortality than individuals without evidence of periodontal disease and dental caries. However, this analysis was only based on adults from one London site dated to the post-Black Death period.

In children, dental caries in deciduous dentition can provide information about early life diet, reflecting the access to resources the parents had (Cameron et al., 2006; Rohnbogner and Lewis, 2016) and potentially changes in diet through time (Halcrow et al., 2013). Deciduous tooth decay can also affect the development of permanent dentition (Skeie et al., 2006). In relation to health, severe tooth decay may cause pain, affecting the ability of a child to eat, resulting in lower weight and shorter height (Sheiham, 2006). It is also related to respiratory infections (Mehtonen et al., 2019), immunosuppression (Rajonson et al., 2017), and nutritional deficiencies like scurvy (Planerova et al., 2017).

4 | Materials and methods

4.1 Materials

4.1.1 Introduction

Human skeletal remains excavated from London and extra-London sites in England (Figure 4.1), dated between AD 900 and 1550, were analysed for evidence of childhood physiological stress. These sites are:

- Wharram Percy (Yorkshire).
- St Oswald's Priory (Gloucestershire).
- St Anne's Charterhouse (Coventry).
- Barton Upon Humber (Lincolnshire).
- Stoke Quay (Ipswich).
- East Smithfield (London).
- Guildhall Yard (London).
- Merton Priory (London).
- St Mary Graces (London).

The criteria for selecting sites was based on primarily dating which allowed differentiating between pre and post-Black Death periods, availability of osteological material of individuals under the age of 35 years for primary analysis by the author or secondary data that included analyses of childhood stress and adolescent growth.

Data were derived both from primary analysis of osteological material by the author, and using secondary databases of previous analyses from published skeletal reports, unpublished 'grey' literature and online databases (see sections 4.1.2 and 4.1.3). As the main objective of this investigation was the analysis of health during growth and development, rather than any accumulative health effects during adulthood, the sample was limited to individuals from intra-uterine life to 35 years of age. The definition of youth, both in current and medieval populations, includes individuals up to 25 years (World Health Organization, 2014*b*; Lewis, 2016*b*). However, in times of crisis, nutritional stress can cause delays in growth and maturation beyond this period (Barrett et al., 2001; Galler and Barrett, 2001; Lewis et al., 2016). This cut-off also allowed an examination of females of reproductive age, which may have impacted the health of the perinates and young children (Gowland, 2015).

To evaluate health in the period leading to the Black Death, the sample was divided into three subsamples based on previous investigations like Lewis (2016*b*). These period were: pre-Black Death, referring to the period previous to the Black Death :

1. **Pre-Black Death (Pre-BD):** dated between AD 900 and 1348 and referring to the period previous to the Black Death.
2. **Black Death (BD):** plague pits dated between AD 1348 and 1350 referring to the period during the Black Death.
3. **Post-Black Death (Post-BD):** dated between AD 1350 and 1550 referring to the period during the medieval period after the Black Death.

When possible, the pre-Black Death period was further divided into early pre-Black Death (AD 900-1100) and late pre-Black Death (AD 1100-1348) subperiod. The criterion for the subperiod classification is based on historical analyses (Hatcher, 1977; Dyer, 1994, 2002; Benedictow, 2004), which discriminate between a period stability during the early period, and a period of expansion followed by retraction and crisis during the late-pre-Black Death period, as well as paleodemographic studies for populations in London (DeWitte, 2010, 2017) which found a decline in health in the period leading to the Black Death.

Evaluation of childhood health in the period leading to the Black Death (Late pre-Black Death) was done by comparing indicators of health status the early and late pre- Black Death subperiods. This analysis was complemented with the additional comparison between pre- and post-Black Death subperiods.

The following map (Figure 4.1) shows the analysed sites. The primary criteria for the selection of the sites were location and period. For this reason, only sites with skeletal remains dated before and after the Black Death were chosen. Special attention was placed on sites with remains dated after the Black Death, or that could be classified within the early or late pre-Black Death subperiods. Additional criteria for site selection was the information available of the site. Sites with more contextual evidence available such stable isotopes studies like Farber (2020) for Stoke Quay and (Fuller et al., 2003; Richards et al., 2002) for Wharram Percy, aDNA identification of pathologies like Mays et al. (2001) for Wharram Percy, as well as sites with evidence of poverty like St Oswald's Priory (Bryant and Heighway, 2003), Stoke Quay (Rielly, 2020), Barton-Upon-Humber (Walter and DeWitte, 2017), which which may reflect stress experienced before the Black Death were prioritised.

The selection of sites was also based on availability of skeletal material for primary analysis. Due to the COVID-19 pandemic, part of this primary analysis was possible and was supplemented with available secondary data.

Figure 4.1: *Location of the primary and secondary study sites*

4.1.2 Primary data

4.1.2.1 Wharram Percy, Yorkshire

Wharram Percy was a rural village located in the valley near the northwest scamp of the Yorkshire Wolds (Harding and Wrathmell, 2007). The settlement held two manors (North and South Manor areas) (Smith, 2009). Skeletal remains analysed in this investigation came from the village's churchyard. There is much discussion about the exact occupation of the manors and church (Smith, 2009). However, the latter was thought to have been founded in AD 950. Individuals buried at Wharram Percy lived not only in the Wharram Percy community but also in surrounding rural areas including Rainsthorpe, Thixendale, Burdale and Towthorpe (Harding and Wrathmell, 2007). While the site was abandoned in the 16th century, when the Hilton family who owned drove out the residents to make space for sheep farming which was becoming more lucrative (Price et al., 2011), the church continued its use until the mid-20th century (Harding and Wrathmell, 2007). The manor was owned by the Percy family until AD 1403 when it became the property of the Hilton family (Smith, 2009). Although the manor was owned by the Percy family, Smith (2009) notes that during the 400 years of occupation of the site, the Percy family was mostly absent from the settlement. They were unlikely to have lived in the village and to have been buried in the churchyard. This would have had an impact on the control and relationship the peasants living in the region had with their lords.

Although Wharram Percy was a rural community, trading, as well as its location 32 km north-east from York, exposed it to similar pathogens as urban settings (Lewis and Gowland, 2007). This likely resulted in the population experiencing stress, especially in the late medieval period. This was observed in previous osteological analyses which detected individuals with tuberculosis, and children with evidence of rickets, more often observed in urban settings (Mays et al., 2007).

The churchyard was excavated numerous times from 1950 to 1990 (Marlow-Mann and Wrathmell, 2012), with some additional excavations in 2005 (Roskams, 2012). The site has been the subject of numerous investigations including the Wharram Percy Project funded by English Heritage (Wrathmell, 2022). The skeletal remains are currently curated by Historic England in Portsmouth, England. During excavation and following radiocarbon dating, the burials were divided into several phases listed in the excavation report: phase 1: AD 950-1066 (late Anglo-Saxon), phase 2: AD 1066-1348 (earlier medieval), phase 3: AD 1348-1540 (later medieval), phase 4: AD 1540-1850 (post-medieval) (Mays et al., 2007).

Although phases are differentiated, many individuals were dated to more than one phase. This analysis focused on 123 individuals dated to the pre- or post-Black Death periods (phases 1, 2 and 3). The resulting sample was mostly concentrated in phase one and two with 120 individuals. Individuals from the Wharram Percy site represents 42.7% of the primary sample and 9.2% of the entire extra-London sample.

Previous research into the Wharram Percy collection included previous bioarchaological studies of health like Lewis et al. (1995); Lewis (2002*a*), stable isotope analyses using teeth and ribs of adults and non-adults (Britton et al., 2015; Fuller et al., 2003; Richards et al., 2002) provide information od diet and weaning practices, and aDNA analysis to identify the presence of the bacterium *M.Tuberculosis* (Mays et al., 2001). These studies were considered when interpreting results from the primary osteological analysis.

4.1.2.2 St Anne's Charterhouse, Coventry

St Anne's Charterhouse is a Carthusian monastery located in Coventry and founded in AD 1381 initially by William, Lord Zouch of Harringworth and then by Richard II in AD 1385. The site was excavated between 1965-87 by Brian Hobley, and Margaret Rylatt with the assistance of Michael Stokes and Iain Soden (Soden and Rylatt, 1995). After the dissolution of the monasteries in AD 1539, most of the buildings were demolished, with only one stone building remaining still (Citron, 2018). The burials were dated through stratigraphy and dated finds. The site was divided into four phases based on the construction of the monastery:

phase I: constructed in 1385; phase II: constructed by AD 1417; phase III: constructed by AD 1450, and phase IV: constructed by 1475. Some burials were dated to more than one period. However, for the scope of this investigation, all burials were categorised as belonging to the post-Black Death period (Citron, 2018; Soden and Rylatt, 1995). This site constituted the smallest collection analysed (n=15) and represented the majority of the post-Black Death sample outside London.

There are very few records from the charterhouse. The available data indicates that the monastery had no land of its own and depended on benefactors to survive. This resulted in instability as the manor where the church was built changed owners multiple times (Soden and Rylatt, 1995).

The socioeconomic status of the residents of the church is also difficult to establish. Although Carthusian monks are generally educated and from the higher classes, most of the monks in St. Anne's Charterhouse were likely uneducated and recruited from merchants and middle-class families from the local urban area (Soden and Rylatt, 1995). This is further supported by evidence of financial struggles by the church, which records show had been on multiple occasions on the verge of bankruptcy (Soden and Rylatt, 1995).

Although it is unclear who is buried in its cemetery, there are records of a few notable individuals graves. However, there is no evidence that any of the excavated remains belong to them. The pattern of burials indicates the possibility of reburials and family members being buried together (Soden and Rylatt, 1995). This indicates that it is likely that individuals from outside of the clergy were buried in the cemetery as well.

Although many individuals may have come from wealthier backgrounds, the struggles of the church may have affected their health. Furthermore, monks may have been subjected to strict diets which included vegetables and fish (Müller, 2010). This may have also affected their health. There is no evidence of how strictly this diet was followed. It was indicated that towards the late medieval period diet restrictions were relaxed (Müller, 2010). Lastly, there is evidence of the church producing ale (Soden and Rylatt, 1995). However, it cannot be determined if it was consumed by its residents.

Towards the end of the 15th century (AD 1478-1479), there was an outbreak of disease that affected the charterhouse. This resulted in its residents receiving charity from local people (Soden and Rylatt, 1995). The struggling situation of St Anne's Charterhouse shows how the post-Black Death period was experienced differently in areas outside London (Bailey, 2021) and may have negatively affected the health of it inhabitants.

4.1.2.3 St Oswald's Priory, Gloucester

St Oswald's Priory was a medieval church and burial ground located Northwest of Gloucester. The church was one of three religious institutions used by the town's population which also included St. Peter's Abbey and Llanthony Secunda Priory (Lewis, 2013). The site was occupied for an extended time from the Roman period to up to AD 1855. The church was initially founded in AD 679 and dedicated to St Peter (Heighway et al., 1999), and by AD 909 it was rededicated to St Oswald (Lewis, 2013). It was later destroyed in AD 1750 (Heighway et al., 1999). During this time it experienced multiple changes and expansions. The church was initially wealthy, as it had Royal connections and was known to keep a lot of gold and relics (Lewis, 2013). Its parish was partially under the jurisdiction of St. Peter's Abbey (Bryant and Heighway, 2003). However, by the 11th century, the church became poor, losing status and becoming overshadowed by the expansion of the neighbouring St Peter's Abbey (Bryant and Heighway, 2003). This may have been reflected in the population buried at the site and analysed in this investigation.

The site was subjected to numerous excavations between 1967 and 1983 (Bryant and Heighway, 2003; Lewis, 2013). The collection included 487 burials and at least 500 skeletons. Skeletal remains were dated as follows (from Heighway et al. (1999): Roman (4th century), Anglo-Saxon (c900-1120), Norman (1120-1230), late medieval (1230-1540), post-medieval (1540-1855).

The study sample comprised 150 individuals dated through radiocarbon and type of burials the Anglo-Saxon and Norman periods (Heighway et al., 1999), or the early and late pre-Black Death subperiods respectively. Individuals from the late medieval period (1230-1540) were not included in this analysis as it was not possible to differentiate between who was buried before and after the Black Death (AD 1348-1350). Individuals from St Oswalds's Priory represented 52.1% of the primary sample and 11.2% of the overall sample for the extra-London region.

4.1.3 Secondary data

To make the analysis more representative, and due to limitations in primary data collection resulting from the lockdown in 2020-21, secondary osteological evidence from previous analyses was included in the investigation. This included data from published and unpublished skeletal reports, research articles, and for London sites, the Wellcome Osteological Database Museum of London Archaeological Archive (Centre for Human Bioarchaeology, 2020). Datasets were also generously provided by Dr Rebecca Watts (UCL), as well as Mary Lewis and Fiona Shapland (University of Reading).

4.1.3.1 Extra-London region

4.1.3.1.1 Barton-upon-Humber, Lincolnshire

Barton-upon-Humber was a small market town located in northern Lincolnshire on the Southern bank of river Humber (Watts, 2013). The site was occupied from the early medieval to the post-medieval period (Figure 4.1). The community had two churches, St Mary's and St Peter's churchyard(Waldron, 2007). The human remains analysed in this thesis came from St Peter's Church.

Individuals buried at St Peter's Church were residents of the town and its surrounding areas.

Barton was considered a rural community (Walter and DeWitte, 2017). Although there is evidence that its inhabitants had multiple occupations related to trade and were in some instances wealthy (McGuire, 2020; Watts, 2013), the community was described by 15th century accounts as poor (Watts, 2013) and most of its residents were labourers involved in agriculture (Waldron, 2007; Watts, 2013). Previous osteological analyses have found evidence of stress reflecting poor nutrition and growth delay (Gilchrist, 2012; Waldron, 2007; Walter and DeWitte, 2017; Watts, 2013).

This churchyard has been the subject of multiple excavations, starting in 1912. However, the most significant excavations were between 1978 and 1984 (Waldron, 2007). Burials were divided into five phases (A-E) spanning AD 950-1855 based on radiocarbon dating Waldron (2007). The analysis in this thesis was based on phases E (AD 950-1150) and D (AD 1150-1300). Although phase C (AD 1300-1500) could have included individuals relevant to this research, it was impossible to distinguish between the pre- and post-Black Death burials and these data were excluded. Secondary data for this site was compiled from multiple sources. The excavation report provided the demographic composition of the sample, the crude prevalence of pathologies, dental conditions and cribra orbitalia (for non-adults) (Waldron, 2007). Watts (2013)'s doctoral thesis provided information about vertebral neural canal (VNC) dimensions, cribra orbitalia, and femoral length for adults. Lewis provided information on puberty stages for individuals aged 10 to 25 years (Shapland and Lewis, 2013, 2014; Lewis et al., 2016). The final study sample was comprised of 522 individuals and represented 38.9% of the extra-London sample.

4.1.3.1.2 Stoke Quay, Ipswich

Stoke Quay is a site located in Ipswich (Figure 4.1). The site was excavated multiple times since 1958. The latest excavation, which resulted in the osteological collection used in this thesis, was done by Oxford Archaeology in 2012 (Brown et al., 2020). The settlement was used from the Anglo-Saxon to the 19th century.

Stoke Quay was an urban site located south of the River Orwell. The site was characterised by the presence of St Augustine's church (Rielly, 2020). Many of its residents participated in the North Sea trading area and likely consumed marine foods. This was confirmed by recent carbon and nitrogen stable isotope analysis (Farber et al., 2017)

The area where the site was located was considered poor (Rielly, 2020), which likely resulted in poor housing and sanitary conditions. This was observed in recent studies which found evidence of intestinal helminth parasites in remains dated to the early medieval period indicating the prevalence of faecal–oral infections (Ryan et al., 2022). These infections likely had an impact on the health and development of its inhabitants (Lewis, 2018). The struggles of the inhabitants of Stoke Quay were further confirmed by previous osteological analyses, which found evidence of pathology in non-adults and adults related to nutritional stress (Brown et al., 2020).

The site was divided into 9 burial phases spanning the prehistoric to the nineteenth century. Data were compiled for individuals from burial phases 4 and 5 (9-12th centuries). This was composed of remains excavated from the medieval church of St Augustine's located at the site. All data used in this thesis came from Oxford archaeology (Brown et al., 2020). These data included frequencies of pathology, oral health and osteometrics in adults. Pubertal growth and VNC dimensions were not recorded at this site. The total sample of individuals under 35 years of age included in this analysis was 531. and represented 39.6% of the total extra-London sample.

4.1.3.2 London region

4.1.3.2.1 Guildhall Yard Cemetery

This site was a lay cemetery and was excavated between 1992 and 1997 by the Museum of London Archaeological Service. From the excavation, 60 individuals were recovered (Centre for Human Bioarchaeology, 2022). The site was divided into two burial phases: AD 1050-1140 and 1141-1230, allowing individuals buried in the early and late pre-Black Death periods to be identified. The study sample comprised 37 individuals under 35 years of age, 10 dated between AD 1050 and 1140 (early pre-Black Death) and 27 dated between AD 1140 and 1230 (late pre-Black Death), were included in the analysis. Individuals from this site represented 5.1% of the total sample from the London region. Data from this site was obtained from Centre for Human Bioarchaeology (2020) and Watts (2013).

4.1.3.2.2 East Smithfield Black Death Cemetery

This site was part of the Old Royal Mint Court. The cemetery was built to bury victims of the Black Death (AD 1348-1350). The excavation took place between 1986 and 1988 (Grainger et al., 2008). The Wellcome Osteological Database currently has 636 individuals registered (Centre for Human Bioarchaeology, 2020).

Due to the specific purpose of the site, it was the one with the most narrow dates (AD 1348-1350). It was the only collection with individuals dated specifically to the pandemic, which allowed further comparisons within the London region.

The population buried at this site also represents one of the most significant groups in this thesis's analysis. The population from this cemetery not only represents the individuals who died during the Black Death in London but also the people who lived or directly descended from people who lived in the period leading to the Black Death. The period of acute crisis before the pandemic (AD 1290-1348) (Dyer, 1994, 2002) is mostly absent from the osteological record. This represents a problem that has been observed in previous investigations (DeWitte and Lewis, 2021). The population buried at the cemetery does not only represent people who died during the Black Death, but also people who lived or directly descended from who were affected and survived the acute crisis period which included other catastrophic events like the Great Famine (AD 1315-1322) (Schofield, 2013). It is expected for this people to show higher levels os stress resulting from experiencing difficulties accessing resources and a greater vulnerability to disease (Benedictow, 2004).

It is important to consider that the composition of this site is unique and reflects a catastrophic context. Black Death cemeteries were not common in England. Most plague victims were buried alongside people who died of other causes. For this reason, it is not possible to compare this directly with an extra-London Black Death cemetery. This was taken into consideration when interpreting results.

The total sample of individuals under the age of 35 years included in this analysis was 400. Individuals from this site represented the only sample dated to the Black Death period and constituted 55.2% of the sample from the London region. Data from this site was obtained from Centre for Human Bioarchaeology (2020) and Watts (2013).

4.1.3.2.3 Merton Priory

This site was an Augustinian Priory. It was excavated between 1977 and 1983 by the Greater London Archaeology Advisory Service and later by the Museum of London Archaeological Service (Centre for Human Bioarchaeology, 2020). Four burial phases were identified: AD 1117-1222, AD 1222-1300, AD 1300-1390, and AD 1390-1536, allowing individuals to be divided into the study periods under investigation. The total sample comprised 676 individuals; 51 individuals were dated to the pre-Black Death period with the remaining 628 dating to the post-Black Death period. Individuals from this site represented 7% of the sample from the London region. Data was compiled from Centre for Human Bioarchaeology (2020) and Watts (2013).

4.1.3.2.4 St Mary Graces

This site was a burial ground associated with the Cistercian Abbey of St Mary Graces and was part of the Royal Mint (Centre for Human Bioarchaeology, 2020). It was excavated between 1986 and 1988 and was dated to after the Black Death. The excavation resulted in 420 burials from which 389 individuals were recuperated. Two burial phases were identified:

AD 1350-1400 and AD 1400-1536, with 196 burials classified as occurring during the post-Black Death period (Grainger and Phillpotts, 2011). This sample was the largest London post-Black Death sample in the study. Individuals from this site represented 27% of the total sample from the London region. Data from this site was compiled from Centre for Human Bioarchaeology (2022) and Watts(2013).

4.1.3.2.5 Spital Square

Spital Square was a large hospital located in London. It was first called St Mary without Bishopsgate and later St Mary Spital. It was dedicated to the care of the poor and sick (Thomas et al., 1997). The site was excavated between 1985 and 1989 by the Museum of London. The cemetery was in use from AD 40-1700 and it was divided into 11 burial phases. Due to maintenance of Wellcome database, only a portion of the dataset was available, which meant that only 40 individuals from this site from certain sectors were included in the study, dating from the late pre-Black Death period (Phases M2, M3 and M4). Individuals from this site represented 5.5% of the total sample from the London region. Data from this site was compiled from Centre for Human Bioarchaeology (2020).

4.2 Methods

4.2.1 Methodological framework

The human body is an open system in constant retroactive feedback with its environment, both “natural” and “cultural” (Goodman and Leatherman, 1998). For this reason, it is important to consider not only the biological changes observed in the osteological record but also the social, cultural, and natural environment in which the studied individual had lived and died to create a more comprehensive image of their health (McElroy, 1990). This investigation employed a multifactorial analysis based primarily on osteological analysis, which included not only multiple methods for the evaluation of nutritional stress in bones but also the integration of additional sources of evidence to contextualise and better interpret the osteological results. These sources were comprised of secondary evidence from previous osteological investigations, primary and secondary historical sources, archaeological and historical contextual evidence involving living conditions and mortuary contexts, as well as information about the natural environment and cultural practices.

4.2.2 Primary osteological analysis

4.2.2.1 Sex estimation

4.2.2.1.1 Non-adults

In this investigation, non-adults were defined as individuals under the age of 18 years. Sex estimation in non-adults is more complicated than in adults as secondary sexual characteristics are not well developed, resulting in reduced sexual dimorphism (Aris et al., 2018). Due to its accuracy being lower than for adults, many studies do not determine sex for non-adults. However, as this investigation includes analysis of pubertal development and delay, it is important to know the sex of non-adults. This allows being able to more accurately compare indicators of delayed puberty and other developmental pathologies between same-sex groups.

There have been several non-invasive approaches to estimating sex in non-adults. These included odontometrics (Aris et al., 2018), osteometrics of the post-cranium (Stull et al., 2017; Luna et al., 2017) and evaluation of morphological differences in the ilium (Schutkowski, 1993; Luna et al., 2017), mandible (Schutkowski, 1993) and humerus (Rogers, 2009) (see section, 3.1.2.2). Nevertheless, their accuracy is not as high as studies involving adults (Rogers, 2009; Schutkowski, 1993). In order to obtain more robust results, this investigation employed a combination of methods involving the analysis of the proximal humerus, the ilium, and the mandible. Sex was estimated by compiling results from the three areas. When all three methods agreed, sex was estimated as likely, when only two agreed, sex was classified as tentative, and if none agreed, sex was described as undetermined.

Accuracy for sex estimation in non-adults is highly dependable upon age, becoming progressively less reliable the younger the individuals is (Rogers, 2009). Independently, these methods would not be able to produce results with the same accuracy as sex estimation methods in adults. However, the combination of the three provided the possibility for more robust results. In this thesis, as secondary data mostly did not provide sex estimation for non-adults, sex estimation for non-adults was only used for analysis of pubertal status and menarche. As pubertal development varies between sexes, it is fundamental to discriminate between males and females for its analysis, particularly the study of menarche (DeWitte and Lewis, 2021). In this thesis, sex was estimated for non-adults over the age of ten, when onset puberty start (Lewis et al., 2016).

4.2.2.1.1.1 Distal humerus

The distal humerus has been used for estimation of sex in adolescents. The method was initially developed for adults by (Rogers, 1999) and the applied to adolescents by Rogers (2009). The latter study focussed on the variation in the humerus morphology based on four features of the distal posterior humerus that contribute to the carrying angle of the arm, which is approximately 10-15 degrees in males and 20-25 degrees in females (Rogers, 1999, 2009) (Figure 4.2).

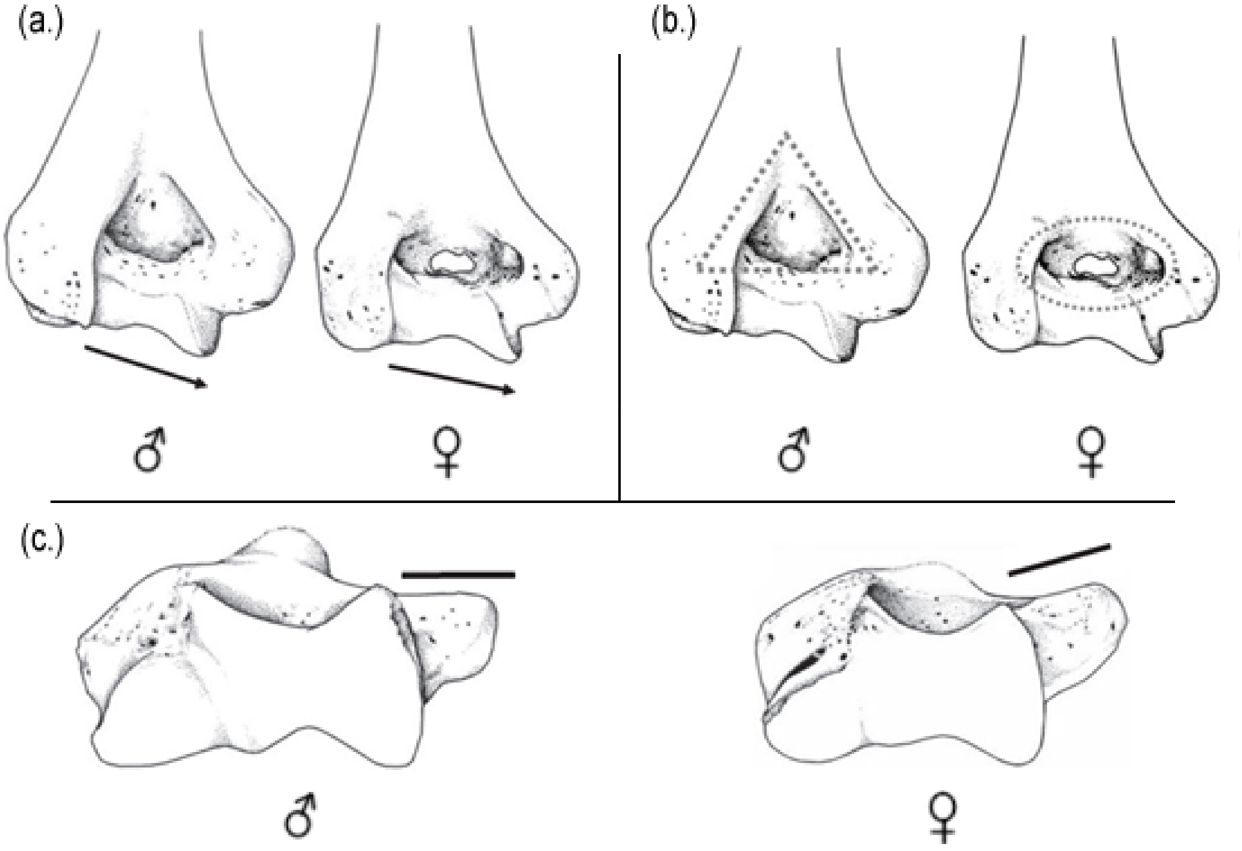


Figure 4.2: *Distal humerus method showing (a) trochlear symmetry and constriction, (b) olecranon fossa shape, and (c) angle of medial epicondyle for males and females adapted from Vance et al. (2011, p. 4).*

4.2.2.1.1.2 Ilium

The study of the ilium in non-adults has shown statistically significant morphological differences between males and females. In this investigation indicators from methods described by Schutkowski (1993); Weaver (1980), which were found to have a statistically significant difference between males and females in individuals over 3 months old (Irurita Olivares and Alemán Aguilera, 2016), were used. This method included observations of the angle and depth of the greater sciatic notch, as well as the elevation of the auricular surface. (Luna et al., 2021) also developed a method based on the observation of the auricular surface of the ilium. However, the method was developed based on a small collection (n=34) of a modern population from Coimbra, Portugal and has not been further tested. For this reason, it was not used in this investigation.

4.2.2.1.1.3 Mandible

The morphology of the mandible has been used to estimate sex in non-adults (Franklin et al., 2007; Schutkowski, 1993). In this investigation, each mandible was observed, and the protrusion of the chin region was classified as female, probably female, undetermined, probably male or male. Similarly to the criteria used for the ilia, this method was chosen due to being tested as having statistically significant among the traits observed by Schutkowski (1993) (Irurita Olivares and Alemán Aguilera, 2016) (Figure 4.3).

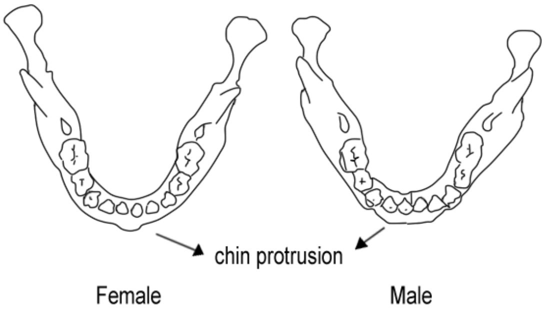


Figure 4.3: *Morphology of the mandible showing the differences in the protrusion of the chin between males and females adapted from Schutkowski (1993, p. 200).*

4.2.2.1.2 Adults

For the adults (18-35 years), sex was estimated based on morphological traits of the pelvis, and if necessary, the skull. The pelvis is known to be the most reliable feature to estimate sex in adults (Buikstra and Ubelaker, 1994) due to reproductive and hormonal factors (Sinhorini et al., 2019). However, in many cases, the pelvis is not available and the skull is used to estimate sex (Buikstra and Ubelaker, 1994). Although the morphology of the skull remains stable in adulthood, authors like Walker (1995) have pointed out that skull changes can lead to interpreting gracile young men under the age of 30 as female and older, postmenopausal women over 45 as male. This problem is particularly prominent when looking at the glabella. As this investigation focuses on individuals under the age of 35 years, the first could potentially become a problem. However, a more recent investigation by Lesciotto and DeMars (2018) has tested the correlation between age and cranial traits in a sample of 272 African-American and European-American individuals aged 20–92 years from the Hamann-Todd Human Osteological Collection. The study found little impact of age on cranial trait expression, with the highest variance (5%) in relation to age observed in the mental eminence in African-American males.

4.2.2.2 Age estimation

4.2.2.2.1 Non-adults

In this investigation due to different methods being used, non-adults were separated in two categories for age estimation: perinates (to refer to individuals under 40 WIU (weeks *in utero*)) and non-adults, which refers to individuals between birth and 17 years of age.

4.2.2.2.1.1 Perinates

Perinates were aged based on dental development following the table developed by AlQahtani et al. (2010). Long bone diaphyseal length of the femur and humerus were aged as well based on charts developed by Kiserud et al. (2017). However, these were not used to age individuals but to compare osteometric aging to age estimated from dental record to identify *in utero* stress (see growth delay section, 4.2.2.4.1.1).

4.2.2.2.1.2 Non-adults (birth-17 years)

Similarly to perinates, age estimation in non-adults (birth-17 years) was primarily based on tooth formation and eruption utilising the London Atlas of Human Development and Eruption development by (AlQahtani et al., 2010). This atlas was developed based on radiographs of known-age individuals from collections held at the Royal College of Surgeons of England and the Natural History Museum, London, UK and known age-at-death skeletal remains from the Christ Church Spitalfields, London collection, and Moorrees et al. (1963*a*,*b*) stages of development. This atlas was shown to be more accurate than previous similar charts developed by Schour and Massler (1940*a*,*b*) and Ubelaker (1989) (AlQahtani et al., 2014).

If necessary, for age was further discriminated through ossification and epiphyseal fusion of long bones and vertebrae (Buikstra and Ubelaker, 1994) that excluded bones used for puberty stage estimation like the distal radius, proximal ulna and capitulum of the humerus (Lewis et al., 2015). To allow the primary and secondary data to be combined in the final analysis, broad age categories were used when considering the mortality profiles. Non-adults were placed into three age cohorts: birth-5 years, 6-12 years, and 13-17 years. To explore the infant and child mortality, those aged under 5 years, this group were further divided into one-year age categories based on dental development, up to 4.99 years. For the puberty stage assessment (see section 4.2.2.1.1), adolescents aged 10-25 years were analysed.

4.2.2.2.2 Adults

Age in adults (18-35 years) was estimated through morphological changes in the pubic symphysis based on standards described by Brooks and Suchey (1990), auricular surface based on descriptions made by Lovejoy et al. (1985), and sternal rib ends (İşcan et al., 1984). As this investigation is focused on individuals under the age of 35 years, special attention was placed on epiphyseal fusion that occurs in adulthood. However, similarly to age estimation in non-adults, indicators used to estimate pubertal delay such as the distal radius were not included in the estimation of age. Adults were primarily placed into 2 broad age categories: 18-25 years and 26-35 years representing young and middle-aged adults. Special attention was placed on the medial epiphyseal clavicle which fuses completely by approximately the age of 30 years (White and Folkens, 2005).

4.2.2.3 Puberty stage estimation

Nutritional stress and adverse environmental conditions can lead to pubertal delay as the body cannot support the biological requirements. For this reason, assessing pubertal age becomes fundamental to understanding childhood stress (Lewis et al., 2015, 2016). This investigation evaluated pubertal age and then compared it with average expected ages for pubertal development to evaluate delay. Puberty stage assessment was based on the methods outlined by Lewis et al. (2016); Shapland and Lewis (2013, 2014) and described below.

4.2.2.3.1 Peak Height Velocity (PHV)

Growth experienced during puberty is not uniform, but consists of a period of acceleration and deceleration. Peak height velocity refers to the maximum moment of growth velocity within the "growth spurt" characteristic of pubertal development (Abbassi, 1998). Assessing the stage of PHV can be used to estimate the stage of pubertal development of the skeleton. This analysis was based on the following indicators:

1. **Hook of hamate development:** classified according to standards described by Shapland and Lewis (2013) and go from stage G (hood absent) to stage I (hook complete) (Figure 4.4).
2. **Mandibular canine root development:** classified through developmental stages defined by Demirjian (Demirjian and Levesque, 1980). Stages E, F, G and H correspond to the periods from pre- to post-puberty respectively (Figure 4.5).

3. **Morphology of the cervical vertebral body:** C3-C5 are classified following a 6-stage scheme described by Shapland and Lewis (2014) (Figure 4.5).
4. **Hand phalanges fusion:** the proximal, middle and distal phalanges are classified as unfused (1), unfused (2), complete, equal with and capped (Lewis et al., 2015).
5. **Distal radius, proximal ulna and capitulum of the humerus fusion:** classified as unfused, fusing or complete (Lewis et al., 2015).

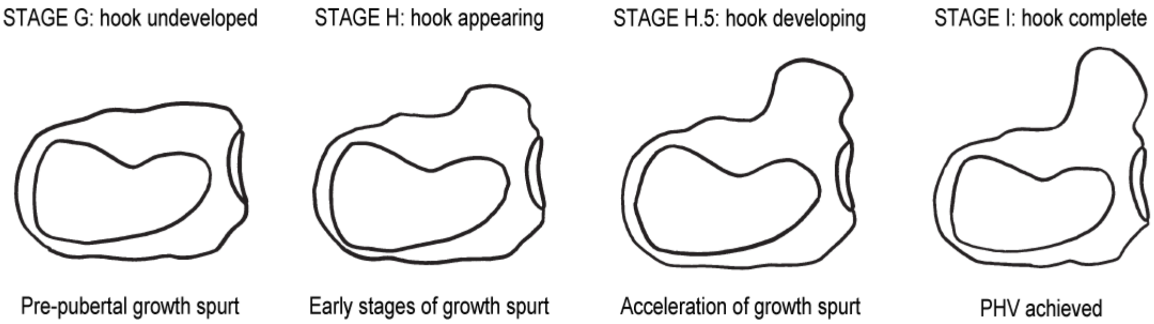
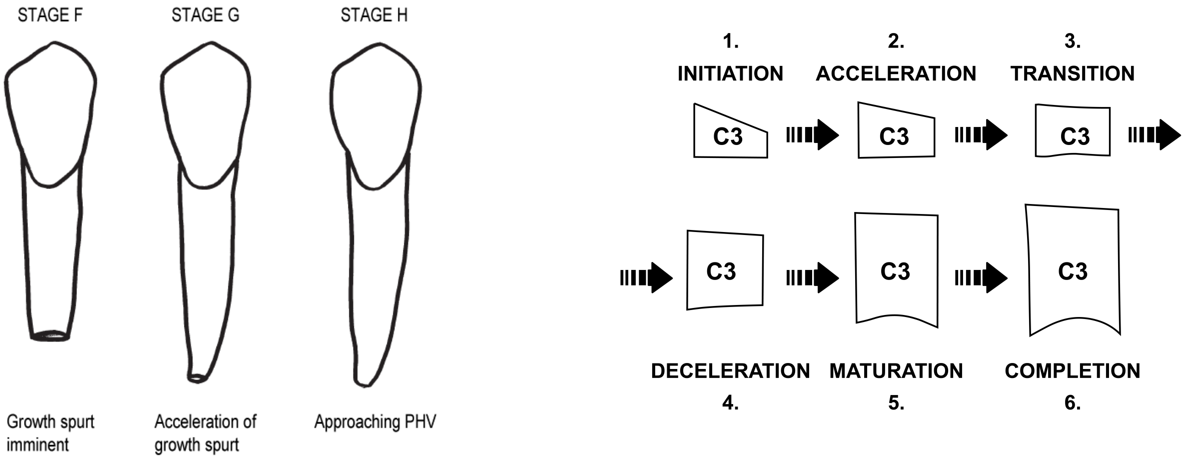


Figure 4.4: Relationship between the development of the hook of hamate and pubertal growth adapted from Shapland and Lewis (2013, p. 305)



(a)

(b)

Figure 4.5: *Relationship between mandibular canine development and pubertal growth (a) and the cervical vertebrae development and pubertal growth (b) adapted from Shapland and Lewis (2013, p. 305) and Shapland and Lewis (2014, p. 145) respectively.*

4.2.2.3.2 Menarche

In females, puberty is assessed through the estimation of whether menarche has occurred or not. For this purpose the following indicators were observed:

1. **Iliac crest:** classified as unfused, fusing, or complete (Lewis et al., 2015).
2. **Distal phalanx fusion:** preferably the distal phalanx of the second finger, or if unavailable, any distal hand phalange (Lewis et al., 2015).

The fusion of the iliac crest and distal phalanx of the second finger has been associated with menarche (Shapland and Lewis, 2013). This was observed in longitudinal clinical studies analysing pubertal development of 212 randomly selected Swedish children born between 1955 and 1958 who were examined yearly (Hägg and Taranger, 1982).

Average ages for pre-menarche and post-menarche females were estimated in order to evaluate trends between sub-periods. In this investigation, puberty was assessed for individuals between the ages of 10 and 35. This was due to two reasons: growth delay during periods of crisis being expected (Galler and Barrett, 2001), and previous bioarchaeological analyses of puberty signalling the presence of individuals up to 25 years of age still in their deceleration phase of the growth spurt at their time of death (Lewis et al., 2016).

4.2.2.4 Indicators of childhood stress

4.2.2.4.1 Growth delay

In this investigation, growth delay was assessed by taking osteometrics of the maximum length of diaphyses for non-adults and maximum long bone length for adults. Diaphyseal length was measured in complete bones with unfused epiphysis and maximum long bone length was measured when both of the epiphysis were fused. Bones with only one fused epiphysis were not considered.

4.2.2.4.1.1 Perinates

For this investigation, perinates were defined as individuals aged under 40WIU. This included foetuses, and infants born prematurely. Individuals over this age were included in the category 0-6 years. Similarly, for perinates, growth delay potentially related to intrauterine growth restriction was assessed by comparing age estimation based on dentition and long bone diaphyseal length of the femur and humerus (in mm) following the method used in Hodson (2018). For this purpose, femoral and humeral diaphyseal length was measured for both the right and left side. If both sides were present, the longest measurement was chosen as it was the most likely to be unaffected by pathology. Dental age was estimated using standards described by AlQahtani et al. (2010) and femoral and humeral ages estimation was done based on the World Health Organization Fetal Growth Charts (Kiserud et al., 2017).

Restricted growth affects long bone development more than dental growth. Individuals with age estimations based on osteometrics that are younger than the dental age were identified as having growth restrictions (Hodson, 2018). The number of individuals with restricted growth was compared between periods and regions.

4.2.2.4.1.2 Non-adults

In the case of individuals who had not undergone epiphyseal fusion, diaphyseal length was measured (in mm). The bones selected for analysis were the femur, tibia, humerus, and radius, including the left and right sides. If both sides were present, the longest measurement was chosen as it was the most likely to be unaffected by pathology. Growth delay was identified following the WHO Child Growth Standards, which identifies growth retardation when height and/or weight deviated more than two standard deviations (z-score <= -2) (Geber, 2014; WHO Multicentre Growth Reference Study Group, 2006*b*). In this investigation, growth delay was evaluated by comparing diaphyseal length to growth profiles developed by Maresh (1970) and corrected by Spake and Cardoso (2021). Diaphyseal lengths for these profiles were taken from children under the age of 12 years living in the United States between the years 1927 and 1967 (Maresh, 1970). Z-scores were estimated using R code developed by Spake and Cardoso (2021). The number of individuals identified as having growth delay (Z-score <=-2) was compared between periods/subperiods and regions. The growth profiles developed by (Maresh, 1970) were used as they were based on a well-documented sample and were subsequently corrected by (Spake and Cardoso, 2021) for bioarchaeological analysis. The purpose of this analysis is not to compare past and modern populations but to use the growth profiles as reference to assess differences in growth between two groups dated to different periods in the past.

4.2.2.4.1.3 Adults

The stature has been estimated in skeletal remains based on osteometrics. For example, Hauser et al. (2005) estimated the height of a modern Polish sample (n=91) using a formula that employs the maximum femur length. Mall et al. (2001) estimated stature by utilising linear regression analysis based on three measurements taken from the radius, ulna, and humerus. The study analysed a sample of 143 individuals from the Anatomical Institute in Munich and Cologne, Germany between and estimated stature correctly with an accuracy of 88.49%-94.93%. However, both studies were based on modern groups and the correlation between height and bone length varies between populations (White and Folkens, 2005). This results in error being introduced when the equations are used on a different population. For this reason, to minimise the bias, this investigation directly compared maximum long bone length.

Maximum long bone length measurements (in mm) were taken of the femur, tibia, radius, and ulna were taken for both the left and right sides. When both sides were present, the longest measurement was considered the more accurate. Results were compared between periods and subperiods to see if there were differences in growth between adults. As long bone osteometrics is different between males and females due to sexual dimorphism (Wells, 2007), the analysis was limited to sex-specific comparisons. Statistical analysis included independent t-tests comparing periods and regions.

4.2.2.4.2 Vertebral development and neural canal size

In this investigation, vertebral neural canal size (VNC) was estimated through standards described by Watts (2011, 2013, 2015), which measure the antero-posterior diameter and transversal diameter of L1-5. The antero-posterior and transverse diameter of the lumbar vertebra starts developing *in utero* and reaches adult size between the ages of 3 and 5 years for the former and 15 years for the latter respectively (Watts, 2015). This means that the analysis of neural vertebral canal growth can provide information about stress experienced at specific ages (Watts, 2013). Previous studies have found a correlations between a smaller vertebral neural canal, higher frequencies of stress, and lower life expectancy. This was observed most significantly for the transverse diameter (Newman and Gowland, 2015; Watts, 2011, 2013, 2015) (Figure 4.6).

In this study, antero-posterior and transversal diameters of the lumbar vertebrae were taken for individuals under the age of 35 years. In individuals who were too young to have experienced neurocentral synchondrosis fusion, only the transversal diameter was taken (Newman and Gowland, 2015). Lumbar vertebrae were chosen because their ossification occurs earlier, and they generally have better preservation (Watts, 2013). To reduce intra-observer error, each measurement was taken twice, and an average of the two measurements was used for the analysis (Watts, 2013). Statistical analyses included independent t-tests to compare VNC dimension means.

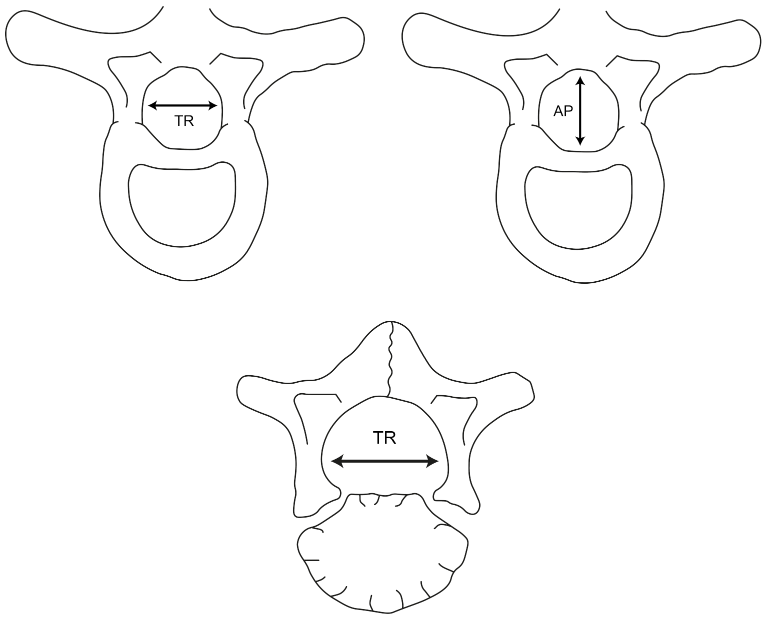


Figure 4.6: *Vertebral neural canal measurements adapted from Newman and Gowland (2015, p. 157)*

4.2.2.4.3 Cribra orbitalia

Orbital roof porosity is present not only in cases of cribra orbitalia, but also trauma, scurvy, and rickets. As the aetiology leading to each lesion is different, the nature of the lesion is different as well. Lesions from cribra orbitalia are characterised as pitting in an outward direction caused by marrow expansion (Brickley, 2018). This pitting is often crescent-shaped, symmetrical, and does not project above the lamellar surface (Klaus, 2017). The shape of the laminar bone is concave as there is no bone deposit related to haemorrhage. This is different to the porosity associated with scurvy where new bone formation results in a flat surface. Additionally, scurvy is associated with changes in other areas of the skeleton (i.e., maxillary and sphenoid bones) (Geber and Murphy, 2012). Therefore, lesions found in the orbits were also scored in the context of similar lesions found in the rest of the body (Klaus, 2017). Special attention was placed on "mixed" indicators, as these nutritional deficiencies can be experienced simultaneously (Brickley, 2018).

Cribra orbitalia was examined macroscopically. Each individual with at least one observable orbit was classified as presenting or not presenting evidence of cribra orbitalia. To better evaluate intragroup susceptibility and frailty, each lesion was graded using the 1-5 scoring scheme developed by Stuart-Macadam (1991):

1. **Stage 0:** Normal bone surface.
2. **Stage 1:** Capillary-like impressions on the bone.
3. **Stage 2:** Scattered fine foramina.
4. **Stage 3:** Large and small isolated foramina.
5. **Stage 4:** Foramina are linked into a trabecular structure.
6. **Stage 5:** Outgrown in trabecular form from the outer surface.

The first two stages represent very mild expressions of the condition that do not reflect periods of stress related to marrow hyperplasia. They are also more difficult to identify correctly and unlikely to be present in secondary data. For this reason, in this investigation, only stages 3 to 5 were considered. Although primary data analysis included both the presence and absence of cribra orbitalia, part of the secondary data only recorded the presence of the condition and did not differentiate between it absence or when the orbits where not available for analysis. For this reason, and to include the entire sample in the analysis, crude rates of cribra orbitalia were estimated.

4.2.2.4.4 Dental enamel hypoplasia (DEH)

Enamel hypoplastic defects are classified into three categories: furrow form, plane form, and pitform (Hillson, 2014). Many researchers include furrow and plane form defects under the label of linear enamel hypoplasia (Goodman and Rose, 1990). Considering the different types of defects present allows for more specific comparisons to be made that may relate to different aetiologies. In this investigation, DEH was identified through macroscopic observation. This recording technique was selected because it allows for primary results to be compared with secondary evidence from previous osteological analyses and it prevents over-recording defects (Hassett, 2014). This follows the indications of the Commission on Oral Health which stated that when the identification of DEH was unsure, it was preferable not to record defects to prevent over-recording them (Hillson, 2014). Both deciduous and permanent teeth were observed for evidence of enamel defects. Dental enamel hypoplasia was recorded in the maxillary canine and mandibular molars 1-3. These teeth were chosen because their development extends from birth to 15 years of age (Moorrees et al., 1963*a*,*b*), allowing researchers to observe stress experienced during a wider frame of time. Each tooth was macroscopically observed for defects and classified as present or absent, and each defect was then classified as furrow, plane or pitted following criteria described by Hillson (2014). For linear defects, age-at-formation of the defect was estimated. The ideal technique for this estimation is observing a ground section of the tooth with a scanning electron microscope (Seow et al., 2005). However, these techniques require the destruction of the tooth which is not plausible for a large sample. Among non-invasive methods, the decile chart based on histological analysis by Reid and Dean (2006) and Holt et al. (2012) is considered to be the most accurate. These charts are preferred over regression equations provided by Goodman and Rose (1990) because they take into consideration the non-linear growth of enamel as well as hidden cuspal enamel. Furthermore, the samples used to estimate tooth growth patterns, which can vary between different groups, included British populations pertinent to this analysis (Martin et al., 2008; Primeau et al., 2015). This investigation used the decile method based on charts developed by Reid and Dean (2000, 2006) and Holt et al. (2012) and modified by Primeau et al. (2015) to adjust for intra- and inter-observer error (Figure 4.7).

This method reduced intra- and inter-observer error. However, it has limitations concerning timing. Only destructive histological analysis allows for observing periods of stress shorter than five days, as well as stress which occurred before the age of two years. For this reason, in this investigation, only stress experienced over the age of two years that lasted more than five days was identified. Results were subsequently used exclusively for comparisons among subgroups subjected to the same methodological limitations. Although dental tissue does not remodel, it can be affected by dental wear which increases with age. For this reason, teeth with signs of wear corresponding with 6 or over according to the wear classification described by Smith (1984) are excluded from the sample. Similarly, if observation of DEH is not possible due to heavy calculus, or dental caries, the tooth was excluded from the analysis as well.

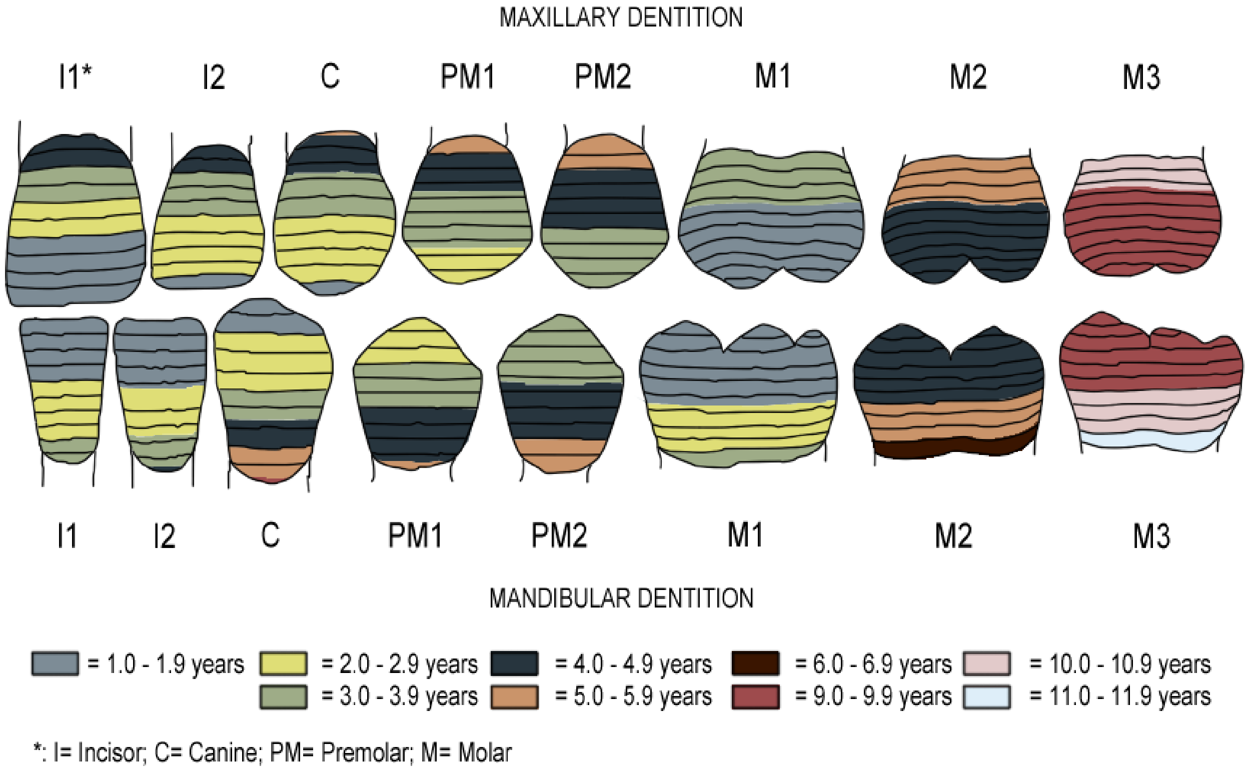


Figure 4.7: *Permanent dentition with deciles showed as coloured horizontal lines adapted from Primeau et al. (2015, p. 386)*

4.2.2.4.5 Spina bifida occulta

Spina bifida occulta is evaluated in the adolescent and adult osteological record, as the condition can only be properly diagnosed in individuals over the age of 15 years whose neural arches are fully fused. The condition was identified when several adjacent neural arches were found open (Lewis, 2018). On the other hand, as its survival rate is smaller, spina bifida aperta is only evaluated in perinatal and infant skeletal remains. However, unlike spina bifida occulta, it is much rarer and difficult to observe (Lewis, 2018). For this reason, more attention was placed on the identification of spina bifida occulta. Other congenital conditions such as anencephaly are rare in the archaeological record. However, if any congenital or specific infection not previously described is observed during osteological analysis, it is recorded as well. Although each pathology and condition listed before is independently identified, the analysis of each lesion and indicator was contextualised by considering other pathologies present in order to avoid over-recording (Rohnbogner and Lewis, 2017).

4.2.2.5 Vitamin deficiencies

4.2.2.5.1 Vitamin C deficiency

Vitamin C deficiency results in scurvy. In this investigation juvenile scurvy was identified following the standards described by Ortner et al. (1999); Ortner and Ericksen (1997). The authors analysed scurvy in non-adults through the presence of scorbutic changes related to haemorrhage in the following bone sections:

1. Cranial vault.
2. Sphenoid, greater wings.
3. Orbit, frontal (roof).
4. Orbit, zygomatic (lateral).
5. Infraorbital foramen.
6. Zygomatic bone, internal surface.
7. Maxilla, posterior surface.
8. Maxilla, alveolar process.
9. Maxilla, alveolar sockets.
10. Palate, process.
11. Mandible, coronoid process; medial surface.
12. Mandible, alveolar processes.
13. Mandible, alveolar sockets.

For adults, residual scurvy analysis was made following standards described by Geber and Murphy (2012), which included the presence of scorbutic changes related to haemorrhage in the following locations as definitive variables:

1. Sphenoid, greater wings.
2. Maxilla, posterior.
3. Palate, process.

4. Maxilla, alveolar process.
5. Mandible, alveolar process.
6. Mandible, coronoid process, medial surface.

For the analysis to be possible, the cranium and mandible must be present. Scurvy was identified as present when at least two of these indicators were found (Geber and Murphy, 2012). Special attention was placed on lesions found on the sphenoid bone as they are considered pathognomonic of the disease (Ortner et al., 1999). Special attention was placed on bilateral lesions on the greater wings of the sphenoid bone as they are considered pathognomonic of the condition (Ortner et al., 1999). In the case of non-adults, when looking at the orbits it was important to distinguish scurvy lesions from indicators of cribra orbitalia. Although both conditions present porosity over the orbital surfaces, scurvy is related to bone formation caused by increased vascularity, making blood vessels penetrate the underlying cortex (Ortner et al., 1999). This new bone formation is not observed in cribra orbitalia, where the porosity is related to red marrow hyperplasia (Stuart-Macadam, 1991).

4.2.2.5.2 Vitamin D deficiency

4.2.2.5.2.1 Rickets

Rickets in non-adults was identified by following the standards described in Brickley and Ives (2008*a*); Ortner and Mays (1998):

1. Mandibular ramus, abnormal medial/posterior bending and porosity.
2. Ribs, costochondral flaring.
3. Ribs, costochondral porosity.
4. Ilium concavity.
5. Leg bones, bowing.
6. Arm bones, bowing.
7. Coxa vara.
8. Long bone diaphysis thickening.
9. Long bone metaphysical flaring/cupping.

10. Long bone metaphysical porosity.
11. Growth plate, porosity.
12. Cranial vault porosity.
13. Orbital roof porosity (these lesions differ from the ones characterising cribra orbitalia and scurvy, as they are larger and more irregular.)

The first 11 indicators are considered indicative, while the last 2 are suggestive, as they can be more difficult to discriminate from similar lesions caused by other conditions such as scurvy and cribra orbitalia (Brickley et al., 2005). Individuals with at least 3 of the indicators were considered affected by the pathology (Watts and Valme, 2018). Rickets was observed in non-adults with unfused epiphyses who had the skull, ribs and wrists/ankles preserved (Lewis, 2002*b*). Similarly to cribra orbitalia, rickets can be observed in active and healed forms. Residual rickets can be present in individuals with fused epiphysis reflecting rickets suffered during formative years. Bowed limbs which followed patterns described by Brickley et al. (2010) were recorded in adults. Careful comparison of similar age cohorts was used to interpret the presence of rickets, residual rickets, and osteomalacia.

4.2.2.5.2.2 Osteomalacia

Identification of osteomalacia is more complicated as its expression is more subtle and it can be confused with other pathologies affecting mineralisation such as osteoporosis. In juveniles and adults osteomalacia was identified through standards described by Brickley et al. (2005):

1. Generalised softening and weakening of the skeleton resulting in bending deformation of the bones of the axial skeleton including the vertebrae, ribs and pelvis.
2. Looser's zones (pseudofractures) observed symmetrically on the superior and inferior pubic rami, the medial femoral neck and medial sub-trochanteric region, throughout the ribs, and the lateral border of the scapula reflecting bone fragility.

Skeletons were evaluated for osteomalacia when the vertebrae, long bones and pelvis were present. Special attention was placed on evidence found on the pelvis of women and they can provide information on obstetrical difficulties (Brickley et al., 2005).

4.2.2.6 Specific infectious diseases

4.2.2.6.1 Tuberculosis (TB)

Tuberculosis is a chronic infectious disease often related to nutritional stress and immunosuppression (Nelson and Wells, 2004). In this investigation TB was identified in non-adults following the standards described by Rohnbogner and Lewis (2017). This includes lytic lesions in the spine in combination with visceral rib lesions as well as widespread new bone formation, osteomyelitis of the mandible and the scapula, and joint involvement. Special attention was placed on lesions in the spine compatible with Pott's disease since it is the most common lesion associated with TB (Rohnbogner and Lewis, 2017; Turgut, 2001). Due to its relationship to immunosuppression (Pawlowski et al., 2012), tuberculosis can act as an opportunistic infection experienced during the course of other conditions. For example, individuals who experience deficiencies in vitamin D have been linked to tuberculosis and other upper respiratory infections. This is because vitamin D plays an important role in immunity, particularly phagocytosis and pathogen digestion (Snoddy et al., 2016). For this reason, particular attention is placed on the overlapping of markers related to nutritional deficiencies, particularly vitamin C and D, and indicators of tuberculosis. In adults, tuberculosis was identified by standards described by (Blondiaux et al., 2015; Santos and Roberts, 2006), which include: visceral surface of the ribs (RL), bone and joint lesions (BJL) and hypertrophic pulmonary osteopathy (HPO) (periosteal new bone deposition on the tubular bones) (Assis et al., 2011). For the analysis of tuberculosis in children, the spine and ribs needed to be preserved. For the analysis in adults, only the ribs must be present.

4.2.2.7 Oral Health

Each tooth, both deciduous and permanent were observed macroscopically. Dental caries were identified merely as present or absent to allow for comparisons between the primary and secondary data (Williamson, 2015).

4.3 Statistical analysis

The statistical analysis included a multi-method approach depending on the types of analysed data. All non-metric variables like cribra orbitalia, rickets, scurvy, tuberculosis, spina bifida, linear enamel hypoplasia presence, caries and periapical lesions were analysed using a non-parametric 2x2 Chi2 test. When analysis was required, but one of the entries was smaller than 5, a two-tailed Fisher's exact test was employed. For this testing, PAST Software was used (Hammer et al., 2009). Comparison between metric variables like VNC dimensions, pubertal stage and pre- and post-menarche ages, and long bone osteometrics of adults were compared using independent t-tests, which allowed comparing means between two groups. For these estimations SPSS 27 software was used. For non-adult diaphyseal length, z-scores were estimated using linear regression using charts developed Maresh (1970), corrected by Spake and Cardoso (2021). Estimations were made using R code developed and tested by Spake and Cardoso (2021).

5 Results

5.1 Primary and secondary data

The health status of 2065 individuals from the three time periods surrounding the Black Death were assessed using both primary data (n=288) collected during the study, and secondary data (n=1777) from published and unpublished sources (Table 5.1).

Data for the extra-London region, comprised primary data for 288 (21.5%) individuals from three sites (St Oswald's Priory, Wharram Percy, St Anne's Charterhouse) and secondary data for 1053 (78.5%) individuals from Barton-on-Humber and Stoke Quay (Brown et al., 2020; Waldron, 2007; Watts, 2013). Information on individuals from the London region was comprised entirely of secondary data compiled from the WORD database (Centre for Human Bioarchaeology, 2020) and Rebecca Watts (Watts, 2013).

The total study sample comprised 2065 individuals. Of these 69.5% (n=1436) dated to the pre-Black Death period, 19.4% (n=400/2065) dated to the Black Death, and 11.1% (n=229/2065) dated to the post-Black Death period. The uneven nature of the sample was more evident when the extra-London and London regions were compared. The London sample had a greater number of individuals dated to the post-Black Death period (n=210/724 or 29%) compared to the extra-London sites (n=19/1341 or 1.4%). This was the smallest subgroup of the sample. The London region also had the only subgroup dated to the Black Death period itself.

Table 5.1: *Composition of the study sample by period, site, and region.*

Region	Site	Pre-Black Death		Black Death		Post-Black Death		Total
		n	%	n	%	n	%	N
Extra-London	**Wharram Percy**	119	96.8	0		4	3.3	123
	St Oswald's Priory	150	100	0		0		150
	St Anne's Charterhouse	0	0	0		15	100	15
	Barton-upon-Humber	522	100	0		0		522
	Stoke Quay	531	100	0		0		531
	Subtotal	1322	98.6	0		19	1.4	1341
London	**Guildhall Yard**	37	100	0		0		37
	East Smithfield	0		400	100	0		400
	Merton Priory	37	72.6	0		14	27.5	51
	St Mary Graces	0		0		196	100	196
	Spital Square	40	100	0		0		40
	Subtotal	114	15.8	400	55.3	210	29	724
Total		1436	69.5	400	19.4	229	11.1	2065

The pre-Black Death period was divided into the early and late pre-Black Death period to allow any period of crisis leading to the Black Death to be identified, in line with previous studies like (DeWitte and Lewis, 2021) (Table 5.2). Of the 1446 individuals dated to the pre-Black Death era, 80% (n=1150) of individuals could be assigned to the more specific early or late periods, with the majority (n=1036 or 71.6%) buried in extra-London cemeteries.

Inevitably, the data available for the secondary sites varied in type and level of detail. These data still enabled a comprehensive analysis to be undertaken when access to skeletal collections like Oxford Castle and Littlemore Priory, Oxfordshire as well as Barton-Upon-Humber and Stoke Quay was disrupted due to COVID-19 restrictions, however meant that most data was recorded at the individual level and could only be reported as crude prevalence rates. Table 5.3 shows the type of data recorded for the secondary sites. The following analysis combines the primary and secondary data to provide an adequate sample size to analyse health before and after the Black Death in the extra-London and London regions.

Table 5.2: *Composition of the study sample in the pre-Black Death period by site.*

Region	**Site**	**Early pre-Black Death**		**Late pre-Black Death**		**Total**
		n	**%**	**n**	**%**	**N**
Extra-London	**Wharram Percy**	7	12.1	51	87.9	58
	St Oswald's Priory	44	29.3	106	70.7	150
	St Anne's Charterhouse	0		0		0
	Barton-upon-Humber	197	66.3	100	33.7	297
	Stoke Quay	191	36	340	64	531
	Subtotal	439	42.4	597	57.6	1036
London[1]	**Guildhall Yard**	10	27	27	73	37
	Merton Priory	0		37	100	37
	Spital Square	0		40	100	40
	Subtotal	10	8.8	104	91.2	114
Total		449	39	701	61	1150

[1] East Smithfield Cemetery and St Mary Graces were not included in this table as the available skeletal remains were dated to the Black Death and post-Black Death respectively

Table 5.3: *Primary and secondary data available for the extra-London and London sites.*

Region	Site	Cribra orbitalia	Puberty	VNC[1]	Skeletal pathology	Spina bifida	Postcranial metrics	Enamel hypoplasia
Extra-London	**Wharram Percy[2]**	Y[3]	Y	Y	Y	Y	Y	Y
	St Oswald's Priory[2]	Y	Y	Y	Y	Y	Y	Y
	St Anne's Charterhouse[2]	Y	Y	Y	Y	Y	Y	Y
	Barton-upon-Humber[4]	Y/CR	Y	Y	CR	CR	Y(F[5])	Y
	Stoke Quay[6]	CR	N	N	CR	Y	Y	Y
London	**Guildhall Yard[7]**	Y[8]	N	Y	CR	CR	Y	Y
	East Smithfield[2]	Y	N	Y	CR	CR	Y	Y
	Merton Priory[2]	Y	N	Y	CR	CR	Y	Y
	St Mary Graces[2]	Y	N	Y	CR	CR	Y	Y
	Spital Square[2]	Y	N	N	CR	CR	Y	Y

[1] VNC = vertebral neural canal; [2] Primary data; [3] Y = data available; CR = crude rate alone; N = data not available; [4] Waldron (2007); Watts (2013) and Lewis and Shapland (M. Lewis, personal communication, 2021); [5] Only adult femoral length available; [6] Brown et al. (2020); [7] Centre for Human Bioarchaeology (2020).

The following analysis combines the primary and secondary data in order to analyse health before and after the Black Death in the extra-London and London regions.

5.2 Demography of the sample

5.2.1 The Pre-Black Death, post-Black Death, and Black Death periods combined

Table 5.4 summarises the demographic composition of the sample. Overall, the sample was composed of 814 adults (n=814/2065; 39.4%) adults and 1251 (n=1251/2065; 60.6%) non-adults. When divided by region, the extra-London sample comprised 462 adults (34.5%) and 879 non-adults (65.6%), while the London region provided data for 352 adults (48.6%) and 372 non-adults (51.4%).

When comparing similar age categories, all age ranges from the extra-London region had a greater number of individuals dated to the pre-Black Death period, reflecting the difference in overall sample sizes. When looking at the London region, the Black Death period showed higher numbers of individuals in all age ranges, followed by the post-Black Death period. The small sample size from the post-Black Death period is a common problem as the period is short and often it is difficult to obtain skeletons dated to the narrow period (e.g., see Lewis (2016*b*)). This difficulties limited some of the analyses.

Table 5.4: *Mortality profiles for the sample by period for the extra-London and the London regions.*

Region	**Age (years)**	**Pre-Black Death**		**Black Death**		**Post-Black Death**		**Total**
		n	**%**	**n**	**%**	**n**	**%**	**N**
Extra-London	**Perinate**	34	100	0		0		34
	0-5	561	99.8	0		1	0.2	562
	6-12	182	98.9	0		2	1.1	184
	13-17	96	99	0		1	1	97
	Non-adult[1]	2	100	0		0		2
	18-25	148	96.7	0		5	3.3	153
	26-35	299	96.8	0		10	3.2	309
	Subtotal	1322	98.6	0		19	1.4	1341
London	**Perinate**	0		5	83.3	1	16.7	6
	0-5	8	7	68	59.1	39	33.9	115
	6-12	22	18.6	73	61.9	23	19.5	118
	13-17	13	19.1	31	45.6	24	35.3	68
	Non-adult	6	9.2	39	60	20	30.8	65
	18-25	25	20.3	63	51.2	35	28.5	123
	26-35	40	17.5	121	52.8	68	29.7	229
	Subtotal	114	15.8	400	55.3	210	29	724
Total		1436	69.5	400	19.4	229	11.1	2065

[1] Non-adult = unaged non-adult

5.2.2 Infant and child mortality profiles

Table 5.5 summarises the age at death profiles for the 540 non-adults under 5 years of age by period and region. The majority of the young non-adult sample came from the extra-London region (n=462/540, 85.6%). This region had all recorded deaths for children under 5 years during the pre-Black Death, while London registered 10.3% (n=8/78) individuals during the pre-Black Death, 57.7% (n=45/78) during the Black Death and 32.1% (n=25/78) during the post-Black Death period.

When looking at the age distribution, the extra-London region showed 31.4% (n=145/462) individuals under the age of 1 year, followed by 25.5% (n=118/462) ages 1-1.99 years and 18.4% (n=85/462) ages 2-2.99 years. The most represented ages in the London region during the pre-Black Death period was 1-1.99 years (n=5/8, 62.5%). The post-Black death was composed by a majority of individuals ages 4-5 years (n=11/25, 44%), while the Black Death had higher representation in the ages 2-2.99 years (n=12/45, 26.7%) followed closely by 3-3.99 and 4-4.99 years (n=10/45, 22.2%).

Table 5.5: *Infant and child mortality profiles for individuals under the age of 5 years by period in the extra-London and London regions.*

Region	**Period**	**0-0.99 years**		**1-1.99 years**		**2-2.99 years**		**3-3.99 years**		**4-4.99 years**		**Total**
		n	**%**	**n**	**%**	**n**	**%**	**n**	**%**	**n**	**%**	**N**
Extra-London	**Pre-Black Death**	145	31.4	118	25.5	85	18.4	71	15.4	43	9.3	462
	Black Death	0		0		0		0		0		0
	Post-Black Death	0		0		0		0		0		0
	Subtotal	145	31.4	118	25.5	85	18.4	71	15.4	43	9.3	462
London	**Pre-Black Death**	1	12.5	5	62.5	0		1	12.5	1	12.5	8
	Black Death	5	11.1	8	17.8	12	26.7	10	22.2	10	22.2	45
	Post-Black Death	0		4	16	5	20	5	20	11	44	25
	Subtotal	6	7.7	17	21.8	17	21.8	16	20.5	22	28.2	78
Total		151	28	135	24.1	102	18.2	87	15.5	65	11.6	540

5.2.3 Sex distribution

Sex could be estimated for 764 adults, representing 37% (n=764/2065) of the total adult sample. Of the sex estimated adults, 58.1% (n=447) were from the extra-London and 41.5% (n=317) were from London sites. Figure (Figure 5.1 shows the sex distribution of the adult sample by period and region. When comparing male and female mortality profiles by region and period, the London sample had a statistically significant higher frequencies of males than females who died during the pre-Black Death period (X^2=11.94, df=1, p=0.0005). There were no statistically significant differences between the sexes of the London and extra-London adults buried during the post-Black Death period. When looking at each region individually, the extra-London region showed statistically higher frequencies of males during the post-Black Death period (two tailed Fisher's exact test p=0.03) compared to the pre-Black Death period. The London period did not show any statistically significant male to female frequencies when comparing pre-, Black Death, and post-Black Death periods.

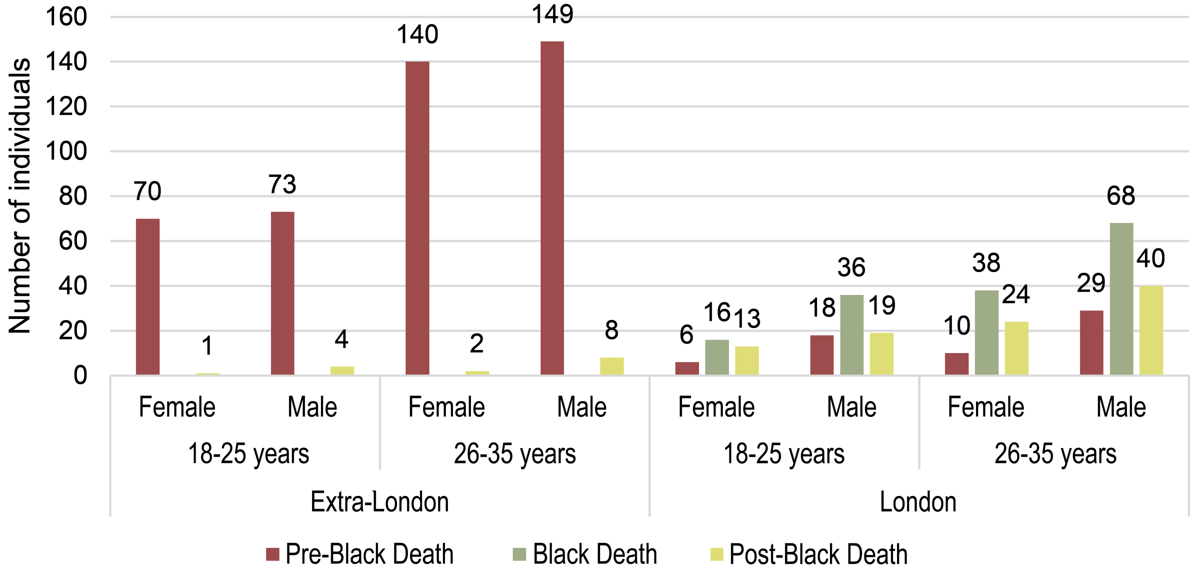


Figure 5.1: *Frequencies of male and female adults by period in the extra-London and London regions.*

5.2.4 Adolescent sample

In this investigation, the analysis of adolescents was extended from 10 to 25 years of age to 10 to 35 years of age to contemplate individuals who may have experienced growth delay pass the age of 25 years. However, as there were no individuals over 25 years of age who had not completed puberty, the age range was limited to 10-25 years of age. This also allowed comparing results to prrevious investigations using the same methodology (e.g. DeWitte and Lewis (2021))

The following figure (5.2) shows the frequencies of individuals aged 10 to 25 by sex and period.

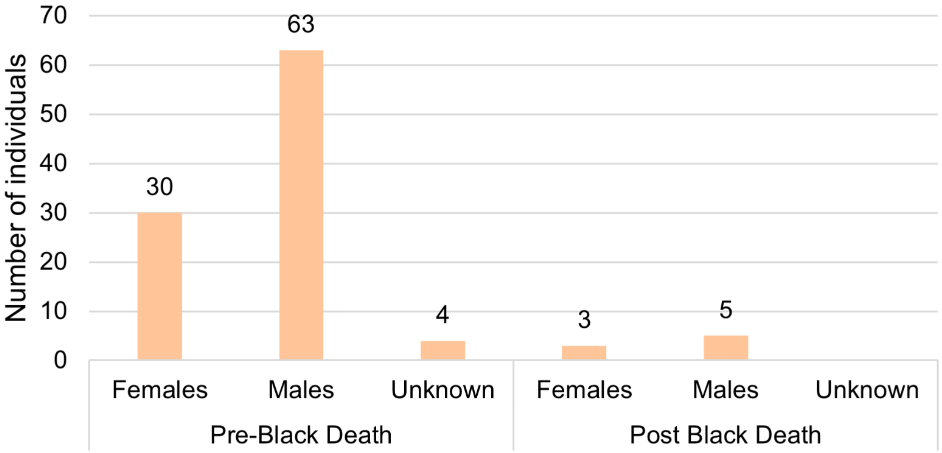


Figure 5.2: *Frequencies of individuals aged 10 to 25 years by sex and period in the extra-London region.*

5.2.5 Pre-Black Death period: early and late

Table 5.6 presents the age mortality profiles for the extra-London and London regions for the early and late pre-Black Death sub-periods. The total pre-Back Death sample comprised 397 adults (n=397/1150; 34.5%) and 753 non-adults (n=753/1150; 65.5%). When looking at differences between regions, the extra-London region was composed by 332 (n=332/1036; 32.1%) adults and 704 (n=704/1036; 68%) non-adults, while the London sample was comprised by 65 (n=65/114; 57%) adults and 49 (n=49/114; 43%) non-adults.

Table 5.6: *Mortality profiles for individuals dated in the pre-Black Death period in the extra-London and London regions.*

Region	Age (years)	Early pre-Black Death		Late pre-Black Death		Total
		n	%	n	%	N
Extra-London	**Perinate**	2	6.1	31	94	33
	0-5	174	39.2	270	60.81	444
	6-12	52	34.7	98	65.3	150
	13-17	40	53.3	35	46.7	75
	Non-adult	0		2	100	2
	18-25	47	43.9	60	56.1	107
	26-35	124	55.1	101	44.9	225
	Subtotal	439	42.4	597	57.6	1036
London	**Perinate**	0		0		0
	0-5	3	37.5	5	62.5	8
	6-12	3	13.6	19	86.4	22
	13-17	0		13	100	13
	Non-adult	0		6	100	6
	18-25	1	4	24	96	25
	26-35	3	7.5	37	92.5	40
	Subtotal	10	8.8	104	91.2	114
Total		449	39	701	61	1150

[1.] Non-adult = unaged non-adult

5.2.6 Infant and child mortality profiles

Within the pre-Black Death period, 71.1% (n=384/540) was dated to the early or late pre-Black Death subperiod. Due to the small sample for the London region, statistical comparison was only possible within the extra-London region. When looking at age distribution, the extra-London region had statistically significant more representation of individuals age 0-1.99 years (n=117/376, 31.1%) for both subperiods. The early pre-Black Death subperiod had higher frequencies of individuals age 1-1.99 years (n=44/144, 30.6%, X^2=4.25, df=1, p=0.04), while the late pre-Black Death period had higher frequencies of individuals age 0-0.99 years (n=83/232, 35.8%, X^2=6.13, df= 1, p=0.01). The London sample was much smaller (n=8). Nevertheless, the region had a similar age distribution with its higher frequencies of deaths registered during the late pre-Black Death period for individuals ages 1-1.99 years (n=4/5, 80%) (Table 5.7).

Table 5.7: *Infant and child mortality profiles for individuals under the age of 5 years by subperiod in the extra-London and London regions.*

Region	**Subperiod**	**0-0.99 years**		**1-1.99 years**		**2-2.99 years**		**3-3.99 years**		**4-4.99 years**		**Total**
		n	**%**	**n**	**%**	**n**	**%**	**n**	**%**	**n**	**%**	**N**
Extra-London	**Early pre-Black Death**	34	23.6	44	30.6	30	20.8	24	16.7	12	8.3	144
	Late pre-Black Death	83	35.8	49	21.1	36	15.5	37	16	27	11.6	232
	Subtotal	117	31.1	93	24.7	66	17.6	61	16.2	39	10.4	376
London	**Early pre-Black Death**	1	33.3	1	33.3	0		0		1	33.3	3
	Late pre-Black Death	0		4	80	0		1	20	0		5
	Subtotal	1	12.5	5	62.5	0		1	12.5	1	12.5	8
Total		118	30.7	98	25.5	66	17.2	62	16.2	40	10.4	384

5.2.7 Sex distribution

When looking at sex distribution in adults by subperiod (Figure 5.3), 385 individuals were estimated as female (n=170/385, 44.2%) or male (n=215/385, 55.8%). When comparing frequencies of females and males by subperiod, the only statistically significant difference was observed in individuals buried during the late pre-Black death period, where London had more male deaths than female deaths (X^2=9.79, df=1, p=0.002). No statistically significant differences were observed when the extra-London and London regions were compared.

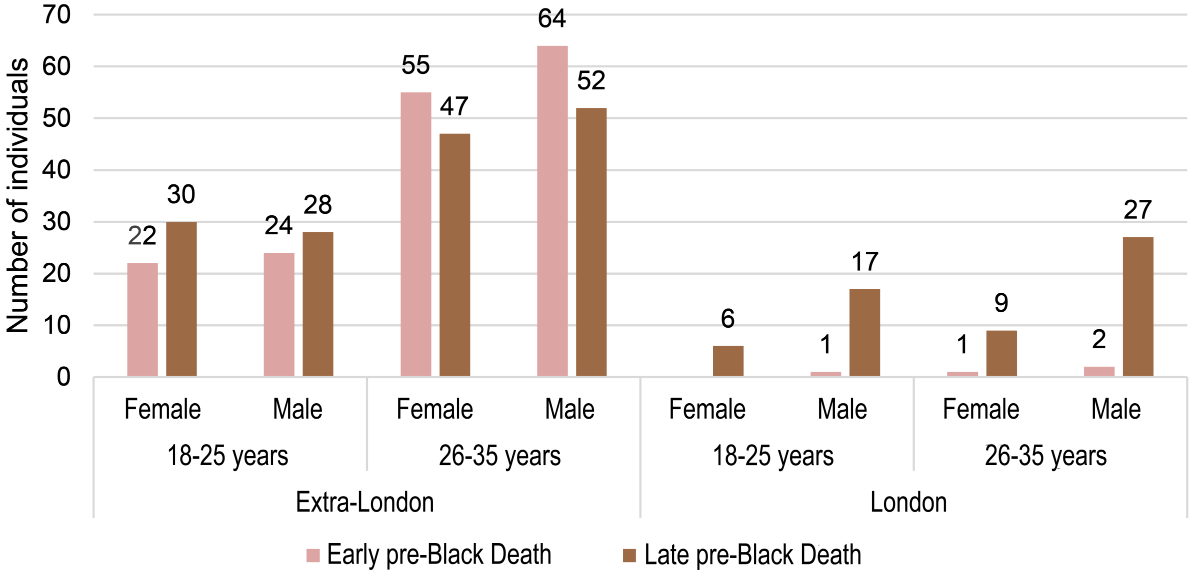


Figure 5.3: *Frequencies of male and female adults by subperiod in the extra-London and London regions.*

5.2.8 Adolescent sample

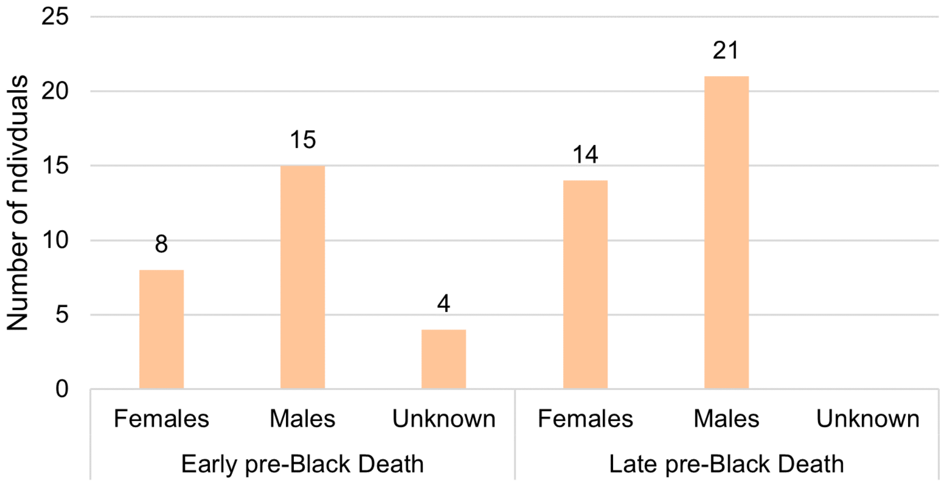


Figure 5.4: *Frequencies of individuals aged 10 to 25 years by sex and subperiod in the extra-London region.*

Figure 5.4 shows the frequencies of individuals aged 10 to 25 by sex and period.

5.3 Osteometrics

5.3.1 Vertebral neural canal size

Vertebral metrics for non-adults over the age of 2.5 years were available only from primary data collection (sites: Wharram Percy, St Oswald's Priory, St Anne's Charterhouse). For adults, vertebral dimensions were available from the primary data collection, as well as from secondary data (sites: Barton-upon-Humber and all the London sites except Spital Square (Watts, 2013).

5.3.1.1 Non-adults

As the evidence of vertebral neural canal size for non-adults is limited to the primary data, the comparisons are only possible for the extra-London region in the pre- and post-Black death periods. The overall sample comprised 58 individuals with fused vertebrae. This was approximately over the age of 2.5 years. Due to the small sample in the post-Black Death period, comparisons between the pre- and post-Black Death groups were limited. Table 5.8 presents a summary of VNC dimensions means by vertebrae and period.

Table 5.8: *VNC dimension means for non-adults by period in the extra-London region.*

Vertebrae	Period	N	Mean	Std. Deviation	Std. Error Mean
L1 AP[1]	**Pre-Black Death**	29	16.50	1.12	0.21
	Post-Black Death	-			
L1 TR	**Pre-Black Death**	33	19.09	1.35	0.24
	Post-Black Death	-			
L2 AP	**Pre-Black Death**	23	16.02	1.09	0.23
	Post-Black Death	1	17.74		
L2 TR	**Pre-Black Death**	34	19.42	1.22	0.21
	Post-Black Death	1	21.27		
L3 AP	**Pre-Black Death**	29	15.30	1.59	0.29
	Post-Black Death	2	14.49	4.07	2.88
L3 TR	**Pre-Black Death**	39	19.64	1.37	0.22
	Post-Black Death	2	18.18	5.29	3.74
L4 AP	**Pre-Black Death**	29	14.32	1.70	0.32
	Post-Black Death	1	18.17		
L4 TR	**Pre-Black Death**	36	19.73	2.10	0.35
	Post-Black Death	1	21.88		
L5 AP	**Pre-Black Death**	27	14.74	1.94	0.37
	Post-Black Death	-			
L5 TR	**Pre-Black Death**	32	22.09	1.97	0.35
	Post-Black Death	-			

[1] L = Lumbar vertebrae; AP = Antero-posterior diameter; - = No data; TR = transversal diameter

Results showed smaller dimensions for L2 and L4 (both AP, L2 = 16.02 ± 1.09 mm, L4 = 14.32 ± 1.7 mm and TR, L2 = 19.42 ± 1.22 mm, L4 = 19.73 ± 2.1 mm) for individuals dated to the pre-Black Death period, while L3 showed smaller means for individuals who died during the post-Black Death period (AP, 14.49 ± 4.07 and TR 18.18 ± 5.29). However, due to the small size of the post-Black Death sample, statistical analysis was not possible.

Within the pre-Black Death period, the overall sample was 41 individuals, 10 (24.4%) individuals dated to the early period and 31 (75.6%) to the later subperiod. Comparisons between subperiods show smaller anterio-posterior and transverse dimensions for L1 (16.22 ± 1.15 mm) and L2 (15.74 ± 1.24 mm), and smaller transverse dimensions for L3 (19.48 ± 1.41 mm) and L5 (21.82 ± 2.14 mm) in the late pre-Black Death period. In the early pre-Black Death period anterio-posterior dimensions were reduced for L3 and L5 (Table 5.9)

Table 5.9: *VNC dimension means for non-adults during the pre-Black Death period*

Vertebrae	Subperiod	N	Mean	Std. Deviation	Std. Error Mean
L1 AP[1]	**Early pre-Black Death**	5	17.35	0.87	0.39
	Late pre-Black Death	18	16.22	1.15	0.27
L1 TR	**Early pre-Black Death**	5	20.33	0.78	0.35
	Late pre-Black Death	21	18.79	1.38	0.30
L2 AP	**Early pre-Black Death**	4	16.91	0.48	0.24
	Late pre-Black Death	13	15.74	1.24	0.34
L2 TR	**Early pre-Black Death**	5	20.39	0.61	0.27
	Late pre-Black Death	21	19.13	1.29	0.28
L3 AP	**Early pre-Black Death**	7	14.55	1.25	0.47
	Late pre-Black Death	15	15.59	1.82	0.47
L3 TR	**Early pre-Black Death**	8	20.43	0.98	0.35
	Late pre-Black Death	20	19.48	1.41	0.32
L4 AP	**Early pre-Black Death**	7	14.11	1.39	0.53
	Late pre-Black Death	13	14.41	1.69	0.47
L4 TR	**Early pre-Black Death**	9	20.99	1.62	0.54
	Late pre-Black Death	18	19.23	2.32	0.55
L5 AP	**Early pre-Black Death**	6	14.31	1.43	0.58
	Late pre-Black Death	15	14.93	2.37	0.61
L5 TR	**Early pre-Black Death**	8	22.59	1.42	0.50
	Late pre-Black Death	18	21.82	2.14	0.50

[1] L = Lumbar vertebrae; AP = Antero-posterior diameter; TR = transversal diameter

5.3.1.2 Adults

5.3.1.2.1 Comparison between periods and subperiods

Table 5.10 and Table 5.11 show the composition of the sample for vertebral neural canal dimension analysis by period and subperiod respectively. As the secondary evidence for VNC was collected for adult individuals over 18 years of age, the following analysis only centred on adults. The overall sample was composed of 242 individuals, 136 (56.2%) from the extra-London region and 106 (43.8%) from the London region. Within the pre-Black Death period (early and late pre-Black Death period), the sample was composed of 112 individuals. This was 46.3% of the total sample of adults with measurable VNC dimensions.

Table 5.10: *Demographic composition of the adult sample with VNC dimensions by estimated sex and period.*

Region	**Sex**	**Pre-Black Death**		**Black Death**		**Post-Black Death**		**Total**
		n	**%**	**n**	**%**	**n**	**%**	**N**
Extra-London	**Female**	64	100	0		0		64
	Male	64	90.1	0		7	9.9	71
	Unknown	1	100	0		0		1
	Subtotal	129	94.9	0		7	5.2	136
London	**Female**	6	13.6	20	45.5	18	40.9	44
	Male	3	4.8	42	67.7	17	27.4	62
	Unknown	0		0		0		0
	Subtotal	9	8.5	62	58.5	35	33	106
Total		138	57	62	25.6	42	17.4	242

Table 5.11: *Demographic composition of the adult sample with VNC dimensions by estimated sex for the pre-Black Death period.*

Region	**Sex**	**Early pre-Black Death**		**Late late-Black Death**		**Total**
		n	**%**	**n**	**%**	**N**
Extra-London	**Female**	24	47.1	27	52.9	51
	Male	32	62.8	19	37.3	51
	Unknown	0		1	100	1
	Subtotal	56	54.4	47	45.6	103
London	**Female**	1	16.7	5	83.3	6
	Male	0		3	100	3
	Unknown	0		0		0
	Subtotal	1	11.1	8	88.9	9
Total		57	50.9	55	49.1	112

The following tables show the vertebral neural canal dimensions for the extra-London (Table 5.12) and London (Table 5.13) regions by period. The next tables exhibit VNC dimensions for the extra-London (Table 5.14) and London (Table 5.15) region during the pre-Black Death period regions by period. the next table exhibit VNC dimensions for the extra-London region during the pre-Black Death period.

Table 5.12: *Vertebral neural canal dimensions for the extra-London region by period*

Vertebrae	Period	N	Mean	Std. Deviation	Std. Error Mean
L1 AP[1]	**Pre-Black Death**	94	16.47	1.23	0.13
	Post-Black Death	6	17.20	1.11	0.45
L1 TR	**Pre-Black Death**	102	21.50	1.65	0.16
	Post-Black Death	6	21.91	1.13	0.46
L2 AP	**Pre-Black Death**	96	15.55	1.75	0.18
	Post-Black Death	6	16.31	2.00	0.82
L2 TR	**Pre-Black Death**	103	21.59	1.81	0.18
	Post-Black Death	7	22.14	2.28	0.86
L3 AP	**Pre-Black Death**	99	14.59	1.61	0.16
	Post-Black Death	7	15.37	1.78	0.67
L3 TR	**Pre-Black Death**	107	21.67	1.49	0.14
	Post-Black Death	7	21.93	2.15	0.81
L4 AP	**Pre-Black Death**	97	14.79	1.80	0.18
	Post-Black Death	6	15.81	1.53	0.63
L4 TR	**Pre-Black Death**	107	22.06	1.95	0.19
	Post-Black Death	7	22.60	1.96	0.74
L5 AP	**Pre-Black Death**	91	16.02	2.28	0.24
	Post-Black Death	4	17.59	1.62	0.81
L5 TR	**Pre-Black Death**	98	24.49	2.26	0.23
	Post-Black Death	4	25.72	2.04	1.02

[1] L = Lumbar vertebrae; AP = Antero-posterior diameter; - = No data; TR = transversal diameter

Table 5.13: *Vertebral neural canal dimensions for the London region by period*

Vertebrae	Period	N	Mean	Std. Deviation	Std. Error Mean
L1 AP[1]	**Pre-Black Death**	9.00	16.86	0.93	0.31
	Black Death	54.00	16.02	2.16	0.29
	Post-Black Death	31.00	16.06	1.18	0.21
L1 TR	**Pre-Black Death**	9.00	21.45	1.50	0.50
	Black Death	56.00	20.86	1.79	0.24
	Post-Black Death	32.00	21.11	1.38	0.24
L2 AP	**Pre-Black Death**	9.00	16.03	1.06	0.35
	Black Death	57.00	14.76	1.41	0.19
	Post-Black Death	31.00	15.25	1.67	0.30
L2 TR	**Pre-Black Death**	9.00	21.94	1.43	0.48
	Black Death	58.00	21.08	1.61	0.21
	Post-Black Death	32.00	21.30	1.21	0.21
L3 AP	**Pre-Black Death**	9.00	15.14	0.94	0.31
	Black Death	55.00	13.82	1.47	0.20
	Post-Black Death	31.00	14.62	1.56	0.28
L3 TR	**Pre-Black Death**	9.00	22.02	1.22	0.41
	Black Death	58.00	20.91	1.59	0.21
	Post-Black Death	30.00	21.59	1.27	0.23
L4 AP	**Pre-Black Death**	8.00	15.79	1.11	0.39
	Black Death	51.00	13.82	1.67	0.23
	Post-Black Death	30.00	14.64	1.78	0.32
L4 TR	**Pre-Black Death**	8.00	22.69	1.34	0.47
	Black Death	53.00	21.30	1.86	0.26
	Post-Black Death	31.00	21.79	1.78	0.32
L5 AP	**Pre-Black Death**	6.00	16.95	0.84	0.34
	Black Death	42.00	15.45	2.28	0.35
	Post-Black Death	27.00	15.94	2.36	0.45
L5 TR	**Pre-Black Death**	8.00	24.86	1.49	0.53
	Black Death	46.00	24.12	2.55	0.38
	Post-Black Death	28.00	24.39	2.44	0.46

[1] L = Lumbar vertebrae; AP = Antero-posterior diameter; TR = transversal diameter

Table 5.14: *Vertebral neural canal dimensions for the extra-London region during the pre-Black Death period*

Vertebrae	Subperiod	N	Mean	Std. Deviation	Std. Error Mean
L1 AP[1]	**Early pre-Black Death**	39	16.16	1.31	0.21
	Late pre-Black Death	35	16.59	1.21	0.20
L1 TR	**Early pre-Black Death**	44	21.39	1.66	0.25
	Late pre-Black Death	38	21.34	1.57	0.25
L2 AP	**Early pre-Black Death**	39	15.24	1.46	0.23
	Late pre-Black Death	37	15.61	1.65	0.27
L2 TR	**Early pre-Black Death**	43	21.71	2.11	0.32
	Late pre-Black Death	40	21.39	1.56	0.25
L3 AP	**Early pre-Black Death**	44	14.50	1.60	0.24
	Late pre-Black Death	36	14.47	1.60	0.27
L3 TR	**Early pre-Black Death**	47	21.45	1.38	0.20
	Late pre-Black Death	40	21.68	1.63	0.26
L4 AP	**Early pre-Black Death**	41	14.95	1.69	0.26
	Late pre-Black Death	38	14.59	2.03	0.33
L4 TR	**Early pre-Black Death**	45	22.28	2.02	0.30
	Late pre-Black Death	41	21.71	1.95	0.31
L5 AP	**Early pre-Black Death**	40	16.30	2.09	0.33
	Late pre-Black Death	36	15.63	2.26	0.38
L5 TR	**Early pre-Black Death**	43	24.61	1.99	0.30
	Late pre-Black Death	38	23.99	2.45	0.40

[1] L = Lumbar vertebrae; AP = Antero-posterior diameter; TR = transversal diameter

Table 5.15: *Vertebral neural canal dimensions for the London region during the pre-Black Death period*

Vertebrae	Subperiod	N	Mean	Std. Deviation	Std. Error Mean
L1 AP	**Early pre-Black Death**	1	18.51		
	Late pre-Black Death	8	16.65	0.74	0.26
L1 TR	**Early pre-Black Death**	1	19.98		
	Late pre-Black Death	8	21.63	1.49	0.53
L2 AP	**Early pre-Black Death**	1	17.33		
	Late pre-Black Death	8	15.87	1.01	0.36
L2 TR	**Early pre-Black Death**	1	21.58		
	Late pre-Black Death	8	21.99	1.52	0.54
L3 AP	**Early pre-Black Death**	1	17.05		
	Late pre-Black Death	8	14.90	0.65	0.23
L3 TR	**Early pre-Black Death**	1	21.83		
	Late pre-Black Death	8	22.05	1.30	0.46
L4 AP	**Early pre-Black Death**	1	15.85		
	Late pre-Black Death	7	15.79	1.20	0.45
L4 TR	**Early pre-Black Death**	1	22.36		
	Late pre-Black Death	7	22.74	1.44	0.54
L5 AP	**Early pre-Black Death**	1	16.28		
	Late pre-Black Death	5	17.08	0.86	0.39
L5 TR	**Early pre-Black Death**	1	24.24		
	Late pre-Black Death	7	24.94	1.59	0.60

[1.] L = Lumbar vertebrae; AP = Antero-posterior diameter; TR = transversal diameter

The extra-London region showed smaller dimensions for all VNC measurements during the pre-Black Death period compared to the post-Black Death period. When comparing early and late pre-Black Death periods, the latter exhibited smaller dimensions with the exception of L1AP, L2AP and L3TR. The London region showed a different pattern where the post-Black Death period individuals had smaller VNC dimensions. Due to the small size of the sample, comparison within the pre-Black Death period was not possible. The Black Death period exhibited smaller VNC than both pre- and post-Black Death periods. This was statistically significant for L2AP (t(64) 2.6, p=0.012), L3AP (t(62) 2.6, p=0.012), L3TR (t(65) 2, p=0.048), L4AP (t(57) 3.2, p=0.048) and L4TR (t(59) 2, p=0.047) for comparisons between the pre-Black Death period and the Black Death period and for L3AP (t(84) -2.39, p=0.019), L3TR (t(86) -2, p=0.044), and L4AP (t(79) -2.1, p=0.04) for comparisons between the Black Death and post-Black Death periods.

Comparisons between regions for the same period categories showed similar tendencies. The extra London region showed more constricted VNC dimensions for individuals who died during the pre- Black Death period (with the only exception of L1TR), while the London regions exhibited smaller dimensions during the post-Black Death period. The latter being statistically significant for L1AP (t(35) 2.2, p=0.037).

Table 5.16 shows the means for the VNC dimensions with statistically significant differences when comparing regions. The post-Black Death only period was the only one for which there was a statistically significant difference observed. The following table (5.17) shows the means for the VNC dimensions with statistically significant differences when comparing periods. The only region in which these difference were found was London.

Table 5.16: *Statistically significant differences in VNC dimensions for the post-Black Death period by region.*

Vertebrae	Region	N	Mean	Std. Deviation	Std. Error Mean
L1AP[1]	**Extra-London**	6	17.20	1.11	0.45
	London	31	16.06	1.18	0.21

[1] L = Lumbar vertebrae; AP = Antero-posterior diameter; TR = transversal diameter

Table 5.17: *Statistically significant differences in VNC dimensions by period for the London region.*

Vertebrae	Period	N	Mean	Std. Deviation	Std. Error Mean
L2AP[1]	**Pre-Black Death**	9	16.03	1.06	0.35
	Black Death	57	14.76	1.41	0.19
L3AP	**Pre-Black Death**	9	15.14	0.94	0.31
	Black Death	55	13.82	1.47	0.20
L3TR	**Pre-Black Death**	9	22.02	1.22	0.41
	Black Death	58	20.91	1.59	0.21
L4AP	**Pre-Black Death**	8	15.79	1.11	0.39
	Black Death	51	13.82	1.67	0.23
L4TR	**Pre-Black Death**	8	22.69	1.34	0.47
	Black Death	53	21.30	1.86	0.26
L3AP	**Black Death**	55	13.82	1.47	0.20
	Post-Black Death	31	14.62	1.56	0.28
L3TR	**Black Death**	58	20.91	1.59	0.21
	Post-Black Death	30	21.59	1.27	0.23
L4AP	**Black Death**	51	13.82	1.67	0.23
	Post-Black Death	30	14.64	1.78	0.32

[1]. L = Lumbar vertebrae; AP = Antero-posterior diameter; TR = transversal diameter

5.3.1.2.2 Sex distribution

The following tables 5.18-Table 5.24 show the difference between males and females by period in the extra-London region and the London region by period and within the prre-Black Death period. Due to small sample, the post-Black Death period was not analysed in the extra-London regionthe extra London region was only.

Table 5.18: *Vertebral neural canal dimensions for the extra-London region during the pre-Black Death period by sex*

Vertebrae	Sex	N	Mean	Std. Deviation	Std. Error Mean
L1 AP[1]	**Female**	48	16.63	1.13	0.16
	Male	45	16.28	1.32	0.20
L1 TR	**Female**	52	21.42	1.60	0.22
	Male	49	21.61	1.72	0.25
L2 AP	**Female**	44	15.48	1.62	0.24
	Male	51	15.60	1.88	0.26
L2 TR	**Female**	50	21.41	1.46	0.21
	Male	52	21.64	2.33	0.32
L3 AP	**Female**	50	14.76	1.67	0.24
	Male	49	14.43	1.55	0.22
L3 TR	**Female**	54	21.76	1.42	0.19
	Male	53	21.57	1.57	0.22
L4 AP	**Female**	45	14.63	1.84	0.27
	Male	51	14.90	1.77	0.25
L4 TR	**Female**	52	21.95	1.71	0.24
	Male	54	22.20	2.16	0.29
L5 AP	**Female**	47	15.78	2.10	0.31
	Male	44	16.28	2.46	0.37
L5 TR	**Female**	50	24.16	2.24	0.32
	Male	48	24.83	2.26	0.33

[1] L = Lumbar vertebrae; AP = Antero-posterior diameter; - = No data; TR = transversal

Table 5.19: *Vertebral neural canal dimensions for the London region during the late pre-Black Death period by sex*

Vertebrae	Sex	N	Mean	Std. Deviation	Std. Error Mean
L1 AP[1]	**Female**	6	17.26	0.78	0.32
	Male	3	16.05	0.66	0.38
L1 TR	**Female**	6	21.68	1.73	0.71
	Male	3	20.98	1.03	0.59
L2 AP	**Female**	6	16.57	0.71	0.29
	Male	3	14.96	0.79	0.46
L2 TR	**Female**	6	22.43	1.46	0.60
	Male	3	20.97	0.81	0.47
L3 AP	**Female**	6	15.50	0.91	0.37
	Male	3	14.41	0.52	0.30
L3 TR	**Female**	6	22.32	1.32	0.54
	Male	3	21.43	0.89	0.51
L4 AP	**Female**	5	15.93	0.58	0.26
	Male	3	15.57	1.88	1.09
L4 TR	**Female**	5	22.62	1.32	0.59
	Male	3	22.80	1.65	0.96
L5 AP	**Female**	4	17.25	0.90	0.45
	Male	2	16.36	0.25	0.18
L5 TR	**Female**	5	24.46	1.23	0.55
	Male	3	25.52	1.91	1.10

[1] L = Lumbar vertebrae; AP = Antero-posterior diameter; - = No data; TR = transversal

Table 5.20: *Vertebral neural canal dimensions for the London region during the Black Death period by sex*

Vertebrae	Sex	N	Mean	Std. Deviation	Std. Error Mean
L1 AP[1]	**Female**	17	16.20	1.14	0.28
	Male	37	15.94	2.51	0.41
L1 TR	**Female**	18	20.82	1.67	0.39
	Male	38	20.89	1.87	0.30
L2 AP	**Female**	18	15.19	1.24	0.29
	Male	39	14.56	1.46	0.23
L2 TR	**Female**	19	21.21	1.46	0.33
	Male	39	21.01	1.69	0.27
L3 AP	**Female**	16	14.40	1.20	0.30
	Male	39	13.58	1.52	0.24
L3 TR	**Female**	17	20.99	1.47	0.36
	Male	41	20.87	1.66	0.26
L4 AP	**Female**	14	14.05	1.21	0.32
	Male	37	13.74	1.82	0.30
L4 TR	**Female**	16	21.67	1.49	0.37
	Male	37	21.15	2.00	0.33
L5 AP	**Female**	11	15.14	2.31	0.70
	Male	31	15.56	2.30	0.41
L5 TR	**Female**	13	24.01	2.37	0.66
	Male	33	24.16	2.65	0.46

[1] L = Lumbar vertebrae; AP = Antero-posterior diameter; - = No data; TR = transversal

Table 5.21: *Vertebral neural canal dimensions for the London region during the Black Death period by sex*

Vertebrae	Sex	N	Mean	Std. Deviation	Std. Error Mean
L1 AP[1]	**Female**	16	15.99	1.03	0.26
	Male	15	16.14	1.36	0.35
L1 TR	**Female**	16	20.85	1.13	0.28
	Male	16	21.37	1.58	0.39
L2 AP	**Female**	15	15.14	1.79	0.46
	Male	16	15.36	1.60	0.40
L2 TR	**Female**	16	21.11	1.25	0.31
	Male	16	21.49	1.19	0.30
L3 AP	**Female**	15	14.69	1.55	0.40
	Male	16	14.56	1.62	0.40
L3 TR	**Female**	15	21.57	1.39	0.36
	Male	15	21.61	1.18	0.30
L4 AP	**Female**	16	14.85	1.87	0.47
	Male	14	14.41	1.70	0.45
L4 TR	**Female**	16	22.08	2.05	0.51
	Male	15	21.48	1.44	0.37
L5 AP	**Female**	14	15.80	1.76	0.47
	Male	13	16.10	2.94	0.82
L5 TR	**Female**	14	24.65	2.52	0.67
	Male	14	24.13	2.43	0.65

[1] L = Lumbar vertebrae; AP = Antero-posterior diameter; - = No data; TR = transversal

Table 5.22: *Vertebral neural canal dimensions for the extra-London region during the early pre-Black Death period by sex*

Vertebrae	Sex	N	Mean	Std. Deviation	Std. Error Mean
L1 AP[1]	**Female**	16	16.30	1.26	0.32
	Male	23	16.06	1.36	0.28
L1 TR	**Female**	19	21.01	1.28	0.29
	Male	25	21.69	1.88	0.38
L2 AP	**Female**	13	15.08	1.50	0.42
	Male	26	15.32	1.46	0.29
L2 TR	**Female**	16	21.18	1.24	0.31
	Male	27	22.02	2.45	0.47
L3 AP	**Female**	19	14.75	1.87	0.43
	Male	25	14.31	1.38	0.28
L3 TR	**Female**	20	21.44	1.25	0.28
	Male	27	21.46	1.49	0.29
L4 AP	**Female**	16	14.56	1.87	0.47
	Male	25	15.20	1.54	0.31
L4 TR	**Female**	18	22.04	1.35	0.32
	Male	27	22.45	2.37	0.46
L5 AP	**Female**	18	15.63	1.76	0.42
	Male	22	16.85	2.22	0.47
L5 TR	**Female**	19	23.99	1.71	0.39
	Male	24	25.11	2.09	0.43

[1] L = Lumbar vertebrae; AP = Antero-posterior diameter; - = No data; TR = transversal

Table 5.23: *Vertebral neural canal dimensions for the extra-London region during the late pre-Black Death period by sex*

Vertebrae	**Sex**	**N**	**Mean**	**Std. Deviation**	**Std. Error Mean**
L1 AP[1]	**Female**	23.00	16.71	1.06	0.22
	Male	11.00	16.29	1.53	0.46
L1 TR	**Female**	24.00	21.46	1.81	0.37
	Male	13.00	21.16	1.11	0.31
L2 AP	**Female**	22.00	15.74	1.68	0.36
	Male	14.00	15.35	1.70	0.45
L2 TR	**Female**	25.00	21.46	1.69	0.34
	Male	14.00	21.29	1.42	0.38
L3 AP	**Female**	20.00	14.64	1.49	0.33
	Male	16.00	14.25	1.76	0.44
L3 TR	**Female**	22.00	21.82	1.55	0.33
	Male	18.00	21.51	1.74	0.41
L4 AP	**Female**	21.00	14.34	2.00	0.44
	Male	16.00	14.76	2.07	0.52
L4 TR	**Female**	23.00	21.69	2.02	0.42
	Male	17.00	21.81	1.97	0.48
L5 AP	**Female**	21.00	15.40	2.29	0.50
	Male	15.00	15.96	2.27	0.59
L5 TR	**Female**	22.00	24.17	2.70	0.58
	Male	16.00	23.73	2.10	0.52

[1] L = Lumbar vertebrae; AP = Antero-posterior diameter; - = No data; TR = transversal

Table 5.24: *Vertebral neural canal dimensions for the London region during the late pre-Black Death period by sex*

Vertebrae	Sex	N	Mean	Std. Deviation	Std. Error Mean
L1 AP[1]	**Female**	5.00	17.01	0.54	0.24
	Male	3	16.05	0.66	0.38
L1 TR	**Female**	5.00	22.02	1.69	0.76
	Male	3	20.98	1.03	0.59
L2 AP	**Female**	5.00	16.42	0.68	0.30
	Male	3	14.96	0.79	0.46
L2 TR	**Female**	5.00	22.60	1.57	0.70
	Male	3	20.97	0.81	0.47
L3 AP	**Female**	5.00	15.19	0.56	0.25
	Male	3	14.41	0.52	0.30
L3 TR	**Female**	5.00	22.42	1.45	0.65
	Male	3	21.43	0.89	0.51
L4 AP	**Female**	4.00	15.95	0.67	0.33
	Male	3	15.57	1.88	1.09
L4 TR	**Female**	4.00	22.69	1.52	0.76
	Male	3	22.80	1.65	0.96
L5 AP	**Female**	3.00	17.57	0.76	0.44
	Male	2	16.36	0.25	0.18
L5 TR	**Female**	4.00	24.51	1.42	0.71
	Male	3	25.52	1.91	1.10

[1] L = Lumbar vertebrae; AP = Antero-posterior diameter; - = No data; TR = transversal

Comparisons between males and females for individuals from the extra-London region was only possible for the pre-Black Death period. Females showed smaller VNC dimensions for

L1 TR, L2, L4 and L5 (AP and TR), while males showed smaller dimensions for L1 AP and L3. However, these differences are very small.

Comparisons between males and females for individuals from the extra-London region was only possible for the pre-Black Death region. There were no significant differences between sexes.

The London region exhibited a tendency for males to have smaller VNC dimensions for all periods. This was observed in L1-L3, L4AP and L5AP during the pre-Black Death period, L1AP and L2-L4 during the Black Death. The only statistically significant differences were observed during the pre-Black Death period in L2AP (t(3.7) 3, p= 0.045) and during the Black Death period in L3AP (t(35.3) 2.1, p= 0.041). Comparisons between males and females were not possible for the post-Black Death period.

When comparing females and males in individuals dated to the early and late pre-Black Death periods, similarly as before, the differences between sexes were not statistically significant. In the London region, comparisons were only possible during the late pre-Black Death. No clear differences between males and females were observed.

When comparing same -sex categories, it was only possible to compare the pre-Black Death and the Black Death periods in London and the early and late pre-Black Death in the extra-London region. Results showed that in London females showed smaller dimensions of VNC during the black Death. This differences were statistically significant for L1AP (t21)=2.093, p=0.049), L2AP (t(22)=2.571, p=0.017), and L4AP (t(17)=3.294,p=0.004). Males also showed smaller dimensions during the Black Death period for all vertebrae excepting L2TR. However, these differences were not statistically significant. When comparing early and late pre-Black Death period in the extra-London region. there were not statistically significant differences observed.

5.3.2 Long bone osteometrics

No long bone data were available for non-adults from the extra-London sites of Barton-upon-Humber and Stoke Quay, while only femoral measurements were available for the adults at Barton-upon-Humber. The following analysis explores growth attainment using diaphyseal length of non-adults under the age of 12 years, and maximum long bone length for adults over the age of 18 years whose epiphyses were fused. These age ranges were chosen as the analysis of non-adult growth was done utilising charts by Maresh (1970) and modified by Spake and Cardoso (2021), which only include non-adults up to the age of 12 years. Analysis of adults did not include any growth chart and were analysed by comparing individuals from different periods and sexes. It was not possible to analyse growth for individuals ages 13-17 years as many had only one epiphysis fused and it was not possible in this investigation to measure the diaphyseal length.

5.3.2.1 Perinates

From the 40 individuals composing the sample, 30 had either measurable femora or humeri (n=30/40, 75%) and 12 were aged based on dentition (n=12/40, 30%).

From the total sample, 56.7% (n=17/30) individuals had both femur and humerus measurements to compare. Due to the small sample, analysis was only possible for the extra-London period during the pre-Black Death period, and within this period, the late pre-Black Death subperiod. The following table (5.25 shows age estimation comparison between the femur and the humerus based on The World Health Organization Fetal Growth Charts (Kiserud et al., 2017).

Table 5.25: *Comparison between age of gestation between femoral and humeral length by period for the extra-London region*

Region	Period	No difference		Femoral-Humeral[1]		Total
		n	%	n	%	N
Extra-London	**Pre-Black Death**	9	56.3	7	43.8	16
London	**Black Death**	0		0		0
	Post-Black Death	1	100	0		1
Total		10	58.8	7	41.2	17

[1] Differences in age estimation based on the humerus and the femur.

When comparing femoral and humeral age estimation, 7 individuals showed younger ages for humeral length than for femoral age (n=7/16, 43.8%) during the pre-Black Death period. From this sample, 6 were dated to the pre-Black Death subperiod (n=6/14, 42.9%). Statistical analysis was not possible.

Due to primary data collection including age estimation based on dentition, when looking at differences between early and late pre-Black Death subperiods comparison between dental, femoral, and humeral age estimation was possible (Table 5.26 and 5.27).

Table 5.26: *Comparison between age of gestation between femoral and humeral length by period for the extra-London region.*

Period	No difference		Femoral-Humeral[1]		Total
	n	%	n	%	N
Early pre-Black Death	1	50	1	50	2
Late pre-Black Death	3	33.3	6	66.7	9
Total	4	36.4	7	63.6	11

[1] Differences in age estimation based on the humerus and the femur.

Table 5.27: *Comparison between age of gestation between estimated dental age and diaphyseal length by period for the extra-London region.*

Period	**No difference**		**Dental-Diaphyseal**^{2}		**Total**
	n	**%**	**n**	**%**	**N**
Early pre-Black Death	1	100	0		1
Late pre-Black Death	8	57.1	6	42.9	14
Total	9	60	6	40	15

[2] Differences in age estimation based on dentition and diaphyseal length.

From the overall sample, 40% (n=6/15) showed humeral lengths that estimated ages younger than those based on the femoral lengths. This was all concentrated in individuals dated to the late pre-Black Death. When comparing age estimated from dentition and diaphyses, 7 out of 11 individuals (63.6%) had estimated gestational ages for the diaphyses. From this, 6 or 85.7% were for individuals dated to the late pre-Black Death and one was dated to the early Black-Death subperiod.

All of the observed differences were for the dental age to be the oldest age estimated, followed by gestational age based on femoral length, with the humeral length consistently providing the youngest ages.

5.3.2.2 Non-adults growth profiles

5.3.2.2.1 Pre-, post-, and Black Death periods

This analysis utilised measurements from 272 individuals under the age of 12 years from the extra-London and London regions and dated to the pre-, Black Death and post-Black Death periods.

A total of 680 bones were analysed. The following tables (5.28 and 5.29) show the distribution of recorded bones by period and subperiod respectively.

Table 5.28: *Distribution of recorded bones by period for non-adults ages 0 to 12 years from the extra-London and London regions.*

Region	**Period**	**Humerus**		**Radius**		**Ulna**		**Femur**		**Tibia**		**Fibula**		**Total**
		n	**%**	**n**	**%**	**n**	**%**	**n**	**%**	**n**	**%**	**n**	**%**	**N**
Extra-London	**Pre-Black Death**	60	20.3	49	16.6	48	16.2	64	21.6	51	17.2	24	8.1	296
	Post-Black Death	0		0		0		0		0		0		0
London	**Pre-Black Death**	7	15.9	4	9.1	15	34.1	5	11.4	9	20.5	4	9.1	44
	Black Death	50	19.6	34	13.3	33	12.9	53	20.8	65	25.5	20	7.8	255
	Post-Black Death	20	23.5	13	15.3	7	8.2	16	18.8	22	25.9	7	8.2	85
Total		137	20.2	100	14.7	103	15.2	138	20.3	147	21.6	55	8.1	680

Table 5.29: *Distribution of recorded bones by subperiod for non-adults ages 0 to 12 years from the extra-London and London regions.*

Region	**Subperiod**	**Humerus**		**Radius**		**Ulna**		**Femur**		**Tibia**		**Fibula**		**Total**
		n	**%**	**n**	**%**	**n**	**%**	**n**	**%**	**n**	**%**	**n**	**%**	**N**
Extra-London	**Early pre-Black Death**	11	22.9	8	16.7	8	16.7	9	18.8	7	14.6	5	10.4	48
	Late pre-Black Death	36	18.7	31	16.1	32	16.6	44	22.8	34	17.6	16	8.3	193
London	**Early pre-Black Death**	0		0		5	100	0		0		0		5
	Late pre-Black Death	7	18	4	10.3	10	25.6	5	12.8	9	23.1	4	10.3	39
Total		54	19	43	15.1	55	19.3	58	20.4	50	17.5	25	8.8	285

From a total of 680 recorded bones, 245 (36%) had a Z-score under -2SD. Figures showing the distribution of z-scores for each bone by period for each region was detailed in the appendix (see , section C.1).

Tables 5.30- 5.33 show a summary of the estimated Z-scores for each bone analysed in this thesis. Growth delay was identified when Z-scores were under -2SD.

In the extra-London sites, the fibula and femur bones provided the smallest values when compared to the means provided by the charts. The fibula presented n=15/24 (62.5%) and the femur exhibited n=33/64 (51.6%) individuals with Z-scores under -2SD. Within the London region, 33.3% (n=5/15 individuals) of the femoral lengths and 36.4% (n=8/22 individuals) tibial lengths fell below the 2SD level during the post-Black Death period, compared to 20% (n=1/5) and 11.1% (n=1/9) in the pre Black Death period respectively, and 26.4% (n=14/53) and 28.8% (n=19/66) during the Black Death, but none of the differences were significant and numbers of bones were small. When grouping all bones, higher frequencies of bones with evidence of growth delay were during the post-Black Death in London (n=27/82, 32.9%). The extra-London region showed higher frequencies of bones with evidence of growth delay during the pre-Black Death period compared to the London region (n=139/296, 47%; Two tailed Fisher exact test, p<0.001).

Table 5.30: *Z-score summary for diaphyseal length of the humerus and femur in non-adults by period and region.*

Region	**Period**	**Humerus**							**Femur**						
		⩽-2SD		**SD**		**⩾2SD**		**Total**	**⩽-2SD**		**SD**		**⩾2SD**		**Total**
		n	**%**	**n**	**%**	**n**	**%**	**N**	**n**	**%**	**n**	**%**	**n**	**%**	**N**
Extra-London	**Pre-Black Death**	26	43.3	33	55	1	1.7	60	33	51.6	29	45.3	2	3.1	64
	Black Death	0		0		0		0	0		0		0		0
	Post-Black Death	0		0		0		0	0		0		0		0
	Subtotal	26	43.3	33	55	1	1.7	60	33	51.6	29	45.3	2	3.1	64
London	**Pre-Black Death**	0		6	85.7	1		7	1	20	4	80	0		5
	Black Death	10	20	40	80	0		50	14	26.4	38	71.7	1	1.9	53
	Post-Black Death	7	36.8	12	63.2	0		19	5	33.3	10	66.7	0		15
	Subtotal	17	22.4	58	76.3	1	1.3	76	20	27.4	52	71.2	2	1.4	73
Total		43	31.6	91	66.9	2	1.5	136	53	38.7	81	59.1	3	2.2	137

Table 5.31: *Z-score summary for diaphyseal length of the ulna and tibia in non-adults by and period and region.*

Region	**Period**	**Ulna**							**Tibia**						
		⩽-2SD		**SD**		**⩾2SD**		**Total**	**⩽-2SD**		**SD**		**⩾2SD**		**Total**
		n	**%**	**n**	**%**	**n**	**%**	**N**	**n**	**%**	**n**	**%**	**n**	**%**	**N**
Extra-London	**Pre-Black Death**	21	43.8	25	52.1	2	4.2	48	25	49	23	45.1	3	5.9	51
	Black Death	0		0		0		0	0		0		0		0
	Post-Black Death	0		0		0		0	0		0		0		0
	Subtotal	21	43.8	25	52.1	2	4.2	48	25	49	23	45.1	3	5.9	51
London	**Pre-Black Death**	8	53.3	6	40	1	6.7	15	1	11.1	8	88.9	0		9
	Black Death	10	30.3	23	69.7	0		33	19	28.8	46	69.7	1	1.5	66
	Post-Black Death	3	42.9	4	57.1	0		7	8	36.4	14	63.6	0		22
	Subtotal	21	38.2	33	60	1	1.8	55	28	28.9	68	70.1	1	1	97
Total		42	40.8	58	56.3	3	2.9	103	53	35.8	91	61.5	4	2.7	148

Table 5.32: *Z-score summary for diaphyseal length of the radius and fibula in non-adults by and period and region.*

Region	**Period**	**Radius**							**Fibula**						
		⩽-2SD		**SD**		**⩾2SD**		**Total**	**⩽-2SD**		**SD**		**⩾2SD**		**Total**
		n	**%**	**n**	**%**	n	**%**	**N**	**n**	**%**	**n**	**%**	**n**	**%**	**N**
Extra-London	**Pre-Black Death**	19	38.8	28	57.1	2	4.1	49	15	62.5	8	33.3	1	4.2	24
	Black Death	0		0		0		0	0		0		0		0
	Post-Black Death	0		0		0		0	0		0		0		0
	Subtotal	19	38.8	28	57.1	2	4.1	49	15	62.5	8	33.3	1	4.2	24
London	**Pre-Black Death**	0		4	100	0		4	1	25	3	75	0		4
	Black Death	12	34.3	22	62.9	1	2.9	35	7	35	13	65	0		20
	Post-Black Death	3	23.1	10	76.9	0		13	1	16.7	4	66.7	1	16.7	6
	Subtotal	15	28.9	36	69.2	1	1.9	52	9	30	20	66.7	1	3.3	30
Total		34	33.7	64	63.4	3	2.9	101	24	44.4	28	51.9	2	3.7	54

Table 5.33: *Z-score summary for overall diaphyseal length in non-adults by period and region.*

Region	**Period**	**Total diaphseal length**						
		⩽-2SD		**SD**		**⩾2SD**		**Total**
		n	**%**	**n**	**%**	**n**	**%**	**N**
Extra-London	**Pre-Black Death**	139	47	146	49.3	11	3.7	296
	Black Death	0		0		0		0
	Post-Black Death	0		0		0		0
	Subtotal	139	47	146	49.3	11	3.7	296
London	**Pre-Black Death**	11	25	31	70.5	2	4.6	44
	Black Death	72	28	182	70.8	3	1.2	257
	Post-Black Death	27	32.9	54	65.9	1	1.2	82
	Subtotal	110	28.7	267	69.7	6	1.6	383
Total		249	36.7	413	60.8	17	2.5	679

5.3.2.2.2 The pre-Black Death period: early and late pre-Black Death subperiods

When discriminating between subperiods within the pre-Black Death period, 118 individuals were analysed. The overall sample was composed of 285 bones from which 113 (39.5%) presented evidence of growth delay.

Figures showing the distribution of z-scores for each bone by subperiod for each region was detailed in the appendix (see , section C.1). The scores were compiled in tables (5.34 and 5.37) to summarise the number of individuals with growth delay (⩽-2SD).

When comparing early and late pre-Black Death subperiods, the extra-London region showed a higher percentage of bones with evidence of growth delay in all bones for the late pre-Black Death subperiod (humerus: 14/36, 38.9%; radius: 12/31, 38.7%; ulna: 13/32, 40.6%; femur: 21/44 47.7%; tibia: 17/34, 50%; fibula: 10/16, 62.5%). For the London region, the only comparison possible was when observing the ulna. Records from this bone showed higher frequencies of growth delay in the early pre-Black Death subperiod (n=3/5, 60%). However, these differences were not statistically significant. When grouping all bones, the extra-London region presented higher frequencies of bones with evidence of growth delay during the late pre-Black Death period (X^2=7.05, df=1, p=0.008).

Table 5.34: *Z-score summary for diaphyseal length of the humerus and femur in non-adults by subperiod and region.*

Region	**Period**	**Humerus**							**Femur**						
		⩽-2SD		**SD**		**⩾2SD**		**Total**	**⩽-2SD**		**SD**		**⩾2SD**		**Total**
		n	**%**	**n**	**%**	**n**	**%**	**N**	**n**	**%**	**n**	**%**	**n**	**%**	**N**
Extra-London	**Early pre-Black Death**	3	27.3	7	63.6	1	9.1	11	4	44.4	4	44.4	1	11.1	9
	Late pre-Black Death	14	38.9	22	61.1	0		36	21	47.7	22	50	1	2.3	44
	Subtotal	17	36.2	29	61.7	1	2.1	47	25	47.2	26	49.1	2	3.8	53
London	**Early pre-Black Death**	0		0		0		0	0		0		0		0
	Late pre-Black Death	0		6	85.7	1	14.3	7	1	20	4	80	0		5
	Subtotal	0		6	85.7	1	14.3	7	1	20	4	80	0		5
Total		17	31.5	35	64.8	2	3.7	54	26	44.8	30	51.7	2	3.5	58

Table 5.35: *Z-score summary for diaphyseal length of the ulna and tibia in non-adults by and subperiod and region.*

Region	**Period**	**Ulna**							**Tibia**						
		⩽-2SD		**SD**		**⩾2SD**		**Total**	**⩽-2SD**		**SD**		**⩾2SD**		**Total**
		n	**%**	**n**	**%**	**n**	**%**	**N**	**n**	**%**	**n**	**%**	**n**	**%**	**N**
Extra-London	**Early pre-Black Death**	2	25	5	62.5	1	12.5	8	2	28.6	3	42.9	2	28.6	7
	Late pre-Black Death	13	40.6	18	56.3	1	3.13	32	17	50	16	47.1	1	2.9	34
	Subtotal	15	37.5	23	57.5	2	5	40	19	46.3	19	46.3	3	7.3	41
London	**Early pre-Black Death**	3	60	2	40	0		5	0		0		0		0
	Late pre-Black Death	5	50	4	40	1	10	10	1	11.1	8	88.9	0		9
	Subtotal	8	53.3	6	40	1	6.7	15	1	11.1	8	88.9	0		9
Total		23	41.8	29	52.7	3	5.5	55	20	40	27	54	3	6	50

Table 5.36: *Z-score summary for diaphyseal length of the radius and fibula in non-adults by and subperiod and region.*

Region	**Period**	**Radius**							**Fibula**						
		⩽-2SD		**SD**		**⩾2SD**		**Total**	**⩽-2SD**		**SD**		**⩾2SD**		**Total**
		n	**%**	**n**	**%**	**n**	**%**	**N**	**n**	**%**	**n**	**%**	**n**	**%**	**N**
Extra-London	**Early pre-Black Death**	2	25	5	62.5	1	12.5	8	2	40	2	40	1	20	5
	Late pre-Black Death	12	38.7	19	61.3	0		31	10	62.5	6	37.5	0		16
	Subtotal	14	35.9	24	61.5	1	2.6	39	12	57.1	8	38.1	1	4.8	21
London	**Early pre-Black Death**	0		0		0		0	0		0		0		0
	Late pre-Black Death	0		4	100	0		4	1	25	3	75	0		4
	Subtotal	0		4	100	0		4	1	25	3	75	0		4
Total		14	32.6	28	65.1	1	2.3	43	13	52	11	44	1	4	25

Table 5.37: *Z-score summary for overall diaphyseal lengths in non-adults by region during the pre-Black Death period.*

Region	**Period**	**Total diaphseal length**						
		⩽-2SD		**SD**		**⩾2SD**		**Total**
		n	**%**	n	**%**	n	**%**	**N**
Extra-London	**Early pre-Black Death**	15	31.2	26	54.2	7	14.6	48
	Late pre-Black Death	87	45.1	103	53.4	3	1.6	193
	Subtotal	102	42.3	129	53.5	10	4.2	241
London	**Early pre-Black Death**	3	60	2	40	0		5
	Late pre-Black Death	8	20.5	29	74.4	2	5.1	39
	Subtotal	11	25	31	70.5	2	4.6	44
Total		113	39.7	160	56.1	12	4.2	285

5.3.2.3 Adults

For this investigation, measurements of the maximal length of long bones were taken. This included femur, tibia, fibula, humerus, radius, and ulna. As sexual dimorphism affects height and limb length, this analysis only considered individuals whose sex was estimated as female or male. A total of 398 individuals and 1283 bones were measured.

Tables 5.38 and 5.39 show the distribution of measurable bones by sex and period from the extra-London and London regions. There were 193 recorded bones for the extra-London (94 females and 99 males) and 205 for the London (77 females and 128 males) region. Due to the small sample, analysis was only possible for the pre-Black Death period for the extra-London region (including early and late pre-Black Death subperiods), as well as the Black Death period for the London region.

Table 5.38: *Distribution of recorded bones by period for adults from the extra-London and London regions.*

Region	**Period**	**Sex**	**Humerus**		**Radius**		**Ulna**		**Femur**		**Tibia**		**Fibula**		**Total**
			n	**%**	**n**	**%**	**n**	**%**	**n**	**%**	**n**	**%**	**n**	**%**	**N**
Extra-London	**Pre-Black Death**	**Female**	74	22	62	18.4	49	14.5	63	18.7	56	16.6	33	9.8	337
		Male	71	20.3	59	16.9	63	18.1	64	18.3	58	16.6	34	9.7	349
	Post-Black Death	**Female**	2	20	1	10	2	20	2	20	2	20	1	10	10
		Male	7	18.9	5	13.5	3	8.1	10	27	9	24.3	3	8.1	37
London	**Pre-Black Death**	**Female**	13	31	8	19.1	9	21.4	3	7.1	6	14.3	3	7.1	42
		Male	11	17.2	14	21.9	9	14.1	13	20.3	12	18.8	5	7.8	64
	Black Death	**Female**	26	23.2	21	18.8	10	8.9	20	17.9	25	22.3	10	8.9	112
		Male	42	19.4	37	17.1	23	10.7	47	21.8	53	24.5	14	6.5	216
	Post-Black Death	**Female**	11	21.2	10	19.2	2	3.9	13	25	14	26.9	2	3.9	52
		Male	13	20.3	9	14.1	7	10.9	15	23.4	18	28.1	2	3.1	64
Total			270	21	226	17.6	177	13.8	250	19.5	253	19.7	107	8.3	1283

Table 5.39: *Distribution of recorded bones by subperiod for adults from the extra-London and London regions.*

Region	**Subperiod**	**Sex**	**Humerus**		**Radius**		**Ulna**		**Femur**		**Tibia**		**Fibula**		**Total**
			n	**%**	**n**	**%**	**n**	**%**	**n**	**%**	**n**	**%**	**n**	**%**	**N**
Extra-London	**Early pre-Black Death**	**Female**	21	22.34	20	21.3	15	16	18	19.2	13	13.8	7	7.5	94
		Male	26	22.6	20	17.4	21	18.3	22	19.1	17	14.8	9	7.8	115
	Late pre-Black Death	**Female**	39	21.4	31	17	25	13.7	34	18.7	33	18.1	20	11	182
		Male	33	17.4	32	16.8	34	17.9	34	17.9	36	19	21	11.1	190
London	**Early pre-Black Death**	**Female**	1	25	0		1	25	1	25	1	25	0		4
		Male	2	16.7	3	25	2	16.7	2	16.7	2	16.7	1	8.3	12
	Late pre-Black Death	**Female**	12	29.3	8	19.5	8	19.5	5	12.2	5	12.2	3	7.3	41
		Male	9	17.3	11	21.2	7	13.5	11	21.2	10	19.2	4	7.7	52
Total			143	20.7	125	18.1	113	16.4	127	18.4	117	17	65	9.4	690

The following tables show the means for males and females for the extra-London and London region by period (Figures 5.40-5.43) and subperiod (Figures 5.44-5.47). Due to th small size of the sample, it was not possible to analyse bone length from women

Table 5.40: *Long bone length for females from the extra-London region by period.*

Long Bone	Period	N	Mean	Std. Deviation	Std. Error Mean
Femur	**Pre-Black Death**	100	41.87	2.15	0.21
	Post-Black Death	2	44.70	2.40	1.70
Tibia	**Pre-Black Death**	55	34.06	2.39	0.32
	Post-Black Death	2	35.40	1.84	1.30
Fibula	**Pre-Black Death**	32	33.05	1.58	0.28
	Post-Black Death	1	33.20		
Radius	**Pre-Black Death**	61	22.22	1.27	0.16
	Post-Black Death	1	22.95		
Humerus	**Pre-Black Death**	73	30.03	1.51	0.18
	Post-Black Death	2	31.40	0.00	0.00
Ulna	**Pre-Black Death**	49	24.41	1.28	0.18
	Post-Black Death	2	24.53	0.46	0.33

Table 5.41: *Long bone length for males from the extra-London region by period.*

Long Bone	Period	N	Mean	Std. Deviation	Std. Error Mean
Femur	**Pre-Black Death**	110	45.82	2.28	0.22
	Post-Black Death	10	47.80	3.32	1.05
Tibia	**Pre-Black Death**	58	37.00	2.43	0.32
	Post-Black Death	9	38.21	2.33	0.78
Fibula	**Pre-Black Death**	35	35.86	2.69	0.45
	Post-Black Death	3	35.83	0.94	0.54
Radius	**Pre-Black Death**	61	24.90	2.10	0.27
	Post-Black Death	5	25.30	0.65	0.29
Humerus	**Pre-Black Death**	73	32.91	1.83	0.21
	Post-Black Death	7	33.95	2.11	0.80
Ulna	**Pre-Black Death**	65	26.82	1.42	0.18
	Post-Black Death	4	25.79	2.35	1.17

Table 5.42: *Long bone length for females from the London region by period.*

Long Bone	Period	N	Mean	Std. Deviation	Std. Error Mean
Femur	**Pre-Black Death**	6	41.32	1.39	0.57
	Black Death	20	42.13	2.58	0.58
	Post-Black Death	13	42.96	1.90	0.53
Tibia	**Pre-Black Death**	6	33.55	0.75	0.31
	Black Death	25	33.38	1.85	0.37
	Post-Black Death	14	33.80	1.81	0.48
Fibula	**Pre-Black Death**	3	32.83	1.64	0.95
	Black Death	10	32.91	2.16	0.68
	Post-Black Death	2	32.30	1.56	1.10
Radius	**Pre-Black Death**	8	21.89	1.04	0.37
	Black Death	21	22.15	1.29	0.28
	Post-Black Death	10	23.32	3.99	1.26
Humerus	**Pre-Black Death**	13	29.73	0.91	0.25
	Black Death	25	29.96	1.46	0.29
	Post-Black Death	11	30.71	1.91	0.58
Ulna	**Pre-Black Death**	9	24.20	1.12	0.37
	Black Death	10	24.51	1.54	0.49
	Post-Black Death	4	25.38	1.74	0.87

Table 5.43: *Long bone length for males from the London region by period.*

Long Bone	Period	N	Mean	Std. Deviation	Std. Error Mean
Femur	**Pre-Black Death**	13	45.68	2.19	0.61
	Black Death	47	45.15	2.65	0.39
	Post-Black Death	15	45.93	1.79	0.46
Tibia	**Pre-Black Death**	12	36.92	2.33	0.67
	Black Death	53	35.90	2.14	0.29
	Post-Black Death	18	36.81	2.20	0.52
Fibula	**Pre-Black Death**	5	34.58	2.00	0.89
	Black Death	14	35.01	1.94	0.52
	Post-Black Death	2	35.95	0.35	0.25
Radius	**Pre-Black Death**	14	24.26	1.40	0.38
	Black Death	37	24.20	1.26	0.21
	Post-Black Death	9	24.31	1.78	0.59
Humerus	**Pre-Black Death**	11	33.35	2.04	0.62
	Black Death	42	32.46	1.70	0.26
	Post-Black Death	13	33.58	1.23	0.34
Ulna	**Pre-Black Death**	9	25.98	1.56	0.52
	Black Death	23	25.97	0.97	0.20
	Post-Black Death	7	26.46	1.55	0.59

Table 5.44: *Long bone length for females from the extra-London region during the pre-Black Death period.*

Long Bone	**Subperiod**	**N**	**Mean**	**Std. Deviation**	**Std. Error Mean**
Femur	**Early pre-Black Death**	31	41.59	1.81	0.33
	Late pre-Black Death	47	42.23	2.27	0.33
Tibia	**Early pre-Black Death**	13	32.77	1.72	0.48
	Late pre-Black Death	33	34.68	2.32	0.40
Fibula	**Early pre-Black Death**	7	31.84	1.96	0.74
	Late pre-Black Death	20	33.57	1.28	0.29
Radius	**Early pre-Black Death**	20	21.86	0.99	0.22
	Late pre-Black Death	31	22.48	1.37	0.25
Humerus	**Early pre-Black Death**	21	29.50	1.59	0.35
	Late pre-Black Death	39	30.26	1.47	0.24
Ulna	**Early pre-Black Death**	15	23.86	1.14	0.29
	Late pre-Black Death	25	24.79	1.18	0.24

Table 5.45: *Long bone length for males from the extra-London region during the pre-Black Death period.*

Long Bone	**Subperiod**	**N**	**Mean**	**Std. Deviation**	**Std. Error Mean**
Femur	**Early pre-Black Death**	41	45.78	2.14	0.33
	Late pre-Black Death	51	45.78	2.39	0.33
Tibia	**Early pre-Black Death**	17	37.18	3.19	0.77
	Late pre-Black Death	36	36.85	2.01	0.34
Fibula	**Early pre-Black Death**	9	36.80	2.10	0.70
	Late pre-Black Death	22	35.35	2.81	0.60
Radius	**Early pre-Black Death**	20	25.15	1.21	0.27
	Late pre-Black Death	33	25.04	2.42	0.42
Humerus	**Early pre-Black Death**	26	33.31	1.58	0.31
	Late pre-Black Death	34	32.78	1.68	0.29
Ulna	**Early pre-Black Death**	21	27.36	1.17	0.26
	Late pre-Black Death	36	26.55	1.36	0.23

Table 5.46: *Long bone length for females from the London region during the pre-Black Death period.*

Long Bone	Period	N	Mean	Std. Deviation	Std. Error Mean
Femur	**Early pre-Black Death**	1	40.60		
	Late pre-Black Death	5	41.46	1.51	0.67
Tibia	**Early pre-Black Death**	1	33.10		
	Late pre-Black Death	5	33.64	0.80	0.36
Fibula	**Early pre-Black Death**	0a			
	Late pre-Black Death	3	32.83	1.64	0.95
Radius	**Early pre-Black Death**	0a			
	Late pre-Black Death	8	21.89	1.04	0.37
Humerus	**Early pre-Black Death**	1	29.80		
	Late pre-Black Death	12	29.73	0.95	0.27
Ulna	**Early pre-Black Death**	1	24.00		
	Late pre-Black Death	8	24.23	1.19	0.42

a. t cannot be computed because at least one of the groups is empty.

Table 5.47: *Long bone length for males from the London region during the pre-Black Death period.*

Long Bone	Subperiod	N	Mean	Std. Deviation	Std. Error Mean
Femur	**Early pre-Black Death**	2	46.10	1.41	1.00
	Late pre-Black Death	11	45.61	2.34	0.71
Tibia	**Early pre-Black Death**	2	36.10	0.28	0.20
	Late pre-Black Death	10	37.08	2.54	0.80
Fibula	**Early pre-Black Death**	1	35.40		
	Late pre-Black Death	4	34.38	2.24	1.12
Radius	**Early pre-Black Death**	3	24.43	1.03	0.59
	Late pre-Black Death	11	24.21	1.53	0.46
Humerus	**Early pre-Black Death**	2	32.65	2.90	2.05
	Late pre-Black Death	9	33.51	2.00	0.67
Ulna	**Early pre-Black Death**	2	26.10	1.56	1.10
	Late pre-Black Death	7	25.94	1.69	0.64

Results showed that females from the extra-London region had smaller means for all long bone metrics during the pre-Black Death period. Similarly, males showed smaller long bone length for all bones during the pre-Black Death period excepting the ulna and fibula. The only statistically significant difference was observed for the humerus in females (t(72) -7.8, $p<0.001$).

When comparing the pre-Black Death and Black Death periods, females in London exhibited smaller long bone length during the pre-Black Death for all bones except the tibia. Comparisons with the post-Black Death period were not possible. Different to this, when comparing males dated to the pre-, post- and Black Death periods, the last one showed smaller lengths for all bones except. However, the only statistically significant difference was observed when comparing humeral length between the Black Death and post-Black Death periods (t(27.5) -2.6, $p=0.015$).

When comparing the same periods, the London region showed smaller long bone length for both males and females during the pre-Black Death period for all bones except for the humerus in females. Comparisons between individuals dated to the post-Black Death period was only possible for males. Results were similar, showing smaller lengths in the London region for the femur, tibia, humerus and radius. None of these differences were statistically significant.

When differentiating early and late-pre Black Death subperiods, the extra-London region showed smaller long bone lengths for females dated to the early pre-Black Death subperiod for all bones. This was statistically significant for the tibia (t(29.6) -3.1, p=0.005) and ulna (t(30.4) -2.5, p=0.019). Different to this, males showed smaller lengths during the late pre-Black Death subperiod for all bones except the radius. This was statistically significant for the ulna (t(45.6) 2-2, p=0.03). The London region presented a smaller sample size and comparisons were only possible for males. This showed no clear differences between subperiods.

When comparing the same subperiods, the London region exhibited smaller lengths for males during the early period. The late period showed a less clear pattern for males, with smaller lengths for femur, fibula, radius and ulna. Similarly, females exhibited smaller length for the London region for all analysed long bones. However, none of these differences were statistically significant.

5.4 Cribra orbitalia

5.4.1 Presence of cribra orbitalia

The following tables shows the crude prevalence rates of cribra orbitalia by age and sex in the extra-London (Table 5.48) and London (Table 5.49) regions.

Table 5.48: *Crude prevalence rates of cribra orbitalia by age, sex, and period for the extra-London region.*

Age (years)	**Sex**	**Pre-Black Death CO/N**[1]	**Pre-Black Death %**	**Post-Black Death CO/N**	**Post-Black Death %**	**Total N**
Perinate		0/34		0/0		34
0-5		80/561	14.3	0/1		562
6-12		44/182	24.2	0/2		184
13-17		17/96	17.7	0/1		97
Non-adult		0/2		0/0		2
Non-adult subtotal		141/875	16.1	0/4		879
18-25	**Female**	21/70	30	1/1	100	71
	Male	15/73	20.6	1/4	25	77
	Unknown	1/5	20	0/0		5
26-35	**Female**	19/140	13.6	0/2		142
	Male	18/149	12.1	0/8		157
	Unknown	0/10		0/0		10
Adult subtotal		74/447	16.6	2/15	13.3	462
Subtotal		215/1322	16.3	2/19	10.5	1341

[1] CO = number of individuals with cribra orbitalia; N = Number of individuals; Non-adult = unaged non-adult

Table 5.49: *Crude prevalence rates of cribra orbitalia by age, sex, and period for the London region.*

Age	**Sex**	**Pre-Black Death**		**Black Death**		**Post-Black Death**		**Total**
(years)		**CO/N[1]**	**%**	**CO/N**	**%**	**CO/N**	**%**	**N**
Perinate		0/0		0/5		0/1		6
0-5		0/8		0/68		3/39	7.7	115
6-12		1/22	4.6	5/73	6.9	2/23	8.7	118
13-17		2/13	15.4	2/31	6.5	1/24	4.2	68
Non-adult		0/6		0/39		0/20		65
Non-adult subtotal		3/49	7	7/216	3.2	6/107	5.6	372
18-25	**Female**	0/6		3/16	18.6	3/13	23.1	35
	Male	1/18	5.6	7/36	19.4	1/19	5.3	73
	Unknown	0/1		0/11		0/3		15
26-35	**Female**	0/10		5/38	13.2	3/24	12.5	72
	Male	1/29	3.5	9/68	13.2	4/40	10	137
	Unknown	1/1	100	0/15		0/4		119
Adult subtotal		3/65	4.6	24/184	13	11/103	10.7	352
Subtotal		6/114	5.3	31/400	7.8	17/210	8.1	742

[1] CO = number of individuals with cribra orbitalia; N = Number of individuals; Non-adult = unaged non-adult

The overall sample exhibited 13.1% (n=271/2065) of all individuals with evidence of cribra orbitalia. In the extra-London sample, 16.1% of individuals had cribra orbitalia (n=217/1341) with 16.6% (n=74/447) of lesions in the adults compared to 16.1% (n=141/875) in the non-adults. The percentage of lesions in adults and children in the pre-Black Death periods was the same at 16%. The sample sizes was much smaller for the post-Black Death period with only 19 individuals with orbits, and two individuals affected (10.5%, n=19/181).

The pre-Black Death period exhibited the majority of the individuals. Estimated females showed evidence of cribra orbitalia in 40 individuals (19%, n=40/210) and males showed evidence of cribra orbitalia in 14.9% (n=33/222). No statistically significant differences were observed when comparing adult females to males or between same sex categories for the pre- or post-Black Death periods.

The London sample exhibited lower frequencies of individuals with cribra orbitalia than the extra- London region. Overall, 7.5% (n=54/724) highest frequency of cribra orbitalia in the smaller pre-Black Death period was in the non-adults at 6.1%. However, in the post-Black death period adults had twice as much cribra orbitalia than the non-adults at 10.7%. The adult sample was 352, from which 317 had estimated sex (90.1%), with 13.1% (n=14/107) females presenting with cribra orbitalia compared to 11% (n=23/210) males. No statistically significant differences were observed when comparing same age or sex categories, nor when comparing females to males between periods.

When comparing both regions, adults dated to the pre-Black Death period from the London region exhibited statistically significant lower frequencies of cribra orbitalia than the ones from the extra-London region (Two tailed Fisher exact test, p=0.01). (Tables 5.48 and 5.49).

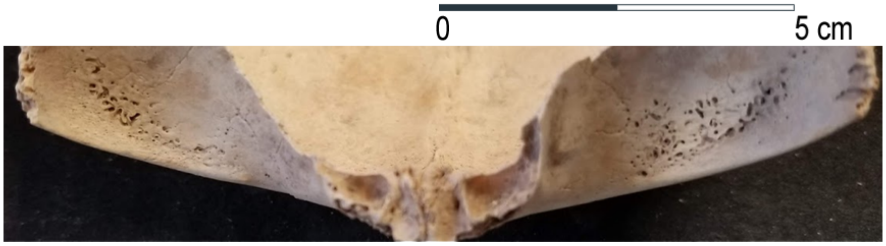


Figure 5.5: *Example of Cribra orbitalia, individual G304, Wharram Percy*

Table 5.50 summarises crude prevalence rates of cribra orbitalia for the extra-London and London regions during the pre-Black Death period.

Table 5.50: *Crude prevalence rates of cribra orbitalia by age, sex during the pre-Black Death period for the extra-London and London regions.*

Region	**Age**	**Sex**	**Early pre-Black Death**		**Late pre-Black Death**		**Total**
	(years)		**CO/N[1]**	**%**	**CO/N**	**%**	**N**
Extra-London	**Perinate**		0/2		0/31		32
	0-5		30/174	17.2	36/270	13.3	444
	6-12		11/52	21.2	28/98	28.6	150
	13-17		9/40	22.5	6/35	17.1	75
	Non-adult		0/ 0		0/2		2
	Non-adult subtotal		50/268	56	70/434	16.1	704
	18-25	**Female**	5/22	22.7	8/30	26.7	52
		Male	4/24	16.7	5/28	17.9	52
		Unknown	0/1		1/2	50	3
	26-35	**Female**	7/55	12.7	9/47	19.2	102
		Male	6/64	9.4	7/52	13.5	116
		Unknown	0/5		0/2		7
	Adult subtotal		22/171	12.9	30/163	18.4	334
Subtotal			71/439	16.2	100/597	16.8	1036
London	**Perinate**		0/0		0/0		500
	0-5		0/3		0/5		8
	6-12		0/3		1/19	5.3	22
	13-17		0/0		2/13	15.4	13
	Non-adult		0/0		0/6		6
	Non-adult subtotal		0/6		3/43	7	49
	18-25	**Female**	0/0		0/6		6
		Male	1/1	100	0/17		18
		Unknown	0/0		0/1		1
	26-35	**Female**	0/1		0/9		10
		Male	0/2		1/27	3.7	29
		Unknown	0/0		1/1	100	1
	Adult subtotal		1/4	25	2/67	3	71
Subtotal			1/10	10	5/104	4.8	114
Total			72/449	16	105/701	15	1150

[1] CO = number of individuals with cribra orbitalia; N = Number of individuals; non-adult = unaged non-adult

The extra-London region was comprised by 49/268 (18.3%) non-adults and 22/171 (12.87%) adults with evidence of cribra orbitalia for the early Black Death period and 70/436 (16.06%) non-adults and 30/161 (18.63%) adults for the late pre-Black Death period.

From the total adult sample, 97% (n=322/332) was estimated as female or male. From this sample, 15.6% (n=12/77) females and 11.4% (n=10/88) males dated to the early pre-Black Death subperiod presented cribra orbitalia. The late pre-Black Death subperiod exhibited 22.1% (n=17/77) females and 15% (n=12/80) males. No statistically significant differences were observed between subperiods for same age and sex categories. Within each period, no differences between males and females were observed.

The samples were too small to make any inferences about cribra orbitalia during the pre-Black Death period in London. In the late pre-Black Death period, 4.8% of individuals (n=5/104) had cribra orbitalia, with the children showing over double the prevalence of the adults at 7% and females showing no evidence of cribra and males exhibiting only 4.3% (n=47) compared to males. No statistically significant differences were observed.

When comparing regions, the only statistically significant difference was observed in the late pre- Black death period where the extra-London region exhibited higher frequencies for adults (Two tailed Fisher's exact text p=0.004).

5.4.2 Severity of cribra orbitalia

Figures 5.6 and 5.7 and Table 5.51 present the prevalence of cribra orbitalia by severity (Stuart-Macadam (1991) Grades 3-5) for the extra-London and London regions.

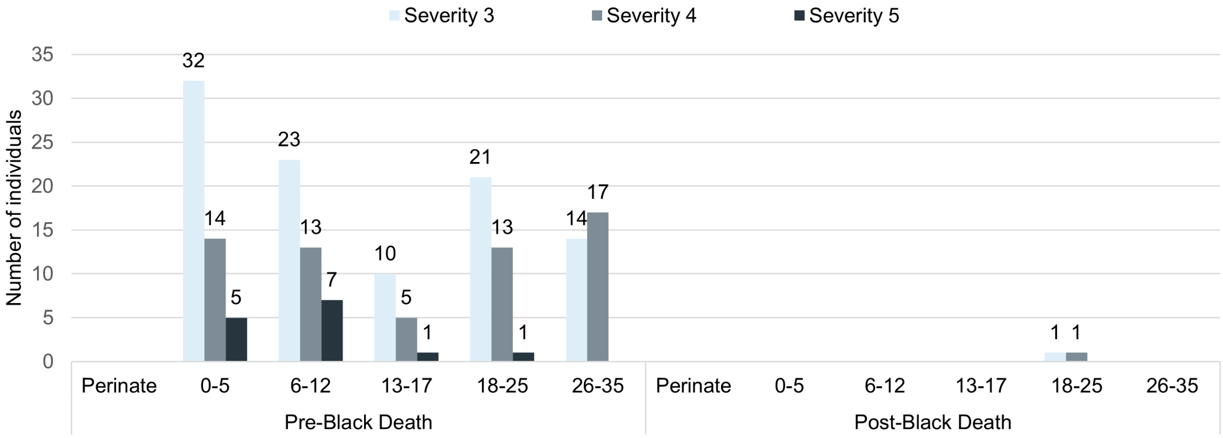


Figure 5.6: *Prevalence of cribra orbitalia by severity and period for individuals from the extra-London region.*

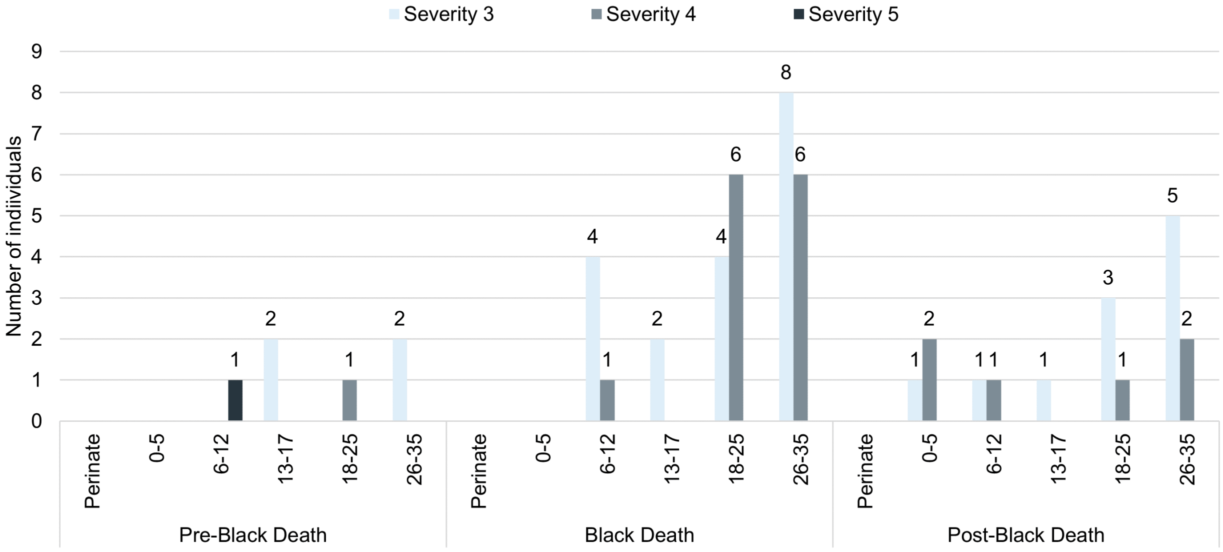


Figure 5.7: *Prevalence of cribra orbitalia by severity and region for individuals from the London region.*

Table 5.51: *Frequencies of cribra orbitalia by severity, period, and region*

Region	**Period**	**Age**	**Severity 3**		**Severity 4**		**Severity 5**		**Total**
			n	**%**	**n**	**%**	**n**	**%**	**N**
Extra-London	**Pre-Black Death**	**Non-adults**	65	59.1	32	29.1	13	11.8	110
		Adults	35	53	30	45.5	1	1.5	66
		Subtotal	100	56.8	62	35.2	14	8	176
	Post-Black Death	**Non-adults**	0		0		0		0
		Adults	1	50	1	50	0		2
		Subtotal	1	50	1	50	0		2
	Subtotal		101	56.7	63	35.4	14	7.9	178
London	**Pre-Black Death**	**Non-adults**	2	66.7	0		1	33.33	3
		Adults	2	66.7	1	33.3	0		3
		Subtotal	4	66.7	1	16.7	1	16.7	6
	Black Death	**Non-adults**	6	85.71	1	14.3	0		7
		Adults	12	50	12	50	0		24
		Subtotal	18	58.1	13	41.9	0		31
	Post-Black Death	**Non-adults**	3	50	3	50	0		6
		Adults	8	72.7	3	27.3	0		11
		Subtotal	11	64.7	6	35.3	0		17
	Subtotal		33	61.1	20	37	1	1.9	54
Total			104	51.5	83	41.1	15	7.4	202

From the overall sample of individuals with cribra orbitalia, 85.6% could be evaluated for severity (n=232/271). In the extra-London both non-adults and adults dated to the pre-Black Death period showed a majority of individuals with cribra severity 3 (non-adults n=65/110, 59.1%; adults n=35/66, 53%), followed by severity 4 (non-adults n=32/110, 29.1%; adults n=30/110 45.5%), and lastly severity 5 (non-adults n=13/110, 11.8%; adults n=1/110, 1.52%). When comparing non-adults to adults, non-adults showed higher frequencies of individuals with cribra orbitalia severity 5 (two-tailed Fisher's exact test p=0.02), and adults exhibited higher frequencies of individuals with cribra orbitalia severity 4 (X^2=4.84, df=1, p=0.03). The post-Black Death period showed only two adult individuals with cribra orbitalia; one with severity 3 and one with severity 4.

The London region presented a smaller sample with a similar composition to the extra-London region. Both non-adults and adults showed higher frequencies of cribra orbitalia severity 3 for all periods with the exception of adults dated to the Black Death and non-adults dated to the post-Black Death. In both these instances, individuals with cribra orbitalia severity 3 and 4 presented equal frequencies (Black Death period adults n=12/24, 50%; Post-Black Death non-adults n=3/6, 50%).

Due to the small sample size for the post-Black Death period in the extra-London region and the pre-Black Death period in London, statistical analysis comparing periods was not possible.

The following table (5.52) and figure (5.8) show the frequencies of cribra orbitalia severity by subperiod for the extra-London region. The London region only presented five individuals with evidence of cribra orbitalia, one adult dating to the early pre-Black Death subperiod with evidence severity 4 and four individuals (two non-adults and two adults) dated to the late pre-Black Death subperiod with cribra orbitalia severity 3.

The extra-London sample showed a higher proportion of severity 3 for non-adults dated to the early (n=22/37, 59.5%) and late pre-Black Death (n=34/63, 54%) subperiods and adults dated to the late pre-Black Death subperiod (n=20/30, 66.7%). Adults dated to the early pre-Black Death period showed a higher proportion of severity 4 (n=11/17, 64.7%). When comparing age categories, adults showed higher frequencies of individuals with cribra orbitalia severity 4 than non-adults (n=11/37, n=29.7%). When comparing same-age categories between subperiods, adults showed higher frequencies of severity 4 during the early pre-Black Death subperiod compared to the late pre-Black Death period (X^2=5.35, df=1, p=0.02) and higher frequencies of severity 3 during the late pre-Black Death subperiod compared to the early pre-Black Death period (X^2=4.32, df=1, p=0.04).

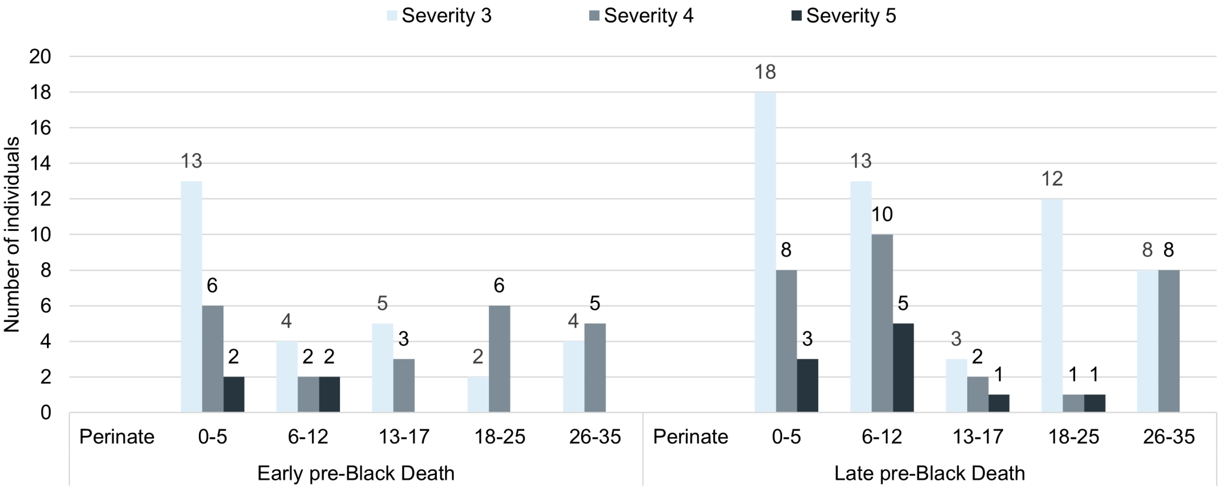


Figure 5.8: *Prevalence of cribra orbitalia by severity and subperiod for individuals from the extra-London region.*

Table 5.52: *Frequencies of cribra orbitalia by severity and subperiod for individuals from the extra-London region.*

Period	**Age**	**Severity 3**		**Severity 4**		**Severity 5**		**Total**
		n	**%**	**n**	**%**	**n**	**%**	**N**
Early pre-Black Death	**Non-adults**	22	59.46	11	29.73	4	10.81	37
	Adults	6	35.29	11	64.71	0		17
	Subtotal	28	51.85	22	40.74	4	7.41	54
Late pre-Black Death	**Non-adults**	34	53.97	20	31.75	9	14.29	63
	Adults	20	66.67	9	30.00	1	3.33	30
	Subtotal	54	58.06	29	31.18	10	10.75	93
Total		82	55.8	51	34.7	14	9.5	147

5.5 Adolescent development

5.5.1 Puberty stage estimation

Puberty stage analysis was limited to primary evidence and data from the site Barton-upon-Humber (M. Lewis, personal communication, 2021); hence, this information is not available for the London region. Adolescence was determined to be between the ages of 10 and 25 years. This age range was chosen as it comprised the period from the start of the development of secondary sexual characteristics until the brain fully matures (**?**Rogol et al., 2002). This age range was also used for previous bioarchaeological analysis such as DeWitte and Lewis (2021), so it allowed for the comparing of results.

In this investigation, one of the reasons the age category was extended to 35 years of age was the possibility that people suffering from undernutrition or disease may have had pubertal development delayed beyond the age of 25 years (Lewis et al., 2015). However, results did not show any individuals over the age of 25 years who had not completed their pubertal development. For this reason and to compare results from this analysis with previous studies, the age cohort was limited to 10-25 years of age.

A total of 105 individuals fell into the adolescent age cohort (10-25 years). The majority of whom were dated to the pre- Black Death period (n=97/105, 92.4%), while eight individuals were dated to the post-Black Death period (n=8/105, 7.6%) (Table 5.53). For this reason, the analysis concentrated in evaluating pubertal development in the pre-Black Death periods (Table 5.54). The majority of the adolescents were male (n=68/105, 64.8%), compared to females (n=33/105, 31.4%) and individuals for whom sex could not be estimated (n=4/105, 3.8%).

Figures 5.9-5.11 present the percentage of adolescents who died within each puberty stage by estimated sex and age during the pre-Black Death period. The completion stage of puberty showed a majority of representation in the overall sample (n=42/105, 43.3%) followed by initiation (onset) (n=13/132, 13.4%). Both males and females showed higher frequencies of individuals in the completion stage of pubertal development (females n=14/30, 46.7%; males n=28/63, 44.4%). For females the following stage with more representation is deceleration (n=6/30, 20%). For males, acceleration (n=9/63, 14.3%) was the second most represented stage of pubertal development. When comparing females and males, no statistically significant differences were observed in either pubertal stage of development.

Table 5.53: *Composition of the adolescent sample by age, sex, and period for the extra-London region*

Age	**Pre-Black Death**					**Post-Black Death**					**Total**				
(years)	**Females**		**Males**		**N**	**Females**		**Males**		**N**	**Females**		**Males**		**N**
	n	**%**	**n**	**%**		**n**	**%**	**n**	**%**		**n**	**%**	**n**	**%**	
10	3	33.3	6	66.7	9	0		0		0	3	33.3	6	66.7	9
11	1	20	4	80	5	2	100	0		2	3	42.9	4	57.1	7
12	0		1	100	1	0		0		0	0	0	1	100	1
13	0		3	100	3	0		0		0	0	0	3	100	3
14	0		2	100	2	0		0		0	0	0	2	100	2
15	1	20	4	80	5	0		0		0	1	20	4	80	5
16	1	20	4	80	5	0		0		0	1	20	4	80	5
17	0		2	100	2	0		1	100	1	0	0	3	100	3
18	1	14.3	6	85.7	7	0		0		0	1	14.3	6	85.7	7
19	3	42.9	4	57.1	7	0		1	100	1	3	37.5	5	62.5	8
20-21	10	41.7	14	58.3	24	1	33.3	2	66.7	3	11	40.7	16	59.3	27
22-25	10	43.5	13	56.5	23	0		1	100	1	10	41.7	14	58.3	24
Total	30	32.3	63	67.7	93	3	37.5	5	62.5	8	33	32.7	68	67.3	101

Table 5.54: *Composition of the adolescent sample by age, sex, and subperiod for the extra-London region.*

Age	**Early pre-Black Death**					**Late pre-Black Death**					**Total**				
(years)	**Females**		**Males**		**N**	**Females**		**Males**		**N**	**Females**		**Males**		**N**
	n	**%**	**n**	**%**		**n**	**%**	**n**	**%**		**n**	**%**	**n**	**%**	
10	0		1	100	1	1	20	4	80	5	1	16.7	5	83.3	6
11	0		1	100	1	1	33.3	2	66.7	3	1	25	3	75	4
12	0		0		0	0		1	100	1	0		1	100	1
13	0		1	100	1	0		0		0	0		1	100	1
14	0		1	100	1	0		1	100	1	0		2	100	2
15	1	33.3	2	66.7	3	0		1	100	1	1	25	3	75	4
16	1	100	0		1	0		0		0	1	100	0		1
17	0		0		0	0		1	100	1	0		1	100	1
18	0		1	100	1	1	50	1	50	2	1	33.3	2	66.7	3
19	1	33.3	2	66.7	3	0		1	100	1	1	25	3	75	4
20-21	4	66.7	2	33.3	6	4	33.3	8	66.7	12	8	44.4	10	55.6	18
22-25	1	20	4	80	5	7	87.5	1	12.5	8	8	61.5	5	38.5	13
Total	8	34.8	15	65.2	23	14	40	21	60	35	22	37.9	36	62.1	58

Due to its small sample, both sexes were grouped together for the post-Black Death period (Figure 5.12). Similarly to the pre-Black Death period, the most represented stage of pubertal development was completion with n=4/8 individuals (50%), followed by initiation (onset) (n=2/8, 25%). When comparing pubertal stages between pre and post-Black Death periods, no statistically significant differences were observed.

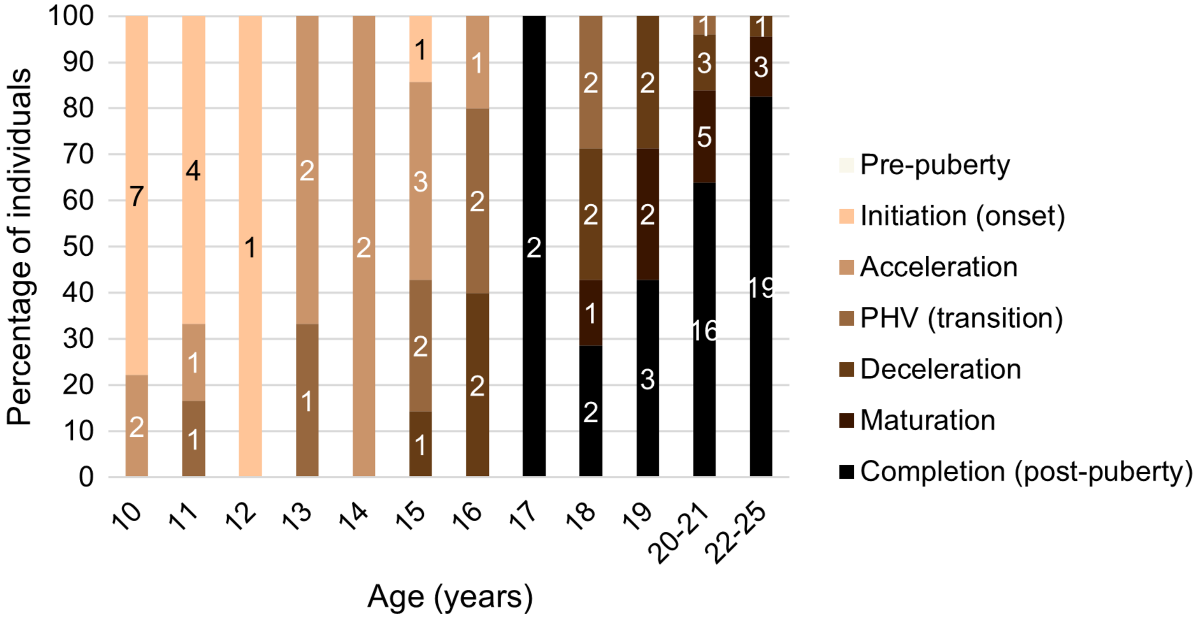


Figure 5.9: *Pubertal stages by age in adolescents dated to the pre-Black Death period in the extra-London region.*

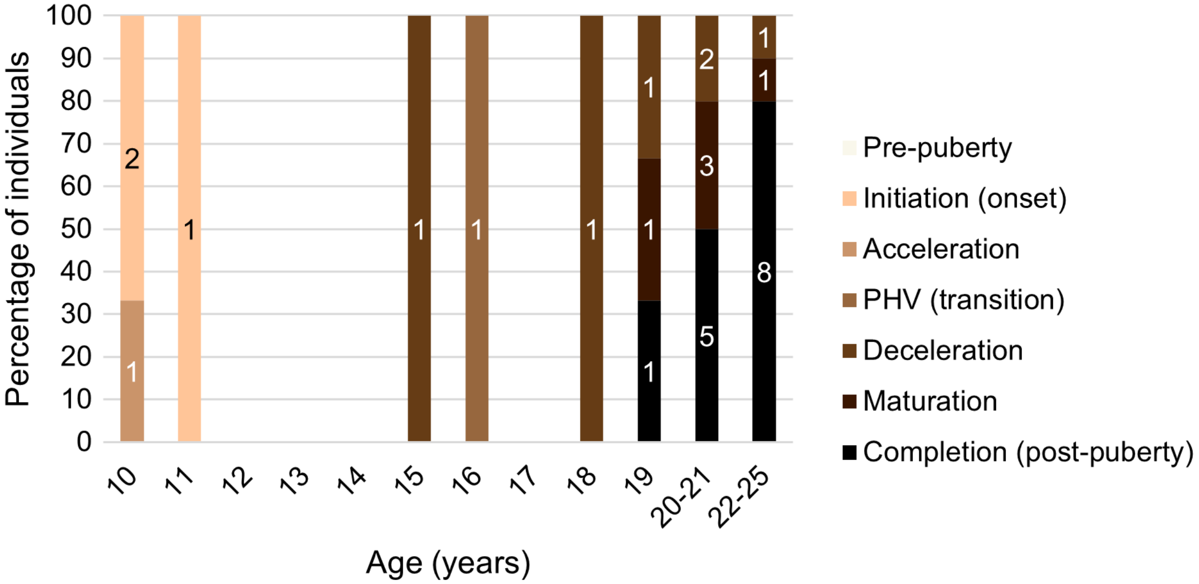


Figure 5.10: *Pubertal stages by age in female adolescents dated to the pre-Black Death period in the extra-London region.*

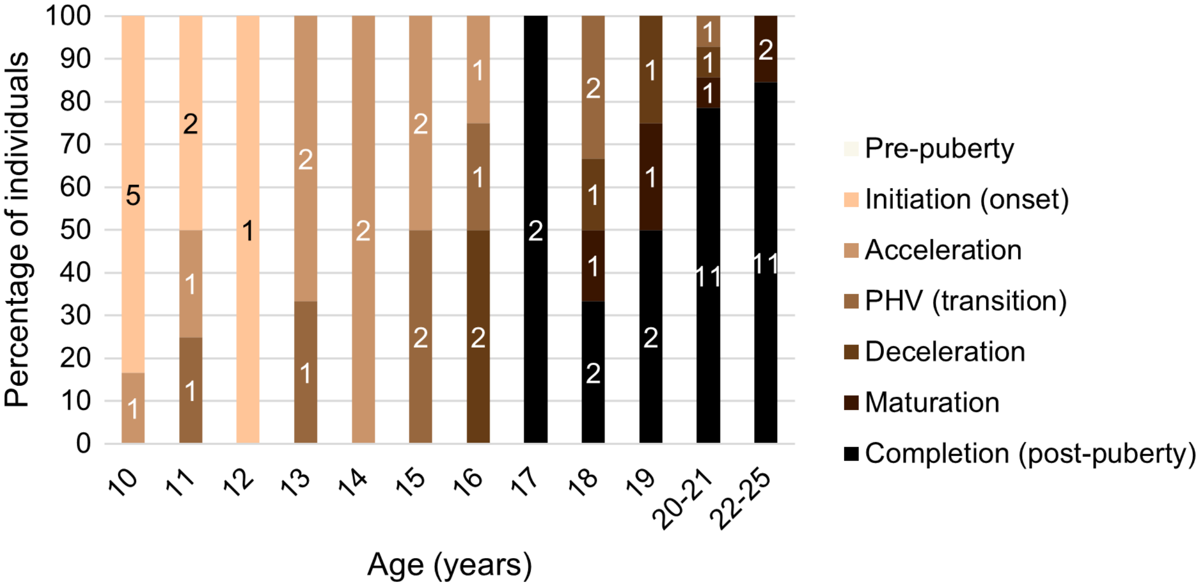


Figure 5.11: *Pubertal stages by age in male adolescents dated to the pre-Black Death period in the extra-London region.*

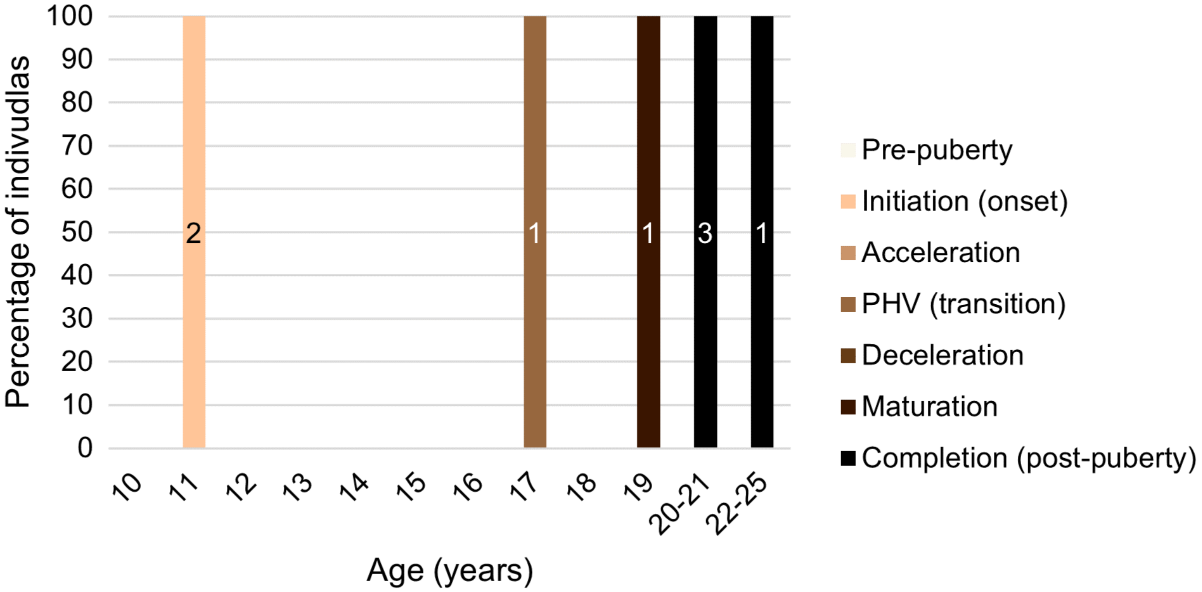


Figure 5.12: *Pubertal stages by age in adolescents dated to the post-Black Death period in the extra-London region.*

Due to the small sample, comparisons with the post-Black Death were not possible. When comparing mean ages for each pubertal stage between males and females dated to the pre-Black Death period, comparisons were only possible for deceleration, maturation, and completion (post-puberty). Females showed higher mean ages for deceleration and completion (post-puberty), while initiation and maturation showed almost equal ages (Table 5.55).

Table 5.55: *Mean ages for each pubertal stage for females and males dated to the pre-Black Death period in the extra-London region.*

Pubertal status	Sex	N	Mean age	Std. Deviation	Std. Error Mean
Initiation (on set)	**Female**	5	10.99	0.34	0.15
	Male	8	10.79	0.82	0.29
Acceleration	**Female**	1	10.05		
	Male	9	13.83	1.85	0.62
PHV (transition)	**Female**	1	15.95		
	Male	10	16.74	3.11	0.98
Deceleration	**Female**	6	19.63	2.43	0.99
	Male	5	18.20	2.36	1.06
Maturation	**Female**	5	20.68	1.42	0.63
	Male	6	20.62	1.94	0.79
Completion (post-puberty)	**Female**	15	22.02	1.87	0.48
	Male	31	21.10	2.02	0.36

Within the pre-Black Death group (Figures 5.13-5.18), the late pre-Black Death period exhibited the highest proportion of individuals (n=39/62, 62.9%). The most represented pubertal stage in both subperiods was completion (early pre-Black Death subperiod n=9/23, 39.1%, late pre-Black Death subperiod n=15/39, 38.5%). When looking at female-male distribution, statistical analysis was only possible for the late pre-Black Death subperiod as the sample dated to the early pre-Black Death subperiod was too small. The early pre-Black Death showed 8 (34.8%) and 15 males (65.2%), while the late pre-Black Death period exhibited 14 females (40%) and 21 males (60%). The completion stage of development was the most represented for all sexes (early pre-Black Death males n=6/15, 40%, late pre-Black Death females n=7/14, 50%, and late pre-Black Death males n=8/21, 38.1%). The only exception was early pre-Black Death females who were represented equally in both the maturation and completion stages of development (n=3/8, 37.5%).

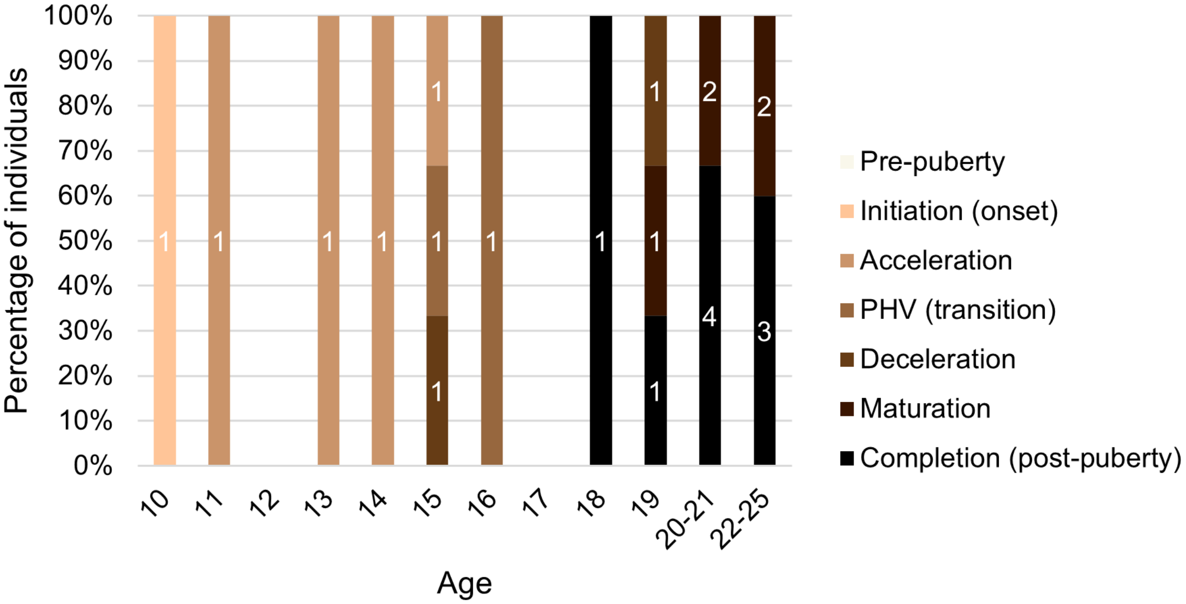


Figure 5.13: *Pubertal stages by age in adolescents dated to the early pre-Black Death sub-period in the extra-London region.*

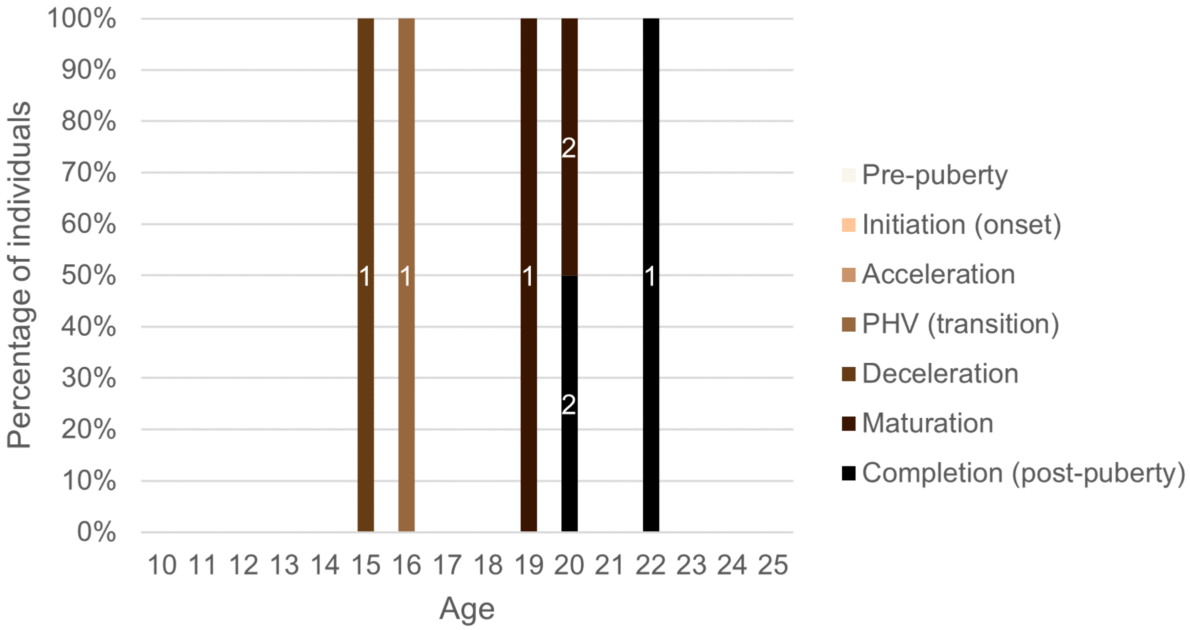


Figure 5.14: *Pubertal stages by age in female adolescents dated to the early pre-Black Death subperiod in the extra-London region.*

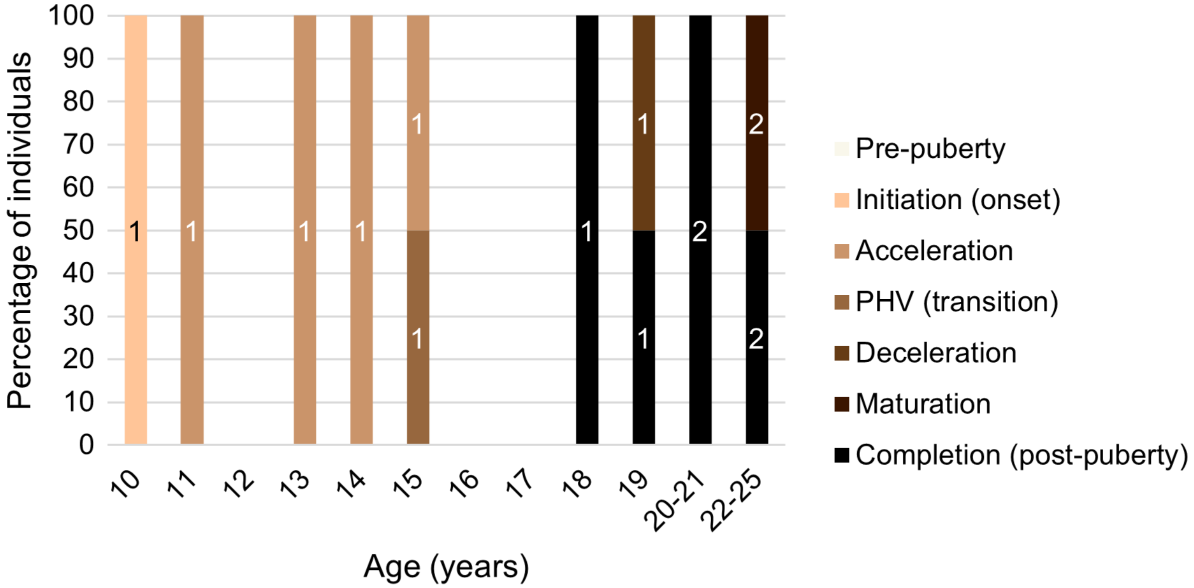


Figure 5.15: *Pubertal stages by age in male adolescents dated to the early pre-Black Death subperiod in the extra-London region.*

Figure 5.16: *Pubertal stages by age in adolescents dated to the late pre-Black Death subperiod in the extra-London region.*

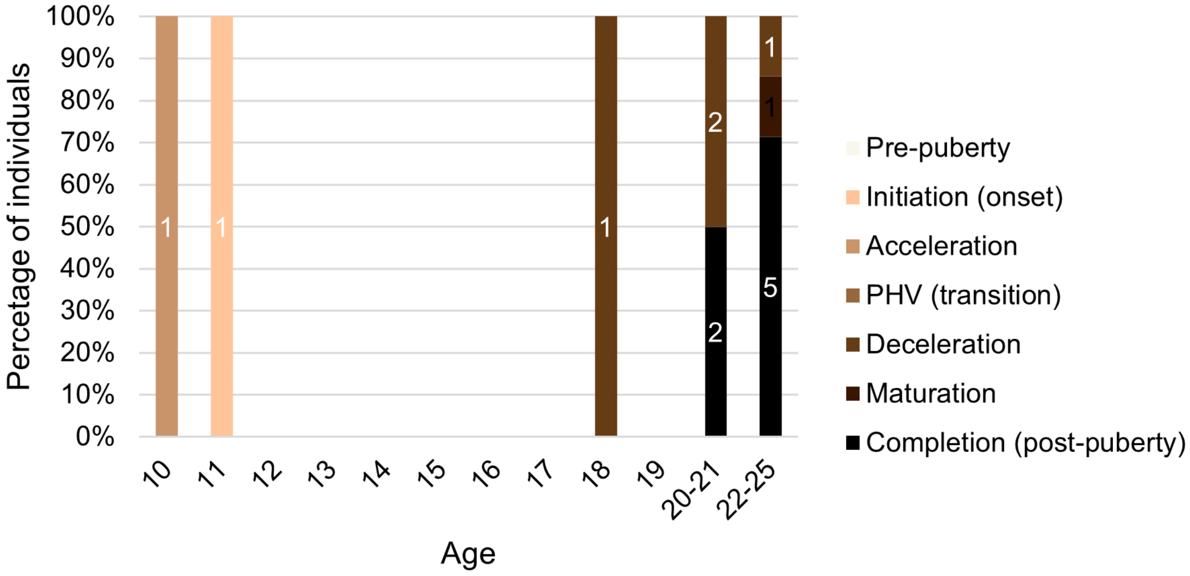


Figure 5.17: *Pubertal stages by age in female adolescents dated to the late pre-Black Death period in the extra-London region.*

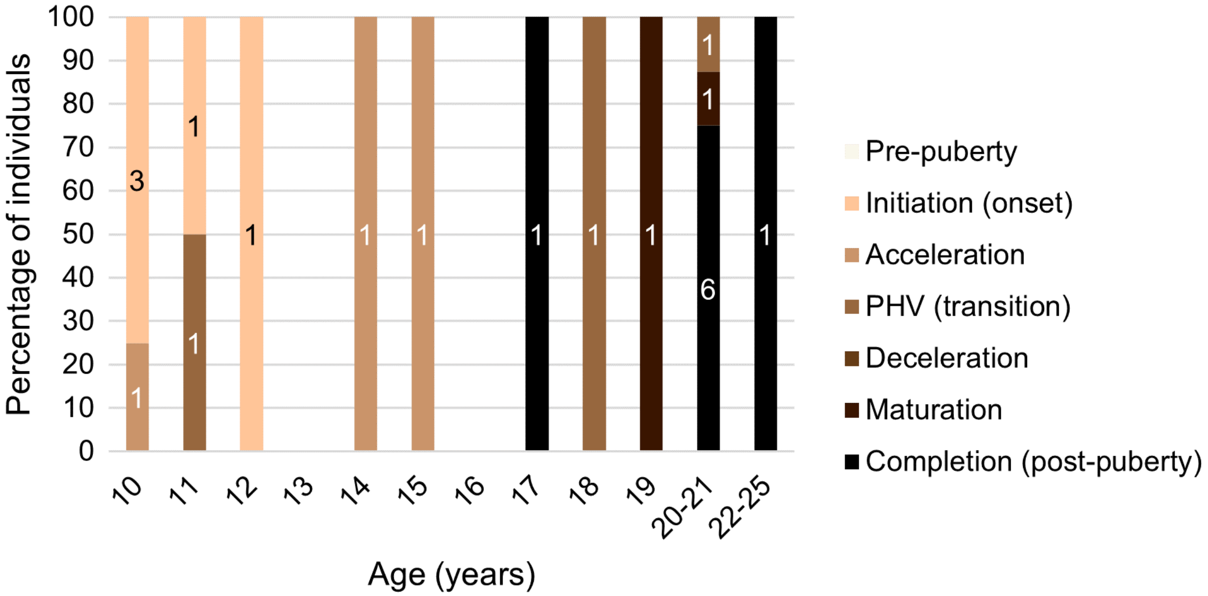


Figure 5.18: *Pubertal stages by age in male adolescents dated to the late pre-Black Death subperiod in the extra-London region.*

When comparing the mean ages for each pubertal stage between subperiods, the late pre-Black Death subperiod showed higher mean ages for stages 1, 3, 4 and 6, while the early pre-Black Death subperiod exhibited higher mean ages for stages 5. Stage 2 presented almost no differences (Table 5.55). None of these differences were statistically significant.

Table 5.56: *Mean ages for each pubertal stage for individuals dated to the early and late pre-Black Death in the extra-London region.*

Pubertal status	Subperiod	N	Mean age	Std. Deviation	Std. Error Mean
Initiation (on set)	**Early pre-Black Death**	1	10.80		
	Late pre-Black Death	8	11.59	1.82	0.64
Acceleration	**Early pre-Black Death**	4	13.60	1.88	0.94
	Late pre-Black Death	5	13.22	2.59	1.16
PHV (transition)	**Early pre-Black Death**	2	15.48	0.67	0.48
	Late pre-Black Death	3	17.07	4.81	2.78
Deceleration	**Early pre-Black Death**	2	17.50	2.83	2.00
	Late pre-Black Death	4	20.75	1.55	0.78
Maturation	**Early pre-Black Death**	5	21.06	1.77	0.79
	Late pre-Black Death	4	20.69	1.62	0.81
Completion (post-puberty)	**Early pre-Black Death**	9	20.77	1.96	0.65
	Late pre-Black Death	15	21.58	2.05	0.53

5.5.2 Age of menarche

The menarche sample comprised 33 females 30 (90.9%) dated to the pre-Black Death and 3 dated to the post-Black Death period (10.1%). Individuals dated to the pre-Black Death period could be estimated to be pre- and post-menarche based on the development of the distal phalanges and the fusion of the iliac crest. Table 5.57 and Figures 5.19 and 5.20 present the mean ages of pre- and post menarcheal females in the pre-Black Death period.

The average ages of pre-menarche and post-mnarche females during the pre-Black Death period were 14.3 years and 21.5 years respectively. In the early pre-Black death cohort the mean age was higher at 15.7 years, decreasing to 14.1 years in the late pre Black Death period. Mean post-menarcheal ages in the early pre-Black death period were earlier at 20.3 years despite menarche appearing to start at an older age (15.7 years) than in the later phase. Conversely, an earlier mean pre-menarcheal age in the later Black Death period was followed by an older post-menarcheal age of 22 years, suggesting an extended developmental period.

This difference in post-menarcheal ages was statistically significant (t(17)=-2.01, p=0.05).

Table 5.57: Pre-menarche and post-menarche mean ages by period

Menarche	Period	N	Mean age	Std. Deviation	Std. Error Mean
Pre-menarche	**Pre-Black Death**	8	14.34	4.23	1.49
	Post-Black Death	2	11		
	Early pre-Black Death	2	15.73	0.32	0.23
	Late pre-Black Death	3	14.18	5.95	3.43
Post-menarche	**Pre-Black Death**	24	21.49	1.82	0.37
	Post-Black Death	1	20		
	Early pre-Black Death	6	20.31	1.10	0.45
	Late pre-Black Death	13	22.03	1.85	0.51

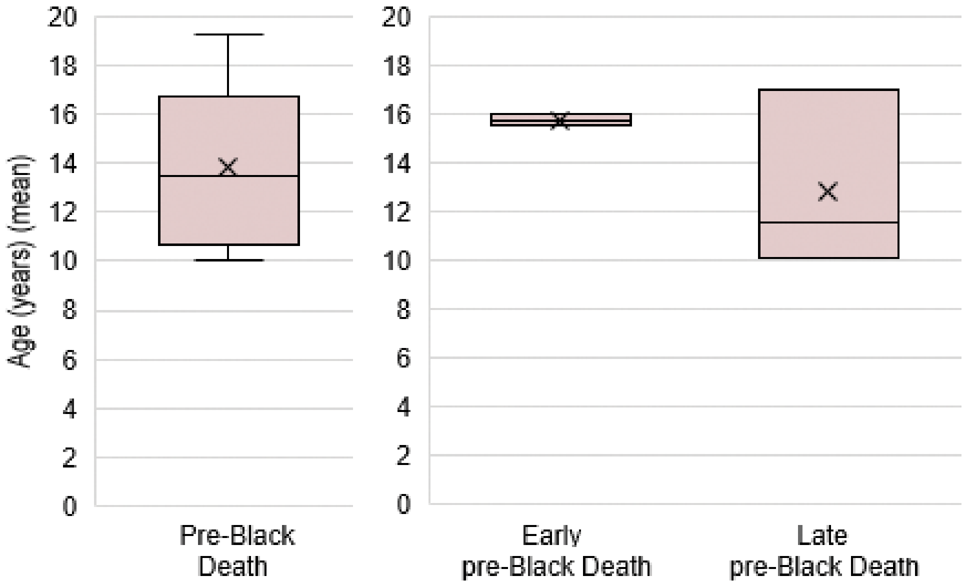


Figure 5.19: Pre-menarche mean age by period and subperiod.

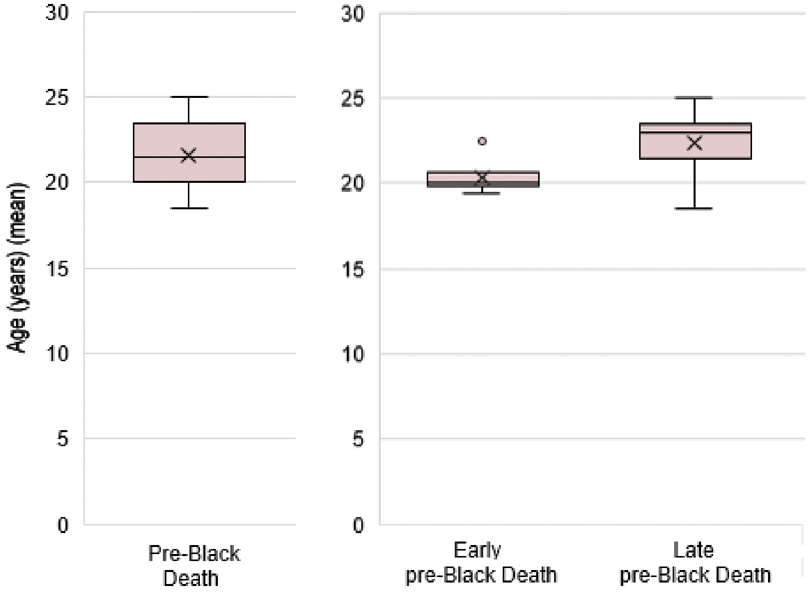


Figure 5.20: *Post-menarche mean age by period and subperiod.*

5.6 Specific infections and vitamin deficiencies

The following tables show the crude prevalence rates of pathology for the extra-London and London regions comparing the pre-Black Death, Black Death, and post-Black Death periods (Tables 5.58 and 5.59).).

The overall sample showed 1.2% (n=25/2065) of individuals with evidence of scurvy, 1% (n=20/2065) with evidence of rickets, 0.5% (n=10/2065) of individuals with tuberculosis. Similarly to when analysing cribra orbitalia, due to the secondary sources of information only reporting cases of pathology rather than presence and absence, only crude prevalence rates could be estimated. This likely reduced the observed rates as it includes skeletons that did not have enough evidence to evaluate a diagnosis.

Table 5.58: *Crude prevalence rates of scurvy, rickets, and tuberculosis by age, sex, and period for the extra-London region*

Age	**Sex**	**Pre-Black Death**							**Post-Black Death**						
(years)		**Scurvy**		**Rickets**		**TB[1]**		**N**	**Scurvy**		**Rickets**		**TB**		**N**
		n	**%**	**n**	**%**	**n**	**%**		**n**	**%**	**n**	**%**	**n**	**%**	
Perinate		0		0		0		34	0		0		0		0
0-5		19	3.4	8	1.4	0		561	0		0		0		1
6-12		2	1.1	1	0.6	0		182	0		0		0		2
13-17		0		1	1	0		96	0		0		0		1
Non-adult		0		0		0		2	0		0		0		0
Non-adult subtotal		21	2.4	10	1.1	0		875	0		0		0		4
18-25	**Female**	0		1	1.4	2	2.7	70	0		0		0		1
	Male	1	1.4	2	2.7	1	1.4	73	0		0		0		4
	Unknown	0		1	20	0		5	0		0		0		0
26-35	**Female**	0		0		2	1.4	140	0		0		0		2
	Male	0		1	0.7	0		149	0		1	12.5	0		8
	Unknown	0		0		0		10	0		0		0		0
Adult subtotal		1	0.2	5	1.1	5	1.1	447	0		1	6.7	0		15
Total		22	1.7	15	1.1	5	0.4	1322	0		1	5.3	0		19

[1] TB = tuberculosis; Non-adult = Unaged non-adult

Table 5.59: *Crude prevalence rates of scurvy, rickets, and tuberculosis by age, sex, and period for the London region*

Age	**Sex**	**Black Death**							**Post-Black Death**						
(years)		**Scurvy**		**Rickets**		**TB**[1]		**N**	**Scurvy**		**Rickets**		**TB**		**N**
		n	**%**	**n**	**%**	**n**	**%**		**n**	**%**	**n**	**%**	**n**	**%**	
Perinate		0		0		0		5	0		0		0		1
0-5		0		1	1.5	2	2.9	68	2	5.1	0		0		39
6-12		0		0		0		73	1	4.4	0		0		23
13-17		0		0		0		31	0		0		2	8.3	24
Non-adult		0		0		0		39	0		1	5	0		20
Non-adult subtotal		0		1	0.5	2	0.9	216	3	2.8	1	0.9	2	1.9	107
18-25	**Female**	0		0		0		16	0		0		0		13
	Male	0		0		0		36	0		0		1	5.3	19
	Unknown	0		0		0		11	0		0		0		3
26-35	**Female**	0		0		0		38	0		1	4.2	0		24
	Male	0		0		0		68	0		1	2.5	0		40
	Unknown	0		0		0		15	0		0		0		4
Adult subtotal		0		0		0		184	0		2	1.9	1	1	103
Total		0		1	0.3	2	0.5	400	3	1.4	3	1.4	3	1.4	210

[1] TB = tuberculosis; Non-adult = Unaged non-adult

When discriminating by period and region, most pathologies are observed during the pre-Black Death period in the extra-London region and in post-Black Death London. This distribution is similar to the overall sample composition. When combining both regions and comparing periods, the pre-Black Death period (extra-London region) showed higher frequencies of scurvy (n=22/1322; 1.7%), while the post-Black Death period (London region) exhibited higher frequencies of rickets (n=4/229, 1.7%) and tuberculosis (n=3/229, 1.3%). However, the differences were not statistically significant. The only statistically significant differences were observed when comparing overall pathologies and scurvy frequencies during the pre-Black Death period in the extra-London region with the Black Death period in the London region, where the first showed higher frequencies (X^2=6.72, df=1, p=0.001 and X^2=6.20, df=1, p=0.01 respectively).

The following table shows frequencies of scurvy, rickets, and tuberculosis by period and sex for the pre-Black Death period (Table 5.60) .

Table 5.60: *Frequencies of scurvy, rickets, and tuberculosis by period and age for the pre-Black Death period for the extra-London region.*

Subperiod	**Age**	**Scurvy**		**Rickets**		**Tuberculosis**		**N**
		n	**%**	**n**	**%**	**n**	**%**	
Early pre-Black Death	**Non-adults**	5	1.9	1	0.4	0		268
	Adults	0		1	0.6	3	1.8	171
	Subtotal	5	1.1	2	0.5	3	0.7	439
Late pre-Black Death	**Non-adults**	16	3.7	6	1.4	0		436
	Adults	1	0.6	4	2.5	2	1.2	161
	Subtotal	17	2.8	10	1.7	2	0.3	597
Total		22	2.1	12	1.2	5	0.5	1036

When comparing the early and late pre-Black Death periods, pathologies were only recorded for the late pre-Black Death extra-London region (n=40/1050, 3.8%). The majority of the cases were vitamin deficiencies (scurvy and rickets) (n=34/1050, 3.2%). When comparing subperiods, the late pre-Black Death period exhibited higher frequencies of scurvy and rickets (n=17/597, 2.8% and n=10/597, 1.7% respectively). However, no statistically significant differences were observed.

5.7 Spina bifida occulta

To assess whether congenital conditions related to maternal health varied between the regions and time periods, spina bifida occulta was recorded. Cases were only recorded in individuals dated to the pre-Black Death period outside London, as the secondary datasets did not differentiate between absence of spina bifida and when the pathology was not observable. For this reason, only crude prevalence rates were considered. As spina bifida occulta is only observable in individuals older than 15, only the age categories 13-17, 18-25 and 25-35 years were considered. The total of the sample was 543 individuals with 210 females, 222 males and 111 individuals of unknown sex (this latter category was composed mostly of individuals ages 13 to 17 (96/111 (86.5%)) 16 or 2.9% had spina bifida occulta (Figure 5.21 and Table 5.61). The only statistically significant difference was observed when comparing the total number of individuals between the early and late pre-Black Death period, when the latter showed higher frequencies (two tailed Fisher's exact test; 11/196, 5.6%, p=0.03). While there was a trend for males to have a higher prevalence of the lesions, this pattern was not statistically significant.

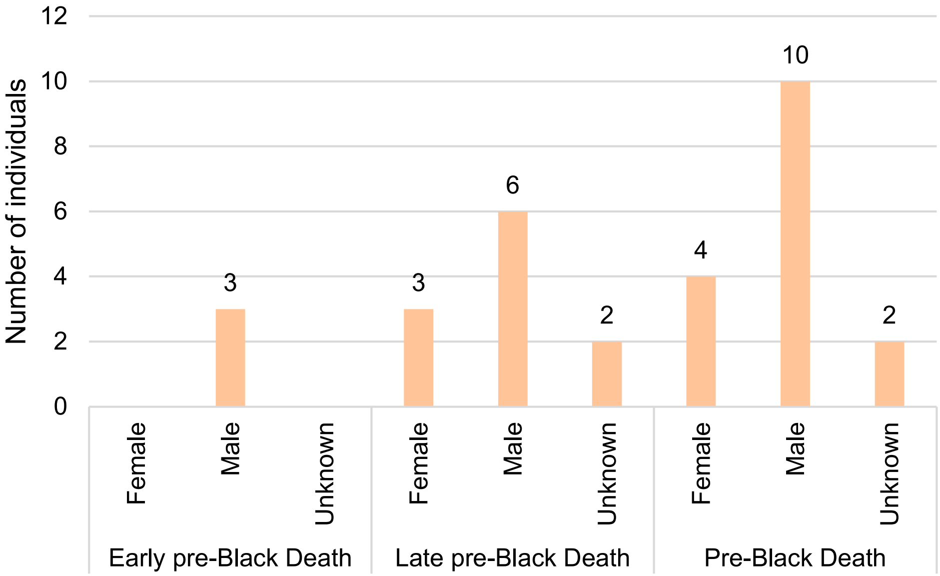


Figure 5.21: *Distribution of spina bifida occulta by period for the extra-London region.*

Table 5.61: *Crude prevalence of spina bifida occulta in individuals aged 13-35 years dated to the pre-Black Death period from the extra-London region.*

Sex	**Early pre-Black Death**		**Late pre-Black Death**		**Pre-Black Death**[1]	
	SB/N[2]	%	**SB/N**	%	**SB/N**	%
Females	0/77		3/77	3.9	4/210	1.9
Males	3/88	3.4	6/80	7.5	10/222	4.5
Unknown	0/46		2/39	5.1	2/111	1.8
Total	3/211	1.4	11/196	5.6	16/543	2.9

[1] Pre-Black Death= includes individuals dated to both early, late pre-Black Death as well an individuals dated to the pre-Black Death period that could not be dated to a specific subperiod; [2] SB= individuals with spina bifida occulta, N= number of individuals.

Spina bifida is cause by a defect of the neural canal (Northrup and Volcik, 2000). However, when looking at the correlation between people with evidence of spina bifida occulta and vertebral neural canal dimensions of the lumbar vertebrae (See , table C.1), no significant differences in mean vertebral neural canal dimensions were observed.

5.8 Dental enamel hypoplasia

5.8.1 Crude rates

Dental enamel hypoplasia was collected from the primary and secondary sites. Primary dental data for the mandibular molars and maxillary canines was only available for the extra-London sites, while information on the tooth types compiled from the secondary data varied, with no information for non-adult teeth available from Barton-upon-Humber. For reasons of comparison, only data for the canine was included in the enamel hypoplasia analysis. Secondary data for dental disease often included which teeth were affected, but do not differenciate unaffected teeth from teeth that were not present or were not observable. Hence, prevalence rates for dental disease are calculated by individuals (i.e. crude prevalence rates). As dental enamel hypoplasia was not observable in perinates, they were not considered for the analysis. similarly, unaged non-adult were not considered either as they did not present dentition. Removing all non-adults from Barton-upon-Humber, perinates and unaged non-adults reduced the total sample size from 2065 to 1647 individuals.

The following table shows the crude prevalence of dental enamel hypoplasia by age, sex, and period for the extra-London and London regions.

From a total of 1647 individuals, 14.7% (287/1958) individuals exhibited evidence of DEH.

Statistical differences were observed when comparing regions where adult females dated to the pre-Black Death period presented higher frequencies of dental enamel hypoplasia in London (9/16, 56.25%, X^2=6.9, df=1, p=0.0009) compared top the extra-London region. Within each region, London showed higher frequencies of dental enamel hypoplasia during the Black Death period (74/184, 40.22%) compared to the pre-Black Death period (14/65, 21.54%). When comparing males to females, the only differences were found in the London region, where females exhibited higher prevalence rates of dental enamel hypoplasia during the pre- and post-Black Death periods (9/16, 56.25%, two tailed Fisher's exact test; 20/37, 54.05%, p=0.0002 and X^2=16.24, df=1, $p<0.0001$).

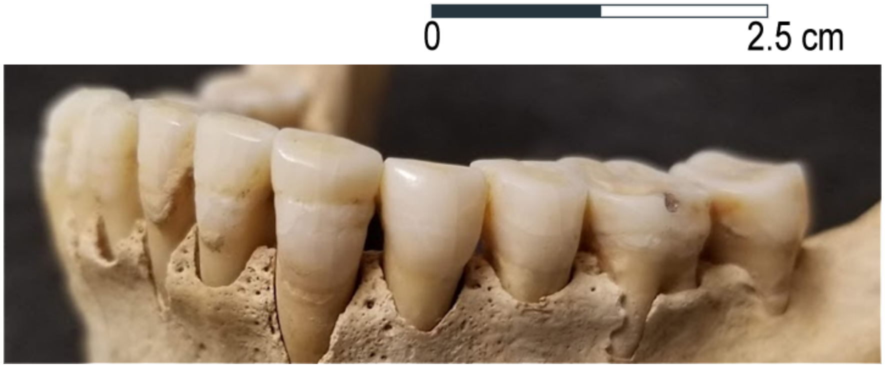


Figure 5.22: *Example of linear enamel hypoplasia, individual NA038, Wharram Percy*

Table 5.62: *Crude prevalence of individuals with linear enamel hypoplasia by age, sex, and period for the extra-London and London regions.*

Region	**Age**	**Sex**	**Pre-Black Death**		**Black Death**		**Post-Black Death**		**N**
	(years)		**DEH/N**[1]	**%**	**DEH/N**	**%**	**DEH/N**	**%**	
Extra-London	**0-5**		2/344	0.6	0/0		0/1		562
	6-12		25/122	20.5	0/0		1/2	50	184
	13-17		10/62	16.1	0/0		0/1		97
	Non-adult subtotal		37/528	7	0/0		1/4	25	532
	18-25	**Female**	19/70	27.1	0/0		0/1		71
		Male	20/73	27.4	0/0		1/4	25	77
		Unknown	1/5	20	0/0		0/0		5
	26-35	**Female**	35/140	25	0/0		1/2	50	142
		Male	30/149	20.1	0/0		3/8	37.5	157
		Unknown	1/10	10	0/0		0/0		10
	Adult subtotal		106/447	23.7	0/0		5/15	33.3	462
	Subtotal		143/975	14.7	0/0		6/19	31.6	994
London	**0-5**		0/8		0/68		0/39		115
	6-12		022		2/73	2.7	2/23	8.7	118
	13-17		0/13		10/31	32.3	4/24	16.7	68
	Non-adult subtotal		0/43		12/172	7	6/86	7	301
	18-25	**Female**	3/6	50	6/16	37.5	9/13	69.2	35
		Male	2/18	11.1	17/36	47.2	6/19	31.6	73
		Unknown	0/1		4/11	36.4	0/3		15
	26-35	**Female**	6/10	60	18/38	47.4	11/24	45.8	72
		Male	2/29	6.9	23/68	33.8	3/40	7.5	137
		Unknown	1/1	100	6/15	40	3/4	75	20
	Adult subtotal		14/65	21.5	74/184	40.2	32/103	31.07	352
	Subtotal		14/108	13	86/356	24.2	38/189	20.1	653
Total			157/1083	14.5	86/356	24.2	44/208	21.2	1647

[1] DEH= Number of individuals with dental enamel hypoplasia; N= Number of individuals.

Table 5.63: *Crude prevalence of individuals with linear enamel hypoplasia by age, sex, and subperiod for the extra-London and London regions.*

Region	**Age**	**Sex**	**Early pre-Black Death**		**Late pre-Black Death**		**N**
	(years)		**DEH/N**[1]	**%**	**DEH/N**	**%**	
Extra-London	**0-5**		0/47		1/270	0.4	317
	6-12		7/33	21.21	6 /98	6.1	131
	13-17		7/21	33.3	3/35	8.6	56
	Non-adults subtotal		14/101	13.9	23/403	5.7	504
	18-25	**Female**	5/22	22.7	7/30	23.3	52
		Male	5/24	20.8	8/28	28.6	52
		Unknown	0/1		0/2		3
	26-35	**Female**	8/55	14.6	18/47	38.3	102
		Male	11/64	17.2	10/52	19.2	116
		Unknown	0/5		0/2		7
	Adults subtotal		29/171	17	45/166	27.1	330
	Subtotal		43/272	15.8	66/564	11.7	836
London	**0-5**		0/3		0/5		8
	6-12		0/3		0 /19		22
	13-17		0/0		0/13		13
	Non-adults subtotal		0/6		0/37		43
	18-25	**Female**	0/0		3/6	50	6
		Male	0/1		2/17	11.76	18
		Unknown	0/0		0/1		1
	26-35	**Female**	1/1	100	5/9	55.56	10
		Male	0/2		2/27	7.41	29
		Unknown	0/0		1/1	100	1
	Subtotal		1/10	10	13/98	13.3	108
	Adults subtotal		1/4	25	13/61	21.3	65
Total			44/282	15.6	79/662	11.9	944

[1] DEH= Number of individuals with dental enamel hypoplasia; N= Number of individuals.

When discriminating between subperiods within the pre-Black Death period, adults showed higher frequencies of dental enamel hypoplasia on individuals dated to the late pre-Black Death subperiod in the Extra-London region. Non–adults showed higher prevalence rates of dental enamel hypoplasia during the early pre-Black Death period. However, these differences were not statistically significant. Due to the small sample size for the London region during the early pre-Black Death comparisons between regions for the early pre-Black Death and between periods in London was not possible.

When looking at differences between males and females, the only statistically difference observed was during the late pre-Black Death subperiod in London where women exhibited higher frequencies of DEH (8/15, 53.33%, Two tailed Fisher exact test, p=0.0008).

5.8.2 True rates

Analysis of deciduous dentition was available only in the primary sample. This allowed estimating true rates for each observed tooth. The following tables show the true rates of dental enamel hypoplasia in deciduous dentition by age for non-adults by period (Table 5.64) and subperiod (Table 5.65).

Table 5.64: *True prevalence of dental enamel hypoplasia al enamel hypoplasia in deciduous teeth from individuals from the primary sample (Wharram Percy, St Oswald's Priory, and St Anne's Charterhouse) by age and period.*

Period	**Age**	**LM2[1]**		**LM1**		**UC**		**Total**	
	(years)	**DEH/N[2]**	**%**	**DEH/N**	**%**	**DEH/N**	**%**	**DEH/N**	**%**
Pre-Black Death	**0-5**	6/43	14	6/54	11.1	5/38	13.2	17/135	12.6
	6-12	0/25		1/21	4.8	1/15	6.7	2/61	3.3
	13-17	0/0		0/0		0/0		0/0	
	Subtotal	6/68	8.87	7/75	9.3	6/53		9/196	9.7
Post-Black Death	**0-5**	0/1		0/1		0/1		0/3	
	6-12	0/1		0/0		0/0		0/1	
	13-17	0/0		0/0		0/0		0/0	
	Subtotal	0/2		0/1		0/1		0/4	
Total		6/70	8.6	7/76	9.21	6/54	11.1	19/202	9.4

[1] LM= Lower molar, UC = Upper canine; [2] DEH = Individuals with evidence of dental enamel hypoplasia, N= number of individuals.

Table 5.65: *True prevalence of dental enamel hypoplasia al enamel hypoplasia in deciduous teeth from individuals from the primary sample (Wharram Percy, St Oswald's Priory, and St Anne's Charterhouse) by age and subperiod.*

Subperiod	**Age**	**LM2**[1]		**LM1**		**UC**		**Total**	
	(years)	**DEH/N**[2]	**%**	**DEH/N**	**%**	**DEH/N**	**%**	**DEH/N**	**%**
Early pre-Black Death	**0-5**	1/7	14.3	2/7	28.6	0/5		3/19	15.8
	6-12	0/4		0/3		0/3		0/10	
	13-17	0/0		0/0		0/0		0/0	
	Subtotal	1/11	9.1	2/10	20	0/8		3/29	10.3
Late pre-Black Death	**0-5**	5/32	15.6	2/37	5.4	5/26	19.2	12/95	12.6
	6-12	0/15		1/12	8.3	1/8	12.5	2/35	5.7
	13-17	0/0		0/0		0/0			
	Subtotal	5/47	10.6	3/49	6.1	6/34	17.6	14/130	10.8
Total		6/79	7.6	5/59	8.5	6/42	14.3	17/159	10.7

[1] LM= Lower molar, UC = Upper canine; [2] DEH = Individuals with evidence of dental enamel hypoplasia, N= number of individuals.

The subsequent section shows a more detailed analysis of dental enamel hypoplasia by tooth for permanent dentition. This was also only possible for the primary sample. Tables (Table 5.66) and (Table 5.67) show the true rates of dental enamel hypoplasia in permanent dentition by age and sex by period and subperiod respectively. As sex was estimated for non-adults from over the age of 10 years in the primary sample, sex categories for the individuals age 13 to 17 years were included as well.

Table 5.66: *True prevalence of dental enamel hypoplasia al enamel hypoplasia in permanent teeth from individuals from the primary sample (Wharram Percy, St Oswald's Priory, and St Anne's Charterhouse) by age, sex, and period.*

Period	**Age**	**Sex**	**LM3[1]**		**LM2**		**LM1**		**UC**		**Total**	
	(years)		**DEH/N[2]**	**%**	**DEH/N**	**%**	**DEH/N**	**%**	**DEH/N**	**%**	**DEH/N**	**%**
Pre-Black Death	**0-5**		0/1		0/2		13/21	61.9	2/5	40	15/29	29
	6-12		0/0		1/9	11.1	7/26	26.9	12/17	70.6	20/52	38.5
	13-17	**Female**	1/1	100	1/2	50	1/1	100	1/1	100	4/5	80
		Male	0/0		0/6		1/6	16.7	2/3	66.7	3/15	20
		Unknown	0/2		2/2	100	1/1	100	1/2	50	4/7	57.1
	Non-adult subtotal		1/4	25	4/21	19	23/55	41.8	18/28	28.6	46/108	39.8
	18-25	**Female**	8/15	53.3	11/20	55	7/20	35	12/68	17.6	38/123	30.9
		Male	1/12	8.3	6/22	27.3	7/22	31.8	13/21	61.9	27/77	49.4
		Unknown	1/2	50	0/1		2/2	100	1/1	100	4/6	66.7
	26-35	**Female**	5/12	41.7	11/20	55	7/19	36.8	12/18	66.7	35/69	50.7
		Male	7/22	31.8	11/27	40.7	5/22	22.7	13/17	76.5	36/88	40.9
		Unknown	0/0		0/0		0/0		0/0		0/0	
	Adult subtotal		22/63	34.9	39/90	43.3	28/85	32.9	51/125	40.8	140/363	38.6
Subtotal			23/67	34.3	43/111	38.7	51/140	36.4	69/153	45.1	186/471	41.6
Post-Black Death	**0-5**		0/0		0/0		0/1		0/0		0/1	
	6-12		0/0		0/1		1/1	100	1/1	100	2/3	66.7
	13-17	**Female**	0/0		0/0		0/0		0/0		0/0	
		Male	0/0		0/1		0/1		0/0		0/2	
		Unknown	0/0		0/0		0/0		0/0		0/0	
	Non-adult subtotal		0/0		0/2		1/3	33.3	1/1	100	2/6	33.3
	18-25	**Female**	1/1	100	0/0		0/0		0/1		1/2	50
		Male	1/3	33.3	1/4	24	0/4		1/4	25	3/15	20
		Unknown	0/0		0/0		0/0		0/0		0/0	
	26-35	**Female**	0/0		0/0		0/2		1/2	50	1/4	25
		Male	0/5		2/7	28.6	0/5		3/7	42.9	5/24	20.8
		Unknown	0/0		0/0		0/0		0/0		0/0	
	Adult subtotal		2/9	22.2	3/11	27.3	0/11		5/14	35.7	10/45	22.2
	Subtotal		2/9	22.2	3/13	23.1	1/14	7.1	6/15	40	12/51	23.5
Total			25/76	32.9	46/124	37.1	52/154	34.2	75/168	44.6	198/522	39.8

[1] LM= Lower molar, UC = Upper canine [2] DEH = Individuals with evidence of dental enamel hypoplasia, N= number of individuals.

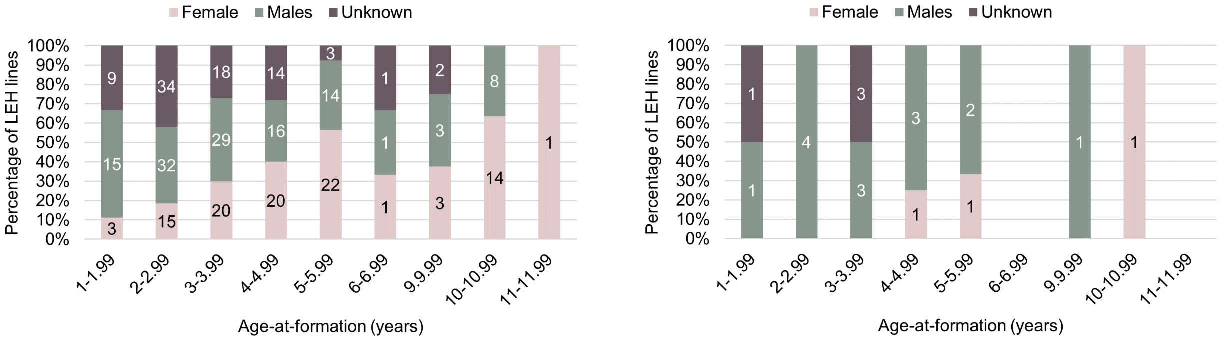
Table 5.67: *True prevalence of dental enamel hypoplasia al enamel hypoplasia in permanent teeth from individuals from the primary sample (Wharram Percy, St Oswald's Priory, and St Anne's Charterhouse) by age, sex, and subperiod.*

Period	**Age**	**Sex**	**LM3[1]**		**LM2**		**LM1**		**UC**		**Total**	
	(years)		**DEH/N[2]**	**%**	**DEH/N**	**%**	**DEH/N**	**%**	**DEH/N**	**%**	**DEH/N**	**%**
Early pre-Black Death	**0-5**		0/1		0/1		1/1	100	0/2		1/5	20
	6-12		0/0		0/1		1/4	25	0/1		1/6	16.7
	13-17	**Female**	1/1	100	1/1	100	1/2	50	1/2	50	4/6	66.7
		Male	0/0		0/2		1/2	50	0/0		1/4	25
		Unknown	0/0		0/		0/0		0/0		0/0	
	Non-adult Subtotal		1/2	50	1/5		4/9	44.4	1/5		7/21	33.3
	18-25	**Female**	2/4	50	1/3	33.3	1/4	25	2/4	50	6/15	40
		Male	0/2		1/3	33.3	1/3	33.3	1/3	33.3	3/11	27.3
		Unknown	1/1	100	0/0		0/0		0/0		1/1	100
	26-35	**Female**	0/1		1/1		1/2	50	1/2	50	3/6	50
		Male	2/7	28.6	3/7	42.9	2/7	28.6	5/6	83.3	12/27	32.4
		Unknown	0/0		0/0		0/0		0/0		0/0	
	Adult subtotal		5/15	33.3	6/14	42.9	5/16	31.3	9/15	60	25/60	41.7
	Subtotal		6/17	35.3	7/19	36.8	9/25	36	10/20	50	32/81	39.5
Late pre-Black Death	**0-5**		0/0		0/1		7/13	53.8	1/1	100	8/15	53.3
	6-12		0/0		1/5	20	3/15	20	6/9	66.7	10/29	34.5
	13-17	**Female**	0/0		0/0		0/0		0/0		0/0	
		Male	0/0		0/3		0/3		2/3	66.7	2/9	22.2
		Unknown	0/2		2/2	100	1/2	50	1/2	50	4/8	50
	Non-adult Subtotal		0/2		3/11		11/33	33.3	10/15	66.7	24/61	39.3
	18-25	**Female**	4/7	57.1	4/10	40	4/9	44.4	5/8	62.5	17/34	50
		Male	0/4		2/8	25.0	2/8	25.0	5/8	62.5	9/28	32.1
		Unknown	0/1		0/1		1/1	100	0/0		1/3	33.3
	26-35	**Female**	1/6	16.7	5/9	55.6	3/9	33.3	6/8	75.0	15/32	46.9
		Male	3/7	42.9	3/11	27.3	2/6	33.3	3/5	60	11/29	37.9
		Unknown	0/0		0/0		0/0		0/0		0/0	
	Adult subtotal		8/25	32.0	14/39	35.9	12/33	36.4	19/29	65.5	53/126	42.1
	Subtotal		8/27	29.6	17/50	34.0	23/66	34.8	29/44	65.9	77/187	41.2
Total			14/44	31.8	24/69	34.8	32/91	35.2	39/64	60.9	109/268	40.7

[1] LM= Lower molar, UC = Upper canine; [2] DEH = Individuals with evidence of dental enamel hypoplasia, N= number of individuals.

5.8.3 Age-at-formation

In this investigation, dental enamel hypoplasia was recorded for mandibular molars and maxillary canines. As age at formation can only be estimated for linear enamel hypoplasia defects, this section only considers that specific type of defect. Age-at-formation was estimated for each LEH defect for the primary data in permanent dentition. A total of 308 defects were recorded, 287 to the pre- (n=287/308, 93.2%) and 21 to the post- (n=21/308, 6.8%) Black Death periods (Figure 5.23).



(a) *(b)*

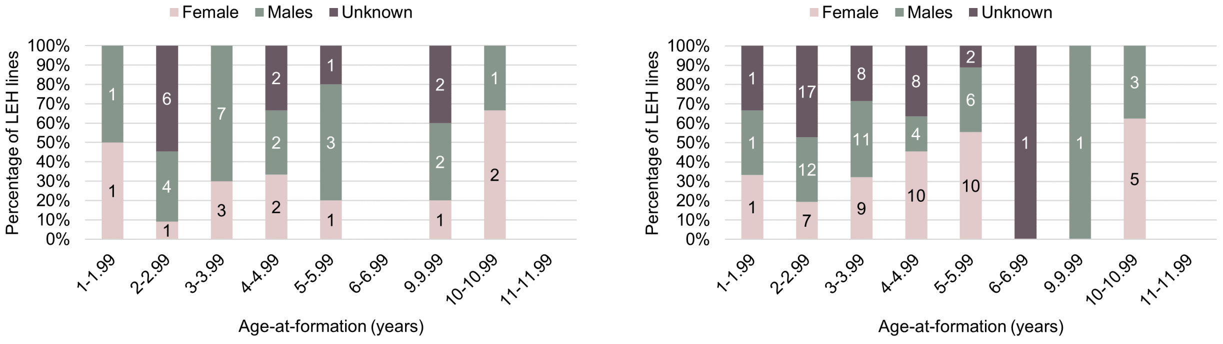
Figure 5.23: *Age-at-formation of LEH in the extra-London region during the pre- (a) and post- (b) Black Death periods*

The most common age-at-formation for the pre-Black Death period was 2-2.99 years (n=81/287, 28.2%) followed by 3-3.99 years (n=67/287, 23.3%). For the post-Black Death period, the most common age was 3-3.99 years (n=6/21, 28.6%) followed by 2-2.99 and 4-4.99 years (n=4/21, 19.1% for both categories). When comparing periods no statistically significant difference was observed.

When looking at differences between males and females in the pre-Black Death period, the most common age-at-formation for females was 5-5.99 (n=22/99, 20.2%) years followed by 3-3.99 and 4-4.99 years (n=20/99, 20.2%). For males the most common estimated age was

2-2.99 years (n=32/107, 29.9%) followed by 3-3.9 years (n=29/107, 27.1%). The post-Black Death period showed a similar pattern in males where the most common age-at-formation was 2-.99 years (n=4/14, 28.6%) followed by 3-3.99 and 4-4.99 years (n=3/14, 21.4%). The female sample was very small with a total sample of 3 recorded lines. Statistical analysis showed no significant differences.

Within the pre-Black Death period, 163 defects were dated to the early (n=42/163, 25.8%) or the late (121/163, 74.2%) pre-Black Death subperiods (Figure 5.24).



(a) *(b)*

Figure 5.24: *Age-at-formation LEH the extra-London region during the early- (a) and late- (b) pre-Black Death subperiods*

When looking within the pre-Black Death subperiod, the most common age-at-formation recorded was 2-2.99 years for both early and late pre-Black Death subperiods (n=11/42, 26.2%; n=36/117, 30.8% respectively). The only statistically significant difference was observed when comparing the number of lines with a defect estimated for 9.99 years where the early pre-Black Death period showed higher frequencies of defects than the late pre-Black Death subperiod (n=5/42, 11.9% and n=1/117, 0.9% respectively; two tailed Fisher's exact test, p=0.005).

Discrimination between males and females showed that during the early pre-Black Death subperiod both females and males had the most common age-at-formation during 3-3.99

years (n=3/11, 27.3% and n=7/100, 35% respectively) followed by 4-4.99 years (n=2/11, 18.2%) for females and 2-2.99 years (n=20/100, 20%). For individuals dated to the late pre-Black Death subperiod, the most common age-at-formation of LEH for females was 4-4.99 years and 5-5.99 years (n=10/42, 23.8%), followed by 3-3.99 years (n=9/42, 21.4%), while for males the most common age was 2-2.99 years (n=12/38, 31.6%) followed by 3.99 years (n=11/38, 29%).

5.9 Oral health

Similarly to DEH, due to the diverse nature of the secondary evidence, analysis of dental caries was focused on crude prevalence of individuals with evidence of caries. For this analysis, the total sample of individuals was reduced from 2065 to 1958. This decrease in the sample size was due to perinates (who didn't have erupted dentition) and unaged non-adults (whose age was not estimated due to lack of dentition) not being included in the analysis.

The following tables show the distribution of individuals with dental caries by age, sex, and period for the extra-London (Table 5.68) and London regions (Table 5.69). The overall sample exhibited 20.3% (n=397/1958) of individuals with evidence of dental caries.

Within each region, the extra-London region showed higher frequencies of dental caries during the post-Black Death period. However, this difference was not statistically significant. Furthermore, the sample size, particularly for non-adults, was very small. Within the London region, statistically significant differences were observed when comparing adults dated to the pre-, post- and Black Death periods. The post-Black Death period presented the highest frequencies of individuals with dental caries (pre-Black Death: n=8/65, 12.3%), followed by the Black Death (n=72/184, 39.1%), and the pre-Black Death period (n=8/65, 12.3%) (pre-Black Death-Black Death: X^2=15.85, df=1, p<0.001; Black Death-post-Black Death: X^2=7.01, df=1, p=0.008; pre-Black Death-post-Black Death: X^2=31.1, df=1, p<0.001).

When comparing regions, the only statistically significant differences were observed for individuals dated to the pre-Black Death period where for non-adults showed higher frequencies in the London region (London: n=9/43, 20.9%, extra-London: 75/839, 8.9%, X^2=6.8, df=1, p=0.009), while adults showed statistically higher frequencies in the extra-London region (extra-London: n=141/447, 31.5%; London: n=8/65, 12.3%, X^2=10.18, df=1, p=0.001).

Within adults, when discriminating between sexes, there were statistically significant differences between males and females in almost all periods/regions. Within the extra-London region, females showed higher frequencies of individuals with dental caries (females n=77/210, 36.7% males n=61/222, 27.5%, X^2=4.19, df=1, p=0.04). The post-Black Death period was too small to analyse. Within the London region, females showed higher frequencies of individuals with dental caries during the pre-Black Death period (females n=5/16, 31.3%, males n=3/47, 6.4%, Two tailed Fisher's exact test, p=0.02), while males exhibited higher frequencies during the post-Black Death (males n=38/59, 64.4%, females n=16/37, 43.2%, X^2=4.14, df=1, p=0.02).

Table 5.68: *Crude prevalence of individuals with dental caries by age, sex, and period for the extra-London and London regions.*

Age	**Sex**	**Pre-Black Death**		**Black Death**		**Post-Black Death**		**Total**
(years)		**Caries/N**[1]	**%**	**Caries/N**	**%**	**Caries/N**	**%**	**N**
0-5		29/561	5.2	0/0		0/1		562
6-12		30/182	16.5	0/0		1/2	50	184
13-17		16/96	16.7	0/0		0/1		97
Non-adult subtotal		75/839	8.9	0/0		1/4	25	843
18-25	**Female**	19/70	27.1	0/0		0/1		71
	Male	10/73	13.7	0/0		1/4	25	77
	Unknown	1/5	20	0/0		0/0		5
26-35	**Female**	58/140	41.4	0/0		2/2	100	142
	Male	51/149	34.2	0/0		6/8	75	157
	Unknown	2/10	20	0/0		0/0		10
Adult subtotal		141/447	31.5	0/0		9/15	60	462
Subtotal		216/1286	16.8	0/0		10/19	52.6	1305

[1] Caries= Number of individuals with dental caries; N= Number of individuals.

Table 5.69: *Crude prevalence of individuals with dental caries by age, sex, and period for the London region.*

Age	**Sex**	**Pre-Black Death**		**Black Death**		**Post-Black Death**		**Total**
(years)		**Caries/N**[1]	**%**	**Caries/N**	**%**	**Caries/N**	**%**	**N**
0-5		0/8		4/68	5.9	0/39		115
6-12		4/22	18.2	4/73	5.5	2/23	8.7	118
13-17		5/13	38.5	9/31	29	6/24	25	68
Non-adult subtotal		9/43	20.9	17/172	9.9	8/86	9.3	301
18-25	**Female**	2/6	33.3	4/16	25	8/13	61.5	35
	Male	0/18		16/36	44.4	10/19	52.6	73
	Unknown	0/1		3/11	27.2	0/3		15
26-35	**Female**	3/10	30	18/38	47.4	8/24	33.3	72
	Male	3/29	10.3	28/68	41.2	28/40	70	137
	Unknown	0/1		3/15	20	3/4	75	20
Adult subtotal		8/65	12.31	72/184	39.1	57/103	55.3	352
Subtotal		17 / 108	15.7	89 / 356	25	65 / 189	34.4	653

[1] Caries= Number of individuals with dental caries; N= Number of individuals.

The following tables show the distribution of individuals with dental caries by age, sex, and subperiod for the extra-London (Table 5.70) and London regions (Table 5.71). When discriminating subperiods within the pre-Black Death period overall, 1109 were dated to either the early or the late pre-Black Death periods. From the overall sample 195 individuals (17.6%) presented evidence of dental caries.

Due to the small sample of the early pre-Black Death period, comparisons between subperiods was only possible for the extra-London region. The only statistically significant difference was observed in adults, where the late pre-Black Death period showed higher frequencies (late pre-Black Death: n=62/161, 38.5%, early pre-Black Death: n=46/171, 26.9%; X^2=5.09, df=1, p=0.02).

When comparing regions, statistically significant differences were observed in individuals dated to the late pre-Black Death subperiod. Adults showed higher frequencies of dental caries in the extra-London region (extra-London n=62/161, 38.5%, London n=7/61, 11.5%,

X^2=15.09, df=1, p<0.001). This was concentrated in differences between males (extra-London n=26/80, 32.5%, London n=3/44, 6.8%, two tailed Fisher's exact test, p=0.002). Different to this, during the same subperiod, the non-adult sample showed higher frequencies of dental caries in the London region (London n=9/37, 24.3%; extra-London region n=42/403, 10.4%, X^2=6.39, df=1, p=0.01). When comparing males and females, women showed higher frequencies in all subperiods/regions. However, no statistically significant differences were observed.

Table 5.70: *Crude prevalence of individuals with dental caries by age, sex, and subperiod for the extra-London region.*

Age	**Sex**	**Early pre-Black Death**		**Late pre-Black Death**		**Total**
(years)		**Caries/N**[1]	**%**	**Caries/N**	**%**	**N**
0-5		12/174	6.9	14/270	5.2	444
6-12		7/52	13.5	22/98	22.5	150
13-17		9/40	22.5	6/35	17.1	75
Non-adult subtotal		28/266	10.5	42/403	10.4	669
18-25	**Female**	4/22	18.2	12/30	40	52
	Male	3/24	12.5	5/28	17.9	52
	Unknown	0/1		0/2		3
26-35	**Female**	20/55	36.4	24/47	51.1	102
	Male	18/64	28.1	21/52	40.4	116
	Unknown	1/5	20	0/2		7
Adult subtotal		46/171	26.9	62/161	38.5	332
Subtotal		74/437	16.9	104/564	18.4	1001

[1] Caries= Number of individuals with dental caries; N= Number of individuals.

Table 5.71: *Crude prevalence of individuals with dental caries by age, sex, and subperiod for the London region.*

Age	**Sex**	**Early pre-Black Death**		**Late pre-Black Death**		**Total**
(years)		**Caries/N**[1]	**%**	**Caries/N**	**%**	**N**
0-5		0/3		0/5		8
6-12		0/3		4/19	21.1	22
13-17		0/0		5/13	38.5	13
Adult subtotal		0/6		9/37	24.3	43
18-25	**Female**	0/0		2/6	33.3	6
	Male	0/1		0/17		18
	Unknown	0/0		0/1		1
26-35	**Female**	1/1	100	2/9	22.2	10
	Male	0/2		3/27	11.1	29
	Unknown	0/0		0/1		1
Adult subtotal		1/4	25	7/61	11.48	65
Subtotal		1/10	10	16/98	16.3	108

[1] Caries= Number of individuals with dental caries; N= Number of individuals.

6 | Discussion

The objective of this investigation was to evaluate differences in childhood health between the pre- (AD 900-1348), post- (AD 1350-1550), and Black Death (AD 1348-1350) periods, discriminating where possible between the early (AD 900-1100) and late (AD 1110-1348) pre-Black Death subperiods. Previous research has suggested a decline in the health status of the London population in the time leading up to the Black Death (DeWitte, 2014, 2017). This may go some way to explaining why the pandemic had such a devastating effect on medieval society. Such holistic studies for the areas outside London are lacking, with research focusing instead on specific conditions such as trauma or mortality rates of a select age group (Lewis, 2016*b*), or specific regions such as Cambridge (Giberti, 2019). This investigation proposed integrating data from multiple sites outside London to provide a more comprehensive understanding of health before the Black Death in England. The sample was divided into two regions: London and extra-London as most of the available investigations focus on the first region which may not be representative of England.

Multiple indicators of health were evaluated in skeletons of males and females of different estimated ages to analyse stress experienced during formative years. To reduce the mortality bias caused by analysing only non-survivors, skeletal remains from both non-surviving non-adults and surviving adults were analysed. To properly integrate all results, the discussion was structured by separating all analysed indicators according to the period of stress they represent (Table 6.1).

Table 6.1: *List of the indicators of stress analysed in this investigation.*

Analysed period of stress	Estimated age of the skeletal sample	Indicators of stress
Perinate and obstetric stress	Perinates	Diaphyseal length
	Children	DEH[1] in deciduous dentition
	>15 years of age	Spina bifida occulta
Childhood stress	Children	Diaphyseal length Infant and child mortality[2] Rickets, scurvy, and tuberculosis Dental caries DEH in permanent dentition Vertebral neural canal dimensions Cribra orbitalia
	Adults	DEH in permanent dentition Cribra orbitalia
Adolescent stress	Adolescents (10-25 years)	Rickets, scurvy, and tuberculosis Vertebral neural canal Long bone length PHV[3] timing Menarche timing

[1] DEH = Dental enamel hypoplasia; [2] birth-5 years of age; [3] PHV= Peak height velocity.

6.1 Perinatal and maternal stress

The first moment of life when an individual can experience stress is while still *in utero*. Stress experienced during pregnancy can have long-lasting consequences throughout the rest of the life of a person (Cetin et al., 2019). For this reason, similar to childhood health analysis, the evaluation of perinates provides valuable information to assess population health. Perinatal health was assessed through the evaluation of conditions which affect development *in utero*. These were observed in both non-surviving perinates as well as surviving children and adults. Perinates were defined as individuals with estimated ages from 24 to 40 weeks *in utero* (WIU) (Lewis, 2007). This included unborn, stillbirths, and premature births. Intrauterine growth restriction in perinates was assessed through the measurement and comparison of the diaphyseal length of the femur and humerus with dental age (Hodson, 2018; Kiserud et al., 2017). Perinatal stress was evaluated in children through the identification of enamel hypoplasia on the deciduous dentition, which can also reflect stress experienced between the second trimester and the first year of life (AlQahtani et al., 2010). Finally, individuals over the age of 15 were evaluated for the presence of spina bifida occulta. This refers to a congenital neural canal defect that may reflect folic acid deficiency during pregnancy (López-Camelo et al., 2005). By utilising variables from people of different ages, it was possible to expand the sample, observing both non-survivors and survivors of different ages and reducing mortality bias. The analysis of perinatal stress provides indirect evidence for maternal health (Gowland, 2015).

There were just 40 perinates in the study sample (1.9% of the total sample), 34 were found in the extra-London region, while 6 were found in London. In the extra-London region, all perinates were dated to the pre-Black Death period, while in London 5 perinates were dated to the Black Death period and one to the post-Black Death period. This distribution is likely to be the result of sample bias and the constraints of the time periods used. In many sites, particularly the ones located in the extra-London region like Wharram Percy, Barton-Upon-Humber, and St Oswald's Priory, most of the perinates were dated to broader periods (e.g. 'medieval') (Mays et al., 2007) which included both the pre- and post-Black Death periods and therefore unsuitable for this investigation. For example, the entire Wharram Percy collection had 36 individuals aged under 40 WIU dated to the medieval period. However, only three had dating which fit the criteria needed for this investigation (the three individuals were dated to the pre-Black Death period). This resulted in a smaller sample for analysis. Other variables that may have played a role are the cultural practices concerning the burial of perinates. Following Roman law, Christian rules forbade the burial of pregnant women and mandated the removal of the unborn individual and subsequent burial of all unbaptised babies (including unborn and stillborn) separately (Bednarski and Courtemanche, 2011). Another possibility is that some of these babies were the product of premature births and were born alive. Due to the religious significance of birth and the importance of baptism, often midwives were allowed to baptise dying infants (Kline, 2003). Additionally, analyses in sites like Raunds Furnells, St. Helen-on-the-Walls, and Wharram Percy show higher frequencies of perinates indicating that the exclusion of stillborn and unbaptised infants was probably not as strictly followed in rural areas as inside London (Lewis and Gowland, 2007). Records show that these rules were preached in more centric urban areas. This means that the lower frequencies of perinates in London may be due to differential burial practices. For this thesis, it was not possible to evaluate whether the perinate had been born alive or not. Further analysis exploring new techniques such as a histological analysis of bone microstructure proposed by Booth (2020) to identify stillborns would be required.

When looking at the London sample, the highest frequencies of perinates are observed during the Black Death period. This is expected as it represents the moment of most significant crisis where women would have struggled to access resources and medical care, resulting in higher maternal and perinatal stress and more pregnancy and birth complications (Benedictow, 2004). Furthermore, pregnancy is characterised by immunosuppression caused by progesterone (Aghaeepour et al., 2017; Kareva, 2020), which would make women more vulnerable to disease. It is also possible that during a period of crisis such as a pandemic, burial practices became less strict due to the high number of victims of the plague being buried in London. However, the sample is too small to make a conclusive analysis.

Due to the small number of perinates found in London sites and the lack of skeletal data from the secondary sources of data from the extra-London sites, only the information from the primary analysis of individuals dated to the pre-Black Death period from the extra-London region (Wharram Percy and St Oswald's Priory) was available for analysis. When looking at the perinates that could be aged based on dentition, 66.7% (n = 8/12) died close to the moment of birth (full-term). All of them died during the third trimester (27 weeks until birth). This is expected from archaeological remains as it is very rare to find younger individuals (Hodson, 2018). During the first trimester of pregnancy, complications can be due to chromosomal abnormalities that make the foetus non-viable (Jurkovic et al., 2013). This differs from the ones most commonly observed during the third trimester that are proportionally more related to the health of the mother and the environment in which the pregnancy develops (Ngoc et al., 2006). These complications can also increase the risk of maternal mortality (Filippi et al., 2016).

The majority of the perinates provided lower estimated ages for the femur than for their dental development, a pattern that became starker when looking at humeral diaphyseal lengths. Dental eruption is considered as a more reliable indicator of age as it is not as affected by stress as the development of long bones (AlQahtani et al., 2010; Hillson, 2005). Observing older ages for dental development reflects that the perinate may have experienced stress during development (Hodson, 2018). This was observed in previous analyses of perinate remains like Hodson (2017) where differences between dental age and femoral and humeral growth were observed. The author finds not only differences between dental age and limb length but also between femora and humeri which indicates that growth disruption did not affect all libs equally. Clinical studies like de Carvalho et al. (2013) have used Doppler analysis to evaluate foetal growth in pregnant women from Brazil. Their investigation related short femora and particularly short humeri length with foetal growth restriction. This indicates the presence of stress in perinatal individuals and possibly their mothers. When comparing early and late pre-Black Death subperiods, the samples were too small for statistical analysis. However, higher frequencies of individuals with possible evidence of stress were observed in the late pre-Black Death period indicating a possible decline in maternal health. Shorter limbs in perinates can signify multiple issues. For example, congenital conditions like Down's syndrome and Achondroplasia can result in shorter limbs (de Carvalho et al., 2013). Additionally, differences in population with the clinical growth charts used to assess age may result in variability within the expected growth. It is important to expand the sample and analysis to further study the possibility of perinatal stress during the pre-Black Death period.

The study of skeletal remains from perinates reflects the weakest individuals who perished from stress. However, it is possible for an individual to experience stress while *in utero* and survive birth. Dental enamel hypoplasia in deciduous dentition provides information about this stress observed in children. The analysis of this indicator showed a similar pattern to the one found in perinates. Due to limitations in the data, only the primary data could be analysed. Results showed a true rate of 12.5% (17/135) for lower molars and upper canine in individuals dated to the pre-Black Death. This indicates stress experienced during the second and third trimester when teeth were forming. This was particularly observed in the 1 to 5 years category. When comparing early and late pre-Black Death subperiods, no clear pattern was observed indicating that stress was likely similar in both subperiods.

The last indicator showing perinatal stress is spina bifida occulta. This indicates specific nutritional deficiencies during pregnancy that did not lead to premature death and can be observed in individuals over the age of 15 years (Lewis, 2018). There are many reasons why spina bifida occulta may occur. Family history of neural tube defects and specific genetic markers can increase the risk of developing the condition (Au et al., 2008). Concerning obstetric care, folic acid deficiency during pregnancy increases the likeness of spina bifida (López-Camelo et al., 2005). Although the general diet of the mother can have an impact on the health of the baby, studies have identified macro-nutrients like vitamin B12 and folic acid as crucial in its development (Elmadfa and Meyer, 2012). Spina bifida occulta was observed in 2.9% (16/543) of individuals dated to the pre-Black Death period in the extra-London region. When comparing between early and late pre-Black Death subperiods, the late pre-Black Death showed a significantly higher prevalence.

This may indicate higher levels of folic acid deficiency in mothers during this period. Males showed higher frequencies of spina bifida occulta than females for the early and late pre-Black Death subperiods. Although this difference was not statistically significant, it agrees with previous investigations which found higher frequencies of the condition in medieval and post-medieval European males (Groza et al., 2016) and in modern populations (Eubanks and Cheruvu, 2009; Fidas et al., 1987). Limited data prevented similar observations from being made in the London samples or other periods. It is important to remember that spina bifida occulta is the least severe form of the condition (see section 3.1.6.2), which allows the individual to survive until older ages. This also means that it reflects milder cases of stress.

The more severe cases of spina bifida aperta were not identifiable in the bioarchaeological record but would be present among perinatal deaths.

Evidence of perinatal stress does not only reflect the condition of the unborn but can also be used as a proxy to explore the health of the pregnant mother (Gowland, 2015). Gowland (2015) proposes that stress experienced by children under the age of 3 years (1000 days) can be used as a proxy for maternal health. This would not only assess the health of the mother but also the health of the person breastfeeding them. In the majority of the cases in medieval England, this was likely the same person as wet-nursing was a practice reserved for the wealthy (Phillips, 2022). However, the distinction must be made as it refers to two separate instances of the life of the progenitor and the baby. Infants and children were among the most vulnerable and likely to die during the medieval period (Robb et al., 2021). There are two most largely traumatic moments in the life of an infant. The first one is birth, when the baby is vulnerable to trauma and hypoxia (Ngoc et al., 2006) and during the weaning process (Lewis, 2018), particularly at the end when the child stops receiving nourishment and immunity from breast milk (Tsutaya et al., 2015).

Concerning maternal mortality, its recognition in the archaeological record is complicated. The identification of pregnancy in the osteological record is unreliable. As mentioned before, Christian law ordered pregnant women to be buried without their foetus (Bednarski and Courtemanche, 2011), making their identification much more difficult. However, this practice was not always followed (Lewis and Gowland, 2007). Archaeothanatology has identified cases of coffin births (when an unborn baby is expelled from the womb of a woman after her death) in medieval Ireland (Le Roy and Murphy, 2020), showing that pregnant women were in some instances buried with their foetuses. Maternal mortality also may extend to people who had recently given birth and died from pregnancy-related complications such as infection (Høj et al., 2003). The World Health Organization defined maternal mortality as deaths during pregnancy or within 42 days after birth (World Health Organization, 2023). Høj et al. (2003) proposes that in impoverished areas where there is a high risk of maternal mortality, this window of 42 days should be extended to 3 months after birth (Høj et al., 2003). This element would make identifying maternal deaths in the bioarchaeological record even more complicated.

As pregnancy was considered part of the realm of women, there are few records of their experiences, mostly based on the perspective of a male relative (Lee, 2002). Although historical records show medical instructions concerning birthing practices (Soranus of Ephesus, 1991). There was also a prominent role of religion and magical thinking related to pregnancy and birth. This is observed in the prayers written in birthing scrolls (Fiddyment et al., 2021). Historical records indicate that miscarriages and premature labour could be related to assault or domestic violence against the mother (Butler, 2011). On rare occasions, it may also be related to the agency of the mother (Evans and Read, 2015) resulting in an intentional abortion. Although these instances reflect stress over the psycho-social health of the mother, they exceed the capabilities and scope of this investigation.

Concerning the health of the mother, it is difficult to estimate maternal mortality during the medieval period. Although some authors highlight that maternal mortality was the main cause of death for young women, authors like Podd (2020) argue that the rates of maternal mortality during the medieval period were smaller than estimated. Podd (2020) identifies the highest risk for maternal mortality in the lower age of first pregnancy (under 15 years of age) and multiple births in older ages. However, this analysis is based on records of pregnancy and births in individuals from the aristocracy and gentry, who had better access to food and medical treatments than lower classes and peasants. This would have resulted in different risk factors than lower economic classes. The individuals from lower classes who compose the analysed samples would have struggled more to access the resources necessary for the proper nutrition and care of pregnant women, particularly concerning the access to meat during the late pre-Black Death period (Müller, 2010), which is rich in protein, iron, folic acid and vitamin B12 all important for the development of a healthy pregnancy. This was observed in Wharram Percy. (Fuller et al., 2003) analysed samples from deciduous dentition of non adults and permanent canine, third molar, and ribs of adult individuals from Wharram Percy. Results from this study showed that some individuals in Wharram

Percy had access to animal protein later in life (Fuller et al., 2003). However, this does not discard the presence of stress. For example the individual EE36 (female of 17-25 years from Wharram Percy (see tables E.1 to E.3)) had likely access to animal protein later during here life (Fuller et al., 2003). However, she also presented evidence of cribra orbitalia indicating that she likely experienced stress during here childhood. This indicates that although access to some resources was attainable, this did not result in adequate intake during the life of the individual.

Furthermore, concerns such as low birth age were unlikely as outside of aristocracy, contemporary concerns and medical textbooks highlight the importance of women waiting until older ages to become pregnant (Soranus of Ephesus, 1991; Podd, 2020). Additionally, economic struggles led most of the population to marry at later ages, postponing pregnancy until after the age of 26 years (Langdon, 2010).

The concern for the health of birthing mothers can be observed in cultural practices surrounding pregnancy and birth. Although the evidence is limited due to the destruction of objects related to birthing rituals during the Dissolution of the Monasteries in AD 1536 (Rawcliffe, 2003), there is evidence that birthing girdles may have been worn before and during pregnancy (Fiddyment et al., 2021). Biomolecular analysis of the stains on a parchment birthing girdle MS632 from the Wellcome Collection (Figure 6.1) found evidence of human peptides, which reflected that the parchment had been used during birth as well as non-human peptides from materials like honey, eggs, milk, and plants which were related to treatments giving during birth (Fiddyment et al., 2021). When analysing maternal stress it is important to differentiate between evaluating stress experienced during the pregnancy and the weaning period. Evidence in this investigation has been presented for both instances. However, they do not provide the same type of information. The relationship between the mother and the foetus is through the physical connection of the placenta (Rutherford, 2019). In this investigation maternal health was observable through perinatal growth, DEH in deciduous dentition and evidence of spina bifida occulta. Results indicated that during the pre-Black Death period, there was a sector of the population which had nutritional deficiencies, particularly folic acid resulting in both maternal and perinatal stress. This stress was likely more prominent towards the late pre-Black Death period when social changes and increased growth in urban centres and marketplaces led to changes in social relations between lords and peasants where the latter were more vulnerable to low wages and less access to resources (Astill, 2010; Dyer, 1994; Hatcher, 1977).

Figure 6.1: *Images of the Wellcome Collection Western MS.632 birthing scroll with prayers written for Saint Quiricus and Julitta (-304). Image taken from Wellcome Collection (Wellcome Collection, 2022)*

After birth, although studies are showing how the health of the mother affects the nutrition of the child during the breastfeeding period (Ahmed et al., 2004; Ross and Harvey, 2003), this relationship is not as direct. During the breastfeeding and weaning period, the child is subjected to external factors which also may interfere with their development (Lewis, 2018) and may not reflect the health of their parent. When looking at infant and child mortality, the extra-London region showed higher frequencies than the London region. During the medieval period, a broad estimate of 30% of child mortality was observed (Augenti and Gilchrist, 2011). In this investigation, the extra-London region showed higher frequencies of child mortality (0-4.99 years) than in London (462/1132, 34.9% and 8/114, 5.6% respectively) during the pre-Black Death period. The rates of child mortality in the extra-London region are slightly higher than expected which may reflect a moment of increased stress. This was more apparent when looking at the differences between early and late pre-Black Death subperiods, where the latter showed statistically higher frequencies of children ages 0-4.99 years of age (144/439, 32.8% and 232/597,38.9%, X^2, df=1, p=0.04). When looking at the age distribution of children under 5 years of age peaked at 1 year (infant mortality). This is expected as it is the period of most vulnerability of the child (Lewis, 2018). The deaths of newborns can be related to complications during birth, fatal congenital conditions which would require immediate medical assistance and disease, illness and, trauma (Lehtonen et al., 2017).

Analysis of the age-at-formation of LEH can provide information on stress specifically related to the weaning period (Gowland, 2015; Uauy et al., 2011). In this investigation, this analysis was only available for the primary analysis from the extra-London region. Results showed higher frequencies of defects for the ages of 2 to 4 years of age for both the pre- and post-Black Death periods. The absence of differences between periods may be the result of similar patterns of stress, perhaps related to the ages of weaning. This would agree with other indicators which show a similar absence of differences. However, it is also important to consider the difficulties of the methodology. Some investigations challenge the accuracy of the macroscopic identification of LEH on the tooth (Hillson, 2014; Martin et al., 2008). In this investigation, to remove bias, a corrected chart created by Reid and Dean (2006) and Holt et al. (2012) and modified by Primeau et al. (2015) was used. The chart is considered more accurate as it takes into account hidden cuspal enamel (Martin et al., 2008; Primeau et al., 2015). Furthermore, the colour modifications introduced by Primeau et al. (2015) were evaluated and showed smaller intra- and inter-observer errors. However, it does not provide the same accuracy as the study of a ground section of the tooth (Seow et al., 2005) or stable isotope analysis (Beaumont et al., 2015).

The estimated age of weaning agrees with previous studies of stable isotopes such as Richards et al. (2002) who calculate it in Wharram Percy between 2 and 4 years but presented older ages than sites like Fishergate House, York (Burt, 2013). When looking at differences between males and females, comparisons were only possible for the pre-Black Death period. Although not statistically significant, males showed younger ages of stress than females, particularly during the late pre-Black Death period. Previous studies about differences by sex in LEH signal that in the first years of life, young males are more susceptible to environmental stress than females (Guatelli-Steinberg and Lukacs, 1999; Infante and Gillespie, 1974). This is because at birth, boys in general have more muscle and less subcutaneous fat than girls, which could develop in greater nutritional requirements and less caloric reserves (Infante and Gillespie, 1974, p. 1057). This could explain the pattern observed in the primary sample.

In summary, in the extra-London period, individuals dated to the late pre-Black Death showed more evidence of perinatal and maternal stress. These differences were not statistically significant when compared to the early pre-Black Death period. However, they show a clear trend indicating an increase in metabolic nutritional stress towards the late pre-Black Death subperiod.

6.2 Childhood health

To analyse childhood health, multiple indicators were observed. This included cribra orbitalia, dental enamel hypoplasia in permanent dentition, vitamin deficiencies such as scurvy, rickets, and tuberculosis which is an infectious disease related to immunosuppression, VNC dimensions, and growth disruptions. These indicators were observed both in non-surviving non-adults and surviving adults. The study of adult remains allowed reducing the mortality bias, expanding the sample size and allowing discrimination between males and females.

Overall results showed little difference between periods in the extra-London region. There were trends identifying higher levels of stress during the late pre-Black Death period. However, few differences were statistically significant. The extra-London region showed a crude rate of cribra orbitalia of 16.2%. The overall rates were smaller than other similar investigations (Yaussy et al., 2016). However, this is expected when estimating crude instead of true rates. In this investigation, crude rates for the extra-London region during the pre-Black Death period was 16.3%. Yaussy et al. (2016) found a true rate of 35% for a sample of 569 adult skeletons from Spital Square, London during the medieval period.

No statistically significant differences were observed when comparing periods or subperiods. The absence of differences in cribra orbitalia between periods could indicate few changes in health status over time during the medieval period. This is consistent with previous studies like the "After the Plague" project (2016-2020) from Cambridge University, which found little differences concerning stress between the pre- and post-Black Death periods (Dittmar et al., 2021a). Although similar rates of cribra orbitalia could suggest that there were no changes in health status between periods, recent investigations have questioned the aetiology of the condition (O'Donnell et al., 2020), as well as the use of the condition as an indicator of pathology or nutritional stress (Zdilla et al., 2022). It is necessary to integrate these results with other indicators to create a broader image of health before the plague.

Similarly to the extra-London region, there were no significant differences in crude prevalence rates of cribra orbitalia between pre-, post-, and Black Death periods (pre-Black Death period 5.3%, Black Death, 7.8% and post-Black Death 8.1%). However, overall rates during the pre-Black Death were significantly lower than in the extra-London region. This could signal that populations outside London experienced more stress before the Black Death. However, the likely explanation is that this difference is due to under-recording the condition for the London region. In this investigation, only severity 3 to 5 (Stuart-Macadam, 1991) was considered. This was because of the difficulties in identifying severity stages 1 (capillary impressions) and 2 (scattered fine foramina). However, when stages 1 and 2 are included, the London region exhibited higher crude prevalence rates of cribra orbitalia than the extra-London region.

Using more recent scoring schemes, such as that by Rivera and Mirazón Lahr (2017) will eliminate these problems in the future, as they begin with Stuart Macadam's Grade 3, but problems with interpreting cribra scores in this static secondary data will persist. This was observed in this thesis, where it was not possible to use it due to the scoring system not being available for all secondary data.

Previous analyses have found that females are more vulnerable to developing cribra orbitalia than men due to higher dietary requirements during fertile years, especially concerning pregnancy and breastfeeding (Walker et al., 2009). In this investigation, this was observed in the extra-London region, where women exhibited higher frequencies of cribra orbitalia during the pre-Black Death period, particularly in individuals ages 18 to 25 years. A similar pattern was observed in London during the post-Black Death period, where women (particularly the ones aged 18 to 25 years) showed higher frequencies of cribra orbitalia. None of the differences were statistically significant, but they show a tendency for young females to experience more stress than men. Similarly to cribra orbitalia, dental enamel hypoplasia shows a period of stress which affects enamel growth. The aetiology of this condition is broad having been related to nutritional stress, infection, and fevers during the period when the tooth was forming (Hillson, 2005). Due to the broad nature of the aetiologies associated with these conditions, they may group very different situations which result in stress under the same category. In some cases, their relationship with stress is contested entirely (Hillson, 2005; Zdilla et al., 2022). For this reason, their analysis individually may not provide much evidence of the nuances of stress experienced by a group. However, similarly to cribra orbitalia, integrated as a part of a multi-method approach, it can aid in identifying trends.

When looking at dental enamel hypoplasia, crude prevalences allowed analysis of individuals with evidence of the defects in primary and secondary data. For the primary data, true rates were estimated. This allowed observing the rates by tooth. Due to the small size of the post-Black Death period sample, comparisons were not possible. When comparing early to late pre-Black Death subperiods, only the adults showed higher frequencies of stress for individuals dated to the latter subperiod (early pre-Black Death= 29/171, 17%, late pre-

Black Death= 45/166, 27.1%; X^2, df=1, p=0.02), while non-adults showed higher frequencies during the early pre-Black Death subperiod (14/101, %13.9, 23/403, %5.7). Observing these results in combination with child mortality results, the higher frequencies of DEH in non-adults may represent instances of stress experienced by surviving children, while children who experienced more stress died younger. However, this could also be the result of differences in recording criteria. To discern if the latter played a role in these differences, true rates for the primary data were estimated. These results showed higher frequencies of overall teeth with evidence of DEH for non-adults dated to the late pre-Black Death subperiod (7/21, 33.3%, 24/61 %39.3 for the early and late pre-Black Death subperiods respectively). This agrees with previous results and shows a trend of stress increasing in the period leading to the Black Death.

When discriminating between males and females, there was a trend to show higher frequencies of DEH in women. This was observed in all ages except ages 18 to 25 years for crude prevalences. When observing true prevalences, all teeth showed higher frequencies of stress for females, except the canine tooth. This is consistent with crude rates which were estimated based on the upper canine tooth. When looking at subperiods, the pattern was similar. Crude prevalences did not show any clear pattern, but when looking at the true rates, females showed higher frequencies for overall teeth for all age categories. This could indicate that, similar to cribra orbitalia, women were experiencing more stress than men.

Stable isotope analyses do not show evidence of differential diet composition between men and women during the medieval period (Burt, 2013; Richards et al., 2002). However, this does not mean that both young girls and boys were treated the same. Medical advice describes young girls as less than men. Little girls were associated not only with biological characteristics but also gendered roles like being more nurturing and wearing female clothing (Kline, 2003). These differences extended for the rest of the life of a girl through more informal education, less pay and more precarious working conditions (Bennett, 2010; Kline, 2003). This does not mean that women were always abandoned. Records of wills during the plague show that many men found forms to ensure their daughters would also inherit (Benedictow, 2004).

However, these beliefs could have been reflected in the early dietary practices of children where, as young boys were considered to need to be prepared for harder work, they may have been preferred in food access, particularly when it was limited.

In London, the crude prevalence of dental enamel hypoplasia showed statistically significant higher rates during the Black Death period compared to the pre-Black Death. However, there were no differences when comparing the pre- and post-Black Death periods. This points to a decrease in health from the pre- to the Black Death periods. As dental enamel hypoplasia reflects stress experienced during formative years (Hillson, 2005), dental enamel hypoplasia identified in victims of the Black Death showed stress experienced during the crisis period prior to the Black Death. The differences with the pre-Black Death period may indicate the worsening of the crisis period in London.

After the Black Death, crude prevalence rates of dental enamel hypoplasia significantly decreased for males (from 38.5% to 15.2%). Females did not show statistically significant differences. This agreed with previous work like DeWitte (2017) which also exhibited a decrease in dental enamel hypoplasia (from 67% during the pre-Black Death period to 59.6% during the post-Black Death period). This may be partially due to an overlap of information. DeWitte (2017) uses information from the sites: Guildhall Yard, St. Nicholas Shambles, St. Mary Graces, and Spital Square. This investigation is based on part of the Spital Square sample as well as Guildhall Yard and St Mary Graces. It also has information from Merton Priory and the East Smithfield Black Death cemetery. This decrease was related to better access to food resources like meat (Bailey, 2021) and fish (Orton et al., 2014). When comparing males to females, females showed statistically higher rates of enamel defect compared to men during the post-Black Death (females = 54.05% and males = 15.25%) and pre-Black Death period (females = 56.3% and males = 8.51%). These results were different from previous investigations like Yaussy et al. (2016) which found few differences between males and females when analysing the site Spital Square. Lewis et al. (2016) highlighted that women in urban areas like London were more vulnerable to some stressors like respiratory infections. Moreover, Goldberg (1986) described that after the Black Death, there were higher frequencies of females migrating to urban centres like York for work opportunities, such as temporary work (Lewis et al., 2016), yearly contracts as servants, or labour in trading. This is further confirmed by Bennett (2010) who explains that after the Black Death, orders for compulsory work were more harshly imposed on females, which resulted in their expulsion from rural environments. This could mean that the females from the sample have a more similar pattern to the extra-London region. This is confirmed by Lewis and Montgomery (2022), who analysed patterns of mobility for adolescents before and after the Black Death. The authors confirmed that after the Black Death, adolescents, particularly women migrated from rural areas to the cities.

It is also important to consider variability within the post-Black Death period, particularly for women. This extends to regional variability observed in how differently manors reacted to the negative impact of the Black Death (Bailey, 2021), as well as temporary variability. Goldberg (1986) highlights how the mortality of the Black Death opened the market for women, who were able to access work as traders and servants more easily than before. However, this short window of time did not last long. By the beginning of the 15th century, women were finding more restrictions on the work they could do (Goldberg, 1986). This is also suggested by Bennett (2010) who doubts the validity of the "golden years" of women after the Black Death period and suggests that although women had more access to work, the quality of this work was not good compared to men. For this purpose, the author cites examples such as the AD 1492 Coventry Leet ordering "idle" women into compulsory service. This leet was shows several issues for the time. Firstly, it was described as "... part of a campaign against harlots in the city" (Goldberg, 2001, p. 97). This shows an attempt to control female bodies, particularly after the relatively higher freedom they had had in finding employment in the close aftermath of the Black Death (Goldberg, 1986, 2001). Lastly, it shows the intersectionality that affected women at the time where many of them although had more freedom did not have permanent employment and had to resort to prostitution (Goldberg, 1986). This variability is observed in the results from this investigation for the London region, where a health improvement is clear for men but not as clear for women. Although this could be related to the smaller sample size. It is also possible that women's health improvement was less significant than the one experienced by men.

Stress in children was also evaluated through the estimation of growth delay. When analysing diaphyseal length, most individuals had estimated negative z-scores for all bones. This means that their bones were shorter than the reference sample provided by Maresh (1970) and corrected by Spake and Cardoso (2021). This is not surprising as the data from Maresh (1970) was derived from a modern healthy population in the United States who would have had better access to resources and health care (Geber, 2014). In addition, the skeletal sample was made up of estimated ages which has a smaller level of precision than working with a known age of a living population. The purpose of comparing the analysed population with a healthy modern sample was not to assess how healthy the past population was, but to use h charts as a reference to compare different periods within the analysed sample. Z-scores that are lower or equal to 2 standard deviations (2SD) below the reference sample reflect growth delay. The rate of bones showing evidence of growth delay for individuals dated to the pre-Black Death period was 47%. Growth delay during catastrophic contexts like the Irish famine (Geber, 2014) showed that 75.7% of children experienced growth retardation. This indicates that children dated to pre-Black Death period in extra-London were experiencing stress. However, it was lower than a severe crisis period. These frequencies of growth delay likely increased towards the beginning of the 14th century with the Great Famine. Comparisons between periods were not possible as the post-Black Death period did not have any diaphyseal length recorded for the extra-London region. When examining differences between the early and late pre-Black Death periods, there was a trend for more individuals with z-scores under 2SD during the late pre-Black Death period. When all the bones were combined, this pattern was statistically significant.

VNC grows independently from a vertebral height but can also be used to evaluate growth disruption (Newman and Gowland, 2015). There was no clear difference between the early and late pre-Black Death periods for the AP dimensions. However, there was a trend of smaller TR dimensions during the late pre-Black Death period. A similar pattern of smaller

VNC dimensions was observed in adult skeletons. When integrating these results with diaphyseal length analysis; this indicates the possibility of higher growth disruption during childhood for individuals dated during the late pre-Black Death period. Similarly to perinatal stress, growth delay shows the difficulties to fulfil nutritional requirements, particularly for children.

The late pre-Black Death period showed more controlled vertebral neural canal dimensions as well as higher frequencies of spina bifida occulta. As both conditions are related to the development of the neural canal (Northrup and Volcik, 2000; Watts, 2013), it is possible that they had similar aetiologies. However, when comparing VNC dimensions between people who had evidence of spina bifida occulta and people who didn't in the primary data, results showed not statistically significant differences (see , section C.2.1).

Specific vitamin deficiencies also provided information about childhood stress. The crude prevalence of vitamin D and vitamin C deficiencies in the study sample was small at 2.2% (n = 45/2065) overall. Most of the cases came from the pre-Black Death period in the extra-London region and occurred in non-adults under the age of 6 years. Due to the small post-Black Death sample, comparisons were not possible. When looking at the pre-Black Death period, the late subperiod again showed higher frequencies (4.5% compared to 1.59%).

The small prevalence of these conditions was expected for the medieval period. When analysing vitamin deficiencies, it is more common to register scurvy or rickets during the post-medieval period. For scurvy, the higher incidents were associated with specific environments that restricted diet (soldiers and sailors) or catastrophic contexts like famines (Mays, 2014). For rickets, its presence has often been linked to industrialisation and pollution which blocked the access to sunlight for workers. This resulted in most studies focusing on the post-medieval period (Pinhasi et al., 2006; Mays et al., 2006). Even when considering catastrophic contexts, such as the Irish famine (1845–1852), rates of specific vitamin deficiencies like scurvy are relatively lower than other broader conditions like cribra orbitalia and DEH (Geber and Murphy, 2012; Geber, 2014). Geber and Murphy (2012) argue that the limited findings concerning scurvy are due to limited diagnostic capabilities. Furthermore, in this investigation crude rates were estimates, which are also smaller than true rates.

For this thesis, the crude rates are likely an underrepresentation of the real incidence of vitamin deficiency in these populations. This is because of several factors. Firstly, both of these conditions can only be diagnosed based on multiple indicators found on the skeleton. Furthermore, to evaluate both the presence and the absence of these diseases, it is necessary for the preservation of several parts of the skeleton like the skull, ribs and distal radius for the identification of rickets (Brickley and Ives, 2008*b*; Ortner and Mays, 1998) and cranium and mandible for scurvy (Geber and Murphy, 2012), which is not always present in the osteological record. Furthermore, in order to include secondary data, this thesis estimated crude rates which as much smaller than true rates. Lastly, skeletal manifestations of scurvy and rickets represent severe instances of the conditions (Lewis, 2018). Vitamin C and D requirements are not very high. The National Institute of Health (NIH) in the United States estimates that children under 13 years require between 15 and 45 mcg of vitamin C and between 10 mcg (400 IU) and 15 mcg (600 IU) of vitamin D. Requirements in adults are higher for vitamin C and the same for vitamin D (Institute of Medicine (US) Panel on Dietary Antioxidants and Related Compounds, 2000; Institute of Medicine (US) Committee to Review Dietary Reference Intakes for Vitamin D and Calcium, 2011) (see , table B.2). It takes sustained malnourishment to develop observable skeletal lesions (Brickley and Ives, 2006). Even when these deficiencies exist, due to the plasticity of the non-adult skeleton, individuals can recuperate without retaining any markers in the skeleton (Lewis, 2018). This makes these conditions very difficult to identify. X-rays can be used in some cases to aide in the identification of rickets and scurvy (Brickley and Ives, 2008*a*). Regretfully, they were not available for this investigation.

The pre-Black Death sample did not include any skeletons dated to the Great Famine. Had those been included it is possible that the rates of scurvy and rickets would have been higher as was observed in other famine contexts (Geber and Murphy, 2012). In London, the Black Death cemetery presented very few instances of either disease, showing that if there was a high prevalence, it did not last a long time. This shows the limitations in the analysis of these conditions, particularly during the medieval period. Results from these conditions are similar to what was observed with other indicators. The extra-London region showed higher prevalence during the pre-Black Death, which was concentrated in the late pre-Black Death period. However, these differences were not statistically significant. The late pre-Black Death period showed higher frequencies of pathological conditions, but the differences were not statistically significant. This result agrees with other indicators that suggest a possible trend for the late pre-Black Death period to have evidence of higher stress, but no robust differences with other periods.

Comparisons between the extra-London and London region showed higher frequencies of scurvy during the pre-Black Death period in the extra-London region, particularly for non-adults, which presented a crude prevalence rate of 21/875, 2.4%. The London regions did not present any evidence of scurvy or rickets for the pre-Black Death period likely due to the small size of the sample. The Black Death period also exhibited low frequencies of the conditions with no cases of scurvy and only one recorded case of rickets (0.3%). The post-Black Death period exhibited the highest crude rates of scurvy and rickets (3/210, 1.4% for both conditions). This could indicate that the population of the extra-London region had more restricted access to fresh fruit and vegetables during the pre-Black Death period. However, these data should be treated with caution most of the scurvy cases were concentrated on the Stoke Quay site. This may be related to Stoke Quay being an urban site near a river (Brown et al., 2020). Sailors made up part of the population of the site. Scurvy is found in higher prevalent among sailors due to limitations in carrying fresh fruit (Rielly, 2020). However, these rates were observed in children, which were unlikely to be subjected to these dietary restrictions. These children may represent a larger part of the population with little access to sources of vitamin C. Stable isotope analysis of populations of Stoke Quay has shown evidence of high marine consumption. This also agrees with zooarchaeological finds and the location of the site (Farber, 2020). One of the most significant changes in medieval diet was related to a moment referred to as the "fish event horizon". This describes a change dated around AD 1000 where widespread consumption of fish was included in the medieval diet. This was observed through a shift from freshwater fish like eel to fish caught in the sea like cod and herring (Harland et al., 2016; Orton et al., 2014; Müldner, 2016). The vitamin content of fishes like cod and herring is low (see , table B.4), which can result in a low intake. Furthermore, the consumption of fish, particularly in urban environments with a lack of hygiene may have led to parasite infections. Although consuming dry and brined fish extends the shelf life of fish, it does not eliminate the risk of parasitosis (Hazards , BIOHAZ) which would have further hinged absorption of nutrients. Low consumption of vitamin C may have particularly affected pregnant and especially breastfeeding women who have higher requirements of vitamin C (see , table B.2). Clinical studies have shown that women who have a low intake of vitamin C produce milk which is also low on the vitamin (Tawfeek et al., 2002). This may have affected young children under the age of 5 years, resulting in more severe cases of scurvy. A similar situation may have affected individuals in London who had also similar access to cod and herring (Orton et al., 2014). It is also possible that the urban environments of Stoke Quay and London contributed to higher frequencies of scurvy and rickets. Lastly, it is important to consider the possibility that due to differential diagnostic criteria among excavations, the evaluations at Stoke Quay resulted in higher frequencies of scurvy. Further studies are required.

Similar to rickets and scurvy, in non-adults, tuberculosis was only observable in two children ages 0-5 years dated to the Black Death period and two teenagers ages 12 to 17 years dated to the post-Black Death period. Due to similar difficulties in the diagnosis of tuberculosis, these frequencies are likely an underrepresentation. Although they may represent a moment of higher crisis during the Black Death, they are likely related to the increased urbanisation of London, where air pollution affected the population, particularly non-adults Lewis (2002*a*).

To summarise, overall results from childhood health analysis show growth disruption and evidence of stress during the pre-Black Death period in the extra-London region. As the post-Black Death sample was small, few comparisons were possible. Those comparisons showed no differences. When comparing the early and late pre-Black Deaths periods there was a trend in all indicators for the late pre-Black Death period to present higher levels of stress. This was in a few instances statistically significant, but in many cases, it only showed a trend. The London region presented clearer patterns showing that the period of the Black Death was the most affected by stress, while individuals dated to the post-Black Death period showed health improvement. One exception was observed for scurvy, rickets, and tuberculosis where individuals dated to the post-Black Death were the most affected period. This could be related to an increased urban environment and changes in diet.

Results for the extra-London period were dissimilar to previous studies in London. Historical and archaeological sources mention a period of better life quality due to better access to food resources and higher wages for the workers and peasants (Bailey and Rigby, 2012; Bailey, 2021; Hatcher, 1977). These results agree with more recent research, also looking at individuals dying outside medieval London (Dittmar et al., 2021a). This may indicate that the extra-London may have experienced stress, but the changes were not as marked as London. Bailey (2021) mentions that the consequences of the Black Death were highly variable throughout England. In this investigation, most of the post-Black Death period came from St. Anne's Charterhouse in Coventry, which was unlikely to be representative of all of the region. It is necessary to expand the early pre-Black Death subperiod sample and include more sites dated to the post-Black period to confirm this conclusion. Additionally, differences in scurvy in Stoke Quay also point out variability in the extra-London region before the Black Death. Similarly to the results obtained from comparing the early and late pre-Black Death period in the extra-London region, both regions showed higher frequencies of stress in females. These differences were only sometimes statistically significant (DEH), and show that women likely experienced more stress than men both before and after the Black Death.

6.3 Adolescent development: puberty, menarche, and final adult stature

The analysis of the adolescent growth spurt and menarche is a relatively new advance in bioarchaeology compared to the evaluation of indicators of stress like cribra orbitalia and dental enamel hypoplasia, and hence, data were only available for the primary sample from the extra-London region. The age of menarche can provide valuable information on population health over generations, and delays in ages of attainment have been linked to poor nutrition, infection and poverty among many other detrimental factors (del Pino et al., 2005; Torres et al., 2019). In the bioarchaeological record, identifying the age of menarche at an individual level rests with the recovery of an ossified but unfused iliac crest, which is rare (Lewis, 2016*b*). Hence an alternative method to explore this feature, developed by DeWitte and Lewis (2021) was employed. This method involved establishing the mean age of women before menarche (including deceleration) compared to the mean ages of women post-menarche (maturation to completion). Information for the post-Black Death period was not available. When comparing the early and late pre-Black Death periods, the earlier period showed higher pre-menarche ages (i.e., the age of attainment was later) when compared to the later period (Early pre-Black Death = 15.73 ± 0.23 years of age and Late pre-Black Death = 14.18 ± 3.43 years of age), while the late pre-Black Death period showed higher mean ages for the post-menarche stage (early pre-Black Death 20.31 ± 0.45 years of age and late pre-Black Death 22.03 ± 3.51 years of age). This was similar to findings from DeWitte and Lewis (2021) who found a similar pattern for Spital Square London. This was 14.38 ± 3.21 years and 13.19 ± 2.90 years for the early and late pre-Black Death pre-menarche mean age respectively and 18.72 ± 3.35 years and 21.58 ± 2.64 years for the early and late pre-Black Death post-menarche mean ages.

Similarly to the study done by DeWitte and Lewis (2021), results in this investigation show older post-menarche mean ages for women during the late pre-Black Death period from the extra-London region. This indicates that women during the late pre-Black Death period were experiencing higher levels of stress which delayed their development.

Although the post-menarche mean ages support delayed menarche timing for the late pre-Black Death period, the pre-menarche stage does not. The reason for this is not due to levels of stress, but due to the age distribution of the sample. The early pre-Black Death period sample does not have individuals ages 10 to 13 years, while the late pre-Black Death period has individuals ages 10 and 11 years. This affects the estimation of the pre-menarche stage means. This highlights a potential problem in the analysis of mean pre- and post-menarche ages. Although the analysis allows for smaller samples, the age distribution of the sample is key. This was controlled in the investigation by DeWitte and Lewis (2021) by comparing the composition of each subperiod sample. However, this was not possible in this analysis due to the absence of young adolescents. This is regretfully a common problem in the analysis of pubertal growth (Lewis et al., 2016).

The mean ages of both pre-menarche and post-menarche for individuals dated to the pre-Black Death period were higher than the results reported by DeWitte and Lewis (2021) for London during the early and late pre-Black Death period. This could indicate that females in the region were experiencing more stress. Further studies expanding the sample to have a better representation of all ages are required.

Results from pubertal analysis in the extra-London region agree with previous results like cribra orbitalia and DEH, which indicate that women likely experienced more stress during the pre-Black Death period, particularly the late pre-Black Death period. This could be due to women being more vulnerable due to being socially considered weaker, being subjected to more informal education and more precarious work, and legally being allowed less autonomy (Bennett, 2010; Kline, 2003). This would make them more vulnerable, particularly in periods of crisis. This also affected the woman's entire cycle showing evidence of stress during childhood, adolescence, and potentially in older ages during pregnancy.

The analysis of menarche is often related to other indicators of stress such as long bone length. DeWitte and Lewis (2021) utilised the tibia length as a proxy to analyse differences in stature between pre- and post-Black Death period, London. In this investigation, due to the small sample, comparisons were only possible between the early and late pre-Black Death subperiods. As adolescents are undergoing the process of epiphyseal fusion, many individuals had only one epiphysis fused. For this reason, it was not possible to measure either the diaphyseal nor long bone length. Results were integrated with long bone length means from the adult sample. The mean length of the femur, tibia, fibula, radius, ulna and humerus were compared between the subperiods. Females showed smaller long bone means during the early pre-Black Death, but only statistically significant for the tibia and ulna (t(29.6) -3.1, p=0.005 and t(30.4) -2.5, p=0.019 respectively). This was statistically significant for the tibia and ulna. Males showed smaller long bone length mean during the late pre-Black Death subperiod for all bones except the radius. This indicates that males were experiencing stress during the late pre-Black period. However, results for females seem to contradict the evidence from pre- and post-menarche mean ages. DeWitte and Lewis (2021) notes that the relationship between puberty stage and height may vary between males and females as well as between populations. The authors suggest that contrary to what was expected, females after the Black Death in London experienced a decrease in height along with earlier age menarche. They explain that due to improved living conditions after the Black Death (Bailey, 2021; Hatcher, 1977), menarche started earlier initiating the fusion of the long bones due to an energy trade-off substituting growth for reproduction. Furthermore, the tibia is the last bone to fuse and stress concentrated in this bone may further signal trade-off substitution during the Black Death period. This discrepancy in height was also observed in DeWitte (2017) and shows the complicated relationship between stature and stress where higher statures are not necessarily indicators of better health. Similarly, in this investigation, the smaller bone length for the tibia of females during the early pre-Black Death period may be related not to higher stress, but to premature bone fusion caused by early menarche. This would agree with previous results which signal a trend towards an increase of stress in women towards the late pre-Black Death subperiod.

For the London region, pubertal analysis was not possible and comparisons between bone length means for females were only possible between the pre-BD and BD periods but not during the post-Black Death period. The pre-Black Death period showed smaller mean lengths for all bones except the tibia. These differences were not significant. Comparisons with previous analyses like DeWitte and Lewis (2021) find similarities as the tibia length was not related to higher stress, but premature fusing caused by early menarche. However, it is important to understand the differences in the analysis between both investigations. DeWitte and Lewis (2021) compare the 1200-1250 AD period with the post-Black Death period (AD 1350-1550) and it is limited to the Spital Square site and does not include groups dated to the Black Death period itself. The authors only analysed tibiae length. This thesis analysed a broader period for the pre-Black Death (AD 900-1348 for pre-Black Death subperiod and 1100-1348 for late pre-Black Death subperiod). The sample included a small sample from Spital Square (see section 4.1.3.2.5) for a description of limitations in the access to this collection), as well as other sites like Guildyard Hall, Merton Priory, and St Mary's Graces. The sample also included populations from the Black Death cemetery East Smithfield, which were not included in the investigation by DeWitte and Lewis (2021).

Results showed for the tibia (bone analysed by DeWitte and Lewis (2021) to evaluate stature), had smaller dimensions during the Black Death. This agrees with the previous study as it also reflects a period of stress higher in the period leading to the Black Death. For DeWitte and Lewis (2021), the closest period to the Black Death was AD 1200-1250. For this thesis, this stress was observed in individuals dated to the Black Death who likely grew up during the period of acute crisis before the plague (AD 1290-1348) (Dyer, 2002). Many victims during the Black Death period had lived or were direct descendants of people who lived through the Great Famine, so it is expected that this period presented the highest prevalence rates of disease and stress. The authors do not look at differences in other bones, so it is not possible to compare results other than tibia length means. Results from this thesis showed smaller dimensions of the tibia during the Black Death period, but other bones had shorter lengths during the pre-Black Death period. This is not what was expected as short tibia length was related to early menarche and other indicators have shown evidence of higher stress during the Black Death period. Further studies of pubertal development and menarche timing of the population dated to the Black Death period are required to better interpret the causes for the shorter tibial length.

Further evidence of higher stress in females is observed in the few cases of tuberculosis recorded. In adults, tuberculosis was recorded only in the five individuals. Four cases were recorded in the extra-London region dated to the pre-Black Death period and one was recorded in the London region. The three case from the extra-London region were women, two were aged 17-25 years and two was aged 25-35 years of age. The only male was dated to the post-Black period in the London region. The incidence of tuberculosis was probably much higher due to the high burden of disease that respiratory illnesses had during the medieval period (Robb et al., 2021). However, considering the low percentage of cases where the illness is visible on the osteological record and the difficulties in its diagnosis (Rohnbogner and Lewis, 2017; Lewis, 2018), the expected rates observable in the osteological record are much smaller. This can be observed in works like Mays et al. (2001) who have evaluated cases of tuberculosis in Wharram Percy through aDNA analysis. Results showed nine cases, from which two individuals were positive for the bacterium without having any spinal lesions and five had no lesions on the ribs (see , table E.4). From the nine individuals identified as having tuberculosis by Mays et al. (2001), six were male and tree were female. All females were under the age of 35 while most men were over the age of 50 years (and one unaged adult). Although the sample is too small to be representative, it provides information signalling that women were likely to become sick and die younger from conditions related to stress and immunosuppression than men. This agrees with previous results from this investigation.

An important factor to take into consideration is also the analysis of individuals identified as male. Males showed a different pattern than females, exhibiting the smallest bone lengths during the Black Death period. The only statistically significant difference was observed when looking at the humeral length mean which was higher during the post-Black Death period. These results seem to contradict what was observed in females. However, the relationship between patterns of pubertal developments and stature varies between sexes and populations (DeWitte and Lewis, 2021). It is possible that males did not go through the process of premature fusion of the long bone epiphyses and longer bone length reflects an improvement in the quality of life for males during the post-Black Death period.

A similar pattern was observed in VNC dimension means, which showed similar values to long bone length in males and dental enamel hypoplasia crude prevalence rates. Individuals dated to the Black Death period exhibited statistically significantly smaller means than during the pre- and post-Black Death periods, particularly for men. Similarly than with long bone length, this indicated that the men during this period experienced more stress, which decreased after the plague.

Finally, oral health provided data to complement the image of health before and after the Black Death. Dental caries is a multifactorial disease (Ferraro and Vieira, 2010). Although it has a relationship with health status, it is a more direct indicator of diet composition (Larsen, 1995). This does not provide the same information as more precise invasive studies on stable isotopes (Pollard, 1998). However, it can show the introduction of softer high carbohydrate foodstuff or softer food which can lead to the easier formation of dental caries (Tayles et al., 2000). The presence of dental caries can both indicate stress (Lewis, 2018) and higher access to resources (Walter et al., 2016). For this reason, it is crucial to contextualise results to properly interpret them.

Results from this investigation showed higher frequencies of dental caries during the post-Black Death period for both regions for adults. This agrees with the inclusion of more cariogenic foods in the diet like wheat bread and ale (Bailey, 2021). Medieval diet was different depending on the socioeconomic class. Higher classes had more access to meat and exotic foods, while peasants' diet was composed mostly of grain and vegetables. This does not mean that peasants did not consume meat, but that their access was limited (Burt, 2013; Müller, 2010). After the Black Death, the lack or workers gave peasants more power to demand higher wages (Hatcher, 1977). Although this was controlled by the government through legislation like the AD 1349 Ordinance of labourer, this was not always followed and it resulted lower classes, like the individuals analysed in this thesis, having access to better foodstuff (Bailey, 2021). This not only included meat but also ale and better-quality bread. During the medieval period bread made from ingredients like peas, beans rye, maslin, and barley, was considered lower quality than while wheat (Dyer, 2023). Both ale and wheat bread from more refined flour are highly cariogenic foods This would have resulted in higher frequencies of dental caries(Mant and Roberts, 2015).

When looking at each region, individuals from the extra-London region dated to the post-Black Death period were mostly from St Anne's Charterhouse. Historic records indicated that the Charterhouse produced ale (Soden and Rylatt, 1995). This agrees with higher frequencies of dental caries observed. In London, the high frequencies of caries signals an improvement in health and diet due to better access to foodstuff after the Black Death, particularly for men.

When looking at differences between males and females, in the extra-London region, women showed higher frequencies of caries in all periods and subperiods (although the post-black Death period was too small to compare males to females). Similarly, in the London region, females showed higher frequencies of dental caries and during the pre-Black Death period. Males showed higher frequencies of dental caries during the post-Black Death.

Increased frequencies of dental caries in females were also noted by Walter et al. (2016), who looked at frequencies of caries in Spital Square and St Mary Graces. This sample overlaps slightly with part of the sample for this analysis. However, this is not the only instance where females presented higher frequencies of dental caries. Analysis of agricultural and pre-industrial societies has shown in some instances higher frequencies of dental caries in women compared to men. This has been explained due to different gender roles concerning labour resulting in differential access to cariogenic foods for females and males (Hillson, 2005). However, this proposition has been challenged. Lukacs (2008) proposes that societies with increased demands on the reproductive system, including an increase in fertility, would result in higher frequencies of caries in females. Walter et al. (2016) also suggests that periods of high stress like famine may contribute to a higher prevalence of dental caries. Results from this investigation may indicate stress experienced by females and non-adults in both regions, particularly for individuals from the extra-London region during the pre-Black Death period.

As mentioned before, frequencies of dental caries in men showed evidence of a larger improvement in their access to resources during the post-Black Death period compared to women. This does not reflect stress, but changes in diet. During this period, there was increased access to resources (DeWitte, 2017). This was due to more demand for work and higher wages. Although there were limitations imposed to demand service and restrict wages, Bailey (2021) explains that in many cases, this was not followed due to the high necessity for workers. This higher access was observed in foodstuff like more refined wheat bread and ale (Dyer, 2023; Bailey, 2021)

In summary, adolescent development and adolescent and young adult diet show that women likely experienced more stress during the pre-Black Death period, particularly during the late pre-Black Death period in the extra-London region. In the London region, stress peaked during the Black Death period and decreased towards the post-Black Death period. This was observed more clearly in men who had better access to foodstuff resulting in higher statures and an increase in dental caries. Women showed more complicated patterns presenting smaller statures related to early menarche rather than higher stress. The differences between London and the extra-London period highlight the significance of analysing sites outside the major urban centre.

6.4 Limitations of the study

This study was originally designed to be based on a sample for primary analysis of over 1000 individuals. The initial planned sample was going to include individuals from Stoke Quay, Barton-Upon-Humber, Littlemore Priory and Oxford Castle. The restrictions put in place during the COVID-19 pandemic limited access to osteological collections. Information from Oxford Castle and Littlemore Priory sites became inaccessible, and Barton-Upon-Humber and Stoke Quay were only available as secondary data. This resulted in the primary data collection being reduced to just 288 skeletons and a substantial portion of the data (86.1 %) being added as secondary. This addition affected the composition of the sample and the quality of the data, as the databases utilised did not always record all the indicators examined in this thesis. For example, the data available from individuals from the London sites and Stoke Quay in the extra-London region did not have information on pubertal development (puberty stage estimation). Similarly, the available data from Stoke Quay did not provide information about vertebral neural canal dimensions.

The sample did not have equal representation in all regions and periods. Due to the limited time frame of the post-Black Death period and difficulties locating securely dated sites outside London, the group from the extra-London region that dated to the post-Black Death period represented the smallest sample in the investigation. This situation is consistent with struggles observed in previous research (Lewis, 2016*b*).

The dating available for sites outside of the London region also differed from the one utilised in studies based in London. Due to the size of the city and the number of deaths caused by the Black Death, a cemetery dedicated to the victims of the plague (East Smithfield Cemetery) was opened in London. This cemetery allows the identification of individuals who lived and died during the catastrophic period. Although there were plague victims and small plague pits identified outside London (e.g. Cambridge (Giberti, 2019; Willmott et al., 2020), these remains were not available for this analysis.

Although there is evidence of victims of the plague, limitations in dating for both the London and the extra-London region prevent identifying individuals dated to the period immediately before the Black Death (AD 1290-1348). This prevents the study of that particular period and evaluates the existence of a crisis immediately before the plague. In the London region, this difficulty was solved by analysing the period previous to the crisis. DeWitte and Wood

(2008) were able to identify a period closer to the Black Death in London between AD 1200 and 1250. This period did not occur immediately before the Black Death (AD 1290-1348) but allowed the authors to define the late pre-black Death period more narrowly. This precise phasing was not available in sites outside London, which meant that the analysis could not compare individuals who were directly affected or died during the crisis period, nor individuals who directly survived the Black Death, but their descendants (DeWitte, 2017). To reduce the bias created by the lack of precise dating, this investigation expanded the scope of the analysis, changing the focus of the study to investigate if there was a broader crisis period before the Black Death. This was done by extending the late pre-Black Death period to AD 1100-1348. This allowed utilising the dating available outside London to evaluate broader trends that may indicate a decline in childhood and adolescent health before the crisis period.

The combination of primary and secondary data also presented some challenges. While secondary data is a valuable resource allowing large numbers of individuals to be included in the study, it is static data, which means information on newly developed methods like pubertal analysis was not available. Hence, there was limited scope to compare information on puberty status, VNC dimensions, number of lines, and age-at-formation of linear enamel hypoplasia. These limitations were present in all secondary datasets but mostly affected comparisons between the London and extra-London regions. For example, pubertal status and age-at-formation of linear enamel hypoplasia data were not available in any of the London sites, narrowing the analysis to only the extra-London region. In addition, the lack of information about the number of bones and teeth in the datasets meant most analyses relied on crude prevalence rates (cribra orbitalia, rickets, scurvy, tuberculosis, spina bifida, enamel hypoplasia). This was most observable in the Barton-Upon-Humber collection, where there was only a record of observed pathologies and conditions without clarifying the difference between the absence of an indicator of stress and the lack of evidence for its evaluation. Mean dental ages for the non-adults were also not always available, hindering growth analysis for some groups. In some cases, ages had to be grouped into broader categories (e.g., 0-5 years, 6-12 years, 13-17 years) to conform to the secondary site databases.

Although this resulted in some limitations of the analysis, the addition of secondary databases allowed increasing the sample size to make it more representative. The age categories permitted comparison of same-age groups, reducing the biases caused by the osteological paradox (Wilson, 2014). Furthermore, where it was possible, for example, child mortality and diaphyseal length, age categories were further narrowed down for a more detailed analysis.

Osteological analysis also has limitations. Many physical conditions are not observable in the osteological record as they do not leave evidence on the bone (e.g. bubonic plague). Other pathologies can leave evidence on the bones, but this is only present in severe or chronic cases. For example, in tuberculosis, evidence of osteological lesions only appears in approximately 3 to 5% of cases (Lewis, 2018). In this investigation, this was considered in the selection of methods (see section 4.2) and in the interpretation of results by considering the possible under-representation of illnesses and other courses of evidence of disease and mortality such as historical records (Robb et al., 2021).

Morphological analysis of and archaeological approaches also struggle to evaluate gender identity and identify individuals outside the binary male-female spectrum (Power, 2020). As it was explained in more detail in chapter 3 (see section 3.1.2.1)

Lastly, this analysis focused on differences by period, region, age, and estimated sex. Many more variables could affect health status. Historical records show differences between small regions in the UK. For example, Bailey (2021) describes areas of County Durham as having a delayed recovery after the Black Death due to having already been impoverished before the plague. However, for some tenants, mass death caused a "one-off financial bonanza" Bailey (2021, p. 71), which brought more revenue and resulted in a different recovery process. This was also sometimes observed within each region as well. Goldberg (1986) remarks that in urban York after the plague, women who migrated from urban areas were able to find work as servants. However, many still struggled, living in impoverished sectors of the town. The full scope of intersectionality, including variations between and within sites (especially for the extra-London region), between smaller subperiods, social class, standing in the community, cultural background, genetic predispositions, and religion, among others, cannot be entirely included in this analysis due to limited contextual evidence for each individual.

There are also intangible social aspects that affect population health. For example, current investigations have highlighted the negative mental health consequences of modern pandemics such as HIV (Eaton and Kalichman, 2020) and COVID-19 (Hossain et al., 2020; Liu et al., 2020). Although there are historical sources that can provide contextual evidence by which we can infer the mental health status of individuals in medieval England, this evidence is limited and its effects cannot be observed in the osteological record. This does not mean that mental health should be ignored. It is important to consider it in the discussion by contextualising the results and how could the evidence of stress have impacted their lives.

7 | Conclusions

This thesis explored childhood health in the periods before and after the bubonic plague pandemic of the 14th century in England. This investigation focused on the period leading to the Black Death to evaluate if childhood health was already deteriorating before the outbreak. For this purpose, health during the formative years and adolescence was evaluated. Indicators of physiological stress which develop *in utero* were also analysed to identify potential stress experienced by the mother (Gowland, 2015).

The bioarchaeological analysis of health has limitations. Many pathologies and conditions which reflect nutritional stress do not leave identifiable lesions on the skeleton (Lewis, 2018). This may be because the condition only affected soft tissue, or due to the individual either recuperating or dying before lesions could be formed (Wilson, 2014). In this investigation, the need to analyse individuals who died within very specific time frames inevitably resulted in small sample sizes, particularly of individuals who died in the years immediately after the Black Death outside London, who tend to be buried in much larger, and less accurately dated cemeteries. Differences in methodology limited the integration of some of the secondary data, particularly because the focus on adolescent health is a recent advance in bioarchaeology, and so data on puberty status and stress indicators such as reduced VNC were not previously recorded. It was also not possible to fully explore ages at menarche before and after the Black Death, particularly in the extra-London sites, due to limited sample sizes. To combat some of these limitations, particular emphasis was placed on dental enamel hypoplasia which was regularly recorded in the non-surviving non-adults and surviving adults. This inclusion allowed for increasing the sample size and reducing the mortality bias caused only by analysing children who had perished (Wilson, 2014). While it was only possible to estimate the biological sex of older non-adults and adolescents in the primary sample, the availability of stress indicator data for adult males and females allowed gender-based differences in childhood to be reconstructed. The use of crude prevalence rates derived from the primary and secondary data, for conditions such as cribra orbitalia, vitamin D and C deficiency and tuberculosis allowed the whole sample to be combined and compared. Despite these constraints, several important conclusions can be drawn from this analysis.

1. **There was also a trend for poor health in the extra-London regions in the period leading to the Black Death period.**

   This included perinatal and maternal stress as well as higher frequencies of pathologies in children. However, when comparing periods, few statistically significant differences were detected. The most significant differences detected were in post-menarche mean ages, where women from the extra-London region showed statistically significant older mean ages during the late pre-Black Death, reflecting growth delay. This agreed with previous studies like (DeWitte and Lewis, 2021) which found similar results for the London region. When the primary and secondary data were combined, crude prevalence rates indicated poor health during the late pre-Black Death period in the extra-London region. This trend affected females more than males suggesting their more vulnerable position in society.

   Although the post-Black Death sample for those outside London was small, there was no evidence that there was an improvement in health following the pandemic. This contrasted with the pattern found in London. In London, individuals dated to the post-Black Death period, particularly men, showed health improvement. However, only a few differences were statistically significant. The analysis of London provided valuable insight into the interpretations of the results from the extra-London region.

2. **The London region showed higher frequencies of indicators of stress during**

the Black Death period.

The Black Death period sample represented populations that had experienced or directly descended from people who had lived through the crisis before the Black Death. His resulted in these results showing not only stress experienced during the plague but also during previous crises like the Great Famine in the decades leading to the Black Death. This population was not equally represented in the extra-London region. As there was no sample dated to the period of the plague. It is possible that the peak moment of crisis in the extra-London region remained unobservable.

3. **Patterns of health after the plague differed between the extra-London and London region.**

   Although the post-Black Death sample for those outside London was small, there was no evidence that there was an improvement in health following the pandemic, compared to the pattern observed in London. This agreed with previous studies such as the "After the Plague" (2016-2020) project from Cambridge University which did not find significant differences between periods (Dittmar et al., 2021a). Alternatively, in London, individuals dated to the post-Black Death period, particularly men, showed health improvement. However, only a few differences were statistically significant. The analysis of London provided valuable insight into the interpretations of the results from the extra-London region.

This thesis has shown that London was a unique urban environment within England, and although it provides valuable sites and information about the impact of the Black Death on the health of the population, it does not illustrate the experience throughout England. Outside London, there was a great variety of living conditions which included serfdom and free paid work, variability in the access to food and resources, and different work opportunities (Bailey, 2021), which affected the health of the population differently. It should be expected that the impact of the pandemic would also have been experienced in a different way. This study has highlighted the importance of continuing this line of investigation beyond London.

7.1 Future work

This thesis has opened multiple lines of investigation that could be continued in future work. Firstly, limitations in the size of the extra-London sample for the post-Black Death period prevented many statistical comparisons with the pre-Black Death period. Expanding the sample size to include multiple sites outside London, like for example the sites that due to the lockdown caused by the COVID-19 pandemic were not accessible for this investigation (Oxford Castle and Littlemore Priory in Oxfordshire) would offer the possibility of studying the changes after the Black Death and particularly the difference between areas. It would also provide the opportunity to better evaluate how the plague affected males and females differently, as previous research in London has shown that women did not experience the same health improvement after the plague as men did.

Results also exhibited differences in health before the Black Death between regions within the extra-London region. This was observed particularly in the Stoke Quay site. Expanding the sample by differentiating urban and rural areas would allow for tackling regional variations and how increased urbanisation interacted with health and the plague.

This thesis showed that stress before the Black Death affected perinatal and adolescent growth. Increasing the study sample by including more and better dated sites, would allow for more robust comparisons between periods. Furthermore, perinatal growth analysis could be enhanced by applying new techniques to reduce bias in evaluating the presence of stress. Correlating pubertal growth with diaphyseal length in the same individual may aid in further studying the discrepancies between stature and pubertal development observed in this thesis, where females may have experienced earlier epiphyseal fusion due to early menarche, rather than increased stress. A larger sample of individuals dying in the pre- and post-Black Death periods outside London would also allow for more data on stress indicators such as reduced VNC to be examined, and in particular, allow more work to be done on its potential relationship to spina bifida occulta. Finally, stable isotope and microscopic analysis of dental enamel hypoplasia may help elucidate potential differences in weaning ages and stress during formative years and further explore *in utero* and possible maternal stress.

Previous research has signalled that the Black Death, although a period of crisis in all of England, was possibly not experienced equally across the country (Bailey, 2021). Combining the bioarchaeological analysis with new zooarchaeological projects such as RATTUS (2023-2027) at the University of York (Orton et al., 2023) could provide more information about how the plague spread to different areas in England. This could be further developed by including the analysis of skeletal remains from plague pits outside London, such as the recent discovery at Thornton Abbey (Willmott et al., 2020).

The focus of this investigation was England. However, the Black Death was a pandemic which also affected the rest of Europe, as well as Asia and Africa (Benedictow, 2004). Results from this investigation could be used in broader collaborations to analyse the plague more globally, and in particular focus on Asia and Africa would bring more attention to areas also affected by the plague that have so far, been neglected.

Finally, although the bubonic plague is more often associated with plagues in the past, the disease has not been eradicated and continues to be a current concern (Raoult et al., 2013; World Health Organization, 2018*b*; Yang, 2017). Information from this thesis can aid in the study of population health concerning the plague in current societies. Furthermore, interpretations from this thesis can be compared with the analysis of other pandemics such as the recent COVID-19 pandemic. The current climate crisis indicates that pandemics and catastrophic events are likely to increase (Lafferty, 2009). This makes the study of pandemics both past and contemporary fundamental. Although the diseases may not be the same, results from this investigation may provide information about the conditions that existed prior to the outbreaks, and inequalities during them, that could be used to better understand modern and future pandemics.

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Appendices

In this section, additional figures and tables with information described in chapter 2, 3, and 5.

B Additional information

The following figure (B.1) shows the distribution of sites with rat remains in England by region and century.

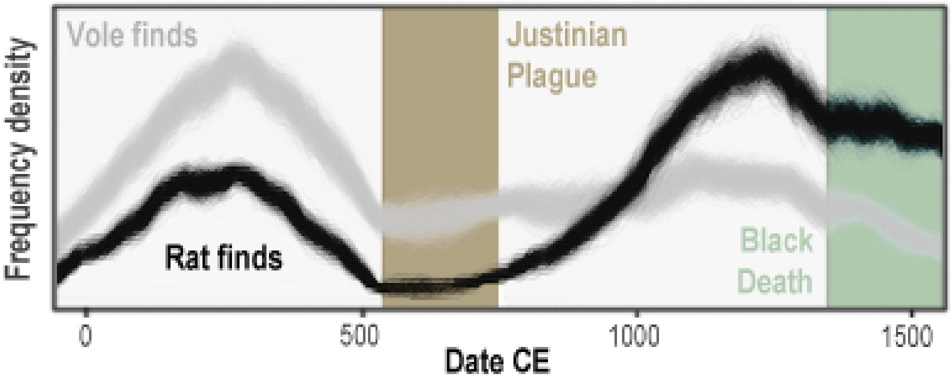


Figure B.1: *Distribution of rat finds through time modified from (Orton et al., 2023).*

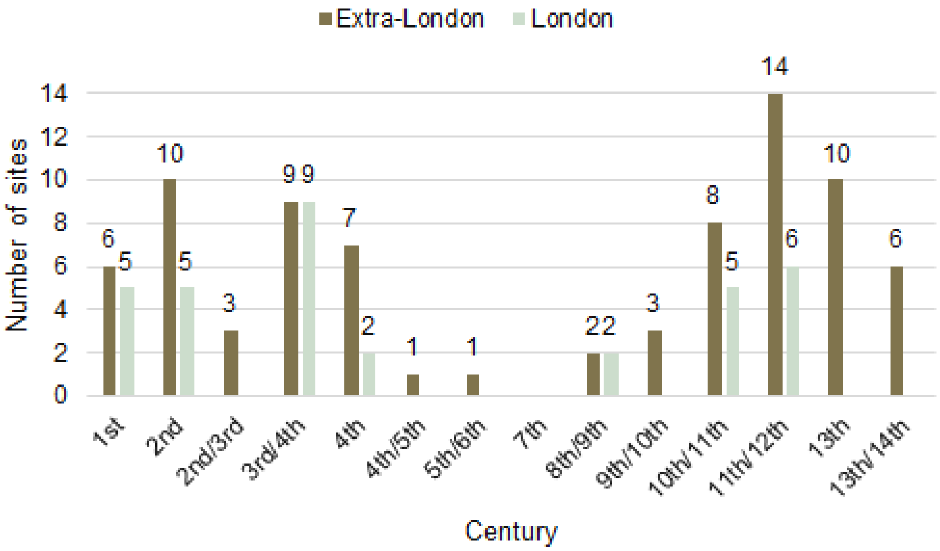


Figure B.2: *Frequencies of sites with rat remains by region and century in England modified from (Rielly, 2010).*

The following tables show the recommended dietary allowances for vitamin C, D (Table B.2), folate, vitamin B12 (Table B.1), iron, and vitamin K (Table B.3).

Table B.1: *Recommended dietary allowances for folate and vitamin B12. Table modified from Institute of Medicine (US) Standing Committee on the Scientific Evaluation of Dietary Reference Intakes and its Panel on Folate, Other B Vitamins, and Choline (1998)*

Age	**General public**		**Pregnancy**		**Breastfeeding**	
	Folate (mcg)	**Vitamin B12 (mcg)**	**Folate (mcg)**	**Vitamin B12 (mcg)**	**Folate (mcg)**	**Vitamin B12 (mcg)**
Birth to 6 months[1]	65	0.4	-	-	-	-
7–12 months	80	0.5	-	-	-	-
1–3 years	150	0.9	-	-	-	-
4–8 years	200	1.2	-	-	-	-
9–13 years	300	1.8	-	-	-	-
⩾14 years	400	2.4	600	2.6	500	2.8

[1] Adequate intake (AI)

Table B.2: *Recommended dietary allowances (RDA[1]) for vitamin C and D. Table modified from Institute of Medicine (US) Panel on Dietary Antioxidants and Related Compounds (2000) and Institute of Medicine (US) Committee to Review Dietary Reference Intakes for Vitamin D and Calcium (2011)*

Age	**Male**		**Female**		**Pregnancy**		**Breastfeeding**	
	Vit C[2] mg	**Vit D mcg (IU[3])**	**Vit C mg**	**Vit D mcg (IU)**	**Vit C mg**	**Vit D mcg (IU)**	**Vit C mg**	**Vit D mcg (IU)**
Birth to 6 months[4]	40	10 (400)	40	10 (400)	-	-	-	-
7-12 months[3]	50	15 (600)	50	15 (600)	-	-	-	-
1-3 years	15	15 (600)	15	15 (600)	-	-	-	-
4-8 years	25	15 (600)	25	15 (600)	-	-	-	-
9-13 years	45	15 (600)	45	15 (600)	-	-	-	-
14-18 years	65	15 (600)	75	15 (600)	80	15 (600)	115	15 (600)
19-69 years	75	15 (600)	90	15 (600)	80	15 (600)	120	15 (600)
⩾70 years	75	20 (800)	90	20 (800)	-	-	-	-
Smokers	Smokers require an additional 35 mg of vitamin C intake per-day than non-smokers							

[1] RDA: "the average daily level of intake sufficient to meet the nutrient of nearly all (97%–98%) healthy individuals", while adequate intake (AI) refers to requirements for nutritional adequacy and is used when there is not sufficient evidence to estimate an RDA (Institute of Medicine. Food and Nutrition Board, 2001); [2] Vit= Vitamin; [3] IU = International unit; [4] Adequate intake (AI)

Table B.3: *Recommended Dietary Allowances for iron and adequate intakes for Vitamin K. Table modified from Institute of Medicine. Food and Nutrition Board (2001)*

Age	**Males**		**Females**		**Pregnancy**		**Breastfeeding**	
	Iron mg	**Vitamin K mg**	**Iron mg**	**Vitamin K mg**	**Iron mcg**	**Vitamin K mcg**	**Iron mcg**	**Vitamin K mcg**
Birth to 6 months[1]	0.27	2	0.27	2				
7–12 months	11	2.5	11	2.5				
1–3 years	7	30	7	30				
4–8 years	10	55	10	55				
9–13 years	8	60	8	60				
14–18 years	11	75	15	75	27	75	10	75
19-49 years	8	120	18	90	27	90	9	90
⩾50 years	8	120	8	120				

[1] Adequate intake (AI)

The following table (Table B.4) shows the nutritional information of the most commonly consumed fish in England during the medieval period. Firstly, freshwater fish (eel) which was found in several sites in England like Stoke Quay (Ipswich), Blue Bridge Lane (Yorkshire), and Fishergate York (Yorkshire) Farber (2020) and secondly, sea fish like cod and herring, which started to become widely consumed after AD 1000. For preservation, cod was salted and dry and herring was brined (Barret, 2016).

Table B.4: *Nutritional information of eel as well as cod and herring both fresh and preserved per 100g modified from U.S. Department of Agriculture, Agricultural Research Service (2019)*

Nutrition	**Eel (fresh)**	**Cod (fresh)**	**Cold (dry and salted)**	**Herring (fresh)**	**Herring (brined)**
Calories (kcal)	184	82	290	158	262
Protein (g)	18.4	17.8	62.8	18	14.2
Calcium (mg)	20	16	160	57	77
Iron (mg)	0.5	0.38	2.5	1.1	1.22
Magnesium (mg)	20	32	133	32	8
Vitamin C (mg)	1.8	1	3.5	0.7	0
Folate (mcg)	15	7	25	10	2
Vitamin B12 (mcg)	3	0.91	10	13.7	4.27
Vitamin A (IU)	3480	40	140	93	860
Vitamin D (D2+D3) (IU)	932	36	161	167	113
Vitamin K (mcg)	0	0.1	0.4	0.1	0.2

C Additional figures and tables

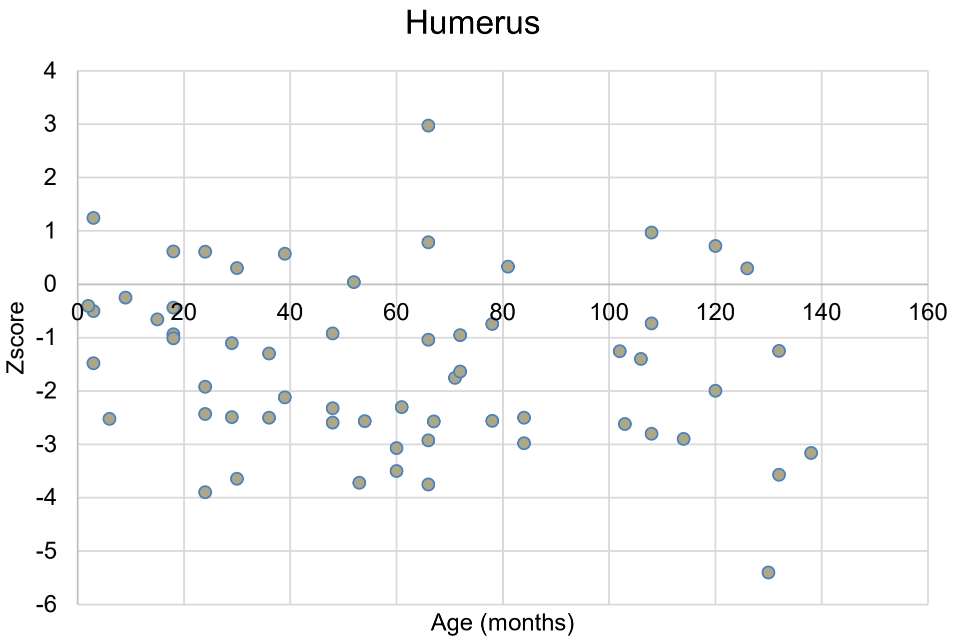
In this section, supplementary figures were listed. These figures expand the analysis of diaphyseal length analysis for non-adults, as well as correlations between VNC dimensions and spina bifida and menarche timing.

Finally, the data recording sheets for perinates, non-adults and adults were listed.

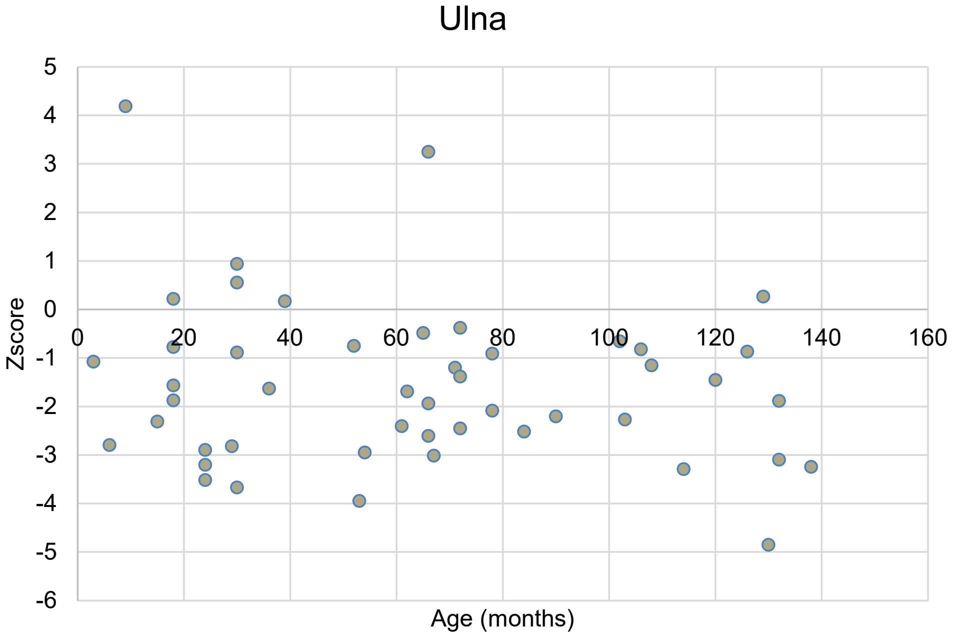
C.1 Diaphyseal - Non-adults (Z-score figures)

C.1.1 Z-score: Comparison between the pre-, post-, and Black Death periods

Figures C.1- C.12 show the distribution of Z-scores for individuals dated to the pre-Black Death period for the extra-London and individuals dated to the pre-, post- and Black Death periods in the London region. The sample of non-adults dated to the post-Black Death period did not present any data. The following figures (C.13- C.22) exhibit the distribution of z-scores for individuals dates to the early and late pre-Black Death for the extra-London and London regions. Due to the small sample, the London region only presented data for the ulna during the early pre-Black Death subperiod.

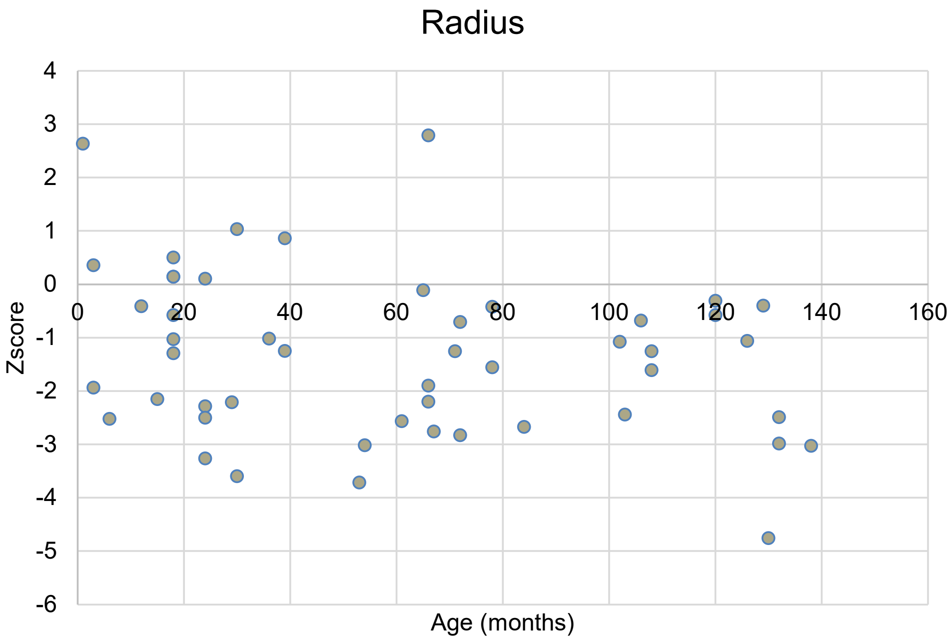


(a) Humerus

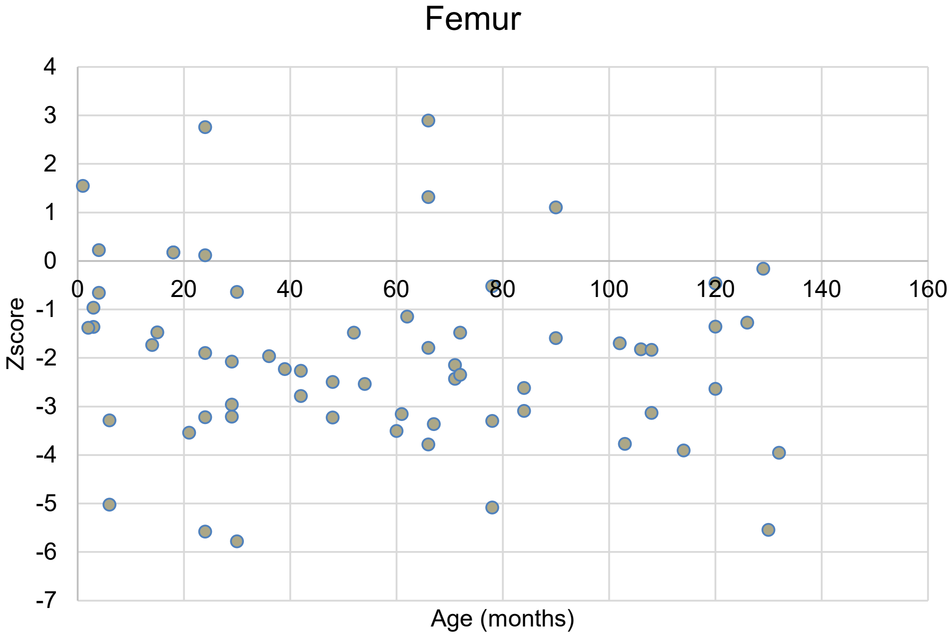


(b) Ulna

Figure C.1: *Z-scores for diaphyseal length for the humerus (a.) and ulna (b.) of individuals from the extra-London region dated to the pre-Black Death period.*

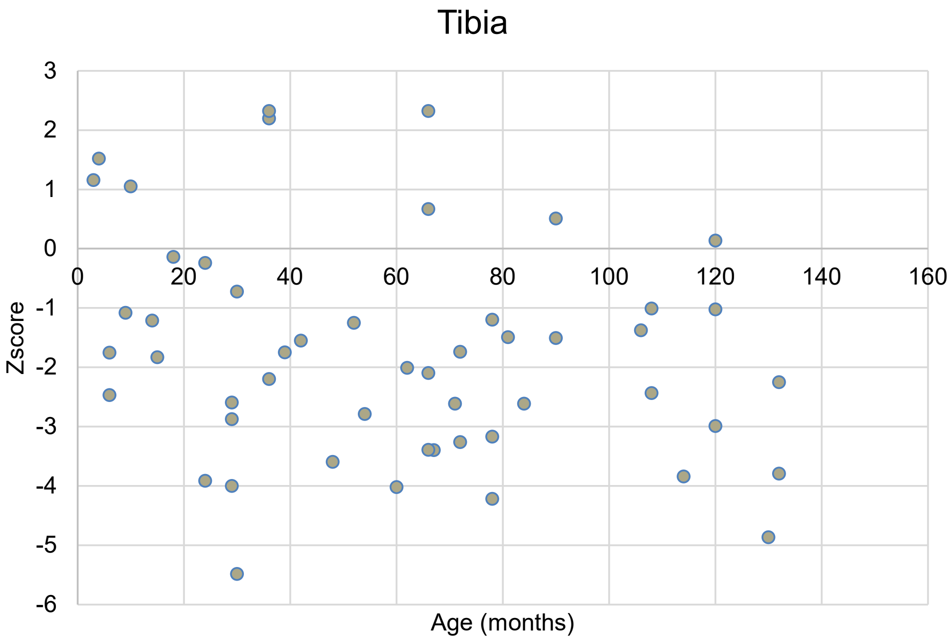


(a) Radius

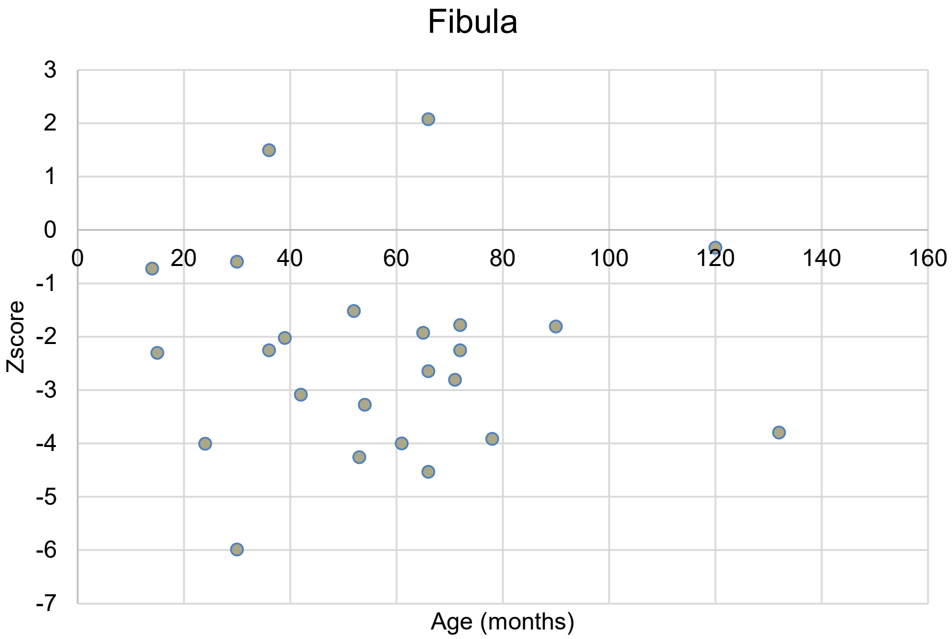


(b) Femur

Figure C.2: *Z-scores for diaphyseal length for the radius (a.) and femur (b.) of individuals from the extra-London region dated to the pre-Black Death period.*

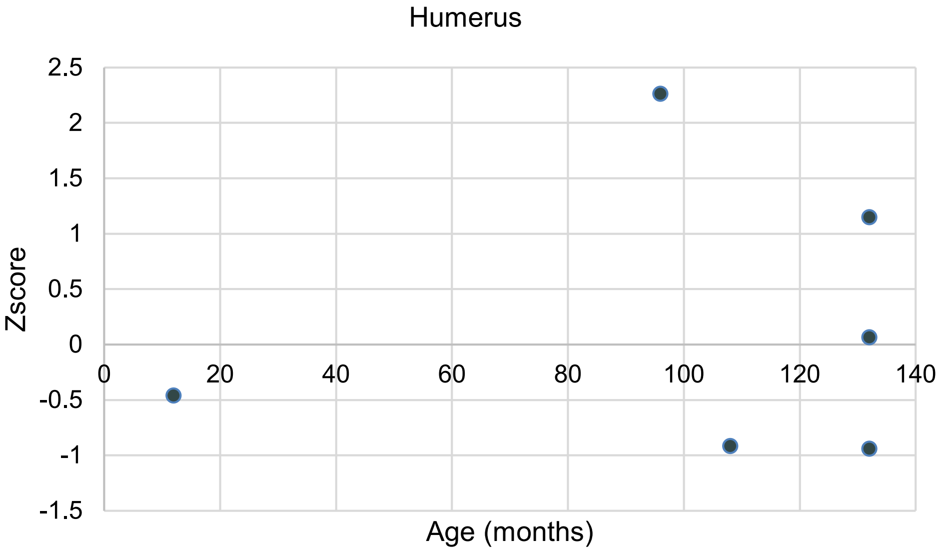


(a) *Tibia*

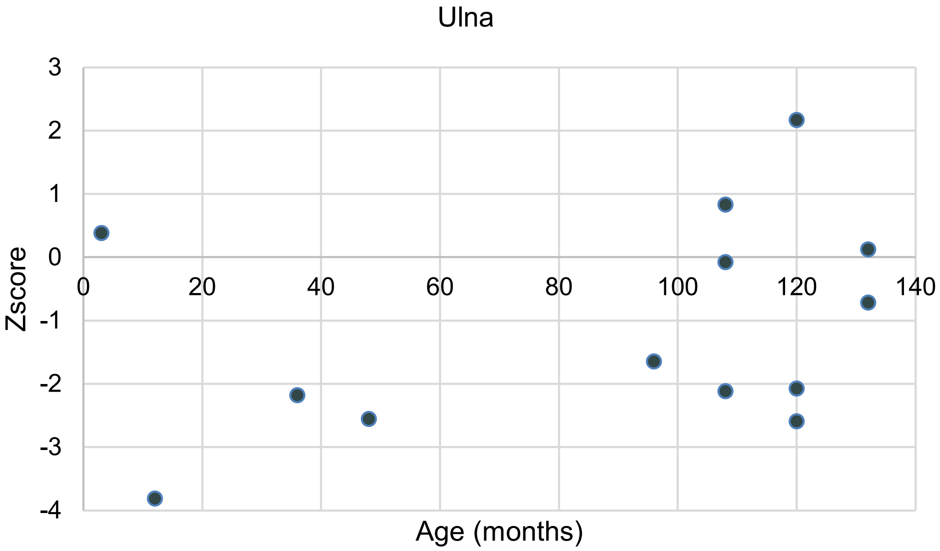


(b) *Fibula*

Figure C.3: *Z-scores for diaphyseal length for the tibia (a.) and fibula (b.) of individuals from the extra-London region dated to the pre-Black Death period.*

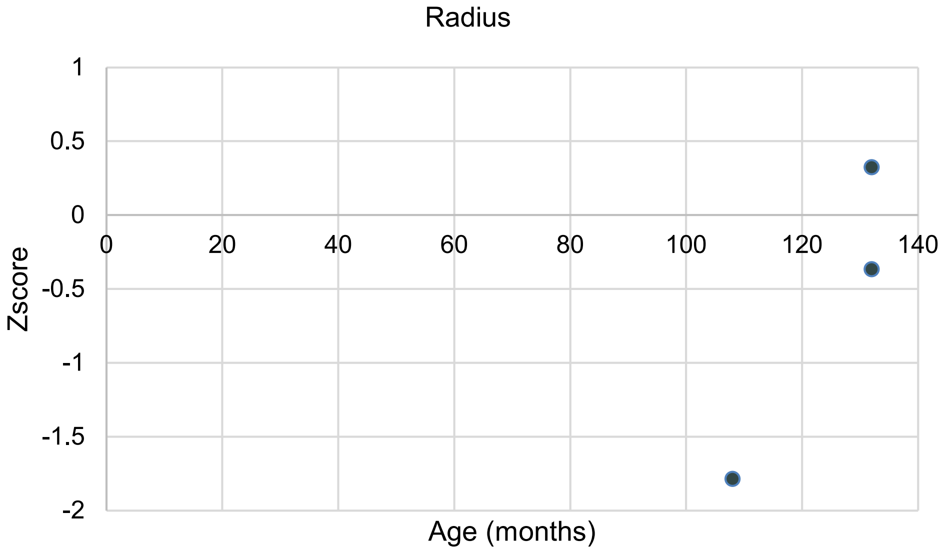


(a) *Humerus*

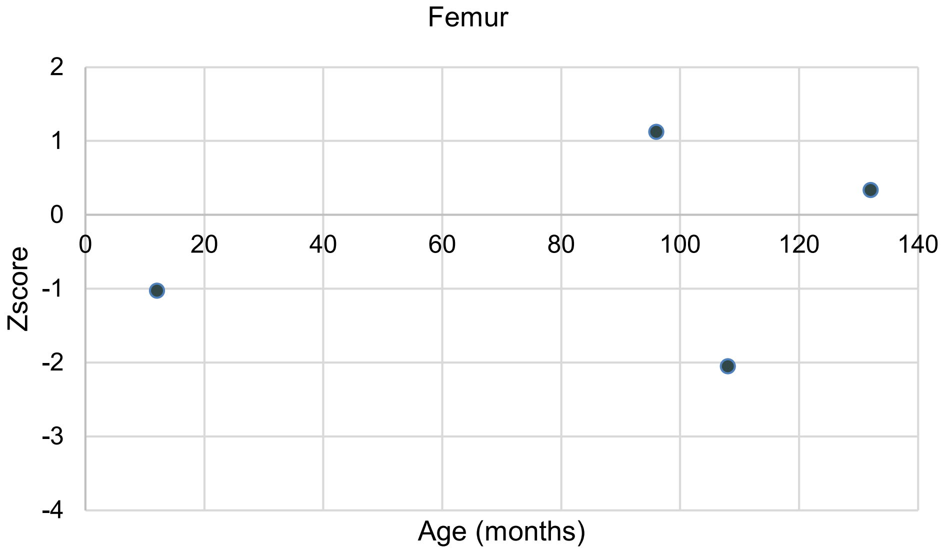


(b) *Ulna*

Figure C.4: *Z-scores for diaphyseal length for the humerus (a.) and ulna (b.) of individuals from the London region dated to the pre-Black Death period.*

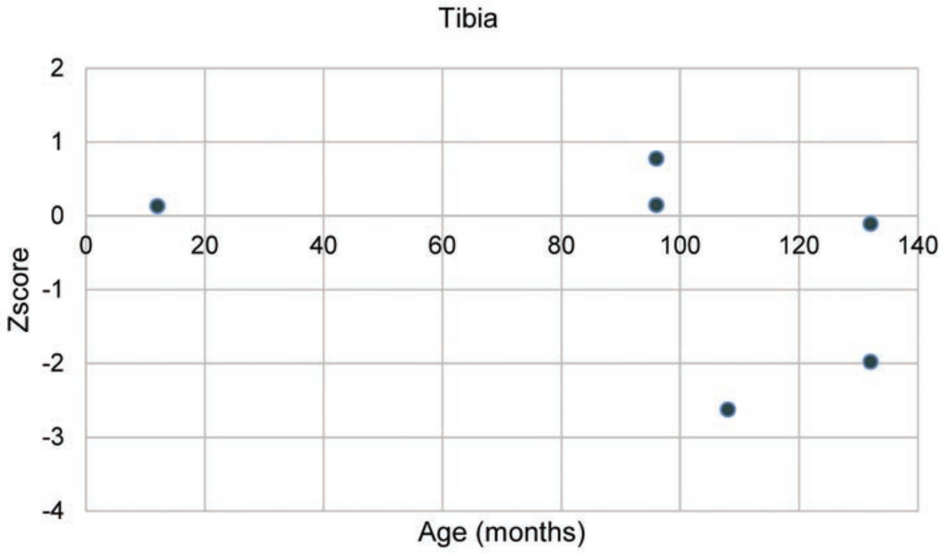


(a) *Radius*

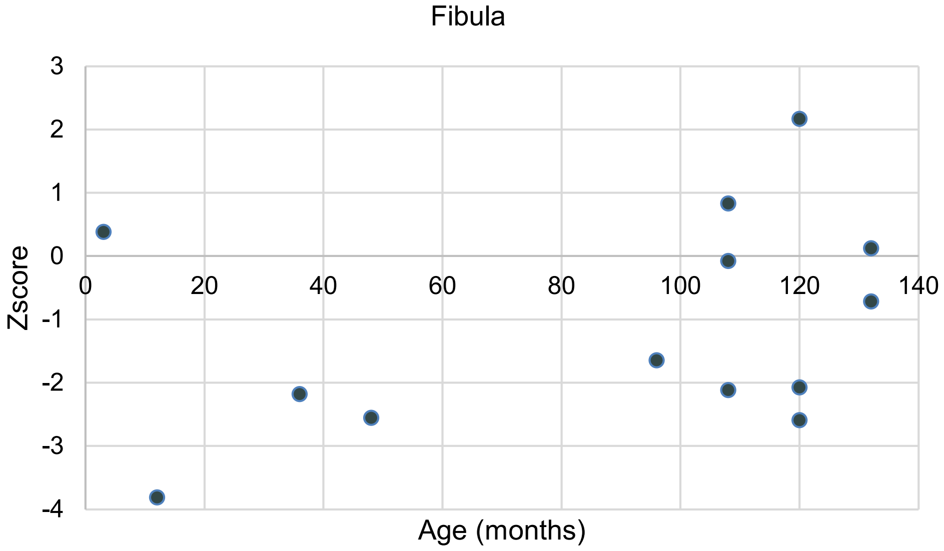


(b) *Femur*

Figure C.5: *Z-scores for diaphyseal length for the radius (a.) and femur (b.) of individuals from the London region dated to the pre-Black Death period.*

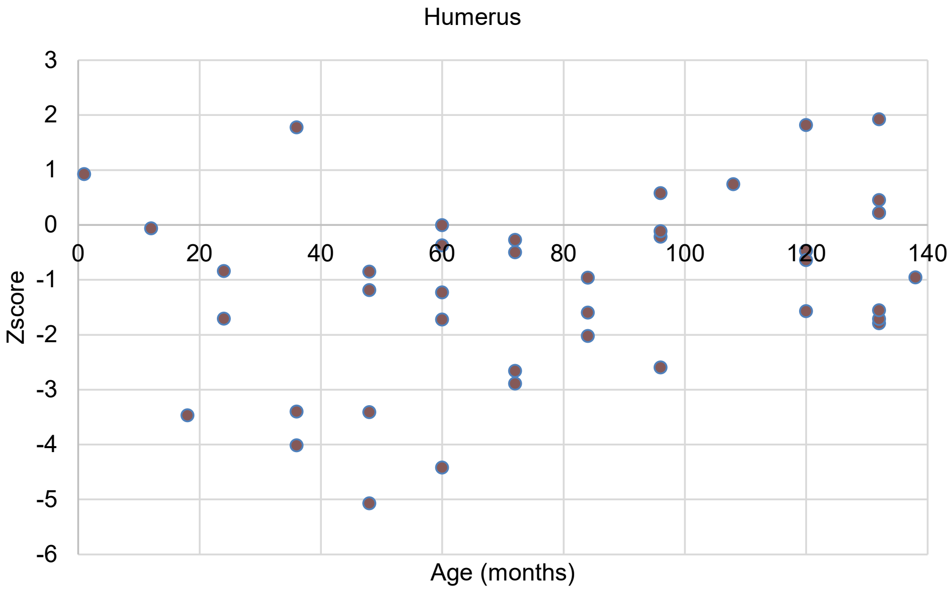


(a) Tibia

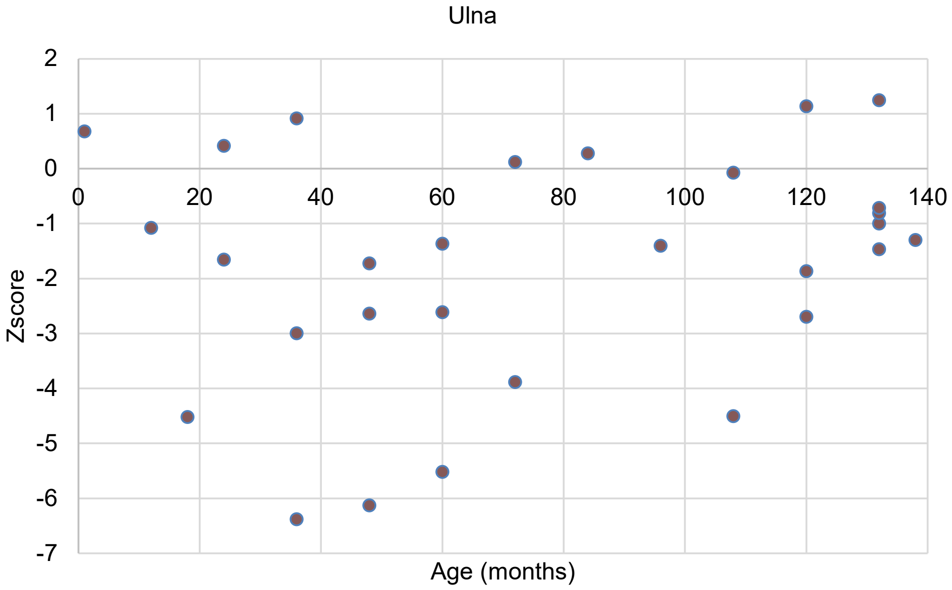


(b) fibula

Figure C.6: *Z-scores for diaphyseal length for the tibia (a.) and fibula (b.) of individuals from the London region dated to the pre-Black Death period.*

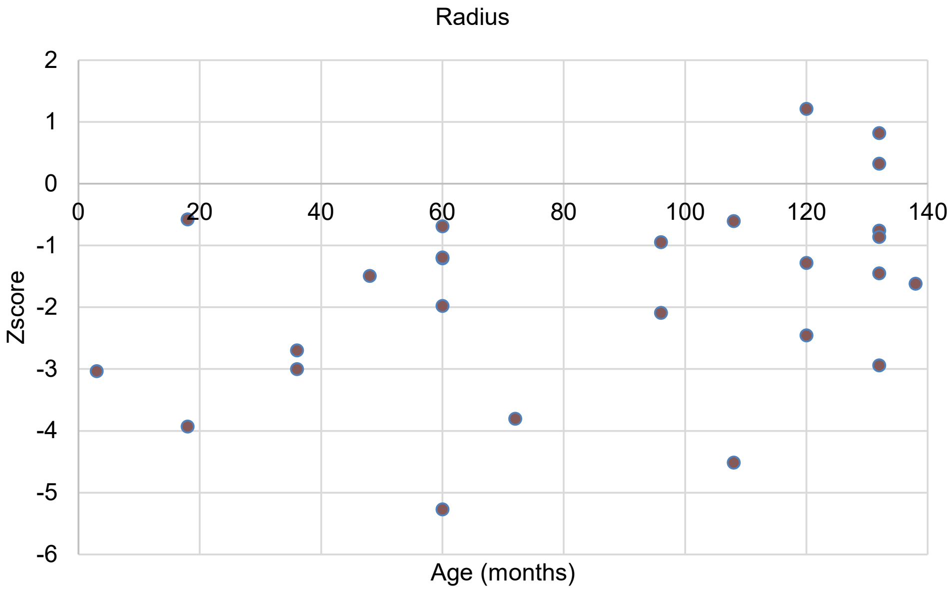


(a) Humerus

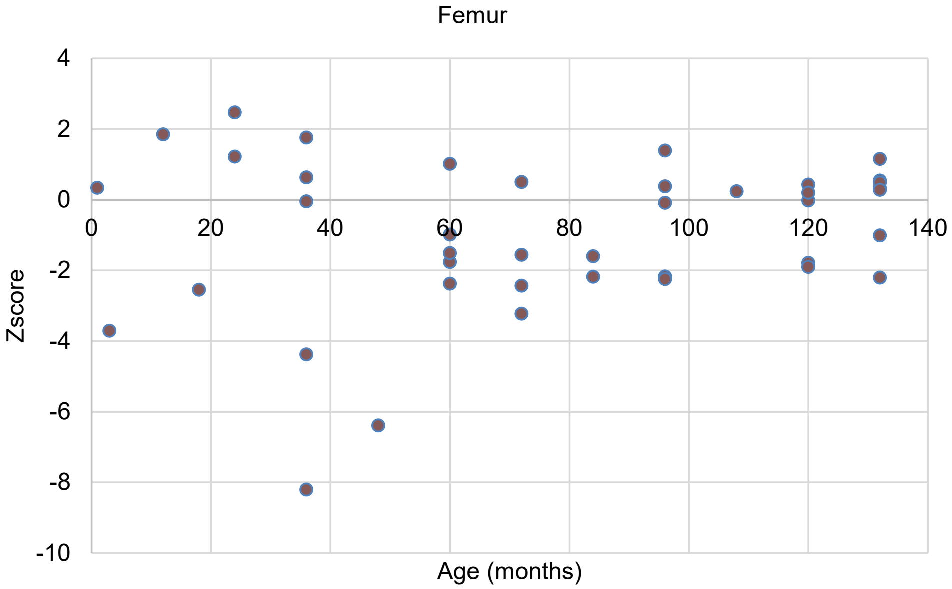


(b) Ulna

Figure C.7: *Z-scores for diaphyseal length for the humerus (a.) and ulna (b.) of individuals from the London region dated to the Black Death period.*

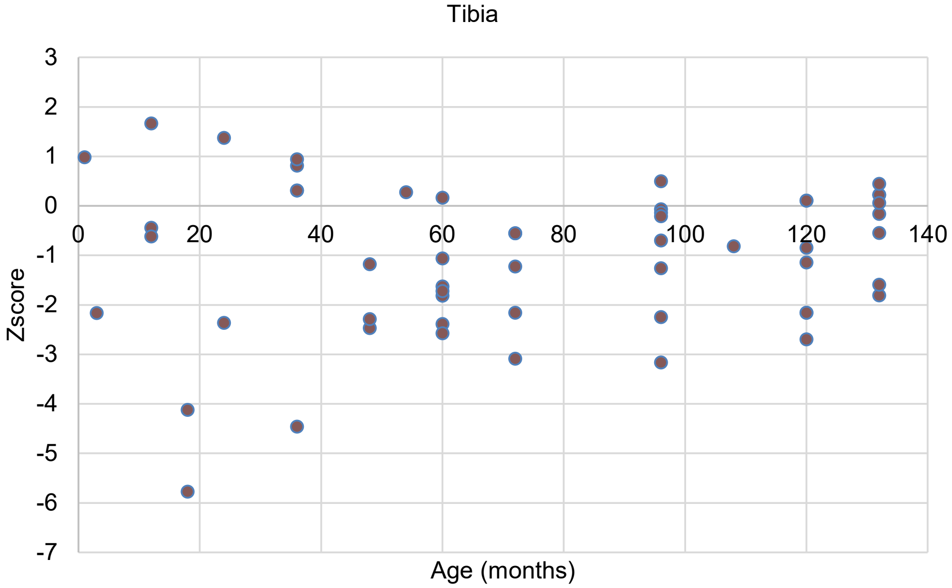


(a) Radius

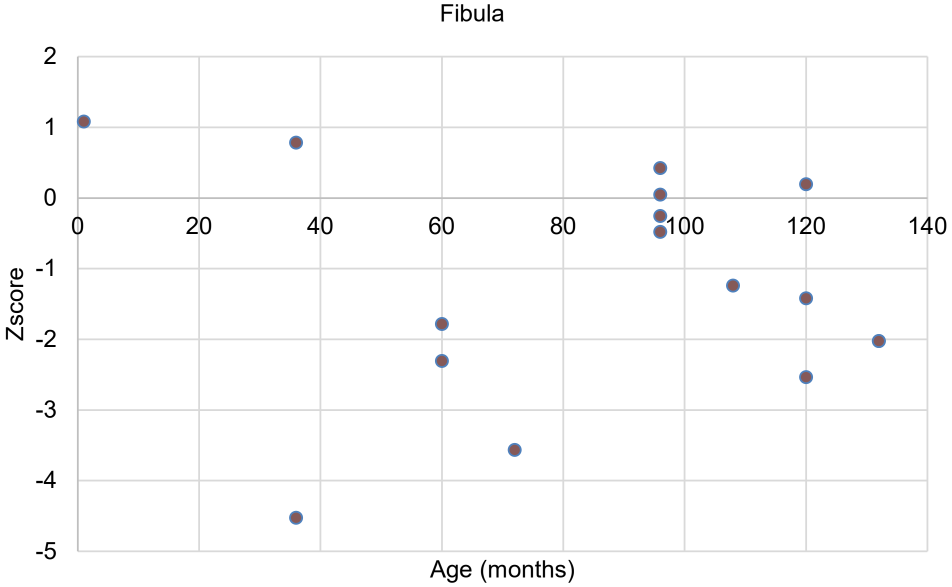


(b) Femur

Figure C.8: *Z-scores for diaphyseal length for the radius (a.) and femur (b.) of individuals from the London region dated to the Black Death period.*

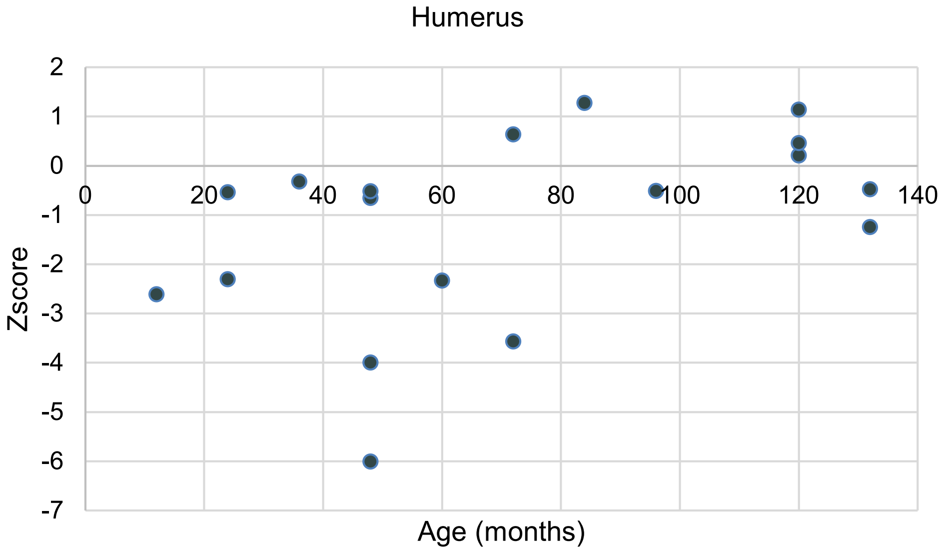


(a) *Tibia*

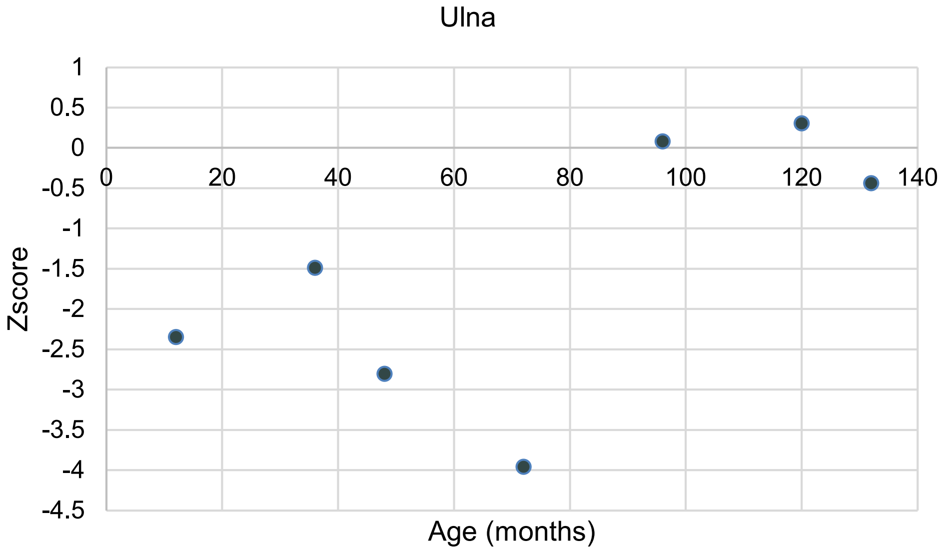


(b) *Fibula*

Figure C.9: *Z-scores for diaphyseal length for the tibia (a.) and fibula (b.) of individuals from the London region dated to the Black Death period.*

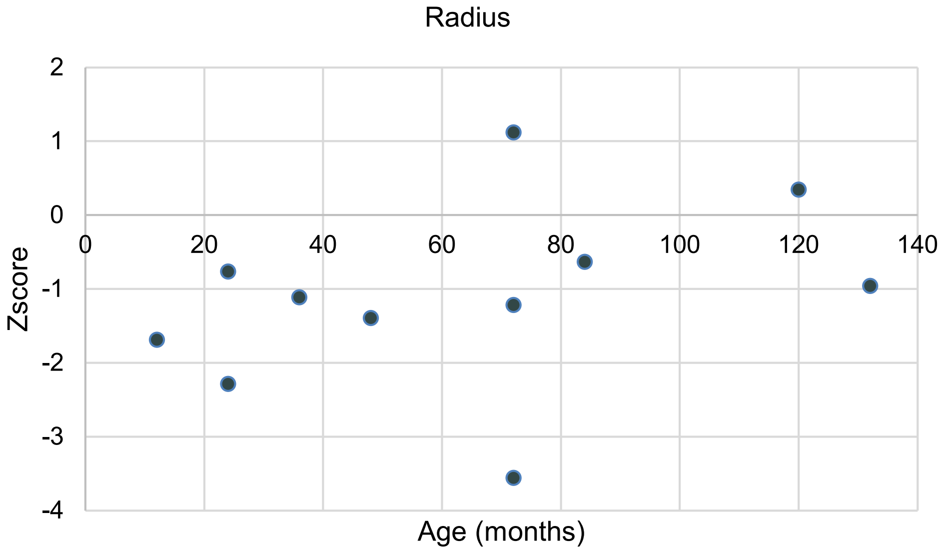


(a) *Humerus*

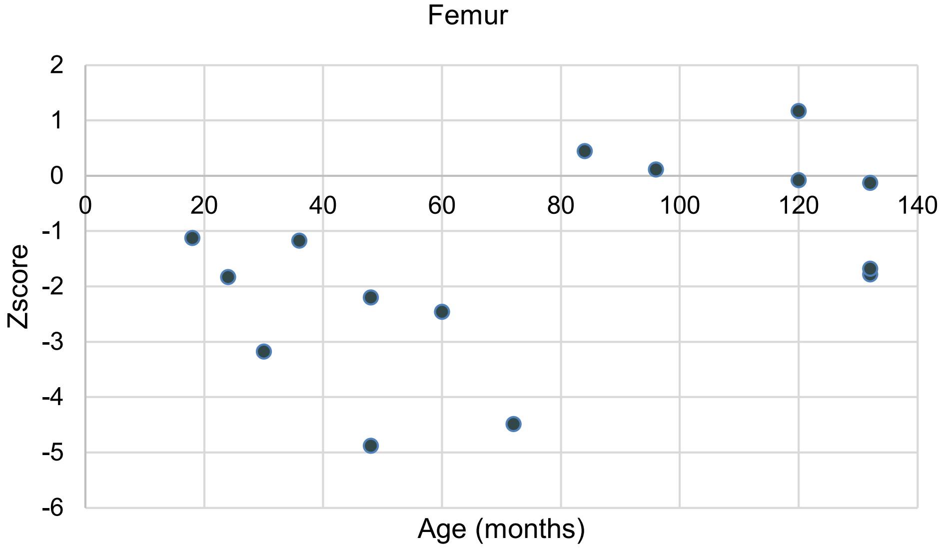


(b) *Ulna*

Figure C.10: *Z-scores for diaphyseal length for the humerus (a.) and ulna (b.) of individuals from the London region dated to the post-Black Death period.*

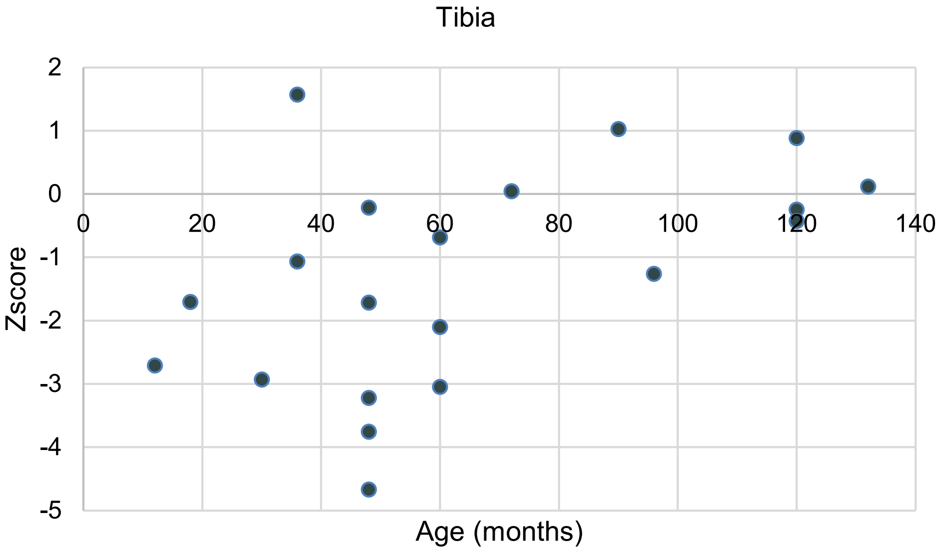


(a) *Radius*

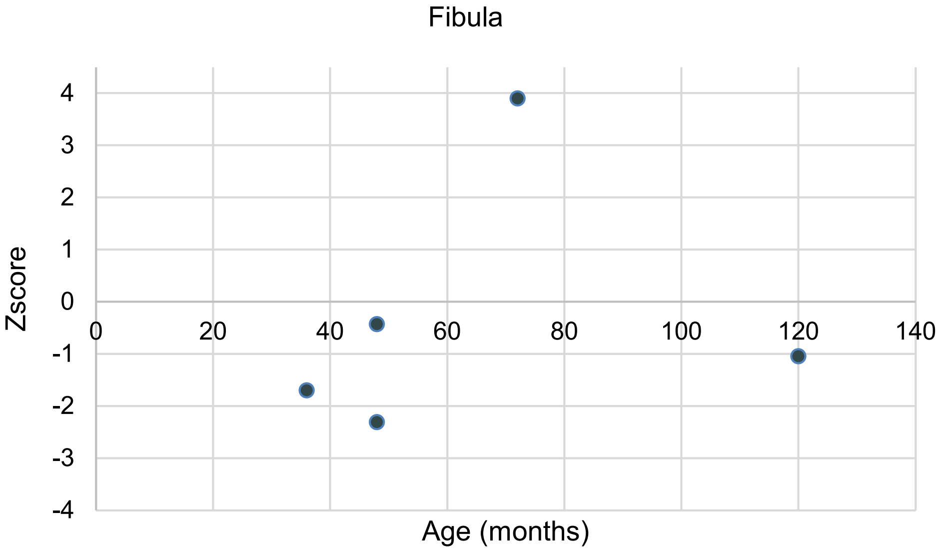


(b) *Femur*

Figure C.11: *Z-scores for diaphyseal length for the radius (a.) and femur (b.) of individuals from the London region dated to the post-Black Death period.*



(a) *Tibia*

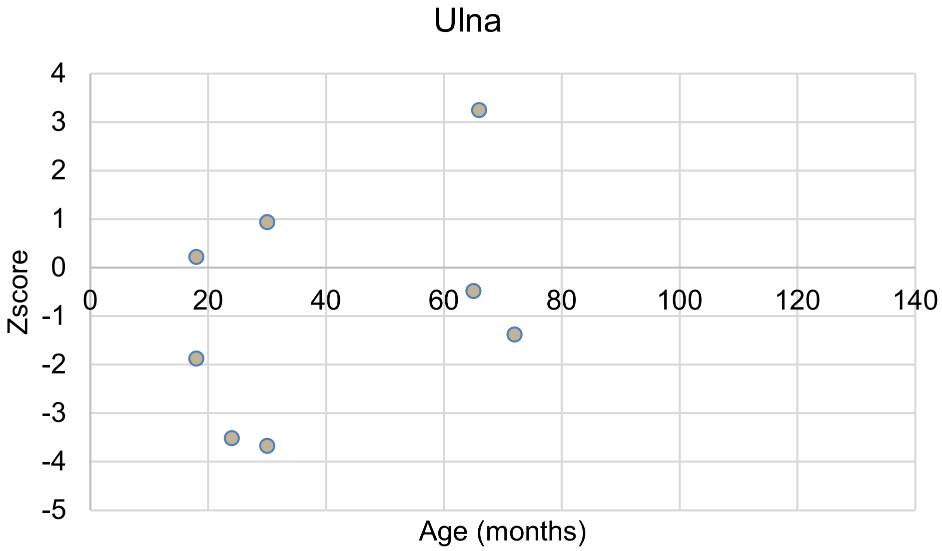


(b) *Fibula*

Figure C.12: *Z-scores for diaphyseal length for the tibia (a.) and fibula (b.) of individuals from the London region dated to the post-Black Death period.*

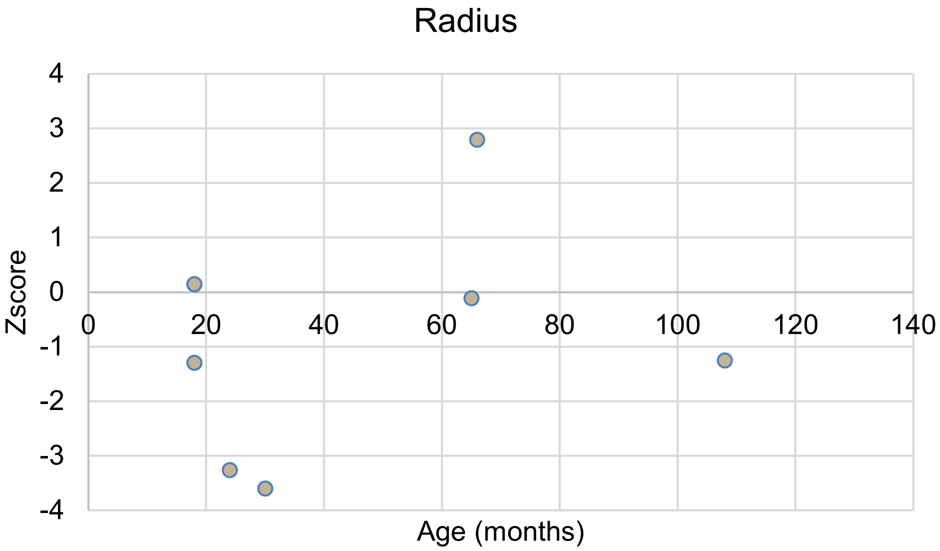
C.1.2 Z-score: The pre-Black Death period - comparison between the early and late subperiods

(a) Humerus

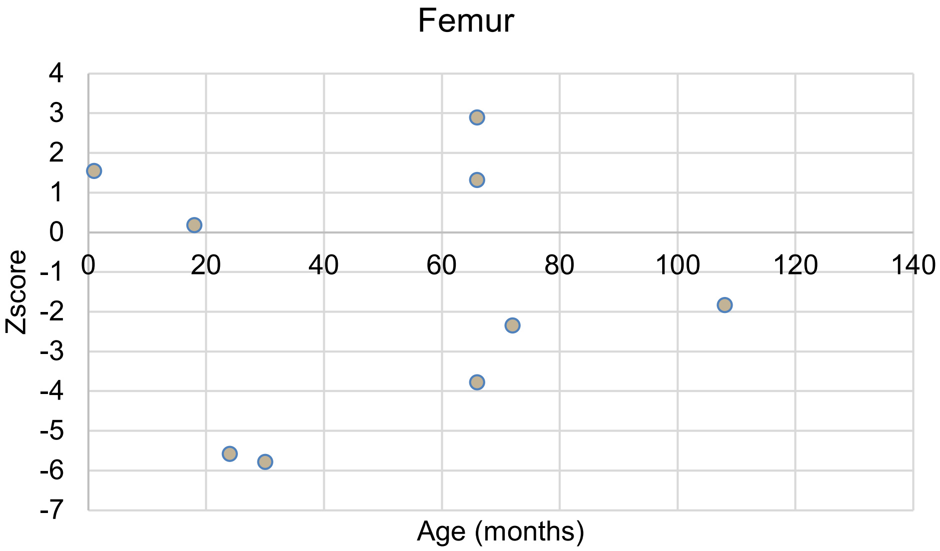


(b) Ulna

Figure C.13: *Z-scores for diaphyseal length for the humerus (a.) and ulna (b.) of individuals from the extra-London region dated to the early pre-Black Death subperiod.*

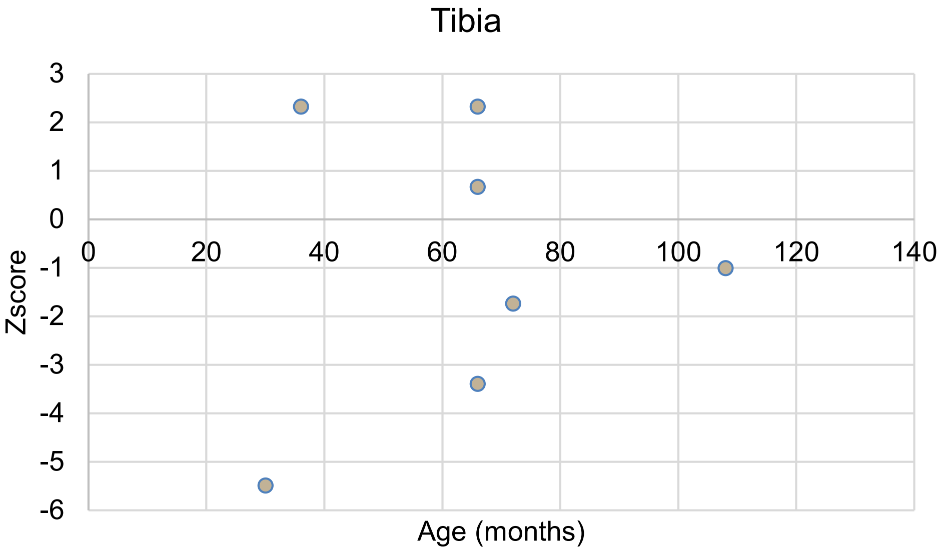


(a) *Radius*

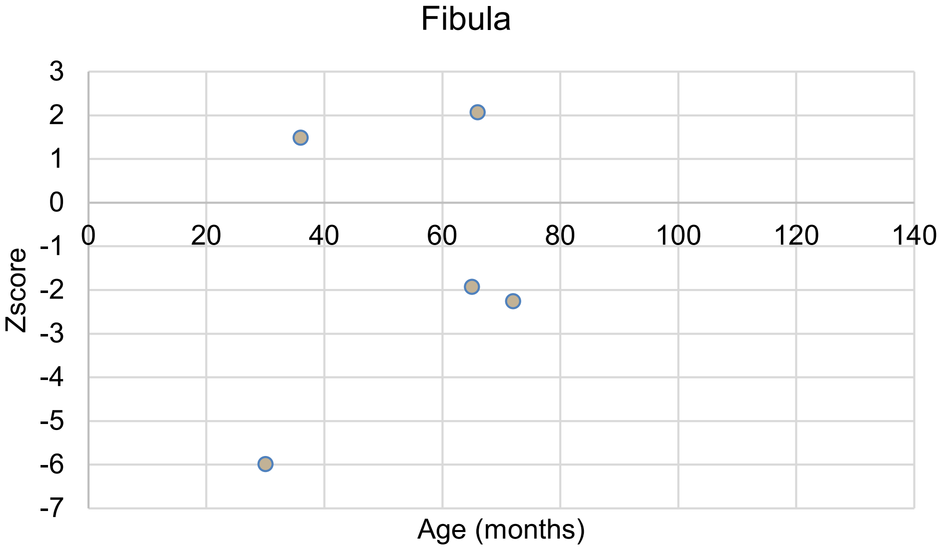


(b) *Femur*

Figure C.14: *Z-scores for diaphyseal length for the radius (a.) and femur (b.) of individuals from the extra-London region dated to the early pre-Black Death subperiod.*

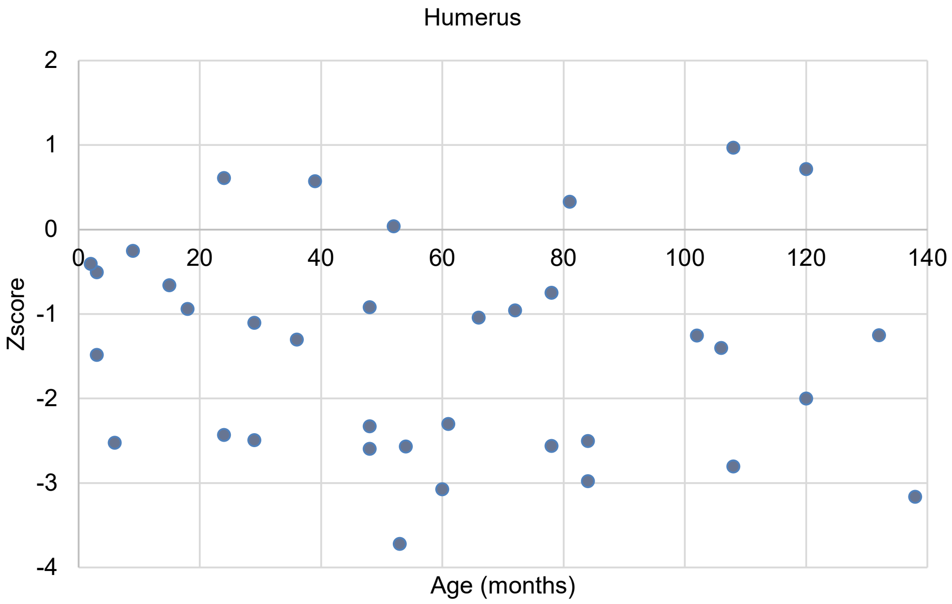


(a) Tibia

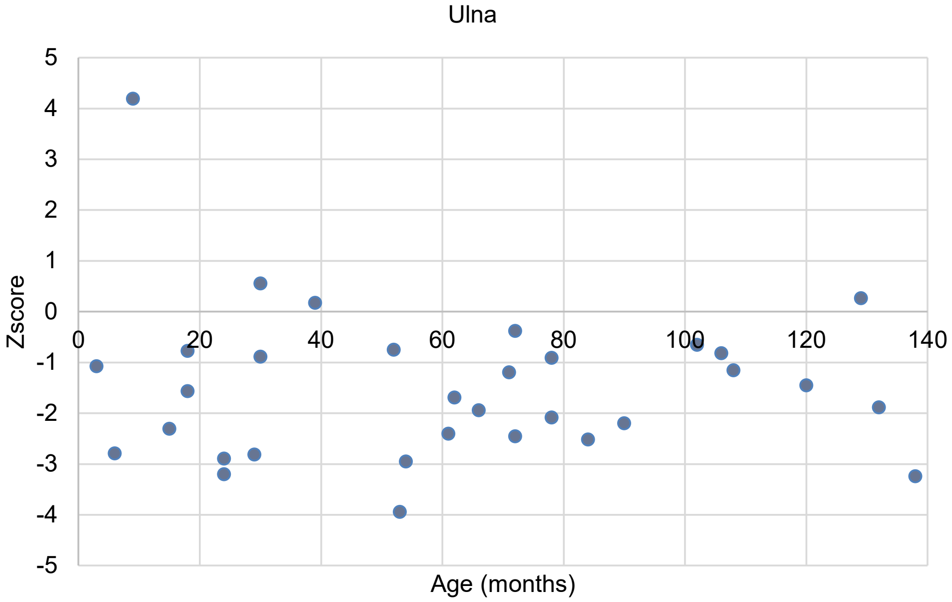


(b) Fibula

Figure C.15: *Z-scores for diaphyseal length for the tibia (a.) and fibula (b.) of individuals from the extra-London region dated to the early pre-Black Death subperiod.*

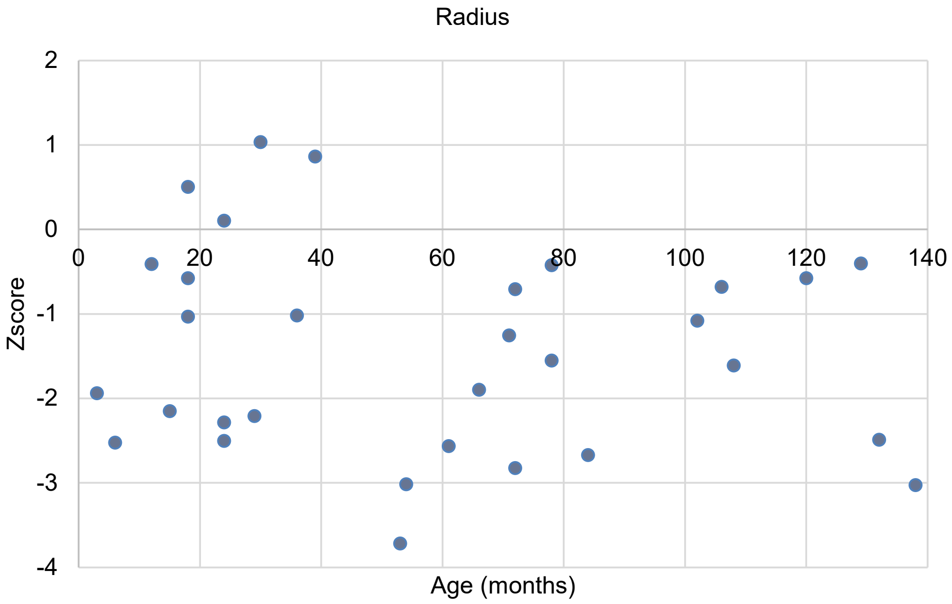


(a) Humerus

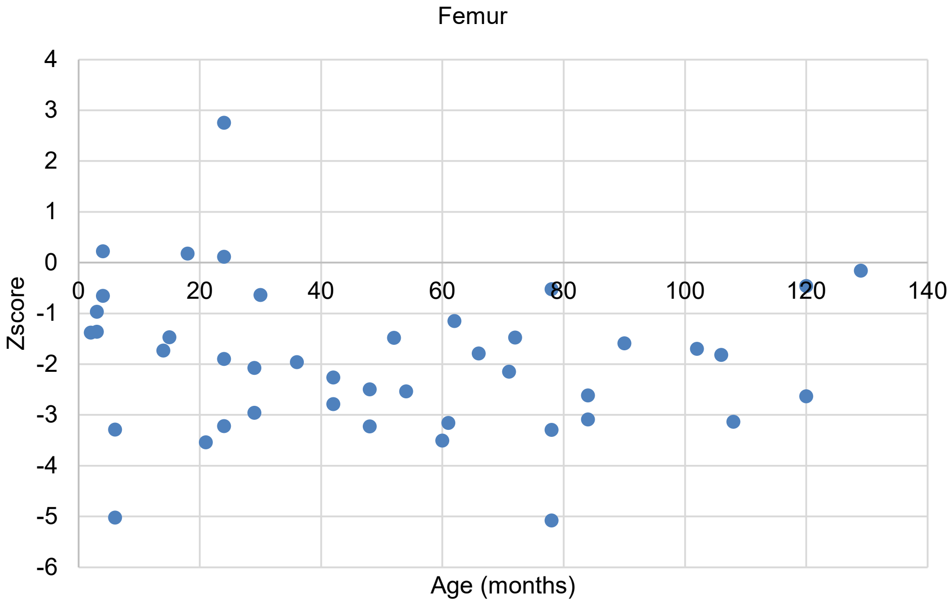


(b) Ulna

Figure C.16: *Z-scores for diaphyseal length for the humerus (a.) ulna (b.) of individuals from the extra-London region dated to the late pre-Black Death subperiod.*

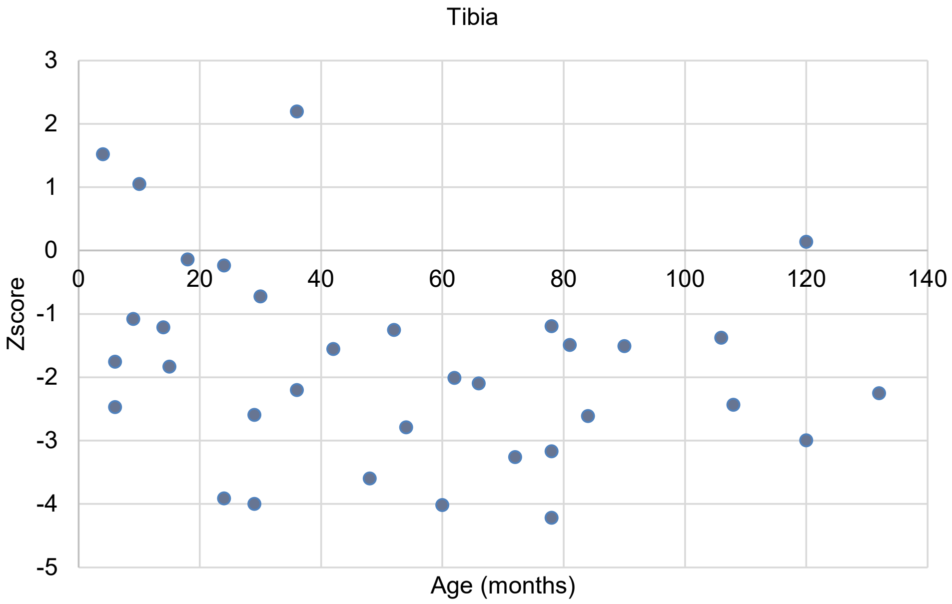


(a) Radius

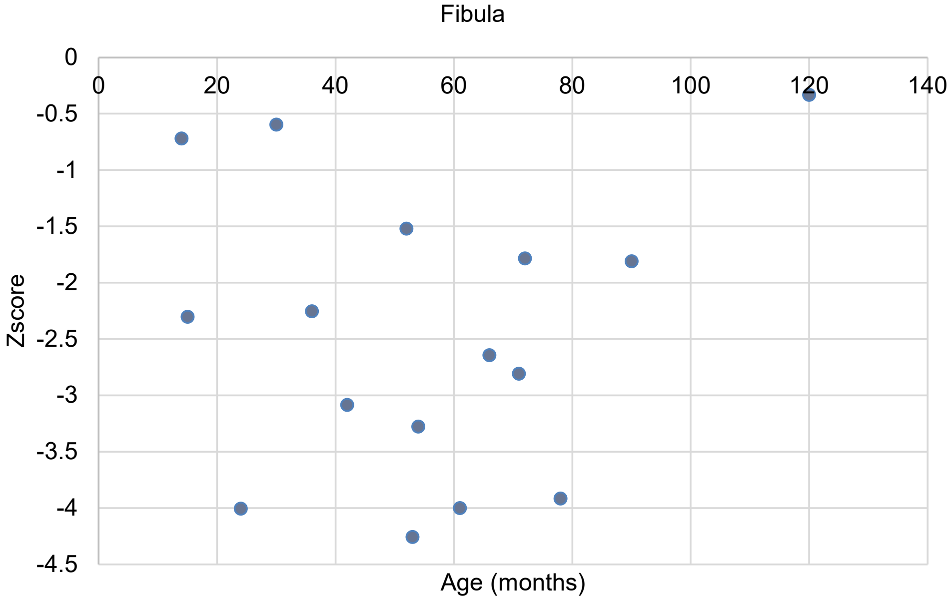


(b) Femur

Figure C.17: *Z-scores for diaphyseal length for the radius (a.) and femur (b.) of individuals from the extra-London region dated to the late pre-Black Death subperiod.*

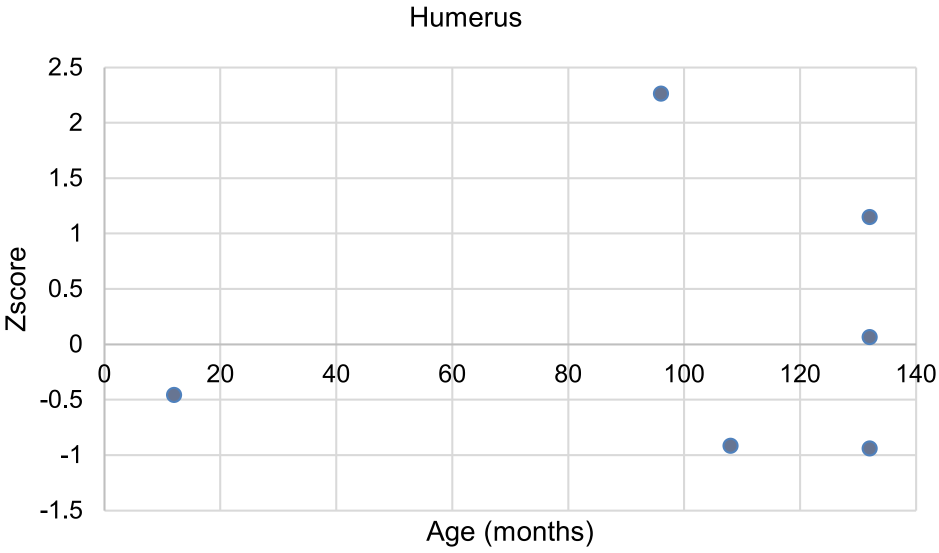


(a) Tibia

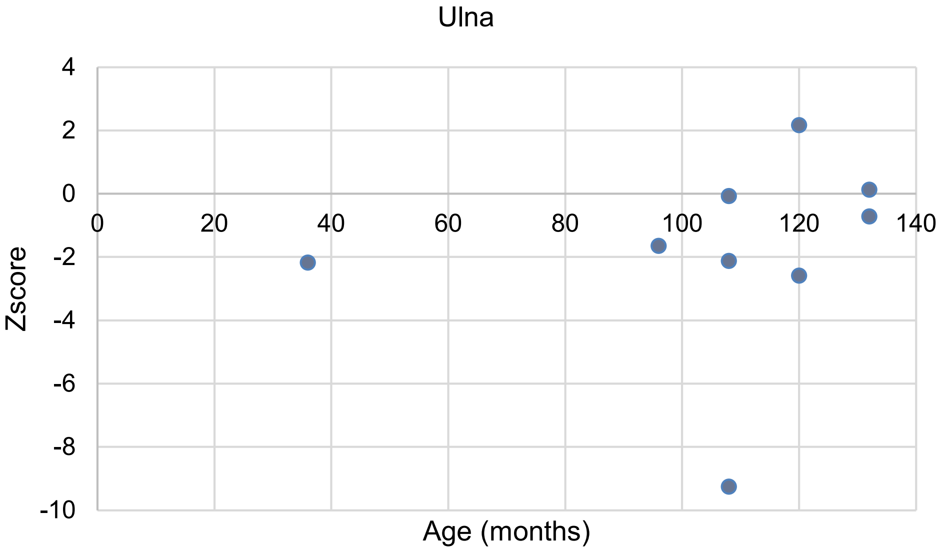


(b) Fibula

Figure C.18: *Z-scores for diaphyseal length for the tibia (a.) and fibula (b.) of individuals from the extra-London region dated to the late pre-Black Death subperiod.*

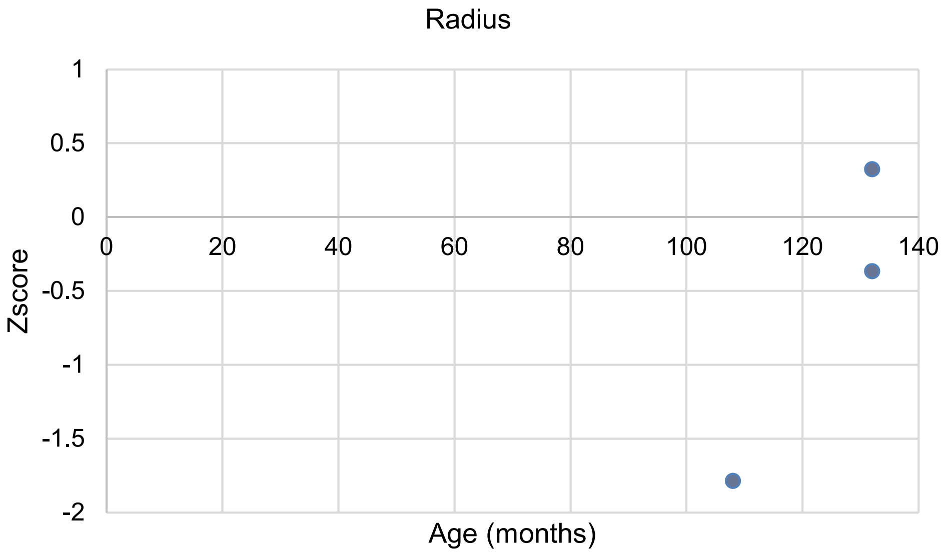


(a) *Humerus*

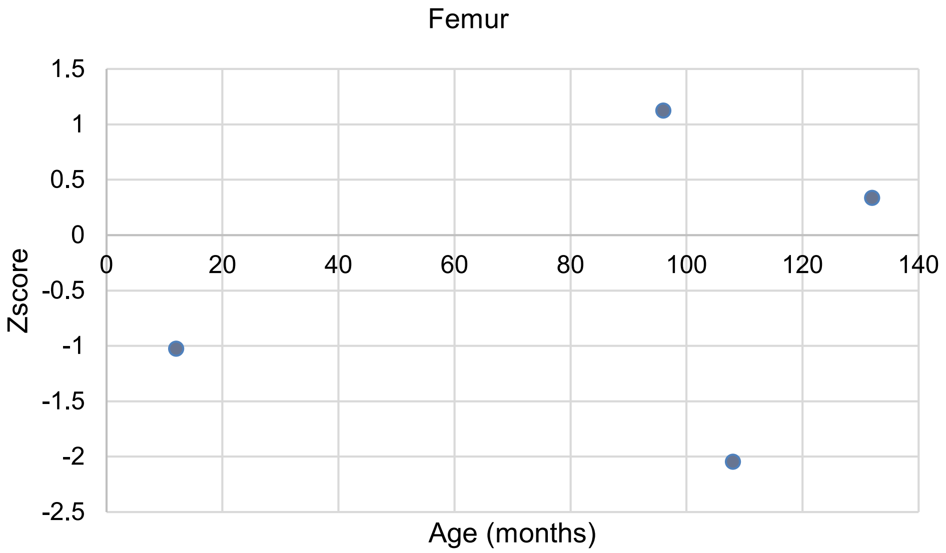


(b) *Ulna*

Figure C.19: *Z-scores for diaphyseal length for the humerus (a.) and the ulna (b.) of individuals from the London during the late pre-Black Death subperiod.*



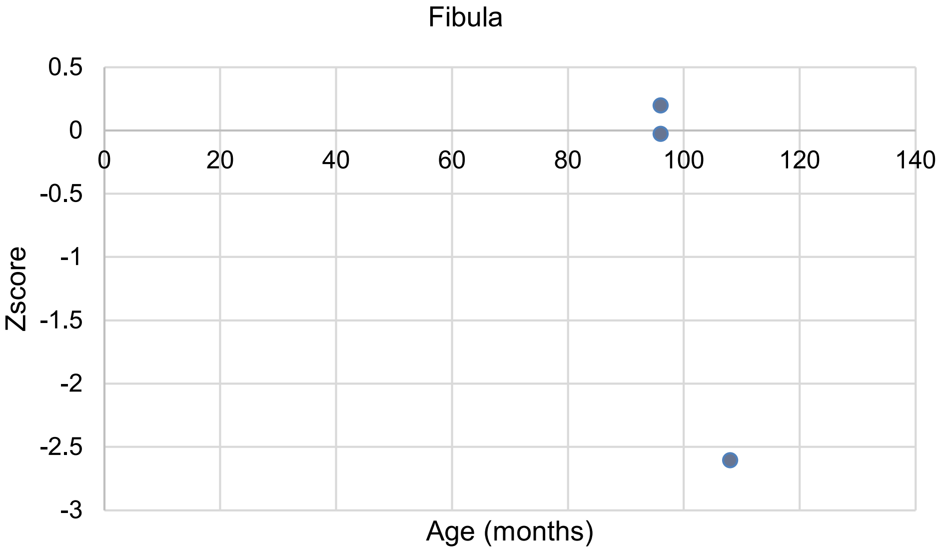
(a) Radius



(b) Femur

Figure C.20: *Z-scores for diaphyseal length for the radius (a.) and the femur (b.) of individuals from the London during the late pre-Black Death subperiod.*

(a) *Tibia*



(b) *Fibula*

Figure C.21: *Z-scores for diaphyseal length for the tibia (a.) and the fibula (b.) of individuals from the London during the late pre-Black Death subperiod.*

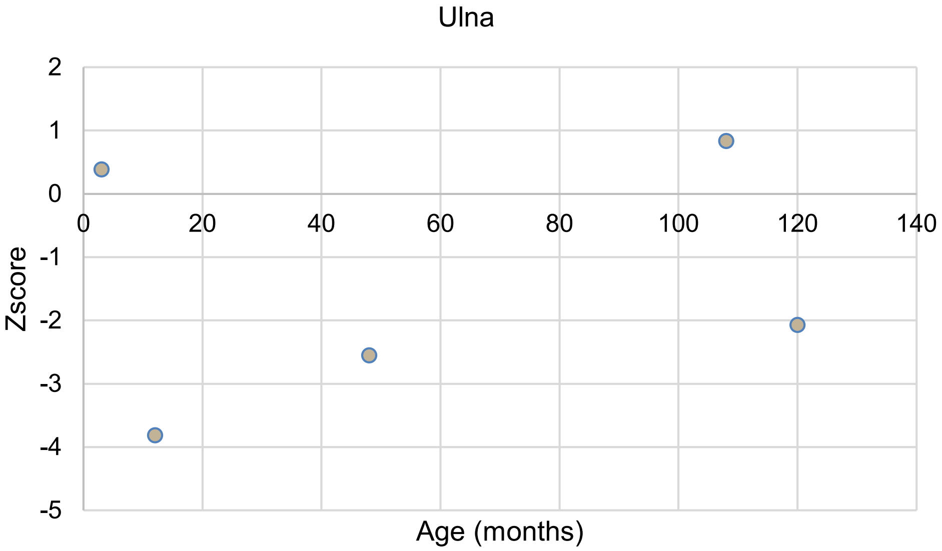


Figure C.22: *Z-scores for diaphyseal length for the ulna of individuals from the London during the early pre-Black Death subperiod.*

C.2 Correlations

C.2.1 Correlations between spina bifida occulta and VNC dimensions

The following table (C.1) shows the correlation between presence of spina bifida and vertebral neural canal dimensions in the pre-Black Death period. In order to be able to do this correlation, only data from the primary analysis was available, which allowed for true prevalences to be estimated. Results showed no statistically significant differences in VNC dimensions between people who had evidence of spina bifida occulta and people who didn't have evidence of the condition.

Table C.1: *Correlation between spina bifida presence and VNC dimensions*

Lumbar vertebrae	Spina bifida	N	Mean	Std. Deviation	Std. Error Mean
L1AP[1]	**Present**	6	16.74	1.43	0.58
	Absent	39	16.61	1.37	0.22
L1TR	**Present**	7	21.57	1.31	0.50
	Absent	41	21.82	1.94	0.30
L2AP	**Present**	9	15.86	1.82	0.61
	Absent	39	15.61	1.86	0.30
L2TR	**Present**	9	21.99	2.96	0.99
	Absent	41	21.79	1.88	0.29
L3AP	**Present**	8	14.59	1.74	0.62
	Absent	42	14.72	1.63	0.25
L3TR	**Present**	8	21.52	1.18	0.42
	Absent	44	21.83	1.50	0.23
L4AP	**Present**	9	15.64	1.91	0.64
	Absent	40	14.71	1.92	0.30
L4TR	**Present**	9	22.26	1.71	0.57
	Absent	45	22.42	2.25	0.33
L5AP	**Present**	7	16.88	0.95	0.36
	Absent	40	15.96	2.60	0.41
L5TR	**Present**	7	25.22	0.64	0.24
	Absent	44	24.71	2.61	0.39

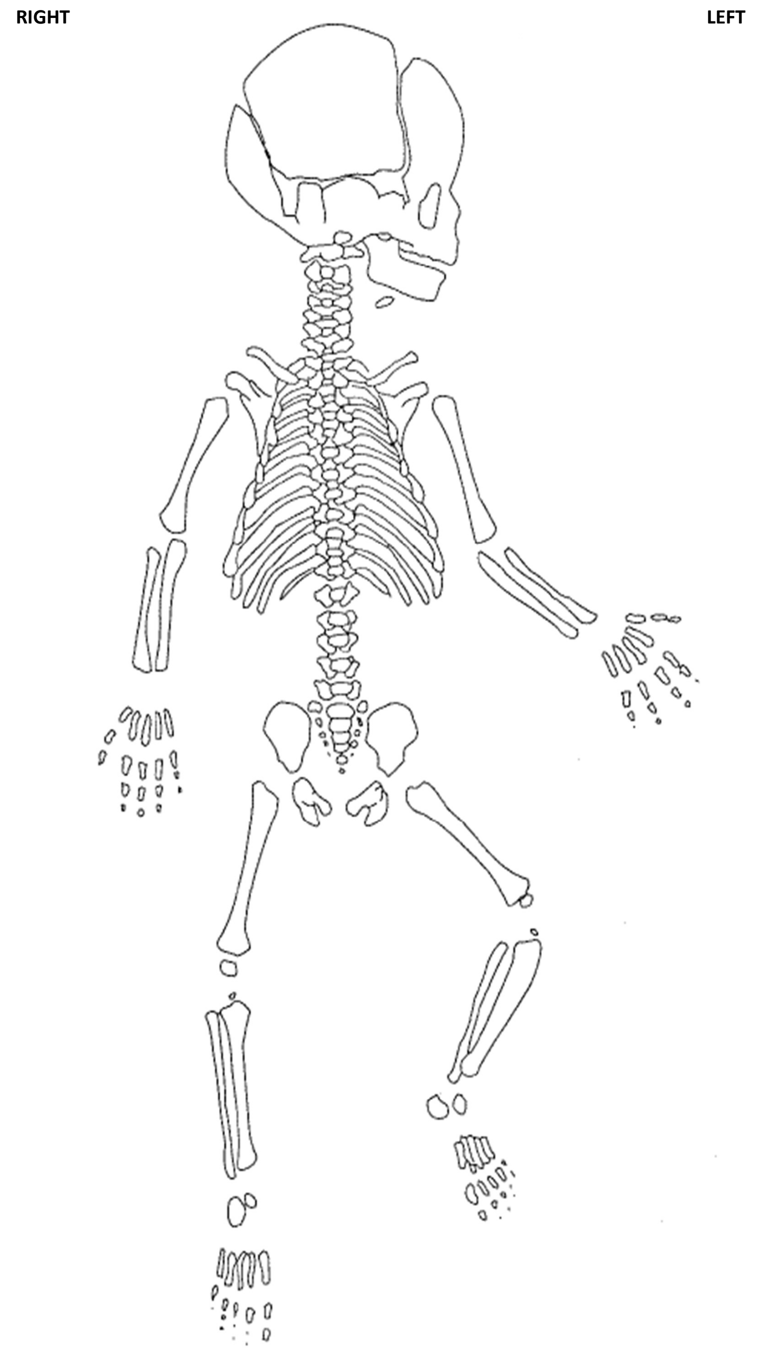
[1] L = lumbar vertebrea; AP = Antero-posterior diameter; TR = Transversal diameter

D Data recording sheets

The following figures show the data recording sheets for the analysis of primary evidence in this thesis. Recording sheets were divided in perinates, non-adults and adults to differentiate age and sex estimation, epiphyseal fusion, dentition and identification of different pathological conditions.

D.1 Perinates

Site: Inv Number: Date: / /20



1

AGE ESTIMATION:

Odontometrics			
Morphological observation of teeth			
Osteometrics	**Measurement (1)**	**Measurement (2)**	**Average**
Scapula			
Radius			
Fibula			

PATHOLOGIES

CRIBRA ORBITALIA		Spina bifida	
Presence		Presence	[Not observable]
Severity (0-5)		Type	
Healed?			
OTHER PATHOLOGIES:			
Comments:			

LONG BONES (Diaphyses)

Bone	**Length (mm) (1)**	**Length (mm) (2)**	**Average**
Femur (left)			
Femur (right)			
Tibia (left)			
Tibia (right)			
Radius (left)			
Radius (right)			
Humerus (left)			
Humerus (right)			
Bone	**Width (mm) (1)**	**Width (mm) (2)**	**Average**
Femur (left)			
Femur (right)			
Tibia (left)			
Tibia (right)			
Radius (left)			
Radius (right)			
Humerus (left)			
Humerus (right)			

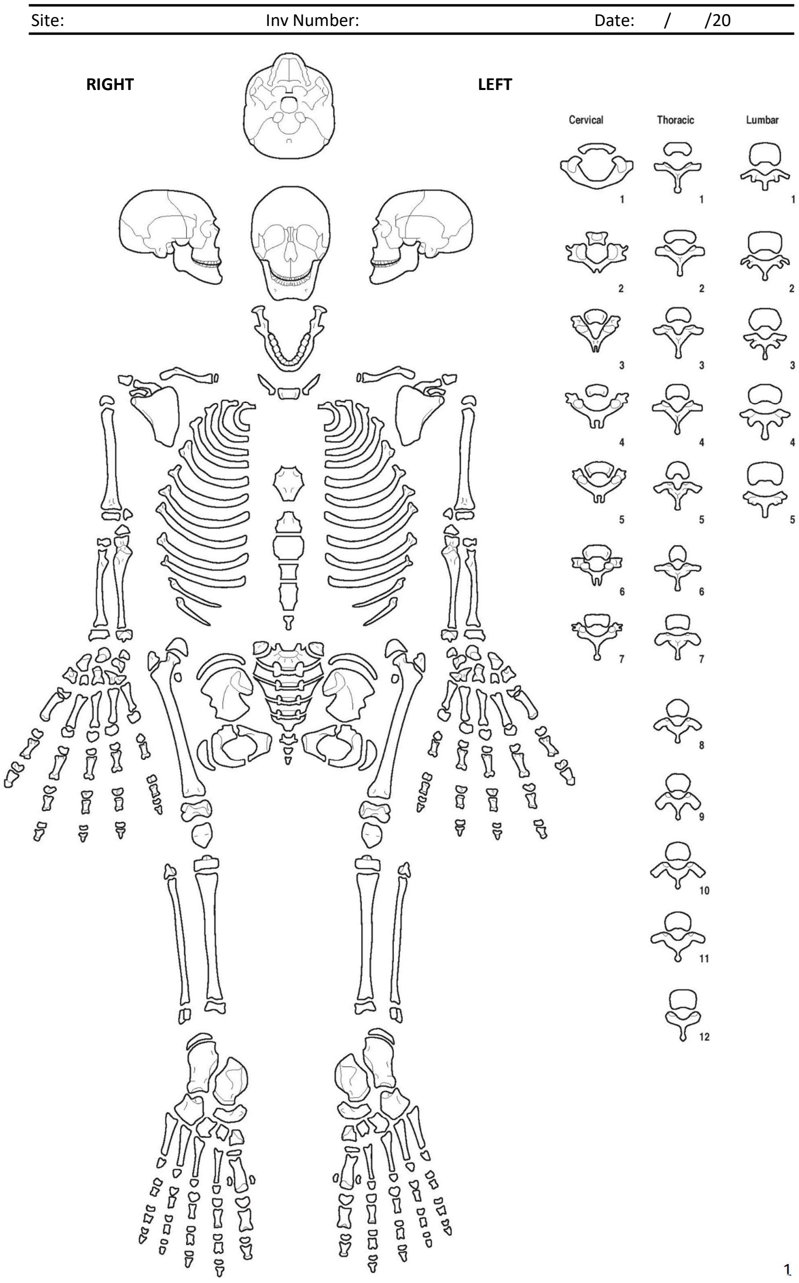
Site: Inv Number: Date: / /20

DENTAL ANALYSIS

	M2	M1	C	P1	P2	C	I2	I1
Height (mm)								
Comments								

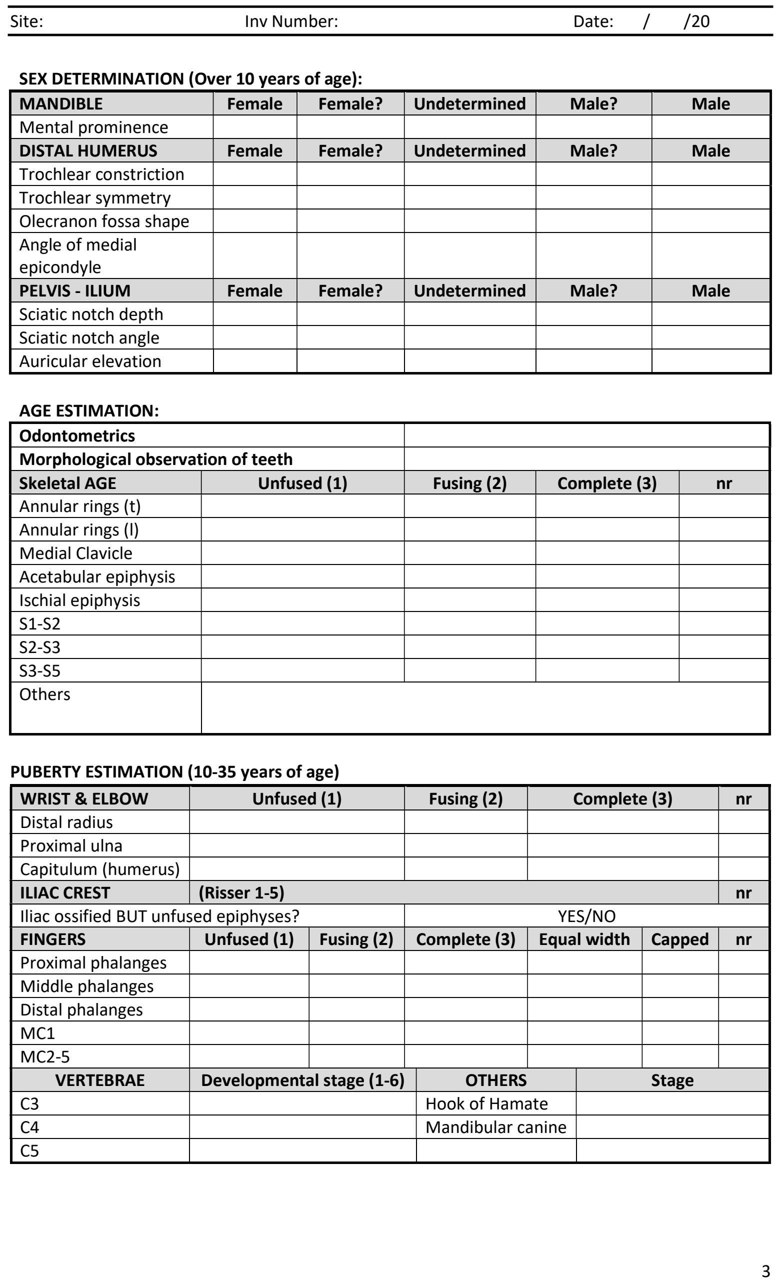
Comments:

D.2 Non-adults



Site: Inv Number: Date: / /20

Comments:



Site: Inv Number: Date: / /20

SEX DETERMINATION (Over 10 years of age):

MANDIBLE	**Female**	**Female?**	**Undetermined**	**Male?**	**Male**
Mental prominence					
DISTAL HUMERUS	**Female**	**Female?**	**Undetermined**	**Male?**	**Male**
Trochlear constriction					
Trochlear symmetry					
Olecranon fossa shape					
Angle of medial epicondyle					
PELVIS - ILIUM	**Female**	**Female?**	**Undetermined**	**Male?**	**Male**
Sciatic notch depth					
Sciatic notch angle					
Auricular elevation					

AGE ESTIMATION:

Odontometrics				
Morphological observation of teeth				
Skeletal AGE	**Unfused (1)**	**Fusing (2)**	**Complete (3)**	**nr**
Annular rings (t)				
Annular rings (l)				
Medial Clavicle				
Acetabular epiphysis				
Ischial epiphysis				
S1-S2				
S2-S3				
S3-S5				
Others				

PUBERTY ESTIMATION (10-35 years of age)

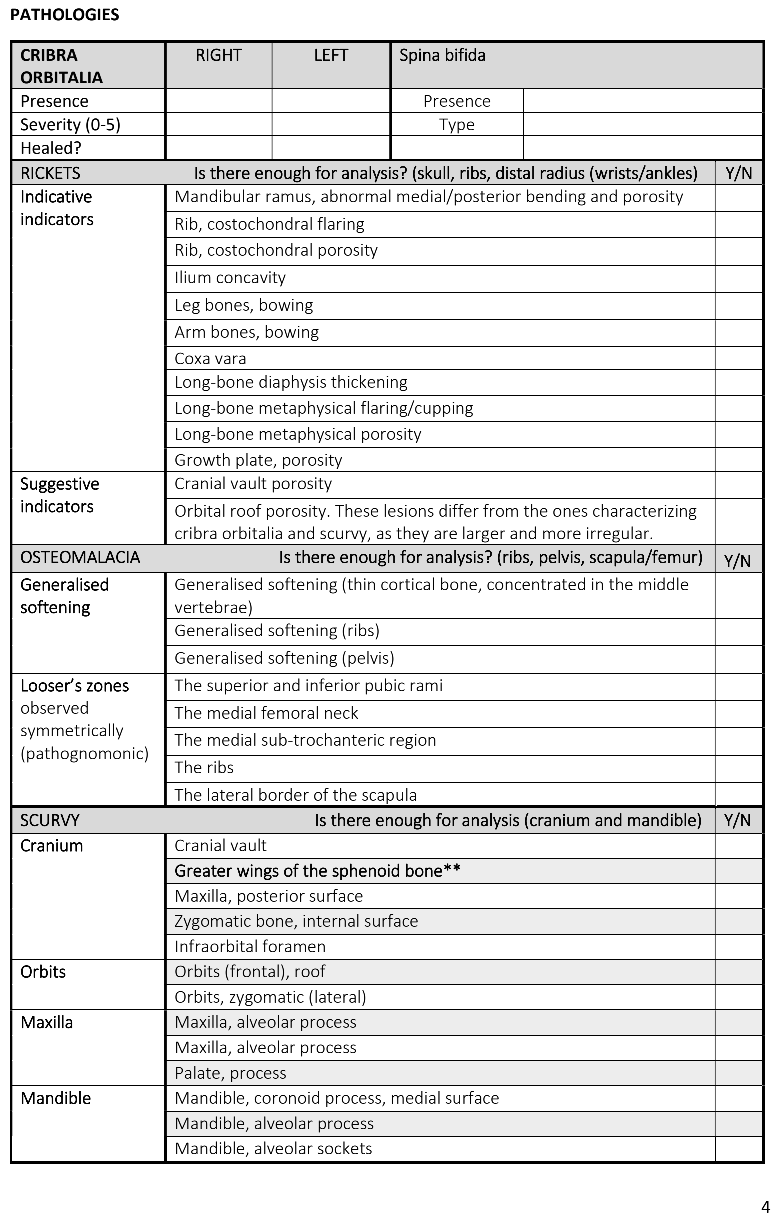
WRIST & ELBOW	**Unfused (1)**	**Fusing (2)**	**Complete (3)**	**nr**
Distal radius				
Proximal ulna				
Capitulum (humerus)				

ILIAC CREST	**(Risser 1-5)**	**nr**
Iliac ossified BUT unfused epiphyses?	YES/NO	

FINGERS	**Unfused (1)**	**Fusing (2)**	**Complete (3)**	**Equal width**	**Capped**	**nr**
Proximal phalanges						
Middle phalanges						
Distal phalanges						
MC1						
MC2-5						

VERTEBRAE	**Developmental stage (1-6)**	**OTHERS**	**Stage**
C3		Hook of Hamate	
C4		Mandibular canine	
C5			

3



PATHOLOGIES

CRIBRA ORBITALIA	RIGHT	LEFT	Spina bifida	
Presence			Presence	
Severity (0-5)			Type	
Healed?				

RICKETS	Is there enough for analysis? (skull, ribs, distal radius (wrists/ankles)	Y/N
Indicative indicators	Mandibular ramus, abnormal medial/posterior bending and porosity	
	Rib, costochondral flaring	
	Rib, costochondral porosity	
	Ilium concavity	
	Leg bones, bowing	
	Arm bones, bowing	
	Coxa vara	
	Long-bone diaphysis thickening	
	Long-bone metaphysical flaring/cupping	
	Long-bone metaphysical porosity	
	Growth plate, porosity	
Suggestive indicators	Cranial vault porosity	
	Orbital roof porosity. These lesions differ from the ones characterizing cribra orbitalia and scurvy, as they are larger and more irregular.	

OSTEOMALACIA	Is there enough for analysis? (ribs, pelvis, scapula/femur)	Y/N
Generalised softening	Generalised softening (thin cortical bone, concentrated in the middle vertebrae)	
	Generalised softening (ribs)	
	Generalised softening (pelvis)	
Looser's zones observed symmetrically (pathognomonic)	The superior and inferior pubic rami	
	The medial femoral neck	
	The medial sub-trochanteric region	
	The ribs	
	The lateral border of the scapula	

SCURVY	Is there enough for analysis (cranium and mandible)	Y/N
Cranium	Cranial vault	
	Greater wings of the sphenoid bone**	
	Maxilla, posterior surface	
	Zygomatic bone, internal surface	
	Infraorbital foramen	
Orbits	Orbits (frontal), roof	
	Orbits, zygomatic (lateral)	
Maxilla	Maxilla, alveolar process	
	Maxilla, alveolar process	
	Palate, process	
Mandible	Mandible, coronoid process, medial surface	
	Mandible, alveolar process	
	Mandible, alveolar sockets	

4

Site: Inv Number: Date: / /20

TUBERCULOSIS	Is there enough for analysis?	Y/N
Chest – Spine, ribs and sacrum	Spondylitis (**spinal** lesions)	
	Pott´s disease	
	Visceral rib new bone formation	
	Lytic lesions of the **spine** and **sacrum**	
Mandible and scapula	Osteomyelitis of the **mandible** and **scapula**	
Joints	**Joint** involvement	

OSTEOMETRICS

VETEBRAL NEURAL CANAL DIMENSIONS

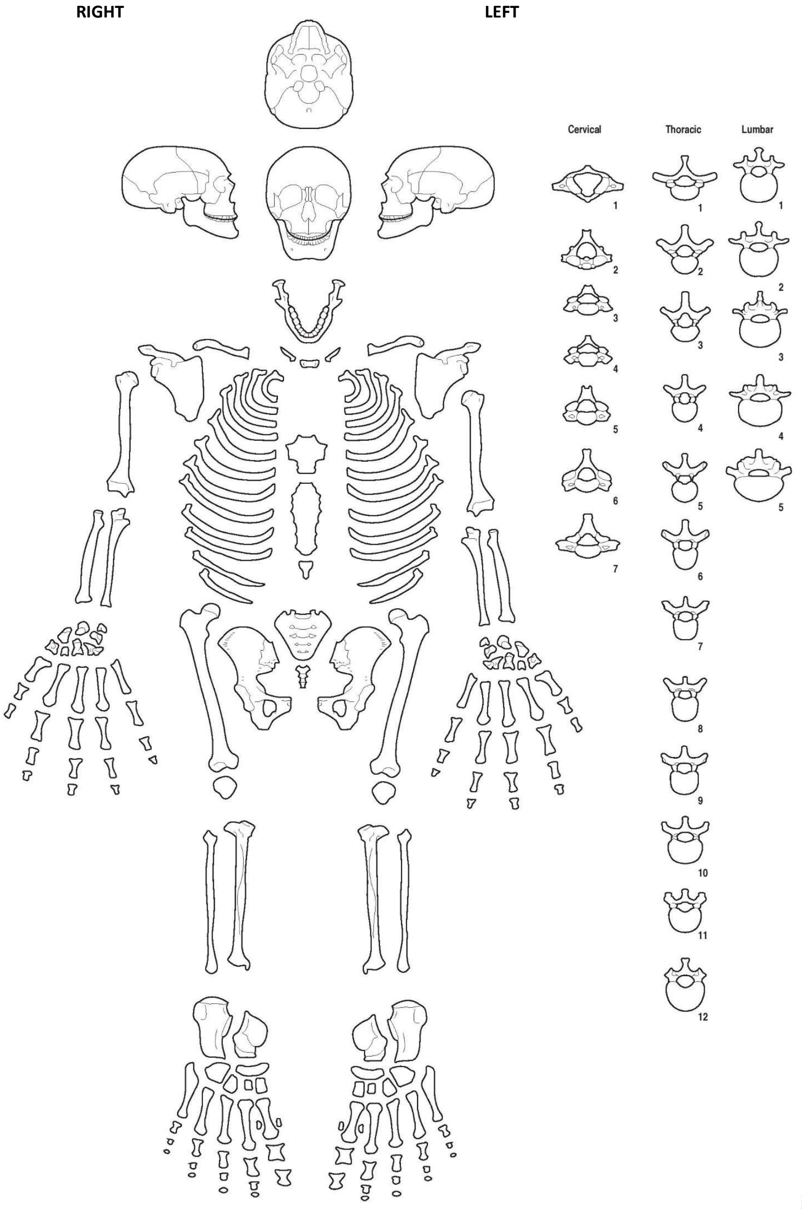
Lumbar vertebrae	Anterio-Posterior AP (1)	Anterio-Posterior AP (2)	Average	Transversal diameter TR (1)	Transversal diameter TR (2)	Average
L1						
L2						
L3						
L4						
L5						

LONG BONES LENGTH

Bones (right side)	Length (mm) (1)	Length (mm) (2)	Fused epifysis?	Bones (left side)	Length (mm) (1)	Length (mm) (2)	Fused epifysis?
Femur (right)				**Femur (left)**			
Tibia (right)				**Tibia (left)**			
Fibula (right)				**Fibula (left)**			
Radius (right)				**Radius (left)**			
Humerus (right)				**Humerus (left)**			
Ulna (right)				**Ulna (left)**			

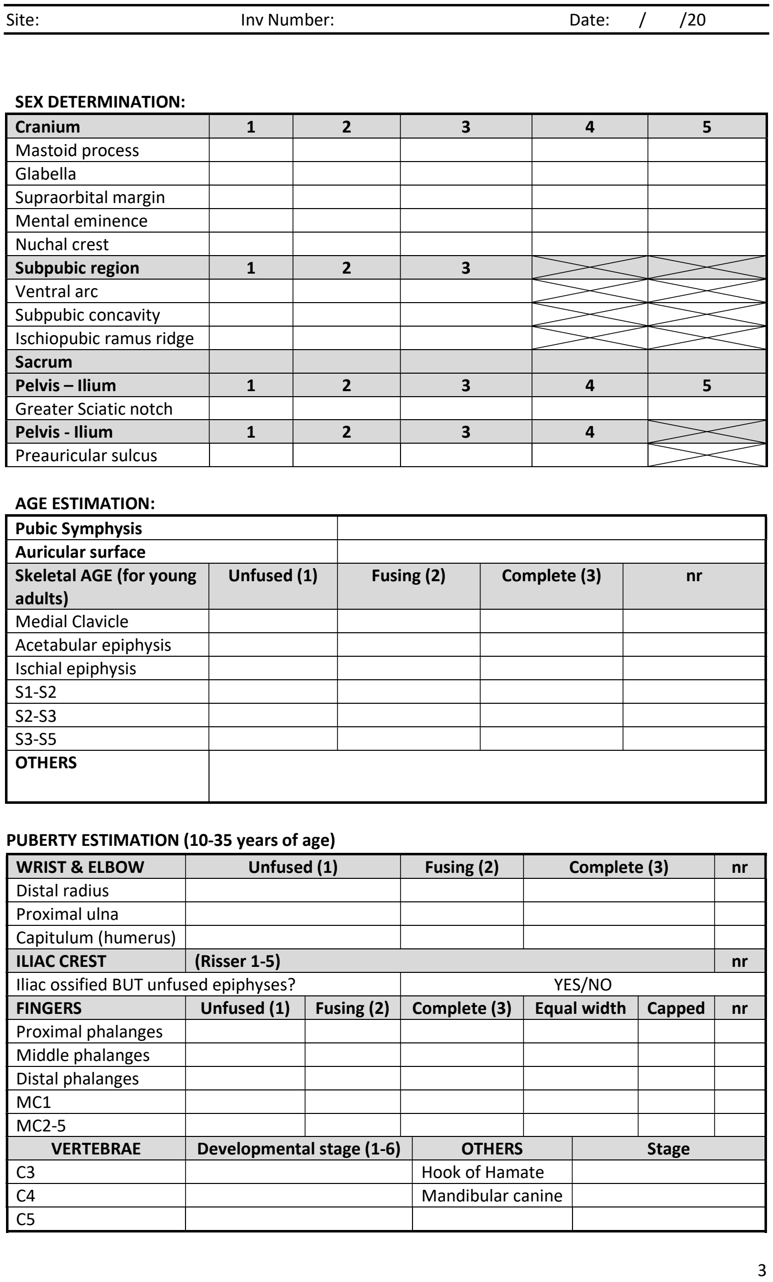
D.3 Adults

Site: Inv Number: Date: / /20



Notes and comments:

2



Site: Inv Number: Date: / /20

SEX DETERMINATION:

Cranium	**1**	**2**	**3**	**4**	**5**
Mastoid process					
Glabella					
Supraorbital margin					
Mental eminence					
Nuchal crest					
Subpubic region	**1**	**2**	**3**		
Ventral arc					
Subpubic concavity					
Ischiopubic ramus ridge					
Sacrum					
Pelvis – Ilium	**1**	**2**	**3**	**4**	**5**
Greater Sciatic notch					
Pelvis - Ilium	**1**	**2**	**3**	**4**	
Preauricular sulcus					

AGE ESTIMATION:

Pubic Symphysis				
Auricular surface				
Skeletal AGE (for young adults)	**Unfused (1)**	**Fusing (2)**	**Complete (3)**	**nr**
Medial Clavicle				
Acetabular epiphysis				
Ischial epiphysis				
S1-S2				
S2-S3				
S3-S5				
OTHERS				

PUBERTY ESTIMATION (10-35 years of age)

WRIST & ELBOW	**Unfused (1)**	**Fusing (2)**	**Complete (3)**	**nr**
Distal radius				
Proximal ulna				
Capitulum (humerus)				

ILIAC CREST	**(Risser 1-5)**	**nr**
Iliac ossified BUT unfused epiphyses?	YES/NO	

FINGERS	**Unfused (1)**	**Fusing (2)**	**Complete (3)**	**Equal width**	**Capped**	**nr**
Proximal phalanges						
Middle phalanges						
Distal phalanges						
MC1						
MC2-5						

VERTEBRAE	**Developmental stage (1-6)**	**OTHERS**	**Stage**
C3		Hook of Hamate	
C4		Mandibular canine	
C5			

PATHOLOGIES

CRIBRA ORBITALIA	RIGHT	LEFT	SPINA BIFIDA	
Presence			Presence	
Severity (0-5)			Type	
Healed?				

OSTEOMALACIA	Is there enough for analysis? (ribs, pelvis, scapula/femur)	Y/N
Generalised softening	Generalised softening (vertebrae)	
	Generalised softening (ribs)	
	Generalised softening (pelvis)	
Looser's zones observed symmetrically (pathognomonic)	The superior and inferior pubic rami	
	The medial femoral neck	
	The medial sub-trochanteric region	
	The ribs	
	The lateral border of the scapula	
SCURVY	Is there enough for analysis (cranium and mandible)	Y/N
Cranium	Sphenoid, greater wings	
Maxilla	Palate, process	
	Maxilla, posterior	
	Maxilla, alveolar process	
Mandible	Mandible, alveolar process	
	Mandible, coronoid process, medial surface	
TUBERCULOSIS	Is there enough for analysis (ribs)	Y/N
TB	Visceral surface of the ribs (RL)	
	Bone and joint lesions (BJL)	
	Hypertrophic pulmonary osteopathy (HPO) (periosteal new bone deposition on the tubular bones)	

OSTEOMETRICS

VETEBRAL NEURAL CANAL DIMENSIONS

Lumbar vertebrae	Anterio-Posterior TR (1)	Anterio-Posterior AP (2)	Average	Transversal diameter (TR) (1)	Transversal diameter (TR) (2)	Average
L1						
L2						
L3						
L4						
L5						

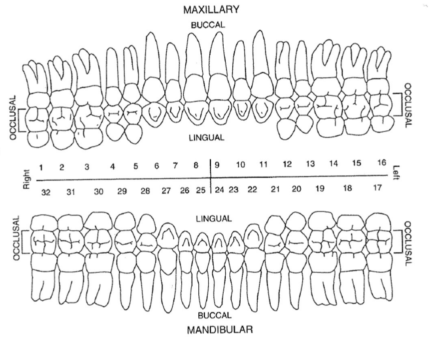
LONG BONE LENGTH

Bone (right side)	Length (mm) (1)	Length (mm) (2)	Bone (left side)	Length (mm) (1)	Length (mm) (2)
Femur (right)			**Femur (left)**		
Tibia (right)			**Tibia (left)**		

Site: Inv Number: Date: / /20

Fibula (right)			**Fibula (left)**		
Humerus (right)			**Humerus (left)**		
Radius (right)			**Radius (left)**		
Ulna (right)			**Ulna (left)**		

DENTAL ANALYSIS



	Mandibular			**Maxillary**	**General**
	M3	**M2**	**M1**	**C**	**Is LEH present?**
DEH Present/absent					
DEH Furrow					
DEH Plane					
DHE Pitted					
LEH Number					
LEH distance (mm)					

Caries (mark in diagram):

Periapical lesions (mark in diagram):

E Molecular and osteological analysis information

The following tables (E.1-E.3) show stable isotope data taken by Fuller et al. (2003) from the Wharram Percy site from individuals who could be dated to the pre- and post-Black Death. Sampling included the second deciduous molar and rib in non-adults dated to the pre-Black Death period, and permanent canine, third molar and rib in adults dated to the pre and post-Black Death period.

Table E.1: *Individuals from the Wharram Percy site dated to the pre- or post-Black Death period sampled and analysed by Fuller et al. (2003) (part 1).*

Inv Number	Period/Subperiod	Sex	Age (years)	Sample	13C	15N	C:N
G339	Late pre-Black Death	Unknown	2-2.5	(dM2[1]) crown	19.6	12.3	3.14
G339	Late pre-Black Death	Unknown	2-2.5	(dM2) cervical root	19.9	10	3.04
G339	Late pre-Black Death	Unknown	2-2.5	(dM2) apical root	19.7	9.3	3.13
G339	Late pre-Black Death	Unknown	2-2.5	rib	20.4	8.1	3.21
G363	Late pre-Black Death	Unknown	2.4-2.5	(dM2) crown	18.7	11.6	3.15
G363	Late pre-Black Death	Unknown	2.4-2.5	(dM2) cervical root	19	10.6	3.17
G363	Late pre-Black Death	Unknown	2.4-2.5	rib	20	8.8	3.24
G576	Pre-Black Death	Unknown	3.1-3.5	(dM2) crown	19.4	11.6	3.05
G576	Pre-Black Death	Unknown	3.1-3.5	(dM2) cervical root	19.5	11.1	3.18
G576	Pre-Black Death	Unknown	3.1-3.5	rib	20.5	9.3	3.22
G614	Pre-Black Death	Unknown	5.4-5.8	(dM2) crown	19.1	11.3	3.12
G614	Pre-Black Death	Unknown	5.4-5.8	(dM2) cervical root	19.4	10	3.18
G614	Pre-Black Death	Unknown	5.4-5.8	rib	20.5	8.5	3.14
G424	Late pre-Black Death	Unknown	5.4-6.5	(dM2) crown	18.8	12.1	3.14
G424	Late pre-Black Death	Unknown	5.4-6.5	(dM2) cervical root	19.1	11.6	3.19
G424	Late pre-Black Death	Unknown	5.4-6.5	(dM2) apical root	19.7	9.9	3.21
G424	Late pre-Black Death	Unknown	5.4-6.5	rib	19.9	8.4	3.2
G500	Late pre-Black Death	Female	8.5-9.5	(dM2) crown	18.9	12	3.17
G500	Late pre-Black Death	Female	8.5-9.5	(dM2) cervical root	19.6	10.4	3.17

[1] dM2 = deciduous second molar

Table E.2: *Individuals from the Wharram Percy site dated to the pre- or post-Black Death period sampled and analysed by Fuller et al. (2003) (part 2).*

Inv Number	**Period/Subperiod**	**Sex**	**Age (years)**	**Sample**	**13C**	**15N**	**C:N**
G500	Late pre-Black Death	Female	8.5-9.5	(dM2[1]) apical root	19.7	9.6	3.19
G500	Late pre-Black Death	Female	8.5-9.5	rib	20.1	8.7	3.25
G500	Late pre-Black Death	Female	8.5-9.5	(dM2) apical root	19.7	9.6	3.19
G500	Late pre-Black Death	Female	8.5-9.5	rib	20.1	8.7	3.25
G658	Late pre-Black Death	Male	10.5-11.5	(dM2) crown	18.1	10.3	3.14
G658	Late pre-Black Death	Male	10.5-11.5	(dM2) cervical root	18.6	9.9	3.18
G658	Late pre-Black Death	Male	10.5-11.5	(dM2) apical root	20	9	3.54
G658	Late pre-Black Death	Male	10.5-11.5	rib	19.7	7.9	3.18
EE36	Pre-Black Death	Female	17-25	(C[2]) crown	19	9.9	3.18
EE36	Pre-Black Death	Female	17-25	(C) cervical root	19.3	8.9	3.19
EE36	Pre-Black Death	Female	17-25	(C) middle root	18.3	8.4	3.17
EE36	Pre-Black Death	Female	17-25	(C) apical root	18.8	8.5	3.24
EE36	Pre-Black Death	Female	17-25	(M3) crown	18.7	9.1	3.17
EE36	Pre-Black Death	Female	17-25	(M3) cervical root	19	7.9	3.13
EE36	Pre-Black Death	Female	17-25	(M3) middle root	19	8	3.16
EE36	Pre-Black Death	Female	17-25	(M3) apical root	18.6	8.4	3.19
EE36	Pre-Black Death	Female	17-25	rib	19.2	9	3.29
G597	Late pre-Black Death	Female	25-35	(C) crown	19.4	9.3	3.18
G597	Late pre-Black Death	Female	25-35	(C) cervical root	19.4	7.6	3.14
G597	Late pre-Black Death	Female	25-35	(C) apical root	19.6	7.7	3.19
G597	Late pre-Black Death	Female	25-35	(M3) crown	19.5	8.2	3.16
G597	Late pre-Black Death	Female	25-35	(M3) cervical root	19.1	8.2	3.18
G597	Late pre-Black Death	Female	25-35	(M3) apical root	19	8.9	3.22
G597	Late pre-Black Death	Female	25-35	rib	19.9	7.8	3.28
CN2	Post-Black Death	Female	25-35	(C) crown	19.6	11.7	3.06
CN2	Post-Black Death	Female	25-35	(C) cervical root	19.3	11.2	3.16
CN2	Post-Black Death	Female	25-35	(C) middle root	19.5	10.6	3.19
CN2	Post-Black Death	Female	25-35	(C) apical root	19.3	10.6	3.16

[1]. dM2 = deciduous second molar; [1]. C = Permanent canine; M3 = Permanent third molar

Table E.3: *individuals from the Wharram Percy site dated to the pre- or post- Black Death period sampled and analysed by Fuller et al. (2003) (part 3).*

Inv Number	Period/Subperiod	Sex	Age (years)	Sample	13C	15N	C:N
CN2	Post-Black Death	Female	25-35	(M3) crown	19.5	11.2	3.16
CN2	Post-Black Death	Female	25-35	(M3) cervical root	19.3	10	3.13
CN2	Post-Black Death	Female	25-35	(M3) apical root	19.3	10.5	3.26
CN2	Post-Black Death	Female	25-35	rib	19.4	10.5	3.13
EE3	Pre-Black Death	Female	25-35	(C) crown	20	11	3.19
EE3	Pre-Black Death	Female	25-35	(C) cervical root	19.7	8.6	3.15
EE3	Pre-Black Death	Female	25-35	(C) middle root	19.5	8	3.17
EE3	Pre-Black Death	Female	25-35	(C) apical root	19.5	8	3.17
EE3	Pre-Black Death	Female	25-35	(M3) crown	19.1	9.4	3.13
EE3	Pre-Black Death	Female	25-35	(M3) cervical root	20.7	9	3.12
EE3	Pre-Black Death	Female	25-35	(M3) middle root	19.7	10.8	3.23
EE3	Pre-Black Death	Female	25-35	(M3) apical root	20	8.6	3.39
EE3	Pre-Black Death	Female	25-35	rib	19.8	10.3	3.29

[1.] C = Permanent canine; M3 = Permanent third molar

The following table (E.4) shows the analysis of tuberculosis on the Wharram Percy collection. The nine individuals were evaluated through aDNA analysis and the tested positive for the bacterium *M. tuberculosis*. From this sample, the only individual who fit the criteria for this investigation was identified as G438, a female who was dated to the late pre-Black Death period and aged under 35 years (25-35 yeas of age).

Table E.4: *Description of possible cases of tuberculosis in Wharram Percy adapted from Mays et al. (2001, p. 302)*

Inv Number	Age (years)	Sex	Period	Lesion		
				Spine	Hip	Other
EE056	⩾50	Male	Medieval[1]	Yes (t, l [2])	No	Yes (ribs)
G438	25-35	Female	Late pre-Black Death	Yes (t, l)	No	Yes (ulna, ribs, ilium)
G482	⩾50	Male	Late pre-Black Death	Yes (l)	No	No
NA026	35-45	Female	Medieval	Yes (t)	-	Yes (ribs. scapula)
NA046	⩾50	Male	Early pre-Black Death	Yes (l)	No	No
NA112	⩾50	Male	Early pre-Black Death	No	Yes	No
NA197	⩾50	Male	Medieval	No	Yes	No
SA013	35-45	Female	Medieval	Yes (t)	No	Yes (ribs)
WCO142	Adult[3]	Male	Medieval	Yes	Yes	Yes (ilium, femora, tibiae fibula, metatarsal, calcanei, foot phalanx)

[1] Medieval = dated within the period AD 890-1500 (with the exception of WCO142 which was dated to AD 900 and 1700. It is not possible to distinguish between pre and post Black Death period); [2] t= thoracic vertebrae; l= lumbar vertebrae; [3] adult = unaged adult.