Stressed at Birth:

Investigating Fetal, Perinatal and Infant Growth and Health Disruption.

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Thesis Submitted for the Degree of Doctor of Philosophy

2017

Abstract

The trajectory and success of fetal, perinatal and infant growth and development is regulated and/or altered by a multitude of intrinsic and extrinsic factors. Both growth and development exhibit a degree of plasticity and thus may fluctuate in response to early life adversity. Non-adult skeletal remains therefore provide a tangible record of growth and health disruption as a consequence of stress in the early life course.

This study represents the first extensive and integrated osteological and palaeopathological assessment of fetal, perinatal and infant growth and health disruption. It seeks to determine skeletal responses to adversity and to provide a comprehensive consideration of the potential pathogeneses, etiologies and contextual factors which can affect intrauterine and postnatal health and growth.

A total of 423 individuals from 15 different archaeological and historical samples, spanning a ~2000-year time period, have been considered for analysis. Assessment reveals a complex and intricate narrative of health and growth disruption, revealing evidence of chronic early life exposure to stress, which resulted in death for these individuals. A total of 192 individuals had both dental and skeletal elements preserved and 20% (*N*=39) of these were found to show significant evidence of growth disruption. Individuals from all time periods are represented, but those from post-Medieval London were found to exhibit the highest frequency and severest evidence of growth disruption. Palaeopathological analysis revealed high prevalence rates of both cranial (70%) and postcranial (30%) lesions, with cranial changes consistently more common throughout all periods and samples. New bone formation was the most commonly identified type of lesion and is considered to reflect evidence of both nutritional and infectious health stressors. Furthermore, it is suggested that socioeconomic status was a dominant factor in regulating exposure to stress. Additionally, periods of rapid cultural change also correlated with increased evidence of fetal and infant stress.

This thesis makes a number of important contributions regarding fetal, perinatal and infant growth and health during the early life course.

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Acknowledgements

I would like to thank my supervisors Dr. Rebecca Gowland and Prof. Charlotte Roberts for their continued support and guidance throughout the completion of this PhD. I would like to further thank Dr. Rebecca Gowland for her unrivalled enthusiasm to this project and for all the wonderful opportunities she has provided for me and my research. I would like to thank the AHRC (Arts and Humanities Research Council) for providing the funding for this PhD. I would also like to extend my thanks to all the staff and fellow students of the Archaeology Department, Durham University, for supporting me throughout the past four years, and who have made me one of the team.

I wish to thank all those who allowed and aided my research project, and who kindly agreed to analysis of their skeletal collections. I wish to thank David Allen (Winchester County Council), Roy and Liz Friendship-Taylor (UNAS; Piddington), David Moon (Oxford County Council), Dr. Rebecca Redfern and Jelena Bekvalac (Museum of London), and Dr. David Hunt (Smithsonian Institute).

I wish to thank all my friends for their endless support, guidance and love. To Sophie, Lauren and Brittney who have been there through it all, and to Cate, Jenny and Marc who never fail to provide emotional or nutritional support.

Finally, I would not have been able to undertake this journey without the continued loving support of my family. My unrelenting thanks to my parents and grandparents, to whom I am eternally grateful, and to Dave for his never-ending patience, care and love.

For my Grandad, who instilled archaeology in my bones.

And for Marc, who illuminated the way.

Chapter 1: Introduction

The field of bioarchaeology emerged in the 1970s and 80s as part of a scholarly movement which aimed to understand the simultaneous biological and cultural significance of lives of people in the past (Armelagos *et al.* 1982, 321; Ortner 2006, XIV; Zuckerman *et al.* 2012, 40-41). The assessment and analysis of skeletal remains has thus long since been recognised as essential in generating meaningful interpretations and perspectives regarding past individuals and populations (Armelagos *et al.* 2009, 269). As Beck states, bioarchaeology is *'…not about how* [past] *people died, but about how they lived'* (2006, 83). Furthermore, where previous decades focussed on individual case studies, unusual pathological 'specimens', and typologies (Buikstra & Cook 1980, 435; Buikstra 2006, 11; Buikstra & Roberts 2012, 768-770; Buzon 2012, 59; Zuckerman *et al.* 2012, 34; Ellison 2018, 619), bioarchaeology today attempts to focus on more general population-based analyses (Wood *et al.* 1992, 344; Roberts 2006, 423; Buikstra & Roberts 2012, 770; Zuckerman *et al.* 2012, 34). It has been recognised that such analyses must not only consider the wider archaeological context, but adopt an interdisciplinary approach to fully explore the potential of their findings (Roberts & Manchester 2010, 274; Zuckerman *et al.* 2012, 35; Martin *et al.* 2013, 7) (For further information on the history of bioarchaeological research see Angel 1981; Armelagos & Van Gerven 2003; Buikstra & Beck 2006).

Analysis of non-adult skeletal remains, including those of fetal, perinatal and infant individuals, is known to provide a wealth of information concerning both their physical and social lives (Baxter 2005, 99; Lewis 2007, 1; Halcrow & Tayles 2008, 190; Finlay 2013, 209-210; Halcrow & Ward 2017, 1). Assessment of both growth and development, and health and wellbeing, can reveal unique insights into early life experiences, and the factors to which individuals were exposed during their brief and precarious lives (Scheuer & Black 2000a, 5; Baxter 2005, 99; Lewis 2007, 1; Agarwal 2016, 130; Halcrow & Ward 2017, 1). Thus, this thesis endeavours to explore growth and health disruption in a multi-sample investigation of fetal, perinatal and infant individuals, elucidating the contextual significance of these findings in light of both ongoing archaeological and clinical investigations.

1.1 The Fetal, Perinatal and Infant Life Course

The earliest stages of the human life course have been defined as those of the fetus, perinate and infant (e.g. Lewis 2007, 2; 2017c, 1; Halcrow & Tayles 2008, 194). This study has classified individuals between 0-36 gestational weeks of age as fetal, those between 36 and 44 gestational weeks of age as perinatal, and individuals considered to be over 44 gestational weeks, up to 6 months of age postpartum, are referred to as infants. Employing this terminology has allowed distinctions to be made between individuals based on their biological age estimates, resulting in individuals also being combined into age groups which reflect the varying extent of their pre- and postnatal experiences.

The fetal, perinatal and infant life stages are ones of demonstrable fragility, where growth, health and wellbeing are malleable, dependent on a complex interplay between both genetic and environmental factors (Cattaneo 1991, 39; Saunders & Hoppa 1993, 128; Bogin 1999, 51; 228-239; King & Ulijaszek 1999, 161; Cardoso 2007, 223). With early life plasticity being a central dynamic in determining our long term developmental outcomes (Joseph & Kramer 1996, 158; Barker 1997, 807; 2012, 186; Barker *et al.* 2002, 1238; Said-Mohamed *et al.* 2018, 4), observing and identifying clear changes and disruption to growth and health in the early life course is an ongoing clinical concern (e.g. Azcorra *et al.* 2016; Fell *et al.* 2016; Fried *et al.* 2017; Holdsworth & Schell 2017; Hujoel *et al.* 2017). Both intra- and extrauterine life is regulated by a multitude of intrinsic and extrinsic factors, all of which the fetus/perinate/infant has limited individual regulation of (Cattaneo 1991, 39; Barker *et al.* 2012, 30). Consequently, assessment of fetal, perinatal and infantile growth and health status provides a tangible reflection of maternal, as well as community, health and wellbeing (Goodman & Armelagos 1989, 239; Redfern 2003, 162; Baxter 2005, 99; Lewis 2007, 20).

Understanding fetal, perinatal and infantile growth and health relies on consideration of the intricate relationship between child, mother and environment (where environment is considered to be any external factor, including those which are social, cultural, dietary or disease related). The mother-infant dyad is one in which previous and existing maternal life course experiences impact upon the growing fetus/perinate/infant (Redfern 2003, 162; Barker *et al.* 2012, 30-31; Gowland 2015, 533; Said-Mohamed *et al.* 2018, 7). Genetic information can pre-determine susceptibility and fragility to disease, as well as provide immunological resistance (Barker *et al.* 2012, 33), whilst environmental factors may limit or aid individuals

in reaching their genetic growth and health potential. With the offspring able to alter and change their developmental pathway due to the high degree of plasticity during the pre- and postnatal periods (Wadhwa *et al.* 2011, 352; Gowland 2015, 530-531; Said-Mohamed *et al.* 2018, 4; Satterlee Blake 2018, 44), growth and health status can reflect the varying maternal experiences and environmental factors experienced during the intrauterine period. Therefore, the relationship between, and wellbeing of, both mother and child is complexly bound, with the mother able to dually regulate offspring growth and health via both genetics and exposure to, and regulation of, environmental conditions.

The environmental conditions experienced during pregnancy by the mother, and thus by proxy the fetus also, can have a significant impact on intrauterine health and growth, as well as birth outcomes (Coussons-Read *et al.* 2012, 651; Dancause *et al.* 2012, 307; Glover 2015, 270; Hoffman 2016, 655). The maternal body attempts to maintain the most optimal intrauterine environment, with it considered that the mother's body instinctively prioritises the developing fetus (Gowland 2015, 533) regardless of her own health status. Consequently, during an ideal pregnancy, the offspring will typically receive nutritional and immunological safeguarding from the mother through her regulation of any environmental factors (Barker 2003, XII; Barker *et al.* 2012, 31; Said-Mohamed *et al.* 2017, 7). Changes in environmental conditions, which may be either beneficial or detrimental to the developing fetus, can alter this maternal regulation through a complicated relationship between offspring, placenta and mother (Wadhwa *et al.* 2011, 353). With fetal/perinatal/infant and maternal health directly correlated, maternal inability to buffer against detrimental environmental conditions can lead to harmful consequences; severe environmental changes – such as high disease loads, limited nutrition, and psycho-social stressors – can limit offspring growth and health, as well as cause deleterious consequences for birth timing and outcome (Zhu *et al.* 2010, 1; Wadhwa *et al.* 2011, 352; Coussons-Read *et al.* 2012, 650; Glover 2015, 270). Skeletal evidence of a poor intrauterine environment, such as growth disruption and pathological lesions, identified in fetal, perinatal and infant individuals, hence reveals not only the health status of these individuals, but also elucidates the health status of the often invisible mother (Gowland 2015, 533).

Significantly, research within the last few decades has also revealed that the environmental conditions to which we are exposed during early prenatal life can impact our gene expression

(epigenetic changes) and phenotype, reflecting the adverse or beneficial conditions experienced *in utero* (Cattaneo 1991, 40; Glover 2015, 277). Ultimately then, no longer is fetal, perinatal and infant growth and health status a reflection of either hereditary (genetic) or short-term environmental changes, but may in fact be the consequence of interaction between the two. Therefore, these epigenetic changes may predispose our susceptibility and/or resilience to certain environments or conditions, simply reflecting the ability of the human body to alter in response to alternative environmental conditions than those anticipated (Wadhwa *et al.* 2011, 352). Consequently, individuals are 'best' adapted for the environment to which they are exposed during intrauterine life (Keinan-Boker 2014, 2). The development of the Barker Hypothesis in the 1980s (Armelagos *et al.* 2009, 261; Keinan-Boker 2014, 2), today known as the Developmental Origins of Health and Disease Hypothesis (DOHaD Hypothesis) (e.g. Barker 2012) has led to extensive research revealing the plethora of long term growth and health implications of a detrimental *in utero* experience (Ulijaszek & Henry 1996, 1; Barker 1997; 2003; 2012; Barker *et al.* 2002; 2012; Hoffman 2016, 656). Today, both epidemiological and auxological research is revealing the wealth of factors that impact birth and life course outcomes, considering the epigenetic impact of environmental and lifestyle factors (e.g. Barker *et al.* 1990; Barker *et al.* 1991; Syddall *et al.* 2005; Glover 2012). Contemporary concerns include the effects of maternal smoking (e.g. Cornelius & Day 2009; Langley *et al.* 2012; He *et al.* 2017), alcohol consumption (e.g. Coathup *et al.* 2017; Mamluk *et al.* 2017; Sundelin-Wahlsten *et al.* 2017) and vitamin/nutrient deficiencies (e.g. Roseboom *et al.* 2000; Rogne *et al.* 2017). Results of these studies have found that individuals exposed to such factors *in utero* have increased risk of diabetes (e.g. Stöger 2008), heart disease (e.g. Barker & Osmond 1986; Barker *et al.* 1989), behavioural disorders (e.g. Armelagos *et al.* 2009, 263; Malaspina *et al*. 2008; Khashan *et al.* 2008; Egliston *et al.* 2007) and obesity (e.g. Vickers *et al.* 2000; Ojha & Budge 2017).

The complexity of epigenetics is further exacerbated when multiple generations or lengthy temporal periods are considered – as in an archaeological context. Epigenetic traits become embedded within the overall genetic code, with the expression of these traits transferred from parent to child, and subsequently grandchild (Glover 2015, 277; Gowland 2015, 534; Satterlee Blake 2018, 43). As intrauterine experiences influence and determine susceptibility to disease over the life course (Barker 1994, 1), it has been suggested that those who experience a detrimental prenatal environment are more likely to be unable to provide an

optimum *in utero* environment for their own offspring as a result of 'ancestral experiences' and an accumulation of risk (Gowland 2015, 534). Ultimately, a predisposition to poor health over an individual's life course is likely to result in similar or additional detrimental exposures in the offspring. This results in a cyclical process whereby epigenetic changes become embedded and cumulative across generations (Gowland 2015, 534-535). This complexity has led to Gowland (2015) conceptualising this mother-infant dyad and intrauterine period in terms of entanglement – where multigenerational, and both intrinsic and extrinsic factors, are entwined in determining the fetal/perinatal/infant life course. Consequently, we must no longer view individuals in isolation, considering only their immediate life course experience, but instead reflect on the life course experiences of their parents and grandparents also.

In addition, pregnancy-related cultural practices, as well as response, treatment and care of the child once born can all also be reflected in individual growth and health status (Finlay 2013, 212; Satterlee Blake 2018, 43 e.g. Wilkie 2013). As Sánchez Romero emphasises (2017, 18), from birth, cultural and care related practices are essential in not only enabling and contributing to survival of the offspring, but also providing the optimum conditions for health, growth and wellbeing. Indeed, growth and health status in turn reflect the social, cultural and economic conditions experienced in early pre- and postnatal life (Sánchez Romero 2017, 18).

As a result, both the intra- and extrauterine environment, and the mother's ability to regulate this, as well as the relationship and interaction between environmental, epigenetic, and genetic factors, must all be considered in relation to individual growth and health. Understanding the complexities of these relationships is however, somewhat problematic, as these multiple mechanisms can cause similar health and growth outcomes for the offspring (Kramer & Joseph 1996, 1254; Kramer 1998, 663; Armelagos *et al.* 2009, 264; See Armelagos *et al.* 2009, 268 for a comparison of how environmental, genetic and epigenetic factors could each be responsible for negative growth and health outcomes). Therefore, defining the precise cause/factor for growth and health disruption, and disentangling environmental factor from genetic is challenging, particularly in regards to archaeological skeletal remains. Yet, what must remain paramount is the acknowledgement that growth and health cannot be simply understood, and instead reflect a complex interplay of factors, which

extend from the immediately experienced pre- and postnatal environments, to intergenerational health and life course experiences.

1.2 The Fetus, Perinate and Infant in Archaeology and Bioarchaeology

The fetus/perinate/infant has long been a marginalised entity, remaining invisible within archaeological discourse until the last two decades (Scott 1999, 5; Lewis 2007, 1; 3; Halcrow & Tayles 2008, 191; Finlay 2013, 209-210; Sánchez Romero 2017, 16). This is partly a consequence of the historical notion, that was once widely held, that such young, fragile, and small individuals were worthless, unable to provide any data of value (Pollock 1983, 1; Lewis 2007, 20; Kamp 2015, 162). As a result, fetal, perinatal and infant individuals have often been marginalised in the archaeological record (Baxter 2005, 2; Halcrow & Tayles 2008, 191; 197; Kamp 2015, 162; Sánchez Romero 2017, 32); commonly missed, misidentified (Lewis 2007, 26; e.g. Ingvarsson-Sundström 2003, 15), or even discarded in the past (Becker 2006, 655). Today increasing interest and awareness of the importance and value of these individuals is leading to their more thorough and careful excavation and assessment. Although osteological assessment of fetal/perinatal/infant individuals is still generally considered to be more difficult than that of older non-adults and adults (Lewis 2018, 113), increasing numbers of studies assessing aspects of fetal/perinatal/infant life are exposing the value and significance of their investigation (Finlay 2013, 212; Halcrow *et al.* 2018, 83). In the last 30-years numerous anatomical and archaeological collections of non-adults have been published and have primarily been used to test methodologies for determining biological age and sex (e.g. Moorrees *et al.* 1963a; 1963b; Redfield 1970; Maresh 1970; Garn *et al.* 1973; Garn & Clark 1975; Fazekas & Kósa 1978; Weaver 1980; Molleson 1990; Molleson & Cox 1993; Schutkowski 1993; Scheuer & Black 1995; Scheuer 1998; Scheuer & Black 2000a; AlQahtani *et al.* 2010; Aleman *et al.* 2012).

The 1980s saw the development of post-processual archaeology, within which gender-related and feminist focuses emerged (Lewis 2007, 1; e.g. Derevenski 1994; 1997; Moore & Scott 1997; Meskell 2001; Diaz-Andreu 2005). These initial studies addressed aspects of the 'marginalised individual' and thus began to consider the roles of women and 'children' within the archaeological record (e.g. Lillehammer 1989). However, many of these early studies focussed on the attitudes of adults towards these younger individuals rather than the experience or perception of the infants/children themselves (Scott 1997, 6-7; Baxter 2005, 17;

Lewis 2007, 3). In particular, non-adults were typically considered in relation to women, and the impact they had on the lives and work of women (Scott 1999; 5; Lillehammer 2000, 17; Lewis 2007, 1). Consequently, it was not until the 1990s that children, specifically fetal, perinatal and infant individuals, were considered as active, rather than passive, participants in life (Lewis 2007, 1; Mays *et al.* 2017, 1; e.g. Sofaer Derevenski 1994a; 1994b; 1997; Baker 1997; Baxter 2005; Halcrow & Tayles 2008; 2011). Much of the literature regarding 'childhood' has tended to focus on the cultural, social and burial environments and treatment they were afforded (e.g. Crawford 1991; Scott 1999; Sofaer Derevenski 2000; Baxter 2005) rather on the physiological data that can be gathered from the skeletal remains themselves (Shilling 2003, 21; 105; Prout 2005, 57). Today, an increasing awareness of the significance of fetal/perinatal/infant skeletal and dental remains is revealing important insights into the life course experiences of the youngest individuals in the past (e.g. Gowland 2017). The highly plastic nature of the skeleton during these early life stages means that not only are fetal/perinatal/infant individuals particularly susceptible to negative environmental onslaughts (Cardoso 2007, 223; Armelagos *et al.* 2009, 267), but their bony remains are more likely to reflect growth and health disruptions as a result. This is why these individuals (fetuses/perinates/infants) are considered to be some of the most sensitive barometers for population health and wellbeing (Lewis 2000, 39; Baxter 2005, 99).

However, correlating concepts of growth and health disruption to archaeological skeletal material is often problematic. While bioarchaeology widely adopts an interdisciplinary approach, utilising methods and comparative data from fields including anthropology and medicine (Martin *et al.* 2013, 1; Zuckerman *et al.* 2012, 35), much of the literature continues to avoid skeletal changes relating to fetal, perinatal and infant individuals, despite these individuals providing unrivalled potential for investigating early life course experiences. With bone turnover in these young individuals exceptionally rapid (Ulijaszek & Henry 1996, 2), responses to growth and health insults present much earlier, and often more severely, as a result of their immature immune system, than they would within an adult individual (Goodman & Armelagos 1989, 239; Perry 2005, 92; Halcrow & Tayles 2008, 336). Furthermore, clinical studies often focus on aspects of human systems unavailable for assessment in the archaeological record, such as soft tissue structures, and diseases which leave no discernible traces on the skeleton (Ortner 2008, 191). This means considering

archaeological and historical individuals is vital in understanding the skeletal mechanisms and responses to detrimental early life intra- and extrauterine experiences.

1.3 Research Aims

This research aims to investigate fetal, perinatal and infantile skeletal growth and health as a response to stress within a variety of temporally disparate archaeological and modern samples. Undertaking detailed macroscopic osteological analysis, and exploring multiple methodological approaches, this project considers the varying stressors which have impacted on early growth and health status. Consequently, intra- and inter-sample comparisons enable consideration of similarities and differences in the skeletal responses to health and growth disruption between individuals from different environments, social statuses and time periods. Thus, this study aims to further current understanding regarding the skeletal evidence for early life exposure to stressors, and to provide a comprehensive consideration of the potential pathogeneses, etiologies and contextual factors which lead to growth and health disruption. Individuals up to the age of six post-partum months have been considered for assessment. This upper age limit was determined to ensure all individuals assessed reflected the very earliest stages of the life course and the immediate pre- and postnatal environments; such a narrow age range meant that individuals assessed would have had a limited duration in which they could respond to, and recover from, pre- and postnatal insults.

1.4 Research Objectives

In order to achieve the intended research aims, this research study has seven primary research objectives:

1. Synthesise and review the corpus of literature regarding the fetus, perinate and infant in archaeological and bioarchaeological discourse. Integrating clinical, anthropological and biological research, consider the developments in fetal/infant studies and the ongoing limitations.
2. Undertake metric, dental and pathological assessment of 423 individuals from 15 archaeological/modern samples. Compile databases of skeletal measurements, dental development and pathological lesions for each individual, where possible.
3. Calculate age-at-death estimates for each individual (where possible) using metric and dental data collected.

4. Assess differences and variations in age-at-death estimates generated, and determine which skeletal element(s) show higher levels of variation/disruption.
5. Record the presence and characteristics of pathological lesions, including their distribution within skeletal elements and the broader skeleton.
6. Calculate the TPR (True Prevalence Rate) of lesions and consider the potential pathogeneses and etiologies of these lesions within and between samples.
7. Examine correlations between evidence of growth disruption and health disruption, whilst integrating contextual information regarding the individuals/samples/time periods analysed.
8. Synthesise metric, dental, pathological and contextual data to observe and explore temporal patterns of growth and health disruption and develop a narrative regarding the fetal, perinatal and infant life course in the past.

1.5 Research Questions

The primary research questions articulated in the study design of this project were as follows:

1. Is there evidence of growth disruption (extreme variation) between skeletal elements of individuals considered within each sample or time period?
2. How many individuals within each sample/time period show evidence of growth disruption?
3. Is evidence of growth disruption consistent between samples/time periods?
4. Which period has individuals with the most extreme evidence of growth disruption and why?
5. Is there a correlation between growth disruption and pathological lesions?
6. Which skeletal elements are most commonly found to have a pathological response and how do these lesions present?
7. What are the etiological, pathogenic and contextual implications of evidence of health disruption?
8. Can we differentiate between new bone formation as part of normal growth and pathological bone formation?

1.6 Thesis Outline

This thesis has been sub-divided into 11 chapters in order to address the research aims, objectives and questions outlined above. Bioarchaeological, anthropological and clinical research has been widely considered throughout this thesis to reflect the multi-disciplinary interest and importance of such research. Consequently, Chapters 2 and 3 will provide a theoretical and biological framework to contextualise the themes, concepts and literature explored within this study. Chapters 4 and 5 outline the skeletal samples and methods utilised in this study. Chapters 6, 7, 8 and 9 are the research manuscripts presented in this thesis. The first three research manuscripts explore health and growth disruption within the contextual parameters of specific time periods. The fourth manuscript, Chapter 9, specifically explores the pathological lesions identified, as a proxy for evidence of health disruption. Chapter 10 draws together the findings of the research manuscripts and discusses them in reference to the research questions listed above. Chapter 11 concludes this thesis by addressing the research aims, objectives and questions, outlining the results of this assessment and detailing the potential future recommendations and directions for fetal, perinatal and infant studies.

The manuscripts in this thesis have been formatted for publication in peer-reviewed research journals and are thus multi-authored between Claire M. Hodson and Dr. Rebecca L. Gowland. All data collection and analysis was conducted by Claire M. Hodson. Dr. Rebecca L. Gowland and Prof. Charlotte A. Roberts were supervisors on this research project and provided support, guidance and editorial suggestions in the development and production of this research project, all four research manuscripts and the final thesis.

A detailed structure of this thesis has been outlined below:

Chapter 2: Investigating the Fetus, Perinate and Infant; The Emerging Bioarchaeological Field

This chapter provides an overview of the history of fetal, perinatal and infant studies and discusses the existing bioarchaeological research in this field to document the major themes so far explored. As fetal/perinatal/infant studies have become a burgeoning field of study, new theoretical and methodological approaches to investigating these young individuals have been outlined. Common debates and ongoing limitations of non-adult studies have also been considered. This chapter will also reflect on the potential problems when working with

skeletal human remains, particularly those of such young individuals, and consider the limitations of retrieval, identification and of existing methods typically employed in analysis. This chapter will also discuss the ethical considerations of working with fetal, perinatal and infant individuals, particularly in regards to the Smithsonian Fetal collection.

Chapter 3: The Beginnings of Life; Understanding Growth, Health and Stress in a Bioarchaeological Context

This chapter discusses in detail the growth and development of both skeletal and dental tissues, considering in particular the biological mechanisms behind cranial and long bone growth. This chapter also considers the definitions and terminology associated with chronological and physiological age, as well as exploring the meaning of growth, health and stress in a bioarchaeological context.

Chapter 4: Materials

This chapter details each of the 15 archaeological or historical collections analysed within this research in chronological order. This chapter provides contextual information regarding the history of each site, as well as information related to the burial, excavation and curation of the individuals assessed. The rationale for inclusion of these sites/collections within this study has also been outlined.

Chapter 5: Methods

This chapter outlines the methods used to both collect and analyse skeletal data for metric, dental and pathological assessment. It considers the limitations of these methods and justifies the use of these methods within this thesis. This chapter also discusses the methodologies that were not employed and included within this study and justifies their exclusion. This chapter also details results of intra- and inter-observer error calculations.

Chapter 6 (Manuscript 1)**:** *Measure for Measure: A comparative study of the impact of stressors on fetal, perinatal and infant growth in Iron Age and Roman Britain.*

This article compares and contrasts growth changes and pathological indicators identified in individuals from Owslebury, Piddington and Barton Court Farm. This paper investigates correlations between pathological indicators and growth changes in the Iron Age and Roman individuals assessed, highlighting an increase in health disruption from rural Iron Age to rural

Roman individuals. Contextual implications for the growth and health disruption identified are also thoroughly discussed. This paper also exposes the need for dental development to be used to more accurately age fetal/perinatal/infant individuals, highlighting the inherent bias in the skeletal methods typically used for age assessment. Assessment of these individuals, using the methodologies outlined to accurately determine health and growth disruption, also supports arguments against infanticide, and emphasises the need for holistic interpretations of non-adult death and burial on rural settlement sites during the Iron Age and Roman periods.

Chapter 7 (Manuscript 2): *Like Mother, Like Child: Investigating change and continuity in fetal, perinatal, infant and maternal health stress in post-Medieval London.*

This article compares and contrasts growth changes and pathological indicators from individuals excavated from post-Medieval London. Although extensive research has been conducted on the adult individuals excavated from these contexts, little consideration of the fetal/perinatal/infant individuals has yet to be afforded. Assessment of such individuals explores the socioeconomic implications of being members of these communities, highlighting geographical, economic and status driven distinctions in growth and health status. This paper illuminates the deplorable and deleterious conditions that many faced during the post-Medieval period in London, revealing evidence of chronic, inescapable illness and poverty. Methodologically, the most revealing implication from the assessment of these individuals is that the *pars basilaris* appears to be a good proxy for dental, and subsequently chronological, age. Comparability between this cranial skeletal element and the dentition suggests prioritisation of growth within these elements, whilst post-cranial, particularly long bone growth, appears to be severely disrupted. Individuals assessed from post-Medieval samples in London were found to show the most extreme and consistent evidence for both growth and health disruption.

Chapter 8 (Manuscript 3): *Little Lives: A metrical and morphological evaluation of a documented 20th Century fetal and infant skeletal collection.*

This article considers growth and health disruption in a clinical sample stored at the Smithsonian Institute in Washington D.C. where cause of death, biological age-at-death, and biological sex are often recorded. This sample, although temporally and geographically disparate from the other samples assessed within this thesis, enables consideration of the accuracy of the methodologies applied to determine growth and health disruption. This paper

examines the differences in health and growth disruption based on biological age, biological sex and cultural background of these individuals, identifying clear differences in growth strategies based on these factors. The implications of targeted collection practices are also extensively considered within this paper, with these the likely cause of the high pathology rates within the sample; individuals were collected on the basis of their health/disease status and cause of death. Analyses of individuals within this fetal collection, and the relationships between growth and health status, hence provide a unique dataset for comparative analysis and aid in interpretation and understanding of these factors in the early life course.

Chapter 9 (Manuscript 4): *Perinatal Pathology in Bioarchaeology: Investigating the Potentials, Problems and Implications.*

This article focusses on the assessment of pathological lesions in fetal, perinatal and infant remains. Using all 423 individuals from the collections analysed throughout this thesis, this paper explores the difficulties and complications of assessing pathological changes in young individuals, considering in particular the ongoing debate surrounding the differentiation between normal and pathological new bone formation. This paper also considers the implications of the location and appearance/type of pathological lesions to consider the etiological and pathogenic differences, contextualising these within the archaeological and historical record to observe changes and continuity in patterns of health disruption over time. In addition, this paper comprehensively considers the DOHaD hypothesis and the implications of multigenerational health disruption, looking to the future and the further avenues of research to be pursued if the interactions between health and disease, and their combined implications on long term health, are to be truly understood throughout history. This paper proposes a new methodological approach to assessing fetal/perinatal/infant pathology and provides a comprehensive overview of the potential causes and consequences of identified lesions.

Chapter 10: Discussion

This chapter brings together findings from the research manuscripts and discuss the results in reference to the research questions outlined for this study. It highlights key findings regarding growth and health disruption and composes a comprehensive and complicated narrative of fetal, perinatal and infant health and growth through time.

Chapter 11: Conclusion and Recommendations

This chapter concludes this thesis by addressing the research aims, objectives and questions of this study. It also considers the potential future recommendations and directions for fetal, perinatal and infant studies.

Chapter 2: Investigating the Fetus, Perinate and Infant – The Emerging Bioarchaeological Field

This chapter provides an overview of fetal, perinatal and infant studies. The history and development of this field will be discussed, considering its origins within archaeological discourse, as well as the emerging focus in bioarchaeology of these young individuals. As a result of these new avenues of research, common themes, debates and interpretations surrounding the recovery and assessment of non-adult remains have also been considered and discussed. This chapter also examines the limitations of working with archaeological human remains, specifically those of fetal, perinatal and infant individuals, and discusses the problems arising from both excavation and analysis. A discussion of the ethical considerations of working with fetal, perinatal and infant individuals is also provided.

2.1 The History and Development of Fetal, Perinatal and Infant Research

The study of non-adults within bioarchaeology is still a relatively recent avenue of scholarly research (Halcrow & Tayles 2008, 191; Mays *et al.* 2017, 38-39); the recognition that these individuals provide important insights into health and the life course has only emerged more substantially over the last two decades (Scott 1999, 5; Cox & Mays 2000, 8; Lewis 2007, 1; 3; Halcrow & Tayles 2008, 191). By contrast, the broader study of childhood and infancy in the past has seen a wealth of multidisciplinary studies, although many of these have focused on the social worlds of children, and past perceptions of childhood (Pollock 1983, 1; Scott 1997, 6-7; Baxter 2005, 17; Lewis 2007, 3; Halcrow & Tayles 2008, 199; e.g. Lillehammer 1989). Though many disciplines, including history, anthropology and sociology, were quick to investigate these young individuals, archaeological investigations were more tentative (Lillehammer 2015, 79-80). Thus, discourse has been dominated by the social context and material culture of children, rather than the individuals themselves (Shilling 2003, 22; 105; Prout 2005, 57; Halcrow &Tayles 2008, 191). Where biological assessment has been afforded it has tended to be sensationalist in nature, focussing on controversial aspects such as infanticide (Sofaer Derevenski 1994a, 8; Lucy 2005, 45; Finlay 2013, 210; e.g. Smith & Kahlia 1992; Mays & Faerman 2001). Today, bioarchaeological studies are rapidly redressing this balance, refuting notions of deliberate disposal and lack of care afforded towards infants in the past (e.g. Gowland & Chamberlain 2002; Millet & Gowland 2015;

Hodson 2017), instead focussing on the unique biological and contextual interpretations they can reveal. However, there is still great potential for the study of fetal, perinatal and infant individuals (Baxter 2005, 93; Halcrow & Tayles 2008, 209).

Historically, two primary strands of childhood discourse emerged; the first of which was predominantly historical, considering the varying treatment of these individuals in the past (Mays *et al.* 2017, 38). The origin of these studies has been widely attributed to Ariès and the publication of *'Centuries of Childhood: A Social History of Family Life'* (1962), translated into English in 1962 (Cunningham 1998, 1197). This work, although denoting a clear realisation that 'children' were important individuals deserving of scholarly consideration, viewed childhood as a modern construction that post-dated the seventeenth century (Ariès 1962; Pollock 1983, 2; Lewis 2007, 2-3; Halcrow & Tayles 2008, 199). Ariès argued that adults, historically, cared less for their children, considering them to have less of an emotional attachment to their offspring due to the high levels of infant mortality commonly experienced in the past (Ariès 1962). He attributes the lack of childhood as a consequence of adults endeavouring to turn offspring into 'mini-adults' – seen in the way offspring were dressed and expected to behave as adults (Ariès 1962). Consequently, the role of the archaeological child and infant has typically been seen as unimportant, inferior to that of adults (Sofaer Derevenski 1997, 193), who in turn are historically considered to have treated infants and children with minimal care, affection and/or respect (Gowland & Chamberlain 2002, 684). As Lewis states, *'…we are led to believe that a child's upbringing was a combination of neglect and cruelty'* (2007, 3). Indeed, such attitudes have become embedded within bioarchaeological and archaeological discourse, and have undoubtedly led to the assumption that non-adults are peripheral and passive in comparison to their adult counterparts.

Such notions have, and continue to be, strongly rejected, with many studies investigating the care afforded to infants and children, as well as the agency and identity of these individuals, in the past (e.g. Sofaer Derevenski 1994a; 1994b; 1997; Baker 1997; Saunders 2000; Kamp 2001; Baxter 2005; Gowland 2006; Halcrow & Tayles 2008; 2011; Lewis 2017b). This has been demonstrated in a range of research contexts, from burial treatment, evidence of material culture, through to social and legal legislation regarding children (Lewis 2007, 3; e.g. Carroll 2011; 2012; Wilkie 2000). Furthermore, infancy and childhood have been

recognised as significant transitions in the life course, and archaeological evidence refutes the idea that such stages of life were not acknowledged or marked within different societies in the past (Lucy 2005, 43-44).

The second strand of childhood discourse is often attributed to the emergence of gender and feminist perspectives within archaeology, where the study of infancy and childhood was first widely addressed (Halcrow & Tayles 2008, 199; 2011, 333; Mays *et al.* 2017, 38; e.g. Moore & Scott 1997; Meskell 2001). These studies focused on 'invisible' demographics, attempting to also examine the socially constructed identities of childhood and adulthood (Mays *et al.* 2017, 38). However, such studies typically sought to emphasise the place and role of women in history and prehistory (Halcrow & Tayles 2008, 199), where non-adults often became 'feminine' and an extension of women's 'work' - confined to the family and household (Baker 1997, 183; 186; Sofaer Derevenski 1997, 192; Scott 1999; 5; Lillehammer 2000, 17; Lewis 2007, 3). By relegating individuals into passive roles non-adults have long been considered as 'others' (Baker 1997, 187; Wyness 2006, 34-35). This 'othering' is also substantiated by the use of terminology such as sub-adult – suggesting these young individuals are inferior and incomplete versions of adults (Prout 2005, 10; 33; Sofaer 2006, 121; Lewis 2007, 2; Halcrow & Tayles 2008, 193).

The best data available to consider the lives of infants and children in the past are their skeletal remains, which provide insights into both their physical and social lives (Lillehammer 2000, 21; Baxter 2005, 93; Lewis 2007, 10; Halcrow & Tayles 2011, 336-338). Bioarchaeology has only recently acknowledged the significance of fetal, perinatal and infant skeletal remains and their ability to provide unique insights into past health, growth, and the social and cultural experiences and care afforded to them in their brief lives (Halcrow & Tayles 2008, 198; 202; 2011, 333; Mays *et al.* 2017, 39 e.g. Saunders 2000; Gowland 2001; Lewis 2007; Dawson 2017; Lewis 2017b). However, the science-theory divide (Gowland and Knüsel 2006, ix; Halcrow & Tayles 2008; 2011, 333-334; Gowland & Thompson 2013, 19; Mays *et al.* 2017, 41) has led to a lack of integration between biological (age, sex and pathology) and social studies regarding concepts of infancy within past populations. Today, biocultural studies are simultaneously addressing both the social and cultural constructions of life course experiences, and the biological and environmental processes affecting the body

(e.g. Robb 2002; Gowland & Knusel 2006; Gowland 2006; Sofaer 2006; Gowland and Thompson 2013).

Furthermore, the publication of specific non-adult osteology texts has improved the identification and understanding of non-adult anatomy (Mays *et al.* 2017, 39; e.g. Scheuer and Black 2000; Baker *et al*. 2005; Schaefer *et al.* 2009). The publication of Lewis' *The Bioarchaeology of Children* (2007) was the first to synthesize the theoretical and biological considerations of non-adult studies (Mays *et al.* 2017, 39). With ongoing research, and increasing publications regarding early life from archaeological samples around the world, our understanding, analysis and interpretation of these young individuals continues to become more precise, comprehensive and nuanced.

For a thorough review of the development of non-adult studies, and a contemporary consideration of the major theoretical and analytical developments see Mays *et al.* (2017).

2.2 Contemporary Research and Current Debates

In a review of American Anthropologist, Schwartzman (2001, 16) found that only 4% of articles published contained detailed information regarding non-adults, whilst only three of those articles were published between 1986 and 2001 (Hirschfield 2002, 612). Such findings are a clear reflection of the continued marginalisation of non-adult individuals within anthropological and archaeological discourse (Baxter 2005, 2). A recent assessment of papers within the SSCIP (Society for the Study of Childhood in the Past) publication '*Childhood in the Past*' found that 75% were of an archaeological focus (Murphy 2017, 3), though only 26.5% focused on burial evidence (Murphy 2017, 4). Within the same volume Mays and colleagues (2017) assessed contributions to seven major anthropological/osteological journals between 2006 and 2015, finding that 15% of articles focussed primarily on non-adults. Despite this enduring legacy of underrepresentation in the literature, non-adult studies, specifically those looking at the youngest individuals (fetuses, perinates and infants) have emerged. Key debates have developed, often surrounding the methodological issues of being able to accurately determine age-at-death, biological sex and disease/health status of non-adult individuals. The following section considers the primary discussions and works that have furthered the study of non-adult skeletal remains.

2.21 Age Estimation in Non-Adult Skeletal Remains

Within bioarchaeology, initial studies concentrated primarily on the methodological approaches to determining biological age and sex (Lewis 2007, 11; e.g. Schour & Massler 1941a; 1941b; Boucher 1955; Maresh 1970; Fazekas & Kósa 1978; Weaver 1980; Schutkowski 1993). Age estimation of non-adults is a central concern as it can aid interpretations of mortality, morbidity, demography, growth, health (both individual and population), and environmental conditions (Lewis 2007, 38; Satterlee Blake 2018, 34). Assessing age-at-death is probably one of the biggest methodological challenges for fetal, perinatal and infant investigations. Genetic and environmental factors interact to create variation in the timing and rate of growth between individuals (Cameron 2002, 13). Consequently, although there are multiple ways to determine age clinically, within bioarchaeology it is much harder to assess as estimation relies on physiological analysis of skeletal and dental remains (Scheuer & Black 2000a, 6; 13; Satterlee Blake 2018, 37). Methodologies primarily consist of metric assessment of skeletal remains, whilst both development and eruption can be considered for dentition. Although this chapter avoids detailed discussion of the methodologies (See Chapter 5 for this), major limitations exist in estimating non-adult age.

A range of reference data has been used to calculate 'standards' for correlations between skeletal growth and chronological age. It is assumed that the biological stage of development/growth is a good proxy for chronological age, consequently diaphyseal length of long-bones has become the standard measurement used to calculate age-at-death (Scheuer & Black 2000a, 4; Lewis 2007, 43). However, today we acknowledge that genetic, environmental and epigenetic factors may all cause variation in the expected growth trajectory of an individual (Goodman & Armelagos 1988, 941-942; Goodman *et al.* 1988, 169-170; Bush & Zvelebil 1991, 5; Scheuer & Black 2000a, 4-5; Satterlee Blake 2018, 35), and are thus critical of methodologies which use metric assessment of growth to estimate age (Lewis 2007, 43). Where possible, assessment of dental development is encouraged as this is considered more accurate (Moorrees *et al.* 1963b, 1490; Bang 1989, 216; Hoppa & Fitzgerald 1999, 3; Gowland & Chamberlain 2002, 677; Satterlee Blake 2018, 38), but fetal, perinatal and infant dentition is small, often not recovered, or simply misidentified (Gowland & Chamberlain 2002, 677; Lewis 2007, 42; Satterlee Blake 2018, 38). Consequently, despite

this critique of metric assessment methodologies, they are still typically used to estimate fetal, perinatal and infant age (Scheuer & Black 2000a, 9).

Many of these skeletal reference samples have been developed using archaeological or historical skeletal remains of deceased individuals – who are unlikely to represent healthy non-adults – and where no documented age or sex has been recorded (e.g. Fazekas & Kósa 1978). Needless to say, documented collections, of sufficient size, are rare (Scheuer & Black 2000b, 17; Lewis 2007, 14). Other methodologies have been developed using samples that are geographically and genetically disparate (e.g. Maresh 1943; 1955; 1970; Gindhart 1973), and equally likely to be dissimilar from the archaeological sample being analysed. Clinical studies have developed references from radiographic and sonographic analyses, rather than metric assessment of dry-bone (e.g. Scheuer *et al.* 1980; Jeanty *et al.* 1984; Jeanty & Romero 1984). As a result, limitations exist in comparing archaeological samples to modern reference standards (Hoppa & Fitzgerald 1999, 18). As growth is known to respond to a multitude of intrinsic and extrinsic factors (Saunders & Hoppa 1993; Bogin 1999, 228-239), comparison of archaeological data to that of modern, known samples also increases variability and inaccuracy in results and interpretations obtained. Even when clear growth differences are identified, interpretation of these differences may be numerous (Saunders *et al.* 1993, 266). However, recent assessment of the Maresh (1970) reference data against WHO standards for normal, healthy growth found that this method does reflect a normal growth pattern, and as such can be used as a reference in bioarchaeological studies (Schillaci *et al.* 2012). Thus, choosing an appropriate reference standard is central to analysis and interpretation of non-adult data. Furthermore, reference methods often pool data from both sexes, particularly methods for estimating age in fetal/perinatal individuals (e.g. Fazekas & Kósa 1978; Scheuer *et al.* 1980), meaning variance between standards and individuals assessed may also be increased (Saunders *et al.* 1993, 266). Though Lewis (2007, 21) lists numerous archaeological sites with large sample sizes, indicating the potential for future, multi-sample research, many of these collections are not of fetal or infant individuals. Consequently, studies of the very youngest individuals are still rare and hindered by the lack of comparable data (Loth & Henneberg 2001, 179; Aleman *et al.* 2012, 606; Halcrow *et al.* 2018, 86). Indeed, Lewis states that one of the biggest limiting factors is the lack '*of modern non-adult skeletal collections*' (2007, 14).

2.22 Assessment of Growth and Development in Non-Adult Skeletal Remains

The study of non-adult growth and development has been a major concern in bioarchaeology for a number of decades (Johnston & Zimmer 1989, 11; Hoppa & Fitzgerald 1999, 1; Halcrow *et al.* 2018, 85 e.g. Johnston 1962) – a focus which has dominated non-adult studies for over 40 years (Lewis 2007, 11). Throughout the last two decades, a wealth of studies has emerged considering growth and development (e.g. Gindhart 1973; Scheuer & Maclaughlin-Black 1994; Huda & Bowman 1995; Cardoso *et al.* 2014). Given the previous discussion surrounding the correlation between growth/development and age-at-death estimation, many such studies are now beginning to consider the limitations of using growth as a proxy for age and are instead investigating the environmental determinants that may have affected growth and development (e.g. Gigante *et al.* 2009; Mays *et al.* 2009; Ruff *et al.* 2013; Zemel 2017; Ives & Humphrey 2017).

Despite the acknowledgement that skeletal growth can no longer be used as an accurate and reliable proxy for chronological age, metric assessment has been significant in leading arguments surrounding infanticide, the deliberate disposal and killing of unwanted perinates/infants (Resnick 1970, 1414). Some archaeologists have used age profiles to support arguments for infanticide (e.g. Smith & Kahlia 1992; Mays 1993; Mays & Faerman 2001; Mays & Eyers 2011). Clustering of individuals around 38-40 gestational weeks/birth has become interpreted as an 'unusual' demographic profile and thus considered to represent deliberate killing when large numbers of skeletal individuals have been recovered (Gowland & Chamberlain 2002, 677; Halcrow *et al.* 2018, 86). However, the regression methodology employed within these studies (Scheuer *et al.* 1980) has been critiqued as it has been found to generate age profiles which mimic the sample used to develop the methodology. Gowland and Chamberlain (2002) used Bayesian statistical assessment to redistribute this age-at-death profile and concluded that infanticide was not a plausible interpretation for the sample investigated. The regression methodology employed increases mimicry between reference and sample population (Bocquet-Appel & Masset 1982, 321; Gowland & Chamberlain 2002, 678), thus, a similar neonatal peak emerged from analysis of the archaeological sample (Mays 1993; Mays & Faerman 2001; Mays & Eyers 2011) as that seen in the method utilised (Sheuer *et al.* 1980). Bayesian analysis considers the likelihood of individuals falling within age categories, in comparison to a natural mortality profile derived from perinatal and infant life tables (Lewis & Gowland 2007, 122), and so redistributes ages by probability (Gowland

& Chamberlain 2002, 684). Indeed, Bayesian analysis is likely the best way to attempt to access 'true' mortality profiles for fetal/perinatal/infant populations (Lewis & Gowland 2007, 127). However, Bayesian analysis is also limited in its application here in that it considers the age distribution of the entire sample rather than generating age estimates for each individual (Lewis & Gowland 2007, 127). This limits detailed assessment of individuals and the potential for identifying growth disruption. Recent reconsideration of the skeletal remains from Ashkelon, Israel – another site where infanticide has been widely considered to have been practiced (Smith & Kahlia 1992) – has revealed that the original age estimations were incorrect (Gilmore & Halcrow 2014). As a result, fetal individuals, whose age-at-death estimates suggest they were unlikely to have survived in the postnatal environment, were also considered to be victims of infanticide within the original investigation (Halcrow *et al.* 2018, 94). Indeed, it has been demonstrated that as bioarchaeologists we need to be extremely careful not only with the methodologies we employ, but the subsequent interpretations we derive.

Furthermore, though age-at-death assessment is important for our understanding of mortality and morbidity profiles, it reveals very little regarding the potential birth point and experience. Although birth is standardly considered to be around the 40 gestational week mark (Satterlee Blake 2018, 35), maternal and offspring experiences can be radically different with birth possible at any point during a large window of time. 'Full-term' birth can happen at any point between 29 and 40 gestational weeks, depending on discrepancies in due date calculation, though most births occur between 38 and 39 gestational weeks (Jukic *et al.* 2013, 2850). Therefore, determining those who experienced premature/overdue birth, stillborn births, intrauterine growth restriction (IUGR) or were small for gestational age (SGA) is complex, as this 'lived' experience is almost impossible to discern from skeletal remains in the archaeological record. Today both premature (before 37 gestational weeks (Franco *et al.* 2007, 518)) and overdue births still occur (Halcrow *et al.* 2018, 93). Though overdue births are more uncommon today due to medical intervention and induction (Satterlee Blake 2018, 35), premature births have a high and increasing prevalence (Ong *et al.* 2015, 983). In fact, males in particular appear to be particularly vulnerable to growth disruption as a result of preterm birth (Haymond *et al.* 2013, 789). However, such experiences are rarely considered in an archaeological context. The point of birth is not recorded on skeletal remains, though can be traced through identification of the neonatal line using histological analysis of the

dentition (Satterlee Blake 2018, 36). As yet, no study has correlated evaluation of the neonatal line with dental and skeletal estimates of age to investigate evidence of prematurity but this is certainly an interesting research prospect.

Within the skeletal record new methodological approaches (e.g. Booth 2016; Booth *et al.* 2016) are beginning to investigate the potential to differentiate between live births and those of stillborn individuals. This research into the bioerosion and diagenesis of internal bone structures suggests that bone composition changes following feeding and the initiation of the gut microbiomes (e.g. White & Booth 2014; Booth 2016; Booth *et al.* 2016). Consequently, individuals who fail to show changes in bone composition, either through histological or micro-CT analysis, are suggested to have not fed and are thus more likely to have been stillborn (Booth *et al.* 2016, 132). This research still has limitations in that it is unknown as to when the infant gut microbiome is 'activated', which factors can lead to this activation, and the effects of differential food intake on the microbiome (Booth *et al.* 2016, 125). Furthermore, this method also cannot distinguish between deliberate avoidance of feeding and an inability to feed. However, such research is starting to further discussion surrounding the timing and outcomes of birth, and will further aid interpretations surrounding that of infanticide.

The use of non-adult terminology, and the sub-division of very young individuals into multiple categories (e.g. fetus, perinate, neonate, infant) which often overlap, adds to the complexity of determining not only true chronological age, but birth experience and outcome (Scheuer & Black 2000b, 9; Satterlee Blake 2018, 35). As an example, individuals who are recorded as being 36 gestational weeks or younger are often referred to as a fetus, however, such terminology implies that they were not born (Halcrow *et al.* 2018, 84). In fact, it may be that they were premature, SGA, IUGR or stillborn, yet currently interpretations are limited in considering these aspects (Halcrow *et al.* 2018, 84). In part this thesis has attempted to minimise this potential uncertainty by using a larger perinatal range than standard practice (36-44 GWA). This was a deliberate action as those dentally aged to be less than 36 GWA, although may have been born prematurely, were unlikely to have survived in the postnatal environment. Similarly, those aged over 44 GWA are more likely to have experienced the postnatal environment to some degree. The complexity arises for those individuals aged dentally to be between 36 and 44 gestational weeks, where their pre- and postnatal

experiences are more ambiguous. However, this thesis uses terminology in a way that attempts to reflect better the developmental and life course stages that the individual experienced.

Utilising both dental and skeletal tissues to examine age is one potential avenue in which to begin exploring SGA and IUGR individuals. Small for gestational age typically refers to individuals whose birth weight falls below the 10th percentile for their gestational age (Bogin 2001, 69; Royal College of Obstetrician & Gynaecologists 2014, 6), but who are overall considered to be healthy and well-nourished (Cyrkowicz & Czekański 1998, 213). SGA babies are those who are genetically 'small' (Cyrkowicz & Czekański 1998, 213), but are considered to be appropriate size given maternal size and ethnicity (Royal College of Obstetrician & Gynaecologists 2014, 6). Furthermore, SGA individuals are considered to be 'symmetrical', where skeletal and physical dimensions are consistently smaller purely as a result of a slower fetal growth rate (Strauss & Dietz 1997, 96; Hay *et al.* 2016, 881). In contrast, IUGR causes asymmetrical growth (Strauss & Dietz 1997, 96; Cameron & Demerath 2002, 162), with variation between skeletal structures due to prioritisation of certain elements, particularly that of the brain (Hay *et al.* 2016, 881). IUGR is often a consequence of *'…a pathological restriction of the genetic growth potential*' (Royal College of Obstetrician & Gynaecologists 2014, 6). Consequently, IUGR is strongly correlated with fetal malformations, infections and placental dysfunction (Cyrkowicz & Czekański 1998, 213; Kliegman 2011, 245; Wu *et al.* 2012, 18-19). Wu and colleagues (2012, 18-19) state that IUGR is responsible for ~50% of non-malformed stillbirths. In addition, IUGR often has implications for the rest of the life course, predisposing individuals to sarcopenia, insulin resistance and diabetes (Hay *et al.* 2016, 881). Of course, SGA and IUGR are strongly correlated though not always synonymous with each other (Kliegman 2011, 245; Royal College of Obstetrician & Gynaecologists 2014, 6; Ong *et al.* 2015, 974). The Royal College of Obstetrician & Gynaecologists (2014, 6) report that 50-70% of SGA babies are constitutionally (genetically) small and are thus, not IUGR. Despite this, both SGA and IUGR individuals are at an increased risk of perinatal/infant mortality, though those suffering from IUGR are of an even greater risk as they suffer more adverse intrinsic and extrinsic experiences (Bogin 2001, 69; Wu *et al.* 2012, 18; Royal College of Obstetrician & Gynaecologists 2014, 6; Schoenwolf *et al.* 2014, 178).

Attempting to identify SGA and IUGR individuals in the archaeological record will require detailed assessment of growth and health. Consideration of age estimations between dental and skeletal structures may allow for similarities and differences between these estimates to be highlighted, potentially exposing variability in growth strategies. Of course methodological limitations of assessing age-at-death, particularly within the skeleton, will hinder analyses of SGA individuals, as the inherent biases within the methods will not reflect individuals who are simply, genetically small. Instead, to access evidence of SGA individuals, consideration of the health status and uniformity in age estimates generated from varying skeletal structures will have to be made. However, lack of pathological changes does not necessarily indicate good health and interpretations of SGA may also have to consider wider population based assessment of size and stature, particularly regarding females within the sample. IUGR is potentially less problematic to identify, with evidence of growth and health disruption possible indicators of such a condition. For now, interpretations of IUGR are perhaps most robust when extremely large differences in skeletal/dental age estimates are identified in individuals, and where clear pathological conditions have been determined. The ability to identify SGA and IUGR within the archaeological record would be significant in furthering interpretations regarding maternal wellbeing and health.

2.23 Palaeodemographic Studies of Non-Adult Skeletal Remains

Age-at-death estimations have also become paramount for studies concerned with fertility, methods in which to quantify fertility, and the impact of fertility rates on environmental adaptation (Lewis 2007, 10; e.g. Buikstra *et al.* 1986; Bogin 1990; Paine & Harpending 1996; 1998; Robbins 2011). These studies arose from a demographic concern to identify and evaluate population structures in prehistory (Brothwell 1971; Scheuer & Black 2000a, 13; Lewis 2000, 39; Lucy 2005, 44; Robbins 2011, 717), with major considerations including the energy expenditure of parturition, childcare and the social and economic benefits of offspring (e.g. Bogin 1997; 1998; Key 2000). Furthermore, infant mortality has also become a palaeodemographic tool, where-by population success and ability to adapt to environmental challenges can be interpreted (Scheuer & Black 2000aa, 5; Lewis 2000, 39; Lewis & Gowland 2002, 125; Halcrow & Tayles 2011, 339). Bogin (1990; 1998) has also investigated the development and evolution of ‘childhood’ as a socially constructed period in which we learn and develop the skills required for adult life.

2.24 Sex Estimation in Non-Adult Skeletal Remains

Sex estimation of non-adult individuals has always been acknowledged to be difficult and unreliable (Hoppa & Fitzgerald 1999, 2; Scheuer & Black 2000a, 1; 15; 2000b, 9; Lewis 2007, 47; Satterlee Blake 2018, 41). The skeletal biology of non-adult individuals is the primary limitation of this assessment, as individuals have not yet fully grown or even developed all of their skeletal structures. Furthermore, the hormonal influxes that alter the morphology of these structures are yet to occur (Hoppa & Fitzgerald 1999, 2; Scheuer & Black 2000a, 15; Lewis 2007, 47). As a result, macroscopic assessment of sex estimation in non-adult individuals is often avoided. However, attempts to determine biological sex based on the dimorphism of the skeleton have been made. Typically, methodologies have utilised similar skeletal structures to those analysed in adult individuals – primarily the bones of the pelvis and cranium (Scheuer & Black 2000a, 15; e.g. Boucher 1955; 1957; Weaver 1980; Schutkowski 1993; Loth & Henneberg 2001; Wilson *et al.* 2008) – however, other attempts have investigated variation in the humerus and dentition (e.g. Black 1978; Rogers 2009; Stull & Godde 2013).

Biological sex is an important factor to consider, particularly in studies of fetal, perinatal and infant individuals as sex differentially affects growth/development (Scheuer & Black 2000a, 4; 15; e.g. Gilsanz *et al.* 1997; Nyati *et al.* 2006f) and frailty (Lewis 2007, 48; Satterlee Blake 2018, 40) – predisposition to health/disease status. Consequently, it is a vital factor which is currently missing from many of our interpretations, this thesis included. The inherent problems and inaccuracy of these methods makes estimating sex for such young individuals a game of chance. Studies testing methods of sex estimation have found that dimorphic traits can vary between populations, that the accuracy of these methods could not be reproduced, and in one case, that sex was only estimated correctly in 64% of individuals (Hunt 1990; Scheuer 2002; Cardoso & Saunders 2008; Vlak *et al.* 2008). In addition, there is substantial overlap between the categories of males and females (Satterlee Blake 2018, 41). Furthermore, given the limitation of estimating age, and subsequently determining stillborn, SGA and IUGR individuals, it is unknown what further complexities/changes these experiences add to the sexual morphology of the skeleton. Today, scientific advances have led to the emergence of studies considering DNA analysis to determine biological sex of infants and children (Hoppa & Fitzgerald 1999, 3; Aiello 2000, VII; Lewis 2000, 40; e.g. Waldron *et al.* 1999; Mays & Faerman 2001). Of course, this has been used to investigate arguments surrounding

infanticide (as more females are typically thought to be 'disposed' of than males). However, Mays & Faerman (2001, 556) found a prevalence of males within their archaeological sample. Given the predisposition for males to be more 'frail' (Lewis 2018, 113) and their alternative growth strategies *in utero* (Barker *et al.* 2012, 32) such a result may not be unexpected, and again support suggestions that these individuals reflect a normal mortality profile. Within this thesis sex estimation has not been attempted on the archaeological individuals as the methodologies are still too variable and inaccurate. The fetal collection at the Smithsonian has recorded biological sex for some of the individuals assessed (Hunt *personal communication*) and thus, where possible analysis has been conducted using these categories (male versus female). Although the author did record sexually dimorphic traits in accordance with Schutkowski (1993), testing this method on the Smithsonian collection has not been afforded within this thesis as no other sample uses sex estimation. It has been detailed that this is a further study for future consideration.

2.25 Palaeopathological Studies of Non-Adult Skeletal Remains

The first study to consider childhood health and palaeopathological evidence was that of Mensforth and colleagues (1978), but it was not until the 1990s that non-adults were routinely incorporated within archaeological and bioarchaeological literature (Mays *et al.* 2017, 38). Assessing pathological changes in non-adults is considered to be particularly challenging (Lewis 2018, 113). This is because, despite increased osteological training, the identification remains from such young individuals is difficult, and understanding of their normal anatomy and growth limited (Lewis 2018, 113). Furthermore, identifying pathological changes, many of which are often subtle or identical to processes of normal growth, is troublesome (Lewis 2018, 113). Despite this, numerous investigations into non-adult health have been undertaken (e.g. Lallo *et al.* 1977; Schultz 1984; 1989; Anderson & Carter 1994; 1995; Lewis and Roberts 1997; Mays *et al.* 2007; Lewis 2011; 2012; 2017a) – with particular focus on metabolic conditions (e.g. Stuart-Macadam 1988; Ortner & Ericksen 1997; Ortner & Mays 1998; Ortner *et al.* 1999; Brickley & Ives 2008). Pathological changes in such young individuals are considered to be particularly important to determine as they provide vital insights into intrauterine as well as maternal health. Fetal, perinatal and infant remains have become synonymous as the most sensitive members of past societies (Goodman & Armelagos 1989, 239; Cox & Mays 2000, 8; Lewis 2000, 39; Halcrow *et al.* 2018, 86), likely to express skeletal changes as a result stress due to their immature immune systems and

higher degree of bone turnover (Lewis 2000, 43; 2002a, 211; 2018, 115; Satterlee Blake 2018, 44). These individuals also provide a unique proxy by which maternal wellbeing, experiences and health can be considered (Satterlee Blake 2018, 34; 43). Indeed, there remain very few cases where expectant mothers have been uncovered, or where individuals are suggested to have died during childbirth (Hoppa & Fitzgerald 1999, 16; Halcrow *et al.* 2018, 86; e.g. Owsley & Jantz 1985; Roberts & Cox 2003, 389-390). Consequently, despite the limited mother-infant evidence in the burial record, the mother-infant nexus can still be extensively explored (Gowland 2015).

Macroscopic assessment of fetal/perinatal/infant pathology is most commonly employed (Satterlee Blake 2018, 42) and some conditions/diseases are clearly identifiable and diagnosable despite the young age and immature development of these individuals. A variety of congenital disorders (e.g. hydrocephaly, anencephaly) as well as diseases such as syphilis, tuberculosis, rubella, and metabolic deficiencies can all be transmitted via the placenta during pregnancy (Lewis 2018, 113). Therefore, the historical assumption that the intrauterine environment is one of a sterile, encapsulated 'bubble' is not accurate and an array of detrimental conditions can affect the health and wellbeing of the offspring. Many case studies exploring evidence of various congenital and specific infectious conditions affecting non-adult individuals have been published (e.g. Richards & Anton 1991; Murphy 1996; Dabernat & Crubézy 2009; Dudar 2010), though evidence still remains limited, primarily as a consequence of the rarity of such conditions and the limitations of preservation and identification. Additionally, many of the predominant causative factors of fetal/perinatal/infant death leave no discernable traces on the skeleton (Lewis 2018, 112), making assessment of early life health disruption in the archaeological record somewhat more troublesome.

Analysis and diagnosis of a specific condition/infection is easier as a clear pattern of distinctive changes emerge (e.g. Hutchinson's incisors and mulberry molars in congenital syphilis). However, non-specific stress, such as metabolic disturbances, general infections, psycho-social stress and sometimes even trauma, is more commonly represented as periosteal new bone formation (NBF) (Lewis 2018, 114). The ongoing major limitation within pathological studies of such young individuals is the differentiation between pathological NBF and that of NBF associated with normal growth (For a comprehensive discussion of

normal fetal/perinatal/infant skeletal growth and development see Chapter 3: Sections 3.1). Methods detailing how to distinguish between the two have yet to be developed (Lewis 2018, 125), though current practice includes considering the location, aspect, and thickness of the NBF, whilst taking into consideration the age-at-death of the individual. Healthy NBF, associated with normal growth, is known to appear in an almost indistinguishable way to pathological NBF, as the process by which the bone forms is identical (Lewis 2000, 42). Consequently, timing of growth spurts must be taken into consideration, and various clinical investigations have suggested that NBF is a common finding in individuals at least over 1 month of age (44 gestational weeks) (Shopfner 1966; Scheuer & Black 2000a, 24; de Silva *et al.* 2003; Kwon *et al.* 2002). Furthermore, NBF has been found to be commonly identified symmetrically on the femur, tibia and humerus, with the NBF being concentric around the diaphysis (De Silva *et al.* 2003, 1124). However, little is still known about the presence and implications of NBF found on individuals younger than 44 gestational weeks, and many pathological conditions do still result in the proliferation of NBF throughout the skeleton, primarily to the long bones (Lewis 2000, 43). Additionally, although bone responds quickly to insults in such young individuals, it is still unknown how long certain conditions take to present on non-adult skeletal remains (Lewis 2000, 40). As skeletal remains of fetal, perinatal and infant individuals are those of the non-survivors, it is not unexpected that pathological indicators would be present reflecting poor health and wellbeing. However, with so many variables regarding the formation of pathological NBF still unknown (timing, severity, extent), disentangling normal from abnormal bone formation remains problematic.

Recent developments in stable isotopic and incremental dentine analyses are furthering pathological assessment, identifying weaning patterns and early life stress (e.g. Fuller *et al.* 2006; Katzenberg 2000; Beaumont *et al.* 2013). Nitrogen ratios have been found to peak during periods of stress (Beaumont & Montgomery 2016), and by conducting incremental analysis, the exact period/timing of this stress can be assessed which is revealing further implications as to intrauterine and postpartum experiences (Beaumont *et al.* 2015). Thus, being able to correlate age-at-death assessment, pathological assessment and isotopic investigation is building a more intricate picture of early life health and stress exposure (Halcrow *et al.* 2018, 97). Again maternal experiences have become central to this narrative and recently the theoretical inclusion of the DOHaD Hypothesis (Developmental Origins of Health and Disease Hypothesis) has become prevalent within interpretations of the early life

course (e.g. Finlay 2013; Klaus 2014; Gowland 2015). This hypothesis, originating from clinical discourse (e.g. Barker & Osmond 1986; Barker 1997), has major implications for fetal, perinatal and infant studies, highlighting the long-term growth and health outcomes of detrimental *in utero* and immediate postnatal environments (Gluckman & Hanson 2006; Armelagos *et al.* 2009, 261; Halcrow *et al.* 2018, 97).

2.3 The Archaeological and Methodological Issues

Bioarchaeological studies of fetal, perinatal and infant individuals are relatively few in comparison to the wealth of multidisciplinary studies regarding older non-adults and adults (Lewis 2000, 39; Halcrow *et al.* 2018, 83). Although this has clearly been a consequence of the attitude historically held towards these individuals (discussed previously), bioarchaeological studies have also been limited by the recovery and excavation of such individuals, the difficulty of identification and analysis, and the methodological limitations of determining age-at-death, biological sex and evidence of pathological changes. As Lewis states, in order to enhance the discipline of fetal, perinatal and infant studies we must first understand the potential and limits of studying non-adult remains (2007, 1). This section discusses these limitations and considers the implications these factors have had on fetal, perinatal and infantile studies.

2.31 Recovery and Excavation

It has long been considered that non-adult remains did not survive the burial environment as well as adults, and that those of infants rarely survived at all (Watts 1989, 377; Scheuer & Black 2000a, 14; Lucy 2005, 44; Lewis 2007, 10-12; 20; Satterlee Blake 2018, 45). Absence of fetal/perinatal/infant remains was considered commonplace and some even considered that such young individuals completely dissolved in the ground (Angel 1969, 434; Lewis 2007, 20). Perpetuation of these views has limited bioarchaeological investigation of these individuals – not only considered to be of less importance, but quite literally, invisible within the burial record - furthering the notion that assessment of these individuals was not only futile, but impossible. Buikstra and Cook (1980) argued that although the importance of non-adult studies was being recognised, poor preservation and recovery, and thus small sample sizes, was hindering analysis and interpretation. However, Katzenberg (2000) reported that, at some sites, non-adult remains can in fact be better preserved than those of their adult counterparts. As a result, it is becoming more widely accepted that the notion of non-adult

skeletal remains not surviving the burial environment is unfounded (Lewis 2007, 37; e.g. Saunders 1992; Buckberry 2000; Hillson 2009). Furthermore, numerous excavations have now been conducted, and continue to be undertaken, from which large samples of non-adult individuals have been carefully excavated and recovered (Lewis 2007, 37).

It has traditionally been considered that fetal/perinatal/infantile remains preserve differentially to that of adults (Hoppa & Fitzgerald 1999, 16; Scott 1999, 109; Scheuer & Black 2000a, 14; 2000b, 10). Guy and colleagues (1997) argued that the physiochemical properties of fetal/perinatal/infant bones were the leading cause as to why they preserved differently. Of course, no one factor is responsible for the level of bone decomposition or preservation, instead multiple factors determine preservation: chemistry, size, shape, density, porosity, age of the bone, groundwater, soil type, temperature, oxygen levels and flora (Scheuer & Black 2000b, 10; Stodder 2008, 81-85; Jackes 2011, 126; e.g. Gordon & Buikstra 1981; Garland & Janaway 1989; Millard 2001). Bone is typically considered to decay in a three-phase process (See Collins *et al.* 2002 for further discussion). Despite this, studies have found it possible to extract DNA, stable isotope signatures and proteins from non-adult skeletons (Mays *et al.* 2017, 43).

Indeed, true understanding of taphonomic processes on non-adult remains is still somewhat limited. Although new research (e.g. Booth *et al.* 2016) is considering the differential diagenesis of bone in burial conditions, and the implications of such, understanding the effect of various factors on fetal, perinatal and infant remains is ongoing. It is considered that infants who have had their first feed, compared to those who have not, will decompose more quickly due to bacteria introduced into the digestive biome (Booth 2014; Booth *et al.* 2016). However, decomposition has also been found to vary depending on age. Bone mineral content varies with age (Guy *et al.* 1997, 224), with the lowest levels found in those aged between one month and one-year-old (Guy *et al.* 1997, 224; Lewis 2007, 25). However, infants contain less trabecular bone than adults and older non-adults, which is known to decompose more rapidly than cortical bone. Additionally, lack of forensic knowledge on how such young individuals decompose today also hinders understanding, as the rarity of cases, particularly those that are buried, means very few studies have been conducted (Lewis 2007, 23). Consequently, funerary treatment and mode of disposal will also differentially affect the

preservation or existence of infant remains within the archaeological record (Roberts 2009, 60-61), which will influence the success of excavation and recovery.

Given both the size and structure of the fetal and infant skeleton it is understandable that such individuals become disarticulated more easily (Lucy 2005, 44). As a consequence of their fragility and size, their remains are more likely to be found scattered or dispersed within an archaeological context. This is in part due to scavenging practices, with once again their small size, making them a prime target for animals (Lewis 2007, 27-28). Scavenging is also likely to be a consequence of the shallower graves in which fetal and infant individuals tend to be buried – it is considered the deeper the grave cut, the more likely an individual is to be well preserved and recovered (Lucy 2005, 44). Crawford (1993) has considered shallow graves to be a practical and logistical consequence of burying a small individual.

In addition, shallow graves are also more susceptible to erosion, ploughing, and post-mortem taphonomic and stratigraphic changes (Johnston & Zimmer 1989, 12; Lewis 2000, 40). Such factors affect the excavation and recovery of these individuals. Furthermore, fetal, perinatal and infant individuals were commonly buried/deposited in alternative parts of sites and cemeteries to that of older individuals and adults (Scott 1999, 109; Lucy 2005, 44; Halcrow *et al.* 2018, 83; Satterlee Blake 2018, 45), often as a consequence of culturally and socially regulated practices as well as biological parameters. When open area excavation is not undertaken it is difficult to ascertain whether all individuals, particularly those of fetal/infant individuals have been fully recovered (Saunders & Barrans 1999, 184; Scheuer & Black 2000b, 9), making skeletal individuals only a subset of a once living population (Scheuer & Black 2000a, 14). As a consequence, it cannot be considered that individuals exhumed and curated represent a true random sample of a population, and thus may not reflect an accurate representation, with regards to both biological and contextual variables (Waldron 2007, 26).

Furthermore, the ability of the excavator to recognise, excavate and collect fetal and infant bones is also paramount (Scheuer & Black 2000a, 14; 2000b, 11; Lucy 2005, 44; Lewis & Gowland 2002, 125). The recovery of smaller skeletal elements, epiphyses and developing bones and teeth relies on a prior knowledge of what to potentially expect when excavating non-adult individuals. In addition, the porosity of these elements means that they often change colour, reflecting chemicals and nutrients in the soil and are thus more easily missed

by a non-specialist excavator (Lewis 2000, 40; 2007, 26). Sieving, despite being recommended within archaeological guidelines (e.g. BABAO Code of Practice), is often not employed due to time pressures of excavation, and so many individuals are likely to remain 'invisible' as a result of archaeological practice (Roberts 2009, 59). Furthermore, lack of specific skeletal recording forms, listing/picturing the potential skeletal elements, means that many excavators are simply unaware and unsure of what to look for (Lewis 2007, 26). Excavation and recovery is also typically hindered by the financial and time constraints of commercial operations (Roberts 2009, 73-80).

2.32 Identification and Analysis

Many texts now thoroughly cover non-adult skeletal anatomy and detail precisely the very small skeletal elements expected to be present before, during and after birth (e.g. Fazekas & Kósa 1978; Scheuer & Black 2000a; Baker *et al.* 2005). Despite this, many individuals, even though excavated and recovered, are sent to faunal specialists because they are mistaken for birds and rodents (Scheuer & Black 2000a, 1; Lewis 2007, 26). Furthermore, those infants who are recognized as human are often accumulated into disarticulated remains bags (e.g. Buckberry 2005). Obviously, analysis of disarticulated human remains is limited due to the nature of the material, though much disarticulated material often remains unassessed (Lewis 2007, 30). Furthermore, a lack of knowledge regarding human anatomy, especially that of non-adults, limits their assessment (Lewis 2018, 113), meaning few actively choose to study these individuals. In addition, it must be acknowledged that depending on the age of the individual, the number of potential bones to be recovered varies; fetal, perinatal and infant individuals have around 263 bony elements (Lewis 2007, 27). Consequently, the number of bones and their rapidly changing morphology can make identification of fetal, perinatal and infantile individuals particularly difficult.

Analysis is hindered by the obvious caveat of all osteological studies – that the individuals studied are the 'non-survivors' of a given population (Wood *et al.* 1992, 344; 349; Lewis 2000, 40; Jackes 2011, 108; Halcrow *et al.* 2018, 93; Satterlee Blake 2018, 40). Consequently, analysis of the biological parameters of age and sex, as well as investigation into pathological changes, is thus not representative of the survivors (Saunders and Hoppa 1993, 128). Therefore, peaks in mortality around 40 gestational weeks of age should not be unexpected; both the birth process and the first few days following are considered to be the

most critical point in an individual's life (Kelnar *et al.* 1995 cited in Halcrow *et al.* 2018, 93; Lewis 2018, 112). Lewis (2018, 112) states that contemporary estimates suggest '…*four million babies die each year worldwide within the first month of life*…'. Furthermore, such individuals are likely to reflect those of greater frailty and with increased health disruption. Today the biggest risks include preterm birth, low birth weight, infection and asphyxia (Lewis 2018, 112; WHO *Newborns: Reducing Mortality*; WHO *Preterm Birth*). Given the limitations and ongoing debates (outlined in section 2.2. Contemporary Research and Current Debates), biological and pathological assessment is particularly complex when considering non-adult remains and interpretations should be carefully constructed and considered.

In addition, the ongoing, inconsistent application of terminology hinders the ability of many studies to be comparable. The non-standardised use of fetus, perinate, neonate, infant, child, juvenile, sub-adult and non-adult, often with a lack of definition for such categories, means individuals are often ascribed to socially constructed age categories, imbued with cultural and social connotations of what that individual should have been (Sofaer Derevenski 1997, 193; Gowland 2006, 144). Thus, terminology employed must always be clearly defined and clarified, with consideration as to the historical, social and cultural implications of these age categories explored.

2.4 Ethical Considerations

Many ethical considerations surround the excavation, collection and analysis of human remains (e.g. Fforde 2004; Sayer 2010). Although many of these concerns centre on the display of individuals within a museum setting to the general public (e.g. Barilan 2006), consideration must be given to the large number of skeletal individuals curated within museums and academic departments (Fforde 2004, 2; Márquez-Grant & Errickson 2017, 193). The collection of human remains, and the origins of large museum collections, have come under scrutiny in recent years (Nilsson Stutz & Tarlow 2013, 7). Of course some envisage human remains as simply material objects, primarily concerned with their scientific value, whilst to others, human remains are imbued with religious, cultural and/or spiritual significance (Nilsson Stutz & Tarlow 2013, 7). The primary issue surrounding these individuals is the rites of the deceased, and whether we as researchers should be able to prioritise our own scientific motivations over those of individuals of the past (Márquez-Grant & Errickson 2017, 193).

Historically, the collection of human remains was to investigate 'race' and studies focused on determining morphological variation between individuals (Fforde 2004, 1). As a consequence, the last 30-years has seen indigenous groups campaign for the return of ancestral remains held within various institutions (Fforde 2004, 2-3). Wide scale repatriation programs (e.g. NAGPRA) have ensured that indigenous groups now have legal protection over the remains of their ancestors (DeWitte 2015, 12), once curated and housed in various museums, some of which are being systematically repatriated. However, within archaeology, where many human remains are excavated as part of rescue excavations, and would be destroyed otherwise, the recovery and curation issues become more complex. Indeed, it has been considered to be paramount that archaeological human remains are accessible to study, so that biological, social, and cultural information regarding past and present populations can be comprehended (Márquez-Grant & Errickson 2017, 194). However, skeletal remains are those of once living people and should be treated as such – the study of human remains is a privilege and not a right (BABAO Ethics and Standards). For a comprehensive review of ethics in bioarchaeology see Walker (2000).

Fetal, perinatal and infant studies are limited with regards to the lack of modern comparative skeletal collections. Donation of non-adult remains to medical study is vital to assess the skeletal and overall bodily mechanisms that have been affected by certain conditions and diseases. Parents are often, understandably, unwilling to donate the bodies of their infants and children to medical science (Lewis 2007, 14). This has been further diminished by the unethical practices, which were undertaken at the Royal Liverpool Children's Hospital (Alder Hey), where pathologists had routinely collected and stored organs, without permission, for research purposes (Carvel 2002, 55; Swain 2002, 95). Although it may be argued that the intentions were not malicious, such practices are entirely unethical.

Consequently, when studying the remains of fetal, perinatal and infant individuals it is imperative to consider the context from which these individuals were recovered and whether consent was given by parents and guardians. Individuals from the Smithsonian Fetal Collection were collected from medical institutions in the early 1900s. Although it must be stressed that this is unclear, it is possible that many of these individuals may have been collected without the knowledge or permission of the parents/family. As a researcher this

collection provides unrivalled opportunity to analyse a ‘known’ sample; however, this study does not overlook the possibility that collection and curation of these remains may be considered contentious.

This study does not attempt to investigate the ethical issues surrounding each of the samples analysed within this thesis, but does ensure that ethical guidelines (BABAO Ethics and Standards) were adhered to in undertaking this assessment. Consequently, the utmost care has been afforded when examining all of the very young and fragile individuals considered within this assessment. It must not be forgotten that these were once living individuals, and despite their short lives, deserve our highest regard and respect.

Chapter 3: The Beginnings of Life; Understanding Growth, Health and Stress in a Bioarchaeological Context

This chapter aims to contextualise the use of the terminology surrounding growth, health and stress throughout this study and provide a detailed background as to the main concepts behind these processes. This chapter explores the principal concepts of human growth and development, centring discussion on the cranium, long bones and dentition during the early stages of life – those of the fetus, perinate and infant. An overview of skeletal growth, its mechanisms and processes is provided, alongside consideration of the ways that bone growth can be disrupted and altered as a result of both intrinsic and extrinsic factors. Furthermore, consideration of the terminology surrounding age, and the distinctions between chronological, biological and physiological age are discussed. Detailed consideration of stress, health and wellbeing is also provided, in particular considering how assessment and interpretation of skeletal pathology can aid our interpretations of past health and wellbeing.

3.1 Introduction to Human Growth

Ontogeny of the human body and its various systems is complex, and understanding the different processes and mechanisms behind development and growth is crucial. However, it must first be acknowledged that growth and development are not interchangeable terms and instead define fundamentally diverse, yet interrelated aspects of human ontogeny (Bogin 2001, 64). Growth is defined as the *'progressive, incremental changes in size and morphology, such as increase in bone length'* over time (Šešelj 2013, 38). That is the fundamental process by which bone dimensions change as a result of increasing physiological progression. Contrastingly, development is considered to be the process by which the skeletal system is considered to 'mature'; the development of epiphyseal centres of ossification, fusion of skeletal elements, or morphological changes associated with sex-related maturation (i.e. puberty) can be defined in terms of development (Bogin 2001, 64; Šešelj 2013, 38).

Anatomically modern humans have been acknowledged as having a uniquely long period of infancy/childhood (Hoppa & Fitzgerald 1999, 8; Bogin 2001, 102; Kuzawa *et al.* 2014, 13010), a period of heightened dependency on older individuals within the community/population (Bogin 2001, 107; Said-Mohamed *et al.* 2018, 6). This lengthy process has been considered to be a result of our unique growth curve (Bogin 1998, 61),

which enables individuals to learn a range of complex social, cultural and behavioural skills (Bogin 2001, 102; Said-Mohamed *et al.* 2018, 6). Thus, growth and development are not merely biological process, but also relate to the maturing of individuals over the life course. Today we ascribe people into socially constructed age categories using not only their stature (growth/height) but their ability to undertake or meet certain criteria (e.g. ability to crawl, walk, talk, vote, drive a car). In addition, we visually see the passing of time through physical manifestations such as wrinkles and grey hairs (Sofaer 2011, 385). The way in which we socially construct and determine age is consequently also strongly correlated with key transitional points in our life course, and the physical study of growth and development often classifies individuals into developmentally functional stages (Bogin 1999, 54; 2001, 64) – those of infant, child, adolescent and adult.

There is an underlying mechanism of all growth, which, regardless of species, is universal (Gesell 1928, 5). Therefore, humans (*Homo sapiens),* although each individual is unique, typically conform to certain biological processes, growth being one such process. Put simply, all humans, although genetically unique, grow according to the same standardised pattern through a series of common biological processes (Tanner 1978, 7; Hillson 2005, 207; Cameron 2012, 20). Indeed, growth is one of the most fundamental processes of life where all living organisms are subject to both the necessities and limitations of it (Gesell 1928, 1). That is not to say that we all grow identically, rather the 'blue print' for human growth is comparable between us all. However, the biology of human development is highly complex and growth manifests in multiple ways, particularly during the fetal, perinatal and infant stages of the life course.

The prenatal period has been described as the most '*spectacular phase of growth*' (Sinclair 1985, 3), both in terms of growth rate and due to the range of structures growing. It is also one of the most fragile and influential periods in an individual's life span, vitally important to future well-being (Tanner 1978, 37; Lejarraga 2012, 40; Sandman *et al.* 2016, 230). The prenatal, or intrauterine, period is typically divided into three trimesters, each of roughly three months' duration (Bogin 1999, 55; 2001, 65; Schoenwolf *et al.* 2014, 1). During these trimesters there are four main stages to prenatal growth: fertilisation, implantation, gastrulation and embryogenesis.

Human growth/development begins with the fertilisation of the ovum, a single large cell, in which the cell membrane of a sperm fuses with the cell membrane of an oocyte (egg) (Bogin 1999, 18; 2001, 64; Sadler 2014, 11; Schoenwolf *et al.* 2014, 14). When fertilised the egg divides into two cells, with each daughter cell containing half of the parent cell material (Sinclair 1985, 3-4; Schoenwolf *et al.* 2014, 14). The fertilized egg (zygote) spends four to five days moving down the fallopian tube before passing into the uterine cavity where it implants in the wall of the uterus; it is during this time that the cells divide steadily so that by the time it implants, the blastocyst constitutes around 150 cells (Tanner 1978, 37; Gluckman & Hanson 2005, 29). It is during this division process (mitosis) that the chromosomes become fundamental to our successful growth and development, replicating themselves to ensure that each daughter cell contains the same genetic information as the parent cell (Tanner 1978, 26; Bogin 2001, 65). It is from this ovum cell and its subsequent initial division that the entirety of the human form develops, an estimated total of 10^{12} cells (Sinclair 1985, 3; Bogin 1999, 18; 2001, 65).

Once implantation has occurred, the blastocyst divides into a bilaminar disc (two-layered disc) (Scheuer & Black 2000a, 32). The outer layer of the blastocyst is termed the trophoblast, and along with the endometrium (uterine wall) forms the placenta (Gluckman 1997, 153; Gluckman & Hanson 2005, 29; Sadler 2014, 38; Schoenwolf *et al.* 2014, 14). The placenta has two primary functions; firstly, the placenta is the structure through which all nutrients and waste will be exchanged (Hahn 1972, 1000; Slack 1991, 16; Sandman *et al.* 2016, 230), and secondly the placenta in central in the production of hormones (Sadler 2014, 98). The inner layer of cells of the blastocyst (embryoblast) go on to from the embryo proper (Sadler 2014, 38; Schoenwolf *et al.* 2014, 14). It is at this point that another small group of cells separates and develop into the amnion (Schoenwolf *et al.* 2014, 16). The amnion is the membrane which forms the embryonic fluid-filled sac which protects and cushions the fetus.

The third phase of growth/development *in utero* is when the embryo undergoes the process of gastrulation. This is when the embryo forms a trilaminar (three-layered disc) and establishes three germ layers: the endoderm, ectoderm and mesoderm (Scheuer & Black 2000a, 32; Bogin 2001, 65; Sadler 2014, 55; Schoenwolf *et al.* 2014, 7). These three layers each give rise to specific tissues and organ systems (Scheuer & Black 2000a, 32; Bogin 2001, 65; Sadler 2014, 67; Schoenwolf *et al.* 2014, 7). The mesoderm also combines with trophoblastic

tissue to from the umbilical cord, connecting fetus to placenta. The process of cell specialisation is also referred to as histogenesis (Tanner 1978, 39). It is during histogenesis that embryogenesis, the last phase of development, occurs, causing tissues such as organs, muscles, nerves and skin, and regions of the body such as head, arms and legs begin to become identifiable (Tanner 1978, 39; Sadler 2014, XII; 3). This moulding of areas of the body into distinguishable shapes, normally completed by the 8th week gestational week, is called morphogenesis and occurs through differential growth and migration of cells (Tanner 1978, 39). During histogenesis, cell division becomes subject to controls which maintain the correct balance of growth and determine when and where further division and specialisation can occur (Sinclair 1985, 17). It is during the period of embryogenesis that most malformations occur, though the initial periods of fertilisation and implantation are equally hazardous. Ten percent of fertilized ova fail to implant and of those that manage to implant 50% are spontaneously aborted (Tanner 1978, 38; Bogin 2001, 68). Such abortions often happen without any knowledge of the mother and are a result of genetic and chromosomal abnormalities, highlighting how complex and intricate the process of growth is, and how reliant human survival is on the optimal conditions for growth and development. Of ova with genetic abnormalities 90% to 95% are spontaneously aborted (Tanner 1978, 38).

The fetal period is classified from the 8th/9th gestational week (Sadler 2014, 3; 89) and is characterised by two distinct processes. The first is the rapid rate of growth of all tissues, with the second being the continued differentiation, specialisation and maturation of cells (Sadler 2014, 89; Schoenwolf *et al.* 2014, 169). Throughout the life course the skeleton experiences various growth spurts, the first of which is during intrauterine life (Sinclair 1985, 3; Gluckman 1997, 153; Bogin 2001, 4). Fetal growth is greatest from the 8th to 16th gestational week during which the fetus will undergo a twenty-five-fold increase in weight (Cameron 2012, 7). Growth in height reaches its peak velocity during the fourth, fifth and sixth gestational months (second trimester) of fetal development (Cameron 2012, 7-8; Sadler 2014, 89; Schoenwolf *et al.* 2014, 169), when the average amount of growth is 1.5 mm per day (Sinclair 1985, 23; Bogin 2001, 4). Therefore, any stress present during these periods is likely to significantly affect function and growth. In the third trimester, growth of the individual slows (Tanner 1978, 41) as its increase in size constricts blood flow and the fetal-maternal '*exchange of nutrients, gases and wastes*' due to pressure it places against the surface of the uterus and placenta (Bogin 1990, 18).

Immediately after parturition, growth increases once more, with average infant body length increasing by 50% within the first year (Sinclair 1985, 26; Saunders & Barrans 1999, 183; Bogin 2001, 77). This heighted rate of growth extends until around three years of age, but gradually decelerates throughout this period (Karlberg 1987, 185; Bogin 2001, 4; Haymond *et al.* 2013, 787; Šešelj 2013, 38). Growth is not seen to accelerate again until puberty (Bogin 2001, 87; Hillson 2005, 207, Lewis 2007, 60; Larsen 2015, 8). Consequently, the rapidity of growth and development within the pre- and postnatal period is an essential component as to why these individuals best reflect physiological adversity. Indeed, the physiological growth of non-adults is considered to be the most sensitive indicator of the social, economic and political environments experienced in the past (Johnston & Zimmer 1989, 13; Lewis 2007, 60). The growth curve for post-parturition exactly mirrors that of the fetal growth curve (Bogin 2001, 4), with a rapid increase in growth and then a steady decline. This means that both second trimester fetal life, and early infancy, are the two most critical periods, during which exposure to stress is likely to be most detrimental to growth and health. In humans the correlation coefficient between length at birth and adult height is only around 0.3, but by the time the individual is two years of age the coefficient rises to nearly 0.8 (Tanner 1978, 43), supporting the notion that it is these early periods/years of development that most influence the rest of the life course.

3.2 The Growth and Development of Skeletal Tissues

The skeletal system develops from both the paraxial mesoderm, lateral plate mesoderm and from the neural crest (Sadler 2014, 125). Bone development proper begins *in utero,* around twelve gestational weeks (Lewis 2007, 61), and just as with postnatal bone growth, the prenatal skeleton can be formed in one of two ways: endochondrally (from hyaline cartilage models) or intramembrously (Scheuer & Black 2000a, 18; Oestreich 2008, 1; 20; Waldron 2009, 12; White *et al.* 2012, 37; Sadler 2014, 125). Flat bones of the cranium and long bones of the limbs differ in their growth mechanism (intramembranous and endochondral ossification respectively) (Schultz 2001, 116; Waldron 2009, 12; White *et al.* 2012, 37). However, although some bones grow purely via one method (intramembranous or endochondral growth), many bones arise from an amalgamation of both processes (Oestreich 2008, 1). Currently, it is still unknown as to why some bones develop from cartilaginous models, whilst others develop within a membrane (Scheuer & Black 2000a, 18).

Bone growth is typically to considered to be regulated by both the paracrine and endocrine systems (Karsenty & Kronenberg 2003, 120; Haymond *et al.* 2013, 787), which control various hormonal influences, affecting key biological process, including both bone modelling and remodelling (Cornish & Martin 2003, 217). Consequently, hormones provide a system of control and regulation for normal human growth and development (Bogin 1999, 353; Stevens & Williams 1999, 200; Helfrecht *et al.* 2017, 1). Major hormonal groups involved in this regulation include thyroid hormones, adrenal hormones, insulin-like growth factors, insulin, cortisol and growth hormones (Karlberg 1987, 191; Stevens & Williams 1999, 200).

Growth hormone (GH) is particularly important in later gestation and postnatal life and is the principle force behind skeletal growth during these periods (Karsenty & Kronenberg 2003, 121). Secretion of GH is considered to be permanently reduced by maternal undernutrition, consequently causing GH deficiency and a disruption in skeletal growth (Barker 1994, 27). GH is also important for the regulation of insulin-like growth factors (IGF) (Karsenty & Kronenberg 2003, 121). IGF are also important regulators in fetal growth (Cameron & Demerath 2002, 164; St. Jacques & Helms 2003, 101), though are also regulated by nutrition. Chronic undernutrition has been found to permanently reduce IGF production (Barker 1994, 27). Insulin is of central importance to fetal growth because as it stimulates mitotic drive and nutrient availability for cell proliferation (Barker 1994, 26). Insulin hormones are able to respond to the nutrient levels of the mother, signalling to the fetus whether there is adequate nutrient availability (Barker 1994, 26). As a result, growth rates are regulated by insulin hormones to ensure they can be accommodated by nutrient supply (Barker 1994, 26). Cortisol is particularly important in the later stages of gestation, as cortisol hormones trigger maturation responses in various fetal tissues (Barker 1994, 26). This is to prepare the fetus for extrauterine life (Barker 1994, 26). In addition, cortisol, like insulin, is also able to signal nutrient insufficiency to the fetus (Barker 1994, 26). Thyroid hormones are important for the process of cell differentiation and also in the regulation and consumption of oxygen by the fetus (Barker 1994, 28).

Bone growth initially results from the mesenchyme, the meshwork of embryonic connective tissues (Mays 1998, 8; Scheuer & Black 2000a, 19; Aubin & Heersche 2003, 44; Sadler 2014, 125). Small areas of dense, clustered mesenchyme give rise to skeletal blastema, small

zones of condensed activity which develop into specific bones. *Hox* genes regulate this skeletal development, ensuring bones are developing in the correct locations (Scheuer & Black 2000a, 19; White *et al.* 2012, 39-40; Sadler 2014, 135). Growth and development of bone consists of the interaction of two primary cells: osteoblasts and osteoclasts. Osteoblasts are those cells which synthesise and secrete bone matrix, while osteoclasts resorb and remove bone (Mays 1998, 6-7; Scheuer & Black 2000a, 30; Aubin & Heersche 2003, 43; White *et al.* 2012, 35). These two types of cells work in unison and are delicately balanced to ensure bone is accurately modelled and remodelled (Scheuer & Black 2000a, 23; Aubin & Heersche 2003, 44; Cornish & Martin 2003, 217). As osteoblasts become trapped in the growing bone matrix they transform into osteocytes (Scheuer & Black 2000a, 23; Aubin & Heersche 2003, 43). Thus, bone growth is not solely the process by which the human body increases its size and morphology, but is also a process in which there is specialisation, destruction, removal and replacement of material (Sinclair 1985, 1-2; Scheuer & Black 2000a, 30; Bogin 2001, 68; White *et al.* 2012, 35). Therefore, the body is in a '*constant state of flux*' with a continuous exchange and replacement of cells and their molecular constituents (Tanner 1978, 26). Although bone cells are already 'coded' to grow in a particular way, muscle usage and activity related stress can greatly alter bone morphology (Tanner 1978, 36; Aubin & Heersche 2003, 43; Oestreich 2008, 13; Duren *et al.* 2013, 49; 54). However, the highly plastic nature of the skeleton also means that bone cells are affected by disease, diet, and social, political, cultural or psycho-sociological stressors.

Bone is a compound of both organic and mineral components: 20-30% of bone is organic, 10% is water, and the rest is mineral (Cole 2003, 1). Bone is comprised of compact, cortical bone and trabecular, or spongy, bone (Mays 1998, 1; Brickley & Ives 2008, 21; Waldron 2009, 12; White *et al.* 2012, 32). Cortical bone forms the thick outer shell of bone, whilst trabecular bone is that of the honeycomb structure found on the internal aspect (Mays 1998, 1; Brickley & Ives 2008, 21; Waldron 2009, 12-13; White *et al.* 2012, 32). However, when cortical bone is modelled, or remodelled, it is not secreted directly as this matrix, but instead as woven bone. Woven bone, sometimes referred to as fibre bone (Ortner 2003, 19; Brickley & Ives 2008, 23), is characterised as immature, highly disorganised bone (Mays 1998, 6; Scheuer & Black 2000a, 30; White *et al.* 2012, 35). It is typical of fetal/perinatal/infant growth as it is formed very quickly (White *et al.* 2012, 35) and is often identified by its grey appearance. Woven bone matures and remodels into lamellar bone, that of a well organised,

linear bone structure (Scheuer & Black 2000a, 30; White *et al.* 2012, 35). Despite both woven and lamellar bone being characteristic of normal bone growth and development, they also can be observed when health insults or trauma are inflicted on the skeleton (Mays 1998, 6; Brickley & Ives 2008, 23; White *et al.* 2012, 34). Response by the skeleton to environmental onslaughts (be they infectious, metabolic, traumatic) results in pathological new bone formation which is identical in process and appearance to normal, healthy new bone formation. This is because the process by which bone grows, remodels and responds is identical regardless of the stimulus. Consequently, where bone growth is proliferative in fetal, perinatal and infant individuals, woven bone is common-place. Distinguishing this normal bone growth from pathological bone growth is consequently highly problematic.

With the aim of the following thesis being to examine the correlation and relationship between growth and exposure to stress through the analysis of infantile skeletal populations, the fundamental biological processes of 'normal' growth must first be outlined. 'Normal' growth is considered in this study to be when an individual grows to full potential without any limiting factor impacting upon this process (e.g. Slack 1991, 9).

3.21 Growth and Development of the Cranium

The cranium is divided into two separate parts, the neurocranium and the viscerocranium. The neurocranium consists of the bones which surround and protect the brain, whilst the viscerocranium form the skeleton of the face (Sadler 2014, 125). The neurocranium can be further divided into the membranous part – the flat bones of the cranial vault – and the chondrocranium – the base of the cranium (Sadler 2014, 125).

Intramembranous growth is within a membrane, fibrous tissue or mesenchyme (Scheuer & Black 2000a, 23; Oestreich 2008, 1; 13) where no cartilaginous precursor is required (Mays 1998, 8; Karsenty & Kronenberg 2003, 120). Instead the membrane is directly ossified by osteoblasts (Mays 1998, 8). The bones of the cranial vault are all intramembranous in origin (Oestreich 2008, 13), as well as the facial bones (viscerocranium) and the clavicle (Scheuer & Black 2000a, 23). The cranial base (chondrocranium) develops endochondrally. The *dura mater* acts on the endocranial surface of the cranial bones as the periosteum, while the true periosteum covers the ectocranial surface and sutures (Oestreich 2008, 16).

The cranial base, one of the most complex skeletal structures, first appears around the fourth gestational week; mesenchyme masses appear at particular sites during this period and spread anteriorly during the second intrauterine month (Scheuer & Black 2000a, 40; St. Jacques & Helms 2003, 78-79). At the end of the first gestational month the vault of the skull begins to develop. The ossification centre for the occipital squama develops posteriorly, as well as those for the temporal squamae, and frontal bones laterally (Scheuer & Black 2000a, 43). These bones begin as curved plates of mesenchyme which gradually extend downwards to meet the forming cranial base (Scheuer & Black 2000a, 43). The parietal bones do not begin ossification until the fetal period (Scheuer & Black 2000a, 43), though there is little agreement of the exact point these bones develop (Scheuer & Black 2000a, 99; St. Jacques & Helms 2003, 86). The parietal bones are characterised by their thickened central eminence from which a network of fine trabecular bone radiates (Scheuer & Black 2000a, 99). The frontal bones have a similar pattern of development, where a network of trabeculae radiate upwards during growth (St. Jacques & Helms 2003, 79). Within the flat bones of the cranium, bone is typically deposited in waves of concentric layers at the perimeter (sutures) of the skeletal element (Karsenty & Kronenberg 2003, 120; Lewis 2017, 3). Throughout pre- and postnatal life, neurocranial bones grow by apposition, with new bone laid down on the outer surface, whilst bone resorption occurs on the inner surface (Sadler 2014, 125).

Intramembranous growth/ossification is the first to commence formation (Scheuer & Black 2000a, 22) and is also considered to be more rapid than endochondral ossification due to the heightened requirement of these structures for support and protection (Scheuer & Black 2000a, 18; 2000b, 14). The speed of growth within bones of the cranium is considered to be a reflection of the rapidity of brain growth during this age (Scheuer & Black 2000b, 14; Lewis 2007, 61; 2017, 3). It is known that the body prioritises growth of particular skeletal and soft-tissue structures, with the brain sitting at the top of this physiological hierarchy (Barker *et al.* 2012, 30; Said-Mohamed *et al.* 2018, 5), consequently requiring the cranial bones to be adequately developed (Karsenty & Kronenberg 2003, 120). It has been found that infants dedicate up to 87% of their resting metabolic rate to brain development (Bogin 2001, 108; 2012, 351; Said-Mohamed *et al.* 2018, 6), thus skeletal development of cranial bones must coincide with this prioritisation. Findings by Humphrey (1998, 62) also established that the cranial bones are some of the first to reach adult proportions: by the end of the first postnatal year the breadth of the frontal bones will have reached 80% of their adult size.

3.22 Growth and Development of Long Bones

Endochondral ossification refers to the process whereby a cartilaginous model (hyaline cartilage model) first develops and this provides a template for bone development and growth (Cole 2003, 1; St. Jacques & Helms 2003, 78; Oestreich 2008, 8). Within endochondral ossification, osteoblasts deposit osteoid to gradually mineralize the cartilaginous model into one of bone (Tanner 1978, 32; Lewis 2007, 61). Within a long bone (i.e. a humerus or femur), the bone develops from a primary centre of ossification (Stevens & Williams 1999, 196; Scheuer & Black 2000a, 18; White *et al.* 2012, 34). Most primary centres of ossification develop prenatally, such as those of the long bones which are all present by the 12th gestational week (Sadler 2014, 133), though some do not appear until postnatal or even adolescent life (Scheuer & Black 2000a, 18; St. Jacques & Helms 2003, 81). Once this primary centre has been established, growth occurs at the growth plate, or metaphyses (White *et al.* 2012, 38). The growth plate is the area between the cartilaginous models of the long bone diaphysis and the epiphysis; it is in this area and space between the two that new bone cells must be added to increase overall bone length (Stevens & Williams 1999, 196; Lewis 2007, 62; Mays *et al.* 2009, 410). Cartilage cells directly beneath the epiphysis divide and pass into the growth plate, aligning themselves into columns of flattened cells (perichondrium) as they descend towards the end of the long bone diaphysis (Acheson 1959, 124; Tanner 1978, 32-33; St. Jacques & Helms 2003, 79; Oestreich 2008, 1). When close to the diaphysis an intercellular substance surrounds the cells so that each column is enclosed in its own sleeve (Tanner 1978, 33). Once enclosed in these sleeves, the cartilage cells enlarge, lose the flattened nature of their appearance and become more disorganised; eventually those nearest the diaphysis of the long bone either die (apoptosis) or are converted into bone cells (Tanner 1978, 33; Aubin & Heersche 2003, 43; Brickley & Ives 2008, 24).

Shortly before birth some of the secondary centres of ossification begin to appear (White *et al.* 2012, 38). These are typically the epiphyses which lie to the proximal or distal ends of the long bones (Scheuer & Black 2000a, 18; White *et al.* 2012, 34). However, for most of the limb bones these secondary centres do not ossify until after birth and are thus often not found/recovered from archaeological remains of fetal/perinatal/infant individuals. Like the long bones, epiphyses undergo the same process of laying down a cartilaginous model before being converted into bone (Tanner 1978, 32). Throughout growth the primary centres of

ossification (typically the diaphyses) are separated from the secondary centres of ossification (epiphyses) by the growth plate. It is not until the rate of ossification exceeds cartilage proliferation that this growth plate narrows, eventually resulting in epiphyseal fusion and the cessation of longitudinal growth (Scheuer & Black 2000a, 18; White *et al.* 2012, 38; Sadler 2014, 135).

The cortical bone of long bones is also covered by an outer surface known as the periosteum, except at the joints, or metaphyses in infant individuals (Mays 1998, 1; St. Jacques & Helms 2003, 80; Brickley & Ives 2008, 21; Kini *et al.* 2012, 29). The periosteum is a fibrous connective tissue containing blood vessels and nerves (Tanner 1978, 35; Schultz 2001, 116; Kini *et al.* 2012, 29) and is also involved in the process of appositional growth (Waldron 2009, 12; Kini *et al.* 2012, 29; White *et al.* 2012, 38). The periosteum is made up of two different layers: the outer layer is purely fibrous but the inner layer consists of cells which multiply and lay down new bone (Tanner 1978, 35; Waldron 2009, 20). Appositional growth of the long bone is intramembranous as no cartilage is present (Tanner 1978, 35), though the bone cells act very much like the cartilage cells in the growth plate at the metaphysis of a long bone, laying down layer after layer to the outer cortical surface of long bones, increasing their diameter (Tanner 1978, 35; White *et al.* 2012, 38).

Where endochondral and intramembranous growth plates meet at the physis and metaphysis a layer, one cell thick, develops. This metaphyseal collar has been termed the periphysis by Oestreich (2008, 3). This collar circumscribes the more mature portions of the growth plate towards the diaphysis from the less mature/more recently developed bone cells and enables appositional/transverse widening at this point (Oestreich 2008, 3). The periphysis thus regulates growth at this point, both limiting and allowing this widening, ensuring growth is happening both endochondrally and intramembrously at the correct rate (Oestreich 2008, 3).

Tempo of growth is known to vary between varying skeletal structures, and even within them (St. Jacques & Helms 2003, 81; Oestreich 2008, 8). The most rapid endochondral growth is at the distal femur whilst the distal radius is known to have a faster rate of growth than its proximal end (Scheuer & Black 2000a, 19; Oestreich 2008, 8). Environmental conditions which may affect rates of growth tend to affect the skeletal structures of greatest growth most prominently, making the largest bones the most likely to reflect growth disruption (Oestreich

2008, 8). Furthermore, vascular activity is also directly correlated to bone growth (Scheuer & Black 2000a, 19); increased vascular activity leads to increased bone growth (Oestreich 2008, 13). This is significant as cortical thickness is thus potentially directly related to health or disease status (Oestreich 2008, 13); cortical thickness appears to be reduced when nutritional status is equally reduced (Mays *et al.* 2009, 410)

Between 34 and 36 gestational weeks fetal long bone growth has often been found to reduce as a result in intrauterine space constriction (Lewis 2007, 63). Consequently, though individuals *in utero* are not weight-bearing, the confined intrauterine environment can lead to pressure on various skeletal elements (Oestreich 2008, 21-22), which may in turn be reflective of health/disease status (e.g. vitamin D deficiency).

3.23 Growth and Development of the Dentition

Teeth and the jaws (mandible and maxilla) have their own growth pattern, autonomous from the rest of the skeleton, and characterised by the two sets of dentition (Mays 1998, 10; Hillson 2005, 207; AlQahtani *et al.* 2010, 481). Humans typically have 20 deciduous teeth and 32 permanent teeth (Mays 1998, 10). Hillson states that the deciduous dentition is associated with the 'shorter', small face of young individuals, and the larger adult dentition is accompanied by a comparable growth in the facial skeleton (2005, 207). The process by which deciduous dentition develop, resorb and consequently become replaced by the permanent, adult dentition provides a useful timeline for determining age-at-death. This process is one which, disregarding any pathological/congenital circumstances, occurs throughout the period of infancy, childhood and adolescence in all individuals with regularity (Hillson 2005, 207).

The dentition, both deciduous and permanent, develops in a systematic, sequential way making formation a good way to estimate age up to early adulthood (Blakey & Armelagos 1985, 371; Huda & Bowman 1995, 139; AlQahtani *et al.* 2010, 481; 2014, 70). Deciduous dentition begins to develop *in utero* from around the sixth week (Scheuer & Black 2000a, 44; AlQahtani *et al.* 2010, 481) meaning assessment of dental development can be utilised in even the youngest individuals. Teeth grow systematically from the tip of the crown to the root (Blakey & Armelagos 1985, 371; Mays 1998, 11; Lewis 2007, 39). This happens at varying

yet relatively precise ages for each tooth (Hillson 1979, 147-148), with both enamel and dentine deposited in regular increments (Šešelj 2013, 44).

Teeth consist of three hard tissues: enamel, cementum and dentine (Mays 1998, 10; Hillson 2005, 146). Unlike bone, dental tissues do not remodel as they lack a blood supply (Mays 1998, 10). However, like bone, teeth are comprised of both organic and inorganic components (Hillson 2005, 146). The predominant inorganic (mineral) components are calcium and phosphate, whilst collagen makes up the organic component (Hillson 2005, 146-148). The proportions of these organic and inorganic components changes over the life course, with a much higher percentage of organic components in developing and newly developed teeth; as teeth 'mature' proteins are removed and a greater mineral content develops (Hillson 2005, 149).

Enamel is a unique mammalian tissue due to its primarily inorganic and acellular composition (Hillson 2005, 155). Enamel formation, commonly referred to as amelogenesis (Hillson 2005, 155; 209), occurs within the enamel epithelium. Amelogenesis consists of three phases – formation, mineralisation and maturation (Mays 1998, 11). Initially an organic matrix is formed, where crystallites are seeded into the matrix, slowly mineralising the structure (Smith 1998, 128). This mineralised matrix has the appearance and features consistent with fully developed enamel, but is actually only one third mineral (Smith 1998, 133; Hillson 2005, 155). Therefore, the third phase of development, maturation, occurs when both proteins (organic) and water components are removed, increasing the size of the crystallites, creating a densely mineralised structure – around 95% mineral (Smith 1998, 128; 133; Hillson 2005, 155). This maturation process occurs both before, during and after eruption of the dentition into the oral cavity. Like bone, the dentition grows appositionally in layers (Smith 1998, 128; Hillson 2005, 209; Armelagos *et al.* 2009, 266), meaning that enamel mineralisation and maturation happens at different times within different individual teeth, with these processes occurring at the tip of the crown first (Hillson 2005, 156).

Within the dentition dentine is the first material to form, before that of enamel (Hillson 2005, 185; 208). Dentine, like enamel, is comprised of both organic and inorganic components. However, unlike enamel, dentine is a living tissue and odontoblasts (dentine cells) continue to secrete and line the sides of the pulp chamber throughout life (Hillson 2005, 184-185).

This is known as secondary dentine and continues after the initial formation phase (Hillson 2005, 185). Growth and formation of primary dentine consists of two phases; the first phase is where the odontoblasts secrete an organic matrix (predentine), this then mineralises to become dentine by the seeding of crystallites into the matrix (Mays 1998, 11; Hillson 2005, 185; 208). Predentine forms initially directly under the tooth cusps, before enamel is mineralised (Hillson 2005, 185). Although the initiation of dentine formation precedes the initiation of enamel formation, once commenced these processes proceed in parallel, with both dentine and enamel laid down layer by layer (Hillson 2005, 185).

3.3 Altered and Affected Growth

Growth is regulated by both intrinsic (genetic) and extrinsic (environmental) factors (Cattaneo 1991, 39; Saunders & Hoppa 1993, 128; Bogin 1999, 51; 228-239; King & Ulijaszek 1999, 161; Scheuer & Black 2000b, 11; Bogin & Rios 2003, 74; Cardoso 2007, 223; Lewis 2007, 60; Duren *et al.* 2013, 49; Ong *et al.* 2015, 975) and the interplay between these various determinants affects and alters the growth process. Importantly, the interaction between genetics and environment is non-linear (Tanner 1978, 117). Though original models of the 1960s and 1970s considered it to be a linear process, the last ten years has seen the emergence of a 'biocultural' view. This view posits a constant and recurring interaction between the human biological aspect of growth and the environmental factors, importantly including where biological changes affect and modify social and cultural behaviour (Bogin 2001, 15; Halfon *et al.* 2014, 348; Larsen 2015, 7).

The human body is highly labile and physiological plasticity is the ability of an individual, group or population to change size and shape in response to environmental factors, be that positively or negatively (Bogin & Loucky 1997, 30; Bogin 2001, 74; Goodman & Martin 2002, 19; Bogin & Rios 2003, 71; Clukay *et al.* 2018, 173). During the early years of growth and development the size and shape of the human skeleton is particularly plastic (Bogin & Rios 2003, 72). The nature of cell development and the constant need to exchange, replace and renew constituents of those cells and develop new cells means the body is always influenced by the environment in which those cells develop. Therefore, the constant turnover of material and the dynamic state of the body allows us and our cells to continually interact, react and adapt to a changing environment (Tanner 1978, 26).

Consequently, assessment of growth has been widely considered within bioarchaeological and anthropological discourse to consider the varying effects of adversity. Growth has long been acknowledged to be particularly susceptible to environmental onslaughts – those such as poor nutrition, reduced socioeconomic status and exposure to pathogens - during the growth period, making non-adults sensitive barometers of overall population health (Saunders & Hoppa 1993, 132; Bogin 1999: 228-239; Hoppa & Fitzgerald 1999, 13; Larsen 2015, 7). Boas (1912) was one of the first scholars to suggest extrinsic factors have a significant role in the regulation of growth. Indeed, determining evidence of growth disruption has long been a central concern within bioarchaeology, and is often utilised as a proxy for health status (Clukay *et al.* 2018, 173; Miller 2018). This has been particularly demonstrated in studies of non-adults, where death is indicative of an inability to adapt or recover from detrimental onslaughts (Goodman & Martin 2002, 19). Thus, as non-survivors of a population, growth disruption is very likely reflected in their skeletal remains.

It has been long considered that there are 'critical periods' of human growth and development, where particular environmental stimuli at such points have greater regulation and impact, channelling growth in accordance to these benefits and/or limitations (Hahn *et al.* 1972, 128; Cameron & Demerath 2002, 159; Gluckman & Hanson 2005, 23; Kuzawa & Quinn 2009, 143; Helfrecht *et al.* 2017, 2). The first 1000 days of life – from conception through to infancy – have been identified as the most fundamental for human growth plasticity (Barker 2012, 187; Said-Mohamed 2018, 4), making them the most influential in shaping future growth and health (Holdsworth & Schell 2017, 1). For developing bodily systems this critical period enables modifications in response to environmental stimuli (Kuzawa & Quinn 2009, 134; Sandman *et al.* 2016, 230). Consequently, environmental effects during the first 1000 days of life can affect cell number, cell type and epigenetic cellular gene expression within particular bodily systems (Kuzawa & Quinn 2009, 134; Chmurzynska 2010, 87). This critical period (the first 1000 days) closely aligns with points in the life course during which the mother is able to buffer offspring against environmental onslaughts to a greater degree (Kuzawa & Quinn 2009, 131; Said-Mohamed *et al.* 2018, 8), where nutrition and immunity can be directly transferred between mother and offspring via both placenta and lactation.

Fetal, perinatal and infant individuals are consequently the most vulnerable to a variety of environmental onslaughts, and their growth is hence a reflection of their experience in the intrauterine environment (Winick *et al.* 1972, 80; Bogin 2001, 68; Oestreich 2008, 20; Kuzawa & Quinn 2009, 132; Kuzawa & Sweet 2009, 3; Mays *et al.* 2009; Dancause *et al.* 2012, 307; Lejarraga 2012, 24). Disease and nutritional deficiency are considered to be the two primary factors that most commonly disrupt growth (King & Ulijaszek 1999, 161; Mays *et al.* 2009, 410). Consequently, fetal development is heavily reliant on the maternal ability to provide adequate nutrition *in utero* (Barker *et al.* 2012, 31; Said-Mohamed *et al.* 2018, 6); maternal malnutrition is the most significant factor in fetal malnutrition (Hales & Barker 2001, 7). The maternal body prioritises the wellbeing of the growing fetus, thus ensuring its optimal growth and development (Gowland 2015, 533; Said-Mohamed *et al.* 2018, 7). However, this requires the mother to have sufficient availability of nutrients to supply the placenta, and relies also on the placenta being able to transport these to the fetus (Gluckman 1997, 153; Barker *et al.* 2012, 30). With particular regards to bone formation and growth, a ready supply of protein and energy is required (Prentice 2003, 255). Poor maternal diet and nutrition is also known to result in a plethora of detrimental outcomes for mother and offspring including lowered birth weight, maternal haemorrhage, pre-eclampsia, IUGR, pre-term birth and birth defects (e.g. neural tube defects) (Winick *et al.* 1972, 86; Prentice 2003, 255; Kuzawa & Quinn 2009, 133; Wu *et al.* 2012, 4). However, nutritional deficiencies (and stress more generally) experienced within the first trimester have been found to be more strongly correlated with these outcomes than when malnutrition is experienced in later gestation/third trimester (Wu *et al.* 2012, 13; Sandman *et al.* 2016, 238). Furthermore, Oestreich (2008, 21) suggests that detrimental environmental factors are able to disrupt bone growth both locally and systemically, supporting current hypotheses that when limited nutritional resources are available, the fetus is able to prioritise growth in particular bodily structures and systems (Barker *et al.* 2012, 30). As a result, there is often a trade-off between structures (Barker *et al.* 2012, 30; Said-Mohamed *et al.* 2018, 5), typically seen in the way longitudinal growth of the long bones may be expended for the benefit of the brain (Aiello & Wells 2002, 330-331; Kuzawa *et al.* 2014, 13010; Sandman *et al.* 2016, 230; Said-Mohamed *et al.* 2018, 5).

Limitations in protein, calorie, vitamin and nutrient intakes all have varying effects on offspring growth (See Wu *et al.* 2012 for extensive discussion of varying nutritional factors).

However, it is not just the quantity but the quality of these factors that is important to successful fetal, perinatal and infant growth (Hahn 1972, 99). Maternal undernutrition of these required elements is associated with SGA, IUGR, low birth weight and premature individuals, with increased morbidity and mortality risks (Wu *et al.* 2012; Farewell *et al.* 2018, 1; Said-Mohamed *et al.* 2018, 6). Placental function is also central to the growth and health of the offspring (Gluckman 1997, 153; Gluckman & Hanson 2005, 33; Luo *et al.* 2010, 93), with faulty placental transfer of nutrients a common underlying cause of fetal malnutrition (Winick *et al.* 1972, 81; Hales & Barker 2001, 7), and thus, subsequently, growth disruption. The placenta is also important in regulating and producing hormones that influence fetal function and growth (Gluckman & Hanson 2005, 33; Kuzawa & Quinn 2009, 135; Sadler 2014, 98), as well as delivering maternal antibodies, oxygen, blood and glucose to the developing offspring. Therefore, the fetus is also able to gain passive immunity against various infections and diseases from the mother through placental transfer (Sadler 2014, 100; Thorsell & Nätt 2016, 3). Consequently, disease and health status, that of exposure to infection pre- and postnatally, is also of fundamental importance for skeletal growth (Mays *et al.* 2009, 410). As the fetus is yet to develop their own functioning/mature immune system they are entirely reliant on maternal antibodies and buffering (Wu *et al.* 2012, 13; Schoenwolf *et al.* 2014, 176). Thus, diseases/infections which are minor in the mother, can be deleterious to the fetus and can even result in death (Schoenwolf *et al.* 2014, 176). As a result, infections have been found to have a profound influence on growth velocity (Blackwell *et al.* 2017, 452; Said-Mohamed *et al.* 2018, 9), thought to be as a result of prioritisation and reallocation of energy resources to immune function (Kuzawa & Quinn 2009, 139; Said-Mohamed *et al.* 2018, 9).

Both nutritional and immune factors can also have a profound impact on skeletal growth as the human body is able to alter hormone regulation as a result of exposure to these environmental conditions (Barker 1994, 132; Godbout & Glaser 2006, 421; Kuzawa & Quinn 2009, 132). As discussed previously (See Section 3.2) a range of hormones control and regulate normal growth and development. However, prenatal exposure to maternal stress can alter regulation of the hypothalamic-pituitary-adrenal (HPA) axis (Armelagos *et al.* 2009, 263; Davis *et al.* 2011, 119; Murgatroyd & Spengler 2011, 1). The HPA axis controls and regulates the body's reaction to 'stress' as well as controlling the immune system (Oberlander *et al.* 2008, 97; Kuzawa & Quinn 2009, 138; Murgatroyd & Spengler 2011, 1; Sandman *et al.*

2016, 231). Ultimately, maternal nutritional, health and endocrine status can alter production, secretion and regulation of the various hormones required for intrauterine growth, as well as the ability to deliver nutrients via the placenta (Gluckman 1997, 154; Dancause *et al.* 2012). Therefore, growth disruption is not solely the physiological response to environmental conditions, but may in fact be regulated on a cellular level by such factors (Murgatroyd & Spengler 2011, 1) (See discussion on epigenetic mechanisms later in this chapter). As a result, disruption to the maternal HPA axis, and subsequent hormone secretion or receptors, can have detrimental effects on fetal skeletal growth (Stevens & Williams 1999, 200; Dancause *et al.* 2012, 307). This is because environmental factors, such as undernutrition and physiological and psychological stress, alter maternal regulation and secretion of hormones, which are signalled to the fetus via the placenta (Barker 1994, 132-133; Kuzawa & Quinn 2009, 133; Wu *et al.* 2012, 13; Thorsell & Nätt 2016, 6). This results in fetal individuals having correlating hormonal (stress) levels as that of the maternal host (Kuzawa & Quinn 2009, 141-142; Davis *et al.* 2011, 119). Glucocorticoids (GC) hormones restrict fetal growth and program endocrine and metabolic systems (Luo *et al.* 2010, 92; Thorsell & Nätt 2016, 3). Though normally fetal levels of GC hormone are much lower than those of the mother, due to the placental barrier, stress can result in overexposure and inhibition of placental enzymes which regulate GC hormone exposure (Cameron & Demerath 2002, 164; Kuzawa & Sweet 2009, 5; Luo *et al.* 2010, 92). As a result, such overexposure to GC hormones as a result of maternal stress has been found to alter fetal HPA axis function, disrupting offspring ability to regulate responses to both physiological and behavioural stressors (Oberlander *et al.* 2008, 97; Davis *et al.* 2011, 119; Thorsell & Nätt 2016, 3). Furthermore, it is well established that chronic and severe episodes of stress greatly affect immune response (Godbout & Glaser 2006, 422; Armelagos *et al.* 2009, 265). It has been hypothesised that hormonal disruption of growth, as a result of stress, affects growth of the thymus, reducing immune function as a consequence (Barker 1994, 28-29; Armelagos *et al.* 2009, 265). However, immune responses are also regulated by the HPA axis and the release of glucocorticoid (GC) hormones (Selye 1973, 695). GC hormones act as anti-inflammatory agents (Godbout & Glaser 2006, 422) and disruption to their secretion will limit immune response. These hormonal disturbances, as a result of exposure to stress, thus render the individual incapable of buffering against further detrimental environmental factors, likely leading to significant changes in growth.

Growth is also a sensitive indicator of the social, political and economic environments to which an infant is exposed (Bogin & Loucky 1997, 17; Thorsell & Nätt 2016, 1). Socioeconomic status (SES) has been identified as a key factor in early life viability and wellbeing (Bogin 2001, 70; García *et al.* 2017, 1). Typically, higher SES correlates with healthier, larger and taller offspring and thus birth weight and growth are used as useful proxies to assess the impact of SES (Bogin 2001, 70). It is considered that offspring success is interrelated with the ability of parents/guardians to invest economically, socially and culturally in their wellbeing (Bogin & Loucky 1997, 17). The higher the socioeconomic status of the individual, the increased preferential access they have to food resources, housing, health care, and general living conditions (Mays *et al.* 2009, 410).

Human growth, and subsequently health, are dynamic processes which begin prenatally and remain throughout the life course (Halfon *et al.* 2014, 344), where environmental factors can influence these outcomes. However, research within the last few decades has revealed a greater intricacy to these processes. Since the 1980s work of Barker and colleagues (e.g. Barker & Osmond 1986; Barker 1994) revealed that the environmental conditions to which we are exposed during early prenatal life can have a significant impact on our future growth and health, whilst our genetic profile may be less influential than previously thought (Luo *et al.* 2006, 39; 2010, 90; Halfon *et al.* 2014, 345-346). These life course models suggested that short and long term health outcomes were no longer a product of purely genetic endowment, but were also regulated by environmental, social, cultural and psychological factors experienced in early life (Halfon *et al.* 2014, 345; Thorsell & Nätt 2016, 1). Consequently, exposure to a variety of conditions/stressors can result in epigenetic changes, those in which our gene expression, rather than the underlying DNA sequence, is altered, reflecting the adverse or beneficial conditions experienced *in utero* (Cattaneo 1991, 40; Chmurzynska 2010, 88; Kuzawa 2012, 327; 329; Mortier & Vanden Berghe 2012, 162; Halfon *et al.* 2014, 346; 350-351; Glover 2015, 277). Epigenetic changes predispose our susceptibility, and/or resilience to disease, through altering the structure and function of various biological systems (Slack 1991, 30-31; Cameron & Demerath 2002, 160; Luo *et al.* 2006, 39; Chmurzynska 2010, 87; Mortier & Vanden Berghe 2012, 162).

Although this ‘fetal origins’ or ‘fetal programming’ hypothesis is now widely accepted, the mechanisms behind epigenetic programming are yet to be fully comprehended (Luo *et al.*

2010, 89; Thorsell & Nätt 2016, 1-2). Epigenetic information, unlike that of the base genetic code, is plastic and in theory inherently reversible (Chmurzynska 2010, 88). Epigenetic programming is a normal physiological process of healthy fetal development, where epigenetic changes regulate cell differentiation (Luo *et al.* 2010, 93; Thorsell & Nätt 2016, 1). However, epigenetic changes can also be 'pathological', resulting from insults experienced perinatally (Luo *et al.* 2010, 93; Thorsell & Nätt 2016, 1). It is these potential 'pathological' epigenetic changes that are of most importance to this study, as the adverse environmental conditions experienced within the archaeological and historical samples assessed may be reflected. Consequently, epigenetic changes are either changes which are permanent or reversible for the individual, and may or may not be inherited depending on the environment experienced. Some epigenetic changes, particularly those resulting from DNA and histone methylation, result in epigenetic changes that are heritable through all future generations (Egger *et al.* 2004, 457; Luo *et al.* 2010, 92; Non *et al.* 2016, 84), resulting in permanent changes in the phenotype (Halfon *et al.* 2014, 346). These methylation mechanisms result in gene 'silencing', where epigenetic signals lock genes into 'off' positions (Egger *et al.* 2004, 457; Phillips 2008, 116; Murgatroyd & Spengler 2011, 5). However, heritability is primarily in genes regulating the epigenetic modification (methylation), rather than the epigenetic change itself (Egger *et al.* 2004; Murgatroyd & Spengler 2011, 7). Consequently, even in these cases it is suggested that almost all health-related epigenetic changes are still potentially reversible with drug treatment and lifestyle changes (Egger *et al.* 2004, 460), meaning they are not necessarily permanent epigenetic mutations. In part, this may be because DNA methylation is particularly sensitive to undernutrition, reliant on adequate availability of methionine and vitamins B6, B12 and folate (Chmurzynska 2010, 87). Undernutrition (as discussed previously) has been commonly associated with increased susceptibility to disease and detrimental life course outcomes (Chmurzynska 2010, 87), yet nutritional supplementation is relatively straightforward to address. However, it is still unclear as to the precise biological mechanisms and limitations of epigenetic therapy (Egger *et al.* 2004, 461; Murgatroyd & Spengler 2011, 11). Today, understanding the processes and regulation behind epigenetic changes is still an ongoing clinical concern, but it is suggested the both the type of environmental influence and its timing are critical in influencing epigenetic expression (Murgatroyd & Spengler 2011, 11; Halfon *et al.* 2014, 346; 353).

Two primary hypotheses emerged: the thrifty phenotype hypothesis and the predictive adaptive response hypothesis. The thrifty phenotype hypothesis posits that poor fetal and infant nutrition, leading to changes in growth strategy, is the primary factor driving long term health consequences (Hales & Barker 2001, 7; Luo *et al.* 2006, 39; Armelagos *et al.* 2009, 263; Chmurzynska 2010, 87; Halfon *et al.* 2014, 348). Fetal malnutrition has been linked to a variety of biological process and the incapacity for these to function correctly (e.g. insulin resistance) (Hales & Barker 2001, 7; Kuzawa & Quinn 2009, 135). As a consequence, the fetus is 'thrifty' in utilising its nutritional stores, prioritising growth in particular elements/structures/organ (Hales & Barker 2001, 7; Luo *et al.* 2006, 39; 2010, 90; Kuzawa & Sweet 2009, 4). This concept of the 'thrifty phenotype', where genetic expression is considered to be able to alter, regulate and buffer the offspring from the environmental factors being faced and predicted, posits that epigenetic changes occur to aid the immediate survival of the child, despite this often having long term impacts on adult health (Cameron & Demerath 2002, 159; Luo *et al.* 2006, 39; 2010, 90; Armelagos *et al.* 2009, 264; Kuzawa & Quinn 2009, 135). Consequently, individuals become 'best' adapted for the environment to which they are exposed during intrauterine life (Mortier & Vanden Berghe 2012, 162; Keinan-Boker 2014, 2; Said-Mohamed *et al.* 2018, 9). In contrast, the predictive adaptive hypothesis suggests that the fetus predicts the postnatal environment given the range of signals it receives from the mother *in utero* (Gluckman & Hanson 2005). This predictive adaptive response does not always provide immediate advantages for survival, instead predicting long term survival strategies (Gluckman & Hanson 2005, 24; Kuzawa & Quinn 2009, 136). For this hypothesis, epigenetic adaptations remain favourable as long as the anticipated and coded for environment remains the same (Gluckman & Hanson 2004, 1735; 2005, 24; Halfon *et al.* 2014, 349; Said-Mohamed *et al.* 2018, 9). If environmental conditions vary from those originally experienced, morbidity, mortality and health risks alter (Hales & Barker 2001, 7; 15; Murgatroyd & Spengler 2011, 1; Halfon *et al.* 2014, 349).

Regardless of the epigenetic mechanism, it is these long term health impacts that the Barker Hypothesis, subsequently referred to today as the Developmental Origins of Health and Disease Hypothesis (DOHaD Hypothesis) has explored (Kramer & Joseph 1996, 1254; Armelagos *et al.* 2009, 261; e.g. Barker & Osmond 1986; Barker 1992; 1994; 2012; Barker *et al.* 2002), revealing a multitude of long term growth and health implications of a detrimental *in utero* experience (Ulijaszek & Henry 1996, 1; Barker 1997; 2003; 2012; Barker *et al.*

2002; 2012; Larsen 2015, 7; Hoffman 2016, 656; Clukay *et al.* 2018, 173). Multiple studies have investigated the correlations between early life course experiences and obesity (e.g. Benyshek 2007; Huang *et al.* 2007; Han *et al.* 2010), cardio vascular health (e.g. Dong *et al.* 2004; Barker *et al.* 2005; Kuzawa & Sweet 2009), diabetes (e.g. Hales & Barker 2001), behavioural disorders (e.g. Van den Bergh *et al.* 2005; Talge *et al.* 2007), fertility (Plana-Ripoll *et al.* 2016), and even academic achievement (e.g. Niederhofer & Reiter 2004).

Longitudinal studies of famine victims have provided researchers with cohorts in which to explore the long term consequences of an adverse intrauterine environment on adult health and growth outcomes. During the Dutch Hunger Winter (1944-1945) individuals exposed in the first trimester to periods of starvation and famine have been found to show epigenetic changes consistent with their bodies adapting to a lower caloric intake (Roseboom *et al.* 2001, 95-97). Thus, when food intake returned to normal the individuals were maladapted for their environment (Bogin *et al.* 2007, 633). This resulted in the individuals having higher body mass indexes (BMIs) as adults, and were consequently more likely to suffer from both obesity and cardiovascular disease (CVD) (Roseboom *et al.* 2001, 96). In contrast, those who experienced malnourishment during their third trimester tended to be of lower birthweight, but did not display similar increase in rates of obesity and CVD (Roseboom *et al.* 2001, 96-97).

The complexity of epigenetics is exacerbated when multiple generations or lengthy temporal periods are considered – as in an archaeological context (Mays *et al.* 2017, 42). Epigenetic traits can become 'embedded', with the expression of these traits transferred from parent to child, and subsequently grandchild (Kuzawa & Quinn 2009, 132; 138; Halfon *et al.* 2014, 349; Glover 2015, 277; Gowland 2015, 534; Thorsell & Nätt 2016, 2; Satterlee Blake 2018, 43). Holland-Jones (2005) has termed this as a 'downstream effect'. Therefore, the fetus is able to adapt its growth strategy in accordance with the maternal phenotype (Gluckman 1997, 154; Kuzawa & Quinn 2009, 132), which can be expressed via '*…nutrients, growth factors, metabolites, hormones, and immune factors that reflect the mother's cumulative experience of the local environment*' (Kuzawa & Quinn 2009, 134). This phenotypic transfer may reflect maternal environmental exposures during pregnancy, maternal environmental exposures during her life course, or intergenerational phenotypic expression of the matrilineal line (Kuzawa & Quinn 2009, 132; 138; Halfon *et al.* 2014, 358; Said-Mohamed *et al.* 2018, 7). In

fact, it has been suggested that an individual's investment in offspring is regulated by grand-maternal experiences, where by amalgamation of matrilineal experiences are signalled to the offspring by the expectant mother (Kuzawa & Quinn 2009, 138). This has been termed 'transgenerational phenotypic inertia' (Kuzawa 2005). This multigenerational complexity has led to Gowland (2015) terming this mother-infant dyad and intrauterine period in terms of entanglement – where multigenerational, and both intrinsic and extrinsic factors, are entwined in determining the fetal/perinatal/infant life course. Consequently, predisposition to various health consequences as a result of unanticipated pre- and postnatal environments has had a fundamental impact on the way bioarchaeologists have interpreted growth and health status within non-adult remains (Mays *et al.* 2017, 42). The realisation that prenatal life can be influenced by previous multi-generational experiences challenges our ability to determine when an individual's biography truly begins (Gowland 2015).

Consequently, in light of these developments, compounding genetic, epigenetic and environmental constraints can all affect skeletal growth and development (Bogin 2001, 68). Understanding the intricate interplay between these factors, and identifying evidence within archaeological samples of these limitations is challenging. As this study aims to further current understanding regarding the skeletal evidence for early life exposure to stressors, growth must be considered a sensitive biological parameter by which evidence of adverse experiences and conditions can be identified. Consequently, by using growth and health as proxies for early life experiences, as well as maternal health and wellbeing, understanding of the critical and fragile early life stages of past individuals can begin to be illuminated.

3.4 Terminology

Throughout this thesis, discipline-orientated terminology has been employed. To understand the implications of this terminology the following section defines and outlines the concepts of health and wellbeing, growth and development, stress and stressors, as well as various age-related terminologies, including the categories of fetus, perinate and infant.

3.41 Age-Related Terminology

Defining the terminology employed throughout this research, regarding age and age-at-death estimates, is imperative, as both are typically used in consideration of growth and development, and have also been used to make inferences regarding morbidity and

environmental conditions (Lewis 2007, 38). Age is a complex biological, chronological and cultural construction (Ginn & Arber 1995, 5; Gowland 2002, 10; Baxter 2005, 95-98; Gowland 2006, 143-144; Lewis 2007, 5; Sofaer 2011, 286-287) and much bioarchaeological literature has considered the varying social and physiological implications of distinctions made between individuals based on these varying categories of age (e.g. Stoodley 2000; Gowland 2001; 2002).

This study uses age estimation as a tool for analysis, in distinguishing evidence of growth and health disruption, by comparing age-at-death estimates generated from a variety of skeletal structures. Age-at-death estimates have been employed as follows: ages generated from assessment of dentition have been referred to as dental age-at-death estimates, and those ages generated from the skeleton as skeletal age-at-death estimates (Huda & Bowman 1995, 136). Methods used in both dental and skeletal assessments are those which measure an aspect of physiological (biological) development (Johnston & Zimmer 1989, 12; Lewis 2007, 38; Couoh 2017, 671). That is, the stage of dental or skeletal development is indicative of a certain physiological point of growth within that individual's trajectory. These physiological stages are then translated into estimates of chronological age (Johnston & Zimmer 1989, 12; Lewis 2007, 7; 38). Chronological age is a more standardised method of comparison across data-sets, especially where historic documentation exists (Huda & Bowman 1995, 136). However, due to variation in human growth and development the biological/physiological stage may differ from true chronological age (See Huda & Bowman 1995; Couoh 2017). Thus, chronological age estimates generated and used throughout this study are indeed just that, estimates. By considering both dental and skeletal ages this research considered variation between the chronological age estimates generated, from the different skeletal parameters. The inclusion and use of the 20th century Smithsonian Fetal Collection will explore the accuracy of age estimation techniques further, although these non-survivors may not be representative of normal fetal growth.

Comparative studies of non-adults have been complicated by this heterogeneous mix of terminology (e.g. juveniles, sub-adults, children) with little concordance between existing studies (Lewis 2007, 2; Falys & Lewis 2011, 710). Typically, within archaeological discourse such terms have been ascribed to define both biological/physiological and chronological age. Thus, social constructs of age have often become entangled with biological definitions

(Lewis 2007, 2). Some scholars recommend the term ‘non-adult’ for classifying such individuals as it refrains from using the preposition, ‘sub’, which has been suggested to infer that the study of young individuals is inferior to the study of adults (Lewis 2007, 2). The terms fetus, perinate, and infant have been used with reference to stages of physiological growth and development (See Table 5.1) and in no way represent the social, or cultural constructs of age that may have been afforded to individuals from the varying skeletal samples assessed. Furthermore, as the relationship between physiological and chronological age is not straightforward, and individuals of the same age may vary greatly in their growth and development, our methods fail in being able to reflect this. Age categories, are by their nature, discrete (See Table. 3.1), whereas age is on a continuum (Bogin 2001, 76; Sofaer 2011, 290).

TABLE 3.1 Definitions of age-related terminology employed within this thesis.

Terminology	**Gestational Age Range (In weeks)**
Fetus	< 36
Perinate	36-44
Infant	> 44

Fetus is often used to define individuals considered to be within the period of intrauterine growth and development. Traditionally, fetal individuals are those between 8 gestational weeks of age and birth (e.g. Lewis 2007, 2). This study has classified individuals between 0-36 gestationalweeks of age as fetal. The upper age limit of this term has been set at 36 gestational weeks of age to allow for a distinction between those individuals estimated to be aged around the time of birth. Perinatal – literally around (*peri*), birth (*natal*) – has been considered to represent individuals as young as 24 gestational weeks of age through to 7 postnatal days (e.g. Lewis 2007, 2). Thus, such a broad definition would, for this study, mean that most individuals would be termed as perinatal, even though up to four months could separate them. This study aimed to distinguish between those individuals who, if born prematurely, were unlikely to have survived in the past, from those who were close to full

term. Thus, the definition of perinate used within this study refers to those between 36 and 44 gestational weeks of age. This range covers the commonly considered point of birth at 40 gestational weeks, and allows a +/- 4 gestational weeks, or a month, either side of this point. The term infant has hence been used to describe those between 44 gestational weeks and 6 months of age (64 gestational weeks of age), which is the upper age limit of this study. These individuals have been classified as a group due to the likelihood that all individuals termed 'infant' were both born and survived for some days/weeks postpartum. Thus, by employing these terms and definitions, this study attempts to distinguish and examine individuals based on their physiological growth and development, as well as potential life course experiences (e.g. Wiley & Pike 1998).

Throughout the rest of this thesis where gestational weeks of age have been recorded or mentioned, the abbreviation GWA will be employed.

3.42 Health and Wellbeing

The construction of health and wellbeing has long been debated, particularly within an archaeological and bioarchaeological context, where only a limited amount of information can be gleaned regarding an individual's overall health and wellbeing status from their skeletal remains. However, attempting to reconstruct health and wellbeing in the past can disclose important demographic and population characteristics – those of mortality and morbidity, and fertility as well as the social, cultural and environmental structures and systems in which individuals functioned (e.g. Goodman *et al.* 1984, 264-265; 1988; Bush 1991, 11; Steckel and Rose 2002, 3). Consequently, non-adults are central to such studies, and as aforementioned, represent sensitive barometers of population health and wellbeing (Lewis 2000, 39; 2007, 20). This is due to both their rapid growth trajectory, which enables health stresses to be more readily identified within the skeleton, and their immature immune system, which makes them more susceptible to adverse pre- and postnatal onslaughts (Goodman and Armelagos 1989, 239; Perry 2006, 92; Halcrow & Tayles 2008, 336).

Health has been defined by the World Health Organization as *'a state of complete physical, mental and social well-being and not merely the absence of disease or infirmity'* (WHO 1946; Constitution). The Oxford English Dictionary defines wellbeing as '*The state of being healthy, happy or prosperous'* (Oxford English Dictionary 2017). Consequently, determining

whether an individual was mentally or socially 'happy' or 'prosperous' is not only subjective but impossible within an archaeological context. As a result, it has been suggested that archaeologists have a penchant for using health and wellbeing as synonyms for disease (Waldron 2009, 9), particularly as even absence of disease/pathological indicators on a skeleton does not infer good health or wellbeing (Waldron 2009, 10). As a consequence, bioarchaeologists must interpret data with regards to the likelihood of the individual's quality of life – affording consideration of their potential socioeconomic status, living and working environments, and dietary intake, as well as their ability to adapt, cope and maintain wellbeing when exposed to variations in these factors (Huber *et al.* 2011, 2; Reitsema and McIlvaine 2014, 181). Halfon and colleagues (2014, 355) determine health to be '*… a developmental capacity that allows an individual to interact successfully with his biological, physical and social environments'.* Thus, a bipartite biological and contextual approach to the study of health and wellbeing in the past is paramount.

It has been considered that health and disease simply represent either ends of a spectrum, being respectively successful and unsuccessful attempts by the body to adapt to environmental conditions (Bush 1991, 11). In fact, Audy (1971, 142) states that health '*does not disappear during an illness to return on recovery but continues, even though it may drop in level while the organism is adapting to the current insult*'. This illustrates that health and wellbeing, disease and illness should not be considered as binary, discrete or even static bodily states, but as consistently fluctuating and changing along a continuum (Goodman *et al.* 1988, 195; Larsen 2015, 9). Thus, health is a multifaceted and complex biological construct, yet, social regulation and perceptions of health must not be overlooked. Disease is often socially patterned, with socioeconomic status, availability of health care, wealth and nutrition all bound in determining individual health status (Halfon *et al.* 2014, 347). Additionally, what might be considered 'healthy' today is unlikely to directly correlate to perceptions of health in the past (King & Ulijaszek 1999, 173-174; Roberts 2009, 154). Furthermore, the direct comparison of modern clinical and anthropological data to archaeological human remains renders interpretations of health in the past to be subjective, particularly as these perspectives of health derive from skeletal remains of the non-survivors (Wood *et al.* 1992; Hoppa & Fitzgerald 1999, 13; Hillson 2005, 225; DeWitte and Stojanowski 2015, 406).

Consequently, this research considers health and wellbeing as part of the same continuum as disease and illness, whereby evidence of pathological lesions likely indicates presence of disease and a subsequent reduction in health status. This study has consistently addressed the contextual implications for the presence of pathological lesions, and as such endeavours to adopt a holistic approach to considering and exploring the concept of health within the fetal, perinatal and infant samples assessed. Therefore, the terms health and wellbeing have been utilised throughout, addressing one of the thesis' primary aims of interpreting pathological changes in association and reflection of the socioeconomic and environmental contexts experienced by these individuals.

3.43 Stress and Stressors

Within this thesis stressors were considered to be those factors which have a negative and detrimental impact upon growth and health (Goodman *et al.* 1988, 169; Goodman & Armelagos 1989, 226; Reitsema & McIlvaine 2014, 181), with evidence of stress, or exposure to stress, interpreted from the growth and health disruption identified (Bush 1991, 11; Bush & Zvelebil 1991, 4). Thus, stress and stressors are correlated with metric and pathological changes, suggested to be indicative of an abnormal physiological response (Bush 1991, 11). Stressors may be considered as factors which influence pre- and postnatal life, such as maternal health/disease status, intra- and extrauterine nutrition, social, cultural and environmental changes, as well as the genetic predisposition and inherited fragility of the child. Consequently, stressors are defined as the multitude of both intrinsic and extrinsic factors which can affect and alter normal growth and health (Goodman & Armelagos 1988, 941-942; Goodman *et al.* 1988, 169-170; Bush & Zvelebil 1991, 5).

The work of Selye (1973) is perhaps the most influential in establishing a concept and theoretical framework for stress (Goodman *et al.* 1988, 173; Armelagos & Goodman 1991, 45). He developed the general-adaptation-syndrome model (GAS), whereby stress is defined as the '*non-specific response of the body to any demand upon it*' (Selye 1973, 692). There are three phases to this model: the alarm, resistance and collapse stages (Selye 1973, 694-695). The first stage considers the initial exposure to stress, which inevitably leads to an excessive bodily response due to the individuals' low resistance (Selye 1973, 694). This is the 'fight or flight' response initiated by the autonomic nervous system which provides a rapid response to the stressor (Murgatroyd & Spengler 2011, 2). The HPA axis also responds to this initial

stressor, but at a much slower rate, though it is central to the long term regulation of stress responsivity (Murgatroyd & Spengler 2011, 2). If this initial stress exposure is severe enough, death can result in the individual at this initial stage (Selye 1973, 694). However, for most stressors, repeated and consistent exposure leads to a resistance stage, where the individual's body has adapted to these pressures and is able to accommodate and maintain the effects of exposure (Selye 1973, 695). The final stage is that of collapse, where the individual has been subject to continued stress exposure and can no longer maintain resistance to this, and consequently either succumbs to the effects of this stressor, or as a result becomes more susceptible to further stressors (Selye 1973, 696). Consequently, this collapse stage ultimately leads to death of the individual (Selye 1973, 696). However, the Selyean concept of stress has been critiqued by many scholars for its focus on physiological stressors, avoiding the psychological stressors which equally impact bodily homeostasis (e.g. Goodman *et al.* 1988, 174-175; Armelagos & Goodman 1991, 45; Bush 1991, 12; Weston 2012, 505). Thus, Selye's concept and framework for stress focuses on the ability of the body to respond, adapt, recover and maintain homeostasis (1973, 695). Indeed, health is then considered not as lack of pathological lesions, but in fact evidence and ability of the skeleton to respond and recover from such insults.

The model of stress (Depicted in Fig. 3.1) typically adopted within bioarchaeology is important due its consideration of environmental, extrinsic factors, as those which can both buffer individuals from stressful onslaughts, as well as generate them (Goodman *et al.* 1988, 175; Goodman & Armelagos 1989, 226). It has been found that social and cultural practices can both mitigate stress, but also act as sources of stress through the adoption of these practices. Furthermore, individual perception of stress and stressors will alter experiences and response to stress, meaning physiological responses to stress, identified through the proxy of skeletal lesions, will be individual (Bush 1991, 17; Bush & Zvelebil 1991, 7). Thus, there is no universal response to stress, nor a universal stressor (Bush 1991, 17).

Bioarchaeological interpretations of health, consequently, rely on identification and interpretation of skeletal and dental lesions (Goodman *et al.* 1988, 177-178; Armelagos & Goodman 1991, 51; Goodman & Martin 2002, 11; Reitsema & McIlvaine 2014, 181). These lesions are commonly referred to as 'stress indicators' (Goodman *et al.* 1988, 169-170; Lewis & Roberts 1997, 581; Goodman & Martin 2002, 12; Reitsema & McIlvaine 2014, 181;

Larsen 2015, 8-58), and can represent a variety of pathological conditions such as specific and non-specific infections, trauma and metabolic disturbances, as well as evidence of growth disruption (Goodman & Martin 2002, 12; Reitsema & McIlvaine 2014, 181). Thus, both growth and health disruption can be used as a proxy for exposure to stress. Although this research does not attempt to ascertain the mechanisms (physiological or psychological) behind the stress responses (i.e. the pathological or growth disruption identified), it must be acknowledged that identifying specific causes of stress is almost impossible due to the vast range of insults which can cause identical physiological responses (Goodman *et al.* 1984, 259; 1988, 178; Bush & Zvelebil 1991, 5; Lewis & Roberts 1997, 584; Temple & Goodman 2014, 186). Due to the nature of bone response, which is either bone formation or destruction, multiple stressors or insults can lead to identical changes within the skeleton (Bush & Zvelebil 1991, 5). Furthermore, if a disease is acute, or system specific (i.e. only affects soft tissue structures) no discernible changes will be observable to the skeleton, plus an individual may die before skeletal changes develop, or recover from insults experienced much earlier in life (Bush & Zvelebil 1991, 5; Cardoso 2007, 231). Consequently, although pathological lesions may present as those indicative of certain conditions and diseases, the aetiological cause, as to why an individual was subject to these insults, can rarely be elucidated. Exacerbating this complexity is the likelihood that individuals experience multiple stressors simultaneously (Goodman *et al.* 1988, 187). Therefore, the concept of stress is one which is both complex, and is still regarded varyingly within existing literature.

Consequently, interpreting 'health' from evidence of pathological lesions (stress markers) and growth disruption is complex, as these identifiable skeletal changes represent a physiological response to overcome or maintain these insults. However, skeletal individuals equally represent the non-survivors, where exposure to stress potentially contributed to their death. In addition, fetal, perinatal and infant individuals represent some of the most vulnerable members of past societies (Goodman & Armelagos 1988, 936; 1989, 226; Rogers 1997, 65-66; Lewis 2002b, 38; Lewis 2007, 5), inferring that stress markers may be more commonly identified within their remains. However, stress indicators, although providing insights into health, cannot reveal the entirety of an individual's health and wellbeing status (Temple & Goodman 2014, 189-190), and contextual considerations of environmental, dietary and cultural factors to which the individual was exposed is essential. Within bioarchaeological studies it is hence recommended that a holistic approach to considering

indicators of stress is adopted to more accurately understand patterns of health in the past (Goodman *et al.* 1988, 169; Goodman 1993, 285).

In addition, some indicators of stress have been used naïvely in interpretations of health in the past. For example, the use of Harris lines as an indicator of growth disruption, and thus exposure to stress has been widely debated (Bush & Zvelebil 1991, 4) (See Section 5.34 for further consideration). As a result, this thesis has avoided assessment of specific 'stress indicators', instead taking a broader approach to considering pathological changes, as recommended (e.g. Goodman *et al.* 1988; Goodman 1993). It is intended that this approach will be more revealing with regard to general health and stress exposure, and interpretations will not be limited by only a select few traits being observed. Consequently, within this thesis, consideration of both health and stress is afforded using pathological and growth changes as a proxy for these interpretations, whereby pathological and growth changes indicate a reduction in health as a consequence of exposure to stress.

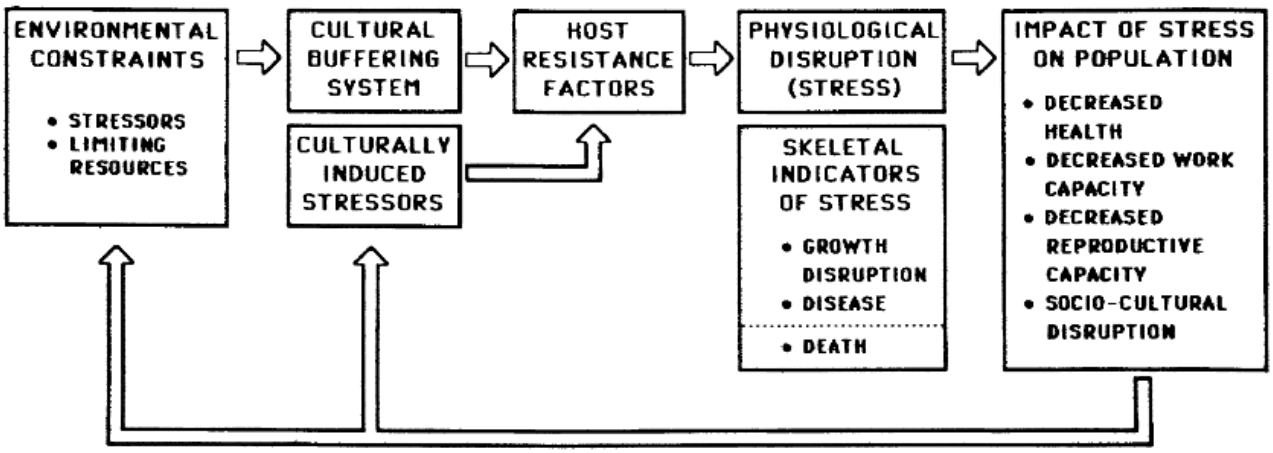


Figure 3.1 A model of stress and stressors, showing the ways in which exposure to stressors can be identified within the skeleton and the contextual implications. Image taken from Goodman & Armelagos (1989, 226).

Chapter 4: Materials

This chapter details the archaeological and historical context of the 15 skeletal samples analysed throughout this thesis. The following sections provide details of the excavation, collection and curation of these remains, as well as the number of individuals analysed from each sample. Although this thesis, and manuscripts herein, are heavily focussed on methodological approaches to exploring growth and health disruption, it is essential to contextualise these findings within the ancient and historical worlds of the samples studied. Differing environments, and societal and cultural organization, will affect interpretations of the growth disruption and health stress identified.

A summary table detailing the sites and their sample sizes in chronological order has been given below (Table 4.1). The dates listed for each skeletal sample are not representative of the whole archaeological sequence for these sites, instead referring only to the periods from which the skeletal individuals assessed come. Additionally, Table 4.2 details samples sizes for all of the fetal, perinatal and infant individuals by historic time period, regardless of the archaeological sites from which they derive. Figure 4.1 illustrates the location of each archaeological site.

4.1 Sample Selection and Rationale

Archaeological samples selected for this study all derive from the south and south-east of England. These archaeological sites were selected due to their relative proximity to each other as well as the known excavation and curation of fetal, perinatal and infant individuals from the sites.

The sites of Owslebury, Piddington and Barton Court Farm were selected for study as they all represented rural sites which transitioned from Iron Age to Roman settlements, yet skeletal individuals recovered primarily reflected varying temporal periods. This has enabled this study (Chapter 6) to investigate health and growth of such young individuals over this significant cultural transition. Although many other Iron Age and Roman rural settlements have been excavated these three sites provided adequate sample sizes for assessment; though sample sizes for Owslebury and Piddington are not large, they are acceptable sample sizes for such young individuals given the archaeological and temporal context.

Medieval and post-Medieval individuals excavated from the City of London, curated and held by the Centre for Human Bioarchaeology at the Museum of London, were also chosen for analysis within this study. This was due to these individuals (N = 184) being available for analysis and representing a large collection from a major urban settlement. Though sample sizes vary greatly between individual sites (See Table 4.1), collectively they enable this study to provide a valuable insight into growth and health consequences of early life in an urban context. Furthermore, this study has deliberately chosen to assess individuals from varying temporal and contextual environments to enable comparisons between samples. Therefore, individuals analysed from the Museum of London (Chapter 7) provide a direct contrast to those from the Iron Age and Roman sites detailed above. As a result, this study attempts to detail similarities and differences in health and health disruption over time (Chapter 9).

The skeletal collection assessed from the Smithsonian Institute, Washington D.C. is the only sample not derived from an archaeological context. Although this collection is geographically disparate to the other samples assessed, comprised of individuals from North-East America (Washington D.C., New York and Columbia), it is a unique historical collection (20th Century) of fetal, perinatal and infant individuals where age, sex, 'ancestry' and cause of death is often recorded. This sample not only reflects a large, documented collection of individuals, but has enabled this thesis to explore health and growth disruption in fetal, perinatal and infant individuals up to the 20th century, providing a unique chronological breadth to this research. By using a documented collection, osteological methods employed to age individuals, as well as assess pathological lesions, have also been able to be tested and evaluated. Although collection bias remains an inherent problem of this sample, inclusion and analysis of these individuals within this thesis enables the furthering of the narrative surrounding fetal, perinatal and infant health and growth disruption over time, a central concern of this research.

TABLE 4.1 Number of individuals assessed in chronological order by archaeological site/collection. Breakdown of the individuals assessed by age for each sample has been provided based on dental development. Where dentition was unable to be assessed, individuals have been recorded in the 'unknown' column.

Time Period (Centuries)	Archaeological Site	Location	Sample Size (*N*) by Chronological Age Category (Based on Dental Development)				Total *N*
			Fetal (*N*)	Perinatal (*N*)	Infant (*N*)	Unknown (*N*)	
3rd BC – 4th AD	Owslebury	Hampshire, U.K.	1	10	2	10	23
1st AD	Piddington	Northamptonshire, U.K.	-	13	3	8	24
1st BC – 4th AD	Barton Court Farm	Oxfordshire, U.K.	1	11	7	33	52
11th AD	St. Benet Sherehog	London, U.K.	-	-	-	3	3
13th AD	Spital Square	London, U.K.	-	-	-	1	1
14th AD	East Smithfield	London, U.K.	-	5	1	2	8
14th – 16th AD	St. Mary Graces	London, U.K.	-	-	1	2	3
16th -17th AD	St. Benet Sherehog	London, U.K.	-	2	7	10	19
16th – 18th AD	Broadgate	London, U.K.	-	2	11	8	21
17th AD	St. Thomas' Hospital	London, U.K.	-	1	3	1	5
17th – 19th AD	St. Bride's Lower	London, U.K.	-	12	17	23	52
18th – 19th AD	Chelsea Old Church	London, U.K.	-	1	4	2	7
19th AD	Cross Bones	London, U.K.	-	18	18	22	58
19th AD	Royal London Hospital	London, U.K.	-	1	-	6	7
Early 20th AD	Fetal Collection, Smithsonian Institute	North East Coast, U.S.A.	7	43	8	82	140
			9	119	82	213	423

TABLE 4.2 Number of individuals assessed in chronological order by historic time period. Breakdown of the individuals assessed by age for each sample has been provided based on dental development. Where dentition was unable to be assessed, individuals have been recorded in the 'unknown' column.

Historical Time Period	**Sample Size (*N*) by Chronological Age Category (Based on Dental Development)**				**Total *N***
	Fetal (*N*)	**Perinatal (*N*)**	**Infant (*N*)**	**Unknown (*N*)**	
Pre-Roman	-	11	2	3	16
Transition	-	13	3	11	27
Roman	1	7	6	25	39
Saxon	-	-	-	2	2
Medieval	-	5	2	8	15
Post-Medieval	-	37	60	72	169
20th Century	7	43	8	82	140
Undated	1	3	1	10	15
	9	119	82	213	423

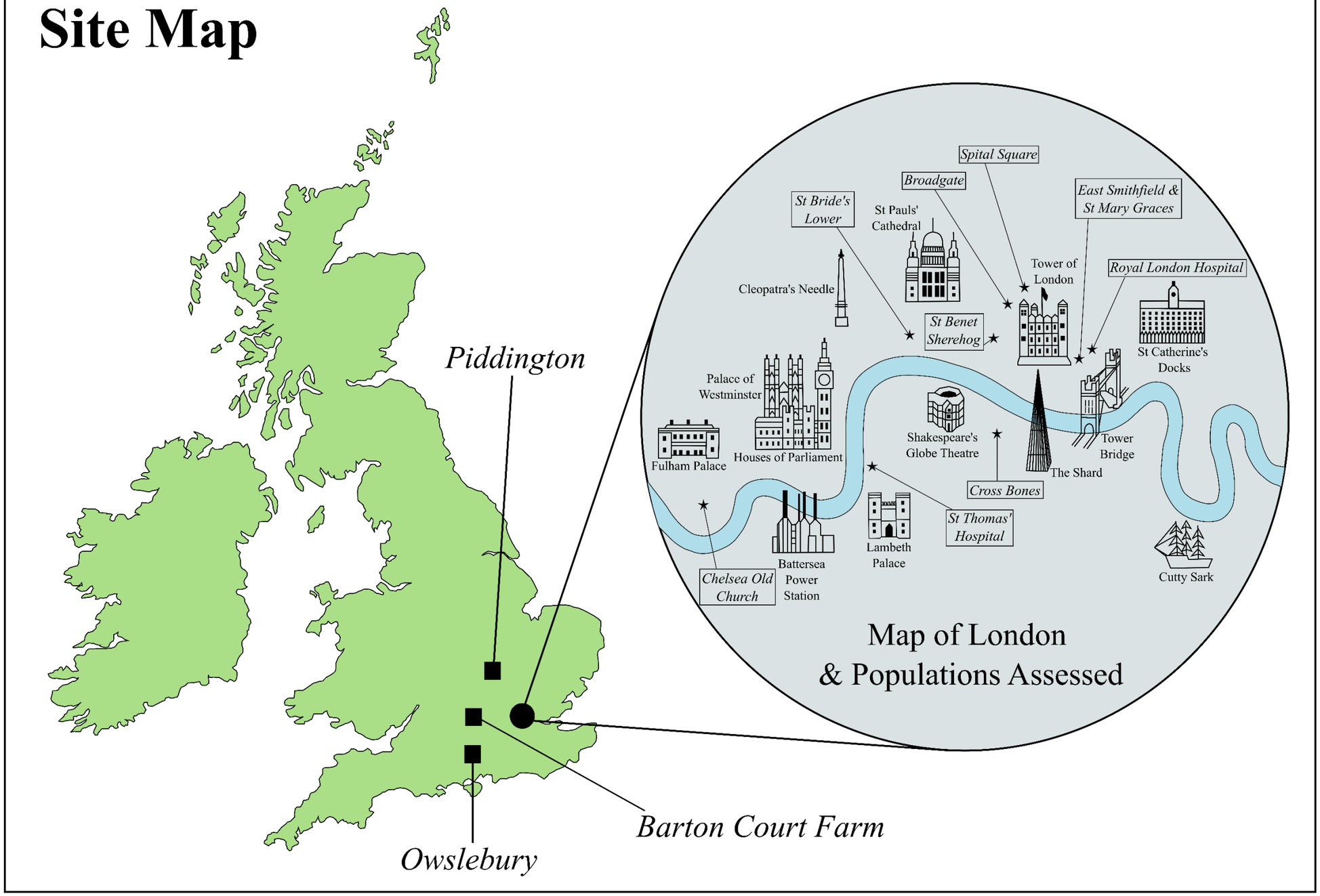


Figure 4.1 Map of the archaeological samples assessed within this thesis.

4.2 Owslebury

Excavations at Bottom Pond Farm, Owslebury, near Winchester, Hampshire were conducted from 1961 to 1972 (Collis 1977, 26; 1994, 106). The site was originally identified when cremated remains were discovered in 1961 as a result of ploughing (Collis 1968, 18). Further trial trenching and aerial reconnaissance occurred in 1962, with small-scale excavation undertaken between 1963-65, and wide-scale excavation between 1966-72 (Collis 1968, 18; 1970, 246; Wells & Collis [No Date], 1). These investigations revealed an archaeological sequence dating from the Bronze Age through to the 4th century AD (Collis 1968, 18; Collis 1977, 26). A banjo enclosure provides the earliest evidence of human activity at the site, succeeded by successive phases of ditches, until the 1st century BC when the site appears to have been extensively remodelled (Collis 1994, 106). This led to a series of ditched enclosures and trackways that were not re-dug and extended until the Roman conquest (Collis 1994, 106). Although this suggests a longevity to inhabitation/usage of the site, the principle phase of occupation was during the Iron Age, which commenced in, what Collis describes as, the 'Belgic period' (1968, 18). Collis distinguishes between occupation periods of '*Iron Age (Early)'* and *'Iron Age (Later)'* (1968; 1970), with the latter being the period he refers to as Belgic.

The Belgic period is typically considered to date from the 1st century BC to the Roman conquest of Britain and references the 'invasion' of people, ideas and objects from Belgic Gaul to the south-east of Britain (Harding 1974, 201; Hill 1995, 79; Cunliffe 2004, 127). This Belgic influence is more typically associated with and referred to as the late La Tène culture (Harding 1974, 201; Hill 1995, 88; Cunliffe 2003, 66; 73-74), a European Iron Age culture. Collis makes no suggestion that those living and working at Owslebury were from the continent, instead seemingly employing the terms 'Belgic' and 'La Tène' to infer cultural and social practices. The presence of Belgic or La Tène cultural influences are suggested to be present by Collis due to a *'warrior inhumation and Belgic cremations inside a rectangular enclosure'* (1968, 18).

Collis suggests that during this predominant phase of occupation (Late Iron Age to early Roman), Owslebury was a 'relatively' wealthy site (1990, 215; 1994, 106). He suggests this due to the excavation of imported ceramic wares from Italy and Spain, as well as Gallo-

Belgic and Gaulish fine potteries (Collis 1994, 106). In addition, silver coinage was recovered from a mid-1st century context (Collis 1994, 106). Such discoveries may indicate that the people of Owslebury were already trading with, and aware of, Roman and 'Romanized' peoples. Collis suggests that Owslebury was inhabited by a '*couple of nuclear families*' (1977, 27; 1994, 108).

Within the internal enclosure ditches, to the north-east of the settlement, three concentrations of burials, referred to as cemeteries, were discovered (Collis 1968, 23-25; Collis 1977, 26). These burials, located proximally to the settlement (Collis 1994; 106), were found to have a combination of funerary practices with both adult and non-adult individuals buried and cremated (Wells & Collis [No Date], 27; Collis 1968, 25). The third cemetery was described by Collis as the infant cemetery, where the burials had been cut into a 1st century BC ditch and cut by a ditch dating from the 1st century AD (1977, 26), identifying them as Iron Age in date. Other non-adult remains were found within the settlement, including in additional ditches and under structures (Collis 1968, 23). Throughout the site, including those in the cemetery, 72 burials were discovered in total, from 70 graves as burials 1 and 35 were double inhumations (Collis 1977, 26). Of the 72 individuals, 33 were found to be non-adults. In total, 24 of these non-adult burials were either fetal, perinatal or infantile, although only 23 could be analysed due to Burial 3 being missing from the collection. Another 46 isolated/disarticulated remains, listed as 'infant', were recorded (Wells & Collis [No Date], 24-27) but these skeletal elements were not included within this thesis as it is unknown whether they represent additional burials or belong to those already afforded burial numbers. The 23 fetal, perinatal or infantile individuals date from the 3rd century BC to the 4th century AD (Wells & Collis [No Date]; Nystrom & Swales [No Date], 1); 14 individuals are from the 1st century BC Belgic Iron Age phase of the site, and five individuals are thought to date from post-Roman conquest - two from the 1st century AD and three from the 3rd century AD. Four individuals remain undated (Wells & Collis [No Date]; Nystrom & Swales [No Date], 1).

4.3 Piddington

Piddington, situated in the south of Northamptonshire, near the Buckinghamshire border, has undergone annual excavation since 1979 as part of a volunteer rescue operation conducted by the Upper Nene Archaeological Society (Upper Nene Archaeological Society 2009 *Briefing Notes*).

These excavations have uncovered an Iron Age settlement, with evidence of an early Roman military presence at the site between AD 45-60 (Upper Nene Archaeological Society 2009 *Phase Descriptions;* Miller 2010, 7), which was followed by successive phases of Romano-British settlement (Friendship-Taylor & Friendship-Taylor 2012). This extensive excavation has provided a rare opportunity to study the Iron Age and Roman phases in significant detail (Upper Nene Archaeological Society 2009 *Interim Report*), making it one of the most extensively explored sites in the country (Selkirk 1996, 57).

Piddington is situated amongst a heavily concentrated area of prehistoric and Roman activity. The Iron Age settlement of Duston is only 6 miles to the north-west, the precursor to the modern town of Northampton (Friendship-Taylor & Friendship-Taylor 2012, 3), with the Iron Age fort of Hunsbury Hill an even shorter distance away. During the Romano-British period Piddington was also situated amongst, and *en route* to, other much larger Roman settlements such as Towcester *(Lactodorum)*, Norton *(Bannaventa)* and Leicester *(Ratae)* (Miller 2010, 6; Friendship-Taylor & Friendship-Taylor 2012). The Watling Street Roman road was also nearby (Miller 2010, 6; Friendship-Taylor & Friendship-Taylor 2012), linking these settlements to a main access route in Roman Britain. Furthermore, evidence of an earlier Roman road has been unearthed to the south of Piddington, and is suggested to have connected the settlements of Duston and Fenny Stratford (*Magiovinium)* (Upper Nene Archaeological Society 2009 *Phase Descriptions*).

Although archaeological evidence has been found at Piddington to suggest activity/presence at the site from Neolithic to Saxon times, the primary archaeological evidence is for an Iron Age and Romano-British settlement (Upper Nene Archaeological Society 2009 *Phase Descriptions*). The site provides evidence of a small Iron Age settlement with multiple roundhouses. These become co-extant with early phases of Romano-British activity at the site, and evidence for the presence of the Roman military being camped at Piddington has also been discovered (Upper Nene Archaeological Society 2009 *Phase Descriptions*). Subsequently, the site appears to have evolved and grown throughout the Roman occupation of Britain, first with a small proto-villa, which was then extended, before a larger villa was built and consistently extended to become a large winged villa compound, with associated

bathhouses, by the 3rd century AD (Miller 2010, 7; Friendship-Taylor & Friendship-Taylor 2012).

In total 34 burials were excavated at Piddington, consisting of six adult burials, 27 fetal, perinatal or infantile burials and one dog burial. All six of the adult individuals, along with a single perinate, are dated to the fourth and fifth century phases of the site. The remaining 26 of the fetal, perinatal and infant burials from Piddington are recoded as being from the 1st century AD. Assessment of the location of the burials show that all of them lie within the villa compound, with the majority of burials seemingly between two of the larger Iron Age roundhouses and respecting the early Roman structures (Hodson 2012, 14). Stratigraphic records suggest that many of the burials have Iron Age features which post-date the interment of the remains, with all of the burials sealed by a layer of dark soil, known to be of Iron Age date (Miller 2010, 12). Thus, the individuals analysed, although considered to be Iron Age, are contemporary with a period of transition, where both Iron Age and Roman occupation was simultaneous, and the transition to a Romanized way of life was beginning at Piddington.

4.4 Barton Court Farm

Barton Court Farm, near Abingdon, Oxfordshire, was excavated between 1972 and 1976 and revealed an archaeological sequence dating from the Neolithic to the Saxon period (Miles 1986). Aerial photographs taken in 1969/70 revealed a series of clear cropmarks covering about two hectares of the Barton Court Farm site (Miles 1986, 4), and indicated multiple phases of occupation. A trackway was also identified which led to Daisy Banks, where it is now known a Romano-British cemetery was located (Miles 1986, 4).

Barton Court Farm is situated in a dense area of archaeological activity; the Upper Thames Valley has a complex network of settlements due to its suitability for inhabitation (Miles 1986, 1). Nearby Barton Court Farm is a Neolithic causewayed enclosure, with possible associated mortuary enclosure and henge monument (Miles 1986, 1). Furthermore, a Bronze Age barrow cemetery and multiple round barrows are also located nearby (Miles 1986, 1). Therefore, it is unsurprising that evidence of prehistoric activity was identified at Barton Court Farm. Multiple pits, postholes and over 1kg of pottery was recovered from the Neolithic phases of the site, along with further structures, enclosures and burials, with at least two of these being infants, identified as Iron Age in date (Miles 1986, 4). However, the

primary archaeological discoveries relate to the Romano-British date of the site; its peak being in the late 4th century AD when the farmhouse/villa consisted of at least eight ground floor rooms, a cellar, and was situated within a large ditched enclosure (Miles 1986, 12). Within this settlement a further building was identified, along with wells, ovens and, to the south-east of the enclosure, an area reserved for infant burial (Miles 1986, 12; 14).

In total, 58 burials were excavated from Barton Court Farm; five of those burials were adults, two being Iron Age and three being Saxon in date (Miles 1986, 6; 18). The remaining 53 burials were all either fetal, perinatal or infantile in age. Two individuals were definitely Iron Age in date, as they were buried at the bottom of Iron Age pits, with a further five suspected to also be from this period, though were stratigraphically less conclusive (Miles 1986, 6). Of the Romano-British individuals, 26 were excavated from shallow pits in the south-east corner of the site, an area of the site seemingly reserved for burial of young infants (Miles 1986, 15). An additional 12 other Romano-British individuals, plus five suspected to be of Romano-British date, were also discovered throughout the settlement. Two of the perinatal/infantile individuals were Saxon (Miles 1986, 18) and one individual, dated from the 1st century BC to the 1st century AD, has been determined as 'transitional' for the purposes of this research. Furthermore, of the 53 fetal, perinatal, and infantile individuals only 52 were analysed due to burial 1151, excavated from the Romano-British 'cemetery', being missing from the collection currently curated within the county museum.

Of note, no Romano-British adult burials were discovered at Barton Court Farm and it is considered that these individuals were instead buried at the cemetery located at Barrow Hills, 800m from the site (Atkinson 1952, 32-34; Miles 1986, 16).

4.5 St. Benet Sherehog

Excavation at the site '1 Poultry', in the city of London, was part of a large-scale redevelopment of the area, encompassing a multitude of streets and locations (Hill & Rowsome 2011, 4). Lying to the south of Poultry and within the Bank Conservation area (Miles *et al.* 2008, 1), this site was originally at the centre of the Roman town of *Londinium* and has been noted as having 'outstanding archaeological survival', with the deep, waterlogged strata preserving organic and environmental remains (Hill & Rowsome 2011, 1).

Thus, excavation at 1 Poultry revealed an archaeological sequence dating from the Roman to post-Medieval periods of London's history.

Antiquarian discoveries at the site of 1 Poultry began in the 19th century, with the famous Bucklersbury mosaic discovered in 1869 (Hill & Rowsome 2011, 1). After almost total destruction during the Second World War, archaeologists were able to reveal more of Roman London's history with the discovery of the temple of Mithras (Hill & Rowsome 2011, 1-3). However, it was not until the 1980s that controlled archaeological excavation took place in the area of Poultry and Bank due to proprietor redevelopment and extension of the DLR (Docklands Light Railway) (Hill & Rowsome 2011, 3). It was these excavations of the 1980s that led to the wide-scale excavation of the site between March 1994 and June 1996 (Miles *et al.* 2008, 1; Hill & Rowsome 2011, 3; Burch *et al.* 2011, 7-9).

In the summer of 1994, the site of St. Benet Sherehog, its burial ground and the underlying church, was discovered at the western limit of the site (Miles *et al.* 2008, 1; Hill & Rowsome 2011, 4; 7; 8), and was found to overlay the old Roman road at Poultry (Cowal 2007a). This church was originally founded in the 11th century but was expanded in the 13th century, remaining in use until its destruction in 1666 during the great fire of London (Cowal 2007a). One of the primary aims of excavation was to remove all burials from the site, be they Medieval or post-Medieval (Miles *et al.* 2008, 3). The church during the Medieval period was found to be a simple structure, approximately 6.1m wide and 10.5m in length (Cowal 2007a). Due to its small size it is considered that during this time it was a private chapel until its expansion in the 13th century. However, it still remained one of the smallest parishes in the City of London (Miles *et al.* 2008, 7).

In total, 280 burials were excavated from the parish church and burial ground of St Benet Sherehog, but only 270 were retained for analysis (Cowal 2007a). Of these 270 individuals, 39 date to the later Medieval period due to their east-west alignment, with the head to the west end of the grave (Cowal 2007a). Of these 39 burials, 23 individuals were found to buried within the church, while a further 15 were external medieval graves (Hill & Rowsome 2011, 12). Of these 39 individuals, 24 were adults and 15 were non-adults, with three of these non-adults being perinatal in age.

4.6 Spital Square

Spital Square, previously known as 'St Mary Spital' (Thomas *et al.* 1997, 2; Cowal 2007b), is located North-East of the City of London on the northern side of Folgate Street (Cowal 2007b). The site is approximately 500m north of Bishopsgate, the primary northern gate into the City of London (Thomas *et al.* 1997, 4). Originally known as the 'Hospital of St Mary without Bishopgate' this was the site of an Augustinian Priory and Hospital, and was amongst the largest hospitals in the country (Thomas *et al.* 1997, 2; Cowal 2007b). Due to extensive excavation and subsequent analysis of the archaeological material and burials from the site, this is considered one of the most comprehensively studied hospitals in the country (Thomas *et al.* 1997, 4).

Discoveries from the site were first made in 1798 with subsequent discoveries in 1892 and 1909 (Thomas *et al.* 1997, 4). However, the first official archaeological investigations took place between 1935 and 1938, due to redevelopment and extension of Spitalfields Market, revealing a range of medieval material and objects (Thomas *et al.* 1997, 4). Full scale, rescue excavations were once again undertaken in 1985, 1988 and 1989 in light of further redevelopment of the area (Thomas *et al.* 1997, 4; Cowal 2007b). These excavations revealed surviving levels of a Roman cemetery, the medieval priory and hospital of St Mary Spital and the post-Dissolution phases of the site (Thomas *et al.* 1997, 5).

In total, 126 individuals were excavated from four areas of the site (*Site codes: NRT85, NRF88, SPQ88 and SSQ88)*, with nine individuals (SSQ88) recovered from the priory (AD 1197-1235), 102 individuals (NRT85, NRF88) recovered from the hospital cemetery (AD 1235-1280), and 15 individuals (SPQ88) recovered from the first phases of the infirmary hall (AD 1280-1320) (Thomas *et al.* 1997, 219; Cowal 2007b). Although these successive phases of the development and expansion of St Mary Spital were recorded, it is the excavation of the 'new' cemetery (AD 1235-1280) to the west of the hospital (OA5) that is of significance. This cemetery was approximately 20m by 27m and contained nine rows of around 25 graves per row (Thomas *et al.* 1997, 37). All graves were aligned east-west (Cowal 2007b), with the head to the west, except for one individual (Thomas *et al.* 1997, 38). All burials were also supine, except for burial 355 which was laid prone (Thomas *et al.* 1997, 38). However, this individual was a neonate and as Thomas *et al.* (1997, 38) suggests, this could be due to the

individual being unbaptised. This was the only perinatal individual recovered from Spital Square.

4.7 East Smithfield

The archaeological site of East Smithfield is situated to the east of the City of London (E1) at the Royal Mint. Lying to the north-east of the Tower of London, the Royal Mint site is approximately two hectares in size (Kausmally 2007). The site was first excavated in 1972 with two small trial trenches being excavated (Grainger *et al.* 2008, 2-3). This was followed by further excavation in 1983 and 1984, before full excavation was conducted between 1986 and 1988 (Kausmally 2007; Grainger *et al.* 2008, 3).

This Black Death cemetery was established somewhere between the end of 1348 and early 1349 as a measure to cope with the plague epidemic facing the City of London (Grainger *et al.* 2008, 1). This cemetery was the first of two established in London to manage the crisis (Kausmally 2007; Grainger *et al.* 2008, 1). No archaeological evidence for substantial buildings or activities at the site were found to predate this cemetery and it is considered that the land was primarily agricultural until its designation as a plague burial ground (Grainger *et al.* 2008, 2-3). A few prehistoric and Roman finds were identified but all recovered from Medieval and post-Medieval deposits (Grainger *et al.* 2008, 3).

The cemetery was organised into two primary areas, the east (OA3) and west (OA2) cemeteries, with both individual and mass graves discovered in both of these locations (Grainger *et al.* 2008, 2; 12). It is thought that up to 2400 individuals may have been buried at the site during its existence (Grainger *et al.* 2008, 2). The significance of this archaeological discovery is that of 759 individuals excavated, 634 were able to be curated and stored for analysis and research (Grainger *et al.* 2008, 2).

The western cemetery (OA2) was situated in the north-west corner of the site and is thought to have been only 50-60% excavated, with the remainder of the cemetery lying beneath the Royal Mint Court's courtyard (Grainer *et al.* 2008, 12). The western cemetery had two mass burial trenches, a mass burial pit and 11 rows of individual graves. The larger of the mass burial trenches was 67m long with the individuals tightly, but carefully placed (Grainger *et al.* 2008, 12). Juvenile and infant remains were often used to fill spaces between the adults

(Grainer *et al.* 2008, 12). The eastern cemetery (OA3) consisted of four rows of individual graves and one mass burial trench (Grainger *et al*. 2008, 17). However, this area had been truncated by later 19th century redevelopment and so the remains were particularly poorly preserved and disturbed in this area (Grainger *et al.* 2008, 17). The mass burial trench in this area was over 125m in length (Grainger *et al.* 2008, 17-18).

Of the 759 individuals excavated, 558 burials were from the western cemetery and 192 individuals from the eastern cemetery (Kausmally 2007). The western cemetery revealed 300 individuals from mass graves, with 258 excavated from individual graves (Kausmally 2007). The eastern cemetery uncovered 102 individuals from mass graves and 90 from single inhumations (Kausmally 2007). Eight fetal, perinatal and infantile individuals were analysed in this thesis, seven from the western cemetery and one from the eastern cemetery.

4.8 St. Mary Graces

St Mary Graces also forms part of the Royal Mint site, excavated between 1986-1988 (Bekvalac 2007a), and was a large burial ground associated with the Cistercian abbey of St Mary Graces (Bekvalac 2007a). St Mary Graces stood to the north-east of the Tower of London, just beyond the boundary of the city wall (Grainger & Phillpotts 2011, 1). This abbey was extant from AD 1350-1540 and was established by Edward III, partly in thanks for his victory at Crécy, his escape from a shipwreck, and his survival of the Black Death (Honeybourne 1952, 16-17; Grainger & Phillpotts 2011, 7). St Mary Graces was also established as a memorial to all those who did perish of the Black Death, with some of the victims buried only meters away at the Royal Mint site (Grainger & Phillpotts 2011, 2).

The site was first excavated in 1972 with two small trial trenches being excavated (Grainger *et al.* 2008, 2-3; Grainger & Phillpotts 2011, 3). This was followed by further excavation in 1983 and 1984, before full excavation was conducted between 1986 and 1988 (Grainger *et al.* 2008, 3; Grainger & Phillpotts 2011, 3). Excavation revealed 420 individuals buried in the churchyard and abbey buildings (Bekvalac 2007a).

The churchyard cemetery, thought to be later 14th century (Grainger & Phillpotts 2011, 33) was located to the north of the church, extending westwards to overlay the earlier Black Death cemetery at the site (Bekvalac 2007a; Grainger & Phillpotts 2011, 33). This

churchyard cemetery went out of use by at the latest AD 1410 (Grainger & Phillpotts 2011, 33), but probably by AD 1405 (Bekvalac 2007a). It is considered that the 221 burials excavated from this area may be representative of an epidemic event; not only do the burials stratigraphically overlie the Black Death burials, but were also buried in 12 neatly aligned rows, following the boundaries of the mass burial trenches (Grainger & Phillpotts 2011, 33). However, over time these burials seem to become less distinct, although an area for infant and juvenile burials seems to have been located to the west of the site (Grainger & Phillpotts 2011, 33). The abbey also contained a number of burials throughout, including in the nave, choir, chancel, chapels, porch and cloister (Bekvalac 2007a). The abbey was in use between AD 1353 and AD 1538 (Bekvalac 2007a). One individual analysed was from the south chapel of the church. This is thought to be one of the lay burials present at the site, with no indication that any area was reserved for Cistercian burial rites or monks (Grainger & Phillpotts 2011, 106).

Of the 420 individual excavated, 389 were retained for analysis, with 283 of the individuals being adults and 106 non-adults (Bekvalac 2007a). Three individuals from this site were found to be fetal/perinatal/infantile and have been analysed; one individual was from the south chapel of the church, with two from the churchyard cemetery.

4.9 St. Benet Sherehog

As previously stated (See Above), the church and burial ground of St Benet Sherehog was excavated during wide-scale excavations at the site of 1 Poultry between 1994 and 1996 (Cowal 2008; Hill & Rowsome 2011, 4). The church, originally established in the 11th century was expanded during the 13th century and later destroyed during the great fire of London in 1666 (Cowal 2007a; 2008). After this destruction, the parish of St Benet Sherehog declined and the land was used by both St Benet Sherehog and St Stephen Walbrook as a burial site until 1853 (Cowal 2008).

Of the 280 individuals excavated, 212 individuals are from the post-fire burial ground, along with 18 burials which are considered to be pre-fire but still 17th century in date (Miles *et al.* 2008, 70). 167 of these post-Medieval burials were adults, with the remainder being non-adults (Cowal 2008). Of these non-adults, 19 were found to be fetal, perinatal or infantile and have thus been analysed in this thesis.

4.10 Broadgate

Broadgate cemetery, located in the east of the City of London, was excavated as part of an extensive development project extending Liverpool Street Station. The cemetery had been destroyed and disturbed in the 19th century when Broad Street and Liverpool Street stations were originally constructed (Dyson *et al.* 1987, 1), but was only fully excavated between 1984 and 1987 by the Museum of London Department of Urban Archaeology, now Museum of London Archaeological Services (MOLAS) (Museum of London 2015). This cemetery is located approximately 200m to the north of the City Wall and has revealed evidence of an archaeological sequence dating from the Roman to post-Medieval period (Dyson *et al.* 1987, 3).

This municipal cemetery, founded in 1569 by the City, was termed the 'New Churchyard' and intended to relieve the overcrowding of parish cemeteries at this time (Schofield & Maloney 1998, 216; Museum of London 2015). However, in practice this New Churchyard became the burial place for members of the poorer classes (Harding 2002, 95). As a result, the individuals were found to be tightly buried, with eight individuals per cubic meter found in some areas (Dyson *et al.* 1987, 8; Schofield & Maloney 1998, 216). Most individuals found were not buried in coffins, reflecting the low social status of the people buried in Broadgate. However, the end of the 17th century saw an increase in the number of richer individuals buried at Broadgate, and thus some lead coffins, and vaults with named individuals, were excavated (Museum of London 2015). Burials were interred at the New Churchyard until at least 1720 (Schofield & Maloney 1998, 216).

Several hundred individuals were excavated from Broadgate, but many were reburied on site, with *circa* 400 individuals being retained for analysis (Museum of London 2015). Of the *circa* 400 individuals retained, 21 were found to be fetal, perinatal or infantile and thus analysed for the purpose of this project.

4.11 St. Thomas' Hospital

Located to the north of St Thomas' Street, this post-medieval cemetery was excavated over an eight-week period in 1991 as part of a redevelopment to New London Bridge House, Southwark (Jones 1991, 3; Bekvalac 2007b). Discovery of a burial ground in this location

had not been considered likely, as documentary evidence was sparse (Jones 1991, 13). However, it was deduced that the burial ground excavated was 17th century and associated with St Thomas' Hospital (Jones 1991, 13-15). The hospital was founded possibly founded in 1106 (Jones 1991, 18), but was re-established at the site just north of St Thomas' Street in the 13th century (Jones 1991, 18; Roberts & Cox 2003, 255). St Thomas' Hospital was one of only three hospitals to survive the dissolution of the monasteries (Roberts & Cox 2003, 319).

The cemetery was only partially excavated, with many individuals remaining *in situ* (Bekvalac 2007b). Excavation revealed a series of three mass burial trenches dating to the 17th century. The burials excavated from this site are considered to be either those of paupers or evidence of an epidemic event (Bekvalac 2007b); there is evidence of rapid burial, with little or no soil found between the multiple layers of persons buried, indicative of a catastrophic burial event (Jones 1991, 31). However, it is thought that the majority of the burials had shrouds, although evidence for the use of coffins is extremely limited (Jones 1991, 30; Bekvalac 2007b).

In total, 227 articulated individuals were recovered, aligned east-west, with a large amount of disarticulated human bone found above, thought to be part of a 17th century charnel pit (Bekvalac 2007b). 193 individuals were retained for analysis and five individuals recovered were either fetal, perinatal or infantile.

4.12 St. Bride's Lower

The site of St Bride's Church has a history dating back to Roman times and has seen seven successive churches built on the site, the first of which was built in the 7th century (Huda & Bowman 1995, 135; Scheuer 1998, 100). The most renowned of the seven churches was designed by Sir Christopher Wren; built following the destruction of the previous church in the Great Fire of London, it opened in 1675 (Huda & Bowman 1995, 135; Scheuer 1998, 100). However, after a cholera epidemic in 1854, parliament forbade burial in the City of London (Huda & Bowman 1995, 135). The remaining church was also destroyed by an air raid in 1940, and so the modern church standing on the site today is a replica of Wren's original design (Scheuer 1998, 100).

Individuals buried in St Bride's Parish are split between the crypt and those in the two external cemetery grounds – The Upper Ground and Lower Ground cemeteries (Scheuer 1998, 103). The individuals analysed within this thesis are those excavated from the Lower cemetery. Excavation of the individuals buried at St Bride's Lower cemetery was undertaken due to redevelopment of the cemetery land and occurred in two primary phases between June 1991 and February 1992 (Miles & Conheeney 2005,1).

St Bride's Lower is located on the west bank of the river Fleet, which still flows beneath Farringdon Street today (Miles & Conheeney 2005, 1). The cemetery was founded in 1610, when the Bishop of London, Dr. Abbot, consecrated the area for the purpose of a new burial ground belonging to St Bride's Church (Miles & Conheeney 2005, 1). The cemetery was formed due to the congestion and overcrowding of the original churchyard (Upper Ground cemetery) associated with St Bride's to the south (Miles & Conheeney 2005, 1; Kausmally 2008). Thus, this alternate cemetery was used throughout the 17th to 19th centuries by those who lived in the parish of St Bride's (Kausmally 2008). However, those individuals excavated are thought to date primarily from the 18th and 19th centuries, and St Bride's Lower cemetery became known variously as the *'lower graveyard, Shoe Lane ground, new churchyard and, later, Fleet Market ground'* (Miles & Conheeney 2005, 1).

St Bride's Lower cemetery is one of the largest post-Medieval skeletal populations recorded from London (Kausmally 2008). Parish registers, although incomplete for the full period of the cemeteries use, detail the period between 1820-1849 exceptionally, with *'name, age at death, abode, date of burial, place of burial and cause of death given for 99% of all the individuals buried in the parish'* (Scheuer 1998, 102). Of the 4520 entries for this period that have been transcribed, 4208 of these individuals were buried in the Upper and Lower Ground cemeteries (Scheuer 1998, 103). Although this information cannot be ascribed to any of the skeletons excavated from these burial grounds, this information is useful in generating a picture 19th century London life and its people.

The individuals from the Lower Ground cemetery were excavated from two primary areas, with a total of 606 recovered (Miles & Conheeney 2005, 5; Kausmally 2008). However, only 544 were retained for analysis, with 47 of those individuals excavated from the vault and 497 excavated from the open yard (Kausmally 2008). Grave cuts were almost impossible to

identify due to the densely packed nature of the burial ground and the constant digging and re-digging of the site for burials (Miles & Conheeney 2005, 5). All of the burials were aligned east-west, except for 30 individuals who were orientated north-south (Miles & Conheeney 2005, 5; Kausmally 2008). Most of the burials excavated showed evidence of being buried in wooden coffins, though very few coffin plates survive or exist to identify these individuals (Kausmally 2008). The individuals were densely buried (Kausmally 2008), likely as a result of their low socio-economic status and the growing population in the parish. The Lower churchyard was the cheapest burial place in the parish and thus was not a preferred burial location, but was heavily used nonetheless throughout the 18th and 19th centuries (Miles & Conheeney 2005, 7). In addition, some of those buried in St Bride's Lower cemetery are likely to be from Bridewell workhouse and Fleet Prison, both of which were in the locality (Miles & Conheeney 2005, 8; Kausmally 2008). Of the 544 individuals retained for analysis, 52 were identified to be fetal, perinatal or infantile in age.

4.13 Chelsea Old Church

Chelsea Old Church, located in the parish of Chelsea, and was one of eight recorded burial grounds in this area, and the earliest of three parish cemeteries (Cowie *et al.* 2008, 19). Chelsea Old Church has a long and colourful history; famous parishioners include King Henry VIII, Elizabeth I and Sir Thomas Moore (Russett & Pocock 2004, 1). The church was originally founded shortly before AD 1120, although recent excavations have revealed evidence of Roman occupation predating this, suggesting the area has at least been inhabited, if not used for religious purposes, before this point (Russett & Pocock 2004, 16-17). Russett & Pocock state that the 15th century saw '*the beginning of the transition from rural backwater to built-up suburb, which was to transform the face of Chelsea over the next 400 years'* (2004, 28). From this point onwards Chelsea and the Old Church transformed and grew. By the 18th century a shift in demographic, due to the building of new brick terraces, for people of moderate means, away from the old village of Chelsea meant many now lived a long way from the Old Church (Russett & Pocock 2004, 108). The Population Book of March 1801 shows the parish of Chelsea had 12,080 inhabitants – this was ten times the population of the previous century (Russett & Pocock 2004, 110). As the population continued to grow, an extension to the churchyard was undertaken in 1790 (Russett & Pocock 2004, 110), although eventually a New Church was required and the Holy Trinity Church opened in 1830 (Russett & Pocock 2004, 108). Although Chelsea Old Church was situated on

the edge of the City of London, and more of a rural area during the 18th and 19th centuries (Museum of London 2009), this period saw a gradual transition, with Chelsea turning into a London suburb (Cowie *et al.* 2008, 13). By the mid-18th century Chelsea was seen as a fashionable resort, and was considered a wealthy, prosperous and healthy area of London, especially in comparison to other areas during this period (Cowie *et al.* 2008, 13).

Chelsea Old Church was completely destroyed by bombing during the Second World War and was rebuilt in the 1950s (Museum of London 2009). During the 1960s the building of a new vicarage and Petyt House saw the discovery of part of the 18th and 19th Old Churchyard (Russett & Pocock 2004, 149; Museum of London 2009). Permission was granted by parliament and the burials were exhumed (Russett & Pocock 2004, 149). In 2000 further redevelopment of the site was undertaken and it was discovered that the churchyard had not been entirely cleared (Russett & Pocock 2004, 149). MOLAS undertook excavations which recovered 290 individuals (Museum of London 2009), some whom were reinterred at Randall's Green Cemetery in Leatherhead (Russett and Pocock 2004, 149). The cemetery is thought to have been in use between 1712 and 1842 due to the coffin plates recovered (Cowie *et al.* 2008, 21).

Excavation revealed two vaults, two brick lined graves and a series of earth cut stacked graves (Museum of London 2009). The majority of individuals were recovered from the latter, with many having wooden coffins although some were found to be lead lined (Museum of London 2009). Interestingly, 25 of the individuals had legible coffin plates meaning biographical information regarding these individuals could be gathered (Cowie *et al.* 2008, 21). The individuals excavated from this cemetery are representative of the high socio-economic status of those living in Chelsea during the 18th and 19th centuries (Museum of London 2009).

Of the 290 individuals exhumed, 198 were retained for analysis (Cowie *et al.* 2008, 21, 40). Seven of those retained were identified as fetal, perinatal or infantile. Two of those analysed were excavated from the pelvic areas of adult female individuals (Cowie *et al.* 2008, 21; Museum of London 2009).

4.14 Cross Bones

The excavation area, of which Cross Bones Cemetery was a part, was located in the parish of St Saviour's, in the borough of Southwark (Brickley *et al.* 1999, 2). The cemetery is located to the east of Redcross Way and to the north of Union Street (Brickley *et al.* 1999, 2; Mikulski 2007). From documentary sources it was known that a post-Medieval burial ground was located in the area, having provided additional burial space for the parish (Brickley *et al.* 1999, 2). A small excavation of the area was undertaken in 1990 to confirm this (Brickley *et al.* 1999, 2), with MOLAS undertaking partial excavation of the Cross Bones burial ground in 1992 as part of redevelopment work for extension of the Jubilee underground line (Mikulski 2007). Excavation lasted from November 1992 through to February 1996 (Brickley *et al.* 1999, 3). Burials located on the site marked for redevelopment were exhumed, whilst those lying outside this area have remained *in situ* (Brickley *et al.* 1999, 3).

Cross Bones burial ground was one of seven burial grounds in the St Saviour's parish (Brickley *et al.* 1999, 5) and may have been established as early as the 16th/17th centuries (Mikulski 2007). It is considered to have originally been a single women's burial ground (a burial ground for prostitutes) for those working in the brothels on Bankside (Brickley *et al.* 1999, 5; Mikulski 2007). However, there is no documented evidence for this, except that the ground remained unconsecrated, in contrast to other burial grounds close by, including that of St Saviour's Workhouse burial ground (Brickley *et al.* 1999, 6). The burial ground came into 'proper' use in 1760 with the agreement of a lease for a new churchyard and from this date, until its closure in 1853 it remained a paupers' cemetery (Brickley *et al.* 1999, 7; Mikulski 2007). In total 148 individuals were excavated from the cemetery (Brickley *et al.* 1999, 4: Mikulski 2007), and are thought to date from the early to late 19th century phases of the site (Mikulski 2007). Those buried in the parish, and excavated and retained for analysis, are considered to be some of the poorest individuals in London at this time.

Excavation revealed that all 148 individuals were buried supine and aligned east-west (Mikulski 2007). Due to the densely packed nature of the burials it was often difficult to identify individual grave cuts, but all burials were in wooden coffins, although only two of the coffin plates discovered were partially decipherable (Brickley *et al.* 1999, 25-26). Evidence of clothing, shoes and burial shrouds were also recovered during excavation (Mikulski 2007).

Of the 148 individuals excavated, a very high proportion were found to be fetal, perinatal or infantile. Consequently, 58 individuals from this site have been analysed as part of this research.

4.15 Royal London Hospital

Located 1.6km east of the City of London, the Royal London Hospital is situated in Whitechapel to the south of Whitechapel Road (Fowler & Powers 2012, 5). Founded in 1740 the hospital was originally located in Aldgate, but construction began on its Whitechapel site in 1752, opening its first block in 1757 (Fowler & Powers 2012, 14-15). The hospital celebrated its awarding of a 'Royal' status in 1990, on its 250th anniversary (Fowler & Powers 2012, XVII).

Excavation was undertaken in 2006 at the Royal London Hospital as part of redevelopment work (Fowler & Powers 2012, 5). Investigations consisted of both watching briefs and excavation, with 262 burials being discovered in the northern part of Area A, formerly known as Bedstead Square (Fowler & Powers 2012, 5). Burials recovered from this area are considered to date between 1825 and 1841/1842, the period of expansion of the burial ground and before a new burial ground was opened to the south (Fowler & Powers 2012, 28). A second area, Area B, was also excavated with the burials unearthed believed to date from 1841 onwards (Fowler & Powers 2012, 5). These burials were exhumed and reinterred without any further analysis being undertaken (Fowler & Powers 2012, 5). Individuals and skeletal elements recovered from Area A are believed to be from unclaimed patients and show evidence of post-mortem and dissection, with some limbs/skeletal elements of multiple individuals interred together or alongside primary inhumations (Fowler & Powers 2012, 28). All of the primary burials were aligned east-west (Fowler & Powers 2012, 28).

Of the *circa* 262 individuals excavated from the Royal London Hospital burial ground, seven individuals were found to be fetal, perinatal or infantile.

4.16 Fetal Collection, Smithsonian Museum of Natural History

The fetal collection, held and curated by the Smithsonian Museum of Natural History, Washington D.C., is a medical collection with the majority of the individuals of known

biological age and sex. The collection was compiled by Aleš Hrdlička who arrived at the museum in 1903 (Hunt *personal communication*). Whilst at the USNM (United States National Museum), now the NMNH (National Museum of Natural History), he corresponded with many active and practicing medical professionals, who donated or exchanged human remains with the museum (Hunt *personal communication*).

Two of the primary, noteworthy donors were Frankline Paine Mall and Daniel Smith Lamb (Hunt *personal communication*)*;* the fetal collection is occasionally termed the Lamb Collection in reference to the latter donor. In total, Hunt lists 26 donors, but notes that from some accession records it is evident that other donors were active, though remain unidentified in the record, instead often Mall, Lamb or Hrdlička are the names given for these individuals (Hunt *personal communication*).

The individuals held within the collection are primarily from the Washington D.C. vicinity and the metropolitan areas on north-east coast of the United States of America. The majority of individuals were collected/donated from medical institutions in Washington D.C., Columbia and New York such as: Columbia Hospital, Freedman's Hospital, Howard University, and University of Maryland School of Medicine (Hunt *personal communication*).

Originally, 365 fetal individuals were collected and curated by the NMNH but today 320 of these are still present, with 45 having been damaged or mixed and as a result have been deaccessioned from the collection (Hunt *personal communication*). Of the 320 remaining the majority have secure biological age, sex and 'ancestry' identification (Hunt *personal communication*). When the collection was originally collected and curated the fetal individuals were assigned as either 'Black', 'White' 'Coloured' or 'Mulato' (Kosa 2002, 85). Although today, ethically, this practice and terminology would be inappropriate, the collection remains distinguished in this way.

Of the 320 individuals available for analysis, 140 were selected for the purposes of this study. These 140 were a random sample of the collection and encompassed individuals donated from various medical institutions and known to exhibit a variety of congenital conditions. The individuals assessed were also of varying biological ages, sex and 'ancestry' (Table 4.3). Only 44% of the collection was analysed due to time constraints of the research project.

TABLE 4.3 Individuals assessed from the Smithsonian Fetal Collection recorded by chronological age, biological sex and ancestry. For chronological age the abbreviation 'm.' is used for months. Ancestry is recorded as W (White), B (Black), C (Coloured) or U (Unknown).

Age Category	**Age (GWA)**	***N***	**Male**			**Female**				**Unknown**		
			W	**B**	**U**	**W**	**B**	**C**	**U**	**W**	**B**	**U**
3 m. *in utero*	13-14	3	1	1			1					
4 m. *in utero*	18	2	1							1		
5 m. *in utero*	22-23	2		2								
6 m. *in utero*	26-27	5	1	1			3					
6.5 m. *in utero*	29	1	1									
7 m. *in utero*	31-32	8	4		1	2	1					
8 m. *in utero*	35-36	3		3								
8-9 m. *in utero*	35-40	2	1	1								
9 m. *in utero*	40	12	1	4		1	5				1	
Full Term	40	3		2		1						
1 Day	40	1					1					
7 Days	41	1		1								
25 Days	43-44	1					1					
40 Days	45-46	1					1					
2 m.	48	1					1					
4 m.	56	1		1								
4.5 m.	58	1			1							
5 m.	60	1					1					
7 m.	68	1					1					
Fetus	< 36	75	25	9	1	26	7	1	1	1		4
Newborn	36-44	6	2	1			2			1		
Died At Birth	36-44	1	1									
Infant	> 44	3		1		1	1					
Child	> 44	4		2			2					
Unknown	-	1	1									
		140	39	29	3	31	28	1	1	3	1	4

Chapter 5: Methods

Multiple methods were employed to determine age-at-death and identify evidence of pathology for the fetal, perinatal and infantile individuals analysed within this thesis. These methods have been employed as both age estimates and pathological lesions have been used throughout this analysis as proxies for evidence of growth and health status. This chapter details these methods, elucidating why they were chosen, as well as critically considering their applicability to this study. Intra- and inter-observer error, and statistical methods employed are also detailed and discussed.

All osteological and palaeopathological assessments undertaken throughout this study have been in compliance with BABAO guidelines for the recording of human remains (Brickley & McKinley 2004), BABAO Code of Ethics (BABAO Ethics & Standards) and the BABAO Code of Practice (BABAO Ethics & Standards). Institution guidelines of the host museums (Museum of London and Smithsonian Institution) were also adhered to throughout data collection. No destructive analysis was performed and all assessment was undertaken macroscopically.

5.1 The Fetal, Perinatal and Infant Samples

Within this thesis a total of 423 fetal, perinatal or infant individuals have been assessed from 15 different archaeological and historical skeletal samples (See Chapter 4: Table 4.1 for detailed information). However, due to the nature of archaeological and historical collections, not all of the individuals had the skeletal elements required for full analysis. Therefore, the overall sample of 423 individuals can be broken down into three sub-samples:

1. Those individuals where both dentition and at least one skeletal element can be assessed to determine age.
2. Those individuals where only dental or skeletal elements can be assessed to determine age.
3. The Smithsonian Collection where age and/or sex and/or ‘ancestry’ is documented for the majority of individuals (See Chapter 4: Table 4.3 for detailed information).

For the first three research papers presented (Chapters 6, 7, and 8) individuals have been assessed within specific temporal contexts: Chapter 6 - Iron Age to Roman transition, Chapter 7 – post-Medieval London, Chapter 8 – 20th century medical collection. Therefore, within each of these chapters there are sub-samples of individuals with(out) dentition and skeletal elements available for assessment. Table 5.1 provides a breakdown of individuals by archaeological sample, detailing the number of individuals (*N*) assessed within each chapter (6, 7, and 8), the number of individuals who had dentition, the number of individuals who had a least one skeletal element available for metric assessment (either femur, tibia, humerus, or *pars basilaris*), and the number of individuals where growth disruption could be investigated (where both dentition and skeletal assessment could be undertaken).

This thesis only considers all 423 individuals in one research paper (Chapter 9). For Chapter 9, an additional 15 individuals from Medieval archaeological samples from London were included in assessment. The limited sample sizes of individuals from these sites means they were excluded from previous analyses but were included within overall pathological assessment. These medieval sites are: Medieval St, Benet Sherehog, Spital Square, East Smithfield and St. Mary Graces. Consequently, of the 423 individuals assessed, 210 had dentition available for assessment, whilst 390 had at least one skeletal element present. Growth disruption, consideration of dental versus skeletal age-at-death estimates, could be undertaken in 192 individuals.

5.2 Dental Ageing

Physiological assessment of dental growth and development is one of the primary methods utilised in archaeological studies to infer chronological age-at-death of non-adult individuals (Moorrees *et al.* 1963b, 1490; Gustafson & Koch 1974, 297; Hillson 2005, 207; Lewis 2007, 38; AlQahtani *et al.* 2014, 7). In non-adult individuals both dental development (the appearance and mineralization of teeth (Šešelj 2013, 39)) and eruption (the process of the tooth emerging through the gum and into the oral cavity (Šešelj 2013, 39)) can be assessed (Moorrees *et al.* 1963b, 1490; Lewis 2007, 38).

TABLE 5.1 Number of individuals, by study sample, with dental and/or skeletal elements available for age-at-death estimation. Number of individuals where assessment of growth disruption (dental versus skeletal age estimation) has been possible is also detailed. Numbers of individuals from the Smithsonian Fetal Collection with documented biological age, biological sex or 'ancestry' have also been provided.

	Total *N*	**Age-at-Death Assessment**		**Assessment of Growth Disruption (*N*)**	**Documented Biological Age**	**Documented Biological Sex**	**Documented 'Ancestry'**
		Dentition (*N*)	**Skeletal Elements (*N*)**				
Owslebury	23	13	16	11	-	-	-
Piddington	24	16	22	15	-	-	-
Barton Court Farm	52	19	48	19	-	-	-
St. Benet Sherehog	3	0	3	0	-	-	-
Spital Square	1	0	1	0	-	-	-
East Smithfield	8	6	6	5	-	-	-
St. Mary Graces	3	1	3	1	-	-	-
St. Benet Sherehog	19	9	17	0	-	-	-
Broadgate	21	13	15	10	-	-	-
St. Thomas' Hospital	5	4	5	4	-	-	-
St. Bride's Lower	52	29	50	28	-	-	-
Chelsea Old Church	7	5	6	5	-	-	-
Cross Bones	58	36	56	36	-	-	-
Royal London Hospital	7	1	6	1	-	-	-
Fetal Collection, Smithsonian Institute	140	58	136	57	50	132	132
	423	210	390	192			

Deciduous tooth development begins before birth at around the sixth gestational week (Scheuer & Black 2000a, 44; AlQahtani *et al.* 2010, 481), mineralising from around the 15th gestational week (Massler *et al.* 1941, 44; Lewis 2007, 38), with all deciduous dentition tending to be complete by around the fourth postnatal year (Liversidge & Molleson 2004, 172). The permanent dentition in contrast begin development around birth, continuing until *circa* 14 years of age, with eruption of the third molar occurring around 17-18 years of age (Lewis 2007, 38). Dental development is sequential, with teeth growing systematically from the tip of the crown to the root (Massler *et al.* 1941, 33; Blakey & Armelagos 1985, 371; Mays 1998, 11). Mineralisation begins with the tooth cusps and ceases with apex closure of the root (Lewis 2007, 39). During crown formation and mineralisation all teeth develop below the alveolar bone level (in the mandibular or maxillary crypt) (Liversidge & Molleson 2004, 173). Deciduous dentition develops and grows at a faster rate than that of permanent dentition, both regarding the enamel and dentine structures (Liversidge & Molleson 2004, 174). For further information regarding dental development see Chapter 3: Section 3.23.

Though both hereditary and environmental factors may affect tooth growth and development (Massler *et al.* 1941, 34; Heuzé & Cardoso 2008, 275), it has been widely established that these show less variability and fluctuation than other growth and development parameters (primarily that of skeletal growth and development) (Gustafson & Koch 1974, 298; Bolaños *et al.* 2000, 98; Humphrey 2000a, 194; Liversidge & Molleson 2004, 172). Furthermore, deciduous dentition is also considered more resilient than permanent dentition to environmental influences (Lewis 2007, 41). Consequently, the sequential development and mineralisation of the deciduous dentition is considered to be more robust, and therefore more accurate and representative when determining an age estimate.

5.21 Dental Ageing Methods Employed

Within this study dental development was recorded in accordance with Moorrees *et al.* (1963a; 1963b) and AlQahtani *et al.* (2010). Scoring of formation for each tooth was recoded in accordance with Moorrees *et al.* (1963a; 1963b) with results falling into one of fourteen stages; the first six stages relate to the formation of the dental crown, with the following eight stages depicting formation of the root and apex. For each tooth, a dental development score was attributed based on the level of growth (e.g. C ½ (Crown ½), Cc (Crown Complete), Ri (Root Initial)) (Moorrees *et al.* 1963a; 1963b). Once tooth formation stages had been

established, age-at-death estimates were calculated based on these stages using the London Atlas of Human Tooth Development and Eruption (AlQahtani *et al.* 2010). If dental development was considered to fall between two age categories, the mid-point between those age groups was recorded, with the largest error/range level for either category afforded.

Based on dental development/eruption charts it should be noted that both the permanent canine, and permanent first molar cusps may be present at birth (e.g. Moorrees *et al.* 1963a; AlQahtani *et al.* 2010). This study was able to detail and record numerous individuals who had at least one of these tooth cusps present and so it should not be forgotten that mixed dentition/dental development is possible from a very young age.

All dental age-at-death estimations have been given in gestational weeks throughout (GWA). Thus, those over 40 GWA are those who are suggested to be post-partum, but for ease of comparison between individuals and methods employed, ages have remained in gestational weeks (e.g. 52 GWA).

5.22 Methodological Limitations

Multiple methods of assessing dental formation/eruption have, over the last century, been developed (Lewis 2007, 39). The most prominent methods that have been typically used within bioarchaeological studies are those of Schour and Massler (1941a; 1941b) and Ubelaker (1978). However, there are limitations to both of these methods, with the former providing no information regarding the sample/material used or of the methods employed, as well as unclear, ill-defined and varying tooth formation stages utilised between the methods (Gustafson & Koch 1974, 298; AlQahtani *et al.* 2014, 70). Furthermore, the number of individuals assessed for the development of these reference methods was limited (Gustafson & Koch 1974, 298). Although these methods were '*ground-breaking for their time'* (Messer & Till 2013, 357; AlQahtani *et al.* 2014, 70), AlQahtani and colleagues produced the London Atlas of Human Tooth Development and Eruption (2010) as an evidenced based atlas, with illustrated tooth development for 31 age categories (AlQahtani 2010; AlQahtani *et al.* 2014, 71). As a result, this was the chosen atlas to establish dental age-at-death estimates for this study as it illustrates development levels of the enamel, dentine and pulp for each tooth (AlQahtani *et al.* 2014, 71), resulting in definitive distinctions between age categories. Furthermore, the drawings for each age category represent the median tooth formation and

eruption for that group (AlQahtani *et al.* 2014, 71). This method was also created using documented age-at-death archaeological collections, as well as current dental patients attending a range of institutions in London (AlQahtani *et al.* 2014, 71). Studies assessing the accuracy of this method also found it to have high levels of reproducibility (AlQahtani *et al.* 2014, 71) and was found to estimate chronological age more accurately that either the methods of Schour and Massler (1941a; 1941b), or Ubelaker (1978) (AlQahtani *et al.* 2014, 71). However, it has been found that this method may underestimate true chronological age (AlQahtani *et al.* 2014, 72-73).

Correlation of physiological dental development (Moorrees *et al.* 1963a; 1963b) to age defined stages (as presented in AlQahtani *et al.* 2010) provides an estimation of chronological age. Dental development has been considered as a good proxy for chronological age, as well as for the trajectory and pace of life histories (Šešelj 2013, 39). Dental development has also been found to be more accurate in younger infants and children, particularly those under 10 years of age (Bolaños *et al.* 2000, 97; 103; Lewis 2007, 39-41; AlQahtani *et al.* 2014, 71). Bolaños *et al.* (2000, 103) suggest this is as a result of the increased number of distinctive developmental stages during infancy and childhood. As a result, dental age-at-death estimates within this study have been used as a marker of 'true' chronological age. However, variation in the timing of tooth formation is not fully understood, and both population and sex differences may account for variation in the timing of dental development (Moorrees *et al.* 1963b, 1494-1497; Lewis 2007, 38-39; Heuzé & Cardoso 2008, 275). It is widely accepted that females reach developmental stages earlier than their male counterparts, with some females being between one and six months ahead in their overall dental development (Hillson 2005, 210; Lewis 2007, 38-39). The canine is considered to be the most sexually dimorphic tooth (Lewis 2007, 39), whilst the central maxillary incisor and first mandibular molar are considered to be the most accurate for estimating age-at-death for both males and females (Bolaños *et al.* 2000, 104). However, it is widely considered that variation in tooth development between individuals and populations is negligible (Ruff *et al.* 2013, 30; AlQahtani *et al.* 2014, 77). Therefore, although there is uncertainty surrounding the suitability and applicability of a reference method for assessment of archaeological individuals (Hillson 2005, 211; Heuzé & Cardoso 2008, 275), the methods developed by Moorrees *et al.* (1963a; 1963b) and AlQahtani *et al.* (2010) are likely the most appropriate for estimating age in unknown fetal, perinatal and infant individuals. This is because both

methods have been developed using large samples of known individuals, and AlQahtani *et al.* (2010) have created average developmental stages using both male and female individuals.

Consequently, within this thesis dental development/formation is considered to the most accurate method to establish age-at-death for non-adult individuals (Bang 1989, 213; Bolaños *et al.* 2000, 98; Lewis 2007, 58). Conversely, it must be remembered that dental development does not always inform us about, or correlate with, skeletal growth and development (Hillson 2005, 213; Šešelj 2013, 39). This is because dental development is less variable (AlQahtani *et al.* 2010, 481) and more robust against environmental factors (e.g. socioeconomic status, health insults, nutritional inconsistencies) than skeletal growth (Acheson 1959, 127; Garn *et al.* 1960, 1053; Bang 1989, 217; Hillson 2005, 207; Lewis 2007, 38; AlQahtani *et al.* 2010, 481; Šešelj 2013, 39; AlQahtani *et al.* 2014, 70).

5.23 Excluded Dental Ageing Methods

For the fetal, perinatal, and infantile individuals within this study, only dental development, not eruption, has been assessed and used to generate age-at-death estimations. This is because eruption only occurs from ~6 months post-partum onwards (See Massler *et al.* 1941; Liversidge & Molleson 2004; Hillson 2005; AlQahtani *et al.* 2010); as 6 months (64 GWA) was the upper age limit for this study it was unlikely that many individuals would show evidence of dental eruption, and those who did would be few in number. Furthermore, as this is an archaeological study, rather than clinical, the presence of any dental remains for such young individuals is limited. Tooth cusps of fetal, perinatal and young infant individuals are very small, easily lost, destroyed or misidentified in archaeological excavations (Gowland & Chamberlain 2002, 677; Lewis 2007, 42; Satterlee Blake 2018, 38). In addition, at this young age the mandibular and maxillary crypts are capacious in relation to the tiny developing tooth cusps which sit in them (Lewis 2007, 26; 42). Consequently, not only does this increase the chance of the dentition being lost both during and post-excavation, but assessment of eruption is impossible as the tooth cusps do not often sit, or remain in their original position (Lewis 2007, 26; 42). Furthermore, there is a clear distinction between the clinical and archaeological definition of eruption. Within clinical contexts eruption is considered to be the emergence of the tooth through the gum, whilst eruption is recorded archaeologically in hard tissues when the tooth emerges from either the mandibular or maxillary crypt. Therefore, differences in the interpretation of eruption often means that archaeological individuals are

recorded to be older than they would be clinically, as their eruption sequence is identified earlier (Lewis 2007, 41).

Eruption is also more variable in response to a range of factors including biological sex, population differences, hormonal and metabolic disturbances, as well as disease status (Moorrees *et al.* 1963b, 1490; Gustafson & Koch 1974, 299; Bang 1989, 217; Hillson 2005, 212; Lewis 2007, 41; e.g. Demirjian 1990; Holman & Jones 1998; Holman & Yamaguchi 2005). Dental eruption also only provides detail of a '*specific phase of short duration*' (Moorrees *et al.* 1963b, 1490; Huda and Bowman, 1995, 138). Consequently, dental eruption is considered to be less reliable than dental development/mineralisation (Moorrees *et al.* 1963b, 1490; Bang 1989, 216; Huda and Bowman, 1995, 138; Lewis 2007, 41) and therefore, dental eruption has not been assessed or considered within any chapters of this study: estimation of age-at-death using the dentition relies on assessment of stage of development alone.

Although histological and radiographic assessment of dental development has commonly been used within archaeological investigations (Gustafson & Koch 1974, 298), this thesis did not employ these techniques to aid estimation of age-at-death. The benefit of histological assessment is that there is no extrapolation and conversation of data to reference methods to estimate age (Huda and Bowman, 1995, 138; 140; Lewis 2007, 42). Instead, age can literally be counted through analysis of the incremental markings within the dental microstructure (Massler *et al.* 1941, 34; Huda and Bowman, 1995, 136; Lewis 2007, 42). Conversely, radiographic assessment is considered to be the least accurate method to estimate age as X-rays only show developing teeth which have mineralised enough to be observable (Huda and Bowman, 1995, 137; Hillson 2005, 225). Consequently, radiographs tend to underestimate age-at-death. However, neither of these methods were employed due to the financial, ethical and time constraints of this project. Given the large sample size (N=423) of individuals assessed within this thesis and their varying locations (See Chapter 4: Table 4.1 and Figure 4.1), the ability to radiograph all individuals would have relied on the samples being able to be removed from their locations and the availability of equipment for use. Furthermore, histological assessment is destructive, requiring thin sections to be taken from the developing teeth. Therefore, access and permission to undertake histological assessment is unlikely to have been granted.

5.3 Skeletal Ageing

Metric assessment of fetal, perinatal and infant skeletal remains is the most commonly employed method for determining chronological age-at-death (Humphrey 2000b, 30; Lewis 2007, 43; Utczas *et al.* 2017, 1). This is likely due to skeletal elements being both better preserved in the archaeological record than the dentition (Gowland & Chamberlain 2002, 677), as well as recognised and collected by the archaeologist. Historically, growth and development were thought to be a good proxy for age estimation (e.g. Fazekas & Kósa 1978; Scheuer *et al.* 1980). Although this may hold true for some populations, particularly those of high socioeconomic status, it has been long recognised that skeletal growth can be severely affected and disrupted by detrimental environmental factors (Cardoso 2007, 223). As a result, although growth, or metric assessment of skeletal elements, should enable assessment of chronological age, in principle it is unlikely to be accurate unless the population and individuals assessed within it have experienced a good environment in which to grow, akin to the modern reference populations.

As with assessment of the dentition, age-at-death estimates derived from metric assessment of long bones have always been given in gestational weeks of age (GWA) throughout.

5.31 Skeletal Ageing Methods Employed

For each individual assessed as many bones as possible of the cranium, upper and lower limbs, and pectoral and pelvic girdles were measured. Although the manuscripts presented rely primarily on age estimates derived from the long bones and *pars basilaris*, it is worth noting that additional measurements were recorded; intra- and inter-element growth disruption has not been considered within any of the papers given, yet this aspect of investigation has been highlighted for future studies (See Chapter 11). All measurements were taken using digital sliding callipers (accuracy of +/-0.02mm), with all results recorded to the hundredth of the millimetre (although it is recognised that this is beyond the scope of human accuracy). All measurements undertaken to the skeletal elements listed were in accordance with the guidelines for assessment outlined in Fazekas & Kósa (1978) and subsequently, Schaefer *et al.* (2009). Results were recorded in a metric database developed using Microsoft Excel.

Throughout the following papers the long bone diaphyseal lengths have been used to calculate chronological age estimates. These were established using the published linear regression equations for long-bone diaphyseal lengths by Scheuer *et al*. (1980). Only the long bones of the skeleton have regression equations available for use in assessment. Where both left and right skeletal elements were available for assessment both were analysed and had age estimates generated, with the average age for that element used in analysis. The chronological age estimate generated has typically been plotted with the error level (+/- *X* gestational weeks of age) given as a range. This has enabled comparison of age-at-death estimates generated from each of the long bones to be compared against one another for each individual to highlight difference in growth between the skeletal elements. Where dental age-at-death estimates are available for consideration, identification of overall skeletal growth disruption is possible.

The *pars basilaris* has also been used within some of the following studies to produce age-at-death estimations. This bone was utilised as it is often recovered archaeologically due to its robust nature (Redfield 1970, 207; Scheuer & Maclaughlin-Black 1994, 377), but also because it is known to be indicative of certain aging thresholds, with both its size and morphology often found to be correlated strongly with age (Redfield 1970; Scheuer & Maclaughlin-Black 1994; Lewis 2007, 44). This is because the base of the cranium is considered to be the most stable area during growth and development (Redfield 1970, 207). Metric assessment of the *pars basilaris* was undertaken in accordance with the methods published by both Fazekas & Kósa (1978) and Schaefer *et al.* (2009), with sagittal length, maximum length and maximum width all recorded where possible. Employing methodology initially established by Redfield (1970), the *pars basilaris* measurements were considered against one another to determine if sagittal length, maximum length or maximum width was the largest measurement recorded. Dimensions of the *pars basilaris* have been identified to correlate with stages of fetal and postpartum growth and development: when maximum width is less than sagittal length the individual is considered to be less than 28 GWA (Scheuer & Maclaughlin-Black 1994); when maximum width is greater than sagittal length, but maximum width is less than maximum length the individual is between 28 GWA and 5 months of age postpartum (60 GWA) (Redfield 1970; Scheuer & Maclaughlin-Black 1994); when maximum width is greater than maximum length the individual is over five months of age, postpartum (Redfield 1970) (See Table 5.2 for summary). Thus, maximum width was

compared against sagittal length and maximum length respectively and according to which measurement was greatest an age bracket was afforded. However, as this only provides large, broad age ranges, measurements were directly compared against those given in Scheuer & Maclaughlin-Black (1994) and more specific age estimations/age estimation ranges were established. As no ranges or error levels were given for this method (Scheuer & Maclaughlin-Black 1994) for each specific age category, where measurements fell within a range of age categories the mean age category has been plotted, with the minimum and maximum age categories used as upper and lower age ranges. For example, a measurement which fell into the 3 weeks, 4 weeks, 7 weeks and 3-months age categories (43-52 GWA), has a mean point of 47.5 GWA, with a range of +/- 4.5 GWA.

TABLE 5.2 Age categories based on the metric dimensions of the pars basilaris.

0 - 28 GWA	Maximum Width less than Sagittal Length
28 - 60 GWA	Maximum Width greater than Sagittal Length Maximum Width less than Maximum Length
60 GWA+	Maximum Width greater than Maximum Length

Additionally, for some of the analyses in the following manuscripts the raw, metric data has been used to compare against reference data and growth charts, with no estimate of age afforded. Reference data that has been used includes the published standards of Fazekas and Kósa (1978) and Maresh (1970) as well as clinical growth charts (See Section 5.33). These reference data sets have been used to show that individuals assessed in this study, of the same chronological age group based on dental development, typically have skeletal metrics which fall below expected. Consequently, this comparison of raw data has once again been employed to provide evidence of growth disruption within the skeletal samples assessed. Multiple reference data sets were utilised to enable all individuals (both pre- and postnatal) and varying skeletal elements to be considered.

5.32 Methodological Limitations

This use of linear regression equations was employed by the author as it is one of the only methods available that provides an error level for age-at-death estimation, and is widely used in other studies making results of this assessment comparable (Lewis & Gowland 2007, 120; e.g. Mays 1993; Lewis 2002a; Halcrow *et al.* 2012). However, this linear regression method has been criticised, suggested to age individuals in a way which mimics the demographic make-up of the sample used to create the regression models (Gowland & Chamberlain 2002, 678; 684; Lewis & Gowland 2007, 120). This has been a common criticism of many age-estimation techniques and methods, with studies found to often reflect the age distribution of individuals within the reference sample (Gowland & Chamberlain 2002, 678; e.g. Bocquet-Appel & Masset 1982). In fact, within this thesis only six individuals were estimated to have long bone elements that were over 46 GWA; the regression equations utilised were developed only considering individuals aged up to 46 gestational weeks of age (GWA) (Scheuer *et al*. 1980). This is in comparison to 82 individuals having dental age estimates which exceed 46 GWA. Therefore, although many of these individuals do show evidence of growth disruption, such clearly defined clustering of skeletal age estimates under 46 GWA is likely to be a product of the method and a mimicry between the reference and sample population. However, to limit the effect of this bias other studies have employed Bayesian statistics to redistribute the age estimations generated (e.g. Gowland & Chamberlain 2002). As discussed previously (See Chapter 2), Bayesian analysis considers the likelihood of individuals falling within age categories, in comparison to a natural mortality profile derived from perinatal and infant life tables (Lewis & Gowland 2007, 122). Therefore, by employing this assessment,

age estimates are redistributed by probability (Gowland & Chamberlain 2002, 684). However, Bayesian analysis considers all individuals as a whole, meaning age estimates cannot be generated for single individuals (Lewis & Gowland 2007, 127). This limits detailed assessment of individuals and the potential for identifying growth disruption. As a result, because this research aims to focus on identifying growth disruption within individuals, Bayesian statistical assessment has not been employed.

Assessment of data collected within this study against a range of reference data will of course incur limitations due to the varying skeletal samples from which the reference methods were constructed. The inherent variation between samples may lead to limitations, and although an appropriate reference sample should be used for comparison, it is almost impossible to do this for archaeological assessments (Scheuer & Black 2000b, 13). As discussed previously regarding dental development, skeletal development also varies between population and is dependent on a range of variables including biological sex, ethnicity and health status (Nyati *et al.* 2006, 135). Growth is sex specific (Sofaer 2011, 287) and males and females are well known to have varying growth strategies (Barker *et al.* 2012, 32). Females are found to skeletally grow and mature more rapidly than males (Humphrey 2000a, 195; Lewis 2007, 48) and males also have a more precarious growth strategy (Barker *et al.* 2012, 32). This is why males are often considered to be more 'frail' and why might expect to see more males with growth disruption and within the archaeological burial record more generally (Lewis 2018, 113). Difference between populations are also evident with American Black non-adults often developing/maturing earlier than American white non-adults (Lewis 2007, 46-47; Nyati *et al.* 2006, 135; 138). African American non-adults have been found to have longer legs than Mexican American and Caucasian American non-adults, whilst Caucasian American individuals have the greatest trunk length of these three groups (Malina *et al.* 1987; Martorell *et al.* 1988; Nyati *et al.* 2006, 135). It is also being found that there is a racial disparity in health, which in turn reflects growth status (Kuzawa & Sweet 2009, 2). It has been considered that racial disparity is closely entwined with socioeconomic status, availability of health care, education and employment, resulting in a perpetuating cycle of disadvantage amongst certain population groups (Kuzawa & Sweet 2009, 2-4). Consequently, although suspected that there are biological and genetic differences in growth timings and tempos between populations, it is less clear as to the extent of these differences and whether they are purely biological difference or ones bound within biocultural and social spheres. Regardless,

the fact that many methods utilised to determine fetal, perinatal or infant age-at-death amalgamate biological sex and ethnicity into their reference measurements adds a potential additional level of error.

Concerns regarding Fazekas and Kósa (1978) have also been raised as this reference method was developed using individuals of unknown age (Scheuer & Black 2000b, 13; Lewis 2007, 44). In addition, others have suggested that disparities between archaeological and radiological assessment will exist due to the comparison of wet versus dry bone (Lewis 2007, 43). Thus, the reference sample of Maresh (1970) may reveal greater metric difference as a result. However, research has suggested that there is no significant difference between archaeological and radiographic measurements (e.g. Warren 1999; Schillaci *et al.* 2012), and that in fact the Maresh data set (1970) generally reflects a normal pattern of human growth (Schillaci *et al.* 2012, 497). Therefore, data given by Maresh (1970) is suggested to be highly suitable for use in observing growth patterns in archaeological and historical skeletal collections (Schillaci *et al.* 2012, 497).

Despite errors between long bone length and gestational age being well documented, such limitations are troublesome to control for (Lewis 2007, 43). This is as a result of all the potential stressors and factors (discussed previously in Chapter 3) which can alter and regulate fetal, perinatal and infant growth. Therefore, assessment and correlation of long bone length to chronological remains the primary method employed for age-at-death determination of fetal, perinatal and infantile individuals in archaeological studies. Thus, to make this study both applicable and comparable to the existing literature, skeletal ages-at-death have been calculated using regression equations (Scheuer *et al.* 1980) as well as utilising reference data sets of Fazekas and Kósa (1978) and Maresh (1970) throughout. Consequently, the need for a consistent and accurate method for ageing fetal, perinatal and infant skeletal remains is highlighted.

Although this thesis in no way attempts to review or overhaul these reference datasets, it highlights the fact that more studies should consider multiple methods of age assessment – those of both dental and skeletal growth – to identify clearer patterns of both growth and health disruption (Šešelj 2013, 44).

5.33 Clinical Assessment of Growth

In an attempt to overcome some of the limitations of the methods listed and discussed above, growth and growth disruption has also been considered utilising a commonly employed clinical methodology.

Growth charts are well established within a clinical context (Altman & Chitty 1997, 174; Beukema *et al.* 2008; Evans 2010, 27; Cole 2012; Dodrill 2016, 267) and a plethora of varying growth charts have been developed for a wide range of fetal, perinatal and infant physiological aspects. Although many of these reference methods consider crown-rump length, biparietal breadth, fetal weight, abdominal circumference and head circumference (e.g. Maresh & Deming 1939; Lubchenco *et al.* 1966; Gindhart 1973; Tanner & Whitehouse 1975; 1976; O'Brien & Queenan 1981; Hohler 1984; Jeanty & Romero 1984; Jeanty *et al.* 1984a; 1984b; Deter & Harrist 1992; Altman & Chitty 1997), characteristics unobservable and unmeasurable within archaeological analyses, growth charts also exist for a variety of long bone diaphyseal lengths. Today, growth charts are usually presented as centiles (Humphrey 2000b, 30) and by comparing fetal, perinatal and infant skeletal measurements to growth charts, the centile(s) within which individuals align are recorded. Centiles are provided whereby the 50th centile represents the median measurement for that age group, with 50% of individuals falling above and below that point (Dodrill 2016, 267). Therefore, the 25th centile is where 25% of individuals fall below this measurement, with the 75th centile being where 25% of individuals are above this measurement. It is suggested that those individuals who fall outside of the 10th and 90th percentiles are clinically significant (Kiserud *et al.* 2017, 18; 20). Today, these extremes signify individuals who require further observation, monitoring, care or intervention. Indeed, those whose skeletal growth falls below the 10th percentile are often found to have negative birth outcomes (Kiserud *et al.* 2017, 21). Thus, by utilising clinical growth charts, and transposing archaeological skeletal measurements on to these charts, this thesis has been able to highlight those individuals who appear to fall into these 'critical' percentiles.

A limitation of this method is that these growth charts are unable to provide age estimates from skeletal dimensions. Consequently, only individuals who had dentition available for assessment could have their skeletal measurements plotted onto these charts. Therefore,

although not used within this thesis to determine accurate age-at-death estimates, employing comparison of skeletal measurements to growth charts has corroborated that many individuals were not only experiencing growth disruption, but experiencing it to an extent where birth outcome and health was likely to be severely affected.

For prenatal (40 GWA or less) the clinical growth charts employed are those derived from the World Health Organisation, as given in Kiserud *et al.* (2017, 19-20). These growth charts are only available for diaphyseal lengths of the femur and humerus, and do not provide different growth centiles by biological sex, nor by ethnicity. Therefore, these growth charts have been utilised in the assessment of archaeological individuals dentally aged to be 40 GWA or less where biological sex and ethnicity is unknown. For postnatal individuals, growth charts given by Maresh (1970) have been used due to the fact this data set has been found to represent normal, healthy growth, and be a suitable reference for archaeological individuals (Schillaci *et al.* 2012, 497). Where individuals of known biological sex have been assessed (individuals from the Smithsonian collection) the specific male or female growth charts have been considered. However, for archaeological individuals where biological sex is unknown, long bone measurements have been plotted against both male and female growth charts.

The major limitation of these growth charts is of course that they have been formulated using modern populations and are consequently less comparable to archaeological skeletal material (Lewis 2007, 72). However, as these charts are not able to determine age-at-death, they are still very informative in providing an insight into those individuals experiencing extremes of growth and growth disruption.

5.34 Excluded Skeletal Ageing Methods

To date, a variety of other ageing methodologies utilising skeletal remains have been established from which chronological age can be interpreted. For fetal, perinatal and early infant individuals these have tended to be the fusion and completion of the tympanic ring, closure of the *sutura mendosa*, fusion of the mental symphysis in the mandible and the closure of the metopic suture.

The application of these methodologies was not employed within this thesis as this investigation aimed to assess and correlate evidence of metric growth disruption with health

status. As none of these methods rely on metric assessment they were not included in analysis. Furthermore, closure of the *sutura mendosa*, fusion of the mental symphysis and closure of the metopic suture are all purported to occur during the first postnatal year. Consequently, as all of the individuals assessed within this study are suggested to be within this age range, the presence/absence of these characteristics would reveal little in regards to determining a more accurate age-at-death estimation.

5.4 Assessment of Growth Disruption

It has been widely accepted that the relationship between metric assessment and chronological age is not a reliable one (Lewis 2007, 43). Consequently, this study has employed metric assessment to calculate age-at-death estimations, not to use as 'true' chronological estimates of age, but to compare against dental age-at-death estimates, which are considered to be more accurate (Cardoso 2007, 223). This practice of comparing skeletal and dental age-at-death estimations has often been employed to identify evidence of physiological stress (Humphrey 2000b, 29; Lewis 2007, 45). This means that throughout the following manuscripts consideration of skeletal age estimations has often been in conjunction with dental age-at-death estimates, with the discrepancies between the two being explored as evidence of both growth and health disruption as a consequence of stress exposure (Lewis 2007, 45).

5.5 Intra-Observer and Inter-Observer Error

To determine the accuracy of metric assessment both intra-observer and inter-observer error or TEM (technical error measurement was calculated). This was employed to determine the level of accuracy in recording measurements taken from the skeleton and demonstrate that the author, and the methods employed, are reliable.

Intra-observer and inter-observer error was calculated for two bones of the skeleton: diaphyseal length of the femur and the maximum length of the *pars basilaris.* These two bones were chosen for assessment as measurements of long bones and the *pars basilaris* are the most commonly used throughout this study to generate age-at-death estimates. Measurements from 10 individuals were collected twice by the author, for each element for calculation on intra-observer error. Measurements from 10 individuals were collected once by

the author, and once by a osteologist (who is not a specialist in fetal/infant analysis) for each of the elements assessed to calculate inter-observer error.

Calculation of Relative TEM (%) was based on the equation:

$$\text{Relative TEM (\%)} = \frac{\text{Absolute TEM}}{\text{VAV}} \text{ X } 100$$

This required both VAV (Variable Average Value) and Absolute TEM to be calculated first and this was undertaken in accordance with the methods outlined by Perini *et al.* (2005) and Ulijaszek and Kerr (1999). Absolute TEM was calculated using the following equation:

$$\text{Absolute TEM} = \sqrt{\frac{\sum d^2}{2\text{n}}}$$

Where:
$\sum d^2$ = Sum of deviation raised to the second power
N = Number of skeletal elements measured

This is where the difference between measurement 1 and measurement 2 is calculated (the deviation) for each of the 10 femoral or *pars basilaris* elements assessed. This deviation is then squared for each of the 10 elements and all 10 are summed, calculating the sum of deviations squared. This is then divided by two times the number of elements assessed – in this study 2 x 10 elements = 20. Thus, the square root of, the sum of deviations squared, over 20, calculates absolute TEM.

Variable average value was calculated by determining the mean measurement (between measurement 1 and measurement 2) for each of the 10 skeletal elements assessed. The ten

measurements were then summed and divided by the number of elements assessed (in this case ten) to generate an overall average for the measurement. Using VAV and Absolute TEM, relative TEM (%) can now be generated.

Tables 5.3 and 5.4 detail the process of calculating Relative TEM (%) for intra-observer error. These tables detail the repeated measurements collected for both the femora and *pars basilaris* elements, the deviations, deviations squared, and average measurements for each of the elements. Sum of deviations squared, Absolute TEM and VAV have been given for both the femoral and *pars basilaris* assessments. Table 5.5 provides the intra-observer and inter-observer error (TEM %) for both the femoral and *pars basilaris* measurements. The low percentages indicate that there is a high rate of precision between these measurements (Perini *et al.* 2005, 87). 10% TEM has been considered an acceptable lever of error (Perini *et al.* 2005, 89), thus, the TEM percentages for this study fall substantially beneath this and indicate that there is a high level of precision in the measurements recorded and used within this study.

TABLE 5.3 Calculations for Sum of Deviations Squared, Variable Average Value and Absolute TEM for intra-observer error of femoral diaphyseal length measurements (mm).

	1	**2**	**3**	**4**	**5**	**6**	**7**	**8**	**9**	**10**
Measurement 1	81.99	60.58	56.25	76.74	51.63	58.5	47.94	57.45	72.07	68.82
Measurement 2	81.95	60.97	56.02	76.25	51.97	58.59	48.05	57.48	73.39	69.09
Deviations	0.04	-0.39	0.23	0.49	-0.34	-0.09	-0.11	-0.03	-1.32	-0.27
Deviations^2	0.0016	0.1521	0.0529	0.2401	0.1156	0.0081	0.0121	0.0009	1.7424	0.0729
Average	81.97	60.775	56.135	76.495	51.8	58.545	47.995	57.465	72.73	68.955

Sum of Deviations Squared = 2.3987

Variable Average Value = 63.2865

Absolute TEM = 0.346316

TABLE 5.4 Calculations for Sum of Deviations Squared, Variable Average Value and Absolute TEM for intra-observer error of pars basilaris maximum length measurements (mm).

	1	**2**	**3**	**4**	**5**	**6**	**7**	**8**	**9**	**10**
Measurement 1	18.86	14.69	16.72	9.65	13.56	14.93	15.91	12.00	13.25	16.37
Measurement 2	18.66	14.92	16.64	9.7	13.64	14.72	15.99	12.05	13.46	16.24
Deviations	0.2	-0.23	0.08	-0.05	-0.08	0.21	-0.08	-0.05	-0.21	0.13
Deviations^2	0.04	0.0529	0.0064	0.0025	0.0064	0.0441	0.0064	0.0025	0.0441	0.0169
Average	18.76	14.805	16.68	9.675	13.6	14.825	15.95	12.025	13.355	16.305

Sum of Deviations Squared = 0.2222

Variable Average Value = 14.598

Absolute TEM = 0.105404

TABLE 5.5 Intra-observer and inter-observer errors (Relative TEM %) for femoral diaphyseal length measurements and pars basilaris maximum length measurements.

Intra-observer Relative TEM	Femoral Length	0.54722 % (0.5%)
	Pars Basilaris Length	0.722044 % (0.7%)
Inter-observer Relative TEM	Femoral Length	0.689823 % (0.7%)
	Pars Basilaris Length	0.702692 % (0.7%)

5.6 Pathological Assessment

Many issues surround the field of fetal/infant osteology and palaeopathology – the most predominant being that of determining true evidence of pathology. Historically, fetal, perinatal and infant remains have been neglected within archaeological literature (Buckberry 2000, 2; Lillehammer 2015, 79) Many excavators simply refrained from exhuming these individuals, or literally disposed of them (Becker 2006, 55), meaning that very few were considered in a worthwhile manner in archaeological reports before the 1980s (Mays *et al.* 2017, 38). However, the last two decades have seen an increasing interest in these young individuals, which has led to a variety of studies focussing on the infant and child within archaeology. Despite this increased interest, pathological analyses of fetal, perinatal and infant individuals have been somewhat limited until recently (e.g. Lewis 2007; Lewis 2017a), and as such disease manifestations, processes and aetiologies are still largely non-specific and unknown.

Assessing pathological changes in non-adults is considered to be particularly challenging (Lewis 2018, 113). Despite this, numerous investigations into non-adult health have been undertaken (e.g. Lallo *et al.* 1977; Schultz 1984; 1989; Stuart-Macadam 1988; Anderson & Carter 1994; 1995; Lewis & Roberts 1997; Ortner & Ericksen 1997; Ortner & Mays 1998; Ortner *et al*. 1999; Mays *et al.* 2007; Brickley & Ives 2006; 2008; Lewis 2011; 2012; 2017a). Pathological changes in such young individuals are considered to be particularly important to

determine as they provide vital insights into intrauterine as well as maternal health. Fetal, perinatal and infant remains have become synonymous as the most sensitive members of past societies (Goodman & Armelagos 1989, 239; Lewis 2000, 39; Halcrow *et al.* 2018, 86), likely to express skeletal changes as a result stress due to their immature immune systems and higher degree of bone turnover (Lewis 2000, 43; 2002a, 211; 2018, 115; Satterlee Blake 2018, 44). These individuals also provide a unique proxy by which maternal wellbeing, experiences and health can be considered (Satterlee Blake 2018, 34; 43).

Macroscopic assessment of fetal/perinatal/infant pathology is most commonly employed (Satterlee Blake 2018, 42) and some conditions/diseases are clearly identifiable and diagnosable despite the young age and immature development of these individuals. A range of causes (disease/infection (specific and non-specific), nutrition (metabolic), trauma (including accidental (i.e. birth trauma) and deliberate trauma) and congenital conditions) can all cause identifiable changes to fetal, perinatal and infant skeletal remains. A variety of congenital disorders (e.g. hydrocephaly, anencephaly) as well as diseases such as syphilis, tuberculosis, rubella, and metabolic deficiencies can all be transmitted via the placenta during pregnancy (Lewis 2018, 113). Various case studies exploring evidence of several congenital and specific infectious conditions affecting non-adult individuals have been published (e.g. Richards & Anton 1991; Murphy 1996; Dabernat & Crubézy 2009; Dudar 2010), though evidence still remains limited, primarily as a consequence of the rarity of such conditions and the limitations of preservation and identification. Additionally, many of the predominant causative factors of fetal/perinatal/infant death leave no discernable traces on the skeleton (Lewis 2018, 112), making assessment of early life health disruption in the archaeological record somewhat more troublesome. When a specific condition is suspected, analysis and diagnosis may be easier as a clear pattern to the pathology emerges of distinctive changes (e.g. Hutchinson's incisors and mulberry molars in congenital syphilis). However, non-specific stress, such as metabolic disturbances, general infections, psychosocial stress and sometimes even trauma, is more commonly represented as periosteal new bone formation (NBF) (Lewis 2018, 114).

5.61 Methodological Limitations

A major hindrance to the development of fetal/infant palaeopathology is the limited literature, both bioarchaeological and clinical, which considers normal skeletal anatomy of the growing

child. The clinical literature is not as concerned with the skeletal manifestations of many diseases, as diagnoses are derived from the soft tissue. Indeed, many conditions leave no, or very few skeletal traces, instead primarily affecting soft tissues; e.g. plague, cholera, small pox and rubella (Ortner 2008, 191). Furthermore, acute conditions may result in pathological lesions being quick to heal, or indeed result in rapid death, meaning no skeletal lesions were able to manifest (Roberts 2000, 145; Ortner 2008, 191-192).

The major limitation however, within pathological studies of such young individuals is the differentiation between pathological NBF and that of NBF associated with normal growth (Lewis 2017, 2-3) (For a comprehensive discussion of normal fetal/perinatal/infant skeletal growth and development see Chapter 3: Sections 3.1). Response by the skeleton to environmental onslaughts (be they infectious, metabolic, traumatic) results in pathological new bone formation. Healthy NBF, associated with normal growth, is known to appear in an almost indistinguishable way to pathological NBF, as the process by which the bone forms is identical (Lewis 2000, 42). Furthermore, NBF is an anticipated observable change in these very young, and rapidly growing, individuals (Lewis 2017, 8). Consequently, distinguishing this normal bone growth from pathological bone growth is consequently highly problematic. Therefore, timing of growth spurts and age-at-death estimations of the individuals assessed must be taken into consideration, as various clinical investigations have suggested that NBF is a common finding in individuals at least over 1 month of age (44 GWA) (Shopfner 1966; Scheuer & Black 2000a, 24; de Silva *et al.* 2003; Kwon *et al.* 2002). However, little is still known about the presence and implications of NBF found on individuals younger than 44 gestational weeks, and many pathological conditions do still result in the proliferation of NBF throughout the skeleton, primarily to the long bones (Lewis 2000, 43). Thus, being able to distinguish between normal patterns of growth, and evidence of abnormal growth as a result of a disease process is complex (See Chapter 9 for further discussion).

Furthermore, bone can only respond in one of two ways to health/disease insults (bone growth and bone destruction) and thus, although there a multitude of possible pathologies, the resulting skeletal changes will be similar (Goodman & Armelagos 1989, 228; Gowland 2004, 139; Ortner 2008, 192-193; Roberts & Manchester 2010, 7). As discussed in Chapter 3: Section 3.2, NBF, regardless of whether it is normal or pathological, is secreted as woven bone, sometimes referred to as fibre bone (Ortner 2003, 19; Brickley & Ives 2008, 23).

Woven bone is immature, highly disorganised bone (Mays 1998, 6; Scheuer & Black 2000a, 30; White *et al.* 2012, 35) typically found in fetal/perinatal/infant individuals who are growing as it is formed very quickly (White *et al.* 2012, 35). However, the distinctive grey appearance of woven bone is also synonymous as evidence of pathological NBF – a physiological response by the body to an environmental stressor. Lamellar bone is bone that has remodelled and matured, as in the normal bone formation process, or can also represent healing bone if the stimulus was pathological. Lamellar bone is well organised, striated bone (Scheuer & Black 2000a, 30; White *et al.* 2012, 35). Consequently, it is imperative to recorded whether NBF is either woven or lamellar in appearance as changes in these states have important implications for pathological assessment. However, although bone responds quickly to insults in such young individuals, it is still unknown how long certain conditions take to present on non-adult skeletal remains (Lewis 2000, 40). Therefore, evidence of lamellar bone - evidence of healing in response to a stressor - would suggest that the individual has been exposed to that stressors for a substantial period of time for the skeleton to not only respond but show evidence of healing.

Biological parameters of age and sex also alter the expression and pattern of pathological lesions (Roberts 2000, 146). This means it is imperative that the pattern and the type of bony response is recorded, and interpretation of these lesions considers both the age-at-death of individuals affected and contextualises them within the archaeological record.

5.62 Determining, Identifying and Recording Pathological Lesions

Due to pathological changes and lesions being so troublesome to identify and record within fetal, perinatal and infant skeletal remains, this thesis employed a strategy whereby no particular ‘stress indicator’ or skeletal element was to be observed, instead considering the whole of the skeleton and recording any potential pathological change. This was to ensure that pathological changes were not overlooked, and prevented the author from observing only certain conditions which tend to be located on specific elements (e.g. metabolic conditions). This resulted in a wide range of changes and lesions being observed in varying skeletal elements and to various aspects of these elements. Furthermore, severity of lesions was inherently variable between individuals.

As normal bone growth and pathological bone formation can currently not be distinguished between, any evidence of NBF was recorded, though a grading scheme was employed to differentiate between extensive NBF and that which was only minor (See below for more detailed explanation). By employing a tripartite strategy to assessing pathological changes (type, severity and location of pathology) along with a grading scheme specifically for NBF, it is suggested that 'true' pathological NBF can be deduced and separated from that of normal NBF. This is because the pattern (location) and severity of NBF, when assessed by age-estimation and known growth patterns, can be seen to vary from the expected pattern of normal NBF.

Assessment of pathology was undertaken macroscopically and relied on detailed and close assessment of the skeletal individuals. Each pathological change/lesion was recorded descriptively by the author and documented photographically. Pathological assessment within this thesis has relied on three primary avenues of investigation:

1. **<u>Type of pathology:</u>**

   Because bone only has two responses, regardless of stimulus/stressors, type of lesion was recorded as either bone formation or bone destruction/resorption. Bone formation was recorded as 'NBF' (New Bone Formation) and bone destruction/resorption was recorded as being 'Lytic'.

   For NBF, type of bone formation was also recorded. Type of bone was recorded as either woven or lamellar bone, and also whether it spiculated (Ortner 2003, 45-64). Lytic lesions were not recorded in this way due to the destructive nature of these lesions.

   Expansion of the metaphyses was also recorded when present. Typically, such changes may be expected in the long bones and ribs and when identified the individual skeletal elements have been detailed. Morphological change (e.g. bowing) was also recorded to each individual skeletal element affected.

2. **Severity of pathology:**

   Severity of pathological change has been recorded in accordance with a grading system established by the author (See Section 5.73 below for details). Grading systems were established to consider severity of NBF, lytic lesions and metaphyseal expansion. For each type of pathology, a severity score of 1, 2 or 3 has been afforded in accordance with the severity description outlined below. It was considered to be imperative that severity was recorded to observe and identify individuals who appear to be more affected by pathological changes. By recording severity, correlations between this variable, the location of pathological lesions and the type of lesion were able to be considered, aiding etiological, pathogenic and contextual interpretations of health stress.

   Furthermore, as it is hypothesised that those with more severe lesions will show greater growth disruption, correlation between severity of lesions and growth disruption was able to be considered.

3. **Location of pathology:**

   Location was firstly documented as cranial or postcranial, then by specific skeletal element (e.g. right tibia, left frontal bone) and then by aspect (e.g. endocranial, ectocranial, medial, lateral). The location was documented in this very specific way so that pathological lesions could be recorded and located as precisely as possible. Furthermore, by documenting the aspect of the bone that was affected it could be determined whether the element was affected circumferentially/across the surface, or whether pathological changes were limited to a particular aspect of the skeletal element. By employing this recording strategy the patterning of pathological lesions could be assessed by skeletal element. Given that some diseases/infections/conditions are known to affect certain elements and certain locations more commonly it was considered that recording the location of pathology was of particular importance.

Table 5.6 provides an example (Skeleton 4 from Cross Bones) of how pathological lesions were recorded for each of the 423 fetal, perinatal and infant individuals assessed. This table

details how results for each of the categories listed above have been recorded, with type of lesion split into 'Type I' (NBF, lytic, metaphyseal extension, morphological change) and 'Type II' (woven, lamellar, spiculated). Once this information was recorded, the data was transferred into an excel spreadsheet – an example of which is given in Table 5.7 (also using Skeleton 4 from Cross Bones as an example). Data was transferred into a database to enable easier statistical assessment of the results. Within this database it is listed whether the individual has cranial and post-cranial elements present and whether there are pathological changes observed to any of these elements. There is then a breakdown of the main skeletal elements with the presence of pathological changes recorded with a 'Y' for 'yes'. Type I and Type II categories are listed at the far end, again with a 'Y' given for 'yes' if that type was recorded within the individual.

5.63 Pathological Definitions, Examples and Grading Systems

The following section outlines and provides examples as to the varying locations, types and severity of lesions identified in assessment of the fetal, perinatal and infant individuals considered within this study. Definitions, as well as photographic examples, have been given for NBF, lytic lesions, metaphyseal expansion and morphological changes. Photographic examples of woven, lamellar and spiculated bone have also been provided. Finally, grading systems from 1 to 3 have been documented photographically and descriptively for NBF, lytic lesions, and metaphyseal expansion.

TABLE 5.6 Example, using Skeleton 4 from Cross Bones, of how pathological lesions were recorded for each individual, listing location, type and severity of the lesion.

Skeleton Number	Sample	Time Period	Dental Age	Cranial or Postcranial	Skeletal Element	Aspect	Type I (NBF/Lytic/Met./Morph.)	Type II (Woven/Lamellar/Spiculated)	Severity (Grade 1-3)
4	Cross Bones	Post-Medieval	46	Cranial	Frontal Bone (Left & Right)	Endocranial	NBF	Woven & Lamellar	2
					Parietal Bone (Left & Right)	Endocranial	NBF	Woven	2
					Occipital Bone	Endocranial	NBF	Woven	2
				Post-Cranial	Tibia	Medial	NBF	Woven	1

TABLE 5.7 Example, again using Skeleton 4 from Cross Bones, of how recorded pathological lesions were transposed into an Excel spreadsheet to enable easier statistical assessment of results. The data presented in this table corresponds to the more detailed information recorded above (Table 5.6). Where 'Y' has been given it records the presence of pathology to that location or type. Due to the expanse of this table, archaeological samples and their time periods have been abbreviated. In this instance 'CB' represents the site of Cross Bones and 'PM' stands for post-Medieval.

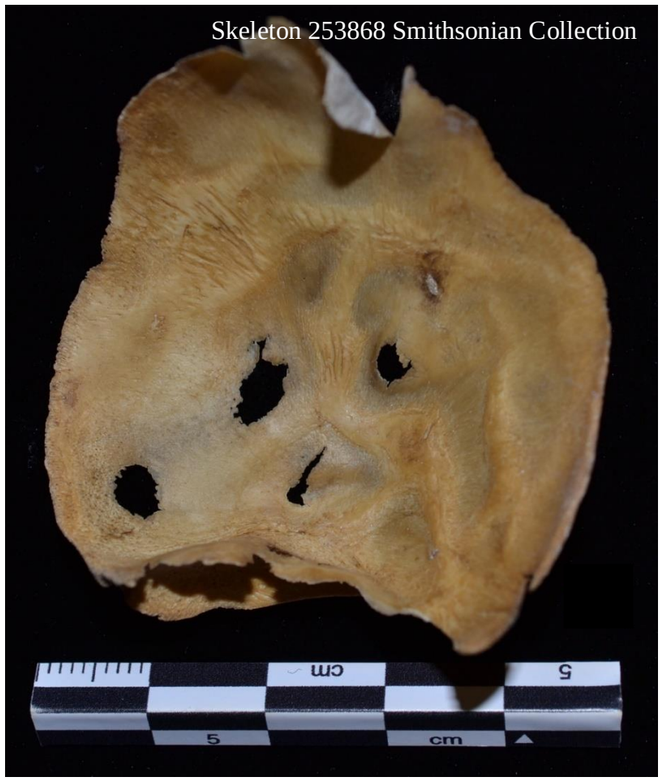
Skeleton Number	Sample	Time Period	Dental Age	Post-cranial Elements	Post- cranial Pathology	Cranial Elements	Cranial Pathology	Frontal	Parietal	Occipital	Temporal	Sphenoid	Zygomatic	Mandible/Maxilla	Unknown Fragment	Humerus	Radius	Ulna	Femur	Tibia	Fibula	Ribs	Vertebrae	Ilia	Scapula	Clavicle	NBF	Lytic	Metaphyseal Expansion	Morphological change	Woven	Lamellar	Spiculated
4	CB	PM	46	Y	Y	Y	Y	Y	Y	Y										Y							Y				Y	Y	

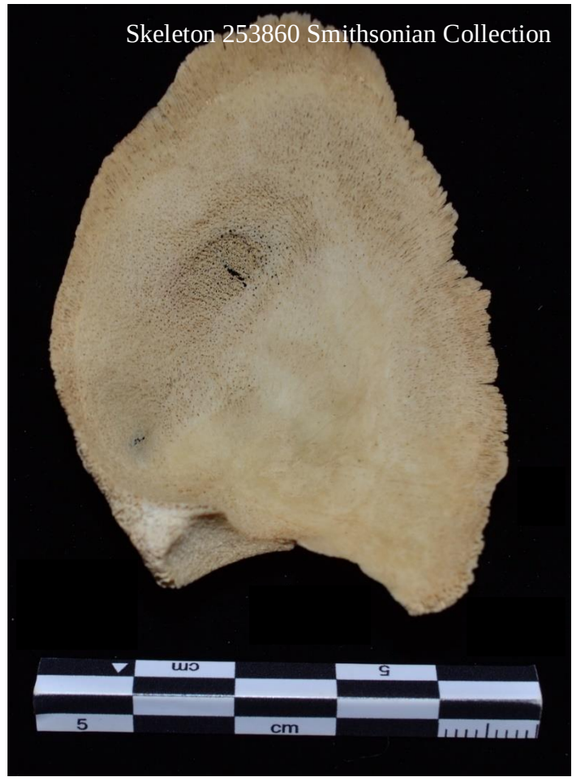
5.63.1 <u>Examples and Definitions:</u>

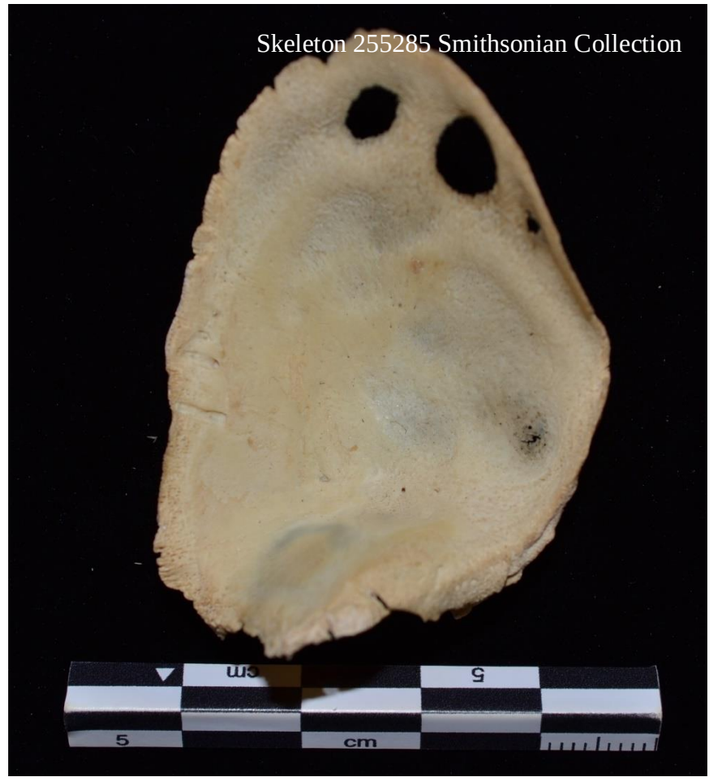
1. New Bone Formation (NBF):
   Where deposits of woven and/or lamellar bone can be identified, typically appearing to be on top of the original cortical bone surface. Multiple layers of NBF may be observed, and along with variation in thickness of the NBF, may provide indication as to the severity and or length of exposure to stress. NBF can be identified on any element and may appear as an isolated patch or cover entire surfaces/aspects.

2. Lytic Lesions:

   Lytic lesions are those where destruction and resorption of the original cortical bone can be identified. Evidence of porosity has also been considered within this category. Variation in the severity and extent of lytic lesions can be observed, with some skeletal individuals having isolated areas/elements showing lytic lesions, whilst others show extensive evidence to multiple skeletal elements. Within the cranial vault, lytic lesions can often be observed as fenestrations, where destruction and thinning of the vault can be observed before complete perforation of the vault occurs. Within the cranial vault these lytic lesions are often found to be in association with areas of bone densification, which typically appear to outline and surround the lytic lesions (as seen in some of the images below).





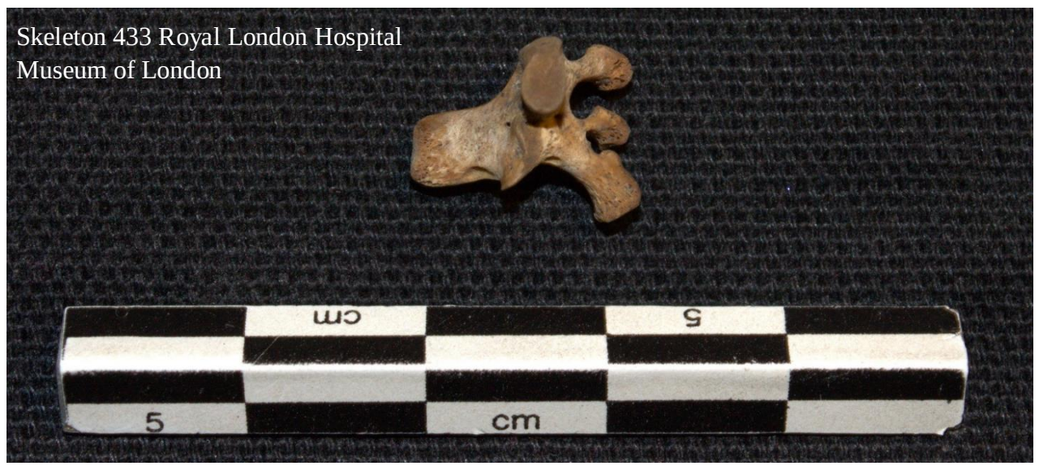


3. Metaphyseal Expansion:

   Metaphyseal expansion refers to the widening (typically laterally) of the metaphysis. This typically occurs at the distal ends of long bones, but can be observed at the proximal ends, as well as in the ribs. Metaphyseal expansion is often identified due to the changes in the metaphyseal margin, which becomes irregular, misshapen and often has a ridge/lip of bone extending past the metaphysis. Metaphyseal expansion can also be identified from observing the inner trabecular bone structure (as seen below), where there is marked expansion in the trabecular structure.

4. Morphological Changes:

   Morphological changes are defined as those where clear shape changes have occurred, typically meaning the process of normal growth and development has been altered. Within this thesis morphological changes primarily refer to the identification of bowing within the long bones. However, as can be seen from the images below, congenital malformations also commonly cause morphological changes. Due to the wide array of changes that may be identified, a grading system for morphological changes has not been employed. Instead each morphological change identified has been described in detail by the author.

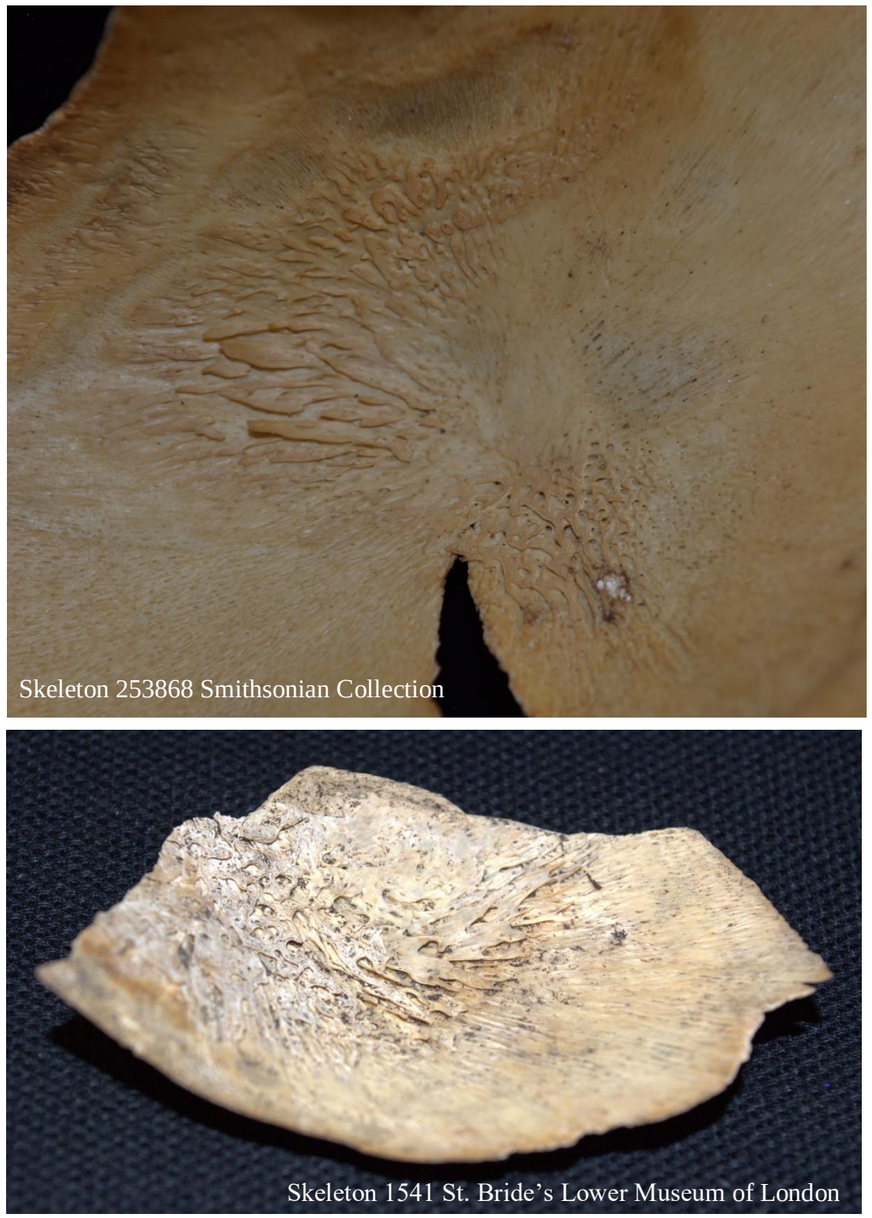


5. Woven Bone:

   Woven bone is immature, highly disorganised bone typically found in fetal/perinatal/infant individuals who are growing as it is formed very quickly (White *et al.* 2012, 35). However, the distinctive grey appearance of woven bone is also synonymous as evidence of pathological NBF – a physiological response by the body to an environmental stressor.

6. Lamellar Bone:

   Lamellar bone is bone that has remodelled and matured or is in the process of doing so. Lamellar bone can represent part of the normal bone formation process, or can also represent healing bone if the stimulus was pathological. Lamellar bone tends to be well organised, striated bone or is bone that is transitioning to this state from woven bone.



Skeleton 253868 Smithsonian Collection

Skeleton 1541 St. Bride's Lower Museum of London

7. Spiculated Bone:

   Spiculated bone typically refers to bone which is perpendicular to the normal growth plate. Thus, the appearance of ‘hair-on end’ bone formation is commonly associated with spiculated bone. However, more generally spiculated bone can refer to bone which is growing in an atypical aspect. Consequently, for the greater wing of sphenoid pictured below the additional bone growth has also been referred to as a bone spicule.

5.63.2 <u>Grading Systems</u>

1. New Bone Formation (NBF):

    a. Grade 1: New bone formation, which may be woven or lamellar in appearance, will be considered to be grade 1 when the NBF is not clearly apparent and the margins are unable to be clearly defined from that of normal cortical bone. Grade 1 NBF is likely to be isolated in location, appearing minimally across the skeletal element.

    b. Grade 2: New bone formation recorded as being grade 2 will be clearly identifiable as a definable area of woven or lamellar bone formation. There will be clear boundaries/borders to the NBF and it will obviously differ from the normal cortical bone of the skeletal element. Grade 2 NBF is likely to be distinguishable as a clear layer of bone on top of the original cortical surface. It is likely that NBF listed within this category will be formed of a single layer though may extend over a large aspect area of the skeletal element.

    c. Grade 3: New bone formation recorded as being grade 3 will be the more severe type of NBF, with clear, multi-layered or thick NBF across a large area/aspect of the skeletal element. The NBF may be woven or lamellar in appearance and is clearly seen to be on top of the original cortical bone.

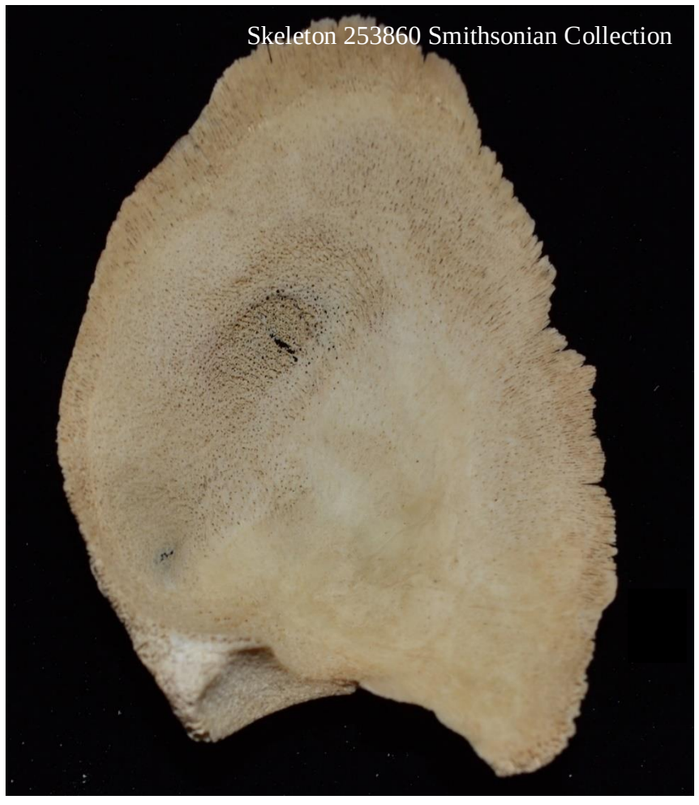
2. Lytic Lesions:

    a. Grade 1: Lytic lesions considered to be grade 1 likely consist primarily of macro-porosity. This porosity will be relatively minor, though may extend over a large skeletal area, and no clear destruction of the cortical bone will be apparent.

    b. Grade 2: Lytic lesions considered to be grade 2 will likely show evidence of some cortical destruction as well as porosity. However, cortical destruction will not be widespread throughout the skeletal element and is instead likely to be in isolated concentrations.

    c. Grade 3: Lytic lesions considered to be grade 3 will show extensive cortical destruction and/or porosity. Destruction will be widespread throughout the element.

Grade 1



Grade 2

Grade 3

3. Metaphyseal Expansion:

    a. Grade 1: Metaphyseal expansion considered to be grade 1 will likely consist of noticeably widened/flared metaphyses which do not appear proportional for the long bone diaphysis. However, despite this expansion no change to the metaphyseal margin or trabecular bone structure will be observed.

    b. Grade 2: Metaphyseal expansion will be considered to be grade 2 when involvement of the metaphyseal margin is apparent. This will result in atypical and misshapen metaphyseal margins often combined with a discernible brim/lip to the metaphysis.

    c. Grade 3: Metaphyseal expansion considered to be grade 3 will be the most severe and where involvement of the trabecular bone structure can be seen. Individuals displaying grade 3 metaphyseal extension will likely have more porous metaphyses and the trabecular structure will appear clearly expanded and widened. Involvement of the metaphyseal margin may still be apparent though this may be lost due to the trabecular expansion.

Grade 1

Grade 2

Grade 3

5.64 Excluded Methods

It has become common throughout palaeopathological literature that for certain skeletal lesions to be synonymous with evidence of 'stress' and health disruption. Consequently, many studies have assessed evidence of *cribra orbitalia* (Facchini *et al.* 2004; Kyselicová *et al.* 2015; Rivera & Lahr 2017), DEH (dental enamel hypoplasia) (Blakey & Armelagos 1985, 371; Armelagos *et al.* 2009, 265; e.g. Goodman & Rose 1990), and Harris lines (e.g. Gindhart 1969), correlating presence of these lesions as evidence for health disruption and disease (Lewis & Roberts 1997, 581). However, this study considered it more appropriate to take a broad approach to recording of pathological lesions, recording all lesions identified rather that targeting particular categories of lesions. It was considered that this would provide a broader and better dataset from which to consider health and wellbeing of the individuals assessed. Therefore, the following analyses avoided consideration of particular stress markers and indicators, instead adopting a broader approach to reveal a more general narrative of health.

DEH is considered to be indicative of physiological disruption and stress (Massler *et al.* 1941, 42; Blakey & Armelagos 1985, 371; Hillson 2005, 168; Franco *et al.* 2007, 518; Armelagos *et al.* 2009, 261; 266) with location of these defects able to be correlated to specific periods of growth and thus, age (Blakey & Armelagos 1985, 371; Armelagos *et al.* 2009, 266). Disruptions in enamel secretion, mineralisation and maturation can be observed as defects on the mature enamel surface and reflect episodic exposure to stress during crown formation (Massler *et al.* 1941, 42; Hillson 2005, 169). Hypoplastic defects occur in hard, well mineralised dentition (Hillson 2005, 170), where there is a local deficiency in the enamel thickness as a result of the experienced disruption (Blakey & Armelagos 1985, 371; Franco *et al.* 2007, 518; Armelagos *et al.* 2009, 266). These bands/lines/pits tend to be linearly orientated around the circumference of the tooth, following the line of the perikymata (Hillson 2005, 170); perikymata being the incremental growth lines within enamel (Hillson 2005, 163). Though many studies have assessed DEH macroscopically, microscopic assessment provides a better indication as to this disruption (Hillson 2005, 172; 174). Thus, microscopic assessment of perikymata and the number of perikymata involved can be tracked through multiple tooth crowns to identify a systemic disruption to tooth formation and development (Hillson 2005, 171).

Multiple aetiologies of DEH have been considered, all of which explore a variety of detrimental life course outcomes; these include premature birth, low birth weight, metabolic disturbances and specific and non-specific infections (Cutress & Suckling 1982, 117; Blakey & Armelagos 1985, 371; Franco *et al.* 2007, 518; Roberts & Manchester 2010, 75-77). As enamel does not repair or remodel (Blakey & Armelagos 1985, 371; Lewis & Roberts 1997, 581; Franco *et al.* 2007, 518), teeth are able to permanently record episodes of physiological stress experienced both intra- and extrauterine (Blakey & Armelagos 1985, 371; Armelagos *et al.* 2009, 265). Thus, being able to identify DEH within fetal/perinatal and infant samples would provide a valuable insight into maternal and intrauterine health and wellbeing, allowing bioarchaeologists to definitively trace growth disruption and stress exposure. Although growth and development of the deciduous dentition can be affected from around the fifth fetal month (Blakey & Armelagos 1985, 371; Armelagos *et al.* 2009, 265), rarely has DEH been identified on fetal, perinatal and infant tooth cusps (Massler *et al.* 1941, 59; e.g. Blakey & Armelagos 1985). This is because the deciduous dentition is typically still required to complete most of its growth and development during these life stages, and both enamel and dentine is poorly mineralised during these initial deposits (Hillson 2005, 210). Hillson (2005, 170) states that DEH can only be observed in well mineralised dental tissues, and due to the early stage of formation of the dentition analysed, where the majority of tooth cusps would not have undergone the second enamel secretion and mineralisation stage, they are not well-developed enough to assess for DEH. Franco and colleagues (2007, 522) also state that the optimum time to observe enamel defects is immediately after tooth eruption. Consequently, due to the majority of the dentition assessed within this thesis being very early in its development process DEH would be almost impossible to identify macroscopically and would certainly require histological assessment to identify. Additionally, it is typical for DEH to be assessed on multiple teeth from the same individual where crown development is complete (e.g. Blakey & Armelagos 1985). Again, due to the immature age of these individuals very few had multiple teeth where crown development was complete, meaning assessment of DEH would have been very limited. Furthermore, due to the ethical considerations of destroying archaeological material, and financial and time constraints (Huda & Bowman 1995, 145), this study considered it unwise to histologically sample the dentition available and as such refrained from using DEH as a pathological criterion.

Similarly, assessment of Harris lines would have required radiographic evaluation of skeletal elements and this study was unable financially and logistically to undertake this assessment. Harris lines have been typically used as evidence of growth arrestment – thought to be indicative of points where growth has been suspended as a result of health disruption – where a radiodense line or band, often identified in long bones, is observed (Mays 1999, 307; Scheuer & Black 2000a, 28; White *et al.* 2012, 430; Larsen 2015, 42). Harris lines are typically identified in the distal and proximal ends of the long bones, with most studies observing these in the bones of the lower limb (femur and tibia) (Mays 1995, 511-512). It has been suggested that these lines are a result of stress and signify growth disruption (Mays 1995, 511; Scheuer & Black 2000a, 28; Lewis 2007, 107). However, many reservations are widely held about this method (Lewis & Roberts 1997, 583; Larsen 2015, 43). It is still unknown whether these Harris Lines represent normal episodes of growth arrestment, where the body is naturally regulating and fluctuating according to its growth trajectory, or whether these do indeed represent periods when growth has been halted, as a way to maintain bodily function, as a result of health disruption (Larsen 2015, 43; e.g. Magennis 1990; Lampl *et al.* 1992; Lampl & Jeanty 2003; Alfonso-Durruty 2011; Papageorgopoulou *et al.* 2011). Mays (1995, 519) found in his study that there was no significant relationship between Harris lines and femoral length, suggesting that they are not always indicative of health stress. However, he interprets absence of Harris lines as a result of catch-up growth and the ability of all the skeletal individuals investigated to have fully recovered from any health insult (Mays 1995, 519). Despite this, other investigations similarly found that Harris lines were present in 10% of individuals when no stressful event had occurred (Gindhart 1969).

The recording of Harris lines is also troublesome, with different authors considering skeletal elements from varying aspects, and also counting lines incongruently – some only counting lines across the whole of the skeletal element, others also counting those which reach at least half way (Lewis & Roberts 1997, 583; e.g. Macchiarelli *et al.* 1994). It has also been found that depending on the type and side of skeletal element investigated, there is variation in the number of Harris lines observed, with the distal tibia and left side of skeletal elements found to exhibit more Harris lines (Hughes *et al.* 1996). Other investigations have attempted to determine the age at which these lines formed (Lewis 2007, 109; White *et al.* 2012, 430; e.g. Hunt & Hatch 1981; Maat 1984; Byers 1991). However, results have been found to have mixed success. Consequently, due to the incongruities in recording and documenting Harris

lines, as well as the ongoing ambiguity surrounding their etiology, this study has not included such assessment within its palaeopathological analysis.

Finally, c*ribra orbitalia* has not been used as a pathological criterion within this thesis. C*ribra orbitalia* (CO) presents as a '*…localised appearance of porotic lesions on the roof of the orbits*' (Facchini *et al.* 2004, 126; Wapler *et al.* 2004, 333; Rivera & Lahr 2017, 1). Primarily due to the physiology of these individuals it would be unexpected for many of them to present with these lesions. Although possible that porosity, in association with CO, may be observed in such young individuals, the orbits are highly vascular and NBF is typically observed within them. This means that porous layers of concentric new bone formation can often be seen and as such identifying porosity, as evidence of CO is unlikely in such young individuals. Furthermore, although cribrotic lesions are commonly recorded, their aetiology has recently been debated (e.g. Wapler *et al.* 2004; Walker *et al.* 2009; Oxenham & Cavill 2010). Typically, *cribra orbitalia* has been attributed to be a consequence of iron deficiency anaemia (Wapler *et al.* 2004, 333; Kyselicová *et al.* 2015, 16; Rivera & Lahr 2017, 1), thought to be the preliminary phase of the condition before other skeletal changes, particularly to the cranial vault (such as porotic hyperostosis), occur (Wapler *et al.* 2004, 333; Rivera & Lahr 2017, 2). However, recent investigations have questioned this (Lewis & Roberts 1997, 583; Zuckerman *et al.* 2012, 44; e.g. Walker *et al.* 2009; Oxenham & Cavill 2010), considering a multifactorial etiology to the lesions (Kyselicová *et al.* 2015, 16). This has been suggested to include tuberculosis, rickets, scurvy and trauma (Kyselicová *et al.* 2015, 16; Rivera & Lahr 2017, 16). Rivera and Lahr (2017, 2) state that complexities in identifying the precise etiology of CO (and pathological lesions more generally) is compounded by the fact that there are many more illnesses than there are skeletal responses. In fact, the duality by which the skeleton can respond to stressors and environmental influences means that there is a very constrained physiological skeletal response (Rivera & Lahr 2017, 2). Consequently, the difficulties in identifying and assessing *cribra orbitalia* within fetal, perinatal and infant individuals, along with the ambiguity over the interpretation of these lesions, means that this thesis has avoided particular assessment of these lesions as markers of stress.

Assessment of vertebral neural canal size and dimensions has also become commonplace within bioarchaeological assessments of non-adult growth, development and physiological

disruption (Watts 2013, 120-121). This is because the fusion of the vertebral neural arches to that of the vertebral body happens during childhood when growth and development is still greatly influenced by the environmental conditions to which you are exposed (Watts 2013, 121). Consequently, it has been discovered that a reduction in VNC measurements reflects disrupted growth and has hence been used as a marker and indicator of stress (e.g. Newman & Gowland 2015). However, investigation of the vertebral neural canal (VNC) size could not be undertaken for this thesis due to the age of the individuals considered for assessment. Neural canal dimensions are considered to be stabilised by around 3-5 years of age (Watts 2013; Newman & Gowland, 156) – thus reflecting early life experiences – however, the neural arches and bodies of the vertebrae are not developed sufficiently or fused in fetal, perinatal and young infant individuals to enable analysis of VNC dimensions (Armelagos *et al.* 2009, 269).

Additionally, due to the limitations of costs, time, availability and ethical permissions as listed previously, radiographic assessment of new bone formation was also not undertaken. Although this would have enabled the thickness of the new bone formation to be established – which is thought to be critical in determining pathological from normal new bone formation – it was beyond the scope of this thesis to be able to radiographically assess the 423 individuals analysed. Instead, this thesis has attempted to propose a methodical, logical approach to recording potential pathological changes in fetal, perinatal and infant individuals, which enables consideration of location, type and severity of change in accordance with estimated age. As no method has yet been proposed to analyse and record pathology within these young individuals this thesis attempts to investigate the potential of recording pathological changes in this way and aims to identify clear correlations between growth and health disruption – it being hypothesised that the greater the health stress experienced (i.e. more numerous and severe pathological lesions), the greater the growth disruption (i.e. the greater the difference between dental and skeletal age-at-death estimates). However, it is resoundingly acknowledged that radiographic investigation of new bone formation, particularly its thickness, would aid in unravelling the ongoing debate surrounding the differentiation of normal from pathological new bone formation (See Chapter 11: Section 11.2 for further discussion).

5.7 Biological Sex Estimation

Biological sex, as discussed in the previous sections, has a clear impact on growth and development, with males and females not only having sexually dimorphic morphology to their skeleton but varying growth strategies. Therefore, determining dental and skeletal growth without consideration of biological sex induces further error, yet sex estimation of non-adult individuals is acknowledged to be notoriously difficult and unreliable (Hoppa & Fitzgerald 1999, 2; Scheuer & Black 2000a, 15; 2000b, 12; Satterlee Blake 2018, 41).

The skeletal biology of non-adult individuals is the primary limitation of this assessment, as individuals have not yet fully grown or even developed the skeletal structures typically required for sex assessment in adult individuals. Furthermore, the hormonal influxes that alter the morphology of these structures are yet to occur (Hoppa & Fitzgerald 1999, 2; Scheuer & Black 2000a, 15 Lewis 2000, 40). Although, hormones are released during fetal development, particularly testosterone during the ~10^{th} gestational week if the individual is male, sexually dimorphic traits are considered not to become clearly apparent until puberty (Saunders 2000; Loth & Henneberg 2001, 179). As a result, macroscopic assessment of sex estimation in non-adult individuals is often avoided.

Despite these limitations attempts to determine biological sex based on the dimorphism of the skeleton have been made. Typically, methodologies have utilised similar skeletal structures to those analysed in adult individuals – primarily the bones of the pelvis and cranium (Scheuer & Black 2000a, 15; Lewis 2007, 47; e.g. Boucher 1955; 1957; Weaver 1980; Schutkowski 1993; Loth & Henneberg 2001; Wilson *et al.* 2008) – however, other attempts have investigated variation in the humerus and dentition (e.g. Black 1978; Rogers 2009; Stull & Godde 2013).

Studies testing methods of sex estimation have found that dimorphic traits can vary between population, that the accuracy of these methods could not be reproduced, and that there is substantial overlap between the categories of males and females (Hunt 1990; Scheuer 2002; Lewis 2007, 48; Cardoso & Saunders 2008; Vlak *et al.* 2008; Satterlee Blake 2018, 41). In fact, most methods, when tested, have failed to yield accuracies of over 70% (Lewis 2007, 48). Males tend to have larger dentition than females (Black 1978), though when tested, sexual dimorphism utilising this method had an accuracy of 75%. Furthermore, sexual

dimorphism was also found to be less in deciduous teeth than in permanent dentition (Lewis 2007, 48-49), and again disease status and environmental factors can cause changes in the tooth crowns (Lewis 2007, 49). Schutkowski (1993) reported that his method for observing sexual dimorphism in the pelvis and mandible was between 70-90% accurate. However, when Loth (1996) tested this, accuracy rates were only found to be between 30-40%. Loth and Henneberg (2001) have further developed this method for using the mandible to determine biological sex, but as this method primarily looks at chin shape (requiring the mandible to be fused at the mental symphysis), most of the individuals considered within this study are too young to be assessed in this way. Sex determination using the mandible is often considered to be the most reliable (Lewis 2007, 58), but given that many of the traits assessed cannot be observed in individuals aged to be less than 6 postpartum months (64 GWA), it was impossible to utilise these methods consistently within this thesis.

The sciatic notch of the ilium has also been prominent in assessment of fetal pelvis, currently thought to be the most dimorphic and accurate variable to assess for biological sex within the ilium (Vlak *et al.* 2008, 309). However, results and interpretations have differed (Lewis 2007, 53) and when tested, sciatic notch depth, breadth and angle was found to only be 61% accurate (Vlak *et al.* 2008, 312). Weaver (1980) developed a method whereby the auricular surface was considered to be elevated in females and non-elevated in males. The original investigation suggested that the method was accurate in 75% of females and 91% of males (Weaver 1980, 191). However, Mittler and Sheridan (1992) found that although males were accurately predicted in 85% of cases, females were only accurate in 58%, arguing that there is a strong male bias within this method. In fact, many of the methodologies developed are suggested to have a strong bias in identifying males (Lewis 2007, 58; e.g. Schutkowski 1993; Wilson *et al.* 2008).

Today, scientific advances have led to the emergence of studies considering DNA analysis to determine biological sex of infants and children (Hoppa & Fitzgerald 1999, 3; Aiello 2000, VII; Lewis 2000, 40; e.g. Waldron *et al.* 1999; Mays & Faerman 2001). However, given the ethical, time and financial constraints of conducting ancient DNA analysis (aDNA), combined with the possibility that no aDNA would be surviving, this was not considered to be a suitable line of investigation within this thesis.

Consequently, within this thesis sex estimation has not been attempted on the archaeological individuals as the methodologies are still too variable and inaccurate. The fetal collection at the Smithsonian has recorded biological sex for some of the individuals assessed (Hunt *personal communication*) and thus, where possible analysis and interpretation has been conducted using these categories (male versus female). Although the author did record sexually dimorphic traits in accordance with Schutkowski (1993), testing this method on the Smithsonian collection has not been afforded within this thesis as no other sample uses sex estimation in its analysis. As such, it has been detailed that this is a further study for future consideration.

5.8 Data Analysis and Statistical Assessment

The statistical methods of assessment employed throughout this study have been deliberately minimal and limited as a result of both the nature of the data sets, sample sizes and research questions considered.

The primary use of statistical tests within this study is to determine whether there are significant differences in skeletal metrics/age estimations when compared to the dental age group. The aim of determining whether there is a significant difference is to identify individuals who present evidence of growth disruption. However, although the data set is large for an archaeological fetal/perinatal/infant study it is still relatively limited in terms of number of individuals assessed when samples are divide by discrete categories, such as dental age estimation. Furthermore, the number of individuals decreases rapidly when those without dentition are excluded from assessment and individuals are grouped by time period/sample and then by dental age. For some of these dental age groups only one or two individuals are present. In addition, regardless of the methods employed there are no two datasets which can be compared; either raw metric data for individuals within an age group is compared against a data point from a reference method (where only an average measurement for that skeletal element is recorded for that age group), or skeletal ages for a particular element are compared against their dental age category. This means that data recorded/calculated in this study is typically only able to be compared against a single point. As a result, the primary statistical assessments employed are t-tests, where the comparative mean can be given by the author (either dental age or reference metric). T-test analysis was undertaken in PAST (developed by

Hammer, Harper and Ryan 2001), a free, downloadable programme which has been specifically designed for paleontological analysis.

For the third manuscript, ANOVA (analysis of variance) has been utilised to consider statistical differences between dental and skeletal age estimates and true documented ages. This method of assessment was able to be used within this manuscript as multiple age estimation methods were able to be tested against documented age. Tests were also undertaken in PAST, and were double checked by running the analyses for a second time in Microsoft Excel. Significance was set to 0.05 (95% confidence), whereby *p*-values below 0.05 were considered to be statistically significant.

However, using the metric data collected, summary statistics (mean, standard deviation, 95% confidence intervals) have also been calculated for select individuals (detailed within the individual manuscripts).

For pathological assessment, limited statistical methods were employed to assess data as pathology cannot be 'quantified' in a way that enables extensive statistical analysis. However, both True Prevalence Rates (TPRs) and chi-squared tests have been utilised, primarily in manuscript 4 (Chapter 9). True Prevalence Rates were calculated by documenting how many individuals out of the 423 analysed had a particular skeletal element(s) present, and then out of those with the element(s), how many showed pathological changes. Within the manuscripts presented, totals have been given for numbers of individuals assessed, along with separate columns of totals for the number of a particular element able to be observed, and then the number found to be affected and showing pathological changes. However, these TPR rates may be better expressed as corrected CPRs (Crude Prevalence Rates) as for some elements, such as long bones, there are bilateral pairs of that element. This is because this study does not calculate prevalence rates for each element by side, instead amalgamating these bilateral pairs. It was considered that this study was not required to consider pathological changes by side, as the focus of investigation was to consider the location of these lesions more generally (i.e. were more appendicular/axial elements affected).

Chi-squared tests for independence at 99.5 % confidence ($p < 0.05$) were also employed for pathological categories to observe whether there was any relationship between various pathological variables. Typically, only two pathological variables have been used for each chi-squared test, but these have been considered by dental age categories, samples and time periods. For example, chi-squared tests were run by dental age group for comparisons between new bone formation to the femur and new bone formation to the tibia. Chi-square results are presented numerically, where $p < 0.05$ shows there is a significant relationship between the variables, whilst p values > 0.05 indicate there is no significant relationship. Chi-Squared values (X^2) have also been given.

Chapter 6: Manuscript 1

Measure for Measure: A comparative study of the impact of 'stressors' on fetal, perinatal and infant growth from three rural Iron Age and Romano-British samples.

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KEY WORDS: Intrauterine; Metric; Pathology; Health; Well-being.

Abstract: *Growth is a dynamic process which responds to environmental as well as genetic factors, making it a useful biological parameter for examining stress in early life. Exposure to stress can leave identifiable skeletal changes, and as such analysis of fetal, perinatal and infantile individuals can reveal important information regarding maternal health, living environments and infant care. This paper examines growth and pathological lesions in 99 individuals, aged between 32 gestational weeks and six months of age (64 gestational weeks of age), from three rural settlements dating from the late Iron Age to Romano-British periods in England. These sites span an important period of social and cultural change in Britain. Growth disruption and pathological lesions were identified in individuals from all three time periods (Pre-Roman, Transition, Roman). The extent and severity of growth and health disruption was found to increase from the 1st century A.D. onwards, during the Roman occupation of Britain. These findings support previous studies which demonstrate a reduction in health as a consequence of the Roman conquest, but are the first to demonstrate that these health deficits were initiated in utero.*

The biology of human growth is complex, particularly during the embryological and fetal stages of life, when it is most prolific (Sinclair 1985; Lejarraga 2002; Bogin 2002). Growth is defined as quantitative, physiological change (Bogin 2001), and is regulated by both intrinsic (genetic) and extrinsic (environmental) factors (Tanner 1978; Hoppa 1992; Cameron 2002; Bogin & Rios 2003). Embedded within each individual is a genetic 'blueprint' for growth, yet our ability to attain this genetic potential is controlled by the environmental conditions to which we are exposed both pre- and postnatally (Cameron 2002; Agarwal 2016). Physiological plasticity enables a degree of modification in skeletal morphology in response to environmental variables (Bogin & Loucky 1997; Bogin & Rios 2003; Agarwal 2016). The

susceptibility of fetal, perinatal and infant individuals to growth and health stress is typically maternally regulated, reliant on maternal ability to protect and buffer offspring against environmental conditions encountered both pre- and postnatally (Barker 1997; Gowland 2015). Maternal diet and nutrition, exposure to disease and infection, as well as social, cultural, political and psychological pressures faced during pregnancy, are all factors that can impede offspring growth. As such, it is essential to acknowledge that indicators of growth and health stress identified on fetal, perinatal and infant individuals, often provide a sensitive indication of maternal, as well as community, health and well-being (Goodman & Armelagos 1989; Hoppa 1992; Redfern 2003). Therefore, assessment of growth has long been utilised in the determination of overall health, with results often compared within and between populations to investigate skeletal responses to stress (Agarwal 2016; e.g. Saunders & Hoppa 1993).

A wealth of research in the medical, bioarchaeological and anthropological literature has investigated the relationship between environmental adversity and birth outcomes (e.g. Bogin & Loucky 1997; Cameron & Demerath 2002; Cardoso 2007; Abu-Saad & Fraser 2010; Bogin & Baker 2012). Detrimental birth outcomes include preterm birth, small for gestational age (SGA), intrauterine growth restriction (IUGR), still birth and birth defects (Winick *et al.* 1972; Prentice 2003; Kuzawa & Quinn 2009; Wu *et al.* 2012), as well as increased morbidity and mortality risks (Wu *et al.* 2012; Farewell *et al.* 2018; Said-Mohamed *et al.* 2018). These detrimental outcomes have often been strongly found to correlate with negative maternal experiences and poor health status during pregnancy (e.g. Chiswick 1985; Goldenberg & Thompson 2003; Abu-Saad & Fraser 2010; Cussons-Read *et al.* 2012; Beaudrap *et al.* 2013; Fell *et al.* 2016; Melby *et al.* 2016). Evidence of growth disruption – defined as the interruption of the 'normal' growth trajectory and inability to attain ones' potential maximum growth – in fetal, perinatal and infantile remains is hence suggestive of a poor *in utero* environment.

Ongoing research into the Developmental Origins of Health and Disease Hypothesis (DOHaD) is exploring the impact that early-life environmental factors have on growth, morbidity and mortality in later childhood and adulthood (e.g. Barker & Osmond 1986; Barker 1997; 2012; Barker *et al.* 2002). This hypothesis has become prevalent within interpretations of the early life course (e.g. Finlay 2013; Klaus 2014; Gowland 2015),

highlighting the long-term growth and health outcomes of detrimental *in utero* and immediate postnatal environments (Gluckman & Hanson 2006; Armelagos *et al.* 2009; Hoffman 2016; Clukay *et al.* 2018; Halcrow *et al.* 2018).

Intrauterine stress can leave specific and non-specific pathological lesions on the skeleton – known as stress indicators (Goodman *et al.* 1988; Lewis & Roberts 1997; Goodman & Martin 2002; Reitsema & McIlvaine 2014; Larsen 2015). These can represent a variety of pathological conditions such as specific and non-specific infections, trauma and metabolic disturbances, as well as evidence of growth disruption (Goodman & Martin 2002; Reitsema & McIlvaine 2014). As fetal, perinatal and infant individuals represent some of the most vulnerable members of past societies (Goodman & Armelagos 1988; 1989; Rogers 1997; Lewis 2002b; 2007), indicators of stress may be more commonly identified within their remains. This is as a consequence of their immature immune system (Goodman and Armelagos 1989; Perry 2006; Halcrow & Tayles 2008), as well as the rapidity of bone turnover within fetal, perinatal and infant individuals (Lewis 2000; 2002a; 2018; Satterlee Blake 2018).

This study examined the relationship between health and growth in 99 fetal, perinatal and infant individuals from the 3rd century B.C. to the 4th century A.D in Britain. Pathological changes, combined with dental and skeletal assessment of biological age-at-death, were recorded and analysed between three samples spanning the Iron Age to Roman transition in Britain.

The concept of Roman Britain has long been the focus of scholarly debate, with ongoing consideration as to the interaction, transition and adoption of Roman cultural, social and economic practices in Britain (Molleson 1992; Hill 1995; Pitts 2008; Redfern *et al.* 2012; Rohnbogner & Lewis 2017). Though settlement patterns, housing structures, religious beliefs and trading protocols may have differed in pre-Roman Iron Age communities, these societies were not necessarily oblivious, or indeed ambivalent, towards Romanised practices. Conversely, it is now widely accepted that there were extensive relations between pre-Roman Britain and the Romanised European mainland (Hill 1995; Pitts 2008). Thus, the people who inhabited pre-Roman and Roman Britain have long been debated - who they were, where they came from and whether they were Roman, Romanised, or native (Webster 2001;

Mattingly 2006; Pitts 2008; Leach *et al.* 2009; Rohnbogner & Lewis 2017). Today, the model of 'Romanisation' is often considered a crude and narrow vision, and instead this significant cultural and social transition is conceptualised as a negotiation of identities (Pitts 2008). Consequently, the transition of Britain from that of Iron Age communities to Roman or Romanised ones, must not be considered as a rapid, brutal and bloody result of warfare. That is not to say that all communities and individuals adopted Roman customs willingly, but the perception that transition was ruthlessly forced on the entire population is untenable.

Despite this, the conquest of Britain brought with it significant changes. Until this point most of the population had lived in rural communities, typically farmsteads or small villages, focussing on agricultural subsistence (Molleson 1992; Hill 1995; Mattingly 2006; Redfern *et al.* 2012). In contrast, the Roman conquest saw the establishment of larger towns and cities (Molleson 1992; Redfern *et al.* 2012; 2015). Public bathing complexes, water systems, sanitation and sewerage systems were also established (Scobie 1986), whilst changes in diet and religious beliefs also occurred (Redfern *et al.* 2012; Rohnbogner & Lewis 2017). The rural landscape of Britain also altered with expansive villa estates established, and the introduction of new crops and farming techniques (Redfern *et al.* 2015). Consequently, with this period of transition, not only was there an influx of new people into Britain but new ideas and practices also.

Recent findings have suggested that despite the introduction of public health systems and improvements in sanitation, population health was seen to decrease in Roman Britain (e.g. Roberts & Cox; Redfern & Roberts 2005; Gowland & Redfern 2010; Redfern *et al.* 2011; 2012). Furthermore, establishment of larger towns and cities added to the increased risk of disease and infection, as high concentrations of people aided easier and quicker transmission of pathogens (Rawson 2003; Roberts & Cox 2003; Redfern & Roberts 2005). In addition, new pathogens were introduced as people from across the empire migrated to Britain (Gowland & Redfern 2010). Despite rural communities often being considered to be healthier than their urban counterparts, high prevalence rates of pathological lesions have also been identified within these groups. (Pitts & Griffin 2012; Redfern *et al.* 2015).

Therefore, this study aims to further consideration as to how this transition impacted on health and growth of fetal, perinatal and infant individuals. Despite increasing interest and

investigation into non-adult health in later time periods, individuals from Iron Age and Roman contexts, particularly rural contexts, are notoriously lacking from analyses (Rohnbogner & Lewis 2017). Assessing 99 individuals from three rural populations, spanning pre-Roman to Roman Britain (3rd Century B.C. to 4th Century A.D.), this study interprets the evidence for intra- and extrauterine growth and health disruption within the context of the rapid social and cultural changes occurring at this time.

Research Context

Owslebury was excavated between 1961 and 1972 (Collis 1977; 1994), with the main phase of inhabitation dating from the mid-2nd century B.C. to the Roman conquest of Britain. Owslebury consisted of a small Iron Age settlement, comprised of a couple of nuclear families (Collis 1977; 1994). In total 23 individuals were analysed, fourteen of whom date from the Iron Age (pre-Roman), five from the Roman period and four remain undated (Wells & Collis [No Date]; Nystrom & Swales [No Date]).

The site of Piddington was originally an Iron Age settlement with evidence of early Roman military presence at the site between 45-60 A.D. (Upper Nene Archaeological Society 2009 *Phase Descriptions;* Miller 2010). This is followed by successive phases of Romano-British settlement (1st to 3rd Centuries A.D.), during which the site evolved into a large villa compound (Miller 2010; Friendship-Taylor & Friendship-Taylor 2012). A total of 26 fetal, perinatal or infant burials are recorded as dating to the 1st century A.D. Of the 26 burials, 25 have been analysed within this study, though only 24 individuals are recorded as one double burial (12a/12b) was comingled.

Barton Court Farm consisted of a large Iron Age enclosure and settlement, which was followed by successive periods of Roman occupation and buildings (Miles 1986). The primary phases of inhabitation date to the Romano-British period, with the peak in the late 4th century A.D. (Miles 1986). To the south-east of this enclosure, an area was excavated which seemed to be reserved for infant burials (Miles 1986). In total, 53 fetal, perinatal or infantile burials were excavated from Barton Court Farm. 52 of these individuals were analysed for this study, 36 of which were Roman. Of the other burials, two were Iron Age (pre-Roman), two were Saxon (Miles 1986), and one individual, who dated from the 1st century BC to the

1st century AD, has been categorised as 'transitional' for the purposes of this research. The remaining 11 individuals were undated.

These sites were selected for assessment as they all represented rural sites which transitioned from Iron Age to Roman settlements, yet skeletal individuals recovered primarily reflected varying temporal periods (pre-Roman, Transition and Roman). Consequently, this study is able to investigate health and growth of fetal, perinatal and infant individuals during this significant cultural transition. The sites are all located in the south of Britain and had adequate sample sizes of non-adult individuals (below 6 months of age) available for assessment.

Categorical terminology of pre-Roman, Transition and Roman have been employed throughout the remainder of this study. These have been used to correlate chronological dates with the Roman conquest. Thus, the terms pre-Roman and Roman, simply reflect that individuals dated to these periods died before or after the roman conquest, whilst transitional individuals died during the period of Roman conquest. These terms have been employed to avoid any ambiguity surrounding whether individuals were Roman, Romanised or native.

Individuals from all three sites have been combined into these temporal categories, with the aim to observe growth and health disruption trends through the transition from pre-Roman to Roman Britain. Individuals who have been listed as either pre-Roman, Transition or Roman have all been securely stratigraphically dated. Dates for these categories have been given in Table 1.2. Any individual where dating was unsecure, or where no stratigraphical information was recorded, have been listed as undated. As the three archaeological sites are all located in the south of Britain, are all rural, and all were originally Iron Age settlements which transitioned into Roman sites, they are similar enough in profile to allow the combining of individuals by time period. Furthermore, consideration of individuals by site would not have enabled such detailed consideration of growth and health disruption over time, as each archaeological site primarily consisted of individuals from a single temporal period.

Table 1.1 outlines the sample sizes for each archaeological site, whilst Table 1.2 details the number of individuals by time period. Where abbreviations have been utilised, 'BCF' is an

abbreviation for Barton Court Farm, 'OW' an abbreviation for Owslebury, and 'PID' an abbreviation for Piddington.

Methods

Gestational age-at-death was estimated for each individual based on dental development and metric assessment of selected long bones, where preserved. It has been widely accepted that the relationship between metric assessment and chronological age is not a reliable one (Lewis 2007). Consequently, this study uses dental age-at-death estimates, which are considered to be more accurate (Cardoso 2007), as estimates of chronological age. Therefore, skeletal age-at-death estimates generated from metric assessment are compared to these dental age estimates for evidence of growth disruption. This practice of comparing skeletal and dental age-at-death estimations is typical when attempting to identify evidence of physiological stress (Humphrey 2000; Lewis 2007).

Diaphyseal lengths of long bones for each individual were taken using digital sliding callipers (accuracy of +/-0.02mm). Metric analyses were taken in accordance with those outlined in Fazekas & Kósa (1978), with results recorded to the nearest hundredth of a millimetre. Gestational age-at-death estimations were calculated from these measurements using the published linear regression equations for long-bone diaphyseal lengths by Scheuer *et al.* (1980). Where both left and right skeletal elements were available for assessment both were analysed and had age estimates generated, with the average age for that element used in analysis. Where individuals only had one element of a bilateral pair available for assessment (e.g. right femur only), that element has been solely used to generate an age estimate. All following analyses of long bone diaphyseal length estimates are where results have been given in this way. This use of linear regression equations was employed as it is one of the only methods available to provide error levels for age estimation, and is widely used in other studies making results of this assessment comparable (Lewis & Gowland 2007; Bonsall 2013; e.g. Mays 1993; Lewis 2002a; Halcrow *et al.* 2012; Rohnbogner & Lewis 2017). Additionally, English fetal, perinatal and infant individuals were used to develop this methodology.

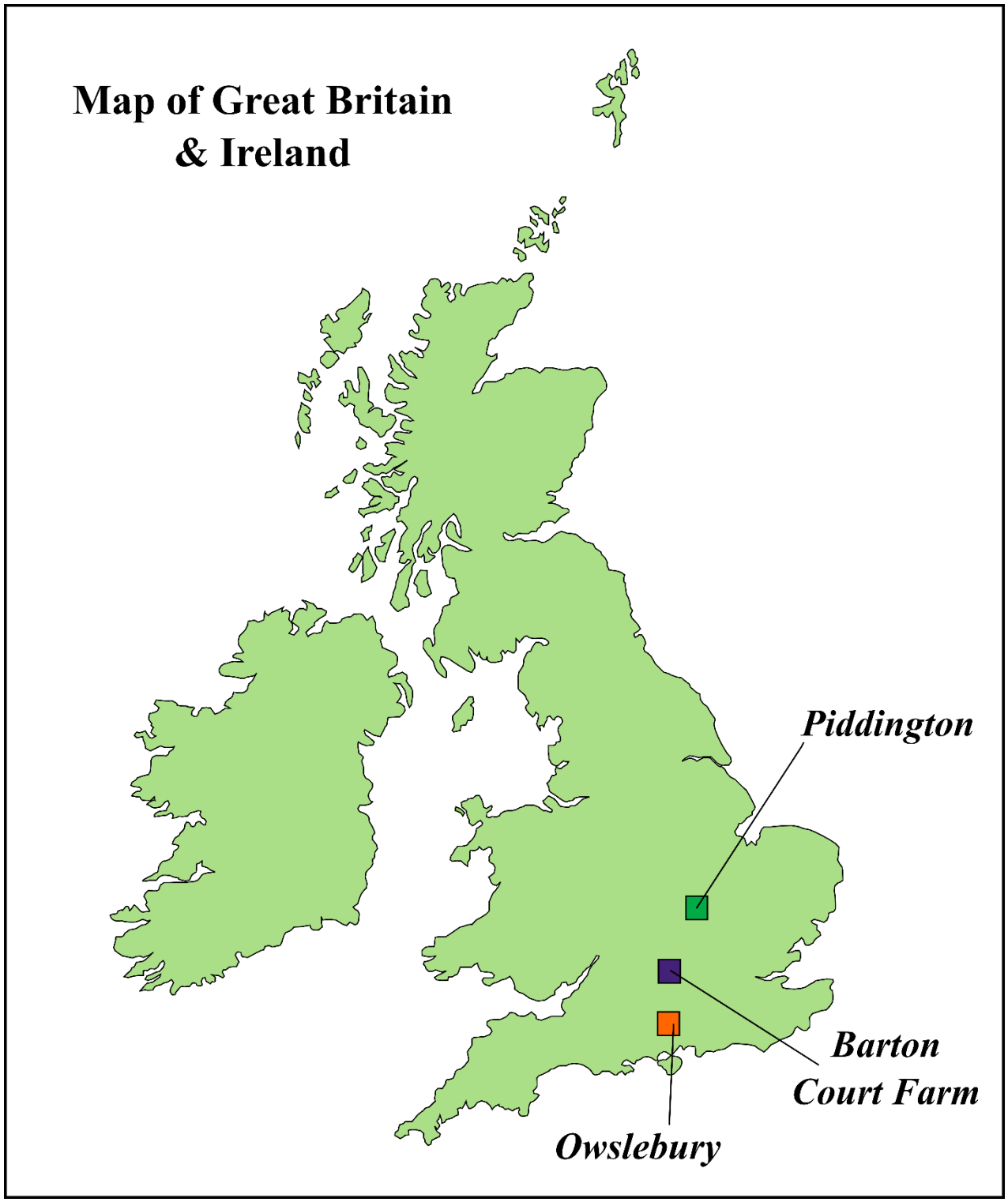

Figure 2. Map of Great Britain and Ireland displaying the location of the three skeletal samples studied; Owslebury, Hampshire, Barton Court Farm, Oxfordshire and Piddington, Northamptonshire.

TABLE 1.1 Total number (N) and percent (% given in brackets) of individuals with dentition and skeletal elements available for assessment by archaeological site. Total number of individuals where growth disruption could be assessed is also given by archaeological site.

Time Period (Centuries)	Archaeological Site	Location	Total *N*	Age-at-Death Assessment		Assessment of Growth Disruption
				Dentition	Skeletal Elements	
3rd BC – 4th AD	Owslebury	Hampshire, U.K.	23	13 (57)	16 (70)	11 (48)
1st AD	Piddington	Northamptonshire, U.K.	24	16 (67)	22 (92)	15 (63)
1st BC – 4th AD	Barton Court Farm	Oxfordshire, U.K.	52	19 (37)	48 (92)	19 (37)
			99	48 (48)	86 (87)	45 (45)

TABLE 1.2 Total number (N) and percent (% given in brackets) of individuals with dentition and skeletal elements available for assessment by time period. Total number of individuals where growth disruption could be assessed is also given by time period.

Time Period (Centuries)	Historical Time Period	Total *N*	Age-at-Death Assessment		Assessment of Growth Disruption
			Dentition	Skeletal Elements	
3rd BC - 1st AD	Pre-Roman	16	13 (81)	13 (81)	11 (69)
1st AD	Transition	27	16 (59)	25 (93)	15 (56)
1st AD – 3rd AD	Roman	39	14 (36)	36 (92)	14 (36)
3rd AD – 4th AD	Saxon	2	-	2 (100)	-
Unknown	Undated	15	5 (33)	10 (67)	5 (33)
		99	48 (48)	86 (87)	45 (45)

TABLE 1.3 Total number (N) and percent (% given in brackets) of individuals by chronological age category, based on dental development age estimate, for each archaeological site.

Archaeological Site	**Total *N***	**Sample Size (*N* (%)) by Chronological Age Category (Based on Dental Development)**			
		Fetal	**Perinatal**	**Infant**	**Unknown**
Owslebury	23	1 (4)	6 (26)	6 (26)	10 (43)
Piddington	24	-	3 (13)	13 (54)	8 (33)
Barton Court Farm	52	1 (2)	4 (8)	14 (27)	33 (63)
	99	2 (2)	13 (13)	33 (33)	51 (52)

TABLE 1.4 Total number (N) and percent (% given in brackets) of individuals with skeletal elements (femur, humerus, tibia, pars basilaris) by archaeological site.

Archaeological Site	**Total *N***	**Sample Size (*N* (%)) by Skeletal Element**			
		Femur	**Humerus**	**Tibia**	***Pars Basilaris***
Owslebury	23	11 (48)	8 (35)	6 (26)	15 (65)
Piddington	24	15 (63)	17 (71)	14 (58)	14 (58)
Barton Court Farm	52	37 (71)	33 (63)	28 (54)	19 (37)
	99	63 (64)	58 (59)	48 (48)	48 (48)

TABLE 1.5 Total number (N) and percent (% given in brackets) of individuals by chronological age category, based on dental development age estimates, for each historical time period.

Historical Time Period	Total *N*	Sample Size (*N*) by Chronological Age Category (Based on Dental Development)			
		Fetal	**Perinatal**	**Infant**	**Unknown**
Pre-Roman	16	-	7 (44)	6 (38)	3 (19)
Transition	27	-	3 (11)	13 (48)	11 (41)
Roman	39	1 (3)	2 (5)	11 (28)	25 (64)
Saxon	2	-	-	-	2 (100)
Undated	15	1 (7)	1 (7)	3 (20)	10 (67)
	99	2 (2)	13 (13)	33 (33)	51 (52)

TABLE 1.6 Total number (N) and percent (% given in brackets) of individuals with skeletal elements (femur, humerus, tibia, pars basilaris) by time period.

Historical Time Period	Total *N*	Sample Size (*N*) by Skeletal Element			
		Femur	**Humerus**	**Tibia**	***Pars Basilaris***
Pre-Roman	16	11 (69)	9 (56)	6 (38)	12 (75)
Transition	27	17 (63)	18 (67)	15 (56)	16 (59)
Roman	39	27 (69)	24 (62)	22 (56)	15 (38)
Saxon	2	1 (50)	2 (100)	-	-
Undated	15	7 (47)	5 (33)	5 (33)	5 (33)
	99	63 (64)	58 (59)	48 (48)	48 (48)

Chronological age estimates generated have been plotted with the error level given as a range in accordance with the +/- gestational weeks given by Scheuer *et al.* (1980) for each skeletal element. All skeletal age-at-death estimations have been given in gestational weeks throughout (GWA).

The *pars basilaris* has also been assessed to determine whether individuals were fetal, perinatal or infantile. This bone was utilised as it is often recovered archaeologically due to its robust nature (Redfield 1970; Scheuer & Maclaughlin-Black 1994), and because it is indicative of certain aging thresholds, with both its size and morphology often found to be correlated strongly with age (Redfield 1970; Scheuer & Maclaughlin-Black 1994; Lewis 2007). Metric assessment of the *pars basilaris* was undertaken in accordance with the methods published by both Fazekas & Kósa (1978) and Schaefer *et al.* (2009), with sagittal length, maximum length and maximum width all recorded where possible. Measurements were directly compared against those given in Scheuer & Maclaughlin-Black (1994). Where a single age estimate has been used for the *pars basilaris*, an average age estimate has been derived from the three potential dimensions analysed.

Direct measurements from the femur and humerus have also been plotted against clinical reference data and growth charts. Centiles are provided whereby the 50th centile represents the median measurement for that age group, with 50% of individuals falling above and below that point (Dodrill 2016). Therefore, the 25th centile is where 25% of individuals fall below this measurement, with the 75th centile being where 25% of individuals are above this measurement. It is suggested that those individuals who fall outside of the 10th and 90th percentiles are clinically significant (Kiserud *et al.* 2017). Indeed, those whose skeletal growth falls below the 10th percentile are often found to have negative birth outcomes (Kiserud *et al.* 2017). For prenatal (40 GWA or less) the clinical growth charts employed are those derived from the World Health Organisation, as given in Kiserud *et al.* (2017). For postnatal individuals, growth charts for the femur and humerus given by Maresh (1970) have been used as this data set has been found to represent normal, healthy growth, and be a suitable reference for archaeological individuals (Schillaci *et al.* 2012).

Dental development is regarded as a more accurate method for estimating gestational age as it is less susceptible to external factors, and thus less easily disrupted (Garn *et al.* 1960; Hillson

2005; AlQahtani *et al.* 2010; Bonsall 2013). Tooth cusp development was recorded in accordance with Moorrees *et al.* (1963) and age-at-death estimates attributed using the dental development atlas developed by AlQahtani *et al.* (2010). If dental development was considered to fall between two age categories, the mid-point between those age groups was recorded, with the largest error/range level for either category afforded. All dental age-at-death estimations have been given in gestational weeks throughout (GWA).

Assessment of the presence and characteristics of pathological lesions was undertaken macroscopically. Due to pathological lesions being troublesome to identify and record within non-adult skeletal remains, this thesis employed a strategy whereby no particular 'stress indicator' or skeletal element was to be observed, instead considering the whole of the skeleton and recording any potential pathological change. Each pathological change/lesion was recorded descriptively by the author and documented photographically.

Location was firstly documented as cranial or postcranial, then by specific skeletal element (e.g. right tibia, left frontal bone) and then by aspect (e.g. endocranial, ectocranial, medial, lateral). The location was documented in this very specific way so that pathological lesions could be recorded and located as precisely as possible. Type of pathology was recorded as either 'NBF' (New Bone Formation) or 'Lytic' for where bone destruction/resorption was evident. For NBF, type of bone formation was also recorded. Type of bone was recorded as either woven or lamellar bone, and also whether it was spiculated (Ortner 2003). Expansion of the metaphyses was also recorded when present, as well as any morphological changes (e.g. bowing to the limbs). As normal bone growth and pathological bone formation can currently not be distinguished between, any evidence of NBF was recorded, though a grading scheme was employed to differentiate between extensive NBF and that which was only minor. Grading systems were also established to consider severity of lytic lesions and metaphyseal expansion. For each type of pathology, a severity score of 1, 2 or 3 has been afforded, with 1 being the least severely affected and 3 being the most. Table 2. outlines the grading systems employed.

TABLE 2. Grading systems for new bone formation, lytic lesions and metaphyseal expansion employed for assessment of pathological lesions within this study.

	Grade 1	Grade 2	Grade 3
New Bone Formation	New bone formation, which may be woven or lamellar in appearance, will be considered to be grade 1 when the NBF is not clearly apparent and the margins are unable to be clearly defined from that of normal cortical bone. Grade 1 NBF is likely to be isolated in location, appearing minimally across the skeletal element.	New bone formation recorded as being grade 2 will be clearly identifiable as a definable area of woven or lamellar bone formation. There will be clear boundaries/borders to the NBF and it will obviously differ from the normal cortical bone of the skeletal element. Grade 2 NBF is likely to be distinguishable as a clear layer of bone on top of the original cortical surface. It is likely that NBF listed within this category will be formed of a single layer though may extend over a large aspect area of the skeletal element.	New bone formation recorded as being grade 3 will be the more severe type of NBF, with clear, multi-layered or thick NBF across a large area/aspect of the skeletal element. The NBF may be woven or lamellar in appearance and is clearly seen to be on top of the original cortical bone.
Lytic Lesions	Lytic lesions considered to be grade 1 likely consist primarily of macro-porosity. This porosity will be relatively minor, though may extend over a large skeletal area, and no clear destruction of the cortical bone will be apparent.	Lytic lesions considered to be grade 2 will likely show evidence of some cortical destruction as well as porosity. However, cortical destruction will not be widespread throughout the skeletal element and is instead likely to be in isolated concentrations.	Lytic lesions considered to be grade 3 will show extensive cortical destruction and/or porosity. Destruction will be widespread throughout the element.
Metaphyseal Expansion	Metaphyseal expansion considered to be grade 1 will likely consist of noticeably widened/flared metaphyses which do not appear proportional for the long bone diaphysis. However, despite this expansion no change to the metaphyseal margin or trabecular bone structure will be observed.	Metaphyseal expansion will be considered to be grade 2 when involvement of the metaphyseal margin is apparent. This will result in atypical and misshapen metaphyseal margins often combined with a discernible brim/lip to the metaphysis.	Metaphyseal expansion considered to be grade 3 will be the most severe and where involvement of the trabecular bone structure can be seen. Individuals displaying grade 3 metaphyseal extension will likely have more porous metaphyses and the trabecular structure will appear clearly expanded and widened. Involvement of the metaphyseal margin may still be apparent though this may be lost due to the trabecular expansion.

Statistical analysis to assess growth disruption was assessed using T-test analyses. T-tests were employed to determine significant differences in skeletal metrics/age estimations when compared by dental age group. T-test analysis was undertaken in PAST (developed by Hammer, Harper & Ryan 2001). Significance was set to 0.05 (95% confidence), whereby *p*-values below 0.05 were considered to be statistically significant. Prevalence rates were calculated for pathological lesions by documenting how many individuals out of the 99 analysed had a particular skeletal element(s) present, and then out of those with the element(s), how many showed pathological changes. Totals have been given for numbers of individuals assessed, along with separate columns of totals for the number of a particular element able to be observed, and then the number found to be affected and showing pathological changes. Chi-squared tests for independence at 99.5 % confidence ($p < 0.05$) were also employed for pathological categories to observe whether there was any relationship between various pathological variables. Chi-squared analyses were employed for pathological lesions by time period (3 x 2) and dental age (7 x 2). Chi-square results are presented numerically, where $p < 0.05$ shows there is a significant relationship between the variables. Chi-Squared values (X^2) have also been given. As the sample sizes were small, Fisher's exact test was used to determine *p* values.

For this study fetal individuals are those assessed to be 36 gestational weeks and under, perinates were classed as those aged 36-44 gestational weeks, and the term infant was used for those over 44 gestational weeks and up to six months of age (64 GWA), the upper age limit of this study.

Tables 1.1 and 1.2 detail the individuals assessed by archaeological site and by time period, listing the number of individuals who had dental and/or skeletal elements available for analysis. Numbers of individuals available for assessment of growth disruption (where both dental and skeletal age estimates were recorded) have also been given. The number of individuals by age category, based on dental age assessment, have been given by site (Table 1.3) and time period (Table 1.5). The number of individuals with various skeletal elements available for assessment (femur, humerus, tibia, *pars basilaris*) have also been given by site (Table 1.4) and time period (Table 1.6).

Results

In total 99 fetal, perinatal and infantile individuals were analysed from across the three sites, 48 of whom had dentition present for analysis, with 86 individuals having at least one skeletal element (femur, humerus, tibia or *pars basilaris*) available for assessment. As a result, growth disruption – comparison between dental and skeletal age-at-death estimations – could be assessed in 45 individuals.

When the frequency of age-at-death estimates were considered for all 99 individuals by chronological age category (fetus, perinate, infant), dental and skeletal methods of assessment were found to yield very different interpretations regarding age (Table 3.). Analysis of dental development revealed that the majority of individuals (*N*=33) were aged to be infants (> 44 GWA). Conversely, assessment of long bone diaphyseal length suggested that almost all of the individuals were perinatal (36-44 GWA). Assessment of long bone length revealed no individuals skeletally determined to be infantile (> 44GWA), with only one individual determined to be fetal in assessment of both the humerus and tibia. Analysis of the *pars basilaris* again suggested that the majority of individuals were perinatal, though four individuals were aged to be infants.

TABLE 3. Frequency of individuals (N) by chronological age category (fetus, perinate, infant) for dental and skeletal methods of age estimation.

	Dentition	**Femur**	**Humerus**	**Tibia**	***Pars Basilaris***
Fetus	2	0	1	1	0
Perinate	13	63	57	47	44
Infant	33	0	0	0	4
Unknown	51	36	41	51	51

Individuals who could be ascribed to a particular time period (*N*=84) were considered by chronological age category (Fig. 2). This pattern, whereby dentition typically generated older age estimates in comparison to skeletal assessment, is clearly observable in the pre-Roman, Transition and Roman samples. Both Transition and Roman samples have many more

individuals dentally aged to be infants than perinates. Three Transition individuals are dentally aged as perinates, compared to 13 individuals determined to be infantile, whilst two Roman individuals are perinatal, and 11 infantile based on assessment of dental development. Only the pre-Roman individuals show an alternating trend, whereby more individuals (*N*=7) are dentally determined to be perinatal rather than infantile (*N*=6). In comparison, disregarding the individuals listed as 'unknown' who did not have particular skeletal elements available for assessment, skeletal age typically determines individuals to be perinatal, rather than fetal or infantile. Only the *pars basilaris* generates skeletal age estimates over 44 GWA.

Assessment of the frequency of age-at-death estimates (in GWA), for both dental development and metric assessment of varying skeletal elements (Fig. 3), further demonstrates that skeletal remains typically generate younger age estimates than assessment of the dentition. For all three of the selected long bones, age-at-death estimations were found to peak between 37 and 40 GWA. In particular, the femora have a very narrow range of age estimations, (36 to 43 GWA), whereas the tibiae have the broadest (32 to 44 GWA). The *pars basilaris*, despite the majority of age estimations also clustering around 39-40 GWA, is identified as the skeletal element with the widest range of age-at-death estimates (38 to 56 GWA).

Of those with dentition (*N*=48), the average dental age-at-death estimate is 46 GWA. From assessment of diaphyseal length, the average age-at-death estimate for those with femora (*N*=63) is 39 GWA; the average age estimate from tibial diaphyseal length (*N*=48) is 40 GWA; and of those with humeri (*N*=58) the average age-at-death estimate is 39 GWA. Therefore, between dental and long bone age estimates there is at least a six-week age difference on average. The average age-at-death estimation of the *pars basilaris* is 41 GWA. However, these averages do not account for whether the dental and skeletal elements analysed derive from different individuals. Therefore, to account for this, individuals have been assessed discretely.

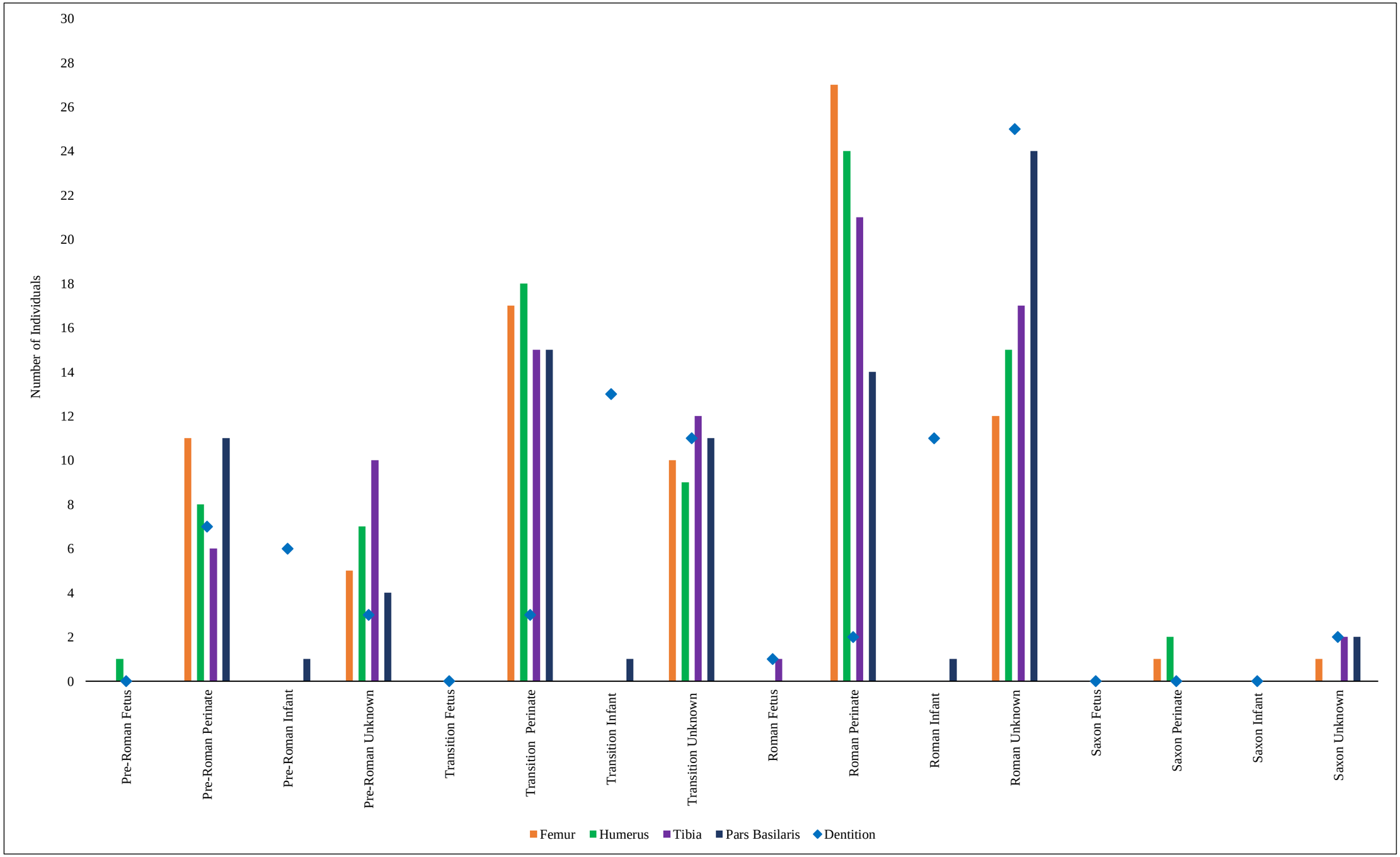

Figure 2. Number of individuals determined to be fetal, perinatal, infantile or unknown by time period (pre-Roman, Transition, Roman or Saxon). Numbers of individuals for each chronological age category are based on assessment of femoral, humeral, tibial and pars basilaris metric assessment, as well as by assessment of dental development.

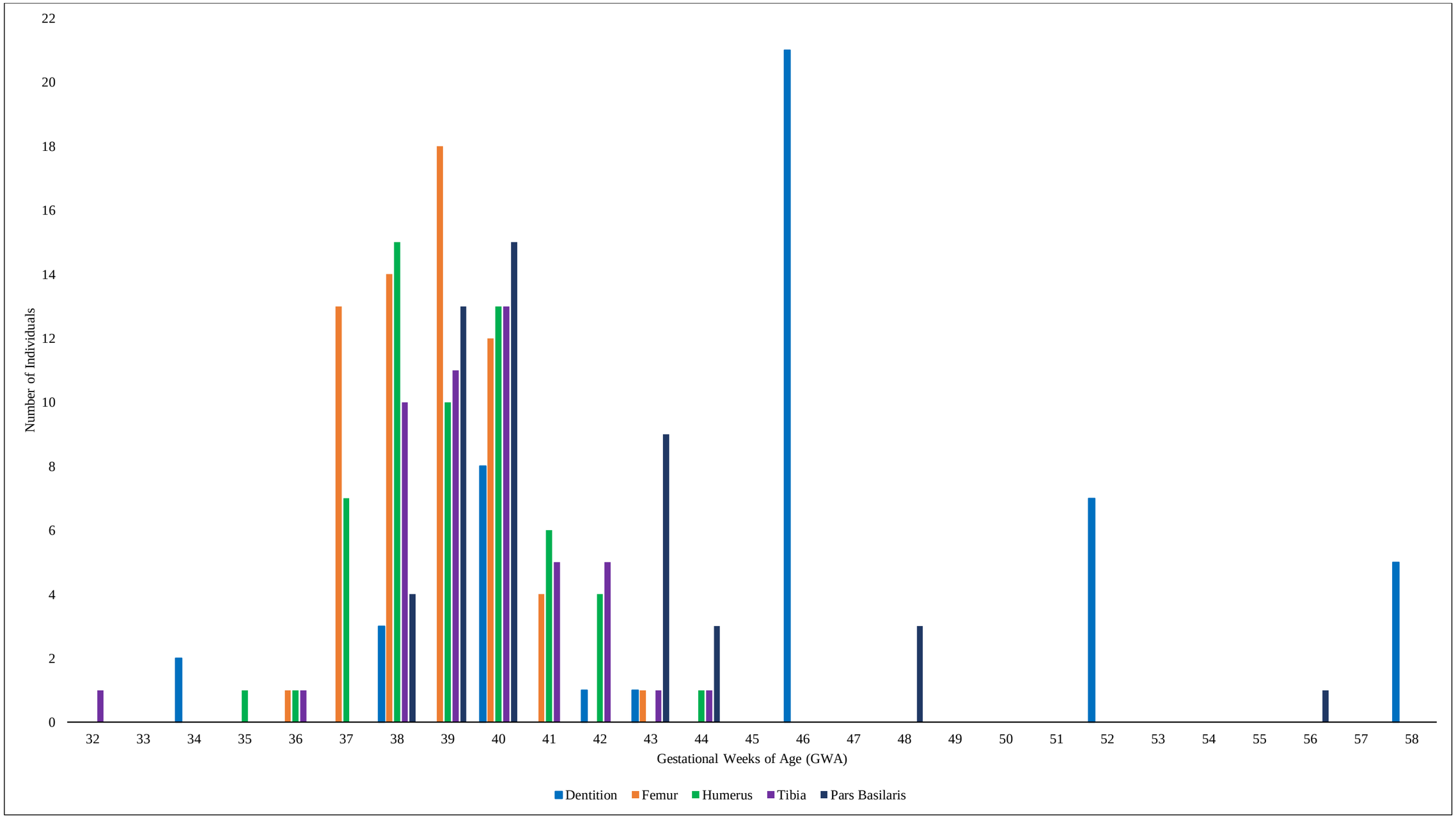

Figure 3. Number of individuals by gestational week based on assessment of femoral, humeral, tibial and pars basilaris metrics, as well as assessment of dental development.

Of the 48 individuals with dentition, 33 also have a femoral age-at-death estimate, with 27 individuals having a tibial age estimate, and 35 individuals a humeral age-at-death estimate. Therefore, a total of 39 individuals have at least one long bone element where diaphyseal length can be assessed for age estimation. This means that for these 39 individuals dental and skeletal age-at-death estimates can be directly compared. Figure 4. plots these 39 individuals, in ascending order according to their dental age estimates, with their respective femoral, humeral and tibial age estimates also plotted where possible.

In total, four individuals have long bone length age estimates which plot above dental age: one individual dentally aged to be 34 GWA (BCF 400), one individual dentally aged to be 38 GWA (BCF 860), and two individuals dentally aged to be 40 GWA (OW 43 and BCF 913). The remaining 35 individuals have long bone length age estimates that are younger than dental development. The majority of these long bone length age estimates fall within the error range of the corresponding dental age estimates, though most are towards the tail end. For the older infants (52 GWA and older), diaphyseal length age estimates are younger than dental ages and error ranges in all but one individual (BCF 905i). However, where error ranges for skeletal estimates are also considered, only three individuals have dental and skeletal age estimates and ranges which do not correspond or overlay. All three of these individuals are dentally aged to be 58 GWA and are from Barton Court Farm. There is one individual from the pre-Roman period, one Roman individual, and one undated individual.

Table 4 displays the average differences, in gestational weeks, between dental age-at-death estimates and those generated from femoral, humeral and tibial diaphyseal length measurements. To obtain accurate differences between dental and skeletal age estimates, only individuals who had both elements available for assessment were used to calculate these averages. Differences have been given for each of the long bone elements, grouped by both time period and dental age. Results suggest that individuals dating to the Transition and Roman periods have marginally greater differences on average between dental and skeletal age estimates than those from the pre-Roman period. Age-at-death estimates derived from the Roman femora are particularly young when compared to dental age-at-death estimates. The average difference between skeletal and dental age-at-death can also be seen to substantially increase as gestational age also increases.

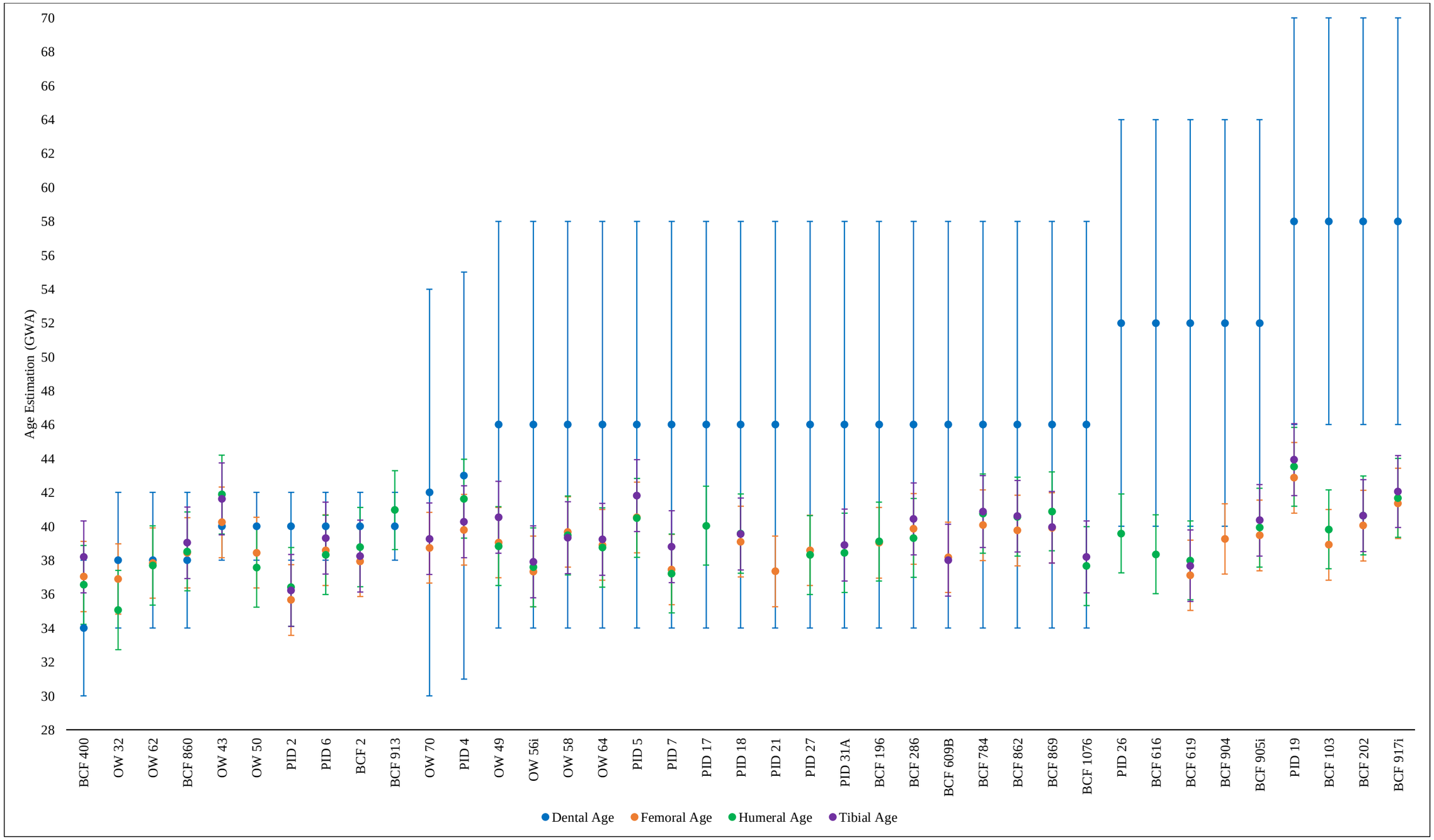

Figure 4. Each individual with a dental age-at-death estimate and their corresponding skeletal age-at-death estimates derived from femoral, humeral and/or tibial diaphyseal length. Error bars have been given for each age estimate in accordance with error ranges provided by AlQahtani et al. (2010) for dental age estimates and by Sheuer et al. (1980) for diaphyseal length estimates. Individuals have been plotted in ascending order according to dental age-at-death estimate.

TABLE 4. Average differences (in gestational weeks) between dental and femoral, humeral and tibial age-at-death estimations. These differences have been categorised by time period and by dental age estimation. Number of individuals (N) is given for each group considered. Results in italics are those where only one individual had both dental and the particular skeletal element available for age-at-death assessment.

	Dental - Femur		**Dental - Humerus**		**Dental - Tibia**	
	N	**GWA**	***N***	**GWA**	***N***	**GWA**
Overall	33	7	35	7	27	6
Pre-Roman	10	5	9	5	6	4
Transition	9	7	12	7	8	6
Roman	10	8	11	7	10	6
Undated	4	9	3	8	3	9
34 GWA	*1*	*-3*	*1*	*-3*	*1*	*-4*
38 GWA	3	0	3	1	*1*	*-1*
40 GWA	5	2	6	1	4	1
42 GWA	*1*	*3*	-	-	*1*	*3*
43 GWA	*1*	*3*	*1*	*2*	*1*	*3*
46 GWA	15	7	16	7	14	6
52 GWA	3	13	4	13	2	13
58 GWA	4	17	4	16	3	17

T-test statistical assessment was undertaken to determine which, if any, skeletal age-at-death estimates were significantly different from dental age-at-death estimates. Skeletal age estimates derived from assessment of diaphyseal length for each skeletal element (femur, humerus and tibia) were compared by dental age group to dental age (in gestational weeks). Summary statistics (mean, 95% confidence intervals and significance (where $p < 0.05$)) were established for each element by age group (Table 5.). Only dental age groups where more than one skeletal age estimate was present have been tested. Results show that for all skeletal elements, average skeletal age follows the trend of increasing with dental age. Statistically

significant differences have been identified between dental and skeletal age-at-death estimations, primarily for the dental age groups of 46 GWA and older.

TABLE 5. Summary statistics and the results of t-test analyses considering evidence of significant differences between dental and skeletal age-at-death estimates.

	Dental Age	Mean Skeletal Age	T-test	Lower 95% Conf. Int.	Higher 95% Conf. Int.	*P*	SIG
Femur	38	37.7	-0.69	35.8	39.6	0.56	N
	40	38.2	-2.54	36.1	40.2	0.06	N
	46	39.0	-26.20	38.4	39.6	2.71E-13	Y
	52	38.6	-17.39	35.3	41.9	0.003	Y
	58	40.8	-19.84	38.0	43.6	0.0002	Y
Humerus	38	37.1	-0.88	32.7	41.5	0.47	N
	40	39	-1.18	36.8	41.2	0.29	N
	46	39.2	-23.21	38.6	49.8	3.62E-13	Y
	52	39.0	-28.38	37.5	40.4	9.60E-05	Y
	58	41.4	-20.72	38.9	43.9	0.0002	Y
Tibia	40	38.8	-1.04	35.2	42.4	0.37	N
	46	39.6	-20.53	38.9	40.2	2.73E-11	Y
	52	39.1	-9.59	21.9	56.2	0.07	N
	58	42.2	-16.56	38.1	46.3	0.004	Y

To determine the significance and potential health implications for evidence of growth disruption, direct comparison of femoral and humeral diaphyseal length measurements (mm) against clinical reference data (WHO: Kiserud *et al.* 2017 and Maresh 1970) was undertaken (Fig. 5.1; 5.2). This was to eliminate any error in converting skeletal diaphyseal length measurements to chronological age estimates, and observe whether any individuals had measurements which fall into, or below, clinically significant centiles (10^{th} and 90^{th} centiles).

Only individuals who had dental age estimates could be considered against these growth charts, where skeletal measurements (mm) were plotted by dental age (GWA).

Assessment of femoral measurements against clinical reference data demonstrates that almost all individuals who are dentally considered to be fetal and perinatal (< 44 GWA) show no evidence of growth disruption. Conversely, two individuals (BCF 400 and OW 43) have femoral diaphyseal lengths that are above the 90th percentile for growth according to WHO standards. A further individual (BCF 860) is also shown to have femoral lengths which align very closely with the 90th growth percentile. Only one individual (PID 2) appears to show significant growth disruption, falling below the 10th centile. When considering these results against age estimates generated from these measurements (Fig. 4), it can be seen the age estimates generated for PID 2 are much younger than the dental estimate. However, due to the large error ranges of the age estimation methods, growth disruption could not be definitively suggested for this individual considering these age estimates alone. Therefore, by directly comparing diaphyseal lengths to clinical data, this individual can be confirmed to show evidence of significant, and likely detrimental, growth disruption. For individuals dentally considered to be infants (> 44 GWA), the majority fall below the 10th growth centile as given by Maresh (1970); all individuals aged 52 and 58 GWA have femoral measurements which are below the 10th growth centile, whilst those aged 46 GWA show a mixed pattern. In total, ten individuals aged 46 GWA (listed on Fig. 5.1) have femoral measurements below the 10th growth centile. Three further individuals aged 46 GWA have femoral measurements which cluster around the 10th growth centile for male individuals, but which still fall below the 10th growth centile for females. Only one individual aged 46 GWA (PID 5) has a femoral measurement which is above the 10th growth centile for either male or female individuals. Consideration of femoral diaphyseal length measurements against clinical data suggests infant individuals (those older than 44 GWA) appear to experience greater levels of growth disruption compared to perinatal and fetal individuals.

Consideration of humeral diaphyseal length measurements has revealed a similar pattern, whereby minimal growth disruption is identified in fetal and perinatal individuals (those younger than 44 GWA), whereas the majority of infant individuals (> 44 GWA) show evidence of growth disruption, falling below the 10th growth centile. Only one fetal individual (OW 32), aged to be 38 GWA, shows evidence of clinically significant growth disruption.

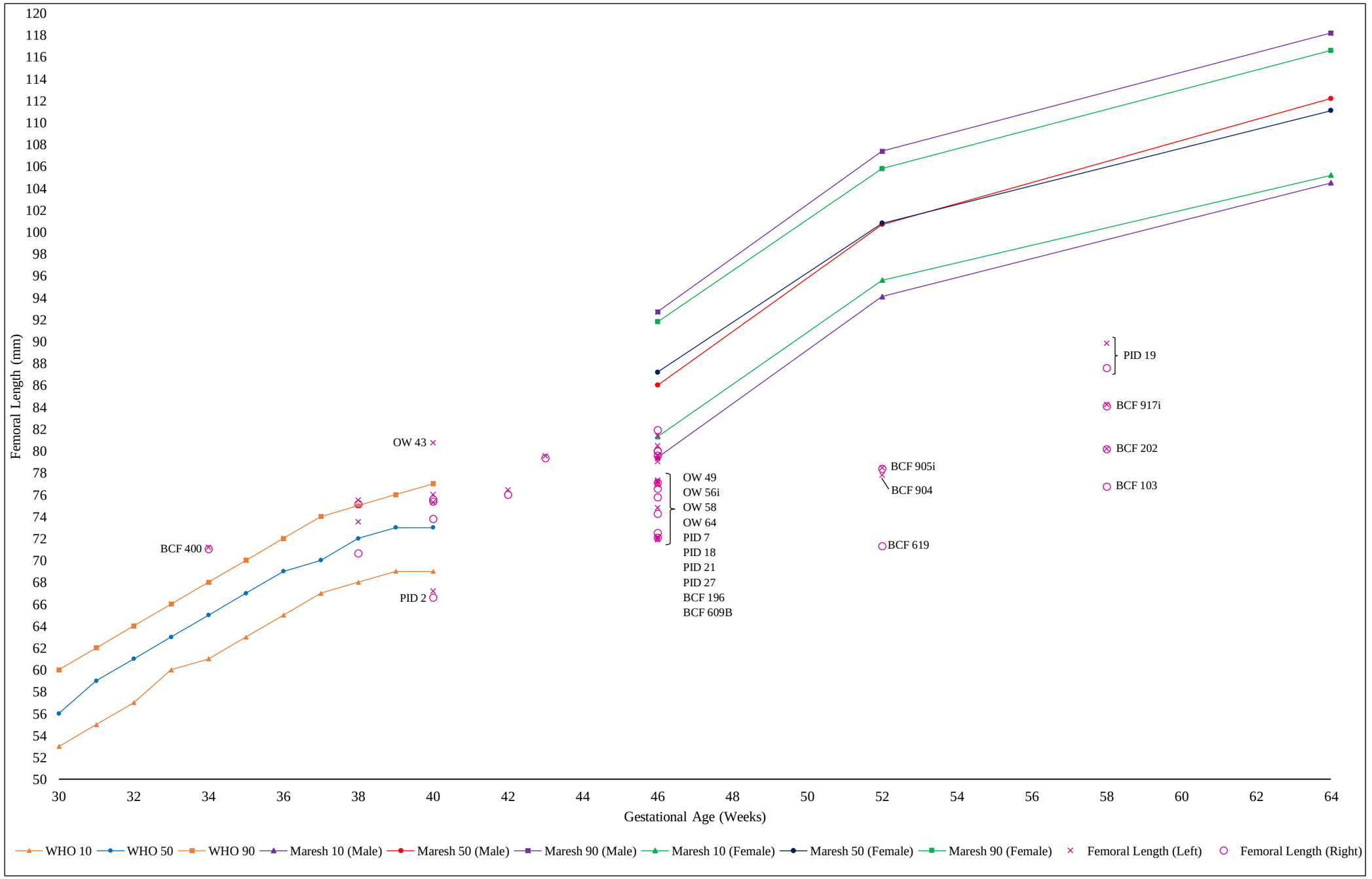

Figure 5.1 Comparison of individuals with femoral diaphyseal length measurements against clinical reference data/growth charts (WHO: Kiserud et al. 2017; Maresh 1970). Femoral measurements taken from the archaeological individuals have been plotted in accordance with their dental age-at-death estimates in GWA.

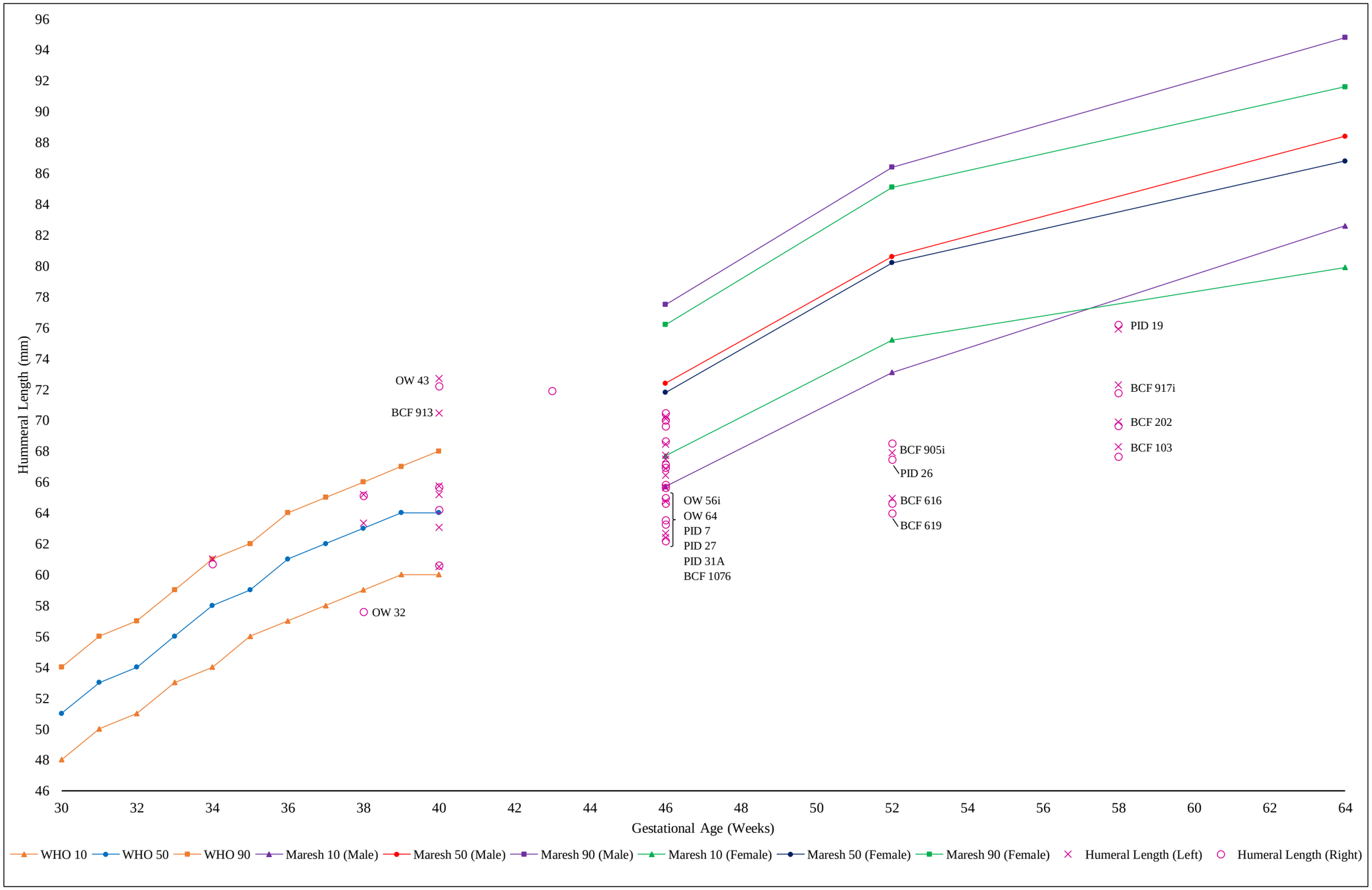

Figure 5.2 Comparison of individuals with humeral diaphyseal length measurements against clinical reference data/growth charts (WHO: Kiserud et al. 2017; Maresh 1970). Humeral measurements taken from the archaeological individuals have been plotted in accordance with their dental age-at-death estimates in GWA.

Again, two individuals (BCF 913 and OW 43), both dentally aged to be 40 GWA, show evidence of growth beyond the 90th percentile. The majority of the fetal individuals can be seen to have humeral measurements which fall between the 10th and 90th growth percentiles. For the infant individuals the humeral diaphyseal length, like that of the femur, can be seen to consistently fall below the 10th centile for individuals aged 52 and 58 GWA. However, these humeral measurements are closer to the 10th centile than those of the corresponding femoral measurements. This may indicate that the humerus is seemingly reflecting less growth disruption. This is corroborated by those individuals aged to be 46 GWA where, although six individuals still fall below the 10th growth centile, more individuals have humeral lengths which fall between the 50th and 10th growth centiles (both male and female) in comparison to femoral diaphyseal lengths. For both femoral and humeral diaphyseal lengths no infant individual has a measurement which falls above the 50th growth percentile. It must also be noted that where bilateral pairs of elements were available for assessment, their measurements are often coupled when plotted.

True Prevalence Rates (TPR) of pathological lesions observed on both the cranial and postcranial elements are presented in Table 6.1. Individuals were found to have high prevalence rates of cranial pathological lesions, with over 50% of individuals affected from all periods. The TPR for the transitional period individuals is exceptionally high for both cranial and postcranial lesions, while the Roman individuals also have high levels of pathology. In total, 165 skeletal elements within the individuals assessed had evidence of NBF, whilst only 5 elements showed evidence of lytic lesions. Of the NBF identified, 89 elements consisted of lamellar bone, 76 of woven and one of spiculated. Additionally, regarding severity, the majority of elements (N=96) were found to be grade two, whilst 71 elements were grade one. Only three skeletal elements were recorded as being grade three. Two skeletal elements showed evidence of metaphyseal expansion, with one pre-Roman individual and one Roman individual affected. Eighteen skeletal elements showed evidence of morphological changes, again with the highest prevalence rate being amongst Transition individuals.

When prevalence rates of pathological lesions are considered by skeletal element (frontal bone, parietal bone, occipital bone, humerus, femur, tibia) the frontal bone in transitional individuals can be seen to have the highest rate of lesions from the cranial elements (Table

6.2). The transitional individuals also have the highest prevalence rates of postcranial pathology, with 55% of individuals showing changes to their tibiae. In total, 83% of individuals observed show pathological changes to the frontal bone, the highest for the cranial elements. Comparatively, 42% of individuals observed showed pathological changes to their tibiae. When the type of pathology is considered by affected skeletal elements, 96-97% of cranial pathologies were found to be NBF, with 60-100% of lesions found to be NBF in the postcranial affected elements.

Assessment of NBF by affected skeletal elements for both type (woven, lamellar, spiculated) and severity (Grade 1, 2, or 3) is presented in Table 6.3. For both the frontal bone and parietal bone all NBF lesions are lamellar, whereas the occipital bone typically has more NBF lesions which are woven in appearance. For the postcranial elements the majority of NBF lesions are woven bone. When divided by skeletal element, typically most NBF lesions were found to have a severity of grade two.

True prevalence rates of pathological lesions were also considered for each time period by dental age (Table 6.4). However, small sample sizes of individuals when broken down by dental age means all of the age categories have high percentage rates of pathological lesions.

Chi-squared statistical assessment of pathological lesions by skeletal element (Table 6.5) (where $p < 0.05$) revealed significant associations between femoral and tibial pathology by time period (X^2=11.348 for both), as well as statically significant association between femoral pathology and dental age (X^2=13.015). Pre-Roman individuals were found to have significantly more tibial pathology than Transition individuals ($p = 0.015$) when using Bonferroni's adjusted p value. Additionally, Transition individuals have significantly more femoral pathology than Roman individuals ($p = 0.004$).

TABLE 6.1 Number and percentage of individuals observed with cranial and postcranial pathology given by time period. Total number of skeletal elements by type of pathology and severity have also been given by time period.

Time Period	*N*	Postcranial Elements		Cranial Elements		NBF	Lytic	Metaphyseal Expansion	Morphological Change	Woven	Lamellar	Spiculated	Severity		
		Observed	Affected N (%)	Observed	Affected N (%)								1	2	3
Pre-Roman	16	13	3 (23)	14	8 (57)	12	4	1	4	2	10	0	2	14	0
Transition	27	24	17 (71)	26	24 (92)	83	0	1	8	46	37	1	36	45	2
Roman	39	35	14 (40)	29	22 (76)	70	1	0	6	28	42	0	33	37	1
						165	5	2	18	76	89	1	71	96	3

Table 6.2 Number and percentage of individuals observed with pathology by skeletal element for each time period. Of those with pathology, the number and percentage of individuals who had lesions which were NBF have been given.

	Frontal Bone			Parietal Bone			Occipital Bone			Humerus			Femur			Tibia		
	Obs.	Affected N (%)	NBF N (%)	Obs.	Affected N (%)	NBF N (%)	Obs.	Affected N (%)	NBF N (%)	Obs.	Affected N (%)	NBF N (%)	Ob.	Affected N (%)	NBF N (%)	Ob.	Affected N (%)	NBF N (%)
Pre-Roman	7	5 (71)	4 (80)	4	3 (75)	2 (67)	13	3 (23)	2 (67)	12	0	-	11	1 (9)	0	9	1 (11)	1 (100)
Transition	17	15 (88)	15 (100)	18	13 (72)	13 (100)	21	13 (62)	13 (100)	21	3 (14)	2 (67)	21	10 (48)	9 (90)	20	11 (55)	11 (100)
Roman	17	14 (82)	14 (100)	16	11 (69)	11 (100)	24	11 (46)	11 (100)	27	2 (7)	1 (50)	29	3 (10)	3 (100)	23	10 (43)	10 (100)
Total	41	34 (83)	33 (97)	38	27 (71)	26 (96)	58	27 (47)	26 (96)	60	5 (8)	3 (60)	61	14 (23)	12 (86)	52	22 (42)	22 (100)

TABLE 6.3 Of those individuals recorded with pathological NBF (see Table 6.2), the number of individuals with woven and/or lamellar lesions has been given for each skeletal element by time period. The frequency of the severity (1, 2, or 3) of the woven and/or lamellar lesions has also been recorded.

	Frontal Bone					**Parietal Bone**					**Occipital Bone**					**Humerus**					**Femur**					**Tibia**				
	Woven	**Lamellar**	**1**	**2**	**3**	**Woven**	**Lamellar**	**1**	**2**	**3**	**Woven**	**Lamellar**	**1**	**2**	**3**	**Woven**	**Lamellar**	**1**	**2**	**3**	**Woven**	**Lamellar**	**1**	**2**	**3**	**Woven**	**Lamellar**	**1**	**2**	**3**
Pre-Roman	0	4 (100)	0	4 (100)	0	0	2 (100)	0	2 (100)	0	1 (5)	1 (5)	0	2 (100)	0	-	-	-	-	-	-	-	-	-	-	1 (100)	0	0	1 (100)	0
Transition	0	15 (100)	7 (47)	7 (47)	1 (7)	0	13 (100)	4 (31)	8 (62)	1 (8)	11 (85)	3 (23)	5 (38)	8 (62)	0	2 (100)	0	0	2 (100)	0	9 (100)	0	5 (56)	4 (44)	0	11 (100)	0	4 (36)	6 (66)	0
Roman	0	14 (100)	4 (29)	9 (64)	1 (7)	0	11 (100)	5 (45)	6 (55)	0	6 (55)	5 (45)	5 (45)	6 (55)	0	1 (100)	0	1 (100)	0	0	2 (67)	1 (33)	3 (100)	0	0	9 (90)	1 (10)	5 (50)	5 (50)	0

TABLE 6.4 Number and percentage of individuals observed with cranial and postcranial pathology given by dental age.

Dental Age	Pre-Roman				Transition				Roman			
	Postcranial		Cranial		Postcranial		Cranial		Postcranial		Cranial	
	Obs.	Affected (N/%)	Obs.	Affected (N/%)	Obs.	Affected (N/%)	Obs.	Affected (N/%)	Obs.	Affected (N/%)	Obs.	Affected (N/%)
34	-	-	-	-	-	-	-	-	1	1 (100)	1	1 (100)
38	2	1 (50)	2	2 (100)	-	-	-	-	1	1 (100)	1	1 (100)
40	2	1 (50)	3	2 (67)	2	2 (100)	2	2 (100)	1	0	1	1 (100)
42	1	1 (100)	1	1 (100)	-	-	-	-	-	-	-	-
43					1	1 (100)	1	1 (100)	-	-	-	-
46	4	0	4	2 (50)	8	7 (88)	10	10 (100)	5	3 (60)	5	4 (80)
52					1	1 (100)	2	2 (100)	5	1 (20)	4	2 (50)
58	1	0	2	1 (50)	1	0	1	1 (100)	1	1 (100)	1	1 (100)

TABLE 6.5 Results of chi-squared analysis (X^2) of pathological lesions by both time period and dental age for various skeletal elements. P results highlighted in bold are those found to be statistically significant.

	Frontal Bone			Parietal Bone			Occipital Bone			Humerus			Femur			Tibia		
	X^2	d.f.	*p*	X^2	d.f.	*p*	X^2	d.f.	*p*	X^2	d.f.	*p*	X^2	d.f.	*p*	X^2	d.f.	*p*
Time Period	3.395	2	0.183	4.682	2	0.96	4.682	2	0.96	2.288	2	0.218	11.348	2	**0.003**	11.348	2	**0.003**
Dental Age	9.199	7	0.149	10.478	7	0.53	7.974	7	0.179	16.846	7	0.223	13.015	7	**0.04**	10.761	7	0.103

Discussion

To understand results of this analysis and their implications, the interplay between a multitude of biological, pathological and contextual factors must be acknowledged. Fetal, perinatal and infant individuals are the most vulnerable to a variety of environmental onslaughts, and their growth is hence a reflection of their experience in the intrauterine environment (Winick *et al.* 1972; Bogin 2001; Lejarraga 2002; Oestreich 2008; Kuzawa & Quinn 2009; Kuzawa & Sweet 2009; Mays *et al.* 2009; Dancause *et al.* 2012). During the *in utero* period a constant interaction between the fetus, placenta and mother is taking place (Harding & Johnston 1995). Consequently, fetal development is heavily reliant on the maternal ability to provide adequate nutrition and protection *in utero* from a variety of harmful environmental stressors (Bateson *et al.* 2004; Barker *et al.* 2012; Said-Mohamed *et al.* 2018). In fact, both mother and placenta typically act as barriers and regulators from external stressors (Barker 2012), prioritising the wellbeing of the developing fetus, and thus ensuring its optimal growth and development (Gowland 2015; Said-Mohamed *et al.* 2018). However, maternal exposure to environmental health stresses results in the 'giving' potential of the mother being limited (Bateson *et al.* 2004). This may interrupt the interaction between fetus, placenta and mother, leading to a host of detrimental birth and life course outcomes (e.g. anaemia, maternal haemorrhage and IUGR) (Wu *et al.* 2012). IUGR can be a result of a limited nutrient and/or oxygen supply *in utero,* whilst spontaneous abortion or stillbirth can be a result of maternal illness, infection, chronic disease and extreme malnutrition (Goldenberg & Thompson 2003; Lewis 2007). Prenatal stress exposure has also been found to correlate with increased frailty and vulnerability of the individual in postnatal life, associated with the subsequent changes in immune function (McDade 2005; Boersma & Tamashiro 2015). The timing of this maternal exposure to stress has been considered to be vital in the long term implications for offspring health and growth. If subjected to stressors prenatally, the most precarious phase of life, the offspring is likely to exhibit physiological, and possible biological, alterations as a result (Bateson *et al.* 2004; Boersma & Tamashiro 2015). Consequently, a mother's disease status, particularly during pregnancy, plays a crucial role in determining the health of her child, both *in utero* and beyond (Gowland 2015).

Birth is one of the most stressful biological events in our life course and heralds a multitude of biological, physical and environmental changes (Bogin 2001; Lewis 2017a). The offspring is entering a world full of pathogens (McDade 2005; Lewis 2017a), and although remains

heavily reliant on the mother, there is a marked reduction in maternal ability to buffer against environmental conditions. Passive immunity from diseases and/or infections, as well as vital nutrients, are instead transferred via lactation (breast milk) (Bogin 2002; Eisenberg *et al.* 2017; Lewis 2017b). Consequently, multiple stressors could cause a detrimental postnatal environment: a reduced or non-existent food/nutrient supply via the mother, exposure to disease and infection, or other social and cultural factors. With postnatal nutrition being vital to healthy growth and development, death of the mother in childbirth, or inability to breast feed could both lead to detrimental growth and health outcomes for the child (Fujita *et al.* 2017).

Age-at-death assessment identified a clear difference between the demographic profiles when considering skeletal versus dental age estimation methodologies (See Table 3.). Dental age-at-death demonstrated a much broader age range, despite less individuals having dentition available for assessment. Skeletal methods for determining age-at-death have long been criticised. In fact, the linear regression method has been strongly critiqued, suggested to age individuals in a way which mimics the demographic make-up of the sample used to create the regression models (Gowland & Chamberlain 2002; Lewis & Gowland 2007). This method has previously been found to cluster individuals around the perinatal period and interpretations of deliberate disposal and killing (infanticide) have been supported from such findings (e.g. Mays 1993; Mays & Faerman 2001; Mays & Eyers 2011).

Many studies have considered the contextual implications of infant burials (e.g. Scott 1999, Pearce 2001, Gowland 2001, Moore 2009), particularly on rural sites, where high numbers of fetal, perinatal and infant burials tend to be uncovered (Hodson 2017). Due to the custom of burying infants within habitation buildings, under floors and in ditches, some authors have interpreted these findings as evidence of infanticide and deviant burial (Scott 1989; Moore 2009; e.g. Heneage Cocks 1921; Mays & Faerman 2001; Mays & Eyers 2011). Ethnographic and historical studies of infanticide have suggested that reasons for such putative killing include *'population control, illegitimacy, inability of the mother to care for the child, greed for power or money, superstition, congenital defects, and ritual sacrifice'* (Resnick 1970; also Rawson 2003; Bonsall 2013). Age-at-death estimations, using skeletal elements, have supported these interpretations, with samples found to show peaks in mortality around 40 GWA (Bonsall 2013; e.g. Smith & Kahila 1992; Mays 1993; Mays & Faerman 2001). However, Gowland and Chamberlain (2002) have demonstrated that assessment of age-at-

death using skeletal regression equations can strongly bias interpretations, with results mimicking the age-at-death profile of the reference sample. In addition, contemporary interpretations of infanticide have failed to consider the pathological data supporting evidence of disrupted health, which is clearly a major consequence of many perinatal, infant and fetal deaths.

Results from this study supports conclusions rejecting infanticide, demonstrating that age-at-death assessment from skeletal elements alone cannot be utilised to substantiate interpretations of infanticide. Indeed, for individuals within this study, dental age-at-death was found to range from 34 to 58 GWA. Conversely, long bone age estimates suggest no individuals assessed are over 44 GWA. As dental estimates are considered much more robust and accurate (Moorrees *et al.* 1963; Gustafson & Koch 1974; Bang 1989; Hoppa & Fitzgerald 1999; Bolaños *et al.* 2000; Humphrey 2000; Liversidge & Molleson 2004; Hillson 2005; AlQahtani *et al.* 2014; Satterlee Blake 2018), it seems pertinent that many more individuals are dentally suggested to be infantile (over 44 GWA) than perinatal or fetal. This suggests that many of the individuals analysed were not killed at birth, but instead likely survived for some days or weeks in the postnatal environment. Furthermore, detailed assessment of pathological changes has shown that individuals from all periods have changes consistent with a reduced intra- and extrauterine environment. Therefore, it is suggested that these individuals perished as a result of the harmful conditions to which they were exposed, rather than as a consequence of deliberate disposal.

Results of dental age assessment has clearly demonstrated that both pre- and postnatal individuals were at risk of growth and health disruption, signifying that both endogenous and exogenous factors were influential in affecting growth and health outcomes. Additionally, this suggests that maternal health and wellbeing was often reduced during pregnancy, as well as postnatally. Given that birth and the transition to this new environment signals the change from a highly regulated to a highly pathogen-loaded environment (McDade 2005; Lewis 2017a), it is not unexpected that more individuals have been found to be postnatal perinates and infants, than prenatal fetuses and perinates. Subsequent inability of the mother, through poor health, to transfer nutrients and immunity via lactation to the offspring would only predispose the infant to further environmental hazards. Individuals from all three time periods show both intrauterine and extrauterine growth disruption. However, the severity of postnatal growth disruption, with a peak in mortality seen at 46 GWA based on dental ages, may

indicate particularly adverse exogenous environmental conditions were predominantly accountable for many of the physiological changes identified (Lewis & Gowland 2007).

For those dentally aged to be 37 gestational weeks or less, it may be considered that they were premature (Tocheri *et al.* 2005). Thus, given the young age estimates of these individuals, it may be considered that these individuals represent those that were premature, still born or died shortly after birth. Furthermore, the limited growth disruption identified within the skeletal remains of these individuals may suggest that they did not survive long enough for observable physiological changes to occur. Individuals born prematurely also face significantly increased risks of mortality and disability (Tocheri *et al.* 2005).

To further counter the skewed analysis that sole consideration of skeletal age-at-death produces, this study considers skeletal and dental age estimates in unison for evidence of growth disruption. When individuals with both of these estimations were considered, the overall average difference between dental and skeletal age estimates was approximately six to seven gestational weeks (Table 4.). However, substantially greater differences were observed when individuals were categorised by time period or dental age. Roman individuals were found to show the greatest average difference between dental and long bone age estimates, followed by transitional individuals. Furthermore, those dentally aged to be 46, 52 and 58 GWA were found to have the greatest differences in dental and skeletal ages. From 46 GWA, skeletal age-at-death estimates become statistically significantly younger than dental development (Table 5.). Comparative assessment of diaphyseal length metrics to published data for fetal, perinatal and infant individuals (Fig. 5 and Fig. 6) corroborates this finding, showing that intrauterine growth appears relatively 'normal', where as extrauterine diaphyseal length measurements fall substantially below these reference data sets, suggestive of a sub-optimal postnatal environment. Furthermore, multiple individuals were shown to fall within or below the 10^{th} growth percentile, indicating severely disrupted growth. Consequently, adverse birth outcomes and subsequent reduction in health and growth status are likely for these individuals.

In total, eight individuals demonstrated a minimum of a 12-week difference between dental and skeletal age-at-death estimates. All of these individuals were dentally aged to be either 52 or 58 GWA. Of those individuals, four are Roman, two are transitional, with one pre-Roman and undated individual also. However, when error ranges for both dental and skeletal

methods are considered, only three individuals have age estimates and ranges which are found not to correspond. Individual 103 from Barton Court Farm shows the largest difference, with an age difference of 19.1 GWA between dental and femoral estimates (Fig. 4).

Of those found to be over 44 GWA (*N*=27), nine individuals are transitional and ten are Roman, whilst only 5 individuals are from the pre-Roman group and three are undated. Therefore, both the transitional and Roman periods have more individuals suggested to be infants and thus, survive into the postnatal period. However, when considered by dental age (Table 1.5) pre-Roman individuals have a similar frequency between perinatal and infant individuals, compared to the transitional and Roman periods which have more individuals aged to be infants. Therefore, it may be that the greater number of older individuals recovered from the transitional and Roman contexts is simply the product of the archaeological sample, with a bias in regards to both retrieval and identification, as well as the potential for selective funerary practices. However, given that the pre-Roman individuals actually have the highest frequency of individuals with dentition (Table 1.2), it would appear that these results are fairly representative of the whole pre-Roman sample, and may substantiate suggestions that postnatal mortality (after one month of age) was particularly high in transitional and Roman samples. This again suggests that postpartum exogenous factors were likely significantly impacting on the health and wellbeing of individuals categorised within the transitional and Roman time periods.

Findings of growth disruption show some relationship to evidence of pathological lesions, with a greater prevalence of both growth and health disruption in the transitional and Roman samples (Table 6.1 and Table 6.2). Statistical assessment of pathological lesions by time period shows that there is a statistically significant association between femoral and tibial pathology by time period (Table 6.5). Furthermore, given that both Transition and Roman individuals had much higher numbers and percentages of individuals with pathological lesions to these elements (Table 6.2) it suggests that there is an increase in health disruption within these periods. Despite small sample sizes, prevalence of pathological lesions also suggests that both pre- and postnatal health was adversely affected (Table. 5.4), with elements from all dental age categories showing pathological changes.

Non-adult pathological lesions are still a topic of much debate, particularly in fetal, perinatal and infantile remains (Ortner 2003). New bone formation, most typically identified and recorded as a pathological response, is also indicative of normal somatic growth where new bone formation is laid down both longitudinally and appositionally (Redfern 2007; Lewis 2017a). Discerning between normal and abnormal bone formation often relies on consideration of location, severity and thickness (e.g. Rana *et al.* 2009; Kwon *et al.* 2002). New bone formation is typically considered to be part of the initial physiological response to stress (Armelagos *et al.* 1991; Goodman & Martin 2002; DeWitte 2014; Larsen 2015) and rapidity of bone turnover in these individuals is considered to result in health insults being quickly reflected on their remains (Kwon *et al.* 2002; Lewis 2017a). However, the non-specificity of new bone formation, as a response to stress, means precise pathogenic/disease processes are rarely discernible (DeWitte 2014). The diagnosis of specific conditions in fetal, perinatal and infant skeletons is complex because they can only respond in a limited number of ways to a multitude of conditions (Ortner 2003; Gowland 2004; Redfern 2007). This means that multiple stressors or insults can lead to identical changes within the skeleton (Bush & Zvelebil 1991). Furthermore, if a disease is acute, or system specific (i.e. only affects soft tissue structures) no discernible changes will be observable to the skeleton, plus an individual may die before skeletal changes develop, or recover from insults experienced much earlier in life (Bush & Zvelebil 1991; Cardoso 2007). Individual responses to disease/infection also vary during pregnancy, and exposure to detrimental environmental insults is likely to result in a variety of outcomes for both mother and offspring (Goldenberg & Thompson 2003; Redfern 2007). Thus, the pathological lesions identified on skeletal remains *'…represent a small percentage of the total disease load in that population'* (Redfern 2003).

By considering lesions by severity, this investigation has attempted to discern that pathological changes can be definitively identified on fetal, perinatal and infant remains. In total, 71 skeletal elements were found to have a severity of 1, with 96 having a severity of 2 and only three elements having a severity of 3 (Table 6.1). For all time periods a severity 2 was found to be most common, followed by severity 1. Of the three skeletal elements with a pathology of severity 3, all were from either the Transition or Roman periods. Even if pathological changes scored to be of severity 1 are discounted - as elements within this category are most likely to be an assimilation of pathological and normal bone changes – pathological changes (of severity 2 or 3) are still highly prevalent throughout all of the time

periods. Furthermore, transitional individuals would still be those found to show the highest percentage of pathological changes, followed by Roman individuals.

Only two individuals, those with skeletal elements of severity 3, appear to show multiple episodes of exposure to stress and responses by the body and skeleton as a result (PID 31B and BCF 1256). This layering of bone formation substantiates the assumption that these lesions are pathological, as normal growth results in a single layer of bone formation (Shopfner 1966; Lewis 2007; Weston 2012). A repeated ability by these individuals to overcome, heal and survive stressful events and conditions is consequently insinuated. As transitional individuals from the 1st Century A.D. have the highest prevalence rates of pathologies, with the pre-Roman individuals the lowest (Table 6.1), it may indicate that those from the period of transition experienced the most 'stressful' pre- and postnatal life, but may equally have been the most resilient in overcoming these stressors. Unfortunately, neither individual (PID 31B and BCF 1256) has both dental and skeletal elements available for assessment, meaning consideration of growth disruption in relation to pathological changes cannot be afforded.

From pathological assessment, new bone formation is widely found within many of the individuals assessed (Table 6.2), but particularly for the transitional and Roman individuals. When considered by type of lesion, woven and lamellar NBF shows a distinct pattern. Within the cranium the majority of lesions appear to be lamellar, suggestive of some initial healing response, whereas the long bones typically have woven NBF suggesting it was an active lesion at time of death. This may indicate a prioritisation of the body for healing within the cranium. Body functions, including immune response, requires major energetic investment by the offspring (McDade 2005). Therefore, if experiencing a stressful intrauterine environment, the fetus does not have sufficient resources to be able to counter all of the stressful impacts. Therefore, prioritisation of skeletal and bodily structures, both in terms of growth and maintenance, occurs. This may explain why different skeletal elements are showing varying stress responses and levels of healing. Such prioritisation may also support why growth and health disruption is found to correlate, with both those in the Transition and Roman samples showing the severest disruption to both factors. High investment in immune activity, in response to pre- and postnatal stressors, is likely to result in a significant disruption in growth

as the offspring diverts resources to a particular function – e.g. immune response over skeletal growth (McDade 2005).

New bone formation has often been correlated with nutritional deficiency. Nutrients such as vitamin C, vitamin D, calcium and iron, are vital for optimal growth and health *in utero* as well as in the initial stages of life outside the womb (Lewis 2007); lack of these nutrients would further make the individuals more susceptible to a range of diseases and infections. The presence of both metaphyseal expansion and morphological changes, consistent with limb bowing, within the samples suggests that vitamin deficiencies are the likely cause of many of the pathological changes observed. Despite it being considered that rickets, and associated bowing deformities, are typically considered to be a disease of childhood, with bowing of the limbs due to weight-bearing and the commencement of crawling and/or walking (Holick 2005), bowing can be observed pre- and perinatally also (e.g. Innes *et al.* 2002). Space constraint prenatally is one of the possible causes of morphological changes. Bonneau and colleagues (2011) found changes in femoral torsion as a result on intrauterine pressure. Therefore, if deficiency was being experienced, and associated 'softening' of the long bones occurred due to changes poor mineralisation of the osteoid matrix, prenatal space restriction could result in the bowing of long bones. Intrauterine rickets has long been reported (e.g. Abbott 1901) as a result of maternal deficiency during pregnancy and subsequent investigations have found that maternal deficiency, as a result of cultural practice or inability to synthesise vitamin D, is still a contemporary concern (Anatoliotaki *et al.* 2003). Like with other deficiencies, the fetus relies on maternal stores of vitamin D for healthy bone growth (Innes *et al.* 2002; Anatoliotaki *et al.* 2003). Evidence of vitamin D deficiency within the individuals analysed suggests that some of the mothers were likely vitamin D deficient.

The high levels of NBF, as a result of subperiosteal haemorrhages, along with the changes at the metaphyses may also be indicative of vitamin C deficiency (Brickley & Ives 2006; Besbes *et al.* 2010). Vitamin C deficiency is a lack of ascorbic acid, which only be ingested from dietary sources (Brickley & Ives 2006). Lack of vitamin C results in weakened blood vessels which are easily ruptured, leading to extensive haemorrhaging (Aufderheride & Rodríguez-Martín 1998; Brickley & Ives 2006; DeWitte 2014). These changes result in NBF within the skeleton, as well as the thinning of the cortex and increased trabecular spacing (Brickley & Ives 2006). It is suggested that it can take several months for associated skeletal changes to become evident in vitamin C deficient individuals, though it is suspected that this

period is significantly reduced in non-adults whose bone turnover and remodelling is much more rapid (Brickley & Ives 2006). However, evidence of vitamin C deficiency within such young individuals is suggestive of extensive maternal malnutrition as the offspring would need to be deficient for multiple months for lesions to be present, suggesting a lack of access to vitamin C prenatally. Consequently, maternal health and the ability of the mother to continue to provide adequate buffering against dietary, health and environmental stressors, can greatly affect the experience of the child, and ultimately its survival.

The Roman conquest of Britain in 43 A. D. fundamentally changed the cultural, economic and demographic landscape of Britain (Redfern *et al.* 2012; Rohnbogner & Lewis 2017), although Roman influence was already present prior to the invasion. The Iron Age population at Owslebury was one that manoeuvred in a complex network and landscape of communities who traded with and were influenced by Roman culture (Collis 1994), whilst those individuals living at Piddington and Barton Court Farm, both sites where Roman villas have been subsequently excavated (Miles 1986; Friendship-Taylor & Friendship-Taylor 2012), were likely to have been strongly influenced by Roman culture, social practices and policies. Indeed, it cannot be known for sure, without further DNA and isotopic analysis, whether the individuals recovered from these sites represent those of native (local) or Roman (non-local) individuals. However, regardless of who they were, they were likely conceived and/or born into a world that was strongly regulated by Roman practices. This can be determined from the imports of pottery, foodstuffs, and even marble, which demonstrate that these sites certainly operated within the complex social and economic network of the Roman Empire. However, the extent of this Roman influence is unknown, yet despite this, the Roman way of life clearly prevailed at these sites. Thus, it must be supposed that adoption of Roman practices influenced and impacted these communities.

Previous studies have highlighted that the Roman invasion of Britain brought a marked reduction in the health of the population in general (Molleson 1989; 1992; Redfern 2008; Redfern & DeWitte 2011; Griffin *et al.* 2011, 545; Redfern *et al.* 2012; Pitts & Griffin 2012), with an increase in the prevalence of infectious disease, joint disease, respiratory disease, metabolic disease, dental disease, and general indicators stress (Roberts & Cox 2003; Lewis 2010). This is attributed to various factors, including the significant population growth, the introduction of new diseases and pathogens by migrants and the army, and new dietary profiles (Redfern 2003; Redfern *et al.* 2012). The growing trade networks and increased

migration to Britain saw new pathogens being introduced to non-immune, 'local' populations (Roberts & Cox 2003). This increase in population also denoted a period of social reorganisation and intensified social stratification (Scobie 1986; Molleson 1992; Roberts & Cox 2003; Pitts & Griffin 2012). This social stratification consequently led to differing access to dietary and nutritional resources, as well as medical care and living conditions contributing to the observed increase in metabolic disease in Roman-Britain (Scobie 1986; Roberts & Cox 2003). Furthermore, social reorganisation and increased social stratification is known to affect population health (Babones 2008), increasing psychosocial stress, which impacts on immunological function (Roberts & Cox 2003). Consequently, it is particularly interesting that the Transition and Roman period groups assessed in this study show greater evidence of health and growth disruption, as it is possible that these changes reflect an increasingly stratified society.

Rural economies were reliant on the market economy and the continual supply of foods. Withholding of resources, or differential access to food products based on social position, was also likely to affect susceptibility to stress and overall health status (Klaus 2014; Rohnbogner & Lewis 2017). Clear social divisions are suggested to have been particularly prevalent within rural settlements, suggesting a very limited population mobility (Pitts & Griffin 2012). Consequently, as social inequality is known to increase susceptibility to poor health and disease (Schell 1997; Griffin *et al.* 2011; Pitts & Griffin 2012), increased social inequality experienced by the transitional and Roman individuals is reflected in their growth and health profiles. As a consequence, it maybe that higher prevalence rates of these disruptions signal that the individuals are those of lower social status. However, growth and health disruption may also be a product of the social and cultural disturbance and instability experienced, where individuals were subjected to *'…unprecedentedly complex world of market forces, new religions, a standing army, and political upheavals'* (Scott 1989).

Despite many of the existing studies focussing on major and minor urban settlements (Rohnbogner & Lewis 2017; e.g. Gowland & Redfern 2010), the pattern of growth and health disruption identified at these rural sites is not markedly different. Though it may be anticipated that rural communities experienced a healthier lifestyle, in fact lack of health care, poor ventilation, limited access to a variety of dietary sources, and close contact with animals all brought increased risks of disease and infection (Rohnbogner & Lewis 2017). An investigation by Pitts and Griffin (2012) found that individuals from rural settlements tended

to have poorer health than individuals from urban settlements, having increased rates of abscesses, enamel hypoplasias, joint disease, and neoplastic disease. This suggests that those living at rural settlements were facing equally compromised living conditions as those inhabiting urban and nucleated settlements. Thus, it appears that many rural individuals likely experienced growth and health disruption as a result.

Behaviour towards and treatment of an infant once born can vary, and social status and culture can dictate the level of care, feeding and health the child has access to (Redfern 2003; Gowland 2004). Changes in maternal health and social and cultural practices associated with the raising and care of infants might also have been influenced and altered as a result of the incoming Roman ideas and practices (Lewis 2010; Rohnbogner & Lewis 2017). Furthermore, the status of the mothers must also be questioned. Servants and slaves were an integral part of the social organization of Iron Age and Romano-British communities, particularly on agricultural and villa estates (Webster 2005; Redfern 2007). Webster (2005) suggests that positions of servitude and slavery were common amongst these communities and, even though little archaeological evidence has been found to substantiate this, it is commonly accepted that there was a large network in the trading of people and slaves within Britain before the Roman conquest. Exposure to, and treatment for disease is also regulated by social status, gender and age, with access to adequate nutrition and healthcare controlled by these culturally contingent factors (Gowland 2004). Consequently, an individual's susceptibility and immune response to disease was reliant on these factors (Gowland 2004). The high levels of pathological lesions identified in the transitional and Roman period individuals thus, might not only be a consequence of social and cultural change, but indicative that the mothers of these infants were those of the lower, lesser status individuals living and working at these sites.

Furthermore, cultural and social practices surrounding pregnancy, birth and wet nursing have also been documented in Roman Britain (e.g. Redfern *et al.* 2012). Soranus recommended that new born offspring were not to be breastfed for the first three weeks of life. It was considered that colostrum was harmful for the infants, and consequently, withholding of this important nutritious and immunological substance from new born infants may have greatly affected survival chances, increasing susceptibility to infection (Edmond *et al.* 2006; Bonsall 2013). In addition, the presence of wet-nurses within Roman Britain may further contribute to

high infant mortality rates, with offspring once again failing to receive adequate nutritional and immunological buffering via breast milk.

Finally, it must be considered that infant mortality, although not any easier to accept, was a consequence of life in ancient communities (Weidemann 1989; Rawson 1986). As Weidemann states '*In Roman times, being a centenarian was remarkable, dying as an infant was not'* (1989). Thus, the abundance of fetal, perinatal and infantile remains excavated and recovered from rural sites may not be unanticipated, and simply reflect the harsh reality of high infant mortality rates. Chamberlain (1997) suggests that up to 50% of individuals born would not reach maturity, highlighting the high mortality rates likely to be reflected within archaeological samples. In addition, many scholars have cited cultural, social and religious reasons as to why infants were buried amongst settlements (Redfern *et al.* 2012; e.g. Gowland & Chamberlain 2002; Moore 2009; Gowland *et al.* 2014; Millet & Gowland 2015). Moore (2009) suggests that associations between fetal, perinatal and infant burials and inhabitation structures as well as a variety of other domestic and agricultural buildings, indicates that burial was '…*not the random disposal of the unwanted or marginalised, but the result of careful choices*'. Therefore, the precarious nature of these early stages of life were well known to these communities and as such, many individuals were not named until a particular age or point of survivorship was achieved (Moore 2009). Consequently, those assessed within this study represent those of the non-survivors, who ultimately succumbed to the deleterious conditions they faced.

Conclusion

This study has provided an important contribution to our understanding of the Iron Age to Roman transition and its impact on growth and health in the youngest members of past societies in a rural context. This synthesis of growth and health assessment provides a robust picture of disruption throughout these populations and time periods, and supports previous studies suggesting that health and well-being deteriorated after the Roman conquest, extending this data into the fetal, perinatal and infant period.

This paper also highlights that dental assessment of age-at-death must be considered where possible for all fetal, perinatal and infant individuals, as skeletal age-at-death has been found to mimic reference data. Thus, interpretations surrounding infant death may be biased when only skeletal elements are assessed for age, and to avoid this, comparison of dental and

skeletal ages should be sought. Furthermore, by assessing individuals in this way it is possible to explore growth disruption on an individual level. This avoids the need for Bayesian statistics, as previous studies have utilised (Gowland & Chamberlain 2002), and means direct comparisons between growth and health data, as well as between individuals can be made. This has enabled this study to investigate temporal differences between growth and health disruption experienced by individuals.

Assessment of pathological lesions has identified that health disruption was prevalent in all three time periods. Pathological lesions were found to increase during the transitional period and remain elevated in the Roman period individuals. Furthermore, individuals of all chronological ages were found to show evidence of health disruption, despite sample sizes being small. This suggests that both intrinsic and extrinsic stressors were impacting the wellbeing of these individuals. This indicates that both the intrauterine and extrauterine environment was compromised, suggesting that maternal health also decreased during the transitional and Roman periods.

This research aimed to consider the contextual implications of this growth and health disruption, but this is a complex and intricate narrative reliant on a multitude of factors including social status, access to dietary resources and health care, as well as the social, cultural and political spheres in which mother and child lived. Determining the key factors in causing this disruption is almost impossible, but it is likely that there is a consistent interplay between many factors, with not one, but multiple stressors, causing the growth and health disruption identified. For all sites, it can be suggested that individuals experienced poor health, though the number of individuals and severity of health disruption experienced is seen to increase with the simultaneous increase in 'Romanisation'. Therefore, although growth and health was clearly disrupted in many individuals from all of these populations, a distinct pattern emerges suggesting pre- and postnatal life was particularly precarious from the 1st Century onwards.

References

Abu-Saad, K. and Fraser, D. 2010; Maternal nutrition and birth outcomes. *Epidemiological Review,* Vol. 32: 5-25.

AlQahtani, S. J. Hector, M. P. and Liversidge, H. M. 2010; The London Atlas of Human Tooth Development and Eruption. *American Journal of Physical Anthropology,* Vol. 142: 481-490.

AlQahtani, S. J. Hector, M. P. and Liversidge, H. M. 2014; Accuracy of Dental Age Estimation Charts: Schour and Massler, Ubelaker, and the London Atlas. *American Journal of Physical Anthropology,* Vol. 154: 70-78.

Anatoliotaki, M., Tsilimigaki, A., Tsekoura, T., Schinaki, A., Stefanaki, S. and Nikolaidou, P. 2003; Congenital rickets due to maternal vitamin D deficiency in a sunny island of Greece. *Acta Paediatrica,* Vol. 92: 389-391.

Armelagos, G. J. and Goodman, A. H. 1991; The concept of stress and its relevance to studies of adaptation in prehistoric populations. *Collegium Antropologicum,* Vol. 15: 45-58.

Armelagos, G. J., Goodman, A. H., Harper, K. N. and Blakey, M. L. 2009; Enamel Hypoplasia and Early Mortality: Bioarchaeological Support for the Barker Hypothesis. *Evolutionary Anthropology,* Vol. 18: 261-271.

Aufderheide, A. and Rodríguez-Martín, C. 1998; *The Cambridge Encyclopedia of Human Paleopathology*. Cambridge: Cambridge University Press.

Babones, S. J. 2008; Income inequality and population health: Correlation and causality. *Social Science and Medicine,* Vol. 66: 1614-1626.

Bang, G. 1989; Age changes in teeth; developmental and regressive. *Age Markers in the Human Skeleton,* Vol. 1: 211-235.

Barker, D.J. 1997; Maternal nutrition, fetal nutrition, and disease in later life. *Nutrition,* Vol. 13:807–13.

Barker, D. J. 2012; Developmental origins of Chronic Disease. *Public Health,* Vol. 126: 185-189.

Barker, D. J. P., Eriksson, J. G., Forsén, T. and Osmond, C. 2002; Fetal origins of adult disease: strength of effects and biological basis. *International Journal of Epidemiology,* Vol. 31: 1235-1239.

Barker, D. J. P., Lampl, M., Roseboom, T. and Winder, N. 2012; Resource allocation in utero and health in later life. *Placenta,* Vol. 33: 30-34.

Barker, D. and Osmond, C. 1986; Infant mortality, childhood nutrition, and ischaemic heart disease in England and Wales. *Lancet,* Vol. 8489: 1077–1081.

Bateson, P., Barker, D., Clutton-Brock, T., Deb, D., D'Udine, B., Foley, R. A., Gluckman, P., Godfrey, K., Kirkwood, T., Mirazón Lahr, M., McNamara, J., Metcalfe, N. B., Monaghan, P., Spencer, H. G. and Sultan, S. E. 2004; Developmental plasticity and human health. *Nature,* Vol. 430: 419-421.

Beaudrap, P., Turyakira, E., White, L. J., Nabasumba, C., Tumwebaze, B., Muehlenbachs, A., Guérin, P., Boum, Y., McGready, R. and Piola, P. 2013; Impact of malaria during pregnancy on pregnancy outcomes in a Ugandan prospective cohort with intensive malaria screening and prompt treatment. *Malaria Journal,* Vol. 12 (1): 139.

Besbes, L. G., Hadded, S., Meriem, C. B., Golli, M., Najjar, M. F. and Guediche, M. N. 2010; Infantile Scurvy: Two Case Reports. *International Journal of Pediatrics,* Article ID 717518: 1-4.

Boersma, G. J. and Tamashiro, K. L. 2015; Individual differences in the effects of prenatal stress exposure in rodents. *Neurobiology of Stress,* Vol. 1: 100-108.

Bogin, B. 2001; *The Growth of Humanity*. New York: Wiley-Liss.

Bogin, B. 2002; *The Evolution of Human Growth.* In N. Cameron & B. Bogin (Eds.) *Human Growth and Development.* London: Elsevier: 287-324.

Bogin, B. & Baker, J. 2012; Low Birth Weight Does Not Predict the Ontogeny of Relative Leg Length of Infants and Children: An Allometric Analysis of the NHANES III Sample. *American Journal of Physical Anthropology,* Vol. 148: 487-494.

Bogin, B. & Loucky, J. 1997; Plasticity, Political Economy, and Physical Growth Status of Guatemala Children Living in the United States. *American Journal of Physical Anthropology,* Vol.102: 17-32.

Bogin, B. & Rios, L. 2003; Rapid morphological change in living humans; implications for modern human origins. *Comparative Biochemistry and Physiology,* Part A, Vol. 136: 71-84.

Bolaños, M. V., Manrique, M. C., Bolaños, M. J. and Briones, M. T. 2000; Approaches to chronological age assessment based on dental calcification. *Forensic Science International,* Vol. 110: 97-106.

Bonneau, N., Simonis, C., Seringe, R. and Tardieu, C. 2011; Study of Femoral Torsion During Prenatal Growth: Interpretations Associated with the Effects of Intrauterine Pressure. *American Journal of Physical Anthropology,* Vol. 145: 438-445.

Bonsall, L. 2013; Infanticide in Roman Britain: A Critical Review of the Osteological Evidence. *Childhood in the Past,* Vol. 6 (2): 73-88.

Brickley, M. and Ives, R. 2006; Skeletal Manifestations of infantile Scurvy. *American Journal of Physical Anthropology,* Vol. 129: 163-172.

Bush, H. and Zvelebil, M. 1991; Pathology and health in past societies: an introduction. In H. Bush and M. Zvelebil (Eds.) *Health in Past Societies: Biocultural interpretations of human skeletal remains in archaeological contexts* (BAR International Series 567). Oxford: BAR Publishing: 3-9.

Cameron, N. 2002; The Human Growth Curve, Canalization and Catch-Up Growth. In N. Cameron & B. Bogin (Eds.) *Human Growth and Development.* London: Elsevier: 1-22.

Cameron, N. and Demerath, E. W. 2002; Critical Periods in Human Growth and Their Relationship to Diseases of Aging. *Yearbook of Physical Anthropology,* Vol. 45: 159-184.

Cardoso, H. F. V. 2007; Environmental Effects on Skeletal Versus Dental Development: Using a Documented Subadult Skeletal Sample to Test a Basic Assumption in Human Osteological Research. *American Journal of Physical Anthropology,* Vol. 132: 223-233.

Chamberlain, A. 1997; Missing stages of life – towards the perception of children in archaeology. In J. Moore and E. Scott (Eds.) *invisible People and Processes: writing gender and childhood into European archaeology.* London: Leicester university Press: 248-250.

Chiswick, M. L. 1985; Intrauterine growth retardation. *British Medical Journal,* Vol. 291: 845-848.

Clukay, C. J., Hughes, D. A., Rodney, N. C., Kertes, D. A. and Mulligan, C. J. 2018; DNA methylation complex genes in relation to stress and genome-wide methylation in mother-newborn dyads. *American Journal of Physical Anthropology,* Vol. 165 (1): 173-182.

Collis, J. R. 1977; Owslebury (Hants) and the problem of burials on rural settlements. In R. Reece (Ed.) *Burial in the Roman World,* CBA Research Report, No. 22. London: The Council for British Archaeology: 26-34.

Collis, J. R. 1994; An Iron Age and Roman Cemetery at Owslebury, Hampshire. In A. P. Fitzpatrick and E. L. Morris (Eds.) *The Iron Age in Wessex: Recent Work.* Salisbury: Trust for Wessex Archaeology Ltd: 106-108.

Cussons-Read, M. E., Lobel, M., Carey, J. C., Kreither, M. O., D'Anna, K., Argys, L., Ross, R. G., Brandt, C. and Cole, S. 2012; The occurrence of preterm delivery is linked to pregnancy-specific distress and elevated inflammatory markers across gestation. *Brain, Behaviour and Immunity,* Vol. 26 (4): 650-659.

Dancause, K. N., Cao, X. J., Veru, F., Xu, S., Long, H., Yu, C., Laplante, D. P., Walker, C. D. and King, S. 2012; Brief Communication: Prenatal and Early Postnatal Stress Exposure Influences Long Bone Length in Adult Rat Offspring. *American Journal of Physical Anthropology,* Vol. 149: 307-311.

DeWitte, S. N. 2014; Differential survival among individuals with active and healed periosteal new bone formation. *International Journal of Paleopathology,* Vol. 7: 38-44.

Dodrill, P. 2016; Typical Feeding and Swallowing Development in Infants and Children. In Groher, M. E. and Crary, M. A. (Eds.) *Dysphagia: Clinical Management in Adults and Children.* Missouri: Elsevier: 253-269.

Edmond, K. M., Zandoh, C., Quigley, M. A., Amenga-Etego, A., Owusu-Agyei, S. and Kirkwood, B. R. 2006; Delayed Breastfeeding Initiation Increases Risk of Neonatal Mortality. *Pediatrics,* Vol. 117 (3): 380-386.

Eisenberg, D. T. A., Borja, J. B., Hayes, M. G. and Kuzawa, C. W. 2017; Early life infection, but not breastfeeding, predicts adult blood telomere lengths in the Philippines. *American Journal of Human Biology,* Vol. 29 (Early View): 1-11.

Farewell, C. V., Thayer, Z. M., Tracer, D. P. and Morton, S. 2018; Prenatal stress exposure and early childhood BMI: Exploring association in a New Zealand context. *American Journal of Human Biology,* [Early View].

Fazekas, I. G. and Kósa, F. 1978; *Forensic Foetal Osteology.* Budapest: Academic Press.

Fell, D. B., Savitz, D. A., Kramer, M. S., Gessner, B. D., Katz, M. A., Knight, M., Luteijn, J. M., Marshall, H., Bhat, N., Gravett, M. G., Skidmore, B. and Ortiz, J. R. 2016; Maternal influenza and birth outcomes: systematic review of comparative studies. *International Journal of Obstetrics and Gynaecology,* Vol. 124: 48-59.

Finlay, N. 2013; Archaeologies of the beginnings of life. *World Archaeology,* Vol. 45 (2): 207-214.

Friendship-Taylor, R. M. and Friendship-Taylor, D. E. 2012; *Iron Age and Roman Piddington: 10th Interim Report and Phase Descriptions of the Late Iron Age Settlements, Military Phase, Roman Villa Complex and Early Saxon Phases at Piddington, Northants.* The Upper Nene Archaeological Society 2103.

Fujita, M., Lo, Y. J. and Brindle, E. 2017; Nutritional, inflammatory, and ecological correlates of maternal retinol allocation to breast milk in agro-pastoral Ariaal communities of northern Kenya. *American Journal of Human Biology,* Vol. 29 (Early View): 1-14.

Garn, S. M. Lewis, A. B. and Polacheck, D. L. 1960; Interrelations in dental development. I. Interrelationships within the dentition. *Journal of Dental Research,* Vol. 39: 1049-1055.

Gluckman, P. D. and Hanson, M. A. 2006; *The Developmental Origins of Health and Disease*. Cambridge: Cambridge University Press.

Goldenberg, R. L & Thompson, C. 2003; The infectious origins of stillbirth. *American Journal of Obstetric Gynecology*, Vol. 189, No. 3: 861-873.

Goodman, A. H. and Armelagos, G. J. 1988; Childhood Stress and Decreased Longevity in a Prehistoric Population. *American Anthropologist,* Vol. 90 (4): 936-944.

Goodman, A. H. & Armelagos, G. J. 1989; Infant and Childhood Morbidity and Mortality Risks in Archaeological Populations. *World Archaeology*, Vol. 21, No. 2: 225-243.

Goodman, A.H. Brooke Thomas, R. Swedlund, A. & Armelagos, G.J. 1988; Biocultural perspectives on stress in prehistoric, historical, and contemporary population research. *Yearbook of Physical Anthropology* 31: 169-202.

Goodman, A.H. and Martin, D. L. 2002; Reconstructing health profiles from skeletal remains. In R. H. Steckel and J.C. Rose (Eds.) *The Backbone of History: Health and Nutrition in the Western Hemisphere.* Cambridge: Cambridge University Press:11-60.

Gowland, R. L. 2001; Playing Dead: Implications of mortuary evidence for the social construction of childhood in Roman Britain. In D. Davis, A. Gardner and K. Lockyear (Eds.) *TRAC 2000.* Oxford: Oxbow: 152-168.

Gowland, R.L. 2004; The social identity of health in late Roman Britain. In B. Croxford, H. Eckardt, J. Meade and J. Weekes (Eds.) *TRAC 2003.* Oxford: Oxbox: pp135-146.

Gowland, R. L. 2015; Entangled Lives: Implications of the Developmental Origins of Health and Disease Hypothesis for Bioarchaeology and the Life Course. *American Journal of Physical Anthropology,* Vol. 158, No. 4: 530-540.

Gowland, R. L. and Chamberlain, A. T. 2002; A Bayesian Approach to Ageing Perinatal Skeletal Material from Archaeological Sites: Implications for the Evidence for Infanticide in Roman-Britain. *Journal of Archaeological Science,* Vol. 79: 677-685.

Gowland, R. L., Chamberlain, A. T., Redfern, R. C. 2014. On the brink of being: re-evaluating infant death and infanticide in Roman Britain, in M. Carroll and E-J. Graham (Eds.), *Infant Health and Death in Roman Italy and Beyond*, Journal of Roman Archaeology Supplementary Series 98, 69-88.

Gowland, R. L. and Redfern, R. C. 2010; Childhood Health in the Roman World: Perspectives from the Centre and Margin of the Empire. *Childhood in the Past,* Vol. 3: 15-42.

Griffin, R. Pitts, M. Smith, R. and Brook, A. 2011; Inequality at Late Roman Baldock, UK. The Impact of Social Factors on Health and Diet. *Journal of Anthropological Research,* Vol. 67, No.4: 533-556

Gustafson, G. and Koch, G. 1974; Age estimation up to 16 years of age based on dental development. *Odontologisk Revy,* Vol.25 (3): 297-306.

Halcrow, S. E. and Tayles, N. 2008; The Bioarchaeological Investigation of Childhood and Social Age: Problems and Prospects. *Journal of Archaeological Method and Theory,* Vol. 15: 190-215.

Halcrow, S. E., Tayles, N. and Elliot, G. E. 2018; The Bioarchaeology of Fetuses. In S. Han, T. K. Betsinger, and A. B. Scott (Eds.) *The Fetus: Biology, Culture, and Society.* New York: Berghahn Books: 83-111.

Halcrow, S. E., Tayles, N., Inglis, R. and Higham, C. 2012; Newborn twins from prehistoric mainland Southeast Asia: birth, death and personhood. *Antiquity,* Vol. 86: 838-852.

Hammer, O., Harper, D. A. T. and Ryan, P. D. 2001; PAST Paleontological Statistics Software Package for Education and Data Analysis. [Online] [Accessed August 2016] Available from: http://palaeo-electronica.org/2001_1/past/issue1_01.htm

Harding, J. E. and Johnston, B. M. 1995; Nutrition and Fetal Growth. *Reproduction, Fertility and Development,* Vol. 7, No. 3: 539-548.

Heneage Cocks, A. 1921; A Romano-British homestead in the Hambleden Valley, Buckinghamshire. *Archaeologia* 71:141–66.

Hill, J. D. 1995; The Pre-Roman Iron Age in Britain and Ireland (ca. 800 B.C. to A.D. 100): An Overview. *Journal of World Prehistory,* Vol. 9 (1): 47-97.

Hillson, S. W. 2005; *Teeth* (Second Edition). Cambridge: Cambridge University Press.

Hodson, C. M. 2017; Between Roundhouse and Villa: Assessing Perinatal and Infant Burials from Piddington, Northamptonshire. *Britannia,* Vol. 48: 195-219.

Hoffman, M. C. 2016; Stress, the Placenta, and Fetal Programming of Behaviour: Genes' First Encounter with the Environment. *American Journal of Psychiatry,* Vol. 173 (7): 655-657.

Holick, M. F. 2005; The Vitamin D Epidemic and its Health Consequences. *The Journal of Nutrition,* Supplement: 2739S-2748S.

Hoppa, R. D. 1992; Evaluating Human Skeletal Growth: An Anglo-Saxon Example. *International Journal of Osteoarchaeology,* Vol. 2: 275-288.

Hoppa, R. D. and Fitzgerald, C. M. 1999; From head to toe: integrating studies from bones and teeth in biological anthropology. In. D. Hoppa and C. M. Fitzgerald (Eds.) *Human growth in the past: studies from bones and teeth*. Cambridge: Cambridge University Press: 1-32.

Humphrey, L. 2000; Growth Studies of Past Population: An Overview and an Example. In M. Cox and S. Mays (Eds.) *Human Osteology in Archaeology and Forensic Science*. London: Greenwich Medical Media Ltd: 23-38.

Innes, A. M., Seshia, M. M., Prasad, C., Al Saif, S., Friesen, F. R., Chudley, A. E., Reed, M., Dilling, L. A., Haworth, J. C. and Greenberg, C. R. 2002; Congenital rickets caused by maternal vitamin D deficiency. *Paediatric Child Health,* Vol. 7 (7): 455-458.

Kiserud, T., Piaggio, G., Carroli, G., Widmer, M., Carvalho, J., Neerup Jensen, L., Giordano, D., Guilherme Cecatti, J., Abdel Aleem, H., Talegawkar, S. A., Benachi, A., Diemert, A., Tshefu Kitoto, A., Thinkhamrop, J., Lumbiganon, P., Tabor, A., Kriplani, A., Gonzalez Perez, R., Hecher, M. A., Gülmezoglu, A. M. and Platt, L. D. 2017; The World Health Organization Fetal Growth Charts: A Multinational Longitudinal Study of Ultrasound Biometric Measurements and Estimated Fetal Weight. *PLoS Medicine,* Vol. 14 (1): e1002220 (1-36).

Klaus, H. D. 2014; Frontiers in the Bioarchaeology of Stress and Disease: Cross-Disciplinary Perspectives From Pathophysiology, Human Biology, and Epidemiology. *American Journal of Physical Anthropology,* Vol. 155: 294-308.

Kuzawa, C. W. and Quinn, E. A. 2009; Developmental Origins of Adult Function and Health: Evolutionary Hypotheses. *Annual Review of Anthropology,* Vol. 38: 131-147.

Kuzawa, C. W. and Sweet, E. 2009: Epigenetics and the embodiment of race: developmental origins of US racial disparities in cardiovascular health. *American Journal of Human Biology,* Vol. 21: 2–15.

Kwon, D. S., Spevak, M. R., Fletcher, K. and Kleinman, P. K. 2002; Physiologic Subperiosteal New Bone Formation: Prevalence, Distribution, and Thickness in Neonates and Infants. *American Journal of Radiology,* Vol. 179: 985-988.

Larsen, C. S. 2015; *Bioarchaeology: Interpreting Behaviour from the Human Skeleton* (Second Edition). Cambridge: Cambridge University Press.

Leach, S., Lewis, M., Chenery, C., Müldner, G. and Eckardt, H. 2009; Migration and Diversity in Roman Britain: A Multidisciplinary Approach to the Identification of Immigrants in Roman York, England. *American Journal of Physical Anthropology,* Vol. 140: 546-561.

Lejarraga, H. 2002; Growth in Infancy and Childhood: A Pediatric Approach. In N. Cameron & B. Bogin (Eds.) *Human Growth and Development.* London: Elsevier: 23-56.

Lewis M. E. 2000; Non-adult palaeopathology: current status and future potential. In M. Cox and S. Mays (Eds.) *Human Osteology in Archaeology and Forensic Science*. London: Greenwich Medical Media Ltd: 39–57.

Lewis, M. E. 2002a; The impact of industrialisation: comparative study of child health in four sites from medieval and post-medieval England (AD 850–1859). *American Journal of Physical Anthropology,* Vol. 119: 211–223.

Lewis, M. E. 2002b; *Urbanisation and Child Health in Medieval and Post-Medieval England.* British Archaeological Reports British Series 229. Oxford: Archaeopress.

Lewis, M. E. 2007; *The Bioarchaeology of Children: Perspectives from Biological and Forensic Anthropology.* Cambridge: Cambridge University Press.

Lewis, M. E. 2010; Life and Death in a Civitas Capital: Metabolic Disease and Trauma in the Children from Late Roman Dorchester, Dorset. *American Journal of Physical Anthropology,* Vol. 142: 405-416.

Lewis, M. E. 2017a; *Paleopathology of Children: Identification of Pathological Conditions in the Human Skeletal Remains of Non-Adults.* London: Academic Press.

Lewis, M. E. 2017b; Childcare in the Past: The Contribution of Palaeopathology. In L. Powell, W. Southwell-Wright, and R. Gowland (Eds.) *Care in the Past: Archaeological and Interdisciplinary Perspectives.* Oxford: Oxbow Books: 23-37.

Lewis, M. E. 2018; Fetal Paleopathology: An Impossible Discipline? In S. Han, T. K. Betsinger, and A. B. Scott (Eds.) *The Fetus: Biology, Culture, and Society.* New York: Berghahn Books: 112-131.

Lewis, M. E. and Gowland, R. L. 2007; Brief and Precarious Lives: Infant Mortality in Contrasting Sites from Medieval and Post-Medieval England (AD 850-1859). *American Journal of Physical Anthropology,* Vol. 134: 117-129.

Lewis, M. E. and Roberts, C. 1997; Growing Pains: the interpretation of Stress Indicators. *International Journal of Osteoarchaeology,* Vol. 7: 581-586.

Liversidge, H. M. and Molleson, T. 2004; Variation in Crown and Root Formation and Eruption of Human Deciduous Teeth. *American Journal of Physical Anthropology,* Vol. 123: 172-180.

Maresh, M. M. 1970; Measurements from roentgenograms. In R. W. McCammon (Ed.) *Human Growth and Development.* Springfield: C. C. Thomas: 157-200.

Mattingly, D. 2006; *An imperial possession: Britain in the Roman Empire*. London: Penguin Books Ltd.

Mays, S. 1993; Infanticide in Roman Britain. *Antiquity* 67: 883–8.

Mays, S. and Eyers, J. 2011. Perinatal infant death at the Roman villa site at Hambleden, Buckinghamshire, England. *Journal of Archaeological Science* 38, 1931-38.

Mays, S. & Faerman, M. 2001; Sex identification of some putative infanticide victims from Roman Britain using ancient DNA. *Journal of Archaeological Science,* Vol. 28: 555-559.

Mays, S., Ives, R. and Brickley, M. 2009; The Effects of Socioeconomic Status on Endochondral and Appositional Bone Growth, and Acquisition of Cortical Bone in Children from 19th Century Birmingham, England. *American Journal of Physical Anthropology,* Vol. 140: 410-416.

McDade, T. W. 2005; Life History, Maintenance, and the Early Origins of immune Function. *American Journal of Human Biology,* Vol. 17: 81-94.

Melby, M. K., Yamada, G. and Surkan, P. J. 2016; Inadequate Gestational Weight Gain Increases Risk of Small-for-Gestational-Age Term Births in Girls in Japan: A Population-Based Cohort Study. *American Journal of Human Biology,* Vol. 28: 714-720.

Miles, D. 1986; *Archaeology at Barton Court Farm, Abingdon, Oxfordshire.* Oxford Archaeological Unit Report 3, CBA Research Report 50. Oxford: Oxford Archaeological Unit.

Miller, Z. E. S. 2010; The Infant Burials from Piddington. In R. M. Friendship-Taylor & D. E. Friendship-Taylor (Eds.), *Iron Age and Roman Piddington: Iron Age, Roman and Anglo Saxon Human Burials and Recent Research 1979-2010.* The Upper Nene Archaeological Society 2010, Fascicule 7: 1-31.

Millet, M & Gowland, R. 2015; Infant and Child Burial Rites in Roman Britain: A Study from East Yorkshire. *Britannia,* Vol. 46: 171-189.

Molleson, T. 1989; Social implications of mortality patterns of juveniles from Poundbury Camp, Romano-British cemetery. *Anthropologischer Anzeiger,* Vol. 47 (1): 27-38.

Molleson, T. 1992; The anthropological evidence for change through Romanisation of the Poundbury population. *Anthropologischer Anzeiger,* Vol. 50 (3): 179-189.

Moore, A. 2009. Hearth and home: the burial of infants within Romano-British domestic contexts. *Childhood in the Past,* Vol. 2: 33-54.

Moorrees, C. F. A. Fanning, E. A. & Hunt, E. E. 1963; Formation and Resorption of Three Deciduous Teeth in Children. *American Journal of Physical Anthropology,* Vol. 21: 205-213.

Nystrom, P. and Mahoney Swales, D. [No Date]; *Report on the Immature Inhumations at Owslebury.* Unpublished.

Oestreich, A. E. 2008; *Growth of the Pediatric Skeleton: A Primer for Radiologists.* New York: Springer.

Ortner, D. J. 2003; *Identification of Pathological Conditions in Human Skeleton Remains.* San Diego: Elsevier.

Pearce, J. 2001; Infants, cemeteries and communities in the Roman provinces. In D. Davis, A. Gardner and K. Lockyear (Eds.) *TRAC 2000.* Oxford: Oxbow: 125-142.

Perry, M. A. 2006; Redefining childhood through bioarchaeology: Toward an archaeological and biological understanding of children in Antiquity. *Archaeological Papers of the American Anthropological Association,* Vol. 15 (1): 89-111.

Pitts, M. 2008; Globalizing the local in Roman Britain: An anthropological approach to social change. *Journal of Anthropological Archaeology,* Vol. 27: 493-506.

Pitts, M. and Griffin, R. 2012; Exploring Health and Social Well-Being in Late Roman Britain: An Intercemetery Approach. *American Journal of Archaeology,* Vol. 116 (No. 2): 253-276.

Prentice, A. 2003; Pregnancy and Lactation. In F. H. Glorieux, J. M. Pettifor and H. Jüppner (Eds.) *Pediatric Bone: Biology and Diseases.* London: Academic Press: 249-269.

Rana, R. S., Wu, J. S. and Eisenberg, R. L. 2009; Periosteal Reaction. *American Journal of Radiology,* Vol. 193: 259-272.

Rawson, B. 1986; Children in the Roman *Familia.* In B. Rawson (Ed.) *The Family in Ancient Rome: New Perspectives.* New York: Cornell University Press: 170-200.

Rawson, B. 2003; *Children and Childhood in Roman Italy.* Oxford: Oxford university Press.

Redfern, R. 2003; Sex and the City: A biocultural investigation into female health in Roman Britain. In G. Carr, E. Swift and J Weekes (Eds.) *TRAC 2002 Proceedings of the Twelfth Annual Theoretical Roman Archaeology Conference.* Oxford: Oxbow Books.

Redfern, R. 2007; The influence of culture upon childhood: an osteological study of iron Age and Romano-British Dorset. In M. Harlow and R. Laurence (Eds.) *Age and Ageing in the Roman Empire.* Portsmouth, Rhode Island: Journal of Roman Archaeology: 171-194.

Redfern, R. 2008; A Bioarchaeological investigation of Cultural Change in Dorset, England (Mid-to-Late Fourth Century B.C. to the End of the Fourth Century A.D.). *Britannia,* Vol. 39: 161-192.

Redfern, R. C. and DeWitte, S. N. 2011; A New Approach to the Study of Romanization in Britain: A Regional Perspective of Cultural Change in Late Iron Age and Roman Dorset Using the Siler and Gompertz-Makeham Models of Mortality. *American Journal of Physical Anthropology,* Vol. 144: 269-285.

Redfern, R. C., DeWitte, S. N., Pearce, J., Hamlin, C. and Egging Dinwiddy, K. 2015; Urban-Rural Difference in Roman Dorset, England: A Bioarchaeological Perspective on Roman Settlements. *American Journal of Physical Anthropology,* Vol. 157: 107-120.

Redfern, R. C. Millard, A. R. and Hamlin, C. 2012; A regional investigation of subadult dietary patterns and health in late Iron Age and Roman Dorset, England. *Journal of Archaeological Science,* Vol. 39: 1249-1259.

Redfern, R. C. and Roberts, C. A. 2005; Health in Romano-British urban communities: Reflections from the cemeteries. In D. N. Smith, M. B. Brickley and W. Smith (Eds.) *Fertile ground: Papers in honour of Susan Limbey.* Oxford: Oxbow Books: 115-129.

Redfield, A. 1970; A New Aid to Aging Immature Skeletons: Development of the Occipital Bone. *American Journal of Physical Anthropology,* Vol. 33: 207-220.

Reitsema, L.J. and McIlvaine, B. K. 2014; Reconciling "stress" and "health" in physical anthropology: What can bioarchaeologists learn from the other subdisciplines? *American Journal of Physical Anthropology,* Vol. 155: 181-185.

Resnick, P. J. 1970; Murder of the Newborn: A Psychiatric Review of Neonaticide. *American Journal of Psychiatry,* Vol. 126, No. 10: 1414- 1421.

Roberts, C. A. & Cox, M. 2003; *Health and Disease in Britain: From Prehistory to the Present Day.* Stroud: Sutton Publishing.

Rogers, A. 1997; Vulnerability, health and healthcare. *Journal of Advanced Nursing,* Vol. 26: 65-72.

Rohnbogner, A. and Lewis, M. E. 2017; Poundbury Camp in Context – a new Perspective on the Lives of Children from urban and rural Roman England. *American Journal of Physical Anthropology,* Vol. 162 (2): 208-228.

Said-Mohamed, R., Pettifor, J. M. and Norris, S. A. 2018; Life History theory hypotheses on child growth: Potential implications for short and long-term child growth, development and health. *American Journal of Physical Anthropology,* Vol. 165: 4-19.

Satterlee Blake, K. A. 2018; The Biology of the Fetal Period: Interpreting Life from Fetal Skeletal Remains. In S. Han, T. K. Betsinger, and A. B. Scott (Eds.) *The Fetus: Biology, Culture, and Society.* New York: Berghahn Books: 34-58.

Schaefer, M. Black, S. & Scheuer, L. 2009; *Juvenile Osteology: A Laboratory and Field Manual.* London: Elsevier Inc.

Schell, L. M. 1997; Culture as a Stressor: A Revised Model of Biocultural Interaction. *American Journal of Physical Anthropology,* Vol. 102: 67-77.

Scheuer, L. and Maclaughlin-Black, S. 1994; Age Estimation from the Pars Basilaris of the Fetal and Juvenile Occipital Bone. *International Journal of Osteoarchaeology,* Vol. 4: 377-380.

Scheuer, L. Musgrave, J. H. & Evans, S. P. 1980; The estimation of late fetal and perinatal age from limb bone length by linear and logarithmic regression. *Annals of Human Biology,* 7 (3): 257-265.

Schillaci, M. A., Sachdev, H. P. S. and Bhargava, S. K. 2012; Technical Note: Comparison of the Maresh Reference Data With the WHO International Standard for Normal Growth in Healthy Children. *American Journal of Physical Anthropology,* Vol. 147: 493-498.

Scobie, A. 1986; Slums, Sanitation, and Mortality in the Roman World. *KLIO,* Vol. 68 (2): 390-433.

Scott, E. 1989; Animal and infant Burials in Romano-British Villas: A Revitalization Movement. In P. Garwood, D. Jennings, R. Skeates and J. Toms (Eds.) *Sacred and Profane: Proceedings of a Conference on Archaeology, Ritual and Religion. Oxford 1989.* Oxford: Oxbow Books: 115-121.

Scott, E. 1999; *The Archaeology of Infancy and Infant Death.* BAR International Series 819. Oxford: Archaeopress.

Shopfner, C. E. 1966; Periosteal bone growth in normal infants: A preliminary report. *American Journal of Roentgenology,* Vol. 97: 154-163.

Sinclair, D. 1985; *Human Growth After Birth* (4th Edition). Oxford: Oxford University Press.

Smith, P. and Kahila, G. 1992; Identification of infanticide in Archaeological Sites: A Case Study from the Late Roman-Early Byzantine Periods at Ashkelon, Israel. *Journal of Archaeological Science,* Vol. 19.: 667-675.

Tanner, J. M. 1978; *Foetus Into Man: Physical Growth from Conception to Maturity.* London: Open Books Publishing Ltd.

Tocheri, M. W., Dupras, T. L., Sheldrick, P. and Molto, J. E. 2005; Roman Period Fetal Skeletons from the East Cemetery (Kellis 2) of Kellis, Egypt. *International Journal of Osteoarchaeology,* Vol. 15 (5): 326-341.

Upper Nene Archaeological Society 2009 *Phase Descriptions* [Online] [Accessed: November 2016]
Available from:
http://www.unas.org.uk/magazine/magview.php?ID=8&date=0910&secshun=2

Webster, J. 2001; Creolizing the provinces. *American Journal of Archaeology,* Vol. 105:209–225.

Webster, J. 2005. Archaeologies of slavery and servitude: bringing 'New World' perspectives to Roman Britain. *Journal of Roman Archaeology* 18:161-179.

Wells, C. and Collis, J. R. [No Date]; *The Burials.* Owslebury Site Report (Unpublished).

Weston, D. A. 2012; Nonspecific infection in palaeopathology: Interpreting periosteal reactions. In A. L. Grauer (Ed.). *A Companion to Paleopathology*. Chichester: Wiley-Blackwell: 492-512.

Wiedemann, T. 1989; Adults and Children in the Roman Empire. New Haven & London: Yale University Press.

Winick, M., Brasel, J. A. and Rosso, P. 1972; Nutrition and Cell Growth. In M. Winick (Ed.) *Nutrition and Development.* London: John Wiley & Sons Inc.: 49-98.

Wu, G. Imhoff-Kunsch, B. and Webb Girard, A. 2012; Biological Mechanisms for Nutritional Regulation of Maternal Health and Fetal Development. *Paediatric and Perinatal Epidemiology,* Vol 26, Suppl.1: 4-26.

Chapter 7: Manuscript 2

Like Mother, Like Child: Investigating change and continuity in perinatal and maternal health stress in Post-Medieval London.

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KEY WORDS: Health; Growth; London; Infant; Fetal; Socioeconomic Status.

Abstract: *Post-Medieval London represented an inherently stressful environment. Poor sanitation, deleterious living and working conditions, and extensive levels of pollution contributed to make London notorious for its unhealthy environment. In response to these harmful conditions, clear changes can be observed within archaeological skeletal samples, revealing evidence of growth and health disruption. Fetal, perinatal and infant individuals are considered to provide the most sensitive depictions of past population health. Consequently, assessment of 169 individuals, aged to be six months or younger (< 64 GWA), from seven post-Medieval archaeological sites in London provide evidence of chronic growth and health disruption. Significant differences were found between dental and skeletal age estimates for all age categories. Furthermore, six of the seven samples assessed show evidence of growth disruption, with individuals from the middling status samples identified as having the highest frequency of individuals affected. However, those of the low status samples were found to show the severest growth disparities between dental and skeletal age estimates. Metric assessment of the pars basilaris has revealed that age estimates derived from this skeletal element show parity with dental age estimates, suggesting it can be utilised as a proxy for dental, and potentially chronological age. Pathological lesions were identified in all of the archaeological samples regardless of social status. However, individuals from Cross Bones and St. Bride's Lower, low status populations, were found to show the most extensive cranial pathological changes. Middling status individuals were also identified to have high rates of pathological lesions. These findings support previous investigations in demonstrating the clear health consequences, to even the youngest members of these populations, of living in London during the post-Medieval period.*

Introduction

Post-Medieval London (16^{th}-19^{th} centuries) was a thriving urban centre which witnessed rapid expansion, particularly with the arrival of the Industrial Revolution (Beier 1978; Lewis

2002a; DeWitte *et al.* 2016). This transition heralded a new technological age and provided unrivalled economic potential (Storey 1992). Despite this progression, social inequalities continued to widen, and a highly stratified society of 'rich' and 'poor' dominated social structure (Lindert 1994; Storey 1992; Lewis 2002b; Beaumont *et al.* 2013). With marked disparities in socioeconomic status known to be causative factors of health disruption and mortality (Saunders & Hoppa 1993; Farmer 1996; Schell 1997; Babones 2008; Cavigelli & Chaudhry 2012; DeWitte *et al.* 2016), life course adversity is often reflected and identified within skeletal remains (e.g. De la Rúa *et al.* 1995; Pinhasi *et al.* 2006; Lewis & Gowland 2007; DeWitte *et al.* 2016; Ives & Humphrey 2017). Social disparity is, in itself, not the direct cause of growth and health disruption, rather social status predisposes and mediates resource access, which in turn affects nutrition, health care, and immunity (Martorell & Habicht 1986; Floud *et al*. 1990; Nicholas & Steckel 1991; Schell 1997; Stinson 2000; Robb *et al*. 2001; Steckel 2009; Halfon *et al.* 2014; DeWitte *et al.* 2016). Indeed, social status is still one of the most prevalent determinants of health today, with those at the lowest social strata often experiencing vastly poorer health and shorter life expectancies that those of the highest (Farmer 1996; Marmot 2005). Today, in low socioeconomic populations, average infant mortality is suggested to be 55 per 1000 live births (WHO Infant Mortality). This is over five times the contemporary rate for infant mortality in the European Region, which is considered to be generally of high socioeconomic status (WHO Infant Mortality). Consequently, despite modern clinical interventions and increased awareness of these associations, social disparity is still one of the biggest factors in determining birth and health outcomes (Phelan *et al.* 2010; Cavigelli & Chaudhry 2012; Robertson *et al*. 2013).

Post-Medieval London was an overcrowded and unsanitary urban centre (Forbes 1972; DeWitte *et al.* 2016). Many migrated to the capital from the countryside in search of work, particularly during the Industrial Revolution (Beier 1978). Migrants to London typically relocated from pastoral and agricultural occupations to those of servants and apprentices, which were unstable, temporary or seasonal occupations of low wage (Beier 1978; DeWitte *et al.* 2016). It is widely considered that post-Medieval London consisted of extremely poor living conditions, associated with increased risks of ill health and mortality (DeWitte *et al.* 2016). Areas of slum housing were notorious hotspots for disease and infection, as a consequence of the extreme poverty faced by many (Beier 1978). Houses were divided up with numerous individuals living within each room, even the cellars, and some landlords are thought to have constructed hovels in the alleyways (Beier 1978; Boulton 2000). Despite this,

a range of dietary sources are considered to have been available, though whether the poor were able to afford or access these resources is questionable. It has been suspected that many of the poor did occasionally consume fish (Mayhew 1985; Picard 2005), though isotopic evidence has yet to corroborate these suggestions (Beaumont *et al.* 2013). Therefore, as conditions in post-Medieval London were demonstrably impoverished for the majority of individuals living and working within this urban centre, it is widely accepted that archaeological skeletal remains from these contexts will have identifiable health and growth changes as result of exposure to such conditions. Social status is thus pivotal in regulating access to nutritional resources, hygienic living conditions and exposure to diseases and infections (Floud *et al.* 1990; Tanner 1994; Dowler & Dobson 1997; DeWitte *et al.* 2016). As the youngest members of both living and dead populations, fetal, perinatal and infant individuals are often found to reflect social conditions and inequalities most severely, meaning analysis of their skeletal remains is paramount for comprehension of the implications of this early life adversity.

The study of non-adult skeletal remains has vastly increased within the last decade, with a proliferation of bioarchaeological studies investigating health and growth disruption in the skeletal remains of children (Humphrey 2000a; Lewis 2002a; 2002b; Halcrow & Tayles 2008). Despite this, fetal, perinatal and infant individuals are currently under-represented in the literature (Halcrow & Tayles 2008; Kamp 2015; Halcrow *et al.* 2017; Lewis 2017c; Sánchez Romero 2017). However, these individuals represent a unique opportunity for tangible considerations of both individual and maternal health and wellbeing, regardless of the archaeological recovery of maternal individuals. Thus, fetal, perinatal and infant individuals, and assessment of their growth and health, provides an unparalleled insight into pre- and postnatal experiences (Scheuer & Black 2000a; Baxter 2005; Lewis 2007; Agarwal 2016; Halcrow & Ward 2017).

The malleable biological processes and parameters of growth and health are ones which commence *in utero,* but continue and remain throughout the life course (Halfon *et al.* 2014). Both pre- and postnatal life is regulated by endogenous and exogenous factors (Cattaneo 1991; Saunders & Hoppa 1993; Bogin 1999; King & Ulijaszek 1999; Cardoso 2007), yet the offspring has limited control over their exposure to these (Cattaneo 1991; Barker *et al.* 2012). Instead, both the intra- and extrauterine environments are largely maternally regulated. The mother-infant dyad is one in which previous and existing maternal life course experiences

impact upon the growing fetus/perinate/infant (Redfern 2003; Barker *et al.* 2012; Gowland 2015; Said-Mohamed *et al.* 2018). Susceptibility and fragility to disease, as well as immunological resistance, can be genetically regulated (Barker *et al.* 2012). Furthermore, offspring experiences rely on maternal ability to supply and buffer nutrients and antibodies via both the placenta and lactation. Consequently, both genetic and environmental factors may limit or aid individuals in attaining their growth and health potential. A high degree of plasticity during the pre- and postnatal periods (Wadhwa *et al.* 2011; Gowland 2015; Agarwal 2016; Said-Mohamed *et al.* 2018; Satterlee Blake 2018), means that growth and health status reflects the varying maternal and environmental exposures encountered. Therefore, fetal, perinatal and infant life stages are inherently fragile, complexly bound to maternal life course experiences and wellbeing. As such, assessment of fetal, perinatal and infantile growth and health status provides a tangible reflection of maternal, as well as community, health and wellbeing (Goodman & Armelagos 1989; Redfern 2003; Baxter 2005; Lewis 2007).

The Developmental Origins of Health and Disease Hypothesis (Barker & Osmond 1986; Barker 1994; 1997; 2003; 2012), has proposed that maternal health, and the environmental factors to which she is exposed, both during and prior to pregnancy, can significantly impact and alter growth and health of the offspring (Gowland 2015). Epigenetic changes are where gene expression, and not the underlying DNA sequence, has been altered in response to the adverse or beneficial conditions experienced *in utero* (Cattaneo 1991; Chmurzynska 2010; Kuzawa 2012; Mortier & Vanden Berghe 2012; Halfon *et al.* 2014; Glover 2015). Such changes predispose individual susceptibility, and/or resilience to disease, by altering the function of various biological systems (Slack 1991; Cameron & Demerath 2002; Luo *et al.* 2006; Chmurzynska 2010; Mortier & Vanden Berghe 2012). Importantly, immune function has been found to alter as a consequence of prenatal stress exposure, altering offspring phenotypic expression, and potentially increasing their susceptibility to disease (Boersma & Tamashiro 2015).

Epigenetic traits can become 'embedded', transferred from parent to child, and subsequently to grandchild, suggesting epigenetic signatures may be transmitted over multiple generations (Kuzawa & Quinn 2009; Halfon *et al.* 2014; Boersma & Tamashiro 2015; Glover 2015; Gowland 2015; Thorsell & Nätt 2016; Satterlee Blake 2018). Holland-Jones (2005) has termed this as a '*downstream effect*'. The realisation that prenatal life can be influenced by

previous multi-generational experiences challenges our ability to determine when an individual's biography truly begins (Gowland 2015). Consequently, fetal, perinatal and infant remains are valuable proxies for maternal and even generational health. However, disentangling the etiological and contextual implications of growth and health disruption identified within the skeletal remains of these individuals is complex, and a holistic interpretation of these factors is required.

Health is a complex biological construct, though what might be considered 'healthy' today is unlikely to directly correlate to perceptions of health in the past (King & Ulijaszek 1999; Roberts 2009). Therefore, although the concept of health has long been fundamental to bioarchaeological studies, there has been a re-emergence of the consideration of 'stress' as a pivotal factor in the regulation of physiological response to adversity (Temple & Goodman 2014). Stressors are considered to be those intrinsic and extrinsic factors and influences which have a negative and detrimental impact upon growth and health (Goodman *et al.* 1988; Goodman & Armelagos 1989; Reitsema & McIlvaine 2014). Stress indicators are the effects of those influences, leaving discernible changes to the skeleton, despite many typically being non-specific in presentation (Goodman *et al.* 1988; Lewis & Roberts 1997; Goodman & Martin 2002; Reitsema & McIlvaine 2014). Growth disruption and pathological lesions are widely considered to be proxies for evidence of exposure to stress (Goodman *et al.* 1988; Armelagos & Goodman 1991; Goodman & Martin 2002; Reitsema & McIlvaine 2014). This is because both are indicative of an abnormal physiological response (Bush 1991). Growth disruption signals an arrestment of growth, suggesting that the body is unable to maintain growth to its full potential, whilst pathological lesions provide direct evidence for conditions/diseases impacting upon the skeleton.

To date, a number of bioarchaeological studies have considered growth and health in London, particularly during the post-Medieval period (e.g. Harvey 1968; Ogden *et al.* 2007; Nitsch *et al.* 2011; Beaumont *et al.* 2013; DeWitte *et al.* 2016; Ives & Humphrey 2017; Newman & Gowland 2017). However, despite this abundance of contemporary research, few studies have considered the fetal, perinatal and infantile individuals in detail. Furthermore, consideration of these young individuals from all of the available archaeological samples has not previously been undertaken. Therefore, this study represents the first to consider fetal, perinatal and infantile health and growth disruption for post-Medieval London, contributing to the ongoing discussion surrounding detrimental early life experiences. The seven post-Medieval samples

will include individuals of differing socio-economic status (low, middling and high status) and locations within and around the City of London (See Fig. 1). The impact of overcrowding, poor sanitation, and socially-differentiated access to resources on the skeletal parameters of growth and non-specific indicators of physiological stress (pathological lesions) will be examined. Construction of a contextualised narrative, exploring the mother-infant dyad will reveal preliminary insights into the health and wellbeing of these individuals in post-Medieval London.

Research Context:

Databases for all post-Medieval sites catalogued, curated and held by the Museum of London (Wellcome Online Database) were searched for individuals determined to be fetal (< 36 gestational weeks of age (GWA)), perinatal (36 - 44 GWA) and infantile (44 GWA – 64 GWA (6-months post-partum)). In total 169 post-Medieval individuals were identified and analysed (Table 1.). Socioeconomic status has been documented as recorded for each sample (Table 1.).

In total, individuals from seven post-Medieval samples were assessed (Fig. 1). The cemeteries of both Broadgate and St Bride's Lower were formed to relieve overcrowding of parish cemeteries (Schofield & Maloney 1998; Miles & Conheeney 2005). Broadgate cemetery was a municipal cemetery, founded in 1569 by the City (Museum of London 2015). This New Churchyard became the burial place predominantly for the poorer classes (Harding 2002). At St Bride's Lower grave cuts were almost impossible to identify due to the densely packed nature of the burial ground (Miles & Conheeney 2005). The Lower churchyard was the cheapest burial place in the parish and heavily used throughout the 18th and 19th centuries (Miles & Conheeney 2005). In addition, some of those buried in St Bride's Lower cemetery are likely to have been inmates in the adjacent Bridewell Workhouse and Fleet Prison (Miles & Conheeney 2005; Kausmally 2008). Similarly, Cross Bones Cemetery, located in Southwark (Brickley *et al.* 1999), was a burial place for the poorest individuals living in London at this time. It is thought to have originally been a single women's burial ground for those working in the brothels on Bankside (Mikulski 2007; Brickley *et al.* 1999). The burial ground came into 'proper' use in 1760, and, until its closure in 1853, remained a paupers' cemetery (Mikulski 2007; Brickley *et al.* 1999). In total 148 individuals were excavated from this site (Brickley *et al.* 1999) and are thought to date from the early to late 19th century phases of the site (Mikulski 2007). St Thomas' is a 17th century burial ground associated with

St Thomas' Hospital (Jones 1991). Excavation revealed three mass burial trenches, considered also to be those of paupers, or victims of an epidemic event (Bekvalac 2007). In contrast, Chelsea Old Church was situated on the edge of the City of London, a rural area during the 18th and 19th centuries (Museum of London 2009). By the mid-18th century Chelsea was considered a wealthy, prosperous and healthy area of London (Cowie *et al*. 2008). The individuals excavated from this cemetery consist of individuals of a higher socio-economic status (Museum of London 2009). Two of the individuals analysed were still *in utero* when buried (Museum of London 2009; Cowie *et al*. 2008). Finally, individuals excavated from the Royal London Hospital Burials are thought to date between 1825 and 1841/1842 (Fowler & Powers 2012). Social status for these individuals is unknown and it maybe that their skeletal remains were retained because the individuals expressed uncommon medical complaints.

TABLE 1.1 Number of individuals with dental and skeletal elements available for assessment by archaeological sample.

Archaeological Sample	**Date *(Century)***	**Status**	**Number of Individuals (*N*)**	**Sample Size (*N*) By Dental and Skeletal Element**				
				Dentition	**Femur**	**Tibia**	**Humerus**	***Pars Basilaris***
St Benet Sherehog	16^{th}-17^{th}	Middle	19	9	9	6	10	9
Broadgate	16^{th}-18^{th}	Low	21	13	8	5	8	12
St Thomas' Hospital	17^{th}	Low	5	4	2	2	2	5
St Bride's Lower	17^{th}-19^{th}	Low	52	29	35	33	40	37
Chelsea Old Church	18^{th}-19^{th}	High	7	5	2	2	5	4
Cross Bones	19^{th}	Low	58	36	41	44	43	43
Royal London Hospital	19^{th}	Unknown	7	1	6	6	6	1
Overall Total			169	97	103	98	114	111

TABLE 1.2 Frequency of individuals aged to be fetal, perinatal or infantile based on dental development. Unknown individuals are those where no dentition was available for assessment.

Population	**Date *(Century)***	**Status**	**Number of Individuals (*N*)**	**Sample Size (*N* (%)) by Chronological Age Category (Based on Dental Development)**			
				Fetal	**Perinatal**	**Infant**	**Unknown**
St Benet Sherehog	16^{th}-17^{th}	Middle	19	0	1	8	10
Broadgate	16^{th}-18^{th}	Low	21	0	2	11	8
St Thomas' Hospital	17^{th}	Low	5	0	1	3	1
St Bride's Lower	17^{th}-19^{th}	Low	52	0	3	26	23
Chelsea Old Church	18^{th}-19^{th}	High	7	0	1	4	2
Cross Bones	19^{th}	Low	58	0	12	24	22
Royal London Hospital	19^{th}	Unknown	7	0	1	0	6
			169	0	21	76	72

Site Map

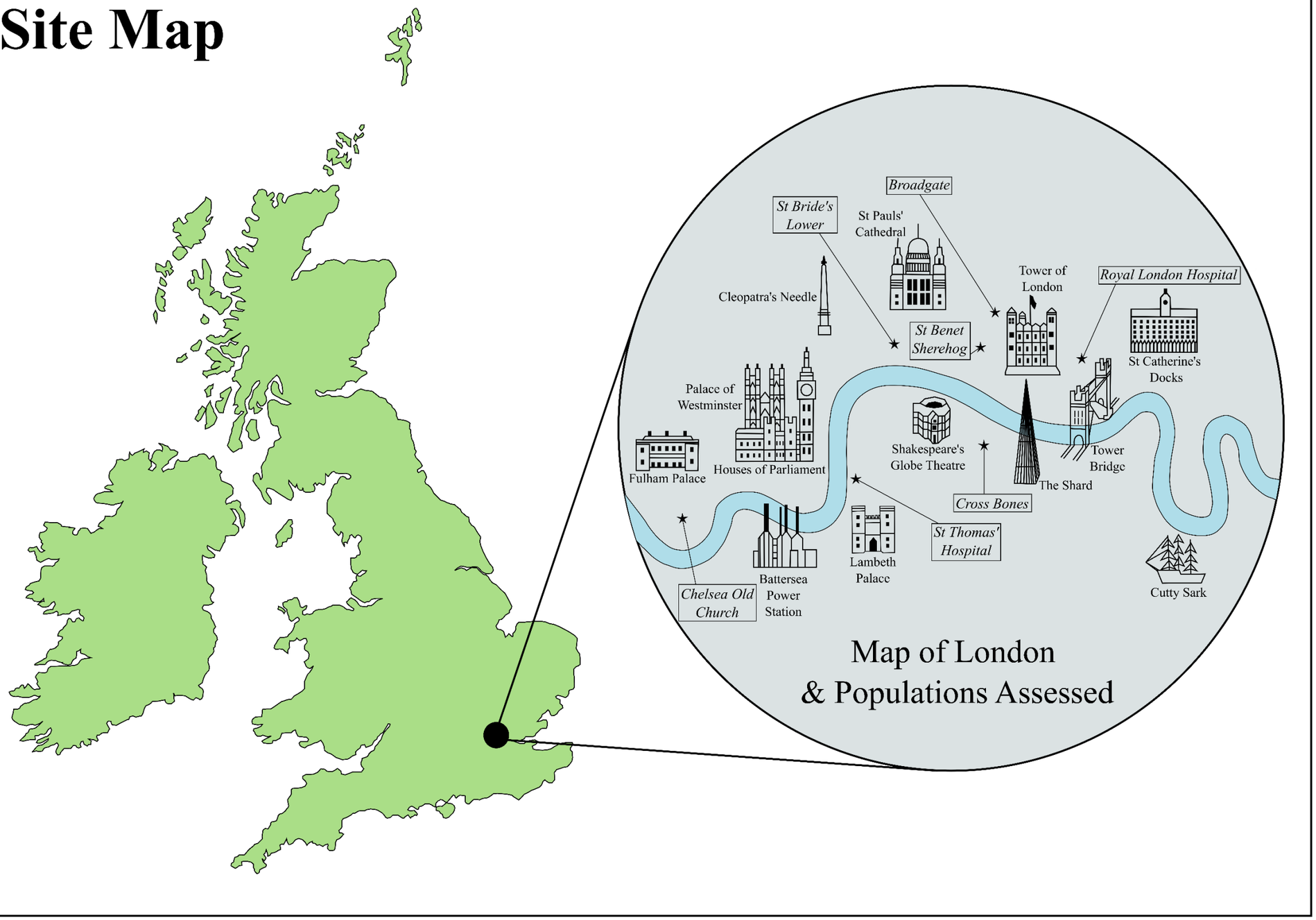

Figure 1. Map detailing the location of the seven post-Medieval populations assessed.

Methods:

Age-at-death estimations based on both dental development, and metric assessment of long bone diaphyseal lengths and the *pars basilaris*, have been calculated.

Dental development is widely utilised to infer chronological age-at-death of non-adult individuals (Moorrees *et al.* 1963b; Hillson 2005; Lewis 2007; AlQahtani *et al.* 2014). Teeth grow systematically from the tip of the crown to the root. This happens at relatively consistent ages for each tooth (Hillson 1979). Thus, as this growth and development follows a regular sequence and trajectory, physiological stage of dental development correlates most closely with chronological age (Bang 1989; Hillson 2005; AlQahtani *et al.* 2014). Though both genetic and environmental factors may affect tooth growth and development (Massler *et al.* 1941; Heuzé & Cardoso 2008), it is widely accepted that tooth development shows less variability and fluctuation than other growth and development parameters (Moorrees *et al.* 1963b; Gustafson & Koch 1974; Bang 1989; Hoppa & Fitzgerald 1999; Bolaños *et al.* 2000; Humphrey 2000a; Liversidge & Molleson 2004; Satterlee Blake 2018). Estimation of chronological age using dental development has also been found to be more accurate in younger infants and children, particularly those under 10 years of age (Bolaños *et al.* 2000; 103; Lewis 2007; AlQahtani *et al.* 2014). Consequently, within this study dentally derived age estimates are considered as a proxy for chronological age.

Tooth cusp development was recorded in accordance with Moorrees *et al.* (1963a; 1963b) and age-at-death estimates attributed using the dental development atlas developed by AlQahtani *et al.* (2010). For individuals where tooth cusp development fell between two age estimates, a midpoint between those two estimates was assigned in gestational weeks, and the maximum error level afforded. The standard developed by AlQahtani *et al*. (2010) was utilised in assessment as it is the most recently established dental development chart, and uses both historical and clinical known age-at-death individuals. Furthermore, this methodology has been found to have high levels of reproducibility (AlQahtani *et al.* 2014). All dental age-at-death estimations have been given in gestational weeks throughout (GWA).

Metric assessment of fetal, perinatal and infant skeletal remains is the most commonly employed method for determining chronological age-at-death (Humphrey 2000b; Lewis 2007; Utczas *et al.* 2017). This is likely due to skeletal elements being both better preserved in the archaeological record than the dentition (Gowland & Chamberlain 2002), as well as recognised and collected by the archaeologist. As with assessment of the dentition, age-at-death estimates derived from metric assessment of long bones have always been given in gestational weeks of age (GWA) throughout.

Metric assessment of the skeletal remains focused on diaphyseal length measurements of the femora, tibiae, and humeri. Measurements were taken using digital sliding callipers (accuracy of +/-0.02mm), with all results recorded to the hundredth of the millimetre, in accordance with the metric analyses outlined in Fazekas & Kósa (1978). Gestational age-at-death estimations were calculated using the published linear regression equations for long bone diaphyseal lengths by Scheuer *et al*. (1980). Only the long bones of the skeleton have regression equations available for use in assessment. Where both left and right skeletal elements were available for assessment both were analysed and had age estimates generated, with the average age for that element used in analysis. The chronological age estimate generated has typically been plotted with the error level (+/- *X* gestational weeks of age) given as a range.

This use of linear regression equations was employed by the author as it is one of the only methods available that provides an error level for age-at-death estimation, and is widely used in other studies making results of this assessment comparable (Lewis & Gowland 2007; e.g. Mays 1993; Lewis 2002a; Halcrow *et al.* 2012). However, this linear regression method has been criticised, suggested to age individuals in a way which mimics the demographic make-up of the sample used to create the regression models (Gowland & Chamberlain 2002; Lewis & Gowland 2007). This has been a common criticism of many age-estimation techniques and methods, with studies found to often reflect the age distribution of individuals within the reference sample (Gowland & Chamberlain 2002; e.g. Bocquet-Appel & Masset 1982). To limit the effect of this bias other studies have employed Bayesian statistics to redistribute the age estimations generated (e.g. Gowland & Chamberlain 2002). Bayesian analysis considers the likelihood of individuals falling within age categories, in comparison to a natural mortality profile derived from perinatal

and infant life tables (Lewis & Gowland 2007). Therefore, by employing this assessment, age estimates are redistributed by probability (Gowland & Chamberlain 2002). However, Bayesian analysis considers all individuals as a whole, meaning age estimates cannot be generated for single individuals (Lewis & Gowland 2007). This limits detailed assessment of individuals and the potential for identifying growth disruption. As a result, because this research aims to focus on identifying growth disruption within individuals, Bayesian statistical assessment has not been employed.

Measurements were also taken from the *pars basilaris*, as due to the changes in morphology, and thus the measurements obtained, the *pars basilaris* is considered to be indicative of certain age thresholds (Redfield 1970; Scheuer & Maclaughlin-Black 1994; Lewis 2007). Maximum width, sagittal length and maximum length were all recorded for the *pars basilaris* when possible. Redfield (1970) established a preliminary method by which to tell broad age categories based on the morphology and size of this bone. Measurements of the *pars basilaris* were assessed using this method (Redfield 1970), with specific gestational weeks attributed using Scheuer and Maclaughlin-Black (1994). A limitation of this method is that no ranges or error levels were given for each specific age category (Scheuer & Maclaughlin-Black 1994). Therefore, where measurements fell within a range of age categories, the mean age category has been plotted, with the minimum and maximum age categories used as upper and lower age ranges. For example, a measurement which fell into the 3 weeks, 4 weeks, 7 weeks and 3-months age categories (43-52 GWA), has a mean point of 47.5 GWA, with a range of +/- 4.5 GWA.

Inherent limitations will be incurred as a result of comparing skeletal metric data collected within this study against a range of reference data. This is due to the varying skeletal samples from which the reference methods were constructed. Although it is almost always suggested that an appropriate reference sample should be used for comparison, it is almost impossible to do this for archaeological assessments (Scheuer & Black 2000b). The application of age estimation methods derived from a specific reference sample to an archaeological sample, far removed temporally and geographically, is a well-known source of error (Bocquet-Appel & Masset 1982). Furthermore, many reference samples have been developed using archaeological or historical skeletal remains of deceased individuals. Therefore, these individuals are unlikely to represent

healthy non-adults, and are often lacking vital biological data such as documented age or sex (e.g. Fazekas & Kósa 1978). Additionally, as growth is known to respond to a multitude of intrinsic and extrinsic factors (Saunders & Hoppa 1993; Bogin 1999), comparison of archaeological data to that of modern, known samples is likely to increase variability and inaccuracy in the results and interpretations obtained. Consequently, the ability to compare archaeological samples to modern reference standards is limited (Hoppa & Fitzgerald 1999). It is therefore important to be mindful of the error ranges associated with each particular ageing technique and interpret the results accordingly. Consequently, for individuals where dental and skeletal age estimates and ranges have been identified to correspond/overlap, an interpretation of growth disruption has not been attributed. Growth disruption has only been reported in those individuals where age estimates show no correlation. Therefore, error ranges specified for both dental development and metric assessment of the long bones have always been afforded. Furthermore, where *pars basilaris* measurements were found to correlate to multiple age categories, the largest applicable error range was used. This was to ensure that for all individuals, growth disruption was only reported where clear distinctions between age estimates could be identified.

Assessment of pathological lesions was undertaken macroscopically. Each pathological change/lesion was recorded descriptively by the author and documented photographically. As identification of pathological lesions within fetal, perinatal and infant individuals is still ambiguous, grading schemes were employed to differentiate between extensive and minor changes. Grading schemes (outlined below) consisted of three discrete categories, where those in categories 2 and 3 likely represent pathological changes, whilst those in category 1 may represent changes which could equally be associated with normal growth and bone formation. As we are still unable to distinguish between normal and pathological changes, particularly for new bone formation (NBF), it was intended that all potential changes were recorded, regardless of their case or etiology. By employing a grading system, the severity and prevalence of lesions to particular elements could be observed, and those lesions considered to be more ambiguous isolated from those definitively identified to be pathological.

Location, type and severity of pathological lesions was recorded for each lesion observed. Table 2. details the variables recorded for each of these categories, whilst Table 3. outlines the grading systems employed to determine severity of NBF, lytic lesions and metaphyseal expansion.

TABLE 2. Categories and variables used in the recording of pathological lesions.

Category	**Variable**
Location	Cranial or Postcranial
Skeletal Element	e.g. Femur, Tibia, Frontal Bone
Aspect	e.g. Endocranial, Anterior, Circumferentially
Type I	NBF, Lytic, Metaphyseal Extension, Morphological change.
Type II	Woven, Lamellar and/or Spiculated
Severity	Grade 1, 2, or 3

T-tests were employed to determine significant differences between skeletal and dental age estimations. T-test analysis was undertaken in PAST (developed by Hammer, Harper & Ryan 2001) where significance was set 95%. Therefore, *p*-values below 0.05 were considered to be statistically significant. Prevalence rates were calculated for pathological lesions for all 169 individuals assessed. Total numbers of individuals and skeletal/dental elements have been given, with the numbers and percentages of individuals showing pathological changes to the element recorded. Chi-squared tests for independence at 99.5 % confidence ($p < 0.05$) were also employed for pathological categories to observe whether there was any relationship between various pathological variables and social status. Chi-square results are presented numerically, where $p < 0.05$ shows there is a significant relationship between the variables. Chi-Squared values (X^2) have also been given. As the sample sizes were small, Fisher's exact test was used to determine *p* values.

TABLE 3. Grading systems for new bone formation, lytic lesions and metaphyseal expansion employed for assessment of pathological lesions within this study.

	Grade 1	Grade 2	Grade 3
New Bone Formation	New bone formation, which may be woven or lamellar in appearance, will be considered to be grade 1 when the NBF is not clearly apparent and the margins are unable to be clearly defined from that of normal cortical bone. Grade 1 NBF is likely to be isolated in location, appearing minimally across the skeletal element.	New bone formation recorded as being grade 2 will be clearly identifiable as a definable area of woven or lamellar bone formation. There will be clear boundaries/borders to the NBF and it will obviously differ from the normal cortical bone of the skeletal element. Grade 2 NBF is likely to be distinguishable as a clear layer of bone on top of the original cortical surface. It is likely that NBF listed within this category will be formed of a single layer though may extend over a large aspect area of the skeletal element.	New bone formation recorded as being grade 3 will be the more severe type of NBF, with clear, multi-layered or thick NBF across a large area/aspect of the skeletal element. The NBF may be woven or lamellar in appearance and is clearly seen to be on top of the original cortical bone.
Lytic Lesions	Lytic lesions considered to be grade 1 likely consist primarily of macro-porosity. This porosity will be relatively minor, though may extend over a large skeletal area, and no clear destruction of the cortical bone will be apparent.	Lytic lesions considered to be grade 2 will likely show evidence of some cortical destruction as well as porosity. However, cortical destruction will not be widespread throughout the skeletal element and is instead likely to be in isolated concentrations.	Lytic lesions considered to be grade 3 will show extensive cortical destruction and/or porosity. Destruction will be widespread throughout the element.
Metaphyseal Expansion	Metaphyseal expansion considered to be grade 1 will likely consist of noticeably widened/flared metaphyses which do not appear proportional for the long bone diaphysis. However, despite this expansion no change to the metaphyseal margin or trabecular bone structure will be observed.	Metaphyseal expansion will be considered to be grade 2 when involvement of the metaphyseal margin is apparent. This will result in atypical and misshapen metaphyseal margins often combined with a discernible brim/lip to the metaphysis.	Metaphyseal expansion considered to be grade 3 will be the most severe and where involvement of the trabecular bone structure can be seen. Individuals displaying grade 3 metaphyseal extension will likely have more porous metaphyses and the trabecular structure will appear clearly expanded and widened. Involvement of the metaphyseal margin may still be apparent though this may be lost due to the trabecular expansion.

Results

Average skeletal age estimates for femoral, humeral and tibial measurements have been calculated for each dental age group. Table 4 details summary statistics (mean, standard deviation and confidence interval (+/-)) for each of the skeletal long bones assessed. T-test analysis was undertaken considering skeletal age estimates, by bone element and age group, in comparison to dental age. Results presented in bold are those where differences between mean dental and skeletal age-at-death estimates were found to be significantly different (where $P < 0.05$). Results of assessment (Table 4.) show that there are significant differences between dental and skeletal age estimates for almost every dental age group and skeletal element. Only those dentally aged to be 39 GWA show no significant differences between their tibial and femoral ages when compared to dentition.

Plotting these mean skeletal age estimates by dental age (Fig. 2) for the femora, humeri and tibiae assessed, demonstrates that the growth profiles for all three of these long bones fall below the 'optimal growth trajectory'. The optimal growth trajectory is simply the assumption that dental and skeletal age estimates should be identical within a single individual. Thus, in an individual of optimal growth, both dental and femoral, humeral and tibial age should correspond. Mean skeletal age estimates are shown to increase with dental age, and despite long bone growth profiles showing a similar trajectory for the majority of age categories, profiles appear to be diverging in the 64 GWA dental category. The femur is shown to have the highest growth trajectory, whilst the tibia has the lowest.

To explore evidence of growth disruption on an individual level, those with dental age estimates and at least one skeletal age estimate (femoral, tibial or humeral) were assessed (Fig. 3). In total 79 individuals were found to have both dental and one skeletal long bone element available for age-at-death assessment; 57 individuals had femora, 71 humeri, and 53 tibiae. Assessment reveals 22 individuals had dental and skeletal age estimates where mean age estimates and age ranges do not overlap, unambiguously indicative of growth disruption. Of these 22 individuals, ten were from the archaeological sample of Cross Bones, six were from St Brides' Lower, three from St Benet Sherehog, and one individual was identified from the sites of Chelsea Old Church, Broadgate, and St Thomas' Hospital.

TABLE 4. Mean skeletal age estimates (GWA) by dental age group for the femora, humeri and tibiae. Results of t-test analyses in bold are those where significant differences were found between mean dental and skeletal age estimates.

Dental Age	(N)	Mean Skeletal Age	S.D.	95% Conf. (+/-) on the mean	T-Test
			FEMUR		
38	8	32.3	1.7	1.4	**-9.48**
39	2	33.9	2.5	22.4	-2.91
43	4	36.2	2.2	3.5	**-6.23**
46	10	36.5	2.6	1.8	**-11.64**
52	23	38.8	2.5	1.1	**-25.14**
58	7	42.4	4.4	4.1	**-9.32**
64	2	47.5	1.1	10.3	**-20.34**
			HUMERUS		
38	8	32.9	2.2	1.8	**-6.53**
39	4	32.5	1.8	2.8	**-7.24**
43	6	36.9	2.3	2.4	**-6.59**
46	13	37.2	2.6	1.6	**-12.23**
52	28	38.6	2.0	0.8	**-35.32**
58	9	42.4	3.5	2.7	**-13.17**
64	3	44.1	3.8	9.4	**-9.09**
			TIBIA		
38	7	32.5	1.9	1.7	**-7.75**
39	3	33.3	2.8	6.9	-3.58
43	4	36.5	3.3	5.3	**-3.95**
46	8	36.9	2.3	1.9	**-9.90**
52	20	38.9	2.8	1.3	**-20.98**
58	8	42.4	4.1	3.4	**-10.70**
64	3	42.6	4.3	10.7	**-8.62**

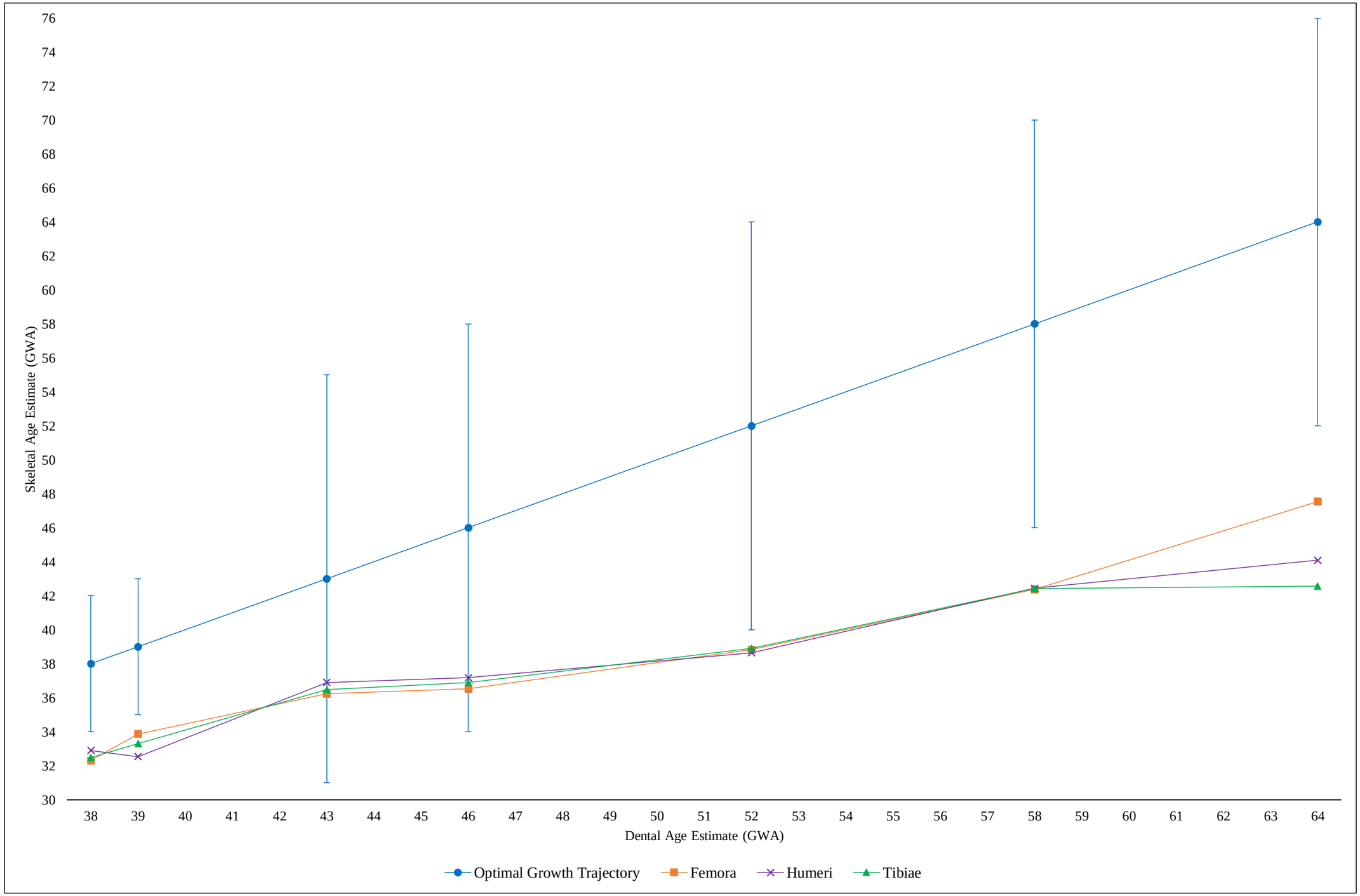

Figure 2. Mean skeletal age estimates (GWA) plotted by dental age. Optimal growth trajectory represents the expected growth profile if dental and skeletal age estimates were found to be identical for each age group. Error bars in accordance with dental age estimates have been plotted for optimal growth trajectory to highlight that average skeletal ages typically fall outside of these error ranges.

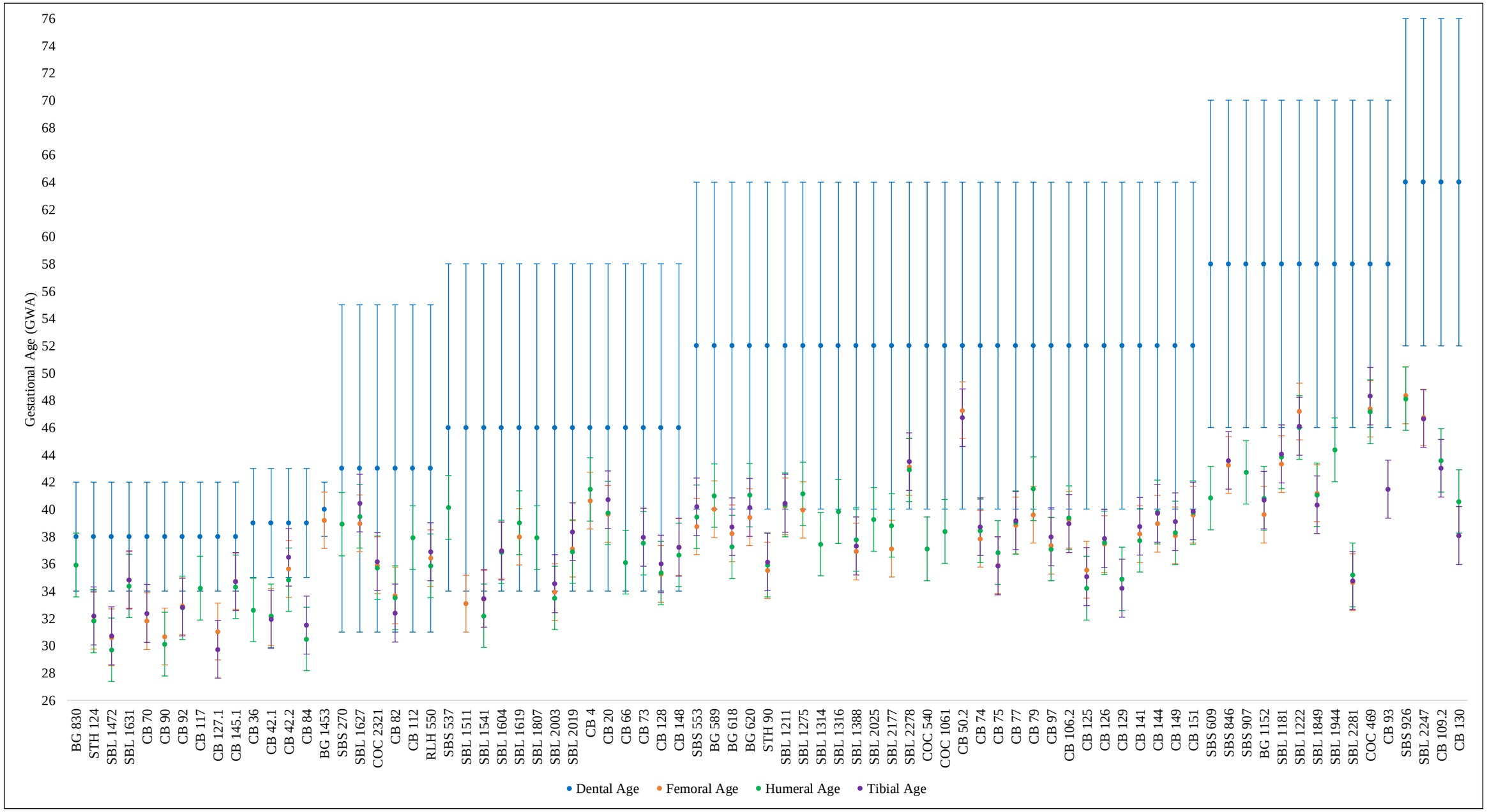

Figure 3. Skeletal and dental age-at-death estimates plotted for each individual with age-estimates available. Error ranges (in GWA) have been afforded to both dental and skeletal age estimates in accordance with the age estimation methodologies employed (AlQahtani et al. 2010; Scheuer et al. 1980). Individuals considered to show evidence of growth disruption are those where dental and skeletal age estimates and ranges do not overlap.

Consideration of individual growth disruption by dental age estimate (Table 5.1), archaeological sample (Table 5.2) and sample status (Table 5.3) was afforded. Results show that those aged 64 GWA and 58 GWA have the highest prevalence of growth disruption. Individuals sampled from St Benet Sherehog and St Thomas' Hospital are the samples with highest prevalence of growth disruption. However, given the small sample sizes for these sites, results may be over-emphasised.

TABLE 5.1 Number and percentage of individuals with growth disruption by dental age.

Dental Age (GWA)	**Total *N***	***N* Growth Disruption**	**% Growth Disruption**
38	10	3	30
39	4	2	50
40	1	0	-
43	6	0	-
46	14	0	-
52	29	6	21
58	11	8	73
64	4	4	100

TABLE 5.2 Number and percentage of individuals with growth disruption by sample.

Sample	**Total *N***	***N* Growth Disruption**	**% Growth Disruption**
St Benet Sherehog	7	4	57
Broadgate	6	1	17
St Thomas' Hospital	2	1	50
St Bride's Lower	24	6	25
Chelsea Old Church	4	1	25
Cross Bones	35	10	29
Royal London Hospital	1	0	0

TABLE 5.3 Number and percentage of individuals with growth disruption by sample status.

Sample Status	Total *N*	*N* Growth Disruption	% Growth Disruption
High	4	1	25
Middle	7	4	57
Low	67	18	27
Unknown	1	0	0

Assessment of the *pars basilaris* displays alternative results with limited evidence of growth disruption and a strong relationship with dental age-at-death estimates (Fig. 4). In total, 78 individuals had dental and *pars basilaris* age-at-death estimates which could be compared. Only three individuals had ages derived from assessment of the *pars basilaris* where no overlap or correlation with dental age and age range is present. Results suggest that maximum length and maximum width most closely correlate with dental age, whilst sagittal length appears to often over-estimate age, particularly for postnatal infant individuals. Averaged age estimates from assessment of the three dimensions of the *pars basilaris* have been considered by dental age (Fig. 5). Though these averaged age estimates show the *pars basilaris* also generates younger age estimates on average than dental age, comparison of these results to average age estimates derived from skeletal long bones (Fig. 2), demonstrates that *pars basilaris* metrics generates estimates which more closely align with dental age.

Pathological assessment of the 169 individuals was undertaken, differentiating lesions by location: cranial and postcranial (Table 6.1). Cranial pathology is more prevalent than postcranial pathology in all skeletal samples, except St Thomas' Hospital. The samples from Cross Bones, St Bride's Lower and Broadgate have very high prevalence rates of cranial lesions. St Thomas has the highest prevalence rate of postcranial lesions, though both St Benet Sherehog and Cross Bones also have high prevalence rates.

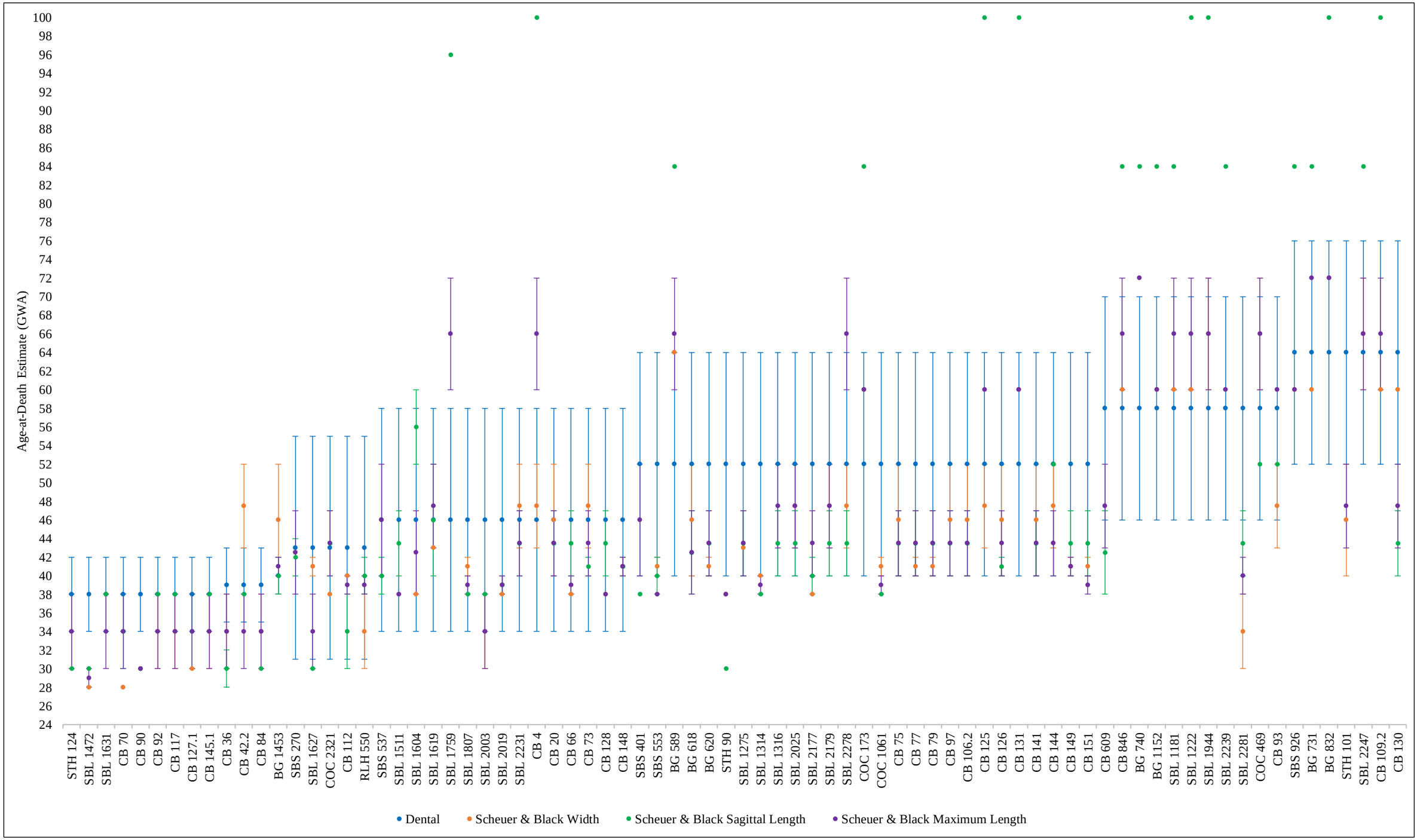

Figure 4. Pars Basilaris and dental age-at-death estimates plotted for each individual with age-estimates available. Error ranges (in GWA) have been afforded in accordance with the age estimation methodologies employed (AlQahtani et al. 2010; Scheuer & Maclaughlin-Black 1994). Individuals considered to show evidence of growth disruption are those where dental and skeletal age estimates and ranges do not overlap.

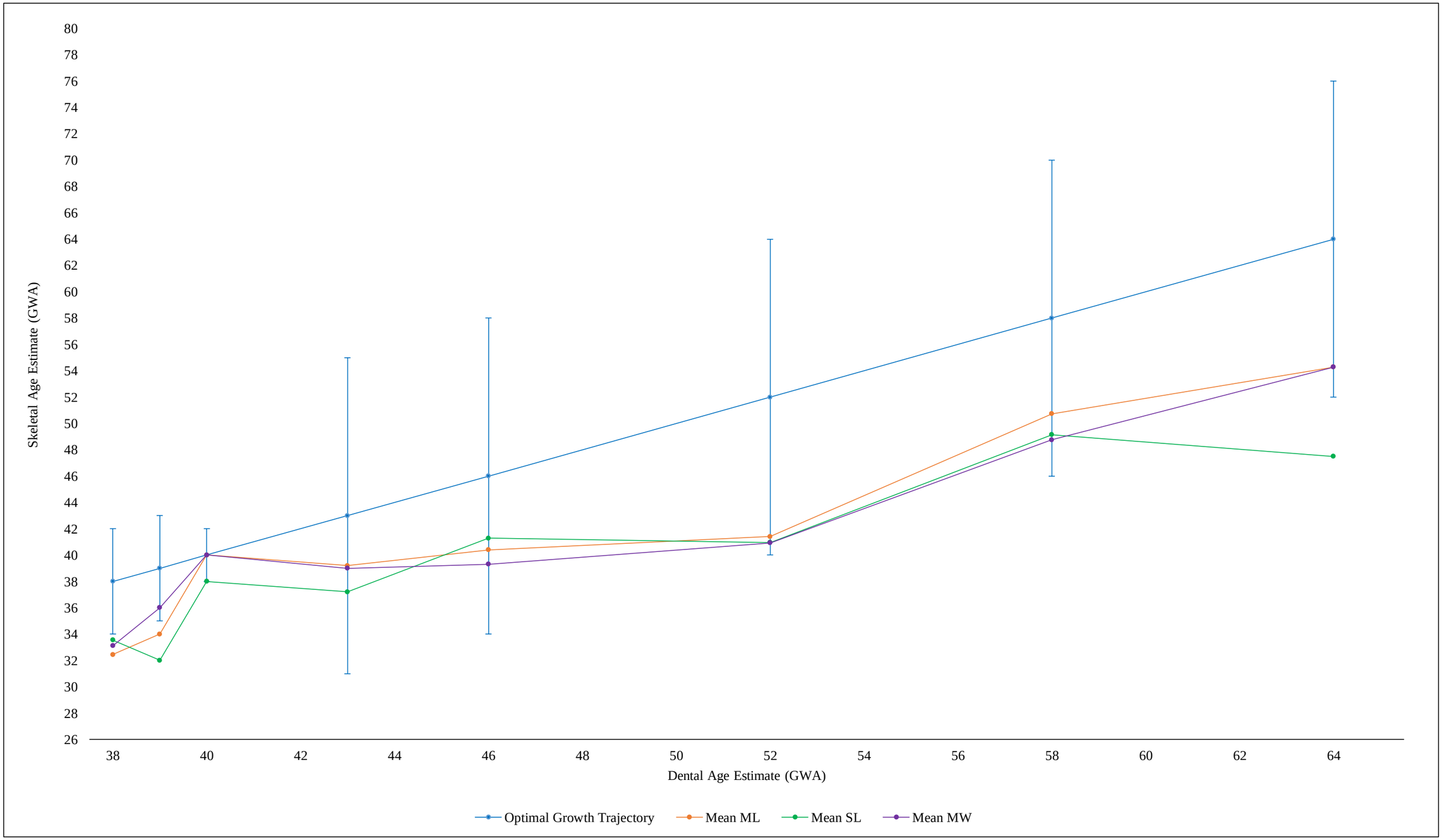

Figure 5. Mean pars basilaris age estimates (GWA) plotted by dental age. Optimal growth trajectory represents the expected growth profile if dental and skeletal age estimates were found to be identical for each age group. Error bars in accordance with dental age estimates have been plotted for optimal growth trajectory to highlight that average skeletal ages typically fall outside of these error ranges.

TABLE 6.1 Number and percentage of individuals observed with cranial and postcranial pathology given by archaeological sample.

Sample	Total *N*	Postcranial		Cranial	
		Observed *N*	Affected *N* (%)	Observed *N*	Affected *N* (%)
Broadgate	21	21	3 (14)	17	12 (71)
Chelsea Old Church	7	6	1 (17)	7	2 (29)
St Bride's Lower	52	52	10 (19)	46	39 (85)
Royal London Hospital	7	7	2 (29)	4	2 (50)
St Benet Sherehog	19	19	10 (53)	16	12 (75)
Crossbones	58	58	24 (41)	54	48 (89)
St Thomas' Hospital	5	3	2 (67)	5	3 (60)

TABLE 6.2 Cranial vault bones by number observed and percentage affected by socioeconomic status. Pathological lesions to each vault element have been documented by location (endo- or ectocranial) and by severity (Grade 1, 2 or 3). Percentages for location and severity have been calculated using only those affected. Occasionally percentages for location and severity by each status group total over 100%. This is because some individuals showed both endo- and ectocranial pathological changes within a single skeletal element and/or had multiple severity scores.

Sample Status	Frontal Bone							Parietal Bone							Occipital Bone						
	Observed (*N*)	Affected (%)	Endocranial (%)	Ectocranial (%)	Severity (%)			Observed (*N*)	Affected (%)	Endocranial (%)	Ectocranial (%)	Severity (%)			Observed (*N*)	Affected (%)	Endocranial (%)	Ectocranial (%)	Severity (%)		
					1	2	3					1	2	3					1	2	3
High	2	100	100	50	50	100	0	2	100	100	0	50	50	0	4	50	100	0	0	100	0
Middle	6	100	100	0	17	83	0	7	100	86	14	0	100	14	14	57	100	0	38	63	0
Low	85	99	99	6	20	67	19	75	99	100	1	30	68	4	109	69	100	0	28	68	5

TABLE 6.3 Postcranial long bones by number observed and percentage affected by socioeconomic status. Pathological lesions to each long bone have been documented by type of lesion (NBF, Metaphyseal Expansion, Morphological Change (e.g. bowing)) and by severity (Grade 1, 2 or 3). Percentages for type of lesion and severity have been calculated using only those affected. Occasionally percentages for lesion type and severity by each status group total over 100%. This is because some individuals showed both multiple types of pathological changes within a single skeletal element and/or had multiple severity scores.

Sample Status	Humerus								Femur								Tibia							
	Observed (*N*)	Affected (%)	NBF (%)	Metaphyseal (%)	Morphological (%)	Severity (%)			Observed (*N*)	Affected (%)	NBF (%)	Metaphyseal (%)	Morphological (%)	Severity (%)			Observed (*N*)	Affected (%)	NBF (%)	Metaphyseal (%)	Morphological (%)	Severity (%)		
						1	2	3						1	2	3						1	2	3
High	6	0	-	-	-	-	-	-	3	33	0	100	0	0	100	0	3	33	100	100	0	0	100	0
Middle	15	27	100	0	0	0	100	0	13	31	75	25	25	25	75	0	9	78	86	14	14	0	100	0
Low	118	12	79	36	50	28	71	14	111	19	67	52	10	5	90	24	101	26	81	27	12	15	73	19

TABLE 6.4 Results of chi-squared analysis (X^2) of pathological lesions by both socioeconomic status and by archaeological sample. P results in bold are those which are statistically significant.

	Frontal Bone			Parietal Bone			Occipital Bone			Humerus			Femur			Tibia		
	X^2	d.f.	*p*	X^2	d.f.	*p*	X^2	d.f.	*p*	X^2	d.f.	*p*	X^2	d.f.	*p*	X^2	d.f.	*p*
Sample Status	8.564	2	**0.014**	3.564	2	0.164	2.812	2	0.24	2.868	2	0.192	0.406	2	0.826	3.356	2	0.223
Archaeological Sample	21.856	5	**0.0001**	16.327	5	**0.005**	5.77	5	0.312	10.66	5	**0.033**	5.313	5	0.255	9.959	5	0.071

Table 6.2. demonstrates that of the cranial vault elements, the frontal bone and parietal bone are more commonly affected that the occipital bone. However, all three elements have a much greater percentage of endocranial to ectocranial lesions. Severity of grade two is seen to be the most prevalent for the individuals assessed, though interestingly the low status individuals typically have more lesions of severity three than either the middle or high status individuals. This pattern is substantiated from pathological assessment of the postcranial long bones (Table 6.3), where only low status individuals have recorded lesions of grade three severity. Analysis suggests that the tibia is the most commonly affected element within all social status groups, though most commonly affected in the middle class individuals. New bone formation has been identified as the primary pathological lesion recorded within these long bones, however, metaphyseal expansion is also prevalent, particularly within the low status sample. Limited statistically significant associations were found between social status and prevalence of pathological lesions (Table 6.4). However, when considered by individual archaeological samples, multiple statistically significant associations emerge. This may suggest that combining archaeological samples by status masks the individual differences between the samples.

Discussion:

Non-adults, particularly those of the youngest age categories (e.g. fetal, perinatal and infant individuals) are widely considered to be the most vulnerable to adverse environmental conditions and experiences (Humphrey 2000a; Newman & Gowland 2017). A consequence of age, both an under-developed immune system (Rogers 1997; Perry 2006; Halcrow and Tayles 2008), and a total reliance on others for care and wellbeing both pre- and postnatally (Lewis 2017a), makes them the most physiologically susceptible members of a community to health and growth disruption. A wealth of intrinsic and extrinsic factors are known to regulate growth and health, and result in adverse birth and life course outcomes (Goodman & Armelagos 1988; Goodman *et al.* 1988; Bush & Zvelebil 1991). Though the reflection of detrimental and limiting factors on the skeletal remains of adults and older children have widely been considered, lack of studies regarding the youngest age categories prevails (Halcrow & Tayles 2008; Halcrow *et al.* 2017; Lewis 2017c).

With recent development of the DOHaD hypothesis, the intrinsic links between maternal and infant wellbeing, and the sociocultural and environmental conditions of pre- and postnatal life

have become of paramount consideration (Barker *et al.* 2002; Agarwal 2016). Ultimately then, fetal, perinatal and infant growth and health status is a reflection of the interaction between both environmental and genetic factors. The intrauterine environment is one which the fetal offspring has limited regulation of, being reliant entirely upon the mother for nutritional and immunological buffering against stressful exposures (Bateson *et al.* 2004; Barker *et al.* 2012; Boersma & Tamashiro 2015). Consequently, identification of health and growth disruption within these young individuals signals an adverse pre- and/or postnatal environment.

Assessment of skeletal growth profiles for the 169 post-Medieval individuals analysed from post-Medieval London has revealed that growth disruption was present within all samples. Consideration of dental and skeletal age estimates has revealed a varying pattern of growth disruption, seemingly dependent on the skeletal element considered. Diaphyseal lengths of long bones have been shown to consistently under-age individuals compared to dental age, though the *pars basilaris* appears more comparable to dental age-at-death estimates. The *pars basilaris* is typically considered to be a robust bone of the base of the cranium (Redfield 1970). The cranial base, is one of the most complex skeletal structures, and first appears around the fourth gestational week (Scheuer & Black 2000a; St. Jacques & Helms 2003). It is known that the body prioritises growth of particular skeletal and soft-tissue structures, with the brain sitting at the top of this physiological hierarchy (Barker *et al.* 2012; Agarwal 2016; Said-Mohamed *et al.* 2018). This prioritisation of the brain requires the cranial bones to be equally adequately developed (Karsenty & Kronenberg 2003). Therefore, the speed of growth within bones of the cranium is considered to be a reflection of the rapidity of brain growth during this age (Scheuer & Black 2000b; Lewis 2007). In fact, infants dedicate up to 87% of their resting metabolic rate to brain development (Bogin 2001; 2012; Said-Mohamed *et al.* 2018), thus, skeletal growth of the cranium must coincide with this prioritisation. When growth disruption is experienced, there is often a trade-off between skeletal structures (Barker *et al.* 2012; Said-Mohamed *et al.* 2018). Typically, longitudinal growth of the long bones is expended for the benefit of the brain and bones of the cranium (Aiello & Wells 2002; Kuzawa *et al.* 2014; Sandman *et al.* 2016; Said-Mohamed *et al.* 2018). This is because energetic resources (e.g. nutrition) are redistributed with certain bodily structures prioritised, typically resulting in the slowing and disruption of skeletal growth (Agarwal 2016). Findings from this study support the conclusion that the *pars basilaris* is prioritised in regards to growth, and is found to be more robust against environmental changes. Hence, the *pars*

basilaris is less likely to reflect evidence of growth disruption, and therefore, more likely to align with dental age-at-death estimates. Consequently, when individuals were considered discretely by dental and *pars basilaris* age, results were found to show parity. Therefore, the growth and development of both dentition and the *pars basilaris* is considered to be resilient against environmental stressors.

Assessment of diaphyseal growth of the long bones shows a much more variable growth profile, with average age estimates, by dental group, almost all falling significantly below dental age. Individuals within the 64 GWA dental category show a diverging growth trajectory between femoral, humeral and tibial elements. Though small sample sizes inhibit the discussion of these findings, it is possible that these skeletal elements are each adopting various growth trajectories. Though growth is known to vary between element, in terms of the growth increment afforded each week/month (e.g. Issel 1986), growth profiles should result in all elements following a similar trajectory. Variation in growth, whereby one element appears to be disrupted or limited, is often considered to be a reflection of poor environment. The bones of the lower limb are considered to have more sensitive growth profiles, as they are some of the most rapidly growing bones of the body (Lewis 2002a). In particular, the tibia has commonly been considered to be the long bone element which shows increased variability and disruption when stress is experienced (Pomeroy *et al.* 2012). Though limited, findings of growth disruption within this study supports these previous conclusions, showing the tibia often reflects the greatest growth disruption. Furthermore, the tibia has also been identified within this study as the long bone which shows the highest prevalence rates of pathology (Table 6.3), corroborating the assumption that this bone is the most sensitive to growth and health stress.

Increased evidence of growth disruption in the older age categories (58-64 GWA) may be indicative of a postnatal drop off in growth. The ability for a mother, regardless of her own health status, to buffer the child from environmental and external factors is greater *in utero* than postnatally (Gowland 2015) – though passive immunity and nutritional buffering can be afforded from breast milk (Eisenberg *et al.* 2017; Lewis 2017b). Interestingly, no individuals dentally aged to be 46 GWA show any evidence of growth disruption, though individuals dentally aged to be both younger and older do. However, Cross Bones is the only site where perinatal individuals (38 and 39 GWA) were found to show growth disruption. Therefore, growth disruption may be suspected to be present in individuals where postnatal buffering is

insufficient to protect them from the extensive pathogenic environment into which they were born. Conversely, those showing prenatal growth disruption represent the most fundamentally deprived, where maternal regulation of the intrauterine environment is highly compromised as a result of deleterious stressors experienced. Thus, those aged 46 GWA may represent those who were able to be buffered to some extent both pre- and postnatally, evident in the lack of growth disruption, despite growth profiles beginning to fall during this age category. However, ultimately those aged 46 GWA were still individuals who succumbed to death, though this may have been so rapid that minimal changes to the skeleton can be observed.

Growth disparities within infants and older children have been widely documented, particularly in regards to varying socioeconomic status and poverty factors (Sinclair 1985). Results of this study suggest that socioeconomic and environmental impacts on growth can be traced in individuals of much younger ages, and even within pre- and perinatal individuals. This study has shown that individuals aged dentally to be as young as 38 GWA show evidence of growth disruption. Growth disruption is a cumulative process, whereby evidence of disruption increases as the individual continues to be exposed to the detrimental stressor. For significant growth changes to occur in individuals as young as 38 GWA, interpretations of chronic exposure to stress are supported.

Evidence from individual assessment of growth disruption also identified Cross Bones and St Bride's Lower as having the most individuals showing prevalence of growth disruption (Table 5.2). Although when considered by percentage of overall sample St Benet Sherehog is found to have the highest prevalence rate, this is likely a product of the small sample sizes considered. In contrast, both Cross Bones and St Bride's Lower have larger samples of individuals, with around ~25-30% of individuals showing growth disruption. Correlation of these disruptions with evidence of pathological lesions shows that Cross Bones and St Bride's Lower have the two highest prevalence rates for cranial pathology (Table 6.1). Furthermore, if findings from St Thomas' Hospital are overlooked, due to small sample size, Cross Bones equally has the most evidence for postcranial pathological lesions. Consideration of growth disruption by social status (Table 5.3) suggests that the middle status individuals show the highest frequency of growth disruption, followed by the low status individuals. However, when individual growth disruption is considered (Fig. 3), those with the greatest evidence of growth disruption (the biggest differences in gestational weeks between skeletal and dental age estimates), are primarily from the Cross Bones and St.

Bride's Lower samples. This suggests that although individuals from the middle status sample show greatest frequency of growth disruption, individuals from the low status samples have the severest evidence of growth disruption.

Pathological lesions corroborate this assumption, showing that the low status samples of Cross Bones and St. Bride's Lower typically have both the highest frequency of, and severest, pathological changes. Despite the prevalence of pathological changes in all samples, with similarly high prevalence rates in the middle status individuals, the severity of changes in the low status samples supports suggestions that disease exposure and susceptibility is increased in those of the lowest social strata. In fact, Southwark, where Cross Bones is located, has been described as *'…nurseries…of the begging poor that swarm within the City*' (Beier 1978), whilst mortality books from the parish of St Bride's reveal that the first year of life was the most perilous (Forbes 1972). Diseases of poverty are typically those associated with nutritional deficiency as a result of reduced food intake and a limited diversity of available foodstuffs (Dowler & Dobson 1997). Food has been suggested to be the most flexible in terms of household expenditure, and thus often the variable most compromised on (Dowler & Dobson 1997). Only those of low status were found to have postcranial lesions recorded within the grade three severity category, and also showed high rates of metaphyseal expansion. These changes are consistent with diseases of nutritional deficiency.

Vitamin C and vitamin D deficiency are the most commonly considered within bioarchaeological literature, however, a diverse range of nutrients and vitamins could be the underlying cause of such changes. Interpretations of vitamin D deficiency can be supposed due to the bowing identified within some of the limb bones assessed (N=10). Though these individuals are too young to be weight-bearing, intrauterine restriction may be responsible for these changes (Abbott 1901). Indeed, similar changes to those observed within this study have been identified clinically in cases of congenital rickets (Innes *et al.* 2002; Anatoliotaki *et al.* 2003). Consequently, such large expansion at the metaphyses, combined with extensive expansion of the trabecular bone structure, suggests that many of the individuals, and subsequently their mothers, were chronically vitamin D deficient. Similarly, scurvy has been identified clinically within fetal and perinatal individuals (Besbes *et al.* 2010) and is considered to occur in individuals where there is poor maternal intake of fresh fruit and vegetables (Brickley & Ives 2006). Skeletal changes consistent with vitamin C deficiency are expansion of the metaphyses and periosteal NBF as a consequence of weakened blood

vessels which easily rupture and haemorrhage (Aufderheide & Rodríguez-Martín 1998; Brickley & Ives 2006; Besbes *et al.* 2010). Vitamin C deficiency is suggested to only be observable in the skeleton after ~6 months of chronic deficiency, though this time may be less in rapidly growing and remodelling bones of non-adults (Brickley & Ives 2006; Mays 2014). However, skeletal evidence for Vitamin C deficiency within the individuals analysed suggests a severe prenatal deficiency, likely as a consequence of maternal deficiency. Such changes would not be observable unless chronic malnutrition was being experienced, and is suggestive that these high prevalence rates of NBF, particularly within the lower status archaeological samples, are reflecting poor maternal health and a deprived dietary intake of sufficient vitamins and nutrients.

Urban centres, such as London, were known for their poverty, poor living and working conditions and heavy pollution (De Witte *et al.* 2016; Newman & Gowland 2017). Mortality rates within such places were extremely high and life expectancy was decreased (Storey 1992; Feinstein 1993). Urban environments of the post-Medieval period are considered to have been places where diseases flourished (Lewis 2002b). Diseases such as cholera, smallpox, measles, whooping cough, tuberculosis, scarlet fever and typhoid are considered to prosper in the deleterious environmental conditions or urban centres at this time (Forbes 1972; Lewis 2002b). Social inequalities have been found to increase disease emergence and spread between individuals (Farmer 1996). The severity of lesions identified within the low status samples assessed may corroborate an infectious origin of the stressors experienced. Furthermore, evidence for syphilis and tuberculosis have been found within the adult populations supporting evidence for a high pathogen filled environment within post-Medieval London (Museum of London: Wellcome Osteological Research Database). Eight individuals (Broadgate = 1, St Thomas' Hospital = 1, Cross Bones = 5, and Royal London Hospital = 1) have evidence of systemic pathological lesions, where new bone formation has been identified throughout the long bones and all recorded as being of grade three severity. These individuals may hence best reflect evidence for infectious disease. Significantly, all of these individuals are from the low status samples, except for the individual from the Royal London Hospital where status is unknown.

Social stratification is suggested to have altered individuals' susceptibility to disease, thus the lower your position within the social hierarchy, the greater the chance of health disruption (Beier 1978; Babones 2008). Multifactorial consequences of this increased predisposition and

susceptibility include living and working environments, diet, access to health care, and education and understanding of healthcare practices (Feinstein 1993; Dowler & Dobson 1997). However, some studies have suggested that there is an intrinsic link between immune function and social position, suggesting exposure to stressful early life environments can alter immunity and regulation of bodily responses to infections (Babones 2008). This means maternal stress, as a consequence of the given factors (e.g. living conditions, poor diet, limited healthcare), can regulate offspring immune response, predisposing individuals to an inability to initiate or maintain sufficient immune responses to disease. Individuals from Cross Bones and St Bride's Lower may therefore show severe pathological changes consistent with this inability, where individuals are unable to regulate and overcome stressors, and in turn again reflect the poor health status of maternal individuals within these populations.

A recent study by Newman and Gowland (2017) has considered older infants and children from some of the same samples in post-Medieval London. Their results also highlight Cross Bones as the sample showing greatest growth and health disruption as a consequence of increased morbidity and mortality risks (Newman & Gowland 2017). However, their findings also highlight the unexpected level of growth disruption in the high status population of Chelsea Old Church, which they attribute to the class defined child rearing practices of the time rather than poverty (Newman & Gowland 2017). Cultural practices of swaddling, staying indoors and the wearing of heavy clothes would have all limited exposure to sunlight and consequently, high status individuals were also commonly found to show evidence of rickets. For low status individuals, increased incidence of vitamin D deficiency are likely the result of living and working conditions, and heavy atmospheric pollution. Pregnancy-related cultural practices, as well as response, treatment and care of the child once born can all also be reflected in individual growth and health status (Finlay 2013; Satterlee Blake 2018; e.g. Wilkie 2013). Furthermore, class defined practices of pregnancy-related care must not be overlooked (Boulton 2000), as distinct differences in experiences for expectant mothers are likely between those of high and low social status.

Many low status women had to return to work as soon as possible after birth of offspring, as household economies often relied on both parents earning an income (Boulton 2000). This means that many children were not breastfed, and instead were left in the care of elderly parents, neighbours or siblings (Boulton 2000). Of course some children were not reared at

all by their family and were instead handed over to authorities (Boulton 2000), though it is suspected this was only intended to be a temporary measure for the majority of parents. However, it has been well established that pauper households were deliberately fragmented, in an attempt for survival (Boulton 2000). Postnatal health and growth of offspring likely reflects these survival strategies, particularly in regards to the lack of breastfeeding practices.

Breastfeeding practices have been found to be strongly regulated by social status (Fildes 1988; 1995; Nitsch *et al.* 2011; DeWitte *et al.* 2016). Perinates and infants who are artificially fed, rather than breastfed, have been found to have increased disease and mortality risks (Fildes 1995; DeWitte *et al.* 2016). Though wet-nursing was popular among the higher social strata, dry-feeding became popular throughout society (DeWitte *et al.* 2016). With dry feeding, perinates and infants were fed a mixture of grains, water, broth and milk (DeWitte *et al.* 2016). Breastmilk, particularly that of colostrum (the initial thick breastmilk available directly after birth), is important for both the nutritional and immunological wellbeing of the infant (Eisenberg *et al.* 2017; Lewis 2017b). Breastmilk enables the transfer of maternal antibodies, as well as triggering the individuals own immune system and functioning (DeWitte *et al.* 2016). Therefore, breastfeeding is the optimum feeding practice as it buffers the offspring from environmental stressors during a period when their own immune function is immature and precarious. Maternal breast milk is also known to have high concentrations of vitamin A, needed to sustain rapid growth in the postpartum environment (Fujita *et al.* 2017, 1-2). Therefore, restriction and withholding of this dietary resource through cultural feeding practices predisposes the offspring to increased disease susceptibility. Lower status women comprehensively adopted the practice of dry feeding, often as it meant they could return to work quickly and was cheap (DeWitte *et al.* 2016). Growth and health disruption, particularly in postnatal individuals from the low status samples assessed may reflect these detrimental feeding practices.

This study has identified individuals from the middling class, those from St Benet Sherehog, to show a reduction in both growth and health status. However, though this middle social class shows the highest frequency of both growth and health disruption, low status individuals show a greater severity to both variables. A higher frequency of individuals showing these changes may then instead be consistent with a greater survival rate for these middling class individuals. Despite being exposed, and suffering from these disruptions, wide-scale identification of these changes within the skeletal samples suggests that, despite

many individuals being exposed to detrimental conditions, many also survived long enough for skeletal changes to occur. Particularly because the severity is reduced in these middle status individuals it may be suggested that they are somehow able to buffer and cope with the detrimental stressors more readily. In contrast, low status individuals show significant evidence of growth disruption, with the biggest difference being over 20 GWA between skeletal and dental estimates. Pathological lesions for the low status individuals were also found, in general, to be more severe. Therefore, despite high prevalence rates of growth and health disruption within the middling class, severity of changes may be more enlightening as to the type and profusion of health stresses experienced.

The importance of a wealth of environmental factors for early life success and wellbeing cannot be downplayed. Disparity in growth between individuals in the first seven years of life has been purported as being almost completely environmentally regulated (Habicht *et al.* 1974). Johnston *et al.* (1976) corroborated this suggestion finding growth variation in between Guatemalan and European children was not found to be hereditary, but instead dependent on living environment. Furthermore, Feinstein (1993) emphasises that poverty above all factors is the most predominant factor regulating health, growth and mortality. Considering this in relation to the known deprived and deleterious living environments of post-Medieval London suggests that conditions experienced by mothers and their offspring pre- and postnatally were paramount in causing growth and health disruption. By the mid-19th century it is suspected that nearly half the population of England lived in urban centres (Schofield 1994). The overcrowding and lack of housing, particularly of sufficient sanitary condition, meant those living within these 'slums' were at higher risk of exposure to disease, infection and thus ultimately, death. Sever postnatal evidence of growth and health disruption is likely a consequence of individuals emerging into a heavily pathogen-loaded environment. Given the potential for an already reduced health and growth status as a result of maternal deprivation, lack of breastfeeding and polluted living conditions only appears to have exacerbated the levels of stress individuals were exposed to.

Despite clear evidence for health and growth disruption, no individuals, from any of the samples, or from any of the social status groups were dentally aged to be fetal. This may be significant in suggesting very few individuals were born, and died, prematurely. However, it is more likely to be a result of dental remains being unavailable for recovery and assessment in the younger individuals, or even that individuals who were born prematurely/still born,

were not buried within the cemetery samples. That is not to say these individuals do not exist within the samples, especially as these detrimental birth outcomes (e.g. prematurity/stillbirth) are strongly correlated with exposure to intrinsic and extrinsic stressors (e.g. Chiswick 1985; Goldenberg & Thompson 2003; Abu-Saad & Fraser 2010; Cussons-Read *et al.* 2012; Beaudrap *et al.* 2013; Fell *et al.* 2016; Melby *et al.* 2016). Instead some of these individuals may instead be reflected in the perinatal or infant individuals, where they subsequently went on to survive for a number of days/weeks afterwards. However, assessment of skeletal diaphyseal lengths revealed that the youngest age estimates generated for the femora, tibiae and humeri were 26, 25 and 22 GWA respectively. Regardless of the potential for skeletal growth disruption, analysis of skeletal remains suggests that premature and/or stillborn individuals are present within the sample. In fact, 40 individuals have femoral age estimates which fall into the fetal age range, with 41 individuals having tibial, and 43 individuals having humeral diaphyseal length measurements which generate fetal age-at-death estimates. Consequently, given the youngest skeletal age estimates generated from diaphyseal length measurements are between 20 and 30 GWA it is likely that these individuals were premature and/or still born regardless of what their dental development would have suggested. Significantly, of the 40 individuals with femoral measurements under 36 GWA, 35 are from the low status sample, three are from the middling group, whilst two are unknown. Similar patterns are observed for the tibiae and humeri, though one high status individual from Chelsea Old Church has the lowest humeral age estimate of 22 GWA. Consequently, these findings suggest that detrimental birth outcomes may show some correlation to social status within these samples.

Finally, despite genetic variation not being considered central to evidence of disruption, increasing research into epigenetics and phenotypic plasticity has established a link between early life stress and intergenerational consequences (Gowland 2015; Mays *et al.* 2017; e.g. Bateson *et al.* 2004). Ultimately, adverse early life experiences decrease longevity and increase frailty (Gowland 2015). Holland Jones (2005) highlights a critical point, acknowledging that adaptive phenotypic consequences become integral to future generations' genetic structure, meaning what was an adaptive response by one individual becomes the basis of '*future life history tactics'* in the offspring. This potentially predisposes offspring to (mal)adaptation in response to the adverse or beneficial environments experienced. Thus, the fetal, perinatal and infant individuals assessed do not represent a static representation of health and wellbeing for post-Medieval London. Conversely, the health and growth

disruption identified within these individuals may represent multigenerational, cumulative consequences of deprived living environments.

Conclusion

With modern infant mortality still known to be substantially reliant on a multitude of socially and environmentally regulated parameters (Marmot 2005), evidence of growth and health disruption, particularly within the samples of individuals from Cross Bones and St Bride's Lower, suggests that socioeconomic status was central in regulating adversity to health and growth. Levels of growth disruption were found to be statistically significant for all age groups suggesting a universally deprived pre- and postnatal environment. Evidence of such significant growth disruption, and high prevalence rates, particularly of cranial lesions, indicates that maternal health was fundamentally reduced to such an extent where buffering of the offspring had been minimal for a substantive period of time. Furthermore, consideration of long-term health consequences and epigenetic changes may mean these individuals reflect cumulative exposure to detrimental early life experiences. Parity between dental age-at-death estimates and those generated from the *pars basilaris* indicates that utilising these measurements as proxies for chronological age is applicable. Therefore, when dentition is absent and unavailable for assessment, it is suggested that metric analysis of the *pars basilaris* provides an adequate substitution. As such, the diaphyseal length of limb bones are indicated to be highly variable and severely altered in response to stressors, correlating with other published findings. Subsequently, diaphyseal lengths of long bones should no longer be used in generating age-estimates which will be used as proxies for chronological age.

Ultimately, assessment of growth and health has revealed that clear patterns of disruption can be identified in the very youngest members of past populations from post-Medieval London. These findings have substantiated ideas of a bounded experience between mother and off-spring, where maternal regulation of both intrinsic and extrinsic factors is central to infant health and wellbeing.

References

Abbott, F. C. 1901; Intrauterine Rickets. *The British Medical Journal,* Vol. 2 (2123): 597-599.

Abu-Saad, K. and Fraser, D. 2010; Maternal nutrition and birth outcomes. *Epidemiological Review,* Vol. 32: 5-25.

Agarwal, S. 2016; Bone Morphologies and Histories: Life Course Approaches in Bioarchaeology. *Yearbook of Physical Anthropology,* Vol. 159: 130-149.

Aiello, L. C. and Wells, J. C. K. 2002; Energetics and the evolution of the genus Homo. *Annual Review of Anthropology,* Vol. 31: 323–338.

AlQahtani, S. J. Hector, M. P. and Liversidge, H. M. 2014; Accuracy of Dental Age Estimation Charts: Schour and Massler, Ubelaker, and the London Atlas. *American Journal of Physical Anthropology,* Vol. 154: 70-78.

AlQahtani, S. J. Hector, M. P. and Liversidge, H. M. 2010; The London Atlas of Human Tooth Development and Eruption. *American Journal of Physical Anthropology,* Vol. 142: 481-490.

Anatoliotaki, M., Tsilimigaki, A., Tsekoura, T., Schinaki, A., Stefanaki, S. and Nikolaidou, P. 2003; Congenital rickets due to maternal vitamin D deficiency in a sunny island of Greece. *Acta Paediatrica,* Vol. 92: 389-391.

Armelagos, G. J. and Goodman, A. H. 1991; The concept of stress and its relevance to studies of adaptation in prehistoric populations. *Collegium Antropologicum,* Vol. 15: 45-58.

Aufderheide, A. and Rodríguez-Martín, C. 1998; *The Cambridge Encyclopedia of Human Paleopathology*. Cambridge: Cambridge University Press.

Babones, S. J. 2008; Income inequality and population health: Correlation and causality. *Social Science and Medicine,* Vol. 66: 1614-1626.

Bang, G. 1989; Age changes in teeth; developmental and regressive. *Age Markers in the Human Skeleton,* Vol. 1: 211-235.

Barker, D. J. P. 1994; *Mothers, Babies, and Disease in Later Life*. London: BMJ Publishing Group.

Barker, D. J. P. 1997; Maternal Nutrition, Fetal Nutrition, and Disease in Later Life. *Nutrition,* Vol. 13 (9): 807-813.

Barker, D. J. P. 2003; *The Best Start in Life: How a Woman's Diet Can Protect her Child from Disease in Later Life.* London: Century Books.

Barker, D. J. P. 2012; Developmental origins of chronic disease. *Public Health,* Vol. 126: 185-189.

Barker, D. J. P., Eriksson, J. G., Forsén, T. and Osmond, C. 2002; Fetal origins of adult disease: strength of effects and biological basis. *International Journal of Epidemiology,* Vol. 31: 1235-1239.

Barker, D. J. P., Lampl, M., Roseboom, T. and Winder, N. 2012; Resource allocation in utero and health in later life. *Placenta,* Vol. 33: 30-34.

Barker, D. and Osmond, C. 1986; Infant mortality, childhood nutrition, and ischaemic heart disease in England and Wales. *Lancet,* Vol. 8489: 1077–1081.

Bateson, P., Barker, D., Clutton-Brock, T., Deb, D., D'Udine, B., Foley, R. A., Gluckman, P., Godfrey, K., Kirkwood, T., Mirazón Lahr, M., McNamara, J., Metcalfe, N. B., Monaghan, P., Spencer, H. G. and Sultan, S. E. 2004; Developmental plasticity and human health. *Nature,* Vol. 430: 419-421.

Baxter, J. E. 2005; *The Archaeology of Childhood: Children, Gender, and Material Culture.* California: AltaMira Press.

Beaudrap, P., Turyakira, E., White, L. J., Nabasumba, C., Tumwebaze, B., Muehlenbachs, A., Guérin, P., Boum, Y., McGready, R. and Piola, P. 2013; Impact of malaria during pregnancy on pregnancy outcomes in a Ugandan prospective cohort with intensive malaria screening and prompt treatment. *Malaria Journal,* Vol. 12 (1): 139.

Beaumont, J., Geber, J., Power, N., Wilson, A., Lee-Thorpe, J. and Montgomery, J. 2013; Victims and Survivors: Stable Isotopes Used to Identify Migrants From the Great Irish Famine to 19th Century London. *American Journal of Physical Anthropology,* Vol. 150: 87-98.

Bekvalac 2007 *St Thomas' Hospital cemetery summary* [Online] [Accessed: November 2016] Available from: https://www.museumoflondon.org.uk/collections/other-collection-databases-and-libraries/centre-human-bioarchaeology/osteological-database/post-medieval-cemeteries/st-thomas-hospital-post-medieval

Beier, A. L. 1978; Social Problems in Elizabethan London. *The Journal of Interdisciplinary History,* Vol. 9 (2): 203-221.

Besbes, L. G., Hadded, S., Meriem, C. B., Golli, M., Najjar, M. F. and Guediche, M. N. 2010; Infantile Scurvy: Two Case Reports. *International Journal of Pediatrics,* Article ID 717518: 1-4.

Bocquet-Appel, J. P. and Masset, C. 1982; Farewell to palaeodemography. *Journal of Human Evolution,* Vol. 11: 321-333.

Boersma, G. J. and Tamashiro, K. L. 2015; Individual differences in the effects of prenatal stress exposure in rodents. *Neurobiology of Stress,* Vol. 1: 100-108.

Bogin, B. 1999; *Patterns of Human Growth* (2nd Edition). Cambridge: Cambridge University Press.

Bogin, B. 2001; *The Growth of Humanity*. New York: Wiley-Liss.

Bogin, B. 2012; *The Evolution of Human Growth.* In N. Cameron & B. Bogin (Eds.) *Human Growth and Development.* London: Elsevier: 287-324.

Bolaños, M. V., Manrique, M. C., Bolaños, M. J. and Briones, M. T. 2000; Approaches to chronological age assessment based on dental calcification. *Forensic Science International,* Vol. 110: 97-106.

Boulton, J. 2000; 'It Is Extreme Necessity That Makes Me Do This': Some 'Survival Strategies' of Pauper Households in London's West End During the Early Eighteenth Century. *International Review of Social history,* Vol. 45: 47-69.

Brickley, M. and Ives, R. 2006; Skeletal Manifestations of infantile Scurvy. *American Journal of Physical Anthropology,* Vol. 129: 163-172.

Brickley, M., Miles, A. and Stainer, H. 1999; *The Cross Bones Burial Ground, Redcross Way, Southwark, London: Archaeological Excavations (1991-1998) for the London Underground Limited Jubilee Line Extension Project* (MOLAS Monograph 3). London: Museum of London Archaeology Service.

Bush, H. and Zvelebil, M. 1991; Pathology and health in past societies: an introduction. In H. Bush and M. Zvelebil (Eds.) *Health in Past Societies: Biocultural interpretations of human skeletal remains in archaeological contexts* (BAR International Series 567). Oxford: BAR Publishing: 3-9.

Cameron, N. and Demerath, E. W. 2002; Critical Periods in Human Growth and Their Relationship to Diseases of Aging. *Yearbook of Physical Anthropology,* Vol. 45: 159-184.

Cardoso, H. F. V. 2007; Environmental Effects on Skeletal Versus Dental Development: Using a Documented Subadult Skeletal Sample to Test a Basic Assumption in Human Osteological Research. *American Journal of Physical Anthropology,* Vol. 132: 223-233.

Cattaneo, C. 1991; Direct genetic and immunological information in the reconstruction of health and biocultural conditions of past populations: a new prospect for archaeology. In H. Bush and M. Zvelebil (Eds.) *Health in Past Societies: Biocultural interpretations of human*

skeletal remains in archaeological contexts (BAR International Series 567). Oxford: BAR Publishing: 39-52.

Cavigelli, S. A. and Chaudhry, H. S. 2012; Social status, glucocorticoids, immune function, and health: can animal studies help us understand human socioeconomic-status-related health disparities? *Hormones and Behavior*, Vol. 62: 295–313.

Chiswick, M. L. 1985; Intrauterine growth retardation. *British Medical Journal,* Vol. 291: 845-848.

Chmurzynska, A. 2010; Fetal programming: link between early nutrition, DNA methylation, and complex diseases. *Nutrition Reviews,* Vol. 68 (2): 87-98.

Cowie, R. Bekvalac, J. and Kausmally, T. 2008; *Late 17th- to 19th-century burial and earlier occupation at All Saints, Chelsea Old Church, Royal Borough of Kensington and Chelsea* (MOLAS Study Series 18). London: Museum of London Archaeology Service.

Cussons-Read, M. E., Lobel, M., Carey, J. C., Kreither, M. O., D'Anna, K., Argys, L., Ross, R. G., Brandt, C. and Cole, S. 2012; The occurrence of preterm delivery is linked to pregnancy-specific distress and elevated inflammatory markers across gestation. *Brain, Behaviour and Immunity,* Vol. 26 (4): 650-659.

De la Rúa C., Izagirre, N., and Manzano, C. 1995; Environmental stress in a medieval population of the Basque country. *Homo*, Vol. 45: 268–289.

DeWitte, S. N., Hughes-Morey, G, Bekvalac, J. and Karsten, J. 2016; Wealth, health and frailty in industrial-era London. *Annals of Human Biology,* Vol. 43 (3): 241-254.

Dowler, E. A. and Dobson, B. M. 1997; Nutrition and poverty in Europe: an overview. *Proceedings of the Nutrition Society,* Vol. 56: 51-62.

Eisenberg, D. T. A., Borja, J. B., Hayes, M. G. and Kuzawa, C. W. 2017; Early life infection, but not breastfeeding, predicts adult blood telomere lengths in the Philippines. *American Journal of Human Biology,* Vol. 29 (Early View): 1-11.

Farmer, P. 1996; Social inequalities and emerging infectious diseases. *Emerging infectious Diseases*, Vol. 2 (4): 259-269.

Fazekas, I. G. and Kósa, F. 1978; *Forensic Foetal Osteology.* Budapest: Academic Press.

Feinstein, J. S. 1993; The Relationship between Socioeconomic Status and Health: A Review of the Literature. *The Milbank Quarterly*, Vol. 71(2): 279-322.

Fell, D. B., Savitz, D. A., Kramer, M. S., Gessner, B. D., Katz, M. A., Knight, M., Luteijn, J. M., Marshall, H., Bhat, N., Gravett, M. G., Skidmore, B. and Ortiz, J. R. 2016; Maternal influenza and birth outcomes: systematic review of comparative studies. *International Journal of Obstetrics and Gynaecology*, Vol. 124: 48-59.

Fildes, V. 1988; Wet nursing. New York: Basil Blackwell Ltd.

Fildes, V. 1995; The culture and biology of breastfeeding: an historical review of Western Europe. In: Stuart-Macadam, P., and Dettwyker, K. (Eds.) *Breastfeeding: biocultural perspectives.* Hawthorne: Aldine De Gruyter: 101-126.

Finlay, N. 2013; Archaeologies of the beginnings of life. *World Archaeology*, Vol. 45 (2): 207-214.

Floud, R., Wachter, K.W. and Gregory, A. 1990; *Height, health and history: nutritional status in the United Kingdom, 1750–1980*. Cambridge: Cambridge University Press.

Forbes, T. R. 1972; Mortality Books for 1820 to 1849 from the Parish of St. Bride, Fleet Street, London. *Journal of the History of Medicine*: 15-29.

Fowler, L. and Powers, N. 2012; *Doctors, Dissection and Resurrection Men: Excavations in the 19th-century burial ground of the London Hospital, 2006* (MOLA Monograph 62). London: Museum of London Archaeology.

Fujita, M., Lo, Y. J. and Brindle, E. 2017; Nutritional, inflammatory, and ecological correlates of maternal retinol allocation to breast milk in agro-pastoral Ariaal communities of northern Kenya. *American Journal of Human Biology,* Vol. 29 (Early View): 1-14.

Glover, V. 2015; Prenatal Stress and Its Effects on the Fetus and the Child: Possible underlying Biological Mechanisms. In M. C. Antonelli (Ed.) *Perinatal Programming of Neurodevelopment* (Volume 10): New York: Springer: 269-283.

Goldenberg, R. L & Thompson, C. 2003; The infectious origins of stillbirth. *American Journal of Obstetric Gynecology,* Vol. 189, No. 3: 861-873.

Goodman, A. H. and Armelagos, G. J. 1988; Childhood Stress and Decreased Longevity in a Prehistoric Population. *American Anthropologist,* Vol. 90 (4): 936-944.

Goodman, A. H. and Armelagos, G. J. 1989; Infant and Childhood Morbidity and Mortality Risks in Archaeological Populations. *World Archaeology,* Vol. 21 (2): 225-243.

Goodman, A.H. and Martin, D. L. 2002; Reconstructing health profiles from skeletal remains. In R. H. Steckel and J.C. Rose (Eds.) *The Backbone of History: Health and Nutrition in the Western Hemisphere.* Cambridge: Cambridge University Press:11-60.

Goodman, A. H., Thomas, R. B., Swedlund, A. C. and Armelagos, G. J. 1988; Biocultural Perspectives on Stress in Prehistoric, Historical, and Contemporary Population Research. *Yearbook of Physical Anthropology,* Vol. 31: 169-202.

Gowland, R. L. 2015; Entangled Lives: Implications of the Developmental Origins of Health and Disease Hypothesis for Bioarchaeology and the Life Course. *American Journal of Physical Anthropology,* Vol. 158, No. 4: 530-540.

Gowland, R. L. and Chamberlain, A. T. 2002; A Bayesian Approach to Ageing Perinatal Skeletal Material from Archaeological Sites: Implications for the Evidence for Infanticide in Roman-Britain. *Journal of Archaeological Science,* Vol. 79: 677-685.

Gustafson, G. and Koch, G. 1974; Age estimation up to 16 years of age based on dental development. *Odontologisk Revy*, Vol.25 (3): 297-306.

Habicht, J. P., Yarbrough, C., Martorell, R., Malina, R. M., and Klein, R. E. 1974; Height and weight standards for preschool children. *Lancet*, 1: 7858.

Halcrow, S. E. and Tayles, N. 2008; The Bioarchaeological Investigation of Childhood and Social Age: Problems and Prospects. *Journal of Archaeological Method and Theory*, Vol. 15: 190-215.

Halcrow, S. E., Tayles, N., and Elliot, G. E. 2017; The Bioarchaeology of Fetuses. In S. Han, T. K. Betsinger, and A. B. Scott (Eds.) *The Fetus: Biology, Culture, and Society.* Berghahn Books (*In Press*).

Halcrow, S. E., Tayles, N., Inglis, R. and Higham, C. 2012; Newborn twins from prehistoric mainland Southeast Asia: birth, death and personhood. *Antiquity*, Vol. 86: 838-852.

Halcrow, S. E. and Ward, S. M. 2017; Bioarchaeology of Childhood. In H. Montgomery (Ed.) *Oxford Bibliographies in Childhood Studies.* New York: Oxford University Press.

Halfon, N., Larson, K., Lu, M., Tullis, E. and Russ, S. 2014; Lifecourse Health Development: Past, Present and Future. *Maternal and Child Health Journal*, Vol. 18: 344-365.

Hammer, O., Harper, D. A. T. and Ryan, P. D. 2001; PAST Paleontological Statistics Software Package for Education and Data Analysis. [Online] [Accessed August 2016] Available from: http://palaeo-electronica.org/2001_1/past/issue1_01.htm

Harding, V. 2002; *The dead and the living in Paris and London: 1500-1670.* Cambridge: Cambridge University Press.

Heuzé, Y. and Cardoso, H. F. V. 2008; Testing the Quality of Nonadult Bayesian Dental Age Assessment Methods to Juvenile Skeletal Remains: The Lisbon Collection Children and Secular Trend Effects. *American Journal of Physical Anthropology,* Vol. 135: 275-283.

Hillson, S. W. 1979; Diet and dental disease. *World Archaeology,* Vol. 11; 147–62.

Hillson, S. W. 2005; *Teeth* (Second Edition). Cambridge: Cambridge University Press.

Holland Jones, J. 2005; Fetal Programming: Adaptive Life-History Tactics or Making the Best of a Bad Start? *Journal of Human Biology,* Vol. 17: 22-33.

Hoppa, R. D. and Fitzgerald, C. M. 1999; From head to toe: integrating studies from bones and teeth in biological anthropology. In. D. Hoppa and C. M. Fitzgerald (Eds.) *Human growth in the past: studies from bones and teeth*. Cambridge: Cambridge University Press: 1-32.

Humphrey, L. 2000a; Interpretations of the growth of past populations. In J. S. Derevenski (Ed.) *Children and Material Culture*. London: Routledge:193–205.

Humphrey, L. 2000b; Growth Studies of Past Population: An Overview and an Example. In M. Cox and S. Mays (Eds.) *Human Osteology in Archaeology and Forensic Science*. London: Greenwich Medical Media Ltd: 23-38.

Issel, E. P. 1985; Ultrasonic measurement of the growth of fetal limb bones in normal pregnancy. *Journal of Perinatal Medicine,* Vol. 13: 305-313.

Ives, R. and Humphrey, L. 2017; Patterns of long bone growth in a mid-19th century documented sample of the urban poor from Bethnal Green, London, UK. *American Journal of Physical Anthropology,* Vol. 163: 173-186.

Johnston, F. E., Wainer, H., Thissen, D., and MacVean, R. 1976; Hereditary and Environmental Determinants of Growth in Height in a Longitudinal Sample of Children and

Youth of Guatemala and European Ancestry. *American Journal of Physical Anthropology,* Vol. 44 (3): 469-476.

Jones H. 1991; *Preliminary Report of Archaeological Excavations at New London Bridge House, London Bridge Street, S.E.1.* Museum of London, Department of Great London Archaeology: London. (Unpublished).

Kamp, K. A. 2015; Children and their Childhoods: Retrospectives and Prospectives. *Childhood in the Past*, Vol. 8 (2): 161-169.

Karsenty, G. and Kronenberg, H. M. 2003; Postnatal Bone Growth: Growth Plate Biology, Modelling, and Remodeling. In F. H. Glorieux, J. M. Pettifor and H. Jüppner (Eds.) *Pediatric Bone: Biology and Diseases.* London: Academic Press: 119-133.

Kausmally, T. 2008 *St Bride's Lower churchyard cemetery summary* [Online] [Accessed: November 2016]
Available from:
https://www.museumoflondon.org.uk/collections/other-collection-databases-and-libraries/centre-human-bioarchaeology/osteological-database/post-medieval-cemeteries/st-brides-lower-post-medieval

King, S. E. and Ulijaszek, S. J 1999; Invisible insults during growth and development: contemporary theories and past populations. In R. D. Hoppa and C. M. Fitzgerald (Eds.) *Human growth in the past: studies from bones and teeth*. Cambridge: Cambridge University Press: 161- 182.

Kuzawa, C. W. 2012; Early environments, developmental plasticity, and chronic degenerative disease. In N. Cameron and B. Bogin (Eds.) *Human Growth and Development* (2nd Edition). London: Elsevier: 325-341.

Kuzawa, C. W., Chugani, H. T., Grossman, L. L., Lipovich, L., Muzik, O., Hof, P. R., Wildman, D. E., Sherwood, C. C., Leonard, W. R. and Lange, N. 2014; Metabolic costs and evolutionary implications of human brain development. *PNAS,* Vol. 111 (36): 13010-13015.

Kuzawa, C. W. and Quinn, E. A. 2009; Developmental Origins of Adult Function and Health: Evolutionary Hypotheses. *Annual Review of Anthropology,* Vol. 38: 131-147.

Lewis, M. E. 2002a; The impact of industrialisation: comparative study of child health in four sites from medieval and post-medieval England (AD 850–1859). *American Journal of Physical Anthropology,* Vol. 119: 211–223.

Lewis, M. E. 2002b; *Urbanisation and Child Health in Medieval and Post-Medieval England.* British Archaeological Reports British Series 229. Oxford: Archaeopress.

Lewis, M. E. 2007; *The Bioarchaeology of Children: Perspectives from Biological and Forensic Anthropology.* Cambridge: Cambridge University Press.

Lewis, M. E. 2017a; *Paleopathology of Children: Identification of Pathological Conditions in the Human Skeletal Remains of Non-Adults.* London: Academic Press.

Lewis, M. E. 2017b; Childcare in the Past: The Contribution of Palaeopathology. In L. Powell, W. Southwell-Wright, and R. Gowland (Eds.) *Care in the Past: Archaeological and Interdisciplinary Perspectives.* Oxford: Oxbow Books: 23-37.

Lewis, M. E. 2018; Fetal Paleopathology: An Impossible Discipline? In S. Han, T. K. Betsinger, and A. B. Scott (Eds.) *The Fetus: Biology, Culture, and Society.* New York: Berghahn Books: 112-131.

Lewis, M. E. and Gowland, R. L. 2007; Brief and Precarious Lives: Infant Mortality in Contrasting Sites from Medieval and Post-Medieval England (AD 850-1859). *American Journal of Physical Anthropology,* Vol. 134: 117-129.

Lewis, M. E. and Roberts, C. 1997; Growing Pains: the interpretation of Stress Indicators. *International Journal of Osteoarchaeology,* Vol. 7: 581-586.

Lindert, P. 1994; Unequal living standards. In R. Floud and D. McCloskey (Eds.) *The economic history of Britain since 1700*. Cambridge: Cambridge University Press: 357–386.

Liversidge, H. M. and Molleson, T. 2004; Variation in Crown and Root Formation and Eruption of Human Deciduous Teeth. *American Journal of Physical Anthropology,* Vol. 123: 172-180.

Luo, Z. C., Fraser, W. D., Julien, P., Deal, C. L., Audibert, F., Smith, G. N., Xiong, X. and Walker, M. 2006; Tracing the origins of 'fetal origins' of adult diseases: Programming by oxidative stress? *Medical Hypotheses,* Vol. 66: 38-44.

Marmot, M. 2005; Social determinants of health inequalities. *Lancet,* Vol. 365: 1099-1104.

Martorell, R. and Habicht. J. P. 1986; Growth in early childhood in developing countries. In F. Falkner and J. Tanner (Eds.) *Human Growth: methodology ecological, genetic, and nutritional effects on growth*. New York: Plenum Press: 241-262.

Massler, M., Schour, I. and Poncher, H. G. 1941; Developmental Pattern of the Child as Reflected in the Calcification Pattern of the Teeth. *American Journal of Diseases of Children,* Vol. 62: 33-67.

Mayhew, H. 1985; *London labour and the London poor. Selections made and introduced by Neuberg, V.* London: Penguin.

Mays, S. 1993; Infanticide in Roman Britain. *Antiquity* 67: 883–8.

Mays, S. 2014; The palaeopathology of scurvy in Europe. *International Journal of Paleopathology,* Vol. 5: 55-62.

Mays, S., Gowland, R., Halcrow, S. and Murphy, E. 2017; Child Bioarchaeology: Perspectives on the Past 10 Years. *Childhood in the Past,* Vol. 10 (1): 38-56.

Melby, M. K., Yamada, G. and Surkan, P. J. 2016; Inadequate Gestational Weight Gain Increases Risk of Small-for-Gestational-Age Term Births in Girls in Japan: A Population-Based Cohort Study. *American Journal of Human Biology,* Vol. 28: 714-720.

Mikulski, R. 2007 *Cross Bones burial ground summary* [Online] [Accessed: November 2016] Available from: https://www.museumoflondon.org.uk/collections/other-collection-databases-and-libraries/centre-human-bioarchaeology/osteological-database/post-medieval-cemeteries/cross-bones-post-medieval

Miles, A. and Conheeney, J. 2005; *A Post-medieval population from London: Excavations in the St Bride's Lower Churchyard 75-82 Farringdon Street, City of London*. London (Unpublished).

Moorrees, C. F. A. Fanning, E. A. & Hunt, E. E. 1963a; Formation and Resorption of Three Deciduous Teeth in Children. *American Journal of Physical Anthropology,* Vol. 21: 205-213.

Moorrees, C. F. A. Fanning, E. A. and Hunt, E. E. 1963b; Age Variation of Formation Stages for Ten Permanent Teeth. *Journal of Dental Research,* Vol. 42: 1490-1502.

Mortier, G. R. and Vanden Berghe, W. 2012; Genomics, Epigenetics and Growth. In N. Cameron and B. Bogin (Eds.) *Human Growth and Development* (2nd Edition). London: Elsevier: 153-172.

Museum of London 2009 *Chelsea Old Church (Post-Medieval) cemetery summary* [Online] [Accessed: November 2016]
Available from:
https://www.museumoflondon.org.uk/collections/other-collection-databases-and-libraries/centre-human-bioarchaeology/osteological-database/post-medieval-cemeteries/chelsea-old-church-post-medieval

Museum of London 2015 *Broadgate (Post-Medieval) cemetery summary* [Online] [Accessed: November 2016]
Available from:
https://www.museumoflondon.org.uk/collections/other-collection-databases-and-libraries/centre-human-bioarchaeology/osteological-database/post-medieval-cemeteries/broadgate-post-medieval

Museum of London *Wellcome Osteological Research Database* [Online] [Accessed July 2017] Available from: https://www.museumoflondon.org.uk/collections/other-collection-databases-and-libraries/centre-human-bioarchaeology/osteological-database

Newman, S. L. and Gowland, R. L. 2017; Dedicated Followers of Fashion? Bioarchaeological Perspectives on Socio-Economic Status, Inequality, and Health in Urban Children from the Industrial Revolution (18th-19th Century) England. *International Journal of Osteoarchaeology,* Vol. 27 (2): 217-229.

Nicholas, S. and Steckel, R. H. 1991; Heights and living standards of English workers during the early years of industrialization, 1770–1815. *Journal of Economic History*, Vol. 51: 937–957.

Nitsch, E.K., Humphrey, L.T. and Hedges, R. E. M. 2011: Using stable isotope analysis to examine the effect of economic change on breastfeeding practices in Spitalfields, London, UK. *American Journal Physical Anthropology*, Vol. 146: 619-628.

Ogden, A. R., Pinhasi, R. and White, W. J. 2007; Gross enamel hypoplasia in molars from subadults in a 16th-18th century London graveyard. *American Journal of Physical Anthropology*, Vol. 133: 957-966.

Perry, M. A. 2006; Redefining childhood through bioarchaeology: Toward an archaeological and biological understanding of children in Antiquity. *Archaeological Papers of the American Anthropological Association,* Vol. 15 (1): 89-111.

Phelan, J. C., Link, B. G. and Tehranifar, P. 2010; Social conditions as fundamental causes of health inequalities: theory, evidence, and policy implications. *Journal of Health and Social Behaviour*, Vol. 51: S28–S40.

Picard, L. 2005; Victorian London. London: Orion Books.

Pinhasi, R., Shaw, P., White, B. and Ogden, A. R. 2006; Morbidity, rickets and long-bone growth in post-medieval Britain - a cross-population analysis. *Annals of Human Biology*, Vol. 33: 372-398.

Pomeroy, E., Stock, J. T., Stanojevic, S., Miranda, J. J., Cole, T. J., and Wells, J. C. K. 2012; Trade-Offs in Relative Limb Length among Peruvian Children: Extending the Thrifty Phenotype Hypothesis to Limb Proportions. *Plos One*¸ Vol. 7 (12): 1-10.

Redfern, R. 2003; Sex and the City: A biocultural investigation into female health in Roman Britain. In G. Carr, E. Swift and J Weekes (Eds.) *TRAC 2002 Proceedings of the Twelfth Annual Theoretical Roman Archaeology Conference.* Oxford: Oxbow Books.

Redfield, A. 1970; A New Aid to Aging Immature Skeletons: Development of the Occipital Bone. *American Journal of Physical Anthropology,* Vol. 33: 207-220.

Reitsema, L.J. and McIlvaine, B. K. 2014; Reconciling "stress" and "health" in physical anthropology: What can bioarchaeologists learn from the other subdisciplines? *American Journal of Physical Anthropology*, Vol. 155: 181-185.

Robb, J., Bigazzi, R., Lazzarini, L., Scarsini, C. and Sonego, F. 2001; Social 'status' and biological 'status': a comparison of grave goods and skeletal indicators from Pontecagnano. *American Journal of Physical Anthropology*, Vol. 115: 213–222.

Roberts, C. A. 2009; *Human Remains in Archaeology*. York: Council for British Archaeology.

Robertson, T., Batty, G. D., Der, G., Fenton, C., Shiels, P. G. and Benzeval, M. 2013; Is socioeconomic status associated with biological aging as measured by Telomere Length? *Epidemiological Review*, Vol. 35: 98–111.

Rogers, A. 1997; Vulnerability, health and healthcare. *Journal of Advanced Nursing,* Vol. 26: 65-72.

Said-Mohamed, R., Pettifor, J. M. and Norris, S. A. 2018; Life History theory hypotheses on child growth: Potential implications for short and long-term child growth, development and health. *American Journal of Physical Anthropology,* Vol. 165: 4-19.

Sánchez Romero, M. 2017; Landscapes of Childhood: Bodies, Places and Material Culture. *Childhood in the Past,* Vol 10. (1): 16-37.

Sandman, C. A., Glynn, L. M. and Davis, E. P. 2016; Neurobehavioral Consequences of Fetal Exposure to Gestational Stress. In N. Reissland and B. S. Kisilevsky (Eds.) *Fetal Development.* Switzerland: Springer International Publishing: 229-265.

Satterlee Blake, K. A. 2018; The Biology of the Fetal Period: Interpreting Life from Fetal Skeletal Remains. In S. Han, T. K. Betsinger, and A. B. Scott (Eds.) *The Fetus: Biology, Culture, and Society.* New York: Berghahn Books: 34-58.

Saunders, S. R. and Hoppa, R. D. 1993; Growth Deficit in Survivors and Non-Survivors: Biological Mortality Bias in Subadult Skeletal Samples. *Yearbook of Physical Anthropology,* Vol. 36: 127-151.

Schell, L. M. 1997; Culture as a Stressor: A Revised Model of Biocultural Interaction. *American Journal of Physical Anthropology,* Vol. 102: 67-77.

Scheuer, L. and Black, S. 2000a; *Developmental Juvenile Osteology*. London: Academic Press.

Scheuer, L. and Black, S. 2000b; Development and Ageing of the Juvenile Skeleton. In M. Cox and S. Mays (Eds.) *Human Osteology in Archaeology and Forensic Science*. London: Greenwich Medical Media Ltd: 9-21.

Scheuer, L. and Maclaughlin-Black, S. 1994; Age Estimation from the Pars Basilaris of the Fetal and Juvenile Occipital Bone. *International Journal of Osteoarchaeology,* Vol. 4: 377-380.

Scheuer, L. Musgrave, J. H. & Evans, S. P. 1980; The estimation of late fetal and perinatal age from limb bone length by linear and logarithmic regression. *Annals of Human Biology*, 7 (3): 257-265.

Schofield, J. and Maloney, C. 1998; *Archaeology in the City of London, 1907-1991: A guide to records of excavations by the Museum of London and its predecessors***.** London: Museum of London.

Sinclair, D. 1985; *Human Growth After Birth* (4th Edition). Oxford: Oxford University Press.

Slack, J. M. W. 1991; *From Egg to Embryon: Regional Specification in Early Development* (2nd Edition). Cambridge: Cambridge University Press.

St. Jacques, B. and Helms, J. A. 2003; Prenatal Bone Development: Ontogeny and Regulation. F. H. Glorieux, J. M. Pettifor and H. Jüppner (Eds.) *Pediatric Bone: Biology and Diseases.* London: Academic Press: 77-117.

Steckel, R. H. 2009; Heights and human welfare: recent developments and new directions. *Explorations in Economic History*, Vol. 46: 1-23.

Stinson, S. 2000; Growth variation: biological and cultural factors. In S. Stinson, B. Bogin, R. Huss-Ashmore and D. H. O'Rourke (Eds.) *Human biology: an evolutionary and biocultural perspective*. New York: Wiley-Liss: 434-438.

Storey, R. 1992; Preindustrial urban lifestyle and health. *MASCA Research Papers in Science and Archaeology*, Vol. 9:33–41.

Tanner, J. 1994; Growth in height as a mirror of standard of living. In J. Komlos (Ed.) *Stature, living standards, and economic development: essays in anthropometric history.* Chicago, IL: University of Chicago Press: 1-9.

Temple, D. H. and Goodman, A. H. 2014; Bioarchaeology has a “health” problem: Conceptualizing “stress” and “health” in bioarchaeological research. *American Journal of Physical Anthropology,* Vol. 155: 186-191.

Thorsell, A. and Nätt, D. 2016; Maternal stress and diet may influence affective behaviour and stress-response in offspring via epigenetic regulation of central peptidergic function. *Environmental Epigenetics,* Vol. 2 (3): 1-10.

Utczas, K., Muzsnai, A., Cameron, N., Zsakai, A. and Bodzsar, E. B. 2017; A comparison of skeletal maturity assessed by radiological and ultrasonic methods. *American Journal of Human Biology,* Vol. 29 (Early View): 1-7.

Wadhwa, P. D., Entringer, S., Buss, C. and Lu, M. C. 2011; The Contribution of Maternal Stress to Preterm Birth: Issues and Considerations. *Clinics in Perinatology,* Vol. 38 (3): 351-384.

WHO Infant Mortality: Global Health Observatory (GHO) Data [Online] [Accessed: September 2017]
Available From:
http://www.who.int/gho/child_health/mortality/neonatal_infant_text/en/

Wilkie, L. A. 2013; Expelling frogs and binding babies: conception, gestation and birth in nineteenth-century African-American midwifery. *World Archaeology,* Vol. 45 (2): 272-284.

Chapter 8: Manuscript 3

Little Lives: A metrical and morphological evaluation of a documented 20th Century fetal and infant skeletal collection.

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KEY WORDS: Growth; Disruption; Age Estimation; Non-Adult

Abstract: *Evaluating growth in order to estimate age-at-death for fetal, perinatal and infant individuals has long been an anthropological concern. Growth standards, from which ages in gestational weeks are derived, have been developed using both documented and archaeological samples. Within bioarchaeology, these methods are commonly employed to generate age estimates, often with little consideration of the comparability between the reference data and sample assessed. This study considers 140 individuals from the Smithsonian Fetal Collection of known age, sex and ancestry. Skeletal growth profiles, plotting mean skeletal and dental ages against documented age, were constructed for these individuals. Skeletal growth profiles for the femur, tibia and humerus were developed. Analysis of dental, skeletal and documented age was also undertaken to explore specific evidence of growth disruption. Results suggest that dental development, when error ranges are included, can be used as a proxy for chronological age within this population. However, both female and black individuals have increased dental development for gestational age, resulting in over-estimations of age-at-death. Conversely, age-at-death estimates based on diaphyseal length were found to consistently show significant underestimation of age, particularly within postnatal age categories, with ages derived from long bone diaphyseal lengths found to cluster around ~40 GWA, a common problem of this age estimation methodology. Furthermore, the tibia was found to be the most sensitive long bone to growth disruption. Metric analysis of the pars basilaris suggests that this element shows parity with both dental and documented age. Consequently, this bone may be suggested to be a good proxy for chronological age. Findings from this study contribute to the ongoing discussion considering the multitude of environmental and biological parameters that can regulate growth in the early life course, highlighting diverging growth strategies between individuals and the potential implications for archaeological assessment and interpretation of age-at-death.*

Introduction

Estimating age-at-death has been a central concern within bioarchaeological and anthropological studies (Falys & Lewis 2011). Such assessment is paramount to the construction of osteobiographies and for biocultural interpretations of the life course (Robb 2002; Sofaer 2006; Gowland 2006; Falys & Lewis 2011). Consequently, research into the 'normal' trajectory of growth and development has been ongoing for more than a century (e.g. Boas 1930; Tanner 1978; Bogin 1999), and has resulted in a well-established chronological sequence for healthy skeletal and dental progression in non-adults. Prior to skeletal maturity, changes in bone and dental size and morphology, together with the ossification and fusion of skeletal elements, provides a wide range of biological parameters to use for age estimation (Falys & Lewis 2011). For fetal, perinatal and infant skeletons, the rapidity of growth during this period provides a high degree of chronological resolution; ages are often estimated within an error range of two or four gestational weeks up until the perinatal period, extending to a range of a few months thereafter until one year of age (e.g. Scheuer *et al.* 1980; AlQahtani *et al.* 2010). Until recently, detailed studies of fetal, perinatal and infant remains were relatively scarce in the bioarchaeological literature (Halcrow *et al.* 2018). However, the importance of these early life stages for understanding longer-term health has stimulated interest in the mother-infant nexus and the intersection of biology and culture at the beginnings of life.

This study aims to investigate the complex relationship between growth and the assessment of age-at-death in fetal, perinatal and infant remains. A large documented medical collection was examined to assess the veracity of multiple methods for estimating age-at-death. This analysis explicitly recognises the contextual nature of this medical collection and the potential for growth disruption and adversity to have impacted on the growth and health of these individuals during their brief and fragile existence. Indeed, it is important to acknowledge that such skeletal collections do not represent a 'biological control': these individuals did not live their short lives and die within a social vacuum. Nor should we be insensitive to the circumstances of structural violence, which may have been responsible for their curatorial end, as opposed to burial (Gindhart 1989; Nystrom 2014). The fetuses, perinates and infants that constitute this collection were conceived and grew within social worlds, and will have been affected by maternal well-being: moulded by genetic, epigenetic and environmental factors. Nevertheless, large, documented samples of fetal and young

infants are scarce and such analyses have the potential to advance our understanding of this neglected demographic in the more distant past (Halcrow *et al.* 2018; Lewis 2018).

Fetal, Perinatal and Infant Growth and Development:

Intrauterine growth represents the most rapid phase of human development (Sinclair 1985; Roth 1992; Gluckman 1997). The developing fetus will receive all of their nutrition, and immunological and environmental buffering from their mother (Gowland 2015); if maternal health and environment is compromised, fetal growth and development will be adversely affected (Gowland 2015). Postnatal maternal nutritional deficiencies or infections may also impact the ability of the mother to provide optimal nutrients via breastfeeding, affecting immunology and increasing disease susceptibility (Fujita *et al.* 2017). The rapidity of growth and development during early life also contributes towards the precariousness of this period, which is characterised as one of enhanced plasticity, when life course trajectories are most easily diverted from the optimum (Roth 1992; Armelagos *et al.* 2009).

The quick and rapid turnover of bone in fetal, perinatal and infant individuals also means that any insults are likely to be reflected more quickly within the skeleton (Lewis 2017). Conversely, more acute insults may heal more rapidly and leave no discernible trace (Bush & Zvelebil 1991, 5; Cardoso 2007, 231). The mother is generally conceptualised as a buffer, absorbing the brunt of adverse environmental conditions at the cost of her own health (Gowland 2015). Evidence of intrauterine growth disruption reflects the fact that the mother no longer retains sufficient stores to adequately buffer or optimally support the fetus/infant (Barker *et al.* 2012). Fetuses with no identifiable growth disruption, therefore, did not necessarily develop within an ideal environment, instead their growth was prioritised and maintained at expense of the mother (Gowland 2015).

Many clinical and anthropological studies have considered both intrinsic and extrinsic factors, such as nutrition, parity, health and socioeconomic status, political changes and psychological health on growth and development (e.g. Schell 1981; Adair 2004), all of which have been found to affect the size of the child at birth, and thus the diaphyseal length of long bones (Lewis 2007). The interplay between such impacts and growth is complex. Growth disruption is not experienced uniformly between individuals, regardless of whether the adversity faced is similar or not, and individual experience, predisposition and susceptibility to disruption will ultimately reflect varying growth and age profiles between individuals

(Rogers 1997; Falys & Lewis 2011; Garcia *et al.* 2017). Those factors, which can have a limiting effect on growth and development, have been termed as non-specific 'stressors' throughout much of the published literature (Goodman *et al.* 1988; Lewis & Roberts 1997; Goodman & Martin 2002) and are referred to as such within this study.

Unravelling Age-Related Terminology:

Age is a complex biological, chronological and cultural construction (Gowland 2002; Baxter 2005; Lewis 2007) and bioarchaeology has utilised chronological age as a standardised unit of analysis with which to investigate past life courses, health and social practice (e.g. Stoodley 2000; Gowland 2001; 2002; 2006). This study uses age estimation as a tool for analysis to distinguish evidence of growth disruption, by comparing age-at-death estimates generated from a variety of cranial and postcranial bones and teeth (Huda & Bowman 1995). Dental and skeletal assessments are those which measure an aspect of physiological (biological) development (Lewis 2007; Couoh 2017) and these are then translated into a chronological age via the medium of modern known age reference standards (Gowland 2006). However, given the ways in which growth and development can be altered and disrupted, age estimates derived from a stage of physiological development do not always correlate to 'true' chronological age (Saunders *et al*. 1993). By investigating individuals of known age-at-death, accuracy of ageing methodologies elements can be assessed, as well as the impact of health stress on growth and development.

As this is a documented population, true chronological age estimates for some of the individuals assessed were provided. Consequently, these 'known' ages have been referred to as documented ages throughout this study. However, although many of the individuals were ascribed an age-at-death at time of curation (see Materials section for more details), the terminology employed lacks standardisation. Thus, this study, though constrained by the terminology employed in collection of the individuals, suggests the use of the terms fetus, perinate and infant to distinguish between individuals based on their physiological growth and development (Table 1.). These age-at-death categories and terminologies were also defined in consideration of the potential life course events these individuals experienced (e.g. Wiley & Pike 1998).

All age estimates throughout this study have been provided in gestational weeks of age, referred to as GWA.

TABLE 1. Definitions of age-related terminology employed within this study.

Fetus	< 36 GWA
Perinate	36-44 GWA
Infant	> 44 GWA

Materials

The fetal and infant collection dates to the first two decades of the 20th century and was compiled by Aleš Hrdlička (Gindhart 1989; Hunt *personal communication*) who, whilst working at the USNM (United States National Museum), now the NMNH (National Museum of Natural History), corresponded with many active and practicing medical professionals, who donated or exchanged human remains with the museum (Hunt *personal communication*). The skeletons derive primarily from Washington D.C., Baltimore, Maryland and the metropolitan areas on the north-east coast of the United States of America (Gindhart 1989). The individuals within the collection are believed to have been acquired between 1903 and 1917 (Gindhart 1989) and collected/donated from medical institutions including Columbia Hospital, Freedman's Hospital, Howard University, and University of Maryland School of Medicine (Hunt *personal communication*).

Originally, 365 fetal individuals were collected and curated by the NMNH, but today 320 of those are still present, with 45 having been damaged or mixed, and consequently deaccessioned (Hunt *personal communication*). Of the 320 skeletons, the majority have a recorded age, sex and 'ancestry' (Hunt *personal communication*). When the collection was originally curated the skeletons were assigned as either 'Black', 'White', 'Coloured' or 'Mulato' (Kósa 2002). Although today, ethically and racially, this practice and terminology is inappropriate and misleading, the collection currently still remains distinguished in this way.

Of the 320 individuals available for analysis, only 140 were assessed for the purposes of this study due to time and access constraints. This sample was randomly chosen and encompassed individuals of a range of gestational ages, sex and ancestry, as well as with a range of medical conditions. As yet there is no database or record of the individuals considering their

preservation, or detailing the skeletal elements available for analysis. Therefore, it was unknown how many individuals in total had dentition and/or skeletal elements available for analysis. Although ideally, only individuals with both dentition and skeletal elements would have been studied, there was no way to determine which individuals would be best for assessment without visually considering each one. Consequently, a random sample was selected. As a result of this selection strategy, no individuals afforded the term 'Mulato' were considered in assessment, and only one 'coloured' individual was analysed. This was not a deliberate selection or assessment bias, but likely reflects the fact that less individuals termed to be 'Mulato' or 'coloured' are listed within the medical sample.

Although this population provides a rare opportunity to consider a historical 'known' fetal/infant population, there are some limitations to the documentation that need to be addressed. Ethical considerations of the collection and curation methods employed have not been widely addressed, but individuals are likely to have been retained as a result of highly unethical procedures (See Gindhart (1989) for a comprehensive discussion of collection and curation practices). Indeed, although black and white individuals broadly comprise of fifty percent of the sample each, Gindhart (1989) does consider that those remains of black individuals may have been targeted for collection. Furthermore, many of the individuals have specific congenital conditions, particularly those of neural tube defects, and thus may have been collected based on the diseases/conditions they presented. As a result, this sample may show elevated levels of particular pathological conditions and associated growth disruption. Black individuals are also considered to have been more likely to present with pathological conditions, as they commonly experienced higher rates of disease/infection in life (Gindhart 1989), and so likely received greater selection for collection.

Ages-at death were recorded by a number of terms – gestational months and weeks/days were provided in varying instances. Table 2 outlines the range of specific terminology that was recorded for individuals within the collection. Conversion of these terms into gestational weeks, to enable easier comparison between individuals, was undertaken and is also presented in Table 2. This has been undertaken in accordance with the standards outlined by Huxley and Angevine (1998) who provide a conversion chart for the transfer of ages reported in gestational and lunar months into those of gestational weeks.

Individuals with no specific age recorded (e.g. months/days) were noted as either a fetus, infant or child. Some individuals were afforded the additional detail of 'Newborn' or 'Died at Birth'. This study assumes that those individuals classified as 'Newborn' or 'Died at Birth' were around or close to full term, particularly as other individuals described as such are aged to be '9 *in utero* months' or 'Full Term'. Table 2 also outlines the age ranges attributed for this various terminology. Within assessment of these individuals, as no precise age-at-death in gestational weeks is known, skeletal and dental age estimates are only considered in correlation to the age ranges ascribed to them based on terminology. Although the categories of 'infant' and 'child' are both classified as simply being comprised of those individuals over 44 GWA, there does seem to be a clear distinction in the historical recording of these individuals. Consequently, it is supposed that 'children' represent older individuals than those classified as 'infants'. However, as no age-at-death estimates in gestational weeks are recorded for these individuals both have simply been classified as over 44 GWA. To determine whether those defined as 'children' are indeed older on average than those classified as 'infants', these categories have remained discrete.

Table 3 details the number of individuals assessed by gestational age/age category, biological sex and 'ancestry'. Individuals in the categories '9 months *in utero*', 'full term' and 'one day old' have been combined as their age estimates are all 40 gestational weeks.

TABLE 2. Age-related terminology used in the Smithsonian Fetal Collection and conversion to terminology and age estimates employed within this study.

Study Terminology	Collection Terminology	Age Estimation (Gestational Weeks of Age)
Fetal Individuals	Fetus	< 36
	3 months in utero	13-14
	4 months in utero	18
	5 months in utero	22-23
	6 months in utero	26-27
	6.5 months in utero	29
	7 months in utero	31-32
	8 months in utero	35-36
Perinatal Individuals	8-9 months in utero	35-40
	9 months in utero	40
	Newborn	36-44
	Died At Birth	36-44
	Full Term	40
	1 Day Old	40
	7 Days Old	41
	25 Days Old	43-44
Infant Individuals	40 Days Old	45-46
	2 Months Old	48
	4 Months Old	56
	5 Months Old	60
	Infant	> 44
	Child	> 44

TABLE 3. Summary of individuals assessed, with individuals listed by recorded chronological age, biological sex and ancestry. For chronological age 'm.' has been used as an abbreviation for months. Ancestry has been recorded as W (White), B (Black), C (Coloured) or U (Unknown).

Age Category	**Age (GWA)**	***N***	**Male**			**Female**				**Unknown**		
			W	**B**	**U**	**W**	**B**	**C**	**U**	**W**	**B**	**U**
3 m. *in utero*	13-14	3	1	1			1					
4 m. *in utero*	18	2	1							1		
5 m. *in utero*	22-23	2		2								
6 m. *in utero*	26-27	5	1	1			3					
6.5 m. *in utero*	29	1	1									
7 m. *in utero*	31-32	8	4		1	2	1					
8 m. *in utero*	35-36	3		3								
8-9 m. *in utero*	35-40	2	1	1								
9 m. *in utero*	40	12	1	4		1	5				1	
Full Term	40	3		2		1						
1 Day	40	1					1					
7 Days	41	1		1								
25 Days	43-44	1					1					
40 Days	45-46	1					1					
2 m.	48	1					1					
4 m.	56	1		1								
4.5 m.	58	1			1							
5 m.	60	1					1					
7 m.	68	1					1					
Fetus	< 36	75	25	9	1	26	7	1	1	1		4
Newborn	36-44	6	2	1			2			1		
Died At Birth	36-44	1	1									
Infant	> 44	3		1		1	1					
Child	> 44	4		2			2					
Unknown	-	1	1									
Totals		140	39	29	3	31	28	1	1	3	1	4

Methods

Gestational age-at-death estimations for each individual were determined based on dental development and metric assessment of selected long bones and the *pars basilaris*, where possible. Dental development provides a useful chronology for life history events and is used to infer chronological age within bioarchaeological studies (AlQahtani *et al.* 2014). Teeth develop in a predictable sequence over the first ~20 years of life (AlQahtani *et al.* 2014; Bang 1989): they have 'morphologically distinct stages of formation and mineralization' which have been found to correspond well with certain age ranges (Bang 1989). Thus, for the dentition, methods of age-at-death estimation rely on assessing the stage of development for each tooth as they grow systematically from the tip of the crown to the root. Tooth cusp development was recorded in accordance with Moorrees *et al.* (1963a; 1963b) and age-at-death estimates attributed using the dental development atlas developed by AlQahtani *et al.* (2010).

Dental development is a more accurate method for estimating gestational age as it is less susceptible to external factors, and thus less easily disrupted (Garn *et al.* 1960; Hillson 2005; AlQahtani *et al.* 2010). Consideration of dental age estimates against the documented chronological age estimates has allowed for assessment of the accuracy of these dental age estimation methods. The London Dental Atlas (AlQahtani *et al.* 2010) has been typically found to underestimate chronological age (by 0.1 of a year (5.2 gestational weeks) on average) based on dental development (AlQahtani *et al.* 2014); however, it underestimates age to a lesser extent than the previous methods developed by Schour & Massler (1941a; 1941b) and Ubelaker (1978). It is commonplace that modern clinical standards are compared to archaeological populations, as absence of tooth development and eruption sequences for past populations, makes it impossible to generate more accurate dental age estimations. Furthermore, evidence from living populations suggests that early development and growth is relatively similar between populations, as long as the environment is optimal (Ruff *et al.* 2013). Thus, the standard developed by AlQahtani and colleagues (2010) is the most recent example of this method, and uses both known age-at-death historical populations and modern clinical data. For individuals for whom tooth cusp development fell between two age estimates a midpoint between those two estimates was assigned in gestational weeks.

Long-bone length is traditionally considered to be useful in determining chronological age (Jeanty & Romero 1984; Jeanty *et al.* 1984). Consequently, diaphyseal length measurements of all present long-bones were recorded for each individual. All measurements were taken using digital sliding callipers in accordance with the metric analyses outlined in Fazekas & Kósa (1978) and Schaefer *et al.* (2009). Results were recorded to the nearest tenth of a millimetre. To interpret these metric data, and consider growth disruption, gestational age-at-death estimations were calculated using the published regression equations for long-bone diaphyseal lengths by Scheuer *et al*. (1980). Two-way ANOVA statistical analysis (analysis of variance) was utilised to analyse differences between skeletal and dental ages by documented age groups.

Metric assessment of the *pars basilaris* was also conducted as due to the changes in morphology, and thus the measurements obtained, it is considered to be indicative of certain age thresholds (Redfield 1970; Scheuer & Maclaughlin-Black 1994; Lewis 2007). Furthermore, previous research by the authors has suggested that age estimates derived from the *pars basilaris* align closely with dental, and thus chronological age. By analysing the *pars basilaris* within a documented age sample this association has been able to be tested. Maximum width, sagittal length and maximum length were all recorded when possible. Measurements of the *pars basilaris* were correlated into chronological age estimates (in GWA) using Scheuer and Maclaughlin-Black (1994). Though this methodology has major limitations, particular its small sample sizes per age group, and its lack of given error or age ranges, it still provides a useful assessment for determining age-at-death. Where measurements fell within a range of age categories, the mean age category has been plotted, with the minimum and maximum age categories used as upper and lower age ranges. For those individuals who were found to fall with the 40 GWA or younger categories Fazekas and Kósa (1978) was also employed to determine a more specific age estimate. However, no methodology as yet exists to metrically consider the *pars basilaris* in older perinates and infants.

Although, in principle, assessment of skeletal dimensions provides an accurate and reliable way to assess age, in reality skeletal growth and development is variable depending on a variety of genetic and environmental factors. Thus, both dental and skeletal age estimates have been derived for each individual where possible to compare against one another. In comparison to other growth systems, such as the skeleton, the dentition is minimally affected

by environmental and nutritional onslaughts (AlQahtani *et al.* 2014; Garn *et al.*1965a; 1965b; Elamin & Liversidge 2013), thus it is suggested that growth disruption can be determined by considering the difference in dental and skeletal age estimates calculated. Consequently, this study considers the accuracy of dental and skeletal methods of age-at-death estimation by comparing estimates generated to known chronological ages. Furthermore, this enables identification and assessment of growth disruption. Despite a variety of other methods being available to aid in determining chronological age (e.g. closure of the mendosal suture and fusion of the tympanic ring to the *pars petrosal*), this investigation intended to observe and investigate growth disruption from metric analysis of the skeleton.

Results

Although selective criteria may have been used in the collection of these individuals, the sample studied was generally reflective of the overall collection structure, in terms of both biological sex (Fig. 1) and age categories. Percentages of individuals by chronological age category, for the whole collection and those sampled were calculated. Two sample t-test analysis shows no statistical significance between the collection and those sampled (*P= 0.99*).

The individuals assessed were categorised by documented chronological age, biological sex and 'ancestry' (Table 3.). Only one individual assessed did not have a recorded age, with biological sex unknown in eight individuals. Of the 140 individuals, only 57 have age estimates which can correlate to a gestational age, with 82 individuals only able to be attributed to one of the broad age categories. In total, 71 males and 61 females were assessed, with 72 individuals recorded as white, 58 recorded as black, and one individual recorded as 'coloured'. Overall, 8 individuals did not have ancestry recorded.

In total, 58 individuals assessed had dentition available for analysis, with 117 individuals having femora, 118 having tibiae, and 122 individuals having humeri available for assessment of diaphyseal length.

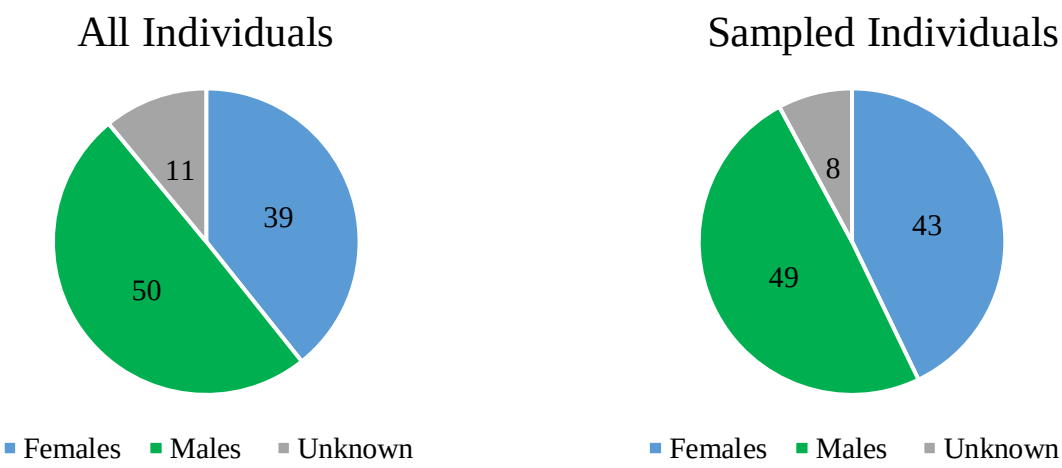

Figure 1. Percentage of individuals, by biological sex, in the total collection and sampled.

Assessment of dental age estimates and femoral, tibial and humeral age estimates, based on diaphyseal length (Scheuer *et al.* 1980), was undertaken for individuals with documented ages (Table 4.). Summary statistics for the individuals assessed are presented in Table 4. For age estimates where only one individual was selected for assessment (categories 41-68 GWA) standard deviation and confidence intervals could not be established. Results in bold are those for which significant differences based on t-test analysis ($p < 0.05$) were found between the skeletal age estimates and known age. Again statistical analysis could not be undertaken where only one individual was present for an age category.

TABLE 4. Mean dental, femoral, tibial and humeral age estimates by documented age-at-death category (GWA). Where documented age-at-deaths generate a range of estimates the mid-point has been utilised. Standard deviation and confidence intervals +/- have also been given for skeletal age estimates. Number of individuals (N) has been given for each skeletal element. Where only one individual was available for assessment statistical analysis was not conducted. Mean age estimates in bold are those found to be significantly different from documented age.

KNOWN AGE		**DENTAL**				**FEMUR**				**TIBIA**				**HUMERUS**			
GWA	***N***	***N***	**Mean**	**Conf. Int. (+/-)**	**S.D.**	***N***	**Mean**	**Conf. Int. (+/-)**	**S.D.**	***N***	**Mean**	**Conf. Int. (+/-)**	**S.D.**	***N***	**Mean**	**Conf. Int. (+/-)**	**S.D.**
13.5	3	0	-	-	-	3	20.1	7.8	3.1	2	20.9	23.1	2.6	3	18.0	9.5	3.8
18	2	0	-	-	-	2	**20.6**	1.0	0.1	1				2	18.5	6.1	0.7
22.5	2	0	-	-	-	2	28.3	27.2	3.0	2	28.2	30.7	3.4	2	27.6	33.0	3.7
26.5	5	3	32.7	5.7	2.3	3	27.3	6.6	2.6	4	27.4	4.5	2.8	3	28.5	4.6	1.8
31.5	9	3	**38.7**	2.9	1.2	8	**28.8**	1.9	2.1	9	**29.3**	2.2	2.6	9	**29.0**	2.0	2.4
35.5	3	3	**39.3**	2.9	1.2	2	38.0	3.0	27.2	2	39.4	34.9	3.9	2	38.7	26.0	2.9
37.5	2	2	39.5	6.4	0.7	2	34.4	14.1	1.6	2	34.1	17.9	2.0	2	33.9	26.4	2.9
40	23	19	**45.2**	2.7	5.5	16	**36.9**	1.4	2.6	17	**37.7**	1.4	2.8	17	**36.4**	1.7	3.2
41	1	1	46	-	-	1	39.1	-	-	1	40.3	-	-	1	39.8	-	-
43.5	2	2	46	-	-	2	38.2	11.0	1.2	2	38.9	16.3	1.8	2	38.1	13.6	1.5
48	1	1	58	-	-	1	40.3	-	-	1	41.0	-	-	1	40.6	-	-
56	1	1	64	-	-	1	42.3	-	-	1	42.8	-	-	1	42.6	-	-
58	1	0	-	-	-	0	-	-	-	0	-	-	-	0	-	-	-
60	1	1	58	-	-	1	45.1	-	-	1	45.6	-	-	1	46.0	-	-
68	1	1	70	-	-	0		-	-	0		-	-	0		-	-

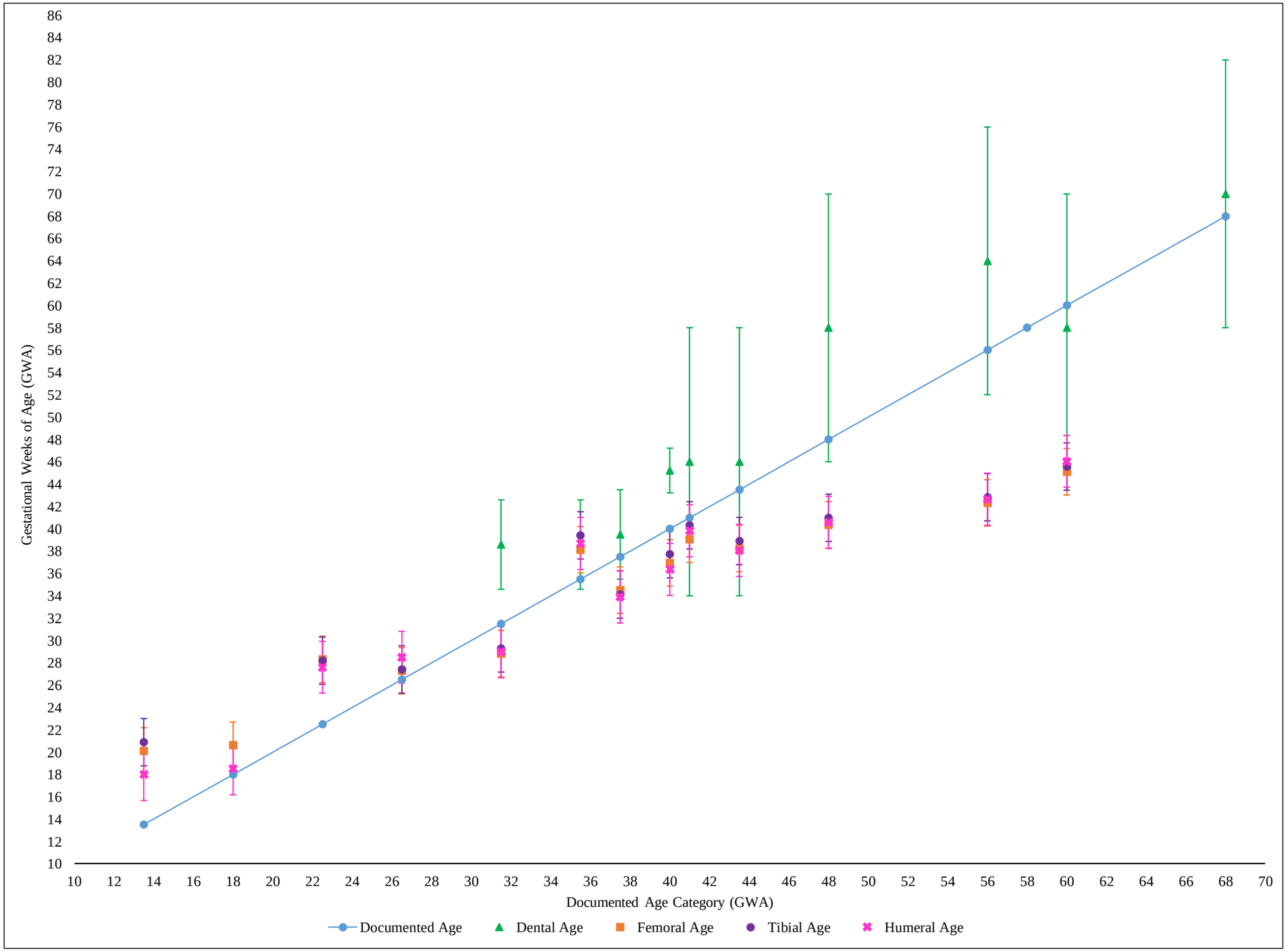

Figure 2. Mean age estimates for dental and skeletal elements by known age category. Error bars of in accordance with AlQahtani et al (2010) and Scheuer et al (1980) have been plotted.

Mean skeletal and dental age estimates by known age category have been presented, with error bars/age ranges given in accordance with AlQahtani *et al.* (2010) and Scheuer *et al.* (1980) (Fig. 2) given as an age range for each skeletal estimate. Dental age estimates have been found to be consistently older on average than documented age, except for the 60 GWA category. Although standard deviation could not be calculated in the older age categories (48-68 GWA), average dental age can still be seen to align with documented age closely. This is in contrast to diaphyseal age estimates, which fall below (younger than) documented age. Consideration of average skeletal and dental estimates, with error ranges provided by the published methods, show average dental age can be seen to correlate with documented age for all age categories except 31-32 GWA (7 months *in utero*) and 40 GWA (9 months *in utero*). Conversely, skeletal age-at-death estimates only correlate with documented age for three of the age categories (18, 26.5 and 41 GWA). For those aged 13.5 GWA, 22.5 GWA and 35.5 GWA average skeletal estimates were found to be older than documented age. For those aged 43.5 GWA and older, average skeletal estimates were all younger than documented age. Given these findings, skeletal age-at-death estimates appear to be generating older estimates than documented age in fetal (prenatal) individuals, whilst generating younger estimates than documented age for infants (postnatal).

For individuals ascribed to the broad categories of fetus, newborn, infant and child, mean dental and skeletal age estimates were calculated (Table 5.). All mean dental and skeletal age estimates increase with documented age. Importantly, the average dental and skeletal ages for those in the 'infant' and 'child' categories do show differences, with those listed as 'children' appearing to be between 3-10 GWA older than 'infants'. Thus, the individuals remain classified in these two distinct groups throughout the remainder of this study. Dental ages all fall within documented age ranges, except for the fetal category. Again, skeletal age estimates show variable results with many estimates younger than documented age range.

ANOVA analysis, where sample size allows, of the dental age and skeletal age estimates generated (using Scheuer *et al.* (1980) and AlQahtani *et al.* (2010)) supports these findings (Table 6.), corroborating significant differences between skeletal and dental age estimates for the

documented age categories of '7 months *in utero',* '9 months *in utero',* and 'Fetus' (where $p < 0.05$).

TABLE 5. Results of mean age estimate, by age category, for dental and skeletal elements assessed. Standard deviations have also been given.

Documented Age Category	Age Estimation (GWA)	Dental Age		Femoral Age		Tibial Age		Humeral Age	
		Mean	SD	Mean	SD	Mean	SD	Mean	SD
Fetus	< 36	39.5	4.3	25.1	4.6	25.1	5.4	24.1	5.6
Newborn	36-44	42	5.6	35.5	3.3	36.1	3.5	33	1.5
Infant	> 44	48	3.5	39.7	5.6	40.2	5.9	40.2	6
Child	> 44	58	10.4	43.7	3	45.2	4.5	43.3	3.3

TABLE 6. Results of ANOVA testing of skeletal and dental age estimates by chronological age category. Results highlighted in bold are statistically significant, where $p < 0.05$.

Age Category	GWA	*F*	*P*
6 m. *in utero*	26-27	3.227	0.075
7 m. *in utero*	31-32	40.26	**5.708E-06**
8 m. *in utero*	35-36	0.07855	0.966
8-9 m. *in utero*	35-40	6.134	0.147
9 m. *in utero*	40	14.64	**1.55E-06**
Fetus	< 36	33.26	**1.24E-17**
Newborn	36-44	2.18	0.191
Infant	> 44	1.673	0.586
Child	> 44	1.564	0.303

To consider individual differences between dental and skeletal age estimates, those with dentition (*N*=58) were plotted with their femoral, humeral and tibial age estimates, derived from diaphyseal lengths (Scheuer *et al.* 1980). One individual was excluded from analysis as dental age was simply determined to be less than 30 GWA. Documented ages were also included, where possible. Dental and skeletal results have been plotted with age ranges (+/- *X* GWA) in accordance with those provided in AlQahtani *et al.* (2010) and Scheuer *et al.* (1980). Figure 3. shows that typically, dental and skeletal age estimates, and documented ages were similar, or fall within the age range of both dental and skeletal age-at-death estimates. Age estimates derived from tibial diaphyseal lengths, however, appear to most commonly fall below other age estimates generated. In total, 17 individuals have tibial age estimates which do not correspond or overlap with documented or dental age estimates. Of these 17 individuals, 12 are those recorded as black, with 4 individuals recorded as white. One individual has no recorded ancestry. Furthermore, 10 of these individuals with tibial growth disruption are female, whilst 6 are male. Again one individual had no recorded biological sex. Of these 17 individuals, 15 have no known pathological condition recorded, with only two individuals, both of 31-32 GWA recorded age (7 months *in utero)* and white females, having anencephaly. The presence of this condition may account for their young age and untimely death.

Mean femoral, humeral and tibial age estimates have been plotted by documented age distinguished by both ancestry (black and white individuals (Fig. 4.1)) and biological sex (male and female (Fig. 4.2)). Of the 56 individuals with documented age (in GWA), 47 had ancestry and 52 had biological sex recorded along with diaphyseal long bone lengths. Figure 4.1 shows that black individuals have greater diaphyseal lengths on average than white individuals, correlating to older age estimates. This is particularly evident for the prenatal individuals, though there appears to be more convergence in the perinatal stages. White individuals appear to have a steeper growth profile compared to black individuals. When compared by biological sex (Fig. 4.2) females can be seen to have a relatively steady and consistent increase between documented and skeletal age-at-death estimates. In comparison, male individuals show a much more varied and irregular growth profile, with both those aged 13.5 GWA and 68 GWA appearing to fall substantially below that of female growth profiles.

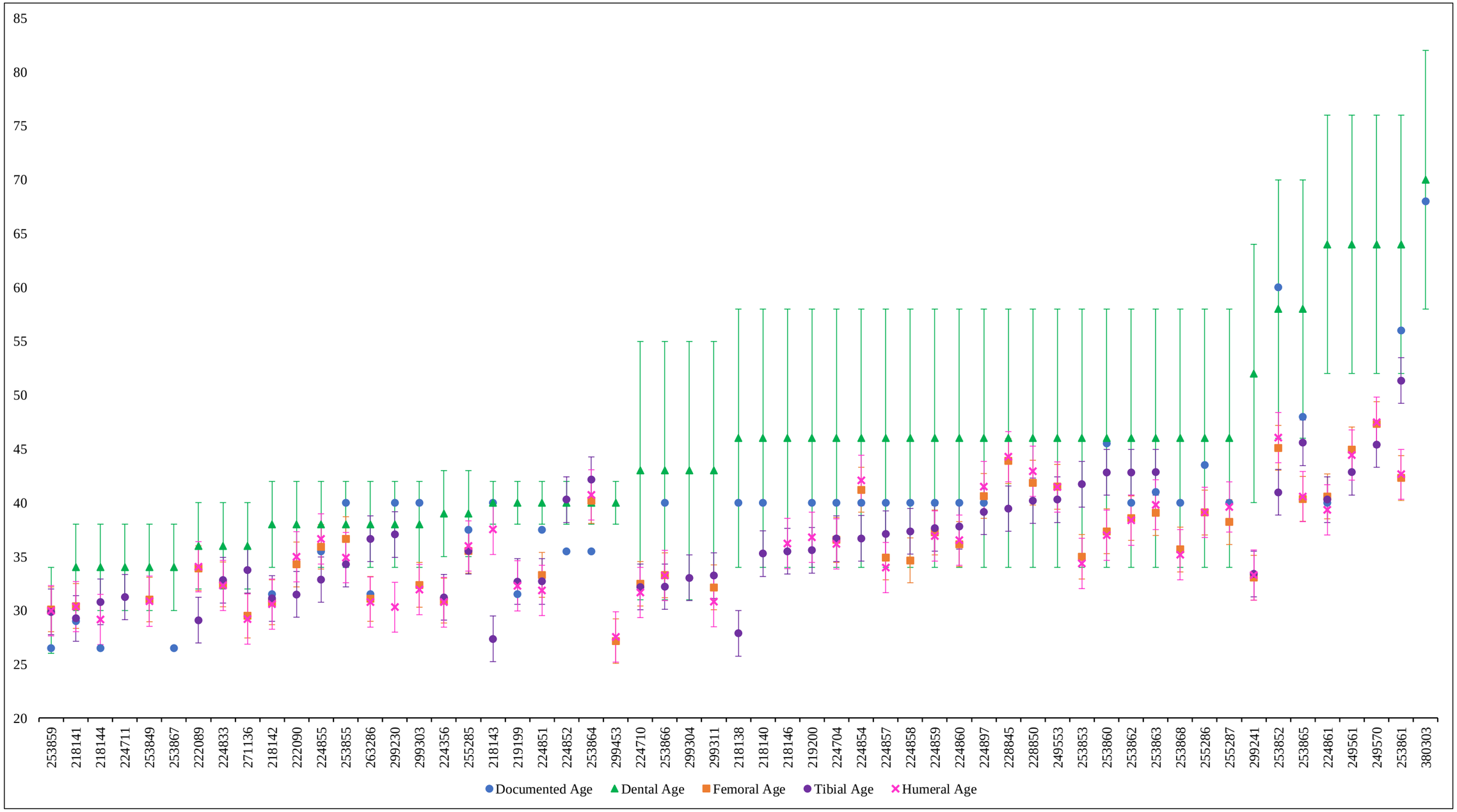

Figure 3. Mean age estimates for individuals with dental and skeletal elements available for assessment. Known chronological age, if available, has also been plotted for these individuals. Error ranges for dental and skeletal age estimates have been plotted in accordance with the reference methods of AlQahtani et al. (2010) and Scheuer et al. (1980).

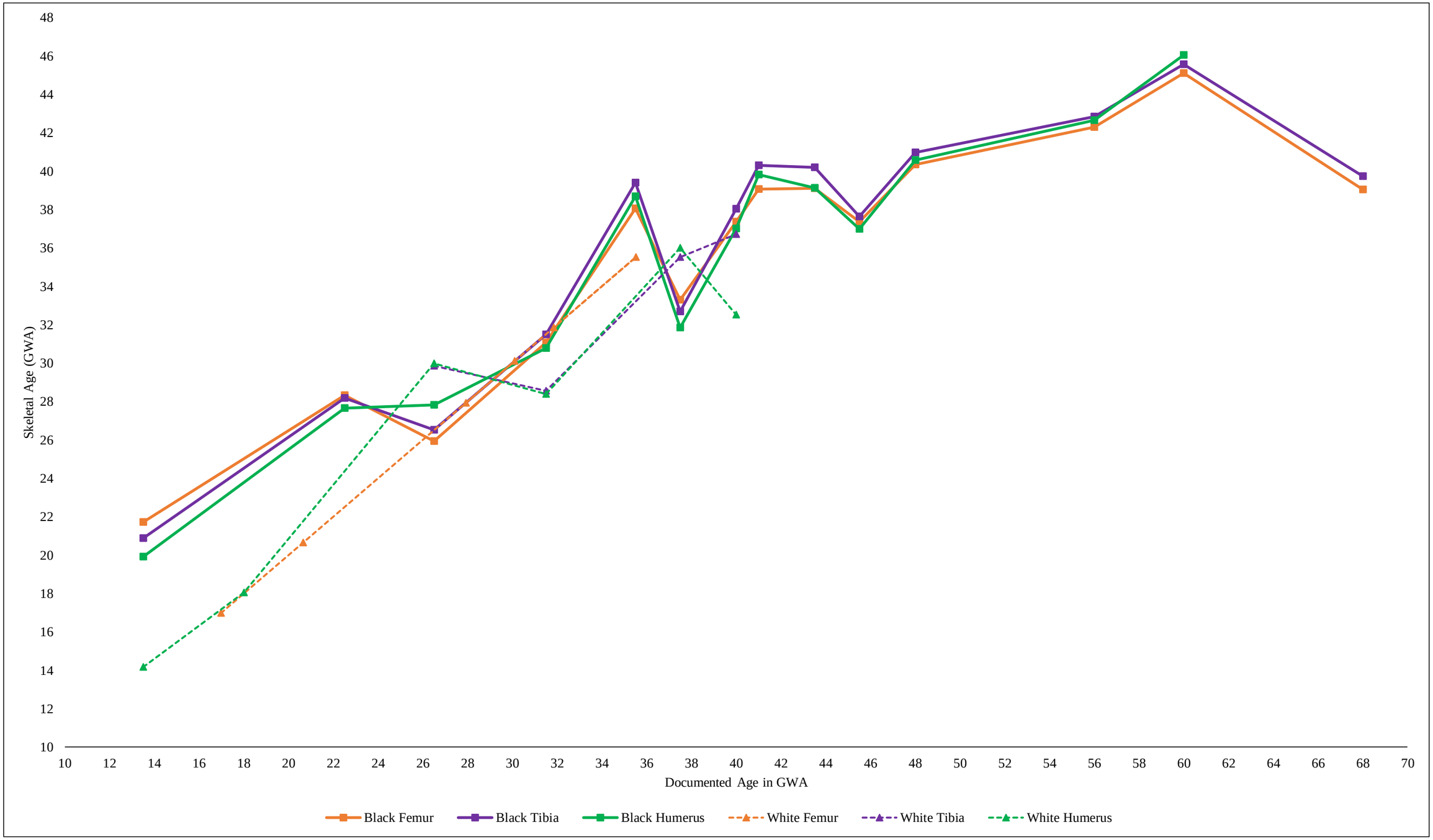

Figure 4.1 Mean femoral, tibial and humeral age estimates by documented age group and ancestry.

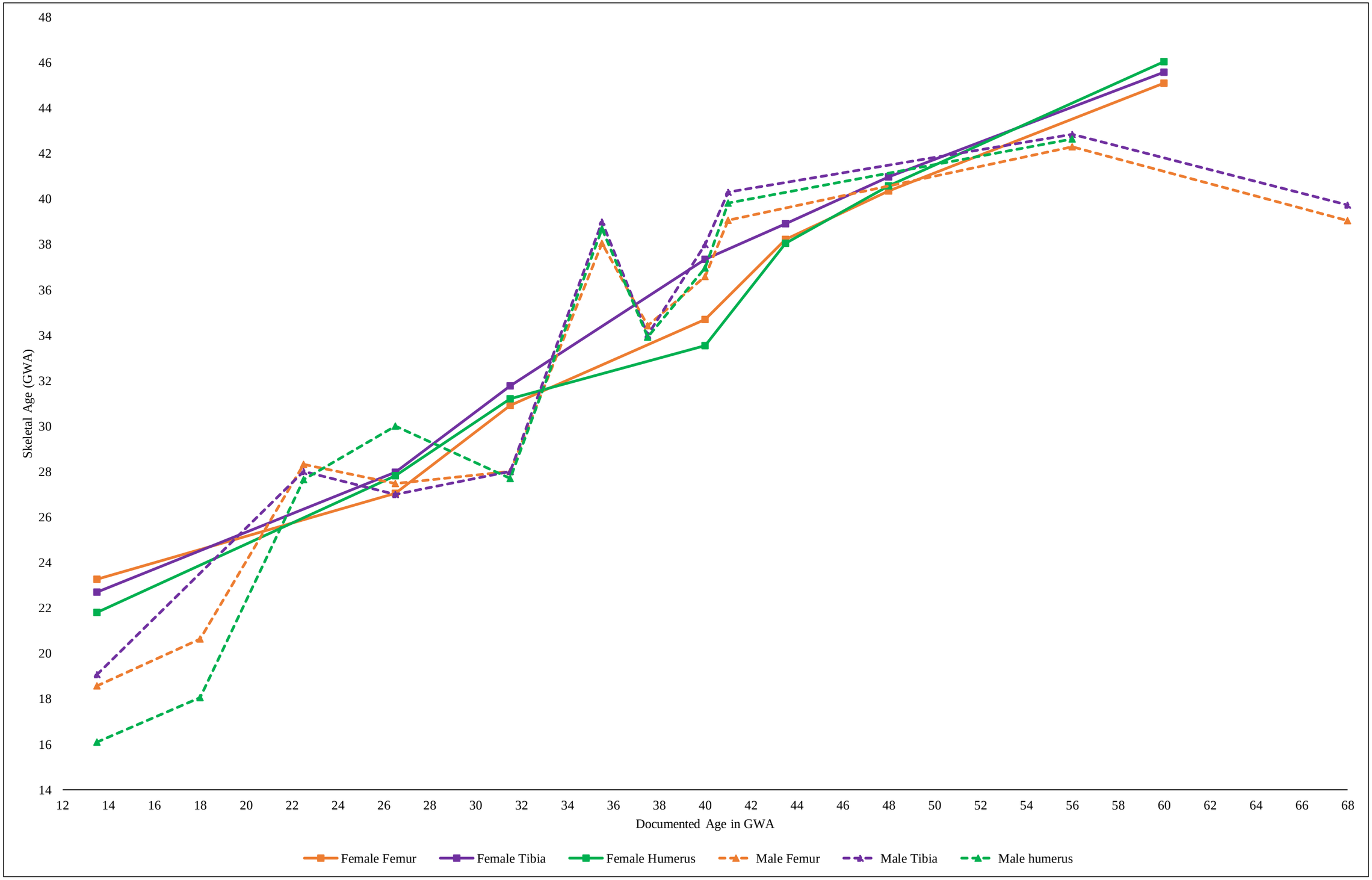

Figure 4.2 Mean femoral, tibial and humeral age estimates by documented age group and biological sex.

Consideration of the average differences between dental, documented and skeletal age estimates complicates the picture of growth disruption. When compared to documented age, the tibia and femur are seen to show the greatest growth disruption. Furthermore, white and female individuals are shown to have greater levels of growth disruption when compared to documented age. This may suggest that despite females having a steadier and more consistent growth trajectory (Fig. 4.2), this growth profile is consistently falling below true documented age. Black individuals are known to have longer diaphyseal lengths than white individuals (Martorell *et al.* 1988; Nyati *et al.* 2006) and thus, the greater growth disruption seen in white individuals compared to documented age may reflect this. Conversely, when skeletal age estimates are compared to dental age estimates, black individuals show a much greater level of growth disruption. Thus, despite skeletal growth closely aligning to chronological age, it appears it is lagging behind dental development. This suggest dental development within black individuals is more rapid than white individuals, and thus may be over-estimating age-at-death. Similarly, the growth disruption evident in female individuals when comparing dental and skeletal estimates may indicate that dental development within female individuals is more rapid than in their male counterparts.

TABLE 7. Average differences between skeletal, dental and documented age estimates.

	Dental - Femur		**Dental - Humerus**		**Dental - Tibia**	
	N	**GWA**	***N***	**GWA**	***N***	**GWA**
Overall	28	8	32	8	31	7
Black	21	10	22	9	22	9
White	6	5	9	5	8	4
Male	14	7	15	6	16	6
Female	13	10	16	10	14	9
	Documented - Femur		**Documented - Humerus**		**Documented - Tibia**	
	N	**GWA**	***N***	**GWA**	***N***	**GWA**
Overall	43	3	43	2	45	3
Black	28	3	27	2	28	3
White	13	4	17	4	14	4
Male	27	2	26	2	27	2
Female	16	4	19	4	16	2

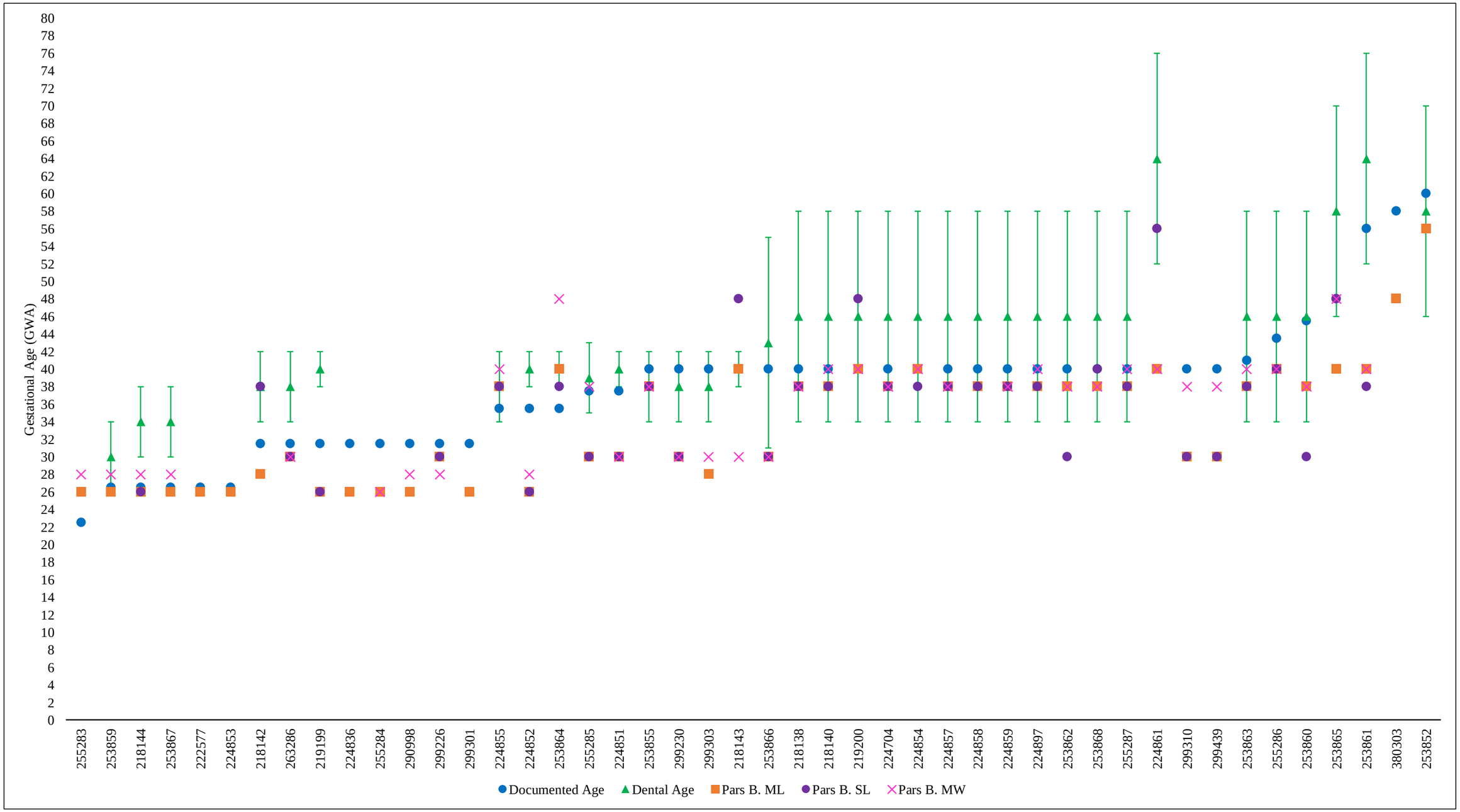

Figure 5. Mean age estimates for individuals with dental and pars basilaris elements available for assessment. Individuals have been plotted in ascending order according to their known chronological ages. Error ranges for dental and skeletal age estimates have been plotted in accordance with the reference methods of AlQahtani et al. (2010) and Scheuer & Maclaughlin Black. (1994).

Comparison between the *pars basilaris* and documented age (Fig. 5) shows that there is greater parity between age estimates derived from this skeletal element and documented age than those of the long bones (Fig. 3). The authors have previously shown that age from the *pars basilaris* is a good approximation of chronological age. This assessment typically supports these conclusions, but further analysis is needed as the methods for correlating metric results and chronological age are limited due to small sample sizes and lack of error range.

Discussion

Analysis of 140 individuals from the Smithsonian Fetal Collection has revealed significant differences both within and between dental and skeletal age-at-death estimates. Although only 57 individuals had documented ages, assessment has revealed considerable variation in the pattern of dental and, more particularly, skeletal development. Fayls and Lewis (2011) suggest that to make comparisons between varying skeletal samples less problematic, broad age categories and ranges should be employed. Many methodologies utilised for estimating age in fetal individuals determine ages in gestational weeks, or week bands. Thus, broad, general age categories are not possible when assessing growth/age within samples of only fetal/perinatal/infant individuals as the ability to distinguish between these young ages would be negated. Rapidity of bone growth and turnover within the pre- and immediate postnatal life also results in quantifiable differences in limb proportions in as little as a week: diaphyseal longitudinal growth in early postnatal life can be as much as 3mm per week (Issel 1985). Although this study has relied on methodologies which derive age estimates as a single gestational week (Scheuer *et al.* 1980; AlQahtani *et al.* 2010), error ranges for these ages have been considered to account for individual variability and plasticity in growth. As a consequence, dental and skeletal age-at-death estimates are regularly found to overlap, suggesting a correlation between the levels of physiological development. In these cases, regardless of by what proportion these age estimates correlate, growth disruption was not implied. By adopting this conservative approach to the data, the evidence of growth disruption identified is more robust.

Dental development has long been considered an accurate method by which age can be attributed to a skeletal individual (Bolaños 2000). Dentition within the individuals assessed was found on average to over-estimate age in comparison to documented age. The method

employed for recording and scoring dental development has typically been found to under-estimate age by 5.2 gestational weeks on average (AlQahtani *et al.* 2014); therefore, these results were not anticipated. When individual dental and skeletal age estimates were analysed (Fig. 3) nine individuals were found to have average dental age and error ranges which show no overlap/correlation to documented age. Of these nine individuals, six were recorded as female and three as male. Assessment of differences between dental and documented ages found that the male individuals have differences of 5 and 4.5 GWA. Conversely, females have differences of 24, 8.5, 7.5, and 6.5 GWA between dental and documented ages. One female (224861) is clearly an outlier with a 24 GWA difference between skeletal and dental ages, however, the females do show slightly larger differences between estimates than the males. However, given the small sample size it is impossible to make any wide-scale assumptions as to the pattern of growth disruption.

Female growth trajectories are considered to vary in comparison to males (Humphrey 2000; Lewis 2007; Sofaer 2011; Barker *et al.* 2012), yet development of the dentition is typically considered to only vary minimally between the biological sexes (Scheuer & Black 2004). Though female growth profiles appear much more regulated and consistent than male profiles when compared to documented age (Fig. 4.2) the clear differences when compared to dental age (Table 7.) support assumptions of very different growth strategies adopted by the biological sexes. Though appearing to have older age estimates (Table 7.), male growth profiles are much more irregular (Fig. 4.2). These findings may support the more 'risky' intrauterine growth strategies employed by male individuals (Ulizzi & Zonta 2002; Barker *et al.* 2012; Lewis 2018), whereby male growth is at the expense of the maternal wellbeing. In comparison, female individuals reflect life course maternal health and wellbeing, and regulate growth accordingly (Barker *et al.* 2012). Males typically grow more rapidly than females, and as a result of prioritisation of skeletal/bodily growth, have limited placental growth in comparison. This compromises their wellbeing by putting them at greater risk of under- or malnourishment. Therefore, though on average generating younger skeletal age estimates, female individuals may reflect a more robust growth strategy. Conversely, males, though found to be experiencing greater growth, reflect a highly variable growth profile, likely to be subsequently associated with increased frailty and reduced maternal health in later pre-/postnatal life (Green 1992; Synnes *et al.* 1994; Ulizzi & Zonta 2002). Consequently, male individuals are suggested to have a higher risk of perinatal death than females (Crawford *et al.* 1987; Green 1992; Synnes *et al.* 1994; Ulizzi & Zonta 2002), thus, the variability shown

in the male growth profiles (Fig. 4.2) may be elucidating this rapid but risky growth strategy. Furthermore, in 17 individuals for whom tibial age estimates fell below all other skeletal estimates, six individuals were found to be male and ten were female. Evidence for fewer males showing growth disruption within this skeletal element may support evidence for male offspring maintaining rapid skeletal growth regardless of maternal health, and thus, in stressful environments, continuing to grow at the expense of maternal health. However, this study found that frequencies of death by biological sex were similar for both pre- and postnatal individuals (Table 3.). However, this may reflect the collection strategy of the sample and not true mortality profiles, thus these findings cannot be substantiated as showing similar mortality risks between males and females.

Mean dental age estimations were found to be significantly different from documented age for those documented as seven, eight and nine months *in utero* (Table 3.). This may be a result of the small sample sizes, with only three individuals having dentition available for assessment in the seven and eight month *in utero* categories. Similarly, the small standard deviation given for these age categories might be a consequence of the limited sample size. However, when average dental age estimates are plotted with error bars (+/- GWA) according to the dental development method employed (AlQahtani *et al.* 2010; Fig. 2), dental age is seen to be comparable to documented age in all but two categories (31.5 GWA and 40 GWA). The significance of the difference between dental and known age is thus diminished; significance only refers to the exact mean itself, making no consideration for the error range which should be afforded. Of particular note is that both female and/or black individuals appear to show accelerate dental development in comparison to documented and skeletal age (Table 7.). Black individuals show much greater differences suggesting that dental development is more advanced within black individuals than white individuals of the same documented age. This may suggest that dental ages for black individuals are overestimating in comparison to documented age. When considered by biological sex, female individuals are shown to have much larger average age estimate differences between skeletal and dental ages. This may again indicate that dental development in more advanced within female individuals than male individuals of the same documented age. Growth is sex specific (Sofaer 2011) and males and females are well known to have varying growth strategies (Barker *et al.* 2012). It is widely accepted that females reach both skeletal and dental developmental stages earlier than their male counterparts (Humphrey 2000; Lewis 2007), with some females being between one and six months ahead in their overall dental development (Hillson 2005; Lewis

2007). However, despite this individual variation in dental development as a result of biological sex and/or 'ancestry' (Table 7.), dental development can be used as a proxy for establishing age-at death as long as error levels for methodologies employed are utilised (Fig. 3).

Growth disruption (where dental and skeletal mean age estimates and error levels do not overlay) was evident in 21 (41.2%) individuals where dental and skeletal long bones were both available for assessment. In total, 14 (32%) individuals have dental/femoral age estimates which do not correlate, 17 (35%) have dental/tibia growth disruption, with 19 (39%) individuals having dental/humeral age estimates which do not correspond. Frequencies of those with disruption were found to show those aged to be 38-40 GWA (*N*=8) and those over 52 GWA (*N*=7) most commonly had growth disruption. A clear distinction between pre- and postnatal birth becomes apparent, with those in the older age categories evidently unable to maintain a growth trajectory which mirrors dental or documented age (Fig. 2). This may be a consequence of the methodology employed, where utilisation of linear regression equations means results from the sample assessed mimic the reference population. Therefore, the clustering of individuals, based on long bone diaphyseal lengths, around the perinatal period might be artificially confining the true range of chronological ages. Furthermore, only individuals up to 46 GWA were considered in the creation of this reference methodology. Therefore, the drop off in growth identified in the postnatal individuals within the infant category might be a product of the methodology. As a result, only where individuals show now correlation or overlap in dental and skeletal age estimates and ranges, has an interpretation of growth disruption been afforded.

Although the femora and humeri were found to most commonly show growth disruption when compared to dental age, the tibiae and femora showed the most disruption compared to documented age (Table 7.). The lower limb is suggested to be the most variable in terms of growth profiles, and thus, though limited, the study supports this conclusion. Furthermore, where compared by both biological sex and ancestry (Fig. 4.1 and 4.2), the humerus, despite generating the lowest age estimates on average in prenatal individuals appears to generate the oldest age estimates in postnatal individuals. Therefore, for both biological sexes and black and white individuals, humeral growth appears to generate older age estimates on average than either femora or tibiae. In particular, the tibia is considered to be more sensitive to health and growth insults than other skeletal elements (Pomeroy *et al.* 2012). Consequently, the fact

that the tibiae within the individuals assessed shows evidence of growth disruption supports these recent studies (Pomeroy *et al.* 2012).

Metric and morphological differences are widely accepted to be present within racially divergent groups (e.g. Nyati *et al.* 2006; Kósa 2002), and this study support these findings. Differences between populations determine that American Black non-adults often develop/mature earlier than American white non-adults (Lewis 2007; Nyati *et al.* 2006). African American non-adults have been found to have longer legs than Mexican American and Caucasian American non-adults, whilst Caucasian American individuals have the greatest trunk length of these three groups (Malina *et al.* 1987; Martorell *et al.* 1988; Nyati *et al.* 2006, 135). When compared to documented age (Table 7.) long bone lengths show less average disruption in black individuals, with white and black individuals appearing to have diverging strategies to long bone growth (Fig. 4.1). In particular, black individuals have been found to show greater femoral and tibial lengths. This is particularly evident within the prenatal individuals, where black individuals show a greater growth profile. White individuals appear to show smaller skeletal diaphyseal lengths compared to black individuals for early prenatal development, correlating to younger skeletal age estimates. However, white individuals appear to show a steeper growth profile and incline, though it is difficult to confirm that this pattern would continue postnatally as white individuals with documented age, as well as long bones, were all 40 GWA or younger. However, if this pattern did continue it might be indicative of a slower postnatal growth profile in black individuals. Thus, despite black individuals appearing to initially have longer diaphyseal lengths, it does appear likely that they would have dropped off and been overtaken by white individuals postnatally. This may be a result of the diverging postpartum environments experienced by black and white individuals, reflecting greater social inequalities and exposure to stressors for the black individuals.

It is widely acknowledged that there is a racial disparity in health, which in turn reflects growth status (Kuzawa & Sweet 2009). It has been considered that racial disparity is closely entwined with socioeconomic status, availability of health care, education and employment, resulting in a perpetuating cycle of disadvantage amongst certain population groups (Kuzawa & Sweet 2009). Consequently, although suspected that there are biological and genetic differences in growth timings and tempos between the black and white individuals assessed,

it is less clear as to the extent of these differences and whether they are purely biological difference or ones bound within biocultural and social spheres.

The maternal-infant nexus is central to evaluating the etiological causes of growth disruption. Maternal regulation of both the pre- and postnatal period is essential to health and wellbeing, whereby a wealth of essential resources are provided by the mother to the offspring (Harding & Johnston 1995; Bateson *et al.* 2004; Barker *et al.* 2012; Said-Mohamed *et al.* 2018). Passive immunological and nutritional buffering safeguards the infant from exposure to a plethora of environmental stressors – the period of birth marks the transition into a world full of pathogens (Lewis 2017) – meaning maternal wellbeing is essential for postnatal wellbeing of the child. Evidence from the individuals assessed shows skeletal growth 'drops off' after ~40 GWA. This pattern of growth disruption within the skeleton may reflect a detrimental postnatal experience. However, it has equally been identified that illness and exposure to stressors later in gestation/pregnancy has a greater effect on fetal growth and health than those experienced earlier in pregnancy (Heinke & Kuzawa 2008). Thus, the fact that the older individuals show more evidence of skeletal growth disruption, both with more skeletal elements affected and greater differences between age-estimates, might not be unexpected. Instead this pattern may reflect the precarious period of later gestation which they experienced, and consequently survived, allowing skeletal changes to manifest. Lack of such significant skeletal growth changes in younger age categories may simply reflect a lack of time for manifestations of growth disruption to develop. In turn, those who died at younger ages, particularly prenatally, may be considered to be more fragile and susceptible to stressors, and consequently were unable to adapt to the conditions experienced.

Gindhart (1989) acknowledges that high infant mortality rates were common among all racial groups in the United States during this time. However, death rates for infants less than one year of age from Washington DC show that over three times more 'Black' infants died than 'White' (Gindhart 1989). Even today, black individuals have higher rates of morbidity and mortality in the United States, suggesting health, wellbeing and thus growth, is related to culturally constructed racial distinctions (Rogers 1997). Black individuals are not especially over-represented within this collection, although are more numerous than white individuals as a consequence of the collection strategy, rather than population demographics. However, growth changes identified have been found to be more common and severe in black individuals suggesting racial disparities may be contributing to this disruption.

Disparity in socioeconomic status is often associated with poorer nutrition, access to health care and living standards (Martorell & Habicht 1986; Floud *et al*. 1990; Nicholas & Steckel 1991; Schell 1997; Stinson 2000; Steckel 2009; Halfon *et al.* 2014; DeWitte *et al.* 2016). Scholars considering racial inequalities have often purported environmental stressors and lack of health care as pivotal causes of morbidity and mortality (Donald 1952). In fact, multiple environmental and psychosocial stressors are known to have a high correlation to detrimental birth outcomes (Chiswick 1985; Goldenberg & Thompson 2003; Abu-Saad & Fraser 2010; Cussons-Read *et al.* 2012; Beaudrap *et al.* 2013; Fell *et al.* 2016; Melby *et al.* 2016) and prolonged exposure to stress can alter functions and regulation of the immune system (Babones 2008; Boersma & Tamashiro 2015). Ultimately, stress exposure weakens the ability for immune response, thus leading to greater disease/infection susceptibility (Rogers 1997). Holmes (1980) suggests that the leading causes of infant deaths in America during the early 20th century were '*respiratory diseases, diarrhea and enteritis, premature birth, congenital debility, atrophy, marasmus, whooping cough, and influenza*'. He also suggests that maternal death was common during this period, although high disease loads in the population meant that many mothers were already of chronically reduced health status (Holmes 1937). However, it must be remembered that although this is a historical population, individuals studied would have had greater access to medical care than archaeological samples of fetal and perinatal individuals that have traditionally been considered. Developments in obstetric care, midwifery and maternal/pregnancy care would have all improved chances of survival and optimal growth and health (Lewis 2007), even though environmental conditions are likely to still have been still poor. Consequently, it may be that these individuals have been collected and curated on the basis of pathology/disease being present. Thus, results, although corroborating with much of the published literature, are ultimately biased by the demographic structure of this collection.

Indeed, the collection of these individuals themselves may be a reflection of the low socioeconomic status of these individuals. Structural vulnerability of poor and socially marginalized groups has been purported as a potential reason for curation and collection of individuals (Nystrom 2014). Social marginalization of these individuals and their parents may have rendered them politically and economically powerless, particularly in regards to preventing acquisition of remains by large institutions. As Gindhart (1989) describes, black individuals became worried about going to hospital for fear of being not only collected, but

deliberately killed to do so. Thus, curation and collection of these individuals may be intrinsically bound to their social and economic status.

Pathological lesions are a further biological marker which can reveal evidence of structural violence. Indeed, Klaus (2012) suggests that socially regulated resources have the ability to affect physiological wellbeing, with paucity of resources as a result of race, sex or age leading to skeletal manifestations indicative of reduced health status (Nystron 2014). Thus, both growth and health disruption is paramount for considering life course experiences. Furthermore, it has been suggested that pathological conditions can affect age-at-death estimation, making results significantly more inaccurate (Sherwood *et al.* 2000). Previous studies, considering neural tube defects, particularly anencephaly and spina bifida, instead found that individuals with these conditions tended to have skeletal elements which overaged the individual (Sherwood *et al.* 2000). Furthermore, the presence of alternative infectious and metabolic conditions is potentially a causative factor of growth disruption. Therefore, the association between health and growth disruption needs to be explored further.

Recorded pathological conditions and causes of death for the individuals assessed include spina bifida, anencephaly, pneumonia, gastrointestinal problems, and mechanical problems, thought to refer to birth trauma. However, pathological lesions were not isolated to only those with known and recorded conditions. In total, 58% of individuals were found to show pathological changes cranial elements, whilst postcranial elements were affected in 17% of the individuals assessed. Though this study avoids detailed assessment of pathological changes, focussing on evidence of growth disruption and the methodologies employed to determine this, health disruption is a likely the impetus behind much of the growth disruption identified. Furthermore, given the inferred low socioeconomic status of the individuals assessed, health disruption is likely.

Conclusion:

Fetal and perinatal individuals have rarely been considered within bioarchaeological discourse, with infant studies primarily focussing on the postnatal period (Halcrow *et al.* 2017). This study has demonstrated that these very young individuals are able to reveal valuable insights into the highly vulnerable stages of early life. Establishing evidence of growth disruption within this fetal, perinatal and infant collection has relied on dual analysis of dental and skeletal age estimates, but importantly has also been able to consider these

estimates in correlation with known, documented ages. Dental age estimation, when error ranges are included, has been found to show a strong relationship with documented age and it is argued can hence be used as a proxy for true age. Skeletal age estimates were found to vary significantly, particularly post-40 GWA, where long bone growth and error ranges were found to be significantly younger than documented age. This is likely a result of the existing methodologies and the way regression based analyses skew results towards the mean. This has resulted in mimicry between sample and reference population age estimates. Thus, the use of long bone diaphyseal length assessment to determine age-at-death should be avoided where possible. Instead the dentition should always be utilised. However, this study has supported initial findings by the authors suggesting that the *pars basilaris* is also a good proxy for chronological age. Again, limitations exist with current methodologies, but further investigation into the universal applicability of this bone to be used for age estimation should be undertaken. Comparison of long bone growth between black and white individuals found, on average, a comparable trajectory between the two, though black individuals were found to be more prevalent when considering evidence of tibial growth disruption. The tibia is considered to be the most sensitive of the long bones and results from this analysis further support these conclusions. Female and male individuals showed variation between their growth strategies, with male individuals reflecting a more fluctuating trajectory. Racial inequalities and environmental adversity are suggested to have been central causes for growth disruption identified, and are indicative of reduced mother-infant wellbeing. This study suggests further assessment of growth disparities utilising this known age collection is paramount for investigating the skeletal evidence of growth disruption in fetal, perinatal and infant remains.

References

Abu-Saad, K. and Fraser, D. 2010; Maternal nutrition and birth outcomes. *Epidemiological Review,* Vol. 32: 5-25.

Adair L. 2004; Fetal adaptations to maternal nutritional status during pregnancy. *American Journal of Physical Anthropology*, Vol. 123, Suppl. 38: 50.

AlQahtani, S. J. Hector, M. P. and Liversidge, H. M. 2010; The London Atlas of Human Tooth Development and Eruption. *American Journal of Physical Anthropology,* Vol. 142: 481-490.

AlQahtani, S. J. Hector, M. P. and Liversidge, H. M. 2014; Accuracy of Dental Age Estimation Charts: Schour and Massler, Ubelaker, and the London Atlas. *American Journal of Physical Anthropology,* Vol. 154: 70-78.

Armelagos, G. J., Goodman, A. H., Harper, K. N. and Blakey, M. L. 2009; Enamel Hypoplasia and Early Mortality: Bioarchaeological Support for the Barker Hypothesis. *Evolutionary Anthropology,* Vol. 18: 261-271.

Babones, S. J. 2008; Income inequality and population health: Correlation and causality. *Social Science and Medicine,* Vol. 66: 1614-1626.

Bang, G. 1989; Age changes in teeth; developmental and regressive. *Age Markers in the Human Skeleton*, Vol. 1: 211-235.

Barker, D. J. P., Lampl, M., Roseboom, T. and Winder, N. 2012; Resource allocation in utero and health in later life. *Placenta,* Vol. 33: 30-34.

Bateson, P., Barker, D., Clutton-Brock, T., Deb, D., D'Udine, B., Foley, R. A., Gluckman, P., Godfrey, K., Kirkwood, T., Mirazón Lahr, M., McNamara, J., Metcalfe, N. B., Monaghan, P., Spencer, H. G. and Sultan, S. E. 2004; Developmental plasticity and human health. *Nature,* Vol. 430: 419-421.

Baxter, J. E. 2005; The Archaeology of Childhood: Children, Gender, and Material Culture. California: AltaMira Press.

Beaudrap, P., Turyakira, E., White, L. J., Nabasumba, C., Tumwebaze, B., Muehlenbachs, A., Guérin, P., Boum, Y., McGready, R. and Piola, P. 2013; Impact of malaria during pregnancy on pregnancy outcomes in a Ugandan prospective cohort with intensive malaria screening and prompt treatment. *Malaria Journal,* Vol. 12 (1): 139.

Boas, F. 1930; Observations on the growth of children. *Science,* Vol. 72: 44-48.

Boersma, G. J. and Tamashiro, K. L. 2015; Individual differences in the effects of prenatal stress exposure in rodents. *Neurobiology of Stress,* Vol. 1: 100-108.

Bogin, B. 1999; *Patterns of Human Growth* (2nd Edition). Cambridge: Cambridge University Press.

Bolaños, M. V., Manrique, M. C., Bolaños, M. J. and Briones, M. T. 2000; Approaches to chronological age assessment based on dental calcification. *Forensic Science International,* Vol. 110: 97-106.

Bush, H. and Zvelebil, M. 1991; Pathology and health in past societies: an introduction. In H. Bush and M. Zvelebil (Eds.) *Health in Past Societies: Biocultural interpretations of human skeletal remains in archaeological contexts* (BAR International Series 567). Oxford: BAR Publishing: 3-9.

Cardoso, H. F. V. 2007; Environmental Effects on Skeletal Versus Dental Development: Using a Documented Subadult Skeletal Sample to Test a Basic Assumption in Human Osteological Research. *American Journal of Physical Anthropology,* Vol. 132: 223-233.

Chiswick, M. L. 1985; Intrauterine growth retardation. *British Medical Journal,* Vol. 291: 845-848.

Couoh, L. R. 2017; Differences between biological and chronological age-at-death in human skeletal remains: A change of perspective. *American Journal of Physical Anthropology,* Vol. 163: 671-695.

Coussons-Read, M. E., Lobel, M., Carey, J. C., Kreither, M. O., D'Anna, K., Argys, L., Ross, R. G., Brandt, C. and Cole, S. 2012; The occurrence of preterm delivery is linked to pregnancy-specific distress and elevated inflammatory markers across gestation. *Brain, Behaviour and Immunity,* Vol. 26 (4): 650-659.

Crawford, M. A., Doyle, W. and Meadows, N. 1987; Gender differences at birth and differences in fetal growth. *Human Reproduction,* Vol. 2: 517–20.

DeWitte, S. N., Hughes-Morey, G, Bekvalac, J. and Karsten, J. 2016; Wealth, health and frailty in industrial-era London. *Annals of Human Biology,* Vol. 43 (3): 241-254.

Donald, H. H. 1952; *The Negro Freedman.* New York: Henry Schuman.

Elamin, F. and Liversidge, H. M. 2013; Malnutrition has no effect on the timing of human tooth formation. *PLoS ONE* 8: e72274.

Falys, C. G. and Lewis, M. E. 2011; Proposing a way forward: A review of standardisation in the use of age categories and ageing technique in osteological analysis. *International Journal of Osteoarchaeology,* Vol. 21: 704-716.

Fazekas, I. G. and Kósa, F. 1978; *Forensic Foetal Osteology.* Budapest: Academic Press.

Fell, D. B., Savitz, D. A., Kramer, M. S., Gessner, B. D., Katz, M. A., Knight, M., Luteijn, J. M., Marshall, H., Bhat, N., Gravett, M. G., Skidmore, B. and Ortiz, J. R. 2016; Maternal influenza and birth outcomes: systematic review of comparative studies. *International Journal of Obstetrics and Gynaecology,* Vol. 124: 48-59.

Floud, R., Wachter, K.W. and Gregory, A. 1990; *Height, health and history: nutritional status in the United Kingdom, 1750–1980.* Cambridge: Cambridge University Press.

Fujita, M., Lo, Y. J. and Brindle, E. 2017; Nutritional, inflammatory, and ecological correlates of maternal retinol allocation to breast milk in agro-pastoral Ariaal communities of northern Kenya. *American Journal of Human Biology,* Vol. 29 (Early View): 1-14.

García, A. R., Gurven, M. and Blackwell, A. D. 2017; A matter of perception: Perceived socio-economic status and cortisol on the island of Utila, Honduras. *American Journal of Human Biology* [Early View].

Garn, S. M. Lewis, A. B. and Polacheck, D. L. 1960; Interrelations in dental development. I. Interrelationships within the dentition. *Journal of Dental Research,* Vol. 39: 1049-1055.

Garn, S. M, Lewis A. B, Blizzard, R. M. 1965a; Endocrine factors in dental development. *J Dent Res,* Vol. 44:243–258.

Garn, S. M, Lewis, A.B, Kerewsky, R. S. 1965b; Genetic, nutritional and maturational correlates of dental development. *J Dent Res,* Vol. 44:228–243.

Gindhart, P. S. 1989; An Early Twentieth-Century Skeleton Collection. *Journal of Forensic Sciences,* Vol. 34 (4): 887-893.

Gluckman, P. D. 1997; Endocrine and nutritional regulation of prenatal growth. *Acta Paediatrica* Supplementary Series, Vol. 423: 153-157.

Goldenberg, R. L & Thompson, C. 2003; The infectious origins of stillbirth. *American Journal of Obstetric Gynecology,* Vol. 189, No. 3: 861-873.

Goodman, A.H. and Martin, D. L. 2002; Reconstructing health profiles from skeletal remains. In R. H. Steckel and J.C. Rose (Eds.) *The Backbone of History: Health and Nutrition in the Western Hemisphere.* Cambridge: Cambridge University Press:11-60.

Goodman, A. H., Thomas, R. B., Swedlund, A. C. and Armelagos, G. J. 1988; Biocultural Perspectives on Stress in Prehistoric, Historical, and Contemporary Population Research. *Yearbook of Physical Anthropology,* Vol. 31: 169-202.

Gowland, R. L. 2001; Playing Dead: implications of mortuary evidence for the social construction of childhood in Roman Britain. In G. Davies, A. Gardner and K. Lockyear (Eds.) *TRAC 2000: Proceedings of the Tenth Annual Theoretical Roman Archaeology Conference, London 2000.* Oxford: Oxbow Books: 152-168.

Gowland, R. L. 2002; Age as an Aspect of Social Identity in Fourth- to Sixth-Century AD England: The Archaeological Funerary Evidence. Ph.D. Thesis, University of Durham, Durham, UK.

Gowland, R. L. 2006; Ageing the past: Examining age identity from funerary evidence. In R. L. Gowland and C. Knüsel (Eds.) *Social Archaeology of Funerary Remains*. Oxford: Oxbow:143-154.

Gowland, R. L. 2015; Entangled Lives: Implications of the Developmental Origins of Health and Disease Hypothesis for Bioarchaeology and the Life Course. *American Journal of Physical Anthropology*, Vol. 158, No. 4: 530-540.

Green, M. S. 1992; The male predominance in the incidence of infectious diseases in children: A postulated explanation for disparities in the literature. *International Journal of Epidemiology,* Vol. 21: 381–386.

Halcrow, S. E., Tayles, N. and Elliot, G. E. 2018; The Bioarchaeology of Fetuses. In S. Han, T. K. Betsinger, and A. B. Scott (Eds.) *The Fetus: Biology, Culture, and Society.* New York: Berghahn Books: 83-111.

Halfon, N., Larson, K., Lu, M., Tullis, E. and Russ, S. 2014; Lifecourse Health Development: Past, Present and Future. *Maternal and Child Health Journal,* Vol. 18: 344-365.

Harding, J. E. and Johnston, B. M. 1995; Nutrition and Fetal Growth. *Reproduction, Fertility and Development,* Vol. 7, No. 3: 539-548.

Heinke, D and Kuzawa, C. W. 2008; Self-reported illness and birth weight in the Philippines: implications for hypotheses of adaptive fetal plasticity. *American Journal of Human Biology*¸ Vol. 20: 538-544.

Hillson, S. W. 2005; *Teeth* (Second Edition). Cambridge: Cambridge University Press.

Holmes, S. J. 1937; *The Negro's Struggle for Survival: A Study in Human Ecology.* Berkeley: University of California Press.

Huda, T. F. J. and Bowman, J. E. 1995; Age Determination From Dental Microstructure in Juveniles. *American Journal of Physical Anthropology,* Vol. 97: 135-150.

Humphrey, L. 2000; Interpretations of the growth of past populations. In J. S. Derevenski (Ed.) *Children and Material Culture*. London: Routledge: 193–205.

Hunt, D. [Personal Communication: October 2015]; *Brief background to the fetal collections housed at the NMNH* (Unpublished).

Huxley, A. K. and Angevine, J. B. 1998; Determination of Gestational Age from Lunar Age Assessments in Human Fetal Remains. *Journal of Forensic Science,* Vol. 43 (6): 1254-1256.

Issel, E. P. 1985; Ultrasonic measurement of the growth of fetal limb bones in normal pregnancy. *Journal of Perinatal Medicine,* Vol. 13: 305-313.

Jeanty, P., Rodesch, F., Delbeke, D. and Dumont, J. E. 1984; Estimation of Gestational Age from Measurements of Fetal Long Bones. *Journal of Ultrasound Medicine,* Vol. 3: 75-79.

Jeanty, P. and Romero, R. 1984; Estimation of Gestational Age. *Seminars in Ultrasound, CT and MRI,* Vol. 5: 121-129.

Klaus, H. D. 2012; The Bioarchaeology of Structural Violence. In D. L. Martin, R. P. Harrod, and V. R. Perez (Eds.) *The Bioarchaeology of Violence. Bioarchaeological Interpretations of the Human Past: Local, Regional, and Global.* Gainesville: University Press of Florida: 29–62.

Kósa, F. 2002; Anthropological study for the determination of the Europid and Negroid characteristics on facial bones of human fetuses. *Acta Biologica Szegediensis,* Vol. 46 (1-2): 83-90.

Lewis, M. E. 2007; *The Bioarchaeology of Children: Perspectives from Biological and Forensic Anthropology.* Cambridge: Cambridge University Press.

Lewis, M. E. 2017; *Paleopathology of Children: Identification of Pathological Conditions in the Human Skeletal Remains of Non-Adults.* London: Academic Press.

Lewis, M. E. 2018; Fetal Paleopathology: An Impossible Discipline? In S. Han, T. K. Betsinger, and A. B. Scott (Eds.) *The Fetus: Biology, Culture, and Society.* New York: Berghahn Books: 112-131.

Lewis, M. E. and Roberts, C. 1997; Growing Pains: the interpretation of Stress Indicators. *International Journal of Osteoarchaeology,* Vol. 7: 581-586.

Malina, R. M., Brown, K. H. and Zavaleta, A. N. 1987; Relative Lower Extremity Length in Mexican American and in American Black and White Youth. *American Journal of Physical Anthropology,* Vol. 72: 89-94.

Martorell, R. and Habicht. J. P. 1986; Growth in early childhood in developing countries. In F. Falkner and J. Tanner (Eds.) *Human Growth: methodology ecological, genetic, and nutritional effects on growth.* New York: Plenum Press: 241-262.

Martorell, R., Malina, R. M., Castillo, R. O., Mendoza, F. S. and Pawson, I. G. 1988; Body Proportions in Three Ethnic Groups: Children and Youth 2-17 Years in NHANES II and HHANES. *Human Biology,* Vol. 60 (2): 205-222.

Melby, M. K., Yamada, G. and Surkan, P. J. 2016; Inadequate Gestational Weight Gain Increases Risk of Small-for-Gestational-Age Term Births in Girls in Japan: A Population-Based Cohort Study. *American Journal of Human Biology,* Vol. 28: 714-720.

Moorrees, C. F. A. Fanning, E. A. and Hunt, E. E. 1963a; Formation and Resorption of Three Deciduous Teeth in Children. *American Journal of Physical Anthropology*, Vol. 21:205-213.

Moorrees, C. F. A. Fanning, E. A. and Hunt, E. E. 1963b; Age Variation of Formation Stages for Ten Permanent Teeth. *Journal of Dental Research*, Vol. 42: 1490-1502.

Nicholas, S. and Steckel, R. H. 1991; Heights and living standards of English workers during the early years of industrialization, 1770–1815. *Journal of Economic History*, Vol. 51: 937–957.

Nyati, L. H., Norris, S. A., Cameron, N. and Pettifor, J. M. 2006; Effect of Ethnicity and Sex on the Growth of the Axial and Appendicular Skeleton of Children Living in a Developing Country. *American Journal of Physical Anthropology*, Vol. 130: 135-141.

Nystrom, K. C. 2014; The Bioarchaeology of Structural Violence and Dissection in the 19th-Century United States. *American Anthropologist*, Vol 116: 765-779.

Pomeroy, E., Stock, J. T., Stanojevic, S., Miranda, J. J., Cole, T. J., and Wells, J. C. K. 2012; Trade-Offs in Relative Limb Length among Peruvian Children: Extending the Thrifty Phenotype Hypothesis to Limb Proportions. *Plos One*¸ Vol. 7 (12): 1-10.

Redfield, A. 1970; A New Aid to Aging Immature Skeletons: Development of the Occipital Bone. *American Journal of Physical Anthropology*, Vol. 33: 207-220.

Robb, J. 2002; Time and biography: Osteobiography of the Italian Neolithic lifespan. In Y. Hamilakis, M. Pluciennik, and S. Tarlow (Eds.) *Thinking Through the Body: Archaeologies of Corporeality*. London: Kluwer Academic/Plenum:153-171.

Rogers, A. 1997; Vulnerability, health and healthcare. *Journal of Advanced Nursing*, Vol. 26: 65-72.

Roth, E. A. 1992; Applications of demography models to paleodemography. In S. R. Saunders and M. A. Katzenberg (Eds.) *Skeletal Biology of Past Peoples: Research Methods*. New York: Wiley-Liss: 175–188.

Ruff, C. B. Garofalo, E. and Holmes, M. A. 2013: Interpreting Skeletal Growth in the Past From a Functional and Physiological Perspective. *American Journal of Physical Anthropology,* Vol 150: 29-37.

Said-Mohamed, R., Pettifor, J. M. and Norris, S. A. 2018; Life History theory hypotheses on child growth: Potential implications for short and long-term child growth, development and health. *American Journal of Physical Anthropology,* Vol. 165: 4-19.

Saunders, S., Hoppa, R. and Southern, R. 1993; Diaphyseal Growth in a Nineteenth Century Skeletal Sample of Subadults from St Thomas' Church, Belleville, Ontario. *International Journal of Osteoarchaeology,* Vol. 3: 265-281.

Schaefer, M. Black, S. & Scheuer, L. 2009; *Juvenile Osteology: A Laboratory and Field Manual.* London: Elsevier Inc.

Schell L. M. 1981; Environmental noise and human prenatal growth. *American Journal of Physical Anthropology,* Vol. 56: 63–70.

Scheuer L. and Black S. (2004) *The Juvenile Skeleton*. London: Elsevier.Stinson

Scheuer, L. and Maclaughlin-Black, S. 1994; Age Estimation from the Pars Basilaris of the Fetal and Juvenile Occipital Bone. *International Journal of Osteoarchaeology,* Vol. 4: 377-380.

Scheuer, L. Musgrave, J. H. & Evans, S. P. 1980; The estimation of late fetal and perinatal age from limb bone length by linear and logarithmic regression. *Annals of Human Biology,* 7 (3): 257-265.

Schour, I. and Massler, M. 1941a; The development of the human dentition. *Journal of the American Dental Association*, Vol. 28:1153–1160.

Schour, I. and Massler, M. 1941b; *Development of human dentition chart* (2nd edition). Chicago: American Dental Association.

Sherwood, R. J., Meindl, R. S., Robinson, H. B. and May, R. L. 2000; Fetal Age: Methods of Estimation and Effects of Pathology. *American Journal of Physical Anthropology,* Vol. 113: 305-315.

Sinclair, D. 1985; *Human Growth After Birth* (4th Edition). Oxford: Oxford University Press.

Sofaer, J. R. 2006; *The Body as Material Culture: A Theoretical Osteoarchaeology*. Cambridge: Cambridge University Press.

Sofaer, J. 2011; Towards a Social Bioarchaeology of Age. In S.C. Agarwal and B.A. Glencross (Eds.) *Social Bioarchaeology*. Oxford: Wiley-Blackwell: 385-311.

Steckel, R. H. 2009; Heights and human welfare: recent developments and new directions. *Explorations in Economic History*, Vol. 46: 1-23.

Stinson, S. 2000; Growth variation: biological and cultural factors. In S. Stinson, B. Bogin, R. Huss-Ashmore and D. H. O'Rourke (Eds.) *Human biology: an evolutionary and biocultural perspective*. New York: Wiley-Liss: 434-438.

Stoodley, N. 2000; From the Cradle to the Grave: Age Organization and the Early Anglo-Saxon Burial Rite. *World Archaeology,* Vol. 31 (3): 456-472.

Synnes, A. R., Ling, E. W. and Whitfield, M. F. 1994; Perinatal outcomes of a large cohort of extremely low gestational age infants (twenty-three to twenty-eight weeks of gestation). *Journal of Pediatrics,* Vol. 125: 952–960.

Tanner, J. M. 1978; *Foetus Into Man: Physical Growth from Conception to Maturity.* London: Open Books Publishing Ltd.

Ubelaker, D. H. 1978; *Human skeletal remains: excavation, analysis, interpretation*. Chicago: Aldine Publishing Co. Inc.

Ulizzi, L. and Zonta, L. A. 2002; Sex Differential Pattern in Perinatal Deaths in Italy. *Human Biology*, Vol. 74 (6): 879-888.

Wiley, A. S. and Pike, I. L. 1998; An Alternative Method for Assessing Early Mortality in Contemporary Populations. *American Journal of Physical Anthropology*¸ Vol. 107: 315-330.

Chapter 9: Manuscript 4

Perinatal Pathology in Bioarchaeology: Investigating the Potentials, Problems and Implications.

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KEY WORDS: *Fetal; Infant; Health; Wellbeing; Stress; Lesions*

Abstract: *Pathological lesions identified within the skeleton represent physiological response to detrimental and harmful onslaughts. These can range in etiology and pathogenicity and may be acute or chronic. Despite this variability, identifiable pathological changes to the skeleton are able to reveal insights into past health, and the biocultural impetus and response to these insults. Investigation into intrauterine and early extrauterine life has recently intensified, revealing how these initial experiences are vital for health and wellbeing across the life course. Yet, relatively little attention has been focused on the identification and interpretation of pathological lesions in fetal, perinatal and infant individuals. Consequently, many uncertainties still surround the study and reporting of non-adult pathology. This study analysed 423 fetal, perinatal and infant skeletons from a wide temporal span (Iron Age to 20th century) to explore the prevalence and characteristics of pathological lesions within these individuals. Addressing the continuing debate surrounding differentiation between normal and pathological new bone formation, this study extensively details location, type and severity of pathological changes by both time period and gestational age. New bone formation was the most typical lesion identified and is often suggestive of systemic, infectious and/or metabolic disturbances. Changes to the frontal bone and tibia were the most prevalent, though lamellar and woven bone respectively were found to be the most common type of NBF identified within these elements. Pathological changes were identified in both pre- and postnatal individuals, indicative of pervasive poor maternal health. This study attempts to provide a systematic way in which to record pathological lesions in non-adults, generating an important narrative regarding the nexus between mother and child, and the impact and experience of health stress in the past.*

Introduction

Assessment of skeletal remains for evidence of pathological lesions has long been considered the most direct way of accessing evidence of disease in the past (Waldron 2009; Rivera & Lahr 2017; Bush & Zvelebil 1991). Associations between a range of diseases (pathogens) and changes to skeletal morphology have been established (pathogenesis) (Roberts & Manchester 2010), with the diagnostic criteria for the interpretation of pathological lesions in skeletal remains constantly being improved (e.g. Ortner 2003; Aufderheide & Rodriguez-Martin 1998). Such analyses, when fully contextualised, enable a consideration of 'local biologies' and the social and cultural implications of skeletal pathologies (Lock 1993; Ortner 2003; Roberts 2017). Central to bioarchaeological interpretation of pathological lesions are factors such as diet, living conditions, sanitation and access to health care (Krenz-Niedbala & Lukasik 2017; Cattaneo 1991), all of which are variables that can determine both exposure and susceptibility to disease.

Diagnosis of skeletal pathology must rely on a strong clinical knowledge-base (Roberts & Manchester 2010) – understanding how disease and poor health affects the body today is imperative for determining the skeletal manifestations in past populations. However, there are a number of limitations to be faced when applying clinical criteria to the bioarchaeological record Firstly, there may be differences between the clinical and archaeological presentation and manifestations of disease; indeed, prior to the antibiotic era, the progress of a number of diseases would have gone unchecked, leading to more severe skeletal changes (Roberts & Manchester 2010). Acute disease rarely affects the bones, meaning palaeopathological evidence is primarily limited to chronic evidence of conditions. Clinicians are primarily concerned with soft tissue manifestations of disease and their observations and interpretations of skeletal lesions are often limited, making direct comparison between modern and archaeological data-sets problematic. Bone has a limited response to health/disease insults (bone growth and bone destruction), meaning a number of pathological lesions have multiple and overlapping etiologies, making identification and interpretation of the lesions an even greater challenge (Goodman & Armelagos 1989; Gowland 2004; Ortner 2008; Roberts & Manchester 2010). Individual expression of disease/illness must also be considered; the same disease will not affect everyone identically (Reitsmea & McIlvaine 2014; Temple & Goodman 2014). As Gowland (2004) states, disease risk and expression is culturally contingent; individuals will be differentially exposed to health risks and treatment of

conditions may be based on gender, status and age. Such factors may also lead to differential access to nutrition, sanitary living conditions and a predisposition to occupation, all of which may result in differences in vulnerability and susceptibility to disease (Gowland 2004). Thus, constructing and establishing health status for archaeological individuals is a complex practice.

The pathological assessment of fetal, perinatal and infantile skeletal remains is further limited by the current corpus of bioarchaeological and medical literature, for which precise description and definition of skeletal pathologies is lacking. In addition, there is limited clinical literature regarding the appearance and differentiation of true pathological bone from that produced during normal growth (Lewis 2017a). The lack of clear photographic and descriptive evidence for the appearance of normal new bone formation (NBF) in the medical and anatomical literature only limits the discussion further. Consequently, the validity of the evidence of pathological change in fetal and infant remains has been brought into question (Lewis 2017a). Multiple methods for identifying the presence of disease/illness have been employed to identify lesions, including macroscopic and microscopic analysis, radiography, bone histology, ancient DNA and isotopic analyses (e.g. Beaumont *et al.* 2015; Faerman 1999). However, although these studies may indicate physiological 'stress' (e.g. malnutrition), or determine the presence of a particular pathogen, definitive etiologies and clear documentation of pathological changes in infants are still lacking. Consequently, macroscopic analysis of fetal, perinatal and infant individuals typically still relies on only a few textual sources (e.g. Ortner 2003; Lewis 2007; Aufderheide & Rodriguez-Martin 2008).

This study presents evidence for pathological lesions, including appearance and distribution in the skeleton, from a large sample of fetal, perinatal and infant skeletons from different time periods. This study describes the location and appearance of putative pathological changes and develops a systematic way to classify and record lesions. Furthermore, this study considers pathological lesions within a biocultural sphere, reflecting on the infant-mother dyad as well as the varying social, cultural and status-dependent environments these individuals were exposed to.

Materials

A total of 423 individuals from 14 different archaeological sites, and one 20th century medical collection, were assessed (Table1.1). The sites date from the Iron Age (3rd century B.C.)

through to the post-medieval period (20th century). Sample sizes by time period have been given in Table 1.2. Individuals in the category labelled 'Transition' date to the 1st Century AD and are from sites which include both Iron Age and Roman material. The undated individuals are all from the sites of Owslebury or Barton Court Farm. The 20th century medical collection is a partially documented sample, with some individuals having recorded biological age and sex.

The samples from Owslebury, Piddington and Barton Court Farm are the only rural sites included and while some of these individuals are associated with Roman villas, the status of these individuals is not known (Collis 1968; 1970; 1977; Friendship-Taylor & Friendship-Taylor 2012; Miles 1986), The post-Medieval samples of Cross Bones, St Brides and Broadgate are low-status individuals from the City of London (Harding 2002; Kausmally 2008; Mikulski 2007; Brickley *et al.* 1999; Hunt *personal communication*). Those from Chelsea Old Church, also located in London, were likely to have been from high-status families living a semi-rural existence within the suburbs (Museum of London 2009). Those from the post-Medieval site of St. Benet Sherehog are of middling status, whilst the individuals curated within the Smithsonian fetal collection are those of low status individuals dating from the 20th century. The remaining individuals Medieval and post-Medieval London are of unknown status.

In this study, fetus refers to those 36 gestational weeks of age (GWA) or younger (Lewis 2018, 1), perinatal is ascribed to those between 36 and 44 GWA (Lewis 2018, 1), and infant identifies those 44 GWA and older (up to 6 months or 64 GWA). Throughout the rest of this study all ages are referred to in GWA for ease of comparison between individuals. Table 1.3 provides the frequency of individuals assessed by dental age.

The total sample analysed (*N*=423) comprises of individuals aged six months (64 gestational weeks of age) or under. Typically, age estimates are derived from long bone and thus, those assessed, were those skeletally determined and recorded to be 64 GWA or younger. As this study was guided by the existing databases and age estimates derived for these individuals, three individuals were found to be dentally over 64 GWA. However, as they were still skeletally aged to be younger than 64 GWA, and were already included within the sample, they have been retained in analysis.

TABLE 1.1 Number of individuals by sample. Status for these samples has also been documented where known.

Sample	**Status**	**Total *N***
Owslebury	Unknown	23
Piddington	Unknown	24
Barton Court Farm	Unknown	52
Medieval St. Benet Sherehog	High	3
Spital Square	Unknown	1
East Smithfield	Unknown	8
St. Mary Graces	Unknown	3
Post-Medieval St. Benet Sherehog	Middle	19
Broadgate	Low	21
St. Thomas' Hospital	Low	5
St. Bride's Lower	Low	52
Chelsea Old Church	High	7
Cross Bones	Low	58
Royal London Hospital	Unknown	7
Fetal Collection, Smithsonian Institute	Low	140
Total		423

TABLE 1.2 Number of individuals by chronological time period.

Time Period	Pre-Roman	Transition	Roman	Saxon	Medieval	Post-Medieval	20th Century	Undated
Total (*N*=423)	16	27	39	2	15	169	140	15

TABLE 1.3 Number of individuals by dental age estimate (in GWA).

Dental Age Estimate (GWA)	30	34	36	38	39	40	42	43	46	52	58	64	70	76	82	Unknown
Total (N=423)	1	7	3	23	6	16	1	11	59	43	21	15	1	1	1	214

Methods

Ages-at-death are provided in gestational weeks and were calculated using dental development. The stage of dental development was recorded using standards outlined by Moorrees *et al.* (1963a; 1963b) and conversion to chronological ages-at-death estimated using AlQahtani *et al.* (2010). Dental development is considered to be a more accurate and reliable measure of age-at-death (Hillson 2005; Bang 1989; Moorrees *et al.* 1963b); the growth of deciduous and permanent dentition has been found to be less susceptible to fluctuations in environmental stress than skeletal growth (AlQahtani *et al.* 2014; Šešelj 2013; Bang 1989; Garn *et al.* 1960; Hillson 2005; AlQahtani *et al.* 2010). Thus, dental development is considered to be genetically regulated, and hence, is most robust against a multitude of perturbations that could be experienced *in utero* (for example: dietary, health, social, cultural stresses) (AlQahtani *et al.* 2014; Ruff *et al.* 2013).

Where this study considers pathological lesions by age, these ages have solely been determined from dental development. Only those individuals where dentition was available for assessment (*N*= 210) have thus been used in assessment of pathological lesions by age estimate. However, as one individual could only be recorded has having dental development of below 30 GWA they have been removed from pathological assessment by age. Consequently, 209 individuals have had pathological lesions considered by gestational age estimate. Three individuals had dental age estimates above 64 GWA (6 months of age) (Table 1.3), however, their skeletal development was suggested to be much younger and falls within the infant age range. Therefore, these three individuals were retained within this study and the subsequent analyses.

Assessment of pathology was undertaken macroscopically and relied on detailed assessment of the skeletal individuals. Each pathological change/lesion was recorded descriptively by the author and documented photographically. Pathological assessment within this thesis has relied on three primary avenues of investigation: location, type and severity of pathology.

1. **Location of pathology:**
   Location was firstly documented as cranial or postcranial, then by specific skeletal element and then by aspect. By employing this recording strategy the patterning of

pathological lesions could be assessed by skeletal element. Given that some diseases/infections/conditions are known to affect certain elements and certain locations more commonly it was considered that recording the location of pathology was of particular importance.

2. **Type of pathology:**
   Type of lesion was recorded as either bone formation, bone destruction/resorption, metaphyseal expansion or morphological change. Bone formation was recorded as 'NBF' (New Bone Formation) and bone destruction/resorption was recorded as being 'Lytic'. For NBF, type of bone formation was also recorded as either woven or lamellar bone, and also whether it spiculated (Ortner 2003).

3. **Severity of pathology:**
   Severity of pathological change has been recorded in accordance with a grading system established by the author (See below for details). Grading systems were developed to consider severity of NBF, lytic lesions and metaphyseal expansion. For each type of pathology, a severity score of 1, 2 or 3 has been afforded.

Table 2. details the variables recorded for each of these categories, whilst Table 3.1 descriptively outlines the grading systems employed to determine severity of NBF, lytic lesions and metaphyseal expansion. Images taken from individuals within this study have been used to support these grading categories (Table 3.2). It is hoped that this grading system will provide a systematic way for fetal, perinatal and infant pathological lesions to be categorised in the future.

True prevalence rates of pathological lesions were calculated by documenting how many individuals out of the 423 analysed had a particular skeletal element(s) present, and then out of those where the element(s) could be observed, how many showed pathological changes. Totals have been given for numbers of individuals/skeletal elements observed and the number affected. Results have typically been given in percentages affected (%) out of total observed (*N*). However, these TPRs may be better expressed as corrected CPRs (Crude Prevalence Rates) as

for some elements, such as long bones, there are bilateral pairs of that element. This study does not calculate prevalence rates for each element by side, instead amalgamating these bilateral pairs. Thus, TPRs may be considered as corrected CPRs.

Chi-squared tests for independence at 99.5 % confidence ($p < 0.05$) were also employed for pathological categories to observe whether there was any relationship between various pathological variables. Chi-square results are presented numerically, where $p < 0.05$ shows there is a significant relationship between the variables. Chi-Squared values (X^2) have also been given.

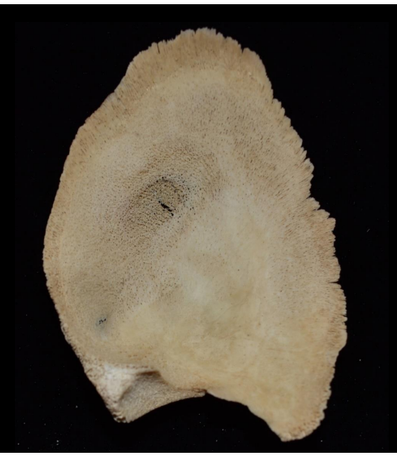
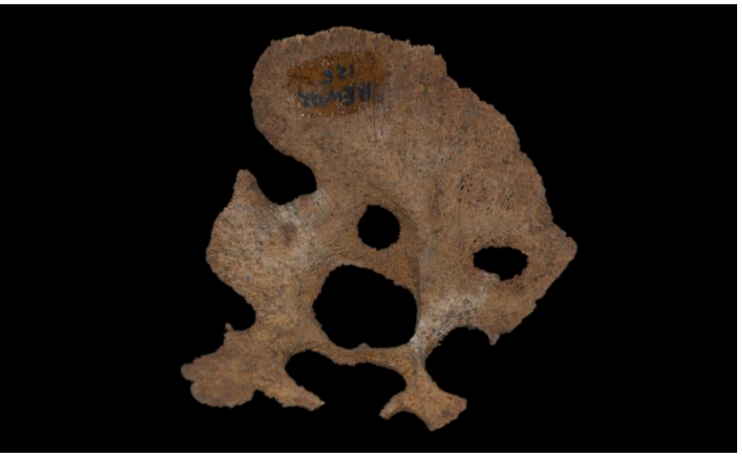
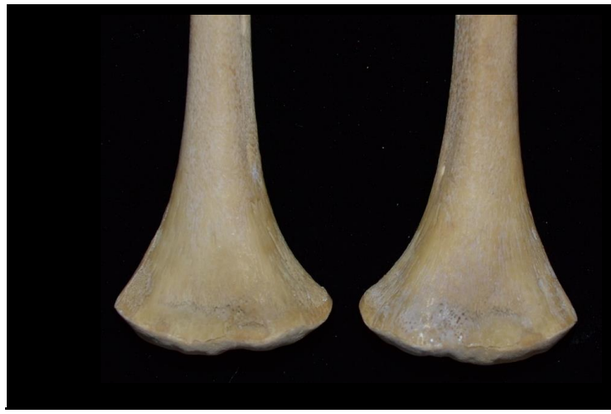
TABLE 2. Categories and variable used in the recording of pathological lesions.

Category	**Variable**
Location	Cranial or Postcranial
Skeletal Element	e.g. Femur, Tibia, Frontal Bone
Aspect	e.g. Endocranial, Anterior, Circumferentially
Type I	NBF, Lytic, Metaphyseal Extension, Morphological change.
Type II	Woven, Lamellar and/or Spiculated
Severity	Grade 1, 2, or 3

TABLE 3.1 Descriptive grading systems for new bone formation, lytic lesions and metaphyseal expansion employed for assessment of pathological lesions within this study.

	Grade 1	Grade 2	Grade 3
New Bone Formation	New bone formation, which may be woven or lamellar in appearance, will be considered to be grade 1 when the NBF is not clearly apparent and the margins are unable to be clearly defined from that of normal cortical bone. Grade 1 NBF is likely to be isolated in location, appearing minimally across the skeletal element.	New bone formation recorded as being grade 2 will be clearly identifiable as a definable area of woven or lamellar bone formation. There will be clear boundaries/borders to the NBF and it will obviously differ from the normal cortical bone of the skeletal element. Grade 2 NBF is likely to be distinguishable as a clear layer of bone on top of the original cortical surface. It is likely that NBF listed within this category will be formed of a single layer though may extend over a large aspect area of the skeletal element.	New bone formation recorded as being grade 3 will be the more severe type of NBF, with clear, multi-layered or thick NBF across a large area/aspect of the skeletal element. The NBF may be woven or lamellar in appearance and is clearly seen to be on top of the original cortical bone.
Lytic Lesions	Lytic lesions considered to be grade 1 likely consist primarily of macro-porosity. This porosity will be relatively minor, though may extend over a large skeletal area, and no clear destruction of the cortical bone will be apparent.	Lytic lesions considered to be grade 2 will likely show evidence of some cortical destruction as well as porosity. However, cortical destruction will not be widespread throughout the skeletal element and is instead likely to be in isolated concentrations.	Lytic lesions considered to be grade 3 will show extensive cortical destruction and/or porosity. Destruction will be widespread throughout the element.
Metaphyseal Expansion	Metaphyseal expansion considered to be grade 1 will likely consist of noticeably widened/flared metaphyses which do not appear proportional for the long bone diaphysis. However, despite this expansion no change to the metaphyseal margin or trabecular bone structure will be observed.	Metaphyseal expansion will be considered to be grade 2 when involvement of the metaphyseal margin is apparent. This will result in atypical and misshapen metaphyseal margins often combined with a discernible brim/lip to the metaphysis.	Metaphyseal expansion considered to be grade 3 will be the most severe and where involvement of the trabecular bone structure can be seen. Individuals displaying grade 3 metaphyseal extension will likely have more porous metaphyses and the trabecular structure will appear clearly expanded and widened. Involvement of the metaphyseal margin may still be apparent though this may be lost due to the trabecular expansion.

TABLE 3.2 Photographic grading systems for new bone formation, lytic lesions and metaphyseal expansion employed for assessment of pathological lesions within this study.

Grade 1	Grade 2	Grade 3
New Bone Formation		
		
Lytic Lesions		
		
Metaphyseal Expansion		
		

Results:

Of the 423 individuals assessed, 209 had dentition available for assessment. Table 4. details the number and percent of individuals by dental age (in GWA) who had cranial and/or postcranial pathological lesions. Results show that, overlooking those age categories where only one individual was assessed, cranial pathology rates are around ~80%, compared to postcranial percentages which are ~30-50%. Chi square analysis found no statistical significant associations between cranial pathology and dental age ($X^2 = 23.93$, d.f. 14, $p = 0.13$) nor between postcranial pathology and dental age ($X^2 = 13.17$, d.f. 14, $p = 0.34$). This supports the finding that pathological lesions are found consistently throughout individuals of all dental ages, showing evidence of both pre- and postnatal pathological changes.

TABLE 4. Prevalence rates of cranial and postcranial pathology by dental age.

Dental Age (GWA)	***N***	**Cranial Pathology *N* (%)**	**Postcranial Pathology *N* (%)**
30	1	1 (100)	0 (0)
34	7	6 (86)	3 (43)
36	3	1(33)	0 (0)
38	23	18 (78)	10 (43)
39	6	5 (83)	1 (17)
40	16	13 (81)	9 (56)
42	1	1 (100)	1(100)
43	11	9 (82)	6 (55)
46	59	54 (92)	19 (32)
52	43	37 (86)	14 (33)
58	21	18 (86)	7 (33)
64	15	8 (53)	4 (27)
70	1	1 (100)	0 (0)
76	1	1 (100)	0 (0)
82	1	0 (0)	0 (0)

TABLE 5.1 Cranial elements by number observed and number affected out of all individuals assessed (N=423).

Skeletal Element	**Observed (*N*)**	**Affected *N* (%)**
Frontal Bone	225	215 (96)
Parietal Bone	178	162 (91)
Occipital Bone	320	162 (51)
Temporal Bone	252	29 (12)
Sphenoid	227	53 (23)
Zygomatic	220	18 (8)

TABLE 5.2 Postcranial long bones by number observed and number affected out of all individuals assessed (N=423).

Skeletal Element	**Observed (*N*)**	**Affected *N* (%)**
Femur	343	63 (18)
Tibia	317	77 (24)
Fibula	277	18 (6)
Humerus	364	39 (11)
Radius	342	21 (6)
Ulna	348	19 (5)

When pathological prevalence rates are considered by skeletal element for both cranial (frontal bone, parietal bone, occipital bone, temporal bone, sphenoid, zygomatic) and postcranial long bones (femur, tibia, fibula, humerus, ulna, radius) the frontal bone and tibia are found to be the most commonly affected elements (Tables 5.1 and 5.2).

When all individuals were assessed by time period for varying skeletal elements (Table 6.) similar patterns emerge; the frontal bone is typically the most affected cranial element and the tibiae the most affected postcranial element. Only within the 20th century sample does the femur marginally show greater prevalence of pathological lesions than the tibia. Saxon and undated individuals have been removed from this analysis, as Saxon individuals gave falsely elevated prevalence rates of pathology due to the small sample size (N=2), and undated individuals (N=15) do not further analysis of pathological lesions by time period.

Consideration of type of lesion by skeletal element (Table 7.) shows that NBF is the most commonly found lesion within both the cranial and postcranial elements assessed. Cranial vault bones then most typically show lytic lesions, though the sphenoid shows a high prevalence rate of morphological change. The limb bones assessed show no evidence of lytic lesions in comparison, though for all limb bones over 20% show evidence of metaphyseal expansion.

As new bone formation is the most commonly identified type of pathological change, consideration of the type of NBF (woven, lamellar, spiculated) was afforded for various cranial and postcranial elements (Table 8.). Woven bone is the most commonly identified type of NBF in all elements assessed, except for within the frontal bone and parietal bone. These elements show a much higher prevalence of lamellar bone formation in comparison. Only the sphenoid was identified to have evidence of spiculated NBF. Within the long bones, woven bone formation is also the most prevalent type of NBF.

Consideration of individuals with dental age estimates for evidence of pathological lesions to various skeletal elements (Table 9.) shows that, despite small sample sizes for some dental ages, there is comparability between prevalence rates of pathology for the various ages. Therefore, it appears that regardless of dental age, skeletal elements show similar prevalence rates of pathological changes. This means there is no pre- or postnatal peak in pathology identified for the majority of these skeletal elements. The only element where a postnatal

peak in pathology may be observed is in the occipital bone. Chi-squared assessment of prevalence of skeletal elements affected by dental age (Table 10.) suggests that there is only a significant association between pathology and dental age for both the occipital bone and parietal bone. Therefore, seemingly, the other skeletal elements show comparable pathological lesions between dental ages.

Considering severity of lesions to the frontal bone and tibia – the elements found to most commonly show pathological lesions – identified that severity of grade two is most commonly seen for both elements, for all dental ages. Grade three severity is the most unlikely score in almost all ages for both the frontal bone and tibia, and both pre- and postnatal individuals have been found to have lesions of this severity. Again this suggests that there is no peak in the prevalence and severity of pathological lesions by age. Furthermore, consideration of NBF, and its type (woven, lamellar) by dental age (Table 12.) for the femur, tibia and humerus has supported these conclusions, showing similar prevalence rates of NBF between ages.

TABLE 6. Skeletal elements by number observed and number found to show pathological changes by time period.

Skeletal Element	Pre-Roman		Transition		Roman		Medieval		Post-Medieval		20th Century	
	Observed (*N*)	Affected *N* (%)	Observed (*N*)	Affected *N* (%)	Observed (*N*)	Affected *N* (%)	Observed (*N*)	Affected *N* (%)	Observed (*N*)	Affected *N* (%)	Observed (*N*)	Affected *N* (%)
Frontal Bone	7	5 (71)	17	15 (88)	17	14 (82)	7	6 (86)	95	94 (99)	76	76 (100)
Parietal Bone	4	3 (75)	18	13 (72)	16	11 (69)	8	8 (100)	85	84 (99)	42	39 (93)
Occipital Bone	13	3 (23)	20	12 (60)	24	11 (46)	7	4 (57)	128	86 (67)	117	43 (37)
Temporal Bone	13	1 (8)	16	3 (19)	19	3 (16)	9	1 (11)	100	11 (11)	86	10 (12)
Sphenoid	11	2 (18)	15	6 (40)	19	6 (32)	9	3 (33)	98	16 (16)	117	19 (16)
Femur	11	1 (9)	21	10 (45)	29	3 (10)	12	4 (33)	134	28 (21)	125	15 (12)
Tibia	9	1 (11)	20	11 (55)	23	10 (43)	7	4 (57)	119	36 (30)	132	14 (11)
Humerus	12	0 (0)	21	3 (14)	27	2 (7)	14	3 (21)	146	19 (13)	135	11 (8)
Radius	10	0 (0)	22	2 (9)	25	0 (0)	14	2 (14)	132	8 (6)	131	8 (6)
Ulna	12	0 (0)	23	2 (9)	23	1 (4)	13	1 (8)	132	7 (5)	133	6 (5)

TABLE 7. Type of pathological lesion by skeletal element. Only those elements affected by pathological changes have been considered. For each skeletal element, type of pathological lesion percentages may go beyond 100% as some individuals were found to have multiple types of changes within a single element.

Skeletal Element	**Affected (*N*)**	**New Bone Formation *N* (%)**	**Lytic Lesions *N* (%)**	**Metaphyseal Expansion *N* (%)**	**Morphological Change *N* (%)**
Frontal Bone	215	204 (95)	13 (6)	-	6 (3)
Parietal Bone	162	151 (93)	7 (4)	-	5 (3)
Occipital Bone	162	153 (94)	9 (6)	-	8 (5)
Temporal Bone	29	16 (55)	6 (21)	-	7 (24)
Sphenoid	53	34 (64)	2 (4)	-	21 (40)
Femur	63	42 (67)	0 (0)	23 (37)	9 (14)
Tibia	77	63 (82)	0 (0)	17 (22)	7 (9)
Humerus	39	28 (72)	0 (0)	10 (26)	3 (8)
Radius	21	13 (62)	0 (0)	8 (38)	4 (19)
Ulna	19	12 (63)	0 (0)	5 (26)	5 (26)

TABLE 8. Type of new bone formation by skeletal element. Only those elements identified as having new bone formation have been considered. For each skeletal element, type of new bone formation percentages may go beyond 100% as some individuals were found to have multiple types of new bone formation within a single element.

Skeletal Element	**Affected (*N*)**	**Affected by Type of New Bone Formation**		
		Woven *N* (%)	**Lamellar *N* (%)**	**Spiculated *N* (%)**
Frontal Bone	204	50 (25)	172 (84)	0 (0)
Parietal Bone	151	34 (23)	121 (80)	0 (0)
Occipital Bone	153	129 (84)	27 (18)	0 (0)
Temporal Bone	16	10 (63)	6 (38)	0 (0)
Sphenoid	34	31 (91)	1 (3)	2 (6)
Femur	42	38 (90)	4 (10)	0 (0)
Tibia	63	58 (92)	5 (8)	0 (0)
Humerus	28	21 (75)	7 (25)	0 (0)
Radius	13	9 (69)	4 (31)	0 (0)
Ulna	12	9 (75)	3 (25)	0 (0)

TABLE 9. Number and percentage of pathological lesions by dental age for both cranial and postcranial elements. Only individuals with dental age estimates have been assessed.

Skeletal Element	Observed (*N*)	Affected (*N*)	Affected by Dental GWA (*N* Affected out of *N* Observed) (%)													
			30	**34**	**36**	**38**	**39**	**40**	**42**	**43**	**46**	**52**	**58**	**64**	**70**	**76**	**82**
Frontal Bone	149	142	1/1 (100)	6/6 (100)	1/1 (100)	16/17 (94)	5/5 (100)	12/13 (92)	1/1 (100)	9/9 (100)	45/48 (94)	29/30 (97)	11/11 (100)	5/5 (100)	1/1 (100)	0/0 (0)	0/1 (0)
Parietal Bone	131	119	0/0 (0)	3/3 (100)	1/1 (100)	7/10 (70)	3/3 (100)	10/10 (100)	1/1 (100)	8/8 (100)	40/44 (91)	31/32 (97)	11/13 (85)	4/5 (80)	0/0 (0)	0/0 (0)	0/1 (0)
Occipital Bone	190	122	0/1 (0)	3/7 (43)	0/2 (0)	6/20 (30)	3/6 (50)	9/15 (60)	1/1 (100)	8/11 (73)	41/57 (72)	27/37 (73)	13/18 (72)	9/12 75)	1/1 (100)	1/1 (100)	0/1 (0)
Temporal Bone	172	25	0/1 (0)	1/7 (14)	0/3 (0)	1/17 (6)	0/4 (0)	4/15 (27)	1/1 (100)	1/10 (10)	10/51 (20)	4/35 (11)	1/17 (6)	2/9 (22)	0/1 (0)	0/1 (0)	0/0 (0)
Sphenoid	160	40	1/1 (100)	3/7 (43)	0/3 (0)	4/17 (24)	2/6 (33)	5/13 (38)	0/1 (0)	1/10 (10)	17/52 (33)	4/33 (12)	1/10 (10)	2/6 (33)	0/1 (0)	0/0 (0)	0/0 (0)
Femur	164	39	0/1 (0)	1/4 (25)	0/3 (0)	2/19 (11)	1/6 (17)	4/11 (36)	1/1 (100)	4/10 (40)	12/46 (26)	6/32 (19)	5/18 (28)	3/12 (25)	0/1 (0)	0/0 (0)	0/0 (0)
Tibia	158	44	0/1 (0)	2/5 (40)	0/3 (0)	4/17 (24)	0/5 (0)	5/12 (42)	0/1 (0)	3/10 (30)	15/50 (30)	6/27 (22)	6/17 (35)	3/9 (33)	0/1 (0)	0/0 (0)	0/0 (0)
Humerus	191	27	0/1 (0)	1/7 (14)	0/3 (0)	4/23 (17)	0/6 (0)	3/13 (23)	0/1 (0)	4/11 (36)	6/52 (12)	3/41 (7)	3/18 (17)	3/14 (21)	0/1 (0)	0/0 (0)	0/0 (0)
Radius	177	15	0/1 (0)	1/6 (17)	0/3 (0)	1/22 (5)	0/6 (0)	1/13 (8)	0/1 (0)	2/11 (18)	3/51 (6)	2/35 (6)	2/16 (13)	2/11 (18)	0/1 (0)	0/0 (0)	0/0 (0)
Ulna	177	13	0/1 (0)	1/7 (14)	0/3 (0)	1/21 (5)	0/5 (0)	1/13 (8)	0/1 (0)	1/11 (9)	2/49 (4)	2/38 (5)	2/15 (13)	2/12 (17)	0/1 (0)	0/0 (0)	0/0 (0)

TABLE 10. Results of chi-squared analysis (X^2) of pathological lesions by dental age for various skeletal elements. P results highlighted in bold are those found to be statistically significant.

	Frontal Bone			**Parietal Bone**			**Occipital Bone**			**Humerus**			**Femur**			**Tibia**		
	***X*²**	**d.f.**	***p***	***X*²**	**d.f.**	***p***	***X*²**	**d.f.**	***p***	***X*²**	**d.f.**	***p***	***X*²**	**d.f.**	***p***	***X*²**	**d.f.**	***p***
Dental Age	22.806	14	0.053	27.969	14	**0.007**	24.318	14	**0.018**	10.805	14	0.67	11.390	14	0.639	8.12	14	0.691

TABLE 11. Assessment of severity of pathological lesions for the frontal bone and tibia by dental age. Only individuals with dental age estimates and pathological changes to either the frontal bone or tibia have been assessed. Again numbers and percentages of severity scores may not match number of individuals affected or 100% as some elements were afforded multiple severity scores due to multiple types of lesions being identified. Additionally, those elements identified as having morphological changes were not assigned a severity grade and therefore are not reflected within this assessment.

Dental Age	**Frontal Bone Affected (*N*)**	**Severity *N* (%)**			**Tibia Affected (*N*)**	**Elements by Severity *N* (%)**		
		Grade 1	**Grade 2**	**Grade 3**		**Grade 1**	**Grade 2**	**Grade 3**
30	1	0 (0)	1 (100)	0 (0)	0	-	-	-
34	6	3 (50)	2 (33)	0 (0)	2	1 (50)	0 (0)	1 (50)
36	1	0 (0)	1 (100)	0 (0)	0	-	-	-
38	16	2 (13)	13 (81)	0 (0)	4	0 (0)	4 (100)	0 (0)
39	5	1 (20)	3 (60)	3 (60)	0	-	-	-
40	12	3 (25)	7 (58)	1 (8)	5	2 (40)	3 (60)	1 (20)
42	1	0 (0)	1 (100)	0 (0)	0	-	-	-
43	9	2 (22)	4 (44)	3 (33)	3	1 (33)	1 (33)	1 (33)
46	45	11 (24)	32 (71)	4 (9)	15	5 (33)	9 (60)	0 (0)
52	29	2 (7)	23 (79)	7 (24)	6	1 (17)	4 (66)	0 (0)
58	11	4 (36)	7 (64)	1 (9)	6	2 (33)	6 (100)	0 (0)
64	5	2 (40)	3 (60)	0 (0)	3	0 (0)	2 (66)	2 (66)
70	1	0 (0)	1 (100)	0 (0)	0	-	-	-

TABLE 12. Assessment of new bone formation and type of new bone formation by dental age for the humerus, femur and tibia.

Dental Age	Humerus				Femur				Tibia			
	Observed *N*	NBF *N* (%)	Woven *N* (%)	Lamellar *N* (%)	Observed *N*	NBF *N* (%)	Woven *N* (%)	Lamellar *N* (%)	Observed *N*	NBF *N* (%)	Woven *N* (%)	Lamellar *N* (%)
30	1	0 (0)	0 (0)	0 (0)	1	0 (0)	0 (0)	0 (0)	1	0 (0)	0 (0)	0 (0)
34	7	1 (14)	1 (100)	0 (0)	4	1 (25)	1 (100)	0 (0)	5	2 (40)	2 (100)	0 (0)
36	3	0 (0)	0 (0)	0 (0)	3	0 (0)	0 (0)	0 (0)	3	0 (0)	0 (0)	0 (0)
38	23	4 (17)	4 (100)	0 (0)	19	1 (5)	1 (100)	0 (0)	17	4 (24)	4 (100)	0 (0)
39	6	0 (0)	0 (0)	0 (0)	6	0 (0)	0 (0)	0 (0)	5	0 (0)	0 (0)	0 (0)
40	13	2 (15)	2 (100)	0 (0)	11	4 (36)	4 (100)	0 (0)	12	5 (42)	5 (100)	0 (0)
42	1	0 (0)	0 (0)	0 (0)	1	0 (0)	0 (0)	0 (0)	1	0 (0)	0 (0)	0 (0)
43	11	2 (18)	1 (50)	1 (50)	10	4 (40)	3 (75)	1 (25)	10	3 (30)	2 (67)	1 (33)
46	52	2 (4)	2 (100)	0 (0)	46	7 (15)	6 (86)	1 (14)	50	10 (20)	10 (100)	0 (0)
52	41	3 (7)	2 (67)	1 (33)	32	4 (13)	4 (100)	0 (0)	27	5 (19)	5 (100)	0 (0)
58	18	2 (11)	2 (100)	0 (0)	18	2 (11)	2 (100)	0 (0)	17	4 (24)	4 (100)	0 (0)
64	14	1 (7)	0 (0)	1 (100)	12	1 (8)	0 (0)	1 (100)	9	1 (11)	0 (0)	1 (100)
70	1	0 (0)	0 (0)	0 (0)	1	0 (0)	0 (0)	0 (0)	1	0 (0)	0 (0)	0 (0)

Discussion

The analysis of this large sample of fetal, perinatal and infant remains has revealed a complex pattern of pathological lesions, with high percentages of individuals from all populations, and of all dental ages, showing evidence of pathological changes. This suggests that many of the individuals experienced a reduced health status.

High prevalence rates of pathological changes, particularly within the cranial elements may be questioned as to whether these changes observed are true pathological indicators. This is particularly pertinent regarding analysis and identification of NBF within the individuals, as this type of lesion in particular has been the subject of much bioarchaeological debate. However, before expanding upon the nuances and evidence this data provides, it is important to consider that this is an assessment of non-survivors. Consequently, patterns of pathological changes observed within these individuals may not be representative of the 'normal' population and indeed may be inflated as a consequence of their frailty and ultimately, untimely death. These individuals died either prior to, during, or shortly after birth in the following days, weeks or months. Hence, the high rates of pathological lesions are not unexpected (Krenz-Niedbala & Lukasik 2017). While not all individuals in the sample would be expected to show skeletal manifestations of disease, as most conditions do not leave skeletal traces (Goodman & Armelagos 1989), many will have perished as a result of disease, infection or other environmental stresses transmitted to them during their most precarious time of life. Furthermore, it must not be overlooked that some of the pathological lesions identified may also be the consequence of birth trauma. In particular, NBF can be a consequence of intra-cranial haemorrhage, which is a common occurrence within modern clinical settings (O'Driscoll *et al.* 1981; Fenichel *et al.* 1984; Looney *et al.* 2007).

It is well documented that fetal, perinatal and infant life is fragile; such individuals are wholly dependent on others for their development and care (Lewis 2017a; 2017b; Ramji 2009; Baxter 2005). Fetal, perinatal and infant death is still widespread; there are 4.5 million infant (under the age of one) deaths per year, and these represent 75% of all children dying under 5 years of age (WHO *Global Health Observatory*). An additional 2.6 million stillbirths are estimated to occur every year (WHO *Sexual and Reproductive Health*). Of these stillbirths, half occur during labour and birth (WHO *Sexual and Reproductive Health*), and of the *'133 million babies born alive each year, 2.8 million die in the first week of life'* (WHO *Maternal*

and Perinatal Health). This emphasises how, even today, the process of birth and the hours and days after are some of the most precarious of our life course. Many of these fetal and infant deaths today are in low and middle-income countries (WHO *Sexual and Reproductive Health*), where access to medical care and treatment is often limited. Within archaeological populations, similar conditions and limited medical knowledge, treatment and care of pregnant women and their children may have been likely**.** Thus, exceptionally high infant mortality, combined with maternal co-morbidity would not be unexpected. It is considered that up to 50% of infants in pre-industrial societies would have perished before reaching adulthood (Chamberlain 1997), whilst maternal mortality rates are purported to be between 7 and 17 per 1000 live births in post-Medieval England (Wrigley & Schofield 1989). Furthermore, the presence of pathological lesions to a high percentage of the individuals assessed simply reflects the high pathogen load and stress exposure that many of these populations are likely to have been subjected to.

Complexities of identifying and diagnosing pathological lesions

The results of pathological assessment demonstrated that the bones of the cranial vault (frontal bone, parietal bone and occipital bone) and the tibiae and femora are most commonly affected by pathological changes. Although these bones - flat bones of the cranium and long bones - differ in their growth mechanism (intramembranous and endochondral ossification respectively) (Schultz 2001), the importance of these skeletal elements within the body may be why these elements typically show pathological changes. It is known that the body prioritises growth of particular bodily and skeletal structures, with the brain sitting at the top of this physiological hierarchy (Barker *et al.* 2012), consequently requiring the cranial bones to be adequately developed.

Bones of the cranial vault are initially formed within the first trimester, with growth in these elements continuing across the plate and at the sutures throughout the intrauterine period (Lewis 2017a). The cranium is a highly vascular structure, and the high number of fissures and foramina means infection and disease can spread rapidly (Chapman *et al.* 2013a; 2013b). As a consequence, this structure is where NBF would perhaps be most expected, both as a result of normal growth and pathological response to health insults. However, growth of the cranial vault elements happens concentrically, with normal NBF originating from the centre of the bone, with growth in dimension occurring at the sutural edges (Lewis 2017a).

Although distinguishing normal from pathological NBF is difficult, evidence of both woven and lamellar NBF, of varying severities has been identified throughout the individuals assessed. For the frontal bone, one individual was found to have only ectocranial lesions, 201 individuals had endocranial lesions, while seven individuals showed evidence of both. None of the individuals assessed showed evidence of a concentric formation to the NBF, with many of the lesions instead appearing to follow, or be bounded by, vascular structures on the endocranial surface. Many lesions were also transverse in organisation, isolated across the endocranial surface, and in 6% of cases were associated with lytic lesions also.

Woven bone is produced when osteoblastic cells produce osteoid rapidly – such as in fetal and infant growth, but also as a result of pathological response (Kini *et al.* 2012). Woven bone is disorganised in appearance and often appears as a fine, porous, grey layer on bones (Kini *et al.* 2012; Schultz 2001). In contrast, lamellar bone is characterised by its regular, linear appearance and is found as a result of healing or as part of the remodelling process in growth and development (Kini *et al.* 2012; Schultz 2001).

When part of the normal growth process of the cranium, it is expected that the NBF would be isolated to the sutural edges of the vault bones. Instead this woven and lamellar bone formation was found across the entirety of the endocranial surface of vault bones; there was no indication that NBF was specific to particular areas, suggesting it is unlikely to be a part of the normal NBF process, and instead a result of pathological bony changes. Additionally, many endocranial lesions also show evidence of meningeal/arterial grooves running though the NBF which has been considered to be indicative of enlarged or atypical vascular structures (Schultz 2001; Rumbaugh & Potts 1966). This may support the suggestion that, due to its highly vascular physiology, the cranium often reflects evidence of pathological changes both first, and perhaps most severely, as infections/systemic conditions would be easily transported to the cranium. It may be that lesions are identified particularly within the cranium as a result of the high bone turnover within these elements, though both normal and pathological NBF would be influenced by this. However, given that the severity of frontal lesions are typically grade two, it is suggested that the majority are not associated with normal NBF as a result of physiological growth. Furthermore, pathological lesions may be identified more readily within the cranial vault bones as the body may be attempting to maintain growth within these elements. Thus, it would follow that if these are the bones where growth is attempted to be maintained that they are equally likely to be the first bones

to show a pathological response. Additionally, if these lesions were associated with normal NBF it would be anticipated that there would be a peak in these lesions from 40 GWA onwards, as the postnatal growth spurt would be increasing NBF at this point. Findings suggest a similar prevalence rate of lesions amongst all dental ages, advocating that these lesions are pathological, and demonstrating that these lesions are not correlated with ages where known growth spurts occur (e.g. 40 GWA onwards).

In contrast, the long bones develop endochondrally, where a cartilaginous precursor is laid down before ossification commences (Lewis 2017a). This endochondral ossification means that new bone develops and grows from the internal aspect of the bone (from within the medullary cavity), and ossifies at the metaphyseal growth plates (Kini *et al.* 2012). The cortical bone is covered by an outer surface known as the periosteum, except for at the joints, or metaphyses in infant individuals (Kini *et al.* 2012). The periosteum is a fibrous connective tissue containing blood vessels and nerves, which helps to protect, nourish and aid in NBF (Kini *et al.* 2012; Schultz 2001). The periosteum is also involved in the process of appositional growth (Kini *et al.* 2012). It is this appositional growth which has caused much debate within bioarchaeology, as again the appearance of woven bone around the diaphyses of skeletal long bones has been argued to be both a pathological response, and part of the normal growth process. In fetal, perinatal and infant individuals the periosteum is both more active (in terms of normal growth) but is also more weakly attached to the cortical bone which it covers (Rana *et al.* 2009). This means that pathological changes to the periosteum can be identified earlier in infants but also tend to be more common and aggressive (Rana *et al.* 2009). The presence of NBF on the diaphyses of long bones can then represent both normal and pathological changes (Kwon *et al.* 2002). Distinguishing between the two has been contested at length, with no definitive method for macroscopic assessment of dry-bone yet established. Recent clinical literature has explored periosteal NBF, seemingly able to distinguish pathological bone on the basis of both the number of layers of NBF and its thickness (Rana *et al.* 2009; Kwon *et al.* 2002).

Kwon *et al.* (2002) found that NBF is often found in individuals aged 1-4 months and is suggestive of normal growth. However, NBF in neonates (younger than 1 month) and older infants (older than 4 months) was not identified and is suggested to be abnormal (Kwon *et al.* 2002). Periosteal NBF, if pathological, is often interpreted as being associated with metabolic disorders, such as vitamin C and D deficiency (Schultz 2001; Lewis 2007; Dawson 2017),

hypervitaminoses A and infections such as congenital syphilis (Kwon *et al.* 2002; Rana *et al.* 2009), soft tissue infections, and leprosy and tuberculosis (Schultz 2001) – all conditions known to cause pathological NBF within the skeleton. Kwon *et al.* (2002) found that 35% of the individuals assessed had NBF to at least one bone, with 55% of individuals between 2 and 3 months of age having the highest rate of NBF. However, it must be noted that the sample considered within Kwon and colleague's analysis (2002) is of individuals who suffered SIDS (Sudden Infant Death Syndrome). SIDS is defined as the sudden, and unexplained, death of an infant below 1 year of age (Moon & Fu 2012; Adams *et al.* 2015). Despite this apparent lack of cause of death, true health status is unknown. Besides, individuals who were premature, small for gestational age, or experienced intrauterine growth restriction are of increased risk of SIDS (NHS; *Sudden Infant Death Syndrome*). Therefore, though minimising the inclusion of 'unhealthy' individuals within their sample, Kwon and colleagues' investigation (2002) was not undertaken on individuals of known health status and remains an assessment of deceased individuals.

Archaeological and historical individuals from this investigation showed that between 33-55% of individuals aged 43-52 GWA showed postcranial pathological changes (Table 4.), with the long bones found to show pathological changes in between 5-24% of all individuals (Table 5.2). For individuals aged 43-52 GWA, 13% of those with femora show pathological lesions, 15% of those with tibiae, and 7% of those with tibiae (figures calculated using Table 9.). When only NBF is considered for the age groups 43-52 GWA (Table 12.), 4-40% of long bones show these changes. Consequently, the rates of pathological lesions identified within these individuals are lower than the published study identified (Kwon *et al.* 2002) and are consequently not suggestive of over-recording. Furthermore, this study revealed that NBF to the limb bones (humerus, femur, tibia) was found throughout the age groups (Table 12.), with those both below 1 month (44 GWA), and over 4 months (56 GWA), showing similar percentages of NBF as those aged between 1-4 months.

The fact that patterns of postcranial pathology observed within this study contradict the findings of Kwon *et al.* (2002) helps substantiate that the NBF identified on the archaeological individuals studied is not associated with normal growth. However, the pattern of bones most typically shown to have NBF are similar to those outlined by Kwon *et al.* (2002), with the tibiae most commonly affected, followed by the humerus and femur, with radius and ulna showing the least prevalence of NBF (Table 7.). However, given than both

metaphyseal changes and morphological changes, consistent with bowing, were also identified within the limb bones (Table 7.) suggests that pathological stimuli were impacting on these individuals. Thus, the prevalence of NBF within the skeletal remains is posited to show evidence of pathological changes.

The tibia is known to be particularly sensitive to health insults (Klaus 2014) and so the high prevalence rate of lesions to this skeletal element within non-survivors of archaeological samples is not unexpected. In total, 25 out of the 63 tibiae (39 %) to show NBF, had lesions located on the anterior. This was the location with the greatest prevalence of lesions, in comparison to those recorded as lateral, medial or circumferential. The anterior tibia is widely considered to be the most sensitive and indicative of pathological changes, particularly regarding NBF. This tendency is considered to be a result of its close proximity to the skin/surface and greater vascularity (Roberts & Manchester 2010). This study supports these assumptions, demonstrating that, of the postcranial bones, the tibia most commonly shows pathological lesions, both in general (Table 5.2), and by time period (Table 6.). Consequently, it may be that, once again, the pattern of pathology is reflecting the pattern of growth; if these bones are suspected of showing evidence of NBF as part of appositional growth, it would be likely that when affected by health insults, these bones would also be first to be affected and show evidence of pathological change. Thus, thickness of the NBF may be imperative in discerning between normal and pathological periosteal NBF, as suggested by Kwon *et al.* (2002).

To conclusively determine whether NBF identified on the 423 individuals assessed are part of normal or pathological growth processes, the NBF would have to be over 2mm in thickness (Kwon *et al.* 2002). As this study did not employ any destructive methods of analysis, nor have the ability to radiograph all 423 individuals, the thickness of the NBF has not been metrically assessed. However, by including assessment of severity within analysis, individuals who showed NBF of severity grade three are suggested to clearly demonstrate evidence of pathological NBF. However, such obvious determination of pathological from normal NBF is not as simple for many of the individuals. Clinical studies do not describe macroscopic features of pathological NBF, instead relying on radiological assessment (e.g. Kwon *et al.* 2002). Thus, diagnostic evidence of pathology currently requires radiographic analysis, whilst macroscopic assessment of archaeological remains still lacks precise diagnostic criteria.

Currently a major limitation in the assessment of pathological lesions is the ambiguity surrounding the body's ability to mount a response to pathological stimuli if indeed experiencing detrimental health stress. Addressing this concern is important for pathological assessment of all individuals not just non-adults, though is particularly pertinent for those individuals who are growing and developing so rapidly (e.g. fetal, perinatal and infant individuals). Weston (2012) has argued that NBF should not be used as an indicator of health stress due to NBF being inhibited under subjection to stress. Ultimately then, NBF should not be identifiable as a physiological response to stress, and instead absence of NBF may be more remarkable in revealing interruption and cessation of growth. However, as Selye has highlighted (1973)**,** there are multiple phases to the stress response. The general-adaptation-syndrome model is defined as the '*non-specific response of the body to any demand upon it*' (Selye 1973, 692). There are three phases to this model: the alarm, resistance and collapse stages (Selye 1973). Within this conceptual framework, although the initial phases of bodily response may be to halt growth as part of the alarm mechanism, the resistance and collapse stages may result in physiological skeletal changes as the body attempts to accommodate, adapt to, and overcome health stresses. Thus, bony changes may be indicative of the body endeavouring to maintain homeostasis (Selye 1973). Klaus (2014) supports this notion suggesting that stress and growth has a much more intricate and bounded relationship than simply an on or off mechanism. Consequently, NBF, both woven and lamellar may be indicative of a healing response. With bones of the cranial vault typically found to have the highest prevalence rates of NBF it is then considered that this is as a result of the body prioritising maintenance and healing within these elements.

Etiological Interpretations

Regarding the etiology of the lesions identified some are consistent with metabolic stress (Lewis 2007; Mensforth *et al.* 1978). Individuals expressing NBF, metaphyseal expansion, and torsion/bowing of the limbs might be more obviously diagnosed with suspected cases of vitamin D and C deficiency. Even today vitamin deficiencies, particularly vitamin D, are common in pregnant women and it has been advised since 2007 that pregnant women in the U.K should take additional supplements of vitamin D (Mahon *et al.* 2010; Scientific Advisory Committee on Nutrition 2007). However, diagnosis is complex and such changes are likely as a result of the mother being unable to provide adequate nutrition to the

individual *in utero* and beyond (Mahon *et al.* 2010). As a result, many other conditions and diseases, which may take a varying form in the adult skeleton, may be expressed as a vitamin deficiency in the developing fetus and perinate. Parasitic infections would cause pathological lesions consistent with vitamin deficiency by restricting the flow of nutrients across the placenta (Barker *et al.* 2012), although various other diseases would also inhibit this ability by reducing the mother's health status and causing her own body to metabolically respond by limiting nutrients to the fetus.

Vitamin C and D deficiencies can affect growth and development of the skeleton; lack of vitamin C results in defective osteoid formation (development of new bone cells) (Brickley & Ives 2006) while vitamin D deficiency affects the calcification of bone (Lewis 2007; Kini *et al.* 2012). Enlarged or widened and porous metaphyses, identified in 29% of the individuals with pathological changes, are also an indication of vitamin D deficiency, in which cartilage at the growth plates does not mineralize properly (Roberts & Manchester 2010). Although some argue that enlarged or 'splayed/flared' metaphyses should only be visible when limbs are weight-bearing (e.g. Holick 2005), a recent clinical investigation has demonstrated that fetal femoral metaphyseal cross-sections are enlarged when their mothers are either vitamin D insufficient or deficient (Mahon *et al.* 2010). Furthermore, this study also found that such changes in metaphyseal dimensions were observable from as early as 19-weeks gestation (Mahon *et al.* 2010). Similarly, Innes and colleagues (2002) found that bowing in association with congenital rickets can be observed pre- and perinatally also. It is posited that prenatal space constraint and restriction *in utero* can alter morphology (e.g. Bonneau *et al.* 2011).

Metaphyseal changes can also indicate the presence of vitamin C deficiency as widening at the metaphyses could suggest chronic bleeding at the joints (Aufderheide and Rodriguez-Martín 2008). Lack of vitamin C can result in blood vessels becoming fragile and easily ruptured (Brickley and Ives 2006; Ortner and Ericksen 1997; Besbes *et al.* 2010). This can lead to haemorrhaging, including in the vessels supplying the metaphyses, but particularly within the cranium (Roberts and Manchester 2010). With many of the individuals assessed exhibiting evidence of NBF to the endocranium, it may indicate that cranial haemorrhaging (Brickley and Ives 2006; Schultz 2001) and chronic bleeding (Ortner *et al.* 1999) was experienced as a result of nutritional deficiencies. Lesions associated with cranial haemorrhaging are typically found around meningeal grooves, on the frontal bones and at the

cruciate eminence of the occipital bone, where they present a web-like appearance (Lewis 2004; 2007). Furthermore, many of the lesions identified are comparable to those considered by Lewis (2004) and Schultz (2001) as evidence of pathological lesions. However, the exact etiology of NBF in the cranium is debated (Lewis 2007) and other possible causes include meningitis, anaemia and venous drainage disorders (Lewis 2004; 2007).

Vitamin A deficiency is a further factor to be considered as we are unable to synthesize this nutrient and are required to get our intake from dietary sources (Fujita *et al.* 2017). Importantly, we require this vitamin for a variety of functions, including both our growth and immunity (Fujita *et al.* 2017). In particular, maternal breast milk is known to have high concentrations of vitamin A as a result of the post-partum infant requiring increased levels of this nutrient to be able to sustain rapid growth (Fujita *et al.* 2017). Vitamin A deficiency is one which is still estimated to claim one million infant lives a year **(**Fujita *et al.* 2017**)** and thus, maternal deficiency of this nutrient within any of the archaeological populations would likely have contributed to the compromised fetal, perinatal and infant health and wellbeing identified. Considering the fact that nutritional deficiency is often experienced in conjunction with other deficiencies, vitamin A deficiency is an etiology which needs further consideration within the bioarchaeological literature.

The presence of NBF is non-specific and multiple causes could be attested (Nade 1983). Klaus (2014) and Weston (2008) have highlighted the multiple mechanisms under which new bone can form, particularly that which involves an inflammatory response. Weston (2008) considers it imperative to note that periosteal NBF is an inflammatory response (a vascular response) by the body to neutralise infection or repair/heal damaged bone. Infections are pathogenic organisms, which typically result in an inflammatory response, but as Weston (2008) states '*…not all such responses are caused by infection'*. Infections are considered to be relatively typical during early development (Eisenberg *et al.* 2017; Degani 2006), and many infections are possible: these include meningitis, rubella, measles, smallpox, puerperal fever and diarrhoeal diseases (Anderson & Gonik 2011; Lewis 2017a). Although the specific pathological lesions for such conditions are unknown, some of the non-specific lesions identified may be attributable to them. In particular, individuals with extensive NBF to the long bones which produces a distinct thickening (See Table 3.2; New Bone Formation Grade 3) may be indicative of osteomyelitis. Osteomyelitis is a result of inflammation elevating the periosteum away from the cortical bone surface and creating a new layer of bone, often

resulting in a thick, bony sleeve of bone around the original cortical surface (Rana *et al.* 2009). Osteomyelitis can be caused by fungal infections, tuberculosis, typhoid fever, congenital syphilis and small pox (Lewis 2017a). However, attributing a specific cause to these lesions is challenging (Schmit & Glorion 2004). The tibia has been identified as the bone which most commonly shows evidence of osteomylitic infections (Rasool 2001). Given that the tibia has similarly been identified within this study to be the most susceptible to pathological changes, and with five individuals having NBF of severity grade three identified on the tibia, osteomyelitis is a likely diagnosis, despite the true etiology remaining unknown. Smallpox is known to increase fetal mortality (Reid 1990), and historical studies of 19th and 20th century populations have shown that as smallpox declined and was eradicated, fetal and maternal mortality also declined (Reid 1990). Smallpox can also be transmitted during pregnancy from mother to the *in utero* child (Woods 2008). Therefore, particularly within the post-Medieval collections, smallpox maybe a further contributing aetiology to consider in relation to the high levels of NBF observed in the individuals from this period.

Endocranial lesions may also result from infectious disease, including tuberculosis, fungal infections (Vattoth *et al.* 2013) and treponemal diseases (Schultz 2001). However, normal vaginal birth has been found to be associated with intracranial haemorrhage (ICH) with no correlation to duration of labour or traumatic/assisted birth (Looney *et al.* 2007). Other studies however, have identified that traumatic birth, breech delivery, and forceps delivery all increase the risks of ICH (O'Driscoll *et al.* 1981; Fenichel *et al.* 1984). ICH has also been considered to be strongly associated with anoxemia (deficiency in oxygen with the arterial blood supply), with symptoms of ICH including appetite loss, vomiting and convulsions (Yongen 1980). Yongen (1980) also found that ICH was strongly correlated to mortality, with 33% of those expressing lesions dying within the first few day/weeks of life. Consequently, evidence of NBF within the cranium of many individuals assessed within this study potentially reflect birth trauma and the morbidity and mortality consequences of such.

The Biocultural Perspective: Implications of fetal, perinatal and infant pathology

The maternal-infant nexus is a relationship that many, multidisciplinary studies, are now considering in more detail due to its ability to influence the morbidity and mortality outcomes during earlier and later life (e.g. Gowland 2015). From a bioarchaeological perspective, this nexus provides insights into maternal health, as well as overall population health (Baxter 2005; Redfern 2003; Goodman & Armelagos 1989). Fetuses, perinates and infants should not

be considered to be autonomous (Gluckman 1997), but instead their fortunes are intrinsically entangled with those of their mothers (Gowland 2015; Redfern 2003). The mother's own health and/or disease status plays a crucial role in determining the health of her child while *in utero* and beyond (Gowland 2015; Wilcox 2010).

The fetus has often been referred to as a parasite (Rivara & Miller 2017), leaching nutritional resources from the mother for optimal growth and development (Gowland 2015). However, the mother has to have sufficient availability of nutrients to supply the placenta, and thus the offspring, and is reliant on the placental transfer of these resources to the fetus (Barker *et al.* 2012; Gluckman 1997). When limited nutritional resources are available, the fetus prioritises growth to particular skeletal structures and bodily systems (Barker *et al.* 2012). As a result, there is often a trade-off between bodily and skeletal structures (Barker *et al.* 2012). Consequently, nutritional status of the mother both at time of conception as well as both prior to and throughout pregnancy can affect the health and wellbeing of the offspring (Barker *et al.* 2012). This is because the child relies on the stored maternal resources of nutrients, including protein and fat (Barker *et al.* 2012). Malnutrition in a pregnant mother can lead to IUGR (intrauterine growth restriction), low birth weight, preterm birth and birth defects – including neural tube disorders (Wu *et al.* 2012), and has been shown to have long-term correlations with rates of obesity, insulin resistance and coronary heart disease (Barker *et al.* 2012). In addition, maternal endocrine regulation – the supply, release and regulation of hormones to the fetus – can also severely affect health and development (Gluckman 1997). Therefore, maternal health and nutrition is central in regulating the health stresses which the offspring faces.

Postnatal health and wellbeing is also dependent on a variety of factors, generally regulated by the mother/caregiver, such as feeding practices and the nutritional status of the breastfeeding mother (Gowland 2015; Ramji 2009). Breastmilk, particularly that of colostrum (the initial thick breastmilk available directly after birth), is important for both the nutritional and immunological wellbeing of the infant (Eisenberg *et al.* 2017; Lewis 2017b). Inability of the ill/nutritionally deficient mother to provide adequate milk would further exacerbate any deficiency experienced by the child and potentially increase their susceptibility to a range of diseases and illnesses post-partum. Birth is not only obstetrically risky for mother and child alike (Reid 1990), but signals the transition for the child from a protected environment into one full of pathogens, and bacteria, all of which the individual needs to build an immune

response to (Lewis 2017a). It is the mother who is required to provide protection, both nutritionally and immunologically, for her child postnatally (Eisenberg *et al.* 2017). However, it cannot be known whether the mothers of the individuals considered in this assessment survived childbirth, or were willing and/or able to breastfeed. Lack of, or limited, maternal nutritional and immunological buffering makes the initial post-partum period even more perilous, and as a result, one in which many infants, both historically and today, are unable to survive.

Both pre- and postnatal individuals were found to show evidence of pathological lesions to multiple skeletal elements, with both similarities in prevalence rates, types of pathological lesions and severity. As such, when all 423 individuals are considered simultaneously, no clear evidence for a particularly stressful pre- or postnatal environment emerges. However, given the archaeological and historical samples assessed it may be insinuated that maternal health was likely universally reduced. As a consequence, pathological lesions identified throughout the 423 fetal, perinatal and infant individuals may have been regulated by the environmental conditions to which these individuals were exposed both pre- and postnatally.

Conclusion:

- Pathology can be identified on fetal, perinatal and infant individuals but it is challenging and there are some ongoing issues/debates, particularly concerning differentiation between normal and pathological NBF, that need to be rectified before standards of recording can be fully implemented.
- This assessment has outlined potential factors (location, type of lesion, severity) for consideration when assessing pathology and has provided a detailed methodology for recording fetal, perinatal and infant pathological lesions.
- This study has shown that NBF is the most commonly found pathological change. These lesions are inherently non-specific resulting in a wide consideration of potential pathogenic and etiological causes.
- This study has demonstrated that NBF can be distinguished from normal new bone formation when severity (grade 1, 2, or 3) and type of lesion (woven or lamellar) is considered. Furthermore, to substantiate this finding, individuals of all ages showed evidence of NBF lesions, countering current medical findings which suggest NBF is typically found in individuals aged between 44 and 56 GWA, as a

result of the postnatal growth spurt. A varying pattern of NBF within these individuals has been identified and is thus suggestive of a pathological stimulus.

- Assessment has suggested that bones of the cranium are most typically affected by adverse conditions. This may be because it is both a highly vascular structure and a vital structure within the body. As a result, if health insults are experienced it may be that we see lesions in the cranium as the body is trying to manage and preserve the health and wellbeing of these structures.
- This study corroborates previous findings identifying the tibia as the most sensitive postcranial element to health stress. NBF, particularly to the anterior of the tibia, was found to be the most prevalent lesions within all long bones. The tibia was still found to be the most commonly affected long bone when lesions were considered by archaeological/historical time period and dental age.
- Pathological lesions, of all types and severity, do not appear to be isolated to a particular age group, with both cranial and postcranial changes found in all age categories. This counters arguments suggesting pathological lesions, in particular those of NBF, are found to be most prevalent in individuals who may be experiencing growth spurts.

It is imperative that investigation into skeletal pathology of fetal, perinatal and infantile individuals continues to develop and attempts to address the challenges raised within this paper. This study provides a large sample of the very youngest individuals from a broad temporal period. It is hoped the methodology for assessing pathological lesions outlined and the findings will provide a useful comparison for future studies. Most importantly, the notion that osteological assessment of such young individuals is futile must be overcome. A wealth of information, regarding health and wellbeing can be discerned from analysis of these individuals, and provides a unique insight into early life experiences, the infant/mother nexus, and ultimately, the physiological response to health stress.

References

Adams, S. M., Ward, C. E. and Garcia, K. L. 2015; Sudden Infant Death Syndrome. *American Family Physician,* Vol. 91 (11): 778-783.

AlQahtani, S. J. Hector, M. P. and Liversidge, H. M. 2010; The London Atlas of Human Tooth Development and Eruption. *American Journal of Physical Anthropology,* Vol. 142: 481-490.

AlQahtani, S. J. Hector, M. P. and Liversidge, H. M. 2014; Accuracy of Dental Age Estimation Charts: Schour and Massler, Ubelaker, and the London Atlas. *American Journal of Physical Anthropology,* Vol. 154: 70-78.

Anderson, B. L. and Gonik, B. 2011; Perinatal Infections. In R. J. Martin, A. A. Fanaroff and M. C. Walsh (Eds.) *Fanaroff and Martin's neonatal-perinatal medicine: diseases of the fetus and infant.* Missouri: Elsevier Mosby: 399-422.

Aufderheide, A. C. and Rodriguez-Martin, C. 2008; *The Cambridge Encyclopedia of Human Palaeopathology.* Cambridge: Cambridge University Press.

Bang, G. 1989; Age changes in teeth; developmental and regressive. *Age Markers in the Human Skeleton,* Vol. 1: 211-235.

Barker, D. J. P., Lampl, M., Roseboom, T. and Winder, N. 2012; Resource allocation in utero and health in later life. *Placenta,* Vol. 33: 30-34.

Baxter, J. E. 2005; The Archaeology of Childhood: Children, Gender, and Material Culture. California: AltaMira Press.

Beaumont, J., Montgomery, J, Buckberry, J and Jay, M. 2015; Infant Mortality and Isotopic Complexity: New Approaches to Stress, Maternal Health and Wellbeing. *American Journal of Physical Anthropology,* Vol. 157: 441-457.

Besbes, L. G., Hadded, S., Meriem, C. B., Golli, M., Najjar, M. F. and Guediche, M. N. 2010; Infantile Scurvy: Two Case Reports. *International Journal of Pediatrics,* Article ID 717518: 1-4.

Bonneau, N., Simonis, C., Seringe, R. and Tardieu, C. 2011; Study of Femoral Torsion During Prenatal Growth: Interpretations Associated with the Effects of Intrauterine Pressure. *American Journal of Physical Anthropology,* Vol. 145: 438-445.

Brickley, M. and Ives, R. 2006; Skeletal Manifestations of Infantile Scurvy. *American Journal of Physical Anthropology¸* Vol. 29: 163-172.

Brickley, M. Miles, A. and Stainer, H. 1999; *The Cross Bones Burial Ground, Redcross Way, Southwark, London: Archaeological Excavations (1991-1998) for the London Underground Limited Jubilee Line Extension Project* (MOLAS Monograph 3). London: Museum of London Archaeology Service.

Bush, H. and Zvelebil, M. 1991; Pathology and health in past societies: an introduction. In H. Bush and M. Zvelebil (Eds.) *Health in Past Societies: Biocultural interpretations of human skeletal remains in archaeological contexts* (BAR International Series 567). Oxford: BAR Publishing: 3-9.

Cattaneo, C. 1991; Direct genetic and immunological information in the reconstruction of health and biocultural conditions of past populations: a new prospect for archaeology. In H. Bush and M. Zvelebil (Eds.) *Health in Past Societies: Biocultural interpretations of human skeletal remains in archaeological contexts* (BAR International Series 567). Oxford: BAR Publishing: 39-52.

Chamberlain, A. 1997; Missing stages of life – towards the perception of children in archaeology. In J. Moore and E. Scott (Eds.) *Invisible People and Processes: writing gender and childhood into European archaeology.* London: Leicester University Press: 248-250.

Chapman, P. R., Bag, A. K., Tubbs, R. S., and Gohlke, P. 2013a; Practical Anatomy of the Central Skull Base Region. *Seminars in Ultrasound, CT and MRI*: 381-392.

Chapman, P. R., Gaddamanugu, S., Bag, A. K., Roth, N. T. and Vattoth, S. 2013b; Vascular Lesions of the Central Skull Base Region. *Seminars in Ultrasound, CT and MRI*: 459-475.

Collis, J. R. 1968; Excavations at Owslebury, Hampshire: An Interim Report. *Antiquaries Journal,* Vol. 48 (1): 18-31.

Collis, J. R. 1970; Excavations at Owslebury, Hampshire: A Second Interim Report. *Antiquaries Journal,* Vol. 50: 246-261.

Collis, J. R. 1977; Owslebury (Hants) and the problem of burials on rural settlements. In R. Reece (Ed.) *Burial in the Roman World,* CBA Research Report, No. 22. London: The Council for British Archaeology: 26-34.

Dawson, H. 2017; Precious Things: Examining the Status and Care of Children in Late Medieval England through the Analysis of Cultural and Biological Markers. In L. Powell, W. Southwell-Wright, and R. Gowland (Eds.) *Care in the Past: Archaeological and Interdisciplinary Perspectives.* Oxford: Oxbow Books: 53-69.

Degani, S. 2006; Sonographic findings in fetal viral infections: a systemic review. *Obstetrical and Gynaecological Survey,* Vol. 61 (5): 329-336.

Eisenberg, D. T. A., Borja, J. B., Hayes, M. G. and Kuzawa, C. W. 2017; Early life infection, but not breatfeeding, predicts adult blood telomere lengths in the Philippines. *American Journal of Human Biology,* Vol. 29 (Early View): 1-11.

Faerman, M. 1999; Ancient DNA diagnosis of bone pathology in infancy and early childhood. *American Journal of Physical Anthropology,* Suppl. 28:125.

Fenichel, G. M., Webster, D. L., Wong, W. K. T. 1984; Intracranial Hemorrhage in the Term Newborn. *Archives of Neurology,* Vol. 41 (1): 30-34.

Friendship-Taylor, R. M. and Friendship-Taylor, D. E. 2012; *Iron Age and Roman Piddington: 10th Interim Report and Phase Descriptions of the Late Iron Age Settlements,*

Military Phase, Roman Villa Complex and Early Saxon Phases at Piddington, Northants. The Upper Nene Archaeological Society 2013.

Fujita, M., Lo, Y. J. and Brindle, E. 2017; Nutritional, inflammatory, and ecological correlates of maternal retinol allocation to breast milk in agro-pastoral Ariaal communities of northern Kenya. *American Journal of Human Biology,* Vol. 29 (Early View): 1-14.

Garn, S. M. Lewis, A. B. and Polacheck, D. L. 1960; Interrelations in dental development. I. Interrelationships within the dentition. *Journal of Dental Research,* Vol. 39: 1049-1055.

Gluckman, P. D. 1997; Endocrine and nutritional regulation of prenatal growth. *Acta Paediatrica* Supplementary Series, Vol. 423: 153-157.

Goodman, A. H. and Armelagos, G. J. 1989; Infant and Childhood Morbidity and Mortality Risks in Archaeological Populations. *World Archaeology,* Vol. 21 (2): 225-243.

Gowland, R. L. 2004; The social identity of health in late Roman Britain. In B. Croxford, H. Eckardt, J. Meade, and J. Weekes (Eds.) *TRAC 2003: Proceedings of the Thirteenth Annual Theoretical Roman Archaeology Conference.* Oxford: Oxbow Books: 135-146.

Gowland, R. L. 2015; Entangled Lives: Implications of the Developmental Origins of Health and Disease Hypothesis for Bioarchaeology and the Life Course. *American Journal of Physical Anthropology,* Vol. 158, No. 4: 530-540.

Harding, V. 2002; *The dead and the living in Paris and London: 1500-1670.* Cambridge: Cambridge University Press.

Hillson, S. W. 2005; *Teeth* (Second Edition). Cambridge: Cambridge University Press.

Holick, M. F. 2005; The Vitamin D Epidemic and its Health Consequences. *The Journal of Nutrition,* Supplement: 2739S-2748S.

Hunt, D. [Personal Communication: October 2015]; *Brief background to the fetal collections housed at the NMNH* (Unpublished).

Innes, A. M., Seshia, M. M., Prasad, C., Al Saif, S., Friesen, F. R., Chudley, A. E., Reed, M., Dilling, L. A., Haworth, J. C. and Greenberg, C. R. 2002; Congenital rickets caused by maternal vitamin D deficiency. *Paediatric Child Health*, Vol. 7 (7): 455-458.

Kausmally, T. 2008 *St Bride's Lower churchyard cemetery summary* [Online] [Accessed: November 2016] Available from: https://www.museumoflondon.org.uk/collections/other-collection-databases-and-libraries/centre-human-bioarchaeology/osteological-database/post-medieval-cemeteries/st-brides-lower-post-medieval

Kini, U. and Nandeesh, B. N. 2012; Physiology of Bone Formation, Remodeling, and Metabolism. In I. Fogelman, G. Gnanasegaran, and H. van der Wall (Eds.) *Radionuclide and Hybrid Bone Imaging.* Verlag Berlin Heidelberg: Springer.

Klaus, H. D. 2014; Frontiers in the Bioarchaeology of Stress and Disease: Cross-Disciplinary Perspectives From Pathophysiology, Human Biology, and Epidemiology. *American Journal of Physical Anthropology,* Vol. 155: 294-308.

Krenz-Niedbala, M. and Lukasik, S. 2017; Skeletal Evidence for Otitis Media in Mediaeval and Post-Mediaeval Children from Poland, Central Europe. *International Journal of Osteoarchaeology,* Vol. 27: 375-386.

Kwon, D. S., Spevak, M. R., Fletcher, K. and Kleinman, P. K. 2002; Physiologic Subperiosteal New Bone Formation: Prevalence, Distribution, and Thickness in Neonates and Infants. *American Journal of Radiology,* Vol. 179: 985-988.

Lewis, M. E. 2004; Endocranial Lesions in Non-Adult Skeletons: Understanding their Aetiology. *International Journal of Osteoarchaeology,* Vol. 14: 82-97.

Lewis, M. E. 2007; *The Bioarchaeology of Children: Perspectives from Biological and Forensic Anthropology.* Cambridge: Cambridge University Press.

Lewis, M. E. 2017a; *Paleopathology of Children: Identification of Pathological Conditions in the Human Skeletal Remains of Non-Adults.* London: Academic Press.

Lewis, M. E. 2017b; Childcare in the Past: The Contribution of Palaeopathology. In L. Powell, W. Southwell-Wright, and R. Gowland (Eds.) *Care in the Past: Archaeological and Interdisciplinary Perspectives.* Oxford: Oxbow Books: 23-37.

Lewis, M. E. 2018; Fetal Paleopathology: An Impossible Discipline? In S. Han, T. K. Betsinger, and A. B. Scott (Eds.) *The Fetus: Biology, Culture, and Society.* New York: Berghahn Books: 112-131.

Lock, M. M. 1993; Cultivating the Body: Anthropology and Epistemologies of Bodily Practice and Knowledge. *Annual Review of Anthropology,* Vol. 22: 133-155.

Looney, C. B., Smith, J. K., Merck, L. H., Wolfe, H. M., Chescheir, N. C., Hamer, R. M. and Gilmore, J. H. 2007; Intracranial hemorrhage in asymptomatic neonates: prevalence on MR images and relationship to obstetric and neonatal risk factors. *Radiology,* Vol. 242 (2): 535-541.

Mahon, P., Harvey, N., Crozier, S., Inskip, H., Robinson, S., Arden, N., Swaminathan, R., Cooper, C., The SWS Study Group, and Godfrey, K. 2010; Low maternal vitamin D status and fetal bone development: cohort study. *Journal of Bone and Mineral Research,* Vol. 25 (1): 14-19.

Mensforth, R., Lovejoy, C. O., Lallo, J. W. and Armelagos, G. J. 1978; The Role of Constitutional Factors, Diet, and Infectious Disease in the Etiology of Porotic Hyperostosis and Periosteal Reactions in Prehistoric Infants and Children. *Medical Anthropology,* Vol. 2 (1): 1-59.

Mikulski, R. 2007 *Cross Bones burial ground summary* [Online] [Accessed: November 2016] Available from: https://www.museumoflondon.org.uk/collections/other-collection-databases-and-libraries/centre-human-bioarchaeology/osteological-database/post-medieval-cemeteries/cross-bones-post-medieval

Miles, D. 1986; *Archaeology at Barton Court Farm, Abingdon, Oxfordshire*. Oxford Archaeological Unit Report 3, CBA Research Report 50. Oxford: Oxford Archaeological Unit.

Moon, R. Y. and Fu, L. 2012; Sudden Infant Death Syndrome: An Update. *Pediatrics in Review,* Vol. 33: 314-320.

Moorrees, C. F. A. Fanning, E. A. and Hunt, E. E. 1963a; Formation and Resorption of Three Deciduous Teeth in Children. *American Journal of Physical Anthropology,* Vol. 21:205-213.

Moorrees, C. F. A. Fanning, E. A. and Hunt, E. E. 1963b; Age Variation of Formation Stages for Ten Permanent Teeth. *Journal of Dental Research,* Vol. 42: 1490-1502.

Museum of London 2009 *Chelsea Old Church (Post-Medieval) cemetery summary* [Online] [Accessed: November 2016] Available from: https://www.museumoflondon.org.uk/collections/other-collection-databases-and-libraries/centre-human-bioarchaeology/osteological-database/post-medieval-cemeteries/chelsea-old-church-post-medieval

Nade, S. 1983; Acute Haematogenous Osteomyelitis in Infancy and Childhood. *Journal of Bone and Joint Surgery,* Vol. 65B (2): 109-119.

NHS *Sudden Infant Death Syndrome (SIDS)* [Online] [Accessed March 2018] Available from: https://www.nhs.uk/conditions/sudden-infant-death-syndrome-sids/

O'Driscoll, K., Meagher, D., Macdonald, D., and Geoghegan, F. 1981; Traumatic intracranial haemorrhage in firstborn infants and delivery with obstetric forceps. *BJOG: International Journal of Obstetrics and Gynaecology,* Vol. 88 (6): 577-581.

Ortner, D. J. 2003; *Identification of Pathological Conditions in Human Skeleton Remains*. San Diego: Elsevier.

Ortner, D. J. 2008; Differential diagnosis of skeletal lesions in infectious disease. In R. Pinhasi and S. Mays (Eds.) *Advances in Human Palaeopathology.* Chichester: Wiley and Sons: 191-214.

Ortner, D. J. Kimmerle, E. H. & Diez, M. 1999; Probable Evidence of Scurvy in Subadults from Archaeological Sites in Peru. *American Journal of Physical Anthropology,* Vol. 108: 321-331.

Ortner, D. J. and Ericksen, M. F. 1997; Bone Changes in the Human Skull Probably Resulting from Scurvy in Infancy and Childhood. *International Journal of Osteoarchaeology,* Vol. 7: 212-220.

Ramji, S. 2009; Impact of infant and young child feeding and caring practices on nutritional status and health. *Indian Journal of Medical Research,* Vol. 130: 624-626.

Rana, R. S., Wu, J. S. and Eisenberg, R. L. 2009; Periosteal Reaction. *American Journal of Radiology,* Vol. 193: 259-272.

Rasool, M. 2001; Primary subacute haematogenous osteomyelitis in children. *The Journal of Bone and Joint Surgery,* Vol. 83 (1): 93-98.

Redfern, R. 2003; Sex and the City: A biocultural investigation into female health in Roman Britain. In G. Carr, E. Swift and J Weekes (Eds.) *TRAC 2002 Proceedings of the Twelfth Annual Theoretical Roman Archaeology Conference.* Oxford: Oxbow Books.

Reid, A. 1990; Death before Birth: Fetal Health and Mortality in Historical Perspective (Review). *Journal of Interdisciplinary History,* Vol. 41 (4): 621-623.

Reitsema, L.J. and McIlvaine, B. K. 2014; Reconciling “stress” and “health” in physical anthropology: What can bioarchaeologists learn from the other subdisciplines? *American Journal of Physical Anthropology,* Vol. 155: 181-185.

Rivera, A. C. and Miller, E. M. 2017; Pregnancy and immune stimulation: re-imagining the fetus as a parasite to understand age-related immune system changes in US women. *American Journal of Human Biology* (Early View): 1-6.

Rivera, F. and Lahr, M. M. 2017; New evidence suggesting a dissociated etiology for *cribra orbitalia* and porotic hyperostosis. *American Journal of Physical Anthropology* (Early View): 1-21.

Roberts, C. 2017; Navigating Approaches to Impairment, "Disability" and Care in the Past: The Need for Reflection. In L. Powell, W. Southwell-Wright, and R. Gowland (Eds.) *Care in the Past: Archaeological and Interdisciplinary Perspectives.* Oxford: Oxbow Books: xi-xviii.

Roberts, C. and Manchester, K. 2010; *The Archaeology of Disease*. Stroud: The History Press.

Ruff, C. B. Garofalo, E. and Holmes, M. A. 2013: Interpreting Skeletal Growth in the Past From a Functional and Physiological Perspective. *American Journal of Physical Anthropology,* Vol 150: 29-37.

Rumbaugh, C. L. and Potts, D. G. 1966; Skull changes associated with intracranial arteriovenous malformations. *The American Journal of Roentgenology, Radium Therapy and Nuclear Medicine,* Vol. 98 (3): 525-534.

Schmit, P. and Glorion, C. 2004; Osteomyelitis in infants and children. *European Radiology,* Vol. 14: 44-54.

Schultz, M. 2001; Paleohistopathology of Bone: A New Approach to the Study of Ancient Diseases. *Yearbook of Physical Anthropology,* Vol. 44: 106-147.

Scientific Advisory Committee on Nutrition 2007. *Update on vitamin D. Position statement by the Scientific Advisory Committee on Nutrition.* The Stationery Office; London: 2007.

Selye, H. 1973; The evolution of the stress concept. *American Scientist,* Vol. 61: 692-699.

Šešelj, M. 2013; Relationship Between Dental Development and Skeletal Growth in Modern Humans and Its implications for Interpreting Ontogeny in Fossil Hominins. *American Journal of Physical Anthropology*, Vol. 150: 38-47.

Temple, D. H. and Goodman, A. H. 2014; Bioarchaeology has a "health" problem: Conceptualizing "stress" and "health" in bioarchaeological research. *American Journal of Physical Anthropology*, Vol. 155: 186-191.

Vattoth, S., DeLappe, R. S., and Chapman, P. R. 2013; Endocranial Lesions. *Seminars in Ultrasound, CT and MRI*, Vol. 34: 393-411.

Waldron, T. 2009; *Palaeopathology*. Cambridge: Cambridge University Press.

Weston, D. A. 2008; Investigating the Specificity of Periosteal Reactions in Pathology Museum Specimens. *American Journal of Physical Anthropology*, Vol. 137: 48-59.

Weston, D. A. 2012; Nonspecific infection in palaeopathology: Interpreting periosteal reactions. In A. L. Grauer (Ed.). *A Companion to Paleopathology*. Chichester: Wiley-Blackwell: 492-512.

WHO (World Health Organisation) *Global Health Observatory* [Online] [Accessed July 2017] Available from: http://www.who.int/gho/child_health/mortality/neonatal_infant_text/en/

WHO (World Health Organisation) *Maternal and Perinatal Health* [Online] [Accessed July 2017] Available from: http://www.who.int/maternal_child_adolescent/topics/maternal/maternal_perinatal/en/

WHO (World Health Organisation) *Sexual and Reproductive Health* [Online] [Accessed July 2017] Available from: http://www.who.int/reproductivehealth/topics/maternal_perinatal/stillbirth/en/

Wilcox, A. J. 2010; *Fertility and Pregnancy; An Epidemiologic Perspective*. Oxford: Oxford University Press.

Woods, R. 2008; Late-Fetal Mortality: Historical Perspectives on Continuing Problems of Estimation and Interpretation. *Population*, Vol. 63 (4): 591-614.

Wrigley, E. A. and Schofield, R. S. 1989; The Population History of England 1541-1871. Cambridge: Cambridge University Press.

Wu, G. Imhoff-Kunsch, B. and Webb Girard, A. 2012; Biological Mechanisms for Nutritional Regulation of Maternal Health and Fetal Development. *Paediatric and Perinatal Epidemiology,* Vol 26, Suppl.1: 4-26.

Yongen, G. 1980; Neonatal Intracranial Hemorrhage. *Tianjin Medical Journal*, Vol. 6.

Chapter 10: Discussion

This chapter integrates the results of the four previous manuscripts, considering evidence of growth and health disruption within and between the archaeological and historical samples of fetal, perinatal and infant individuals. This chapter discusses the implications of these results, contextualising findings with regards to the environmental, social and cultural conditions to which individuals were exposed. This discussion aims to address the research questions stated within the introduction of this thesis and to develop a holistic and nuanced interpretation of fetal, perinatal and infant growth and health disruption within the different time periods analysed.

This discussion is divided into three main sections, the first seeks to examine evidence of growth disruption, the second evidence of health disruption, and the third section considers the link between the two, contextualising findings within the archaeological and historical frameworks of the samples assessed. Within these main sub-sections, discussion of dental and skeletal age-at-death and pathological assessment is presented, and considered with respect to etiological and pathogenic implications. This discussion concludes with an exploration of how this analysis of fetal, perinatal and infant individuals has revealed unique insights into the precarious experiences of early life.

10.1 Growth Disruption:

Examining and considering evidence of growth disruption has long been a central concern of many anthropological and clinical studies (Kaplan 1954, 780; e.g. Johnston 1962; Tanner 1963; Maresh 1970; Tanner 1978; Johnson & Zimmer 1989). Many of these were primarily interested in the effects of a range of environmental factors on growth and attempted to correlate these environmental stresses with evidence of growth disruption (Kaplan 1954, 780; e.g. Tanner 1981; Johnston & Zimmer 1989; Bogin & Loucky 1997; Bogin *et al.* 2007). Today, implications of a reduced *in utero* and postnatal environment and experience have been widely considered (Holland Jones 2005, 22; e.g. Barker 1997; 2012; Barker *et al.* 2002; 2012), highlighting a complex interaction between evidence of growth disruption and exposure to stress. Stress is a complex concept (See Chapter 3; Section 3.43 for discussion) with growth disruption typically considered to be an indicator of stress, whereby detrimental conditions have affected the optimal development and growth of the individual (Goodman *et*

al. 1988, 169; Goodman & Armelagos 1989, 226; Reitsema & McIlvaine 2014, 181). Determining the etiology of growth disruption is challenging, relying on a multifaceted consideration of both intrinsic and extrinsic factors (Johnston *et al.* 1976; Goodman & Armelagos 1988, 941-942; Goodman *et al.* 1988, 169-170; Bush & Zvelebil 1991, 5). For archaeological individuals the identification of growth disruption is of greater complexity as the age of the individual, and thus their expected growth attainment, their sex, and the social and environmental conditions to which they were exposed, must all be considered in relation to their growth and development. These variables are ones which can be attributed and estimated using various archaeological and bioarchaeological methodologies. However, there are inherent limitations when interpreting skeletal remains for past populations.

Growth disruption is identified using age estimates calculated from dental and skeletal elements, whereby stress and stressors may be correlated with metric changes and inconsistencies between growth profiles, indicative of an abnormal physiological and skeletal response (Bush 1991, 11). For individuals identified within this thesis where skeletal age-at-death estimates lag considerably behind dental age estimated, growth disruption has been posited. As all individuals were assessed using the same methods, the same inherent limitations exist in all samples. The methods employed were used due to their ease of application, their comparability to other published studies, and their ability to provide error ranges for the age estimates derived.

Individuals analysed throughout this thesis derive from varying skeletal samples (See Chapter 4; Tables 4.1 and 4.2). Consequently, variation between the archaeological samples and the reference sample from which age estimation methodologies were produced, may result in higher levels of inaccuracy and variability (Lewis 2007, 38; e.g. Demirjian 1990; Saunders *et al.* 1993). Furthermore, age estimation methods derived in clinical/anthropological contexts (e.g. Maresh 1970) are based on samples of healthy non-adults, limiting the comparison of non-survivors from archaeological contexts (Saunders & Hoppa 1993; Lampl & Johnston 1996). It has been suggested that the direct comparison of modern clinical and anthropological data to archaeological human remains renders interpretations of health in the past problematic (Wood *et al.* 1992; Hoppa & Fitzgerald 1999, 13; DeWitte & Stojanowski 2015, 406). Furthermore, intrinsic genetic variables which affect growth, such as ancestry and sex (See Chapter 8 for evidence and discussion), cannot be controlled for in the archaeological samples. However, current literature suggests that any disparities as a result of

these biological differences should not be significant (Ruff *et al.* 2013, 30); Habicht *et al.* (1974) first reported that variation in growth in the first seven years of life is almost exclusively regulated by the environment, not genetics. In fact, optimal *in utero* experiences will make individuals from different populations comparable (Ruff *et al.* 2013, 30). Despite clear differences in growth trajectories when individuals of known biological sex and ancestry were assessed (Chapter 8), when error ranges for age estimates were included there was parity between dental and documented ages. Consequently, within this study, wide age ranges, based on error levels given within the methodologies utilised, have been afforded in order to encompass variability between dental and skeletal age estimates, so that small differences are not interpreted as evidence of growth disruption. By applying error ranges to both dental and skeletal data the chance of age estimates over-lapping ranges increases. Where growth disruption is reported here, it is only when there are substantial differences between physiological age estimates, beyond the error ranges of the methods utilised. As this study is not primarily concerned with the demographic profiles of the samples assessed, but evidence of growth disruption, what is significant here are the differences between dental and skeletal age estimates on an individual level.

As dental development is considered to be less susceptible to environmental stressors than skeletal growth (Gustafson & Koch 1974, 298; Bolaños *et al.* 2000, 98; Humphrey 2000a, 194; Liversidge & Molleson 2004, 172), dental age estimates have been used throughout this thesis as a proxy for chronological age. Consideration of documented age, against age estimates derived from dental development, found a close relationship between the two (See Chapter 8). Further comparison of documented age estimates to the *pars basilaris* corroborated that dental age is a relatively accurate proxy for chronological age. This is because parity was found between documented and *pars basilaris* age estimates and dental and documented age estimates. Thus, the *pars basilaris* appears to be a good proxy for dental development and dental development a good proxy for chronological age. It is considered that the *pars basilaris*, over those of the long bones, is a reliable skeletal element for age estimation. This is because formation at the base of the cranium appears to show a prioritisation in growth and hence be more robustly buffered from environmental stressors in comparison to postcranial elements. Further research into the applicability of the *pars basilaris* to estimate age accurately is subsequently required, though analysis of this bone in manuscript two also demonstrated comparable age-at-death estimations between this element and dental age.

Consequently, the methodology and practices assumed within this study are suggested to be robust in distinguishing true evidence of growth disruption.

10.11 Interpreting Evidence of Growth Disruption:

Of the 423 individuals assessed within this study, 209 had dentition available for assessment and 390 had at least one long bone measurement which could be assessed for age-at-death. However, only 192 individuals had both dental and skeletal elements available for assessment. Of these, 175 individuals with dentition and long bone measurements (femoral, tibial and humeral) have been plotted in Figure 10. 1 – individuals have been plotted in ascending order according to dental age estimates, with the time period from which they derive denoted by the data marker used. Of these, 39 individuals display clear evidence of growth disruption, where no skeletal age estimates or age ranges overlap with dental age estimates and ranges (Table 10.1). Only eight individuals have long bone age estimates that exceed dental age: three individuals from Barton Court Farm, one Owslebury individual, two East Smithfield individual, and two individuals from the Smithsonian Fetal collection. Given these limited numbers of individuals, particular within a sample of deceased non-adults, it may be that these individuals reflect evidence of healthy growth.

TABLE 10.1 Number of individuals (%) with growth disruption between dental age and femoral, tibial and humeral age.

	N	**Growth Disruption (*N*)**	**Growth Disruption (%)**
Dental - Femoral	139	57	41
Dental - Tibial	130	50	38
Dental - Humeral	159	63	40

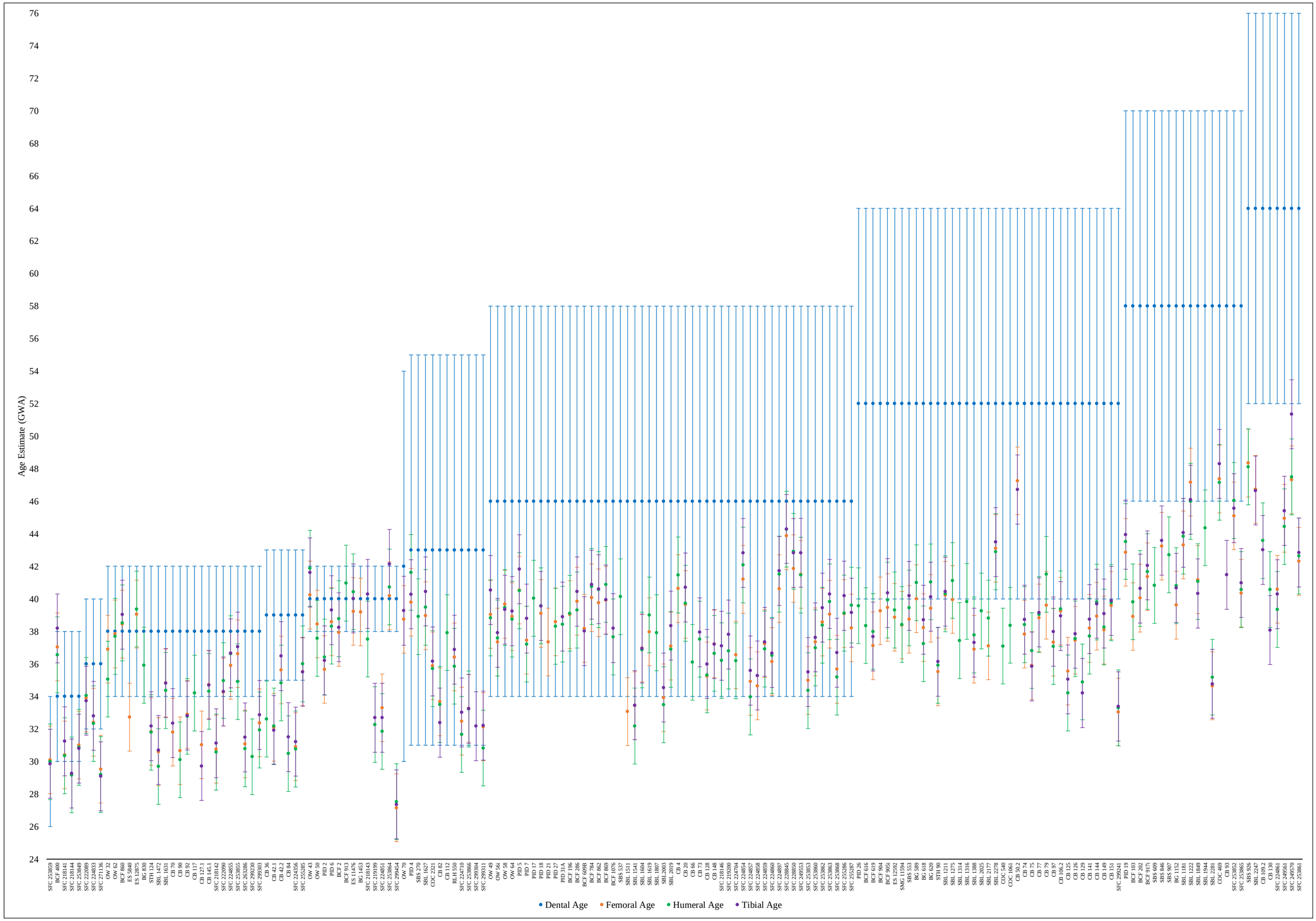


Figure 10.1 Skeletal and dental age-at-death estimates plotted for each individual with age-estimates available. Error ranges (in GWA) have been afforded to both dental and skeletal age estimates in accordance with the age estimation methodologies employed (AlQahtani et al. 2010; Scheuer et al. 1980). Individuals considered to show evidence of growth disruption are those where dental and skeletal age estimates and ranges do not overlap.

However, many more individuals are suggested to have growth disruption when skeletal elements are considered individually (Table 10.1), which suggests that the femur is most frequently affected, followed by the humerus and tibia. This pattern of growth disruption identified was unanticipated given that the femur and tibia are known to be the most sensitive long bones to environmental stress (Pomeroy *et al.* 2012). However, when individual differences between dental and skeletal estimates are considered, for those found to show evidence of growth disruption, the tibia was found to have the severest evidence of growth disruption, with one individual showing a difference of 26GWA between dental and skeletal estimates (Table 10.2). Thus, the susceptibility of the tibia to environmental and social stress (Pomeroy *et al.* 2012) may be reflected in the severity, rather than the frequency, of growth disruption.

When considered by dental age those determined to be 39, 52, 58 and 64 GWA were found to have the greatest number of individuals showing growth disruption (Table 10.3). These large differences may be attributed to variable causes. Firstly, it may be that inherent limitations of the methods employed are affecting these results. Skeletal age-at-death estimates have been generated using Scheuer *et al.* (1980) who only developed these regression equations using individuals aged up to 46 GWA (Scheuer *et al.* 1980, 258). Thus, eight individuals have skeletal age estimates above this 46 GWA threshold, suggesting that this method of skeletal age estimation may not be appropriate for these individuals (Gowland & Chamberlain 2001; 2002).

However, such severe growth disruption in these older infants may result from an adverse postnatal environment. Once born, individuals are fundamentally more exposed to increased numbers of environmental, social and cultural stressors (Lewis & Gowland 2007). Immunological buffering, typically provided by the mother through feeding, is not automatically afforded and relies on her capability to feed the child. Death in childbirth and maternal nutritional deficiency can result in compromised or non-existent maternal feeding**,** with inadequate substitute foods used instead. Furthermore, cultural practices regarding infant feeding vary and social factors may be implicated within some of these individuals. Thus, it must be considered that the post-partum environment provides increased nutritional challenges for these infants, which in turn increases their susceptibility and vulnerability to a range of disease processes. In addition, care of these individuals, with regard to their environment and socialisation, has been found to be central to their healthy growth and

development (Hannault 2002). Keeping an infant warm, and social interaction with a child has been found to increase success of growth (Hannault 2002), though social practices such as swaddling, and keeping infants in doors – which were considered to be 'good practice' – can be similarly detrimental (Brickley & Ives 2008; Newman & Gowland 2017). Thus, although the intrauterine environment is highly variable and significantly regulated by maternal practices and experiences, extrauterine life is even more precarious, in which the infant can be exposed to a broader range of potential stressors. Furthermore, for the older infants, more severe growth disruption might simply reflect a longer period in which skeletal evidence can manifest, meaning much of this disruption is likely to have commenced *in utero.*

Growth disruption has also been found to vary depending on the gestational age at which an insult or stressors was experienced. 'Critical periods' for growth and development have long been acknowledged, and fetal growth is considered to be most prone to disruption (Armelagos *et al.* 2009, 267). Heinke and Kuzawa (2008) have demonstrated that growth is affected much more severely later on *in utero* in response to reduced maternal health status. Although growth is known to be prolific within the first trimester, when limb bones can grow up to 3mm within a week (Issel 1985), it slows down as gestation increases, to around 1mm a week in the third trimester (Issel 1985). This is a result of both the reduced need and capacity for growth to occur. Growth of certain skeletal structures is known to be prioritised *in utero* – the cranium in particular (Barker *et al.* 2012, 30). Consequently, the individuals who show excessive growth disruption in the older gestational age categories may reflect a more severe disruption experienced immediately prior to birth, where the growth of other skeletal elements was prioritised. Furthermore, if the mother was experiencing poor health immediately prior to birth, it may be that the illness/stressors were likely to have continued (either briefly or more chronically) into the postnatal period. This is turn may be reflected in the offspring's growth and development and their increased vulnerability after birth.

Considering the individuals by time period, the post-Medieval individuals have the highest growth disruption (Table 10.4). There is an increase in growth disruption throughout the time period up to the post-Medieval period, where after this, the 20th century individuals show a marginal reduction in growth disruption. Growth disparities thus may reflect the increasingly stressful and pathogen-filled environments over time. Furthermore, increasing socioeconomic

stratification and poverty factors may explain this increase in growth disruption throughout the periods.

TABLE 10.2 Differences (GWA) between dental age estimates and skeletal age estimates for individuals found to show evidence of growth disruption to the femur, tibia and humerus.

Age Difference (GWA)	**Femora**	**Tibia**	**Humeri**
	N=57	*N*=50	*N*=63
4	1	1	1
5	2	3	2
6	4	2	3
7	7	7	4
8	1	3	6
9	-	-	1
12	-	-	1
13	12	9	7
14	5	6	13
15	8	1	5
16	4	3	6
17	4	6	7
18	3	2	2
19	3	2	1
20	-	-	2
21	-	2	1
22	1	-	-
23	2	1	2
24	-	1	-
25	-	-	1
26	-	1	-

TABLE 10.3 Number of individuals with growth disruption by dental age.

Dental Age Estimate (GWA)	*N*	Growth Disruption (*N*)	Growth Disruption (%)
30	1	0	0
34	7	1	14
36	3	1	33
38	23	11	48
39	6	4	67
40	16	4	25
42	1	0	0
43	11	0	0
46	59	3	5
52	43	28	65
58	21	15	71
64	15	8	53
70	1	0	0
76	1	0	0
82	1	0	0

TABLE 10.4 Percentage of individuals, of those with both dental and skeletal elements available for assessment, with growth disruption by time period.

Time Period	*N*	Growth Disruption (*N*)	Growth Disruption %
Pre-Roman	13	1	8
Transition	16	2	13
Roman	14	5	36
Medieval	7	3	43
Post-Medieval	97	46	47
20th Century	57	17	30
Undated	5	1	20

10.2 Health Disruption:

Although health may typically be considered as the overall wellbeing of an individual and the lack of any disease processes being present, health is in fact a much more variable construct based on individual response to potential insults (Goldenberg & Thompson 2003). Ultimately what might affect one individual, may not affect another, while interpretations of 'healthiness' are contingent on the perception of health and its representation within that society (Goodman *et al.* 1988; Roberts 2009: 154; Huber *et al.* 2011; Reitsema and McIlvaine 2014: 181).

Health and healthiness is also considered to be an interpretation which, within bioarchaeology, we have to be very cautious in attributing (Waldron 2009, 10). In fact, what we can reconstruct is not in fact health, but evidence of disease, yet lack of disease does not equate to health (Waldron 2009, 9). Both psychological factors, which cannot be identified within the archaeological record, and the paradoxical limitations of evidence of disease, hinder and complicate interpretations. Few diseases cause physical changes to the skeleton, meaning diagnosis of conditions is limited, especially when the skeleton can only respond in a limited number of ways to a multitude of conditions (Ortner 2003, 45; Gowland 2004, 139). Thus, the pathological lesions identified on skeletal remains *'…represent a small percentage of the total disease load in that population'* (Redfern 2003, 150). However, any response at all by the skeleton is suggestive of an attempt by the body to adapt to the conditions experienced (Klaus 2014). Although many studies use woven bone as an indicator of the very initial stages and exposure of stressors, this bony response has been considered by some to instead simply be the very first stage of healing (Klaus 2014). Weston (2012) argues that all bodily growth (including woven bone) ceases under adverse circumstances, whereas others argue that the skeleton is still able to mount a response to adversity (Klaus 2014). Although new bone formation is the most commonly considered non-specific indicator of stress, other skeletal changes can be observed and this study has attempted to also consider bone destruction and altered morphology.

However, it must be remembered that all of the individuals assessed within this study were non-survivors. Consequently, they represent all of those who could not maintain and regulate homeostasis. With fetal, perinatal and infant individuals, this study assumes that these individuals do not represent victims of infanticide, partially due to lack of evidence

(Goodman & Armelagos 1989, 228), but also because infant mortality is known to have been relatively common in the past (Guy *et al.* 1997, 221). Therefore, these individuals are those who either suffered from an intrinsic genetic or congenital abnormality, or who were unable to buffer and respond to a multitude of stressors experienced. The similarity in the ways disease processes present, as well as the likelihood that many disease process will never or very rarely affect the skeleton means constructing 'normal' profiles for expected amounts of disease within this population is inherently problematic.

10.21 Evidence of Health Disruption:

Of the 423 individuals considered within this study, 386 were found to have either cranial or postcranial skeletal elements present for analysis. Individuals from all samples and time periods were found to show evidence of lesions. Individuals within this study have been found to have an overall prevalence rate of 70% for cranial pathological lesions and 30% for postcranial lesions. When considered by time period, cranial lesions were found to be most prevalent in the Transition and post-Medieval samples, and postcranial lesions in the Transition sample. These high rates of pathological lesions are not unexpected (Krenz-Niedbala & Lukasik 2017, 375) given that these individuals represent the non-survivors.

10.22 Pathological Lesions –Their Location and Presentation:

The bones most commonly found to show evidence of pathological lesions are the frontal bone and the tibia. It might be considered that these bones are most commonly found to show pathological lesions as a result of various mechanisms, including the stressor/insult being experienced, the severity of that insult and the importance of growth prioritisation within that element.

It is suggested that the bones of the cranial vault are typically affected due to both their highly vascular structure, and the need for growth to be prioritised within these elements (Humphrey 2000a; Barker *et al.* 2012, 30; Chapman *et al.* 2013a, 386). Thus, as a result of their vascular nature, NBF might be considered to be more prolific as the body, and bone, attempt to maintain and continue growth within these elements, despite stress being experienced. If indeed growth does completely arrest when stress is experienced (Weston 2012) it might be that the cranial elements are the last to see this cessation, and as a result show more prolific evidence of a pathological response.

Periosteal NBF has long been considered to be a non-specific pathological lesion, and thus indicator of stress. Weston (2008; 20012) has found that the periosteum responds in an identical way to a multitude of varying etiologies, highlighting how specific causes cannot be attributed. Furthermore, she considers that the typical association of periosteal NBF with pathological processes of infection, rather than other causes, may be over-exaggerating the level of infectious disease processes in the past. As manuscript 4 discusses, there are complexities when interpreting the implications of NBF in fetal, perinatal and infant individuals. Normal somatic growth results in NBF which mirrors the appearance of pathological bone formation – woven bone is commonly deposited during the rapid phases of growth during pre- and postnatal life (Lewis 2007; 2017a). Early clinical investigations found that it was difficult to distinguish between pathological and normal bone formation (e.g. Shopfner 1966), and subsequently, such opinions have been widely disseminated within bioarchaeological literature (Ribot & Roberts 1996; Lewis 2004; Weston 2012; Lewis 2017a). However, recent clinical investigations have revealed that NBF is not commonly found in individuals less than 44GWA and more than 56GWA (Kwon *et al.* 2002). This suggests that when NBF is identified on individuals who are aged to be younger or older than this range (44 -56 GWA), the impetus can be suspected to be pathological. However, this interpretation is limited in that it fails to consider then how fetal, perinatal and infant bones continue to grow. Consequently, it may be that NBF in association with normal growth is present beyond these suggested age ranges, however, NBF is particularly marked and obvious during this 44-56 GWA window due to the rapid postpartum growth spurt. Thus, it may be suggested that it is during this narrow age range that normal and pathological NBF is most difficult to determine and distinguish between. Further investigations into these distinctions are required, though this study has detailed extensive NBF (of severity 2 and 3) that is present in individuals both within and beyond this age range.

Lewis suggests that evidence of NBF which is a result of pathological processes is more likely to be localised and unilateral (2007, 135), compared to NBF associated with normal growth which will be circumferential and a thick single layered deposit (Shopfner 1966; Lewis 2007: 135; Weston 2012: 497). However, many health insults, particularly those of metabolic disturbances are systemic and thus unlikely to affect individual elements. Thus, evidence of NBF bilaterally does not mean that pathological processes are not a potential cause. Furthermore, the periosteum is more susceptible to disturbance in these young individuals, because it is less securely attached to the cortical surface – this makes it more

likely to rupture and respond to health insults (Resnick 2002: 2397; Lewis 2007: 135). Thus, NBF would be expected to be more commonly identified in these individuals as a result of both pathological and normal processes. However, differentiating between normal and pathological formation within the archaeological record is problematic. It is suggested that future studies require analysis of location, type, laterality, thickness (including evidence of multiple layers) and should be considered in relation to estimated age-at-death.

Weston (2012, 506) has argued that NBF should not be used as an indicator of stress due to NBF being inhibited under subjection to stress. However, as Selye (1973) has highlighted**,** there are multiple phases to the stress response, so although the initial phases may be to halt growth as part of the alarm mechanism, the body then attempts to adapt and accommodate these stresses – thus it is likely that bony responses can occur. Klaus (2014) supports this notion suggesting that stress and growth has a much more intricate and bounded relationship than simply an on or off mechanism (Klaus 2014, 295-299). Thus, NBF itself, even in the woven response stages, may indeed reflect a healing response, whereby the body has stabilised and is expending energy in order to attempt to maintain and accommodate stress exposure. As ever, the paradox of this is that lack of lesions may indicate both an absence of stress and an inability for the individual to respond to the stress.

New bone formation was the most common type of lesion identified in the individuals assessed, for all age groups and for all time periods. It could be argued that some of the instances of NBF recorded represent normal bone formation – perhaps in the severity grade one category - and thus the identification of pathological lesions has been over-represented here. However, the NBF recorded in these individuals was similar to that recorded in other studies describing the presentation of these lesions, and corresponds to locations where pathological changes are believed to be most expected (e.g. anterior tibia). Although periosteal NBF can occur on any bone surface, the anterior tibia has been found to commonly show these lesions. This tendency is considered to be a result of its close proximity to the skin/surface and greater vascularity (Roberts & Manchester 2010, 173). The tibia was found to be the most commonly affected postcranial element with regards to pathological lesions identified. Thus, patterns of pathology identified appear to correlate with the known expression of conditions and helps substantiate that this bone is one of the primary skeletal elements to show pathological changes. Furthermore, the majority of both cranial and postcranial skeletal lesions were recorded as being of severity two, suggesting these lesions

were typically more defined/thicker than those of severity grade one, which are perhaps more likely to be associated with normal NBF.

Within the cranium NBF is typically concentric (Lewis 2017a, 3), and for the frontal bone and parietal bone in particular, NBF is found in patterns which do not appear to correlate to concentricity but instead vascular structures – typically mirroring and outlining the paths of vascular structures in a manner considered to be abnormal (Schultz 2001, 128; Rumbaugh & Potts 1966, 532-534). Furthermore, for some individuals there are multiple isolated lesions, again not consistent with a normal pattern of growth. Within the long bones NBF is much harder to distinguish as pathological, with diagnosis typically relying on the thickness and extent of the formation (Kwon *et al.* 2002). For some individuals analysed (*N*=35), there is a circumferential layer of new bone formation, with many of the pathological lesions extending to the metaphyses. The thickness, in many cases of this NBF, is also indicative of its pathological nature. Although thickness was not accurately recorded, and requires radiological assessment, this is a consideration for future study. Only individuals aged between 44-56GWA are thought to show periosteal NBF on the long bones as part of a normal growth process (Kwon *et al.* 2002). In contrast to these clinical findings, individuals of all ages were found to show evidence of NBF in this study.

If NBF is indeed considered to reflect stress exposure, it might be anticipated that these detrimental insults would quickly be reflected within fetal, perinatal and infant skeletal remains, as bone growth and turnover is exceptionally rapid at this point (Lewis 2007). Consequently, these individuals may rapidly reflect the stressors and insults being faced just prior to death. These initial, active phases of bony response to a variety of stressors manifests as rapidly deposited disorganised and porous (woven/fiber) bone (Schultz 2001, 115; Ortner 2003, 206; Lewis 2007, X; Kini *et al.* 2012, 30). If and when healing commences, this NBF becomes remodelled, gradually becoming orientated into linear, organised and smooth cortical bone (Schultz 2001, 115; Ortner 2008, 198; Kini *et al.* 2012, 30; Larsen 2015, 87). Depending on the type of bone identified, it is possible to determine whether physiological responses were active, healing or healed at time of death (Mays 1998, 179-182).

In the majority of the individuals assessed, for postcranial lesions NBF was primarily woven in appearance. This indicates that although the body had time for a physiological response to manifest, the stressors and impetus for this lesion are likely to have still been active at the

time of death. For cranial bones, particularly the endocranial surface, NBF was primarily lamellar, indicative of an initial healing response. It is suggested healing may have been prioritised within the cranial elements so as to maintain healthy skeletal growth and brain development. The fact that many of the cranial lesions did show a healing response may indicate that many were able to mount an immune response to the health stress experienced. However, evidence within the majority of individuals for both lamellar (within the cranium) and woven (within postcranial elements) bone formation may also suggest that individuals were exposed to multiple stressors, or multiple episodes of stress. In total 21 individuals show evidence of both woven and lamellar bone formation within a single skeletal element. Differential healing stages may indicate that whilst an immune response was attempted, ultimately the individual was unable to overcome the stressor(s) experienced. However, given that many individuals also show evidence of growth disruption it may also be considered that bone turnover, and consequently healing bony responses, were slower in these individuals. Of course, it must not be overlooked that the sample of fetal, perinatal and infant individuals assessed is that of the non-survivors, and given the health and growth disruption identified, likely represent those who succumbed to death as a result of exposure to various stressors.

Evidence for metaphyseal expansion (flaring) is considered to be indicative of metabolic disturbances where both bone and vascular structures are weakened, leading to morphological changes within skeletal elements, particularly the long bones (Brickley & Ives 2008). Metabolic disorders are equally correlated to evidence of NBF (Brickley & Ives 2008), thus co-occurrence of NBF and metaphyseal expansion within individuals assessed suggests many may have been suffering from metabolic disturbances and deficiencies. Morphological changes and metaphyseal expansion were both found to be more prevalent in dentally older (> 40GWA) individuals. This may similarly indicate that the postnatal environment compromised individuals in different ways. Metaphyseal expansion may be more apparent in older individuals as a consequence of increased weight-bearing, where unmineralized osteoid leads to the bowing of the limbs (Brickley & Ives 2008). However, metaphyseal expansion has been identified in those as young as 19 GWA *in utero* (Mahon *et al.* 2010, 14), suggesting that metabolic disturbances do result in morphological changes prior to bone loading. Furthermore, skeletal elements which have decreased mineralization, as a consequence of nutritional deficiency, are thus, ‘soft’, and can display morphological changes (i.e. bowing) as a result of space restriction *in utero* (Bonneau *et al.* 2011).

10.23 Interpreting Pathological Lesions as Health Disruption

Regarding the etiological, pathogenic and contextual implications of these pathological lesions a very complex and nuanced narrative emerges of health disruption. It can be considered that given the prevalence rates of pathological lesions and the presence of these within all age categories, time periods, and expressed in multiple ways, shows it is likely that many of these populations experienced chronic health stress. To understand the intrinsic and extrinsic interpretations of theses pathological changes aetiological and contextual implications are considered.

10.24 Pathogenic Interpretations:

Given the pathological lesions recorded, it is likely that two main pathogenic causes stimulated these changes – metabolic disorders and infectious disease (both specific and non-specific). However, it must not be overlooked that some of these changes may equally represent congenital conditions and more traumatic processes such as birth trauma.

Metabolic changes are considered to be the most prevalently recorded lesions as malnutrition often results in general indicators of ill health (Lewis 2007, 66; 97; Mensforth *et al.* 1978). Many nutrients and micronutrients are vital for our growth and health, particularly *in utero*. Thus, although metabolic disorders are often considered to leave non-specific pathological lesions, their known presentation correlates with many of the lesions identified on individuals studied.

Nutrients such as vitamin C, vitamin D, calcium and iron, are vital in the initial stages of life outside the womb (Lewis 2007, 98); lack of these nutrients would further make the individuals more susceptible to a range of diseases and infections. Individuals expressing NBF, metaphyseal expansion and torsion/bowing of the limbs might be considered to have experienced vitamin D and C deficiency. Vitamin C and D deficiencies can affect growth and development of the skeleton; lack of vitamin C results in defective osteoid formation (development of new bone cells) (Brickley & Ives 2006, 163) while vitamin D deficiency affects the mineralization of bone (Lewis 2007, 121; Kini *et al.* 2012, 55). Lack of vitamin C can result in blood vessels becoming fragile and easily ruptured (Brickley and Ives 2006, 163; Ortner and Ericksen 1997, 213). This can lead to haemorrhaging, including in the vessels supplying the metaphyses, but particularly within the cranium (Roberts and Manchester 2010, 235). Some of the individuals with evidence of NBF to the endocranium

may have suffered cranial haemorrhaging (Brickley and Ives 2006, 168; Schultz 2001, 117) and chronic bleeding (Ortner *et al.* 1999, 328) as a result of nutritional deficiencies. Vitamin A deficiency is a further factor to be considered as we are unable to synthesize this nutrient independently and are required to get our intake from dietary sources alone (Fujita *et al.* 2017, 1). Importantly, we require this vitamin for a variety of functions, including both our growth and immunity (Fujita *et al.* 2017, 1). In particular, maternal breast milk is known to have high concentrations of vitamin A as a result of the post-partum infant requiring increased levels of this nutrient to be able to sustain rapid growth (Fujita *et al.* 2017, 1-2).

Infectious diseases are less readily identifiable in fetal and young infants. Although many infectious diseases can transfer to the fetus through the placenta, many lesions may still be soft-tissue related, or present later in life. For example, congenital syphilis is known to cause Hutchinson incisors and mulberry molars (Ortner 2003; Ogden *et al.* 2007), and although dentition does develop and is present *in utero*, definitively identifying these changes in such young individuals is difficult. Conditions such as tuberculosis are known to attack vertebral bodies and joints, such as the femoral head and acetabulum, but these structures are not sufficiently ossified at this age for lesions to be identifiable. Furthermore, the bones do not tend to be affected until later on in the disease process. Thus, bony response to specific infectious diseases is limited. Non-specific pathological lesions, such as NBF, may be an indicator of infectious conditions in some instances, but it would be difficult to differentiate between etiologies (Nade 1983, 113).

Infections are considered to be relatively typical during early development (Eisenberg *et al.* 2017, 1; Degani 2006, 329), and many infections/diseases are possible: these include meningitis, rubella, measles, smallpox, puerperal fever and diarrhoea (Lewis 2017a). Although the specific pathological lesions for such conditions are unknown, some of the non-specific lesions identified may be attributed to such conditions. In particular, extensive and thick NBF to the long bones may be indicative of osteomyelitis. Osteomyelitis is a result of inflammation elevating the periosteum away from the cortical bone surface and creating a new layer of bone, often resulting in a thick, bony sleeve of bone (Rana *et al.* 2009, 265). Additionally, Rana *et al.* (2009) suggest that bilateral evidence of periosteal NBF is evidence of systemic disease response, although some contradict this sentiment, suggesting bilateral formation is more indicative of normal growth processes (Lewis 2007, 135).

Congenital conditions were known to be present in eight individuals from the Smithsonian Fetal Collection, and six individuals from the other samples are suspected of presenting congenital changes. Congenital conditions are those in which either genetic or environmental factors adversely affect the developing fetus. Within the 20th century population assessed a number of individuals were diagnosed with spina bifida, hydrocephaly, anencephaly and iniencephaly (Hunt, *personal communication*). Scalloping and fenestration of the cranial vault and cranial base elements is suggestive of either increased cranial pressure or haemorrhage (Chapman *et al.* 2013b, 462), often as a result of one of the previously listed conditions.

10.25 Etiological Interpretations:

The etiological implication behind these various disease processes include disease load of the population, nutrition, the pre- and postnatal environment, and both genetic and epigenetic factors.

During the *in utero* period there is a constant interaction between the fetus, placenta and mother (Harding & Johnston 1995), with the mother and placenta typically acting as barriers and regulators from external stressors (Barker 2012, 187). This dyad between mother and child ensures the optimal *in utero* environment, prioritising the needs of the unborn child and increasing the chances of survival (Gowland 2015, 4). Consequently, maternal nutrition is pivotal for the health and wellbeing of offspring, as well as their optimum growth and development. Maternal nutritional status both during and after pregnancy, and in fact even throughout her whole life course, is vital in regulating the nutritional resources available for the offspring. However, if the mother is experiencing nutritional or health stress, the 'giving' potential of the mother is limited. This may result in the interrupted interaction between fetus, placenta and mother, leading to a host of detrimental conditions and birth outcomes arising (e.g. anaemia, maternal haemorrhage and IUGR) (Wu *et al.* 2012, 4).

One of the earliest studies into maternal nutritional disruption studied famine victims from the First World War (Ivanovski 1923). This study was one if the initial publications to highlight the intrinsic link between skeletal growth, health and diet. Maternal nutritional status and weight can regulate growth and development of the fetus, and thus the diaphyseal lengths of long bones (Hauspie *et al*. 1994; Adair 2004), as well as reduce immune function for both mother and child, which can result in physiological and psychological stress, disease

and death (Lewis 2007, 66). IUGR (Intrauterine Growth Restriction) can also be a result of a limited nutrient and/or oxygen supply *in utero,* whilst stillbirth can be a result of maternal illness, infection, chronic disease and extreme malnutrition (Lewis 2007, 134; Goldenberg & Thompson 2003). Thus, a mother's disease status, and her biocultural experiences, play a crucial role in determining the health of her child, both *in utero* and beyond (Gowland 2015).

In addition, postnatal health and wellbeing is also dependent on a variety of factors, generally regulated by the mother/caregiver, such as feeding practices and the nutritional status of the breastfeeding mother (Gowland 2015; Ramji 2009, 625). Breastmilk, particularly that of colostrum (the initial thick breastmilk available directly after birth), is vital for both the nutritional and immunological wellbeing of the infant (Eisenberg *et al.* 2017, 2; Lewis 2017b, 31). Inability of the ill/nutritionally deficient mother to provide milk would further exacerbate any deficiency experienced by the child and potentially increase their susceptibility to a range of diseases and illnesses post-partum.

The point of birth is one of the most stressful biological events in our life course and heralds a multitude of biological, physical and environmental changes (Bogin 2001, 69). Birth is not only obstetrically risky for mother and child alike (Reid 1990, 621), but signals the transition for the child from a protected environment into one full of pathogens, and bacteria, all of which the individual needs to build an immune response to (Lejarraga 2012). It is the mother who is required to provide protection, both nutritionally and immunologically, for her child postnatally (Eisenberg *et al.* 2017, 6).

Obstetric death is one factor which must also be considered in light of perinatal mortality. Today rates of maternal death in the U.K. are low (8.5 women in 100,000 (MBRRACE-UK Maternal Death 2016 *Lay Summary*)) but it is unlikely that these rates were so low for archaeological and historical populations. Wrigley and colleagues (1997, 236) have estimated that mortality rates in England between 1580-1837 were between 4.7 and 17 per 1000 births. In particular, death rates of infants were known to be substantially high for the post-Medieval samples (Forbes 1972). Obstetric death can be caused by obstructed labour, breech birth, haemorrhage and placental detachment (Lewis 2007, 34). All of these conditions were likely experienced in archaeological populations, yet without modern medical care, would have led to high levels of obstetric death (Lewis 2007, 34). Obstetric death is significant because it clearly has implications for health and wellbeing of mother and offspring alike – chances of

survival would be poor and so many individuals (mother and child) likely died as a consequence. However, for those offspring who did survive without their mother there may have been long term health consequences, particularly as a result of compromised feeding strategies.

In addition, this study cannot overlook the theoretical implications of epigenetics and the DOHaD Hypothesis. Epigenetic research has expanded rapidly over the last decade, and theoretical implications of this work have started to be incorporated within the corpus of bioarchaeological literature (e.g. Gowland 2015). Fetal individuals must not be considered to be autonomous (Gluckman 1997, 153), as bounded or discrete entities, but instead their life course is intrinsically entangled with that of their mothers (Gowland 2015; Redfern 2003, 162). Epigenetic changes are those in which gene expression can be altered as a result of the environmental determinants being experienced *in utero* (Cattaneo 1991, 40). Such changes affect the phenotype, simply the expression of our genetic 'code' (Cattaneo 1991, 40). This genetic adaptation to the conditions experienced *in utero* can cause long term health consequences for the fetus (Barker 1997; 2012; Barker *et al.* 2012; 2002). Changes such as an increased risk of heart disease and diabetes have been found to be related to adverse early-life environments (Barker 2012). Epigenetic changes are known to directly impact on several generations (Holland Jones 2005). Barker coined the idea of a '100 years of nutritional flow' (2012, 31) showing the multigenerational impact of a single detrimental *in utero* experience. This is a theoretically important concept when considering fetal, perinatal and infant individuals who may be considered 'frail', having an inherent biological inability to withstand many of the stressors experienced in the early life course.

10.3 Understanding Early Life Disruption: Stressed at Birth?

The following discussion considers the evidence for growth and health disruption presented and contextualises these results in regards to the archaeological and historical context of these samples. Correlations between growth and health disruption show that certain samples and age groups appear to have experienced more stressful starts than others.

Although levels of growth and health disruption may be considered to be relatively high in these samples, early life mortality was exceptionally high in many of these populations (Volk & Atkinson 2008, 103), and thus many individuals are likely to have died as a result of adverse early life experiences. Within preindustrial societies 'children' would have

constituted at least one third of the population (Chamberlain 2006, 178) and infant mortality is often considered to be somewhere between 30-50% (e.g. Schultz 2001, 129; Gowland 2001, 155**;** Wiedemann 1989, 12-16). Vulnerability is considered to be at its peak in the first year of life, where susceptibility to disease and death is highest – this vulnerability decreases with age, although it is not until after the first year that survival chances significantly increase (Lewis 2007, 5). Coupled with the environmental and social stressors that many individuals would have been exposed to, death of those within the most precarious age bracket is not unlikely, and thus, reflection of this fragility in growth and health disruption is not unexpected.

The relative lack of individuals aged dentally to be between 38 and 42 GWA is very intriguing. This is typically considered to be the point at which birth is most likely to occur, and as a result we may expect an increase in the number of individuals dying at around this time. Of all 423 individuals assessed, 46 fell within this age category based on dental estimates, with 11 aged to be younger than 38GWA and 152 over 42 GWA. Although obstetric risks are present regardless of what point birth occurs, both pre-term and post-term births have been found to have higher rates of mortality (Jeanty & Romero 1984, 127). Thus, although we may expect to see a higher frequency of individuals aged between 38-42 GWA to represent perinatal mortality associated with birth, we must remember that in the past, as today, birth can occur at multiple points and result in very differing outcomes. Those under 38 GWA and over 42 GWA at birth are considered to be riskier and have higher associated mortality. Those individuals assessed to be under 38 GWA potentially represent those of premature birth who were unable to survive either birth or postnatal experiences. In contrast, a higher representation of individuals in the post-42 GWA category could represent both individuals who were born at full-term and who succumbed to death after numerous weeks in the postnatal environment, or may equally represent those of individuals who were post-term and died within the birth process. However, it is uncommon for individuals to be over 2 weeks overdue, suggesting evidence for these individuals might be scarce within the archaeological record. Furthermore, being able to distinguish between individuals who were born and not born, and identifying those who were likely to have been small for gestational age (SGA) becomes of paramount importance to interpretation, as varying implications as to their death could be attributed. Work by Booth *et al.* (2016) is beginning to unravel these complexities, considering evidence of bioerosion as an indication of survivorship, and ultimately distinguishing those who had fed, from those who had not.

10.31 Contextual Interpretations of Growth and Health Disruption

Socioeconomic status, maternal working and living conditions, parity, access to health care, and community response to infants are all factors affecting survivorship and health status. Socioeconomic status is considered to be the most influential in terms of regulating an individual's exposure and susceptibility to growth and health disruption (Saunders & Hoppa 1993; Farmer 1996; Schell 1997; Babones 2008; Cavigelli & Chaudhry 2012; DeWitte *et al.* 2016). In fact, socioeconomic status dictates a multitude of factors including nutrition, exposure to disease, and living and working environments (Kaplan 1954, 791; 797; Johnston *et al.* 1976, 469; Lewis 2007, 22). Thus, exposure to, and treatment for disease is also regulated by social status, gender and age, with access to adequate nutrition and healthcare controlled by these culturally contingent factors (Gowland 2004, 137). Consequently, an individual's susceptibility and immune response to disease was reliant on these factors (Gowland 2004, 137). The high levels of pathological lesions identified in the post-Medieval and Transition periods might be indicative of the socioeconomic status and social and cultural changes these individuals were experiencing. Social inequality is known to increase susceptibility to poor health and disease (Griffin *et al.* 2011, 533; Pitts & Griffin 2012, 254). Consequently, individuals from both of these samples are from known 'transitional periods' – those of the Roman invasion and the Industrial Revolution. Much work has considered the impact of wide scale social, cultural and economic changes on health and growth and found that these periods represent some of the most physiologically 'risky' periods. Increased prevalence rates of NBF in populations of low socioeconomic status and poor living conditions have been identified (Larsen 2015, 88).

Rates of congenital conditions are also known to increase in low socioeconomic and poor environmental conditions (Lewis 2017a, 35; Dudar 2010, 877). Although the 20th century population shows in general a reduction in pathological lesions from the post-Medieval sample, this may be expected due to the improvements in maternal, fetal/infant and obstetric care. However, the low status of these 20th century individuals (Hunt, *personal communication*) may still be reflected in the extremely high prevalence rates of congenital conditions, and may even be suggestive of socially and culturally restricted access to health care. However, given the suspected collection strategy employed in the curation of this sample, congenital defects maybe overrepresented as a result. This is because individuals suffering from these conditions may have been of greater selection bias.

Social and cultural practices and community response to infants can regulate exposure to growth and health stress. Infanthood and childhood have been recognised as social, rather than biological, constructions, with each culture and population able to define these liminal periods independently (Prout 2005; Wyness 2006). The behaviour towards and treatment of an infant once born can vary, and social status and culture can dictate the level of care, feeding and health the child has access to (Gowland 2004, 135; Redfern 2003, 151), having consequences for the health of these individuals.

Although this study refrains from considering the funerary context of these individuals with regards to burial goods and material culture, all of them were deliberately buried and placed within their graves. Although some individuals clearly represent those of lower socioeconomic individuals (e.g. Cross Bones and St. Bride's individuals) none appear to have been casually 'disposed of'. While none had been afforded extravagant burials, or even coffins in most cases, it must be remembered that many of their adult counterparts were afforded identical treatment in death. Furthermore, the recovery of young individuals from within settlements and dwellings is not uncommon and has been found in sites dating from the Neolithic to the Roman period (Lewis 2007, 31). Thus, the individuals analysed from Owslebury, Piddington and Barton Court Farm are not suggested to represent those deliberately or unwantedly disposed of. Instead they represent individuals buried in the normal tradition for such small individuals, and given the growth and health disruption identified within these samples (Hodson 2017), they display clear evidence of difficult early life experiences. There is no suggestion here that these infants were deliberately neglected or uncared for, despite the clear biological disturbances they experienced. Instead, growth and health disruption is far more likely to be a result of chronic, inescapable exposure to environmental stressors, to which mothers, parents and communities alike were unable to buffer these individuals.

The evidence of greater growth disruption in individuals aged over 40GWA, particularly in those age 52 and 58 GWA may indicate that social and cultural practices were also detrimental to growth and health. Although many infant care practices were untaken with the belief that they were beneficial, often this was unfounded and instead predisposed the child to greater growth and health disruption. Within the Roman, Medieval and post-Medieval

samples assessed swaddling is likely to have been widely practiced. This would have restricted the movement of the infant, but also restricted exposure to sunlight and contribute to vitamin D deficiency (Brickley & Ives 2008; Newman & Gowland 2017). With evidence of metaphyseal expansion and NBF evident metabolic disturbances are widely considered to be an important aetiological cause, particularly in post-medieval individuals.

Variable feeding practices may equally result in growth and health changes. Thus increased growth and health disruption may potentially also be reflective of this cultural practice. In Roman Britain it is known that infants were often sent to the countryside from cities (such as Londinium) as it was considered to be healthier (Scott 1999). Infants would be wet nursed and cared for, typically at villa sites (Scott 1999) – which may equally be a consequence of why we find many young individuals at these sites. Comparably, those of high status in post-Medieval London would be equally likely to employ a wet nurse, as society dictated, and thus immunological buffering was inherently limited in these individuals. This may be a cause of predominantly older individuals (>40GWA) occurring in samples at Chelsea Old Church. In fact, recent investigations by Newman and Gowland (2017) have suggested that growth changes were seen more severely in infancy in the high-status population of Chelsea as a result of fashionable social and cultural practices undertaken. However, dry-feeding became fashionable among the lower social classes (DeWitte *et al.* 2016). Once again, withholding of colostrum and maternal antibodies and nutrition, as a result of these feeding practices, may have resulted in these required resources for optimal postnatal survival being unattained. Consequently, postnatal survivorship, and health and growth, would have been disrupted and it is likely the severe growth and health disruption identified in the postnatal individuals assessed is partly a consequence of some of these practices.

10.4 Conclusions

Assessment of fetal, perinatal and infant skeletal and dental remains provides osteologists with the most intimate evidence of their lives in the past (Lewis 2007, 10). The early life course is commonly considered to be the most sensitive and fragile of the human life sequence (Roth 1992), with these youngest members of past societies a sensitive indicator of social, cultural and environmental parameters and experiences (Goodman & Armelagos 1989). Their immature immune system and rapid growth makes them vulnerable to adverse health and nutritional influences (Goodman and Armelagos 1989, 239; Perry 2006; Halcrow and Tayles 2008, 336). As poor health and nutrition in the prenatal period and/or early

childhood has been shown to have broader health implications in later life, such as compromised immunity, the study of childhood health provides a "sensitive barometer" for overall population health (Lewis 2007, 20).

Pre-industrial populations are typically considered to have significantly higher levels of mortality and morbidity than populations of today (Wrigley & Schofield 1989). This study has demonstrated that when careful assessment and analysis is undertaken, both health and growth disruption can be identified within infant skeletal remains. Although growth disruption is identifiable in all age groups and from all samples and time periods analysed, there are certainly variations in the patterns of growth disruption identified. Individuals from post-Medieval London show the greatest evidence of both growth and health disruptions, while the Pre-Roman individuals show the least evidence of both. This demonstrates that there is a strong temporal relationship between growth and health, pertaining to the varying social, cultural and environmental stressors experienced within these periods. In particular, social transitions and social stratification appear to have had severe impacts on growth and health.

There are a multitude of ways in which socioeconomic status can affect the physiological parameters of growth and health; it has been traditionally considered that low socioeconomic status predisposes individuals to poorer nutrition, living and working environments and thus a higher disease and pathogen load. Consequently, metabolic, infectious and congenital disease processes are all much more likely. Post-Medieval individuals show clear differences between status groups; those from Cross Bones, the lowest status sample assessed, were identified as having the most severe growth and health disruption, whilst those from Chelsea Old Church, the highest status population had the least. However, nuances in interpretation must not be overlooked and although this general relationship between status and disruption is evident, cultural and social practices of maternal and perinatal/infant care still had implications for growth and health of these individuals.

A myriad of varying extrinsic factors have been considered as to the etiological and contextual causation of growth and health disruption identified. Given the variability in the age of the individuals affected, the extent and severity of changes, and the type and location of pathological lesions, it is likely that there are a multitude of factors which affected health and growth.

Furthermore, evidence from these individuals has found that health and growth disruption can be identified both pre- and postnatally. As a consequence, the theoretical implications of the DOHAD hypothesis should also be considered. Although epigenetic changes have not been directly investigated, predisposition to poor health as a result of multi-generational exposure to detrimental and limiting conditions may have resulted in some of these individuals being of increased susceptibility to poor health.

Of course, as discussed throughout, there are many limitations of this study and although attempts to overcome these in the best way possible have been made, individual variation in growth and disease response is always a confounding factor. However, by employing a robust methodology and strategy for investigating health and growth disruption, this study supports the wealth of other literature, with findings highlighting that these individuals are indicative of the most fragile and precarious lives of people in the past.

Ultimately this study has initiated an avenue of study which must continue to be developed and explored, highlighting the essential need to evaluate both dental and skeletal development to synthesise age-at-death data and investigate evidence of growth disruption. By considering multiple populations and attempting to compare and contrast growth disruption over time, this study has been able to evaluate diachronic trends, implicating the significance of socioeconomic status and environmental stressors as key factors for regulating fetal, perinatal and infant growth and health. Today, growth and health disruption is an ongoing clinical concern and thus understanding the ways in which the skeleton responds to stress and insults is imperative. Consequently, holistic consideration of both growth and health disruption supports the suggestion that many of these individuals were ‘stressed’ at birth.

Chapter 11: Conclusions and Recommendations

11.1 Conclusions

The primary focus of this thesis, and manuscripts herein, has been the identification and consideration of health and growth disruption in fetal, perinatal and infant individuals from archaeological and historical populations. Current clinical and anthropological studies are revealing the multitude of ways that early life can be disrupted, detailing both the mechanisms behind, and physiological responses to, these onslaughts (e.g. Barker 1997; 2012; Barker *et al.* 1990; 2002; Abu-Saad & Fraser 2010; Holdsworth & Schell 2017; Hujoel *et al.* 2017). This growing discourse has been pivotal in identifying these young individuals as central to interpretations of overall community and population health (Goodman & Armelagos 1989, 239; Redfern 2003, 162; Baxter 2005, 99; Lewis 2007, 20). Only within the last decade have fetal, perinatal and infant individuals received comprehensive attention in the bioarchaeological literature (Halcrow & Tayles 2008, 191; Mays *et al.* 2017, 38). The failure of archaeologists and bioarchaeologists to acknowledge the potential of these remains for yielding important insights into past health, has resulted in the most perilous stage of life being systematically absent from many bioarchaeological interpretations. Thus, if we are to understand the health and wellbeing of an archaeological sample we must not overlook the most fragile members of these societies. Adopting a holistic approach, considering the vast multidisciplinary discourse on health and growth disruption, this study has been able to consider and contextualise results of assessment to develop a comprehensive narrative on the causes, manifestation and implications of physiological disruption. Therefore, this thesis makes an important contribution to the study of fetal, perinatal and infant remains within bioarchaeology.

This study comprises of a uniquely large sample size of fetal, perinatal and infant skeletal remains. In addition, inclusion and assessment of a documented collection has enabled specific investigation as to the effects of sex and ethnicity on growth and health disruption. Thus, the samples analysed are perfectly suited to the aims of the project and have enabled extensive consideration of growth and health disruption in these young individuals over a lengthy chronological time period.

Within this thesis, the novel approach adopted, comparing age estimations established from assessment of dental development against ages calculated from diaphyseal length of the long bones has proved highly effective in determining growth disruption. It is suggested that such an approach should be commonly employed, where possible, for all future studies of these young individuals. Consequently, this thesis has demonstrated that dental development, and the corresponding age-at-death estimates it generates, is a reliable method whereby proxies of chronological age can be generated. Consideration of a known age-at-death population has helped in establishing this claim. Furthermore, assessment of the *pars basilaris*, a bone considered to be robust against environmental stress, has found a strong parity between dental age and age estimates derived from metric assessment of this cranial element. This research has proven that establishing evidence of growth disruption, by considering skeletal age in comparison to dental age, is a robust methodology. Furthermore, the inclusion of error ranges, as outlined by the age estimation methodologies employed, results in growth disruption only being recorded for individuals where significant differences are found between the age estimates.

Evidence of systemic growth disruption, where multiple skeletal elements were found to be shorter, and generate younger age estimates, than dental age, has thus been identified in all of the samples/time periods considered for analysis. Although only 39 individuals out of the 423 assessed showed this evidence of growth disruption, where all long bone elements had age estimates which failed to correlate or overlap with dental age, many additional individuals were found to have disruption within particular long bone elements (See Chapter 10 and Table 10.1). Overall, considering all individuals with evidence of growth disruption (N = 170) to at least one skeletal element, the post-Medieval period was found to have the highest prevalence of growth disruption. Prevalence rates of growth disruption were not found to be similar between time periods/samples. Instead growth disruption appears to be intrinsically linked to the socioeconomic, cultural and environmental constraints acting upon individuals within a sample.

This thesis has also attempted to the explore evidence of pathological lesions within these individuals, although it is acknowledged that this is a challenging area of research which requires further investigation. Evidence of pathological lesions was found in individuals from all samples and time periods analysed. Cranial lesions were the most commonly identified pathology throughout all samples and time periods (TPR 70%), although prevalence rates

were shown to vary substantially between these categories. Postcranial lesions were consistently less prevalent in all samples (TPR 30%), although again variation in prevalence was identified between time periods. New bone formation was the type of lesion most frequently identified in all individuals. Morphological change and metaphyseal expansion were not regularly identified, except for those in the infant age category. Similarities between samples in type and location of lesion suggests a comparable physiological response between individuals, regardless of the particular heath stress experienced. Yet, disparity in prevalence rates between samples indicates a variable exposure to stress over time – those of post-Medieval and Transition samples showing exceptionally high pathological prevalence rates compared to those of the pre-Roman sample. Inherent variation in the social, cultural and environmental experiences of individuals within these samples has once again been suggested to be a contributing factor to this variation.

Growth and health disruption are both likely to be a result of an amalgamation of detrimental insults. Nutritional deficiency, specific and non-specific infections, as well as congenital conditions, endocrine disorders and psychosocial deprivation (Haymond *et al.* 2013, 787) have all been implicated in growth and health disruption. Consideration of the pathological lesions identified within individuals assessed, typically NBF, in correlation with growth disruption, is argued to indicate that metabolic disturbance was the most likely etiology for many of the physiological disruptions identified. That it not to say that multiple pathogenic and etiological impetuses were not experienced – many disease processes are found to co-occur – but simply that specific disease processes were concealed by this non-specific response. Furthermore, specific diseases often manifest as nutritional deficiency within fetal, perinatal and infant individuals due to their reliance on maternal care, and nutritional and immunological safeguarding. Extracting specific pathogenic, etiological and contextual origins for growth and health disruption is thus challenging when skeletal responses to such insults are often non-specific.

This thesis has attempted to establish unequivocal evidence of growth and health disruption, as widespread methodological limitations and reservations often result in limited assessment of fetal, perinatal and infant individuals. By examining physiological disruptions in a large sample of temporally disparate individuals, this study has attempted to demonstrate that early life disruption can be quantified throughout the archaeological record. Yet, despite this focus on the physical parameters of skeletal remains, comprehensive consideration of social,

cultural and environmental variables has demonstrated that a myriad of factors can influence and affect the early life course. Social and cultural practice, maternal buffering and regulation of the environment are central to overall growth and health, whereby these variables can themselves often be either intrinsically or extrinsically regulated (Goodman & Armelagos 1988, 941-942; Goodman *et al.* 1988, 169-170; Bush & Zvelebil 1991, 5). Consequently, it is argued that understanding the implications of growth and health disruption is complex, particularly as physiological responses are typically non-specific with regards to pathological lesions (Goodman *et al.* 1984, 259; 1988, 178; Bush & Zvelebil 1991, 5; Lewis & Roberts 1997, 584; Temple & Goodman 2014, 186). Given this skeletal mechanism, identifying specific and exact causes of stress and insult is implausible within archaeological material, especially when only macroscopic assessment is undertaken.

Indeed, interpreting health and growth disruption by considering physiological skeletal responses is itself fraught with debate. After all, it might not be so unexpected that growth and health disruption is prevalent when it is the non-survivors of past populations being considered (Krenz-Niedbala & Lukasik 2017, 375). With infant mortality considered to be exceptionally high within past populations – estimates range from 30-50% (e.g. Schultz 2001, 129; Gowland 2001, 155; Chamberlain 1997, 249; Wiedemann 1989, 12-16) - it is anticipated that young individuals frequently succumbed to the detrimental environments and stressors of intra- and extrauterine life. Identification of clear growth changes and pathological lesions, indicative of a reduced health status, are consequently not unexpected, and conversely may only represent a portion of the actual number of individuals experiencing stress. Rapidity of skeletal response within fetal, perinatal and infant individuals means health disruption is often reflected on their skeletal remains (Ortner 2003, 206; Lewis 2007, 60; Schultz 2001, 115; Kini *et al.* 2012, 30) – more so than on those of adults – yet acute or particularly severe insults, where death was almost instantaneous, do not always have time to manifest within the skeleton. It can be argued that the prevalence rates and percentages of those showing growth and health disruption presented within this study ultimately reflect lower estimates of disruption than what may have truly been experienced.

Research objectives outlined within the introduction of this thesis have been thoroughly achieved. As a result, four major findings from this research, which will advance analyses of fetal, perinatal and infant remains, have been established:

1. The *pars basilaris* is a useful proxy for dental ageing, which in turn is a good proxy for chronological age.
2. Diaphyseal lengths of long bones, which are commonly used to age perinates, will skew chronological age estimations to the centre as they are based on regression equations. This means there is a mimicry between reference and sample populations, with results simply reflecting the standard from which they derive. The use of dental age estimation methodologies, instead of these skeletal ones, protects against this bias. Therefore, diaphyseal lengths should no longer be used to estimate age-at-death.
3. The tibia has been demonstrated to be the most sensitive to a range of environmental stressors, showing the greatest evidence of growth and postcranial health disruption. This finding supports previous results, though extends the evidence into the fetal, perinatal and infantile period.
4. Error levels are central to interpretations of both chronological age and growth disruption, with growth disruption only identified in individuals where skeletal and dental age estimates showed no correlation or overlap. Thus, it is vital that error ranges provided by each of the methods are considered to ensure assessment of both age and evidence of growth disruption is robust.

This thesis has demonstrated that growth and health disruption can be identified within these youngest members of past societies and provide valuable insights into early life experiences. Confining fetal, perinatal and infant individuals to grey literature is no longer an option, as sensitive indicators of samples/populations these individuals provide unique opportunities to study these most fragile and precarious lives. Determining growth and health profiles of the samples and individuals assessed, this study has revealed a fluctuating pattern of physiological responses within and between past populations. Consideration of individual experience is imperative - based on age, environment, predisposition to disruption and insults, and time period - revealing the intrinsically variable, yet broadly comparable way the early life course represents the most precarious period of human life. Extensive evidence of physiological disruption throughout the samples assessed reveals a narrative of individuals inherently ‘stressed’ at birth.

11.2 Future Directions and Recommendations

To support and expand findings presented in this study six central future considerations have been outlined.

1. Increased studies and samples sizes of fetal, perinatal and infant individuals recording dental and skeletal development to construct age-at-death profiles.
    a. Further data (skeletal, dental and pathological) to consider known age-at-death individuals. Increasing sample size of known age individuals would further comprehension of growth disruption within archaeological samples.
    b. Increased sample sizes would also enable further statistical assessment to consider age-dependent differences between chronological and skeletal and dental ages.

2. Investigating varying aspects of skeletal growth metrically to determine skeletal elements most commonly disrupted in growth.
    a. Given that the tibia is typically considered to be the most variable and responsive to stress, future studies should investigate other distal segments of long bones (radius, ulna and fibula) to explore intra-limb growth prioritisation and variation.
    b. Consideration of allometry and asymmetrical growth would enable further indication of stress. In particular, clinical studies indicate that SGA offspring have shorter radii and ulnae in comparison to humeri (Brooke et al 1984). Furthermore, asymmetric growth is considered to be indicative of IUGR. Consequently, by determining disproportional growth within and between skeletal elements interpretations surrounding both stress exposure and birth experiences would be aided.
    c. Intra-element growth disruption should be considered; e.g. comparisons between diaphyseal lengths and metaphyseal widths of femora and humeri.
    d. Diaphyseal mid-point width should also be investigated as growth and health disruption is known to prioritise interstitial, rather than appositional growth.

3. Radiographic investigation of fetal, perinatal and infant long bones.
    a. Radiograph long bones with suspected pathological NBF to determine thickness of periosteal NBF in order confirm presence of pathological changes.
    b. Measure total cortical thickness to investigate appositional growth changes. Disruption to growth is not only considered to result in small diaphyseal

diameters, but reduction in cortical thickness also. Investigation would support metric investigation of diaphyseal mid-point width.

4. Isotopic incremental dentine analysis to determine ratios of nitrogen isotopes within samples assessed.
    a. Investigate whether nitrogen isotope ratios are increased in individuals showing more evidence of pathological lesions/severer growth disruption.
    b. Determine nitrogen isotope profiles for individuals, considering peak values and whether these were still rising or dropping at point of death.
    c. Consider whether rising or falling nitrogen isotpe ratios correlate with pathological lesions showing either an initial response (woven bone) or a healing response (lamellar bone formation).

5. CT scanning of fetal, perinatal and infant long bones to assess evidence of bioerosion.
    a. Investigate whether age-dependent differences in bioerosion can be identified, substantiating correlation between age and survival.

6. Histological assessment of the neonatal line.
    a. By determining the presence of the neonatal line, comparison of dental and skeletal growth and health disruption can aid interpretations of prematurity, SGA or IUGR.

The above future directions have been considered as interesting prospective avenues to explore to enhance consideration of fetal, perinatal and infant growth and health disruption.

References

Abu-Saad, K. and Fraser, D. 2010; Maternal Nutrition and Birth Outcomes. *Epidemiological Reviews,* Vol. 32: 5-25.

Acheson, R. M. 1959; Effects of starvation, septicaemias and chronic illness on the growth cartilage plate and metaphysis of the immature rat. *Journal of Anatomy,* Vol. 93: 123-130.

Adair L. 2004; Fetal adaptations to maternal nutritional status during pregnancy. *American Journal of Physical Anthropology,* Vol. 123, Suppl. 38: 50.

Adams, S. M., Ward, C. E. and Garcia, K. L. 2015; Sudden Infant Death Syndrome. *American Family Physician,* Vol. 91 (11): 778-783.

Agarwal, S. C. 2016; Bone Morphologies and Histories: Life Course Approaches in Bioarchaeology. *Yearbook of Physical Anthropology,* Vol. 159: 130-149.

Aiello, L. C. 2000; Foreword: The Development of Juvenile Osteology. In L. Scheuer and S. Black *Developmental Juvenile Osteology*. London: Academic Press.

Aiello, L. C. and Wells, J. C. K. 2002; Energetics and the evolution of the genus Homo. *Annual Review of Anthropology*, Vol. 31: 323–338.

Aleman, I., Irurita, J., Valencia, A. R., Martínez, A., López-Lázaro, S., Viciano, J. and Botella, M. C. 2012; Brief Communication: The Granada Osteological Collection of Identified Infants and Young Children. *American Journal of Physical Anthropology,* Vol. 149: 606-610.

Alfonso-Durruty, M. P. 2011; Experimental assessment of nutrition and bone growth's velocity effects on Harris lines formation. *American Journal of Physical Anthropology,* Vol. 145: 169-180.

AlQahtani, S. J. Hector, M. P. and Liversidge, H. M. 2010; The London Atlas of Human Tooth Development and Eruption. *American Journal of Physical Anthropology,* Vol. 142: 481-490

AlQahtani, S. J. Hector, M. P. and Liversidge, H. M. 2014; Accuracy of Dental Age Estimation Charts: Schour and Massler, Ubelaker, and the London Atlas. *American Journal of Physical Anthropology,* Vol. 154: 70-78.

Altman, D. G. and Chitty, L. S. 1997; New charts for ultrasound dating of pregnancy. *Ultrasound in Obstetrics and Gynecology,* Vol. 10: 174-191.

Anatoliotaki, M., Tsilimigaki, A., Tsekoura, T., Schinaki, A., Stefanaki, S. and Nikolaidou, P. 2003; Congenital rickets due to maternal vitamin D deficiency in a sunny island of Greece. *Acta Paediatrica,* Vol. 92: 389-391.

Anderson, T. and Carter, A. R. 1994; Periosteal reaction in a newborn child from Sheppey, Kent. *International Journal of Osteoarchaeology,* Vol. 4:47-48.

Anderson, T. and Carter, A. R. 1995; An unusual osteitic reaction in a young medieval child. *International Journal of Osteoarchaeology,* Vol. 5:192–195.

Anderson, B. L. and Gonik, B. 2011; Perinatal Infections. In R. J. Martin, A. A. Fanaroff and M. C. Walsh (Eds.) *Fanaroff and Martin's neonatal-perinatal medicine: diseases of the fetus and infant.* Missouri: Elsevier Mosby: 399-422.

Angel, J. L. 1969; The basis of paleodemography. *American Journal of Physical Anthropology,* Vol. 30: 427–438.

Angel, J. L. 1981; History and Development of Paleopathology. *American Journal of Physical Anthropology,* Vol. 56: 509-515.

Ariès, P. 1962; *Centuries of Childhood: A Social History of Family Life*. London: Cape.

Armelagos, G. J. and Goodman, A. H. 1991; The concept of stress and its relevance to studies of adaptation in prehistoric populations. *Collegium Antropologicum*, Vol. 15: 45-58.

Armelagos, G. J. and Van Gerven, D. P. 2003; A Century of Skeletal Biology and Paleopathology: Contrasts, Contradictions, and Conflicts. *American Anthropologist,* Vol. 105 (1): 53-64.

Armelagos, G. J., Carlson, D. and Van Gerven, D. 1982; The theoretical foundations and development of skeletal biology. In: F. Spencer (Ed.) *A History of American Physical Anthropology 1930-1980, Volume 2.* New York: Academic Press: 305-329.

Armelagos, G. J., Goodman, A. H., Harper, K. N. and Blakey, M. L. 2009; Enamel Hypoplasia and Early Mortality: Bioarchaeological Support for the Barker Hypothesis. *Evolutionary Anthropology*, Vol. 18: 261-271.

Atkinson, R. J. C. 1952; Excavations in Barrow Hills Field, Radley, Berkshire, 1944-45. *Oxoniensia,* Vol. 17: 14-35.

Aubin, J. E. and Heersche, J. N. M. 2003; Bone Cell Biology: Osteoblasts, Osteocytes, and Osteoclasts. In F. H. Glorieux, J. M. Pettifor and H. Jüppner (Eds.) *Pediatric Bone: Biology and Diseases.* London: Academic Press: 43-75.

Audy, J. R. 1971; Measurement and diagnosis of health. In P. Shepard and D. McKinley (Eds.) *Environmental: Essays on the planet as a home.* Boston: Houghton Mifflin: 140-162.

Aufderheide, A. C. and Rodriguez-Martin, C. 2008; *The Cambridge Encyclopedia of Human Palaeopathology.* Cambridge: Cambridge University Press.

Azcorra, H., Dickinson, F. and Banik, S. D. 2016; Maternal height and its relationship to offspring birth weight and adiposity in 6 to 10-year old Maya children from poor neighbourhoods in Merida, Yucatan. *American Journal of Physical Anthropology,* Vol. 161: 571-579.

BABAO Ethics and Standards [Online] [Accessed August 2017] Available from: http://www.babao.org.uk/publications/ethics-and-standards/

BABAO Code of Practice [Online] [Accessed August 2017] Available from: http://www.babao.org.uk/assets/Uploads/code-of-practice.pdf

Babones, S. J. 2008; Income inequality and population health: Correlation and causality. *Social Science and Medicine,* Vol. 66: 1614-1626.

Baker, B., Dupras, T. and Tocheris, M. 2005; *The Osteology of Infants and Children*. College Station, TX: Texas A&M University Press.

Baker, M. 1997; Invisibility as a symptom of gender categories in archaeology. In J. Moore and E. Scott (Eds.) *Invisible People and Processes.* London: Leicester University Press: 183-191.

Bang, G. 1989; Age changes in teeth; developmental and regressive. *Age Markers in the Human Skeleton*, Vol. 1: 211-235.

Barilan, M. Y. 2006; Bodyworld and the ethics of using human remains: A preliminary Discussion. *Bioethics,* Vol. 20 (5): 233-247.

Barker, D. J. P. 1994; *Mothers, Babies, and Disease in Later Life*. London: BMJ Publishing Group.

Barker, D. J. P. 1997; Maternal Nutrition, Fetal Nutrition, and Disease in Later Life. *Nutrition,* Vol. 13 (9): 807-813.

Barker, D. J. P. 2003; *The Best Start in Life: How a Woman's Diet Can Protect her Child from Disease in Later Life*. London: Century Books.

Barker, D. J. P. 2012; Developmental origins of chronic disease. *Public Health,* Vol. 126: 185-189.

Barker, D. J. P., Bull, A. R., Osmond, C., and Simmonds, S. J. 1990; Fetal and placental size and risk of hypertension in adult life. *British Medical Journal*, Vol. 301: 259–262.

Barker, D. J. P., Eriksson, J. G., Forsén, T. and Osmond, C. 2002; Fetal origins of adult disease: strength of effects and biological basis. *International Journal of Epidemiology,* Vol. 31: 1235-1239.

Barker, D. J. P, Godfrey, K. M., Fall, C., Osmond, C., Winter. P. D., and Shaheen, S. O. 1991; Relation of birth weight and childhood respiratory infection to adult lung function and death from chronic obstructive airways disease. *British Medical Journal*, Vol. 303: 671–675.

Barker, D. J. P., Lampl, M., Roseboom, T. and Winder, N. 2012; Resource allocation in utero and health in later life. *Placenta,* Vol. 33: 30-34.

Barker, D. and Osmond, C. 1986; Infant mortality, childhood nutrition, and ischaemic heart disease in England and Wales. *Lancet,* Vol. 8489: 1077–1081.

Barker, D. J., Osmond, C., Forsen, T. J., Kajantie, E., and Eriksson, J. G. 2005; Trajectories of growth among children who have coronary events as adults. *The New England Journal of Medicine*, Vol 353 (17): 1802–1809.

Barker, D. J. P., Osmond, C. and Law, C. M. 1989; The intrauterine and early postnatal origins of cardiovascular disease and chronic bronchitis. *Journal of Epidemiological Community Health*, Vol. 43: 237–240.

Bateson, P., Barker, D., Clutton-Brock, T., Deb, D., D'Udine, B., Foley, R. A., Gluckman, P., Godfrey, K., Kirkwood, T., Mirazón Lahr, M., McNamara, J., Metcalfe, N. B., Monaghan, P., Spencer, H. G. and Sultan, S. E. 2004; Developmental plasticity and human health. *Nature,* Vol. 430: 419-421.

Baxter, J. E. 2005; *The Archaeology of Childhood: Children, Gender, and Material Culture.* California: AltaMira Press.

Beaudrap, P., Turyakira, E., White, L. J., Nabasumba, C., Tumwebaze, B., Muehlenbachs, A., Guérin, P., Boum, Y., McGready, R. and Piola, P. 2013; Impact of malaria during pregnancy on pregnancy outcomes in a Ugandan prospective cohort with intensive malaria screening and prompt treatment. *Malaria Journal,* Vol. 12 (1): 139.

Beaumont, J., Geber, J., Powers, N., Wilson, A., Lee-Thorp, J. and Montgomery, J. 2013; Victims and survivors: Stable isotopes used to identify migrants from the Great Irish Famine to 19th century London. *American Journal of Physical Anthropology,* Vol. 150: 87-98.

Beaumont, J., Montgomery, J, Buckberry, J and Jay, M. 2015; Infant Mortality and Isotopic Complexity: New Approaches to Stress, Maternal Health and Wellbeing. *American Journal of Physical Anthropology,* Vol. 157: 441-457.

Beaumont, J. and Montgomery, J. 2016; The Great Irish Famine: Identifying Starvation in the Tissues of Victims Using Stable Isotope Analysis of Bone and Incremental Dentine Collagen. *PLoS ONE,* Vol. 11 (8).

Beaumont, J., Montgomery, J., Buckberry, J. and Jay, M. 2015; Infant Mortality and Isotopic Complexity: New Approaches to Stress, Maternal Health and Wellbeing. *American Journal of Physical Anthropology,* Vol. 157: 441-457.

Beck, L. A. 2006; Kidder, Hooton, Pecos, and the Birth of Bioarchaeology. In J.E. Buikstra and L. A. Beck (Eds.) *Bioarchaeology: The Contextual Analysis of Human Remains*. Amsterdam, Boston: Academic Press: 83-94.

Becker, M. J. 2006; The Archaeology of Infancy and Childhood; Integrating and Expanding Research into the Past. *American Journal of Archaeology,* Vol. 10: 655-658.

Beier, A. L. 1978; Social Problems in Elizabethan London. *The Journal of Interdisciplinary History,* Vol. 9 (2): 203-221.

Bekvalac, J. 2007a *St Mary Graces* [Online] [Accessed: November 2016] Available from: https://www.museumoflondon.org.uk/collections/other-collection-databases-and-

libraries/centre-human-bioarchaeology/osteological-database/medieval-cemeteries/st-mary-graces

Bekvalac 2007b *St Thomas' Hospital cemetery summary* [Online] [Accessed: November 2016] Available from: https://www.museumoflondon.org.uk/collections/other-collection-databases-and-libraries/centre-human-bioarchaeology/osteological-database/post-medieval-cemeteries/st-thomas-hospital-post-medieval

Benyshek, D. 2007; The developmental origins of obesity and related health disorders–prenatal and perinatal factors. *Collegium Antropologicum*, Vol. 31 (1): 11–17.

Beukema, R., Raiche, M. and Turok, D. 2008; Complications of Pregnancy. In S. D. Ratcliffe, E. G. Baxley, M. K. Cline and E. L. Sakornbut (Eds.) *Family Medicine Obstetrics*. Philadelphia: Elsevier: 151-201.

Besbes, L. G., Hadded, S., Meriem, C. B., Golli, M., Najjar, M. F. and Guediche, M. N. 2010; Infantile Scurvy: Two Case Reports. *International Journal of Pediatrics*, Article ID 717518: 1-4.

Black, T. K. 1978; Sexual Dimorphism in the Tooth-Crown Diameters of the Deciduous Teeth. *American Journal of Physical Anthropology*, Vol. 48: 77-82.

Blackwell, A. D., Urlacher, S. S., Beheim, B., von Rueden, C., Jaeggi, A., Stieglitz, J., Trumble, B. C., Gurven, M. and Kaplan, H. 2017; Growth references for Tsimane forager-horticulturalists of the Bolivian Amazon. *American Journal of Physical Anthropology*, Vol. 162: 441–461.

Blakey, M. L. and Armelagos, G. J. 1985; Deciduous Enamel Defects in Prehistoric American From Dickson Mounds: Prenatal and Postnatal Stress. *American Journal of Physical Anthropology*, Vol. 66: 371-380.

Boas, F. 1912; Changes in bodily form of descendants of immigrants. *American Anthropologist*, Vol. 14: 530–62.

Bocquet-Appel, J. P. and Masset, C. 1982; Farewell to palaeodemography. *Journal of Human Evolution,* Vol. 11: 321-333.

Boersma, G. J. and Tamashiro, K. L. 2015; Individual differences in the effects of prenatal stress exposure in rodents. *Neurobiology of Stress,* Vol. 1: 100-108.

Bogin, B. 1990; The Evolution of Human Childhood. *BioScience,* Vol. 40 (1): 16-25.

Bogin, B. 1997; Evolutionary Hypotheses for Human Childhood. *Yearbook of Physical Anthropology,* Vol. 40: 63-89.

Bogin, B. 1998; Evolutionary and biological aspects of childhood. In C. Panter-Brick (Ed.) *Biosocial Perspectives on Children*. Cambridge: Cambridge University Press: 10–44.

Bogin, B. 1999; *Patterns of Human Growth* (2nd Edition). Cambridge: Cambridge University Press.

Bogin, B. 2001; *The Growth of Humanity*. New York: Wiley-Liss.

Bogin, B. 2002; *The Evolution of Human Growth.* In N. Cameron & B. Bogin (Eds.) *Human Growth and Development.* London: Elsevier: 287-324.

Bogin, B. 2012; *The Evolution of Human Growth.* In N. Cameron & B. Bogin (Eds.) *Human Growth and Development.* London: Elsevier: 287-324.

Bogin, B. & Baker, J. 2012; Low Birth Weight Does Not Predict the Ontogeny of Relative Leg Length of Infants and Children: An Allometric Analysis of the NHANES III Sample. *American Journal of Physical Anthropology,* Vol. 148: 487-494.

Bogin, B. & Loucky, J. 1997; Plasticity, Political Economy, and Physical Growth Status of Guatemala Children Living in the United States. *American Journal of Physical Anthropology,* Vol.102: 17-32.

Bogin, B. & Rios, L. 2003; Rapid morphological change in living humans; implications for modern human origins. *Comparative Biochemistry and Physiology*, Part A, Vol. 136: 71-84.

Bogin, B., Varela-Silva, M. I. and Rios, L. 2007; Life history trade-offs in human growth: adaptation or pathology? *American Journal of Human Biology,* Vol. 19: 631–42.

Bolaños, M. V., Manrique, M. C., Bolaños, M. J. and Briones, M. T. 2000; Approaches to chronological age assessment based on dental calcification. *Forensic Science International,* Vol. 110: 97-106.

Bonneau, N., Simonis, C., Seringe, R. and Tardieu, C. 2011; Study of Femoral Torsion During Prenatal Growth: Interpretations Associated with the Effects of Intrauterine Pressure. *American Journal of Physical Anthropology,* Vol. 145: 438-445.

Bonsall, L. 2013; Infanticide in Roman Britain: A Critical Review of the Osteological Evidence. *Childhood in the Past,* Vol. 6 (2): 73-88.

Booth, T. J. 2016; An investigation into the relationship between bacterial bioerosion and funerary treatment in European archaeological human bone. *Archaeometry*, Vol. 58 (3): 484-499.

Booth, T. J., Redfern, R. C., Gowland, R. L. 2016; Immaculate conceptions: Micro-CT analysis of diagenesis in Romano-British infant skeletons. *Journal of Archaeological Science*, Vol. 74: 124-134.

Boucher, B. 1955; Sex differences in the foetal sciatic notch. *Journal of Forensic Medicine,* Vol. 2: 51–54.

Boucher, B. 1957; Sex differences in the foetal pelvis. *American Journal of Physical Anthropology,* Vol. 15: 581–600.

Boulton, J. 2000; 'It Is Extreme Necessity That Makes Me Do This': Some 'Survival Strategies' of Pauper Households in London's West End During the Early Eighteenth Century. *International Review of Social history,* Vol. 45: 47-69.

Brickley, M. and Ives, R. 2006; Skeletal Manifestations of Infantile Scurvy. *American Journal of Physical Anthropology*¸ Vol. 29: 163-172.

Brickley, M. and Ives, R. 2008; *The Bioarchaeology of Metabolic Bone Disease*. Amsterdam/London: Elsevier/Academic Press.

Brickley, M. and McKinley, J. I. (Eds.) 2004; *Guidelines to the Standards for Recording Human Remains*. IFA Paper No. 7. Southampton and Reading: BABAO and IFA.

Brickley, M. Miles, A. and Stainer, H. 1999; *The Cross Bones Burial Ground, Redcross Way, Southwark, London: Archaeological Excavations (1991-1998) for the London Underground Limited Jubilee Line Extension Project* (MOLAS Monograph 3). London: Museum of London Archaeology Service.

Brothwell, D. 1971; Palaeodemography. In W. Brass (Ed.) *Biological Aspects of Demography*. London: Taylor & Francis: 111–128.

Buckberry, J. 2000; Missing, Presumed Buried? Bone Diagenesis and the Under-Representation of Anglo-Saxon Children. *Assemblage,* Vol. 5: 1-17.

Buckberry, J. 2005; Where have all the children gone? The preservation of infant and children's remains in the archaeological record. Paper presented at the *Archaeology of Infancy and Childhood Conference*, 6–8 May 2005, University of Kent, UK.

Buikstra, J. E. 2006; A historical introduction. In J.E. Buikstra and L. A. Beck (Eds.) *Bioarchaeology: The Contextual Analysis of Human Remains*. Amsterdam, Boston: Academic Press: 7-26.

Buikstra, J. E. and Beck, L. A. 2006*; Bioarchaeology: The Contextual Analysis of Human Remains*. London: Elsevier Inc.

Buikstra, J. E. and Cook, D. C. 1980; Palaeopathology: An American Account. *American Review of Anthropology*, Vol. 9: 433-470.

Buikstra, J. E., Konigsberg, L. and Bullington, J. 1986; Fertility and the development of agriculture in the prehistoric Midwest. *American Antiquity*, Vol. 51: 191-204.

Buikstra, J. E. and Roberts, C. 2012; *The Global History of Paleopathology: Pioneers and Prospects.* Oxford: Oxford University Press.

Burch, M. Treveil, P. and Keene, D. 2011; *The development of early medieval and later Poultry and Cheapside: Excavations at 1 Poultry and vicinity, City of London* (MOLA Monograph 38). London: Museum of London Archaeology.

Bush, H. 1991; Concepts of Health and Stress. In H. Bush and M. Zvelebil (Eds.) *Health in Past Societies: Biocultural interpretations of human skeletal remains in archaeological contexts* (BAR International Series 567). Oxford: BAR Publishing: 11- 21.

Bush, H. and Zvelebil, M. 1991; Pathology and health in past societies: an introduction. In H. Bush and M. Zvelebil (Eds.) *Health in Past Societies: Biocultural interpretations of human skeletal remains in archaeological contexts* (BAR International Series 567). Oxford: BAR Publishing: 3-9.

Buzon, M. R. 2012; The bioarchaeological approach to palaeopathology. In A.L. Grauer (Ed.) *A Companion to Paleopathology.* Chichester; Malden, MA: Wiley-Blackwell: 58-75.

Byers, S. 1991; Technical note: calculation of age at formation of radiopaque transverse lines. *American Journal of Physical Anthropology*, Vol. 85: 339-343.

Cameron, N. 2002; The Human Growth Curve, Canalization and Catch-Up Growth. In N. Cameron and B. Bogin (Eds.) *Human Growth and Development.* London: Elsevier: 1-22.

Cameron, N. 2012; The Human Growth Curve, Canalization and Catch-Up Growth. In N. Cameron & B. Bogin (Eds.) *Human Growth and Development.* London: Elsevier: 1-22.

Cameron, N. and Demerath, E. W. 2002; Critical Periods in Human Growth and Their Relationship to Diseases of Aging. *Yearbook of Physical Anthropology,* Vol. 45: 159-184.

Cardoso, H. F. V. 2007; Environmental Effects on Skeletal Versus Dental Development: Using a Documented Subadult Skeletal Sample to Test a Basic Assumption in Human Osteological Research. *American Journal of Physical Anthropology,* Vol. 132: 223-233.

Cardoso, H. F. V., Abrantes, J., and Humphrey, L. T. 2014; Age estimation of immature human skeletal remains from the diaphyseal length of the long bones in the postnatal period. *International Journal of Legal Medicine,* Vol. 128 (5): 809-824.

Cardoso, H. F. V. and Saunders, S. R. 2008; Two Arch Criteria of the Ilium for Sex Determination of Immature Skeletal Remains: A Test of Their Accuracy and An Assessment of Intra- and Inter-observer Error. *Forensic Science International,* Vol. 178 (10): 24-29.

Carroll, M. 2011; Infant death and burial in Roman Italy. *Journal of Roman Archaeology,* Vol. 24: 99-120.

Carroll, M. 2012; 'No part in earthly things'. The Death, Burial and Commemoration of Newborn Children and Infants in Roman Italy. In M. Harlow and L. Larsson Lovén (Eds.) *Families in the Roman and Late Antique World.* London: Continuum International Publishing Group: 41-63.

Carvel D. 2002; Controversies concerning human tissue retention and implications for the forensic practitioner. *Journal of Clinical Forensic Medicine,* Vol. 9:53–60.

Cattaneo, C. 1991; Direct genetic and immunological information in the reconstruction of health and biocultural conditions of past populations: a new prospect for archaeology. In H. Bush and M. Zvelebil (Eds.) *Health in Past Societies: Biocultural interpretations of human skeletal remains in archaeological contexts* (BAR International Series 567). Oxford: BAR Publishing: 39-52.

Cavigelli, S. A. and Chaudhry, H. S. 2012; Social status, glucocorticoids, immune function, and health: can animal studies help us understand human socioeconomic-status-related health disparities? *Hormones and Behavior*, Vol. 62: 295–313.

Chamberlain, A. 1997; Missing stages of life – towards the perception of children in archaeology. In J. Moore and E. Scott (Eds.) *Invisible People and Processes: writing gender and childhood into European archaeology.* London: Leicester University Press: 248-250.

Chamberlain, A. 2006; *Demography in Archaeology.* Cambridge: Cambridge University Press.

Chapman, P. R., Bag, A. K., Tubbs, R. S., and Gohlke, P. 2013a; Practical Anatomy of the Central Skull Base Region. *Seminars in Ultrasound, CT and MRI*: 381-392.

Chapman, P. R., Gaddamanugu, S., Bag, A. K., Roth, N. T. and Vattoth, S. 2013; Vascular Lesions of the Central Skull Base Region. *Seminars in Ultrasound, CT and MRI*: 459-475.

Chiswick, M. L. 1985; Intrauterine growth retardation. *British Medical Journal,* Vol. 291: 845-848.

Chmurzynska, A. 2010; Fetal programming: link between early nutrition, DNA methylation, and complex diseases. *Nutrition Reviews,* Vol. 68 (2): 87-98.

Clukay, C. J., Hughes, D. A., Rodney, N. C., Kertes, D. A. and Mulligan, C. J. 2018; DNA methylation complex genes in relation to stress and genome-wide methylation in mother-newborn dyads. *American Journal of Physical Anthropology,* Vol. 165 (1): 173-182.

Coathup, V., Northstone, K., Gray, R., Wheeler, S., and Smith, L. 2017; Dietary Patterns and Alcohol Consumption During Pregnancy: Secondary Analysis of Avon Longitudinal Study of Parents and Children. *Alcoholism: Clinical and Experimental Research,* Vol. 41 (6): 1120-1128.

Cole, T. J. 2012; Growth References and Standards. In N. Cameron and B. Bogin (Eds.) *Human Growth and Development.* London: Elsevier: 537-366.

Cole, W. G. 2003; Structure of Growth Plate and Bone Matrix. In F. H. Glorieux, J. M. Pettifor and H. Jüppner (Eds.) *Pediatric Bone: Biology and Diseases.* London: Academic Press: 1-41.

Collins, M. J., Nielsen-Marsh, C. M., Hiller, J., Smith, C. I., Roberts, J. P, Prigodich, R. V., Weiss, T. J., Csapo, J., Millard, A. R., and Turner-Walker, G. 2002; The survival of organic matter in bone: A review. *Archaeometry,* Vol. 44: 383-394.

Collis, J. R. 1968; Excavations at Owslebury, Hampshire: An Interim Report. *Antiquaries Journal,* Vol. 48 (1): 18-31.

Collis, J. R. 1970; Excavations at Owslebury, Hampshire: A Second Interim Report. *Antiquaries Journal,* Vol. 50: 246-261.

Collis, J. R. 1977; Owslebury (Hants) and the problem of burials on rural settlements. In R. Reece (Ed.) *Burial in the Roman World,* CBA Research Report, No. 22. London: The Council for British Archaeology: 26-34.

Collis, J. R. 1990; L'impact des processus d'urbanisation sur les sites ruraux: Le cas d'Owslebury, Hants, Angleterre. In A. Duval, J. P. Le Bihan and Y. Menez (Eds.) *Les Gaulois D'Armorique: La Fin De L'Age Du Fer En Europe Tempérée: Actes Du XII Colloque de l'AFEAF Quimper, Mai 1988.* Rennes : Association pour la Diffusion des Recherches Archéologiques dans l'Ouest de la France.

Collis, J. R. 1994; An Iron Age and Roman Cemetery at Owslebury, Hampshire. In A. P. Fitzpatrick and E. L. Morris (Eds.) *The Iron Age in Wessex: Recent Work.* Salisbury: Trust for Wessex Archaeology Ltd: 106-108.

Cornish, J. and Martin, T. J. 2003; Other Factors Controlling Bone Growth and Development: Calcitonin, CGRP, Osteostatin, Amylin, and Adrenomedullin. In F. H. Glorieux, J. M.

Pettifor and H. Jüppner (Eds.) *Pediatric Bone: Biology and Diseases.* London: Academic Press: 217-233.

Cornelius, M. D. and Day, N. L. 2009; Developmental consequences of prenatal tobacco exposure. *Current Opinion in Neurology*, Vol. 22: 121-125.

Couoh, L. R. 2017; Differences between biological and chronological age-at-death in human skeletal remains: A change of perspective. *American Journal of Physical Anthropology,* Vol. 163: 671-695.

Coussons-Read, M. E., Lobel, M., Carey, J. C., Kreither, M. O., D'Anna, K., Argys, L., Ross, R. G., Brandt, C. and Cole, S. 2012; The occurrence of preterm delivery is linked to pregnancy-specific distress and elevated inflammatory markers across gestation. *Brain, Behaviour, and Immunity,* Vol. 26 (4): 650-659.

Cowal L. 2007a *St Benet Sherehog* [Online] [Accessed: November 2016] Available from: https://www.museumoflondon.org.uk/collections/other-collection-databases-and-libraries/centre-human-bioarchaeology/osteological-database/medieval-cemeteries/st-benet-sherehog

Cowal, L. 2007b *Spital Square* [Online] [Accessed: November 2016] Available from: https://www.museumoflondon.org.uk/collections/other-collection-databases-and-libraries/centre-human-bioarchaeology/osteological-database/medieval-cemeteries/spital-square

Cowal, L. 2008 *St Benet Sherehog cemetery summary* [Online] [Accessed: November 2016] Available from: https://www.museumoflondon.org.uk/collections/other-collection-databases-and-libraries/centre-human-bioarchaeology/osteological-database/post-medieval-cemeteries/st-benet-sherehog-post-medieval

Cowie, R. Bekvalac, J. and Kausmally, T. 2008; *Late 17th- to 19th-century burial and earlier occupation at All Saints, Chelsea Old Church, Royal Borough of Kensington and Chelsea* (MOLAS Study Series 18). London: Museum of London Archaeology Service.

Cox, M. and S. Mays (Eds.); *Human Osteology in Archaeology and Forensic Science*. London: Greenwich Medical Media Ltd.

Crawford, S. 1991; When do Anglo-Saxon children count? *Journal of Theoretical Archaeology* Vol. 2: 17-24.

Crawford S. 1993; Children, death and the afterlife in Anglo-Saxon England. *Anglo- Saxon Studies in Archaeology and History,* Vol. 6:83–91.

Crawford, M. A., Doyle, W. and Meadows, N. 1987; Gender differences at birth and differences in fetal growth. *Human Reproduction,* Vol. 2: 517–20.

Cunliffe, B. 2003; *The Celts: A Very Short Introduction.* Oxford: Oxford University Press.

Cunliffe, B. 2004; *Iron Age Communities in Britain: An Account of England, Scotland and Wales from the Seventh Century BC until the Roman Conquest.* London: Routledge.

Cunningham, H. 1998; Histories of Childhood. *The American Historical Review,* Vol. 103 (4): 1195–1208.

Cussons-Read, M. E., Lobel, M., Carey, J. C., Kreither, M. O., D'Anna, K., Argys, L., Ross, R. G., Brandt, C. and Cole, S. 2012; The occurrence of preterm delivery is linked to pregnancy-specific distress and elevated inflammatory markers across gestation. *Brain, Behaviour and Immunity,* Vol. 26 (4): 650-659.

Cutress, T.W. and Suckling, G. W. 1982; The assessment of noncarious defects of enamel. *International Journal of Dentistry,* Vol. 32: 117-122.

Cyrkowicz, A. and Czekański, A. 1998; Intrauterine growth retardation (IUGR) vs small for gestational age fetus (SGA). Diagnostic aspects. *Ginekologia Polska,* Vol. 69 (4): 213-217.

Dabernat, H. and Crubézy, E. 2009; Multiple Bone Tuberculosis in a Child from Predynastic Upper Egypt (3200 BC). *International Journal of Osteoarchaeology,* Vol. 20 (6): 719-730.

Dancause, K. N., Cao, X. J., Veru, F., Xu, S., Long, H., Yu, C., Laplante, D. P., Walker, C. D. and King, S. 2012; Brief Communication: Prenatal and Early Postnatal Stress Exposure Influences Long Bone Length in Adult Rat Offspring. *American Journal of Physical Anthropology,* Vol. 149: 307-311.

Davis, E. P., Glynn, L. M., Waffarn, F. and Sandman, C. A. 2011; Prenatal maternal stress programs infant stress regulation. *Journal of Child Psychology and Psychiatry,* Vol. 52 (2): 119-129.

Dawson, H. 2017; Precious Things: Examining the Status and Care of Children in Late Medieval England through the Analysis of Cultural and Biological Markers. In L. Powell, W. Southwell-Wright, and R. Gowland (Eds.) *Care in the Past: Archaeological and Interdisciplinary Perspectives.* Oxford: Oxbow Books: 53-69.

De la Rúa C., Izagirre, N., and Manzano, C. 1995; Environmental stress in a medieval population of the Basque country. *Homo,* Vol. 45: 268–289.

De Silva, P. G., Evans-Jones, G., Wright, A. and Henderson, R. 2003; Physiological periostitis; a potential pitfall. *Archives of Disease in Childhood,* Vol. 88 (12): 1124-1125.

Degani, S. 2006; Sonographic findings in fetal viral infections: a systemic review. *Obstetrical and Gynaecological Survey,* Vol. 61 (5): 329-336.

Demirjian, A. 1990; Dentition. In F. Falkner and J. M. Tanner (Eds.) *Human Growth: A Comprehensive Treatise*. New York: Plenum Press: 269–297.

Derevenski, J. S. 1994; Perspectives on Children and Childhood. *Archaeological Review from Cambridge,* Vol. 13 (2).

Derevenski J. S. 1997; Engendering children, engendering archaeology. In J. Moore and E. Scott (Eds.) *Invisible People and Processes*. Leicester: Leicester University Press: 192–202.

Deter, R. I. and Harrist, R. B. 1992; Growth Standards for Anatomic Measurements and Growth Rates Derived from Longitudinal Studies of Normal Fetal Growth. *Journal of Clinical Ultrasound,* Vol. 20: 381-388.

DeWitte, S. N. 2014; Differential survival among individuals with active and healed periosteal new bone formation. *International Journal of Paleopathology,* Vol. 7: 38-44.

DeWitte, S. N. 2015; Bioarchaeology and the Ethics of Research Using Human Skeletal Remains. *History Compass,* Vol. 13 (1): 10-19.

DeWitte, S. N., Hughes-Morey, G, Bekvalac, J. and Karsten, J. 2016; Wealth, health and frailty in industrial-era London. *Annals of Human Biology,* Vol. 43 (3): 241-254.

DeWitte, S. N. and Stojanowski, C. M. 2015; The osteological paradox 20 years later: Past perspectives, future directions. *Journal of Archaeological Research,* Vol. 23: 397-450.

Dodrill, P. 2016; Typical Feeding and Swallowing Development in Infants and Children. In Groher, M. E. and Crary, M. A. (Eds.) *Dysphagia: Clinical Management in Adults and Children.* Missouri: Elsevier: 253-269.

Donald, H. H. 1952; *The Negro Freedman.* New York: Henry Schuman.

Dong, M., Giles, W. H., Felitti, V. J., Dube, S. R., Williams, J. E., Chapman, D. P., and Anda, R. F. 2004; Insights into causal pathways for ischemic heart disease: Adverse childhood experiences study. *Circulation,* Vol. 110 (13): 1761–1766.

Dowler, E. A. and Dobson, B. M. 1997; Nutrition and poverty in Europe: an overview. *Proceedings of the Nutrition Society,* Vol. 56: 51-62.

Dudar, C. J. 2010; Qualitative and Quantitative Diagnosis of Lethal Cranial Neural Tube Defects from the Fetal and Neonatal Human Skeleton, with a Case Study Involving Taphonomically Altered Remains. *Journal of Forensic Science,* Vol. 55 (4): 877-883.

Duren, D. L., Seselj, M., Froehle, A. W., Nahhas, R. W. and Sherwood, R. J. 2013; Skeletal Growth and the Changing Genetic Landscape during Childhood and Adulthood. *American Journal of Physical Anthropology*, Vol. 150: 48-57.

Dyson, L. Malt, D. Wellman, T. and White, B. 1987; *Excavations at Broad Street Station: The Broadgate Development Archive Report.* City of London (Unpublished).

Edmond, K. M., Zandoh, C., Quigley, M. A., Amenga-Etego, A., Owusu-Agyei, S. and Kirkwood, B. R. 2006; Delayed Breastfeeding Initiation Increases Risk of Neonatal Mortality. *Pediatrics*, Vol. 117 (3): 380-386.

Egger, G., Liang, G., Aparicio, A. and Jones, P. A. 2004; Epigenetics in human disease and prospects for epigenetic therapy. *Nature,* Vol. 429: 457-463.

Egliston, K-A., McMahon, C., and Austin, M-P. 2007: Stress in pregnancy and infant HPA axis function: conceptual and methodological issues relating to the use of salivary cortisol as an outcome measure. *Psychoneuroendocrinology*, Vol. 32: 1–13.

Eisenberg, D. T. A., Borja, J. B., Hayes, M. G. and Kuzawa, C. W. 2017; Early life infection, but not breastfeeding, predicts adult blood telomere lengths in the Philippines. *American Journal of Human Biology,* Vol. 29 (Early View): 1-11.

Elamin, F. and Liversidge, H. M. 2013; Malnutrition has no effect on the timing of human tooth formation. *PLoS ONE* 8:e72274.

Ellison, P. T. 2018; The evolution of physical anthropology. *American Journal of Physical Anthropology,* Vol. 165: 615-625.

Evans, A. 2010; *The Pocket Podiatry Guide: Paediatrics.* Edinburgh: Elsevier.

Facchini, F., Rastelli, E. and Brasili, P. 2004; Cribra orbitalia and cribra cranii in Roman skeletal remains from the Ravenna area and Rimini (I-IV century AD). *International Journal of Osteoarchaeology*, Vol. 14: 126-136.

Faerman, M. 1999; Ancient DNA diagnosis of bone pathology in infancy and early childhood. *American Journal of Physical Anthropology,* Suppl. 28:125.

Falys, C. G. and Lewis, M. E. 2011; Proposing a way forward: A review of standardisation in the use of age categories and ageing technique in osteological analysis. *International Journal of Osteoarchaeology,* Vol. 21: 704-716.

Farewell, C. V., Thayer, Z. M., Tracer, D. P. and Morton, S. 2018; Prenatal stress exposure and early childhood BMI: Exploring association in a New Zealand context. *American Journal of Human Biology,* [Early View].

Farmer, P. 1996; Social inequalities and emerging infectious diseases. *Emerging infectious Diseases,* Vol. 2 (4): 259-269.

Fazekas, I. G. and Kósa, F. 1978; *Forensic Foetal Osteology.* Budapest: Academic Press.

Feinstein, J. S. 1993; The Relationship between Socioeconomic Status and Health: A Review of the Literature. *The Milbank Quarterly,* Vol. 71(2): 279-322.

Fell, D. B., Savitz, D. A., Kramer, M. S., Gessner, B. D., Katz, M. A., Knight, M., Luteijn, J. M., Marshall, H., Bhat, N., Gravett, M. G., Skidmore, B. and Ortiz, J. R. 2016; Maternal influenza and birth outcomes: systematic review of comparative studies. *International Journal of Obstetrics and Gynaecology,* Vol. 124: 48-59.

Fenichel, G. M., Webster, D. L., Wong, W. K. T. 1984; Intracranial Hemorrhage in the Term Newborn. *Archives of Neurology,* Vol. 41 (1): 30-34.

Fforde, C. 2004; Collecting the Dead: Archaeology and Reburial Issues. London: Duckworth & Co. Ltd.

Fildes, V. 1988; Wet nursing. New York: Basil Blackwell Ltd.

Fildes, V. 1995; The culture and biology of breastfeeding: an historical review of Western Europe. In: Stuart-Macadam, P., and Dettwyker, K. (Eds.) *Breastfeeding: biocultural perspectives*. Hawthorne: Aldine De Gruyter: 101-126.

Finlay, N. 2013; Archaeologies of the beginnings of life. *World Archaeology,* Vol. 45 (2): 207-214.

Floud, R., Wachter, K.W. and Gregory, A. 1990; *Height, health and history: nutritional status in the United Kingdom, 1750–1980*. Cambridge: Cambridge University Press.

Forbes, T. R. 1972; Mortality Books for 1820 to 1849 from the Parish of St. Bride, Fleet Street, London. *Journal of the History of Medicine*: 15-29.

Fowler, L. and Powers, N. 2012; *Doctors, Dissection and Resurrection Men: Excavations in the 19th-century burial ground of the London Hospital, 2006* (MOLA Monograph 62). London: Museum of London Archaeology.

Franco, K. M. D., Line, S. R. P. and de Moura-Ribeiro, M. V. L. 2007; Prenatal and Neonatal Variable Associated with Enamel Hypoplasia in Deciduous Teeth in Low Birth Weight Preterm Infants. *Journal of Applied Oral Science,* Vol. 15 (6): 518-523.

Fried, R. L., Mayol, N. L., McDade, T. W., Kuzawa, C. W. 2017; Maternal metabolic adaptations to pregnancy among young women in Cebu, Philippines. *American Journal of Human Biology,* Vol. 29 [Early View].

Friendship-Taylor, R. M. and Friendship-Taylor, D. E. 2012; *Iron Age and Roman Piddington: 10th Interim Report and Phase Descriptions of the Late Iron Age Settlements, Military Phase, Roman Villa Complex and Early Saxon Phases at Piddington, Northants.* The Upper Nene Archaeological Society 2013.

Fujita, M., Lo, Y. J. and Brindle, E. 2017; Nutritional, inflammatory, and ecological correlates of maternal retinol allocation to breast milk in agro-pastoral Ariaal communities of northern Kenya. *American Journal of Human Biology,* Vol. 29 (Early View): 1-14.

Fuller, B. T., Fuller, J. L., Harris, D. A. and Hedges, R. E. M. 2006; Detection of Breastfeeding and Weaning in Modern Human Infants with Carbon and Nitrogen Stable Isotope Ratios. *American Journal of Physical Anthropology,* Vol. 129 (2): 279-293.

García, A. R., Gurven, M. and Blackwell, A. D. 2017; A matter of perception: Perceived socio-economic status and cortisol on the island of Utila, Honduras. *American Journal of Human Biology,* Vol. 29 (5): 1-16.

Garland, A. N. and Janaway, R. C. 1989; The taphonomy of inhumation burials. In C. Roberts, F. Lee and J. Bintliff (Eds.) *Burial Archaeology: Current Research, Methods and Developments.* BAR British Series 211. Oxford: Archaeopress: 15-37.

Garn, S. M. and Clark, D. C. 1975; Nutrition, Growth, Development, and Maturation: Findings From the Ten-State Nutrition Survey of 1968-1970. *Pediatrics,* Vol. 56 (2): 306-319.

Garn, S. M., Lewis, A. B. and Polacheck, D. L. 1960; Interrelations in dental development. I. Interrelationships within the dentition. *Journal of Dental Research,* Vol. 39: 1049-1055.

Garn, S. M., Sandusky, S. T., Rosen, N. N. and Trowbridge, F. 1973; Economic Impact on Postnatal Ossification. *American Journal of Physical Anthropology,* Vol. 38: 1-4.

Gesell, A. 1928; *Infancy and Human Growth.* New York: The Macmillan Company.

Gigante, D. P., Nazmi, A., Lima, R. C., Barros, F. C. and Victora, C. G. 2009; Epidemiology of early and late growth in height, leg and trunk length: findings from a birth cohort of Brazilian males. *European Journal of Clinical Nutrition,* Vol. 63: 375-381.

Gilmore, H. F. and Halcrow, S. E. 2014; Sense or Sensationalism? Approaches to Explaining High Perinatal Mortality in the Past. In J. L. Thompson, M. P. Alfonso-Durruty and J. J. Crandall (Eds.) *Tracing Childhood: Bioarchaeological Investigations of Early Lives in Antiquity.* Gainesville: University Press of Florida: 123-138.

Gilsanz, V., Kovanlikaya, A., Costin, G., Roe, T. F., Sayre, J. and Kaufman, F. 1997; Differential Effect of Gender on the Sizes of the Bones in the Axial and Appendicular Skeletons. *Journal of Clinical Endocrinology and Metabolism*, Vol. 82 (5): 1603-1607.

Gindhart, P. S. 1969; The frequency of appearance of transverse lines in the tibia in relation to childhood illness. *American Journal of Physical Anthropology*, Vol. 31: 17-22.

Gindhart, P. S. 1973; Growth standards for the tibia and radius in children aged one month through eighteen years. *American Journal of Physical Anthropology*, Vol. 39: 41-48.

Gindhart, P. S. 1989; An Early Twentieth-Century Skeleton Collection. *Journal of Forensic Sciences*, Vol. 34 (4): 887-893.

Ginn, J. and Arber, S. 1995; "Only connect": gender relations and ageing. In S. Arber and J. Ginn (Eds.) *Connecting Gender and Ageing: A Sociological Approach*. Buckingham: Open University Press: 1-14.

Glover, V. 2015; Prenatal Stress and Its Effects on the Fetus and the Child: Possible underlying Biological Mechanisms. In M. C. Antonelli (Ed.) *Perinatal Programming of Neurodevelopment* (Volume 10): New York: Springer: 269-283.

Gluckman, P. D. 1997; Endocrine and nutritional regulation of prenatal growth. *Acta Paediatrica* Supplementary Series, Vol. 423: 153-157.

Gluckman, P. D. and Hanson, M. A. 2004; Living with the past: evolution, development, and patterns of disease. *Science*, Vol. 305: 1733–36.

Gluckman, P. D. and Hanson, M. A. 2005; *The Fetal Matrix: Evolution, Development and Disease*. Cambridge: Cambridge University Press.

Gluckman, P. D. and Hanson, M. A. 2006; *The Developmental Origins of Health and Disease*. Cambridge: Cambridge University Press.

Godbout, J. P. and Glaser, R. 2006; Stress-Induced Immune Dysregulation: Implications for Wound Healing, Infectious Disease and Cancer. *Journal of NeuroImmune Pharmacology,* Vol. 1: 421-427.

Goldenberg, R. L & Thompson, C. 2003; The infectious origins of stillbirth. *American Journal of Obstetric Gynecology*, Vol. 189, No. 3: 861-873.

Goodman, A. H. 1991; Health, Adaptation, and Maladaptation in Past Societies. In H. Bush and M. Zvelebil (Eds.) *Health in Past Societies: Biocultural interpretations of human skeletal remains in archaeological contexts* (BAR International Series 567). Oxford: BAR Publishing: 31-38.

Goodman, A. H. 1993; On the interpretation of health from skeletal remains. *Current Anthropology,* Vol. 34: 281-288.

Goodman, A. H. and Armelagos, G. J. 1988; Childhood Stress and Decreased Longevity in a Prehistoric Population. *American Anthropologist,* Vol. 90 (4): 936-944.

Goodman, A. H. and Armelagos, G. J. 1989; Infant and Childhood Morbidity and Mortality Risks in Archaeological Populations. *World Archaeology,* Vol. 21 (2): 225-243.

Goodman, A. H., Armelagos, G. J. and Rose, J. C. 1984; The Chronological Distribution of Enamel Hypoplasias From Prehistoric Dickson Mounds Populations. *American Journal of Physical Anthropology,* Vol. 65: 259-266.

Goodman, A.H. Brooke Thomas, R. Swedlund, A. & Armelagos, G.J. 1988; Biocultural perspectives on stress in prehistoric, historical, and contemporary population research. *Yearbook of Physical Anthropology* 31: 169-202.

Goodman, A.H. and Martin, D. L. 2002; Reconstructing health profiles from skeletal remains. In R. H. Steckel and J.C. Rose (Eds.) *The Backbone of History: Health and Nutrition in the Western Hemisphere.* Cambridge: Cambridge University Press:11-60.

Goodman, A. H. and Rose, J. C. 1990; Assessment of systemic physiological perturbations from dental enamel hypoplasias and associated histological structures. *Yearbook of Physical Anthropology*, Vol. 33: 59-110.

Goodman, A. H., Thomas, R. B., Swedlund, A. C. and Armelagos, G. J. 1988; Biocultural Perspectives on Stress in Prehistoric, Historical, and Contemporary Population Research. *Yearbook of Physical Anthropology,* Vol. 31: 169-202.

Gordon, C. C. and Buikstra, J. 1981; Soil pH, bone preservation and sampling bias at mortuary sites. *American Antiquity,* Vol. 48: 566–571.

Gowland, R. 2001; Playing Dead: implications of mortuary evidence for the social construction of childhood in Roman Britain. In G. Davies, A. Gardner and K. Lockyear (Eds.) *TRAC 2000: Proceedings of the Tenth Annual Theoretical Roman Archaeology Conference, London 2000.* Oxford: Oxbow Books: 152-168.

Gowland, R. L. 2002; Age as an Aspect of Social Identity in Fourth- to Sixth-Century AD England: The Archaeological Funerary Evidence. Ph.D. Thesis, University of Durham, Durham, UK.

Gowland, R. L. 2004; The social identity of health in late Roman Britain. In B. Croxford, H. Eckardt, J. Meade, and J. Weekes (Eds.) *TRAC 2003: Proceedings of the Thirteenth Annual Theoretical Roman Archaeology Conference.* Oxford: Oxbow Books: 135-146.

Gowland, R. L. 2006; Ageing the past: Examining age identity from funerary evidence. In R. L. Gowland and C. Knüsel (Eds.) *Social Archaeology of Funerary Remains*. Oxford: Oxbow:143-154.

Gowland, R. L. 2015; Entangled Lives: Implications of the Developmental Origins of Health and Disease Hypothesis for Bioarchaeology and the Life Course. *American Journal of Physical Anthropology*, Vol. 158, No. 4: 530-540.

Gowland, R. L. 2017 (*In Press)*; Infants and mothers: linked lives and embodied life courses. In S. Crawford, D. Hadley and G. Shepherd (Eds.) *The Oxford Handbook of the Archaeology of Childhood.* Oxford: Oxford University Press: Chapter 6.

Gowland, R. L. and Chamberlain, A. T. 2002; A Bayesian Approach to Ageing Perinatal Skeletal Material from Archaeological Sites: Implications for the Evidence for Infanticide in Roman-Britain. *Journal of Archaeological Science,* Vol. 79: 677-685.

Gowland, R. L., Chamberlain, A. T., Redfern, R. C. 2014. On the brink of being: re-evaluating infant death and infanticide in Roman Britain, in M. Carroll and E-J. Graham (Eds.), *Infant Health and Death in Roman Italy and Beyond*, Journal of Roman Archaeology Supplementary Series 98, 69-88.

Gowland, R. L. and Knüsel, C. 2006; Introduction. In R. L. Gowland and C. Knüsel (Eds.) *Social Archaeology of Funerary Remains*. Oxford: Oxbow:.i-xii.

Gowland, R. L. and Redfern, R. C. 2010; Childhood Health in the Roman World: Perspectives from the Centre and Margin of the Empire. *Childhood in the Past*, Vol. 3: 15-42.

Gowland, R. L. and Thompson, T. J. U. 2013; *Human Identity and Identification*. Cambridge: Cambridge University Press.

Grainger, I. and Phillpotts, C. 2011; *The Cistercian abbey of St Mary Graces, East Smithfield, London* (MOLA Monograph 44). London: Museum of London Archaeology.

Grainger, I. Hawkins, D. Cowal, L. and Mikulski, R. 2008; *The Black Death cemetery, East Smithfield, London* (MOLAS Monograph 43). London: Museum of London Archaeology Service.

Grantz, K. L., Hediger, M. L. Liu, D. and Buck Louis, G. M. 2018; Fetal Growth Standards: the NICHD fetal growth study approach in context with INTERGROWTH-21st and the World Health Organization Multicentre Growth Reference Study. *American Journal of Obstetrics and Gynecology*, [Early View]: S641-S655.e28.

Green, M. S. 1992; The male predominance in the incidence of infectious diseases in children: A postulated explanation for disparities in the literature. *International Journal of Epidemiology,* Vol. 21: 381–386.

Griffin, R. Pitts, M. Smith, R and Brook, A. 2011; Inequality at Late Roman Baldock; The impact of Social Factors on Health and Diet. *Journal of Anthropological Research,* Vol. 67. (No. 4): 533-556.

Gustafson, G. and Koch, G. 1974; Age estimation up to 16 years of age based on dental development. *Odontologisk Revy,* Vol.25 (3): 297-306.

Guy H., Masset, C. and Baud, C. A. 1997; Infant taphonomy. *International Journal of Osteoarchaeology,* Vol. 7:221–229.

Habicht, J. P., Yarbrough, C., Martorell, R., Malina, R. M., and Klein, R. E. 1974; Height and weight standards for preschool children. *Lancet,* 1: 7858.

Hahn, P. 1972; Lipid Metabolism and Nutrition in the Prenatal and Postnatal Periods. In M. Winick (Ed.) *Nutrition and Development.* London: John Wiley & Sons Inc.: 99-134.

Halcrow, S. E. and Tayles, N. 2008; The Bioarchaeological Investigation of Childhood and Social Age: Problems and Prospects. *Journal of Archaeological Method and Theory*, Vol. 15: 190-215.

Halcrow, S. E. and Tayles, N. 2011; The biological investigation of children and childhood. In S.C. Agarwal and B.A. Glencross (Eds.) *Social Bioarchaeology.* Oxford: Wiley-Blackwell: 333-360.

Halcrow, S. E., Tayles, N. and Elliot, G. E. 2018; The Bioarchaeology of Fetuses. In S. Han, T. K. Betsinger, and A. B. Scott (Eds.) *The Fetus: Biology, Culture, and Society.* New York: Berghahn Books: 83-111.

Halcrow, S. E., Tayles, N., Inglis, R. and Higham, C. 2012; Newborn twins from prehistoric mainland Southeast Asia: birth, death and personhood. *Antiquity*, Vol. 86: 838-852.

Halcrow, S. E. and Ward, S. M. 2017; Bioarchaeology of Childhood. In H. Montgomery (Ed.) *Oxford Bibliographies in Childhood Studies.* New York: Oxford University Press.

Hales, C. N. and Barker, D. J. P. 2001; The thrifty phenotype hypothesis. *British Medical Bulletin,* Vol. 60: 5-20.

Halfon, N., Larson, K., Lu, M., Tullis, E. and Russ, S. 2014; Lifecourse Health Development: Past, Present and Future. *Maternal and Child Health Journal,* Vol. 18: 344-365.

Hammer, O., Harper, D. A. T. and Ryan, P. D. 2001; PAST Paleontological Statistics Software Package for Education and Data Analysis. [Online] [Accessed August 2016] Available from: http://palaeo-electronica.org/2001_1/past/issue1_01.htm

Han, J. C., Lawlor, D. A., and Kimm, S. Y. 2010; Childhood obesity. *Lancet,* Vol. 375 (9727): 1737–1748.

Harding, D. W. 1974; *The Iron Age in Lowland Britain.* London: Routledge.

Harding, J. E. and Johnston, B. M. 1995; Nutrition and Fetal Growth. *Reproduction, Fertility and Development,* Vol. 7, No. 3: 539-548.

Harding, V. 2002; *The dead and the living in Paris and London: 1500-1670.* Cambridge: Cambridge University Press.

Hauspie, R. C., Bergman, P., Bielicki, T. and Susanne, C. 1994; Genetic variance in the in the pattern of the growth curve for height: a longitudinal analysis of male twins. *Annals of Human Biology,* Vol. 21: 347-362.

Hay, W. W., Brown, L. D., Rozance, P. J., Wesolowski, S. R. and Limesand, S. W. 2016; Challenges in nourishing the intrauterine growth-restricted foetus – Lessons learned from studies in the intrauterine growth-restricted foetal sheep. *Acta Paediatrica,* Vol. 105: 881-889.

Haymond, M., Kappelgaard, A-M., Czernichow, P., Biller, B. M. K., Takano, K., and Kiess, W. 2013; Early recognition of growth abnormalities permitting early intervention. *Acta Paediatrica,* Vol. 102: 787-796.

He, Y., Chen, J., Zhu, L-H., Hua, L-L., and Ke, F-F. 2017; Maternal Smoking during Pregnancy and ADHD: Results from a Systematic Review and Meta-Analysis of Prospective Cohort Studies. *Journal of Attention Disorders,* [Early View].

Heinke, D and Kuzawa, C. W. 2008; Self-reported illness and birth weight in the Philippines: implications for hypotheses of adaptive fetal plasticity. *American Journal of Human Biology*, Vol. 20: 538-544.

Helfrecht, C., Hagen, E. H., DeAvila, D., Bernstein, R. M., Dira, S. J. and Meehan, C. L. 2017; DHEAS patterning across childhood in three sub-Saharan populations: Associations with age, sex, ethnicity, and cortisol. *American Journal of Human Biology,* Vol. 30 [Early View]: 1-17.

Heneage Cocks, A. 1921; A Romano-British homestead in the Hambleden Valley, Buckinghamshire. *Archaeologia* 71:141–66.

Heuzé, Y. and Cardoso, H. F. V. 2008; Testing the Quality of Nonadult Bayesian Dental Age Assessment Methods to Juvenile Skeletal Remains: The Lisbon Collection Children and Secular Trend Effects. *American Journal of Physical Anthropology,* Vol. 135: 275-283.

Hill, J. D. 1995; The Pre-Roman Iron Age in Britain and Ireland (ca. 800 B.C. to A. D. 100): An Overview. *Journal of World Prehistory,* Vol. 9 (1): 47-98.

Hill, J. and Rowsome, P. 2011; *Roman London and the Walbrook stream crossing: Excavations at 1 Poultry and vicinity, City of London* (MOLA Monograph 37). London: Museum of London Archaeology,

Hillson, S. W. 1979; Diet and dental disease. *World Archaeology,* Vol. 11; 147–62.

Hillson, S. W. 2005; *Teeth* (Second Edition). Cambridge: Cambridge University Press.

Hillson, S. W. 2009; The World's Largest Infant Cemetery and Its Potential for Studying Growth and Development. *Hesperia Supplement,* Vol. 43: 137-154.

Hirschfield, L. A. 2002; Why Don't Anthropologists Like Children? *American Anthropologist,* Vol. 104 (2): 611-627.

Hodson, C. M. 2012; *Between Roundhouse and Villa: An Investigation into Perinatal Mortality at the Late Iron Age/Early Roman Site of Piddington, Northamptonshire.* (Unpublished MSc Thesis) University of Exeter.

Hodson, C. M. 2017; Between Roundhouse and Villa: Assessing Perinatal and Infant Burials from Piddington, Northamptonshire. *Britannia,* Vol. 48: 195-219.

Hoffman, M. C. 2016; Stress, the Placenta, and Fetal Programming of Behaviour: Genes' First Encounter with the Environment. *American Journal of Psychiatry,* Vol. 173 (7): 655-657.

Hohler, C. W. 1984; Ultrasound Estimation of Gestational Age. *Clinical Obstetrics and Gynecology,* Vol. 27 (2): 314-326.

Holdsworth, E. A. and Schell, L. M. 2017; Maternal-infant interaction as an influence on infant adiposity. *American Journal of Human Biology,* Vol. 29 [Early View].

Holick, M. F. 2005; The Vitamin D Epidemic and its Health Consequences. *The Journal of Nutrition,* Supplement: 2739S-2748S.

Holland-Jones, J. 2005; Fetal Programming: Adaptive Life-History Tactics or Making the Best of a Bad Start? *American Journal of Human Biology,* Vol. 17: 22-33.

Holman, D. J. and Jones, R. E. 1998; Longitudinal analysis of deciduous tooth emergence. II: Parametric survival analysis in Bangladeshi, Guatemalan, Japanese, and Javanese children. *American Journal of Physical Anthropology,* Vol. 105: 209-230.

Holman, D. J. and Yamaguchi, K. 2005; Longitudinal Analysis of Deciduous Tooth Emergence: IV. Covariate Effects in Japanese Children. *American Journal of Physical Anthropology,* Vol. 126: 352-358.

Holmes, S. J. 1937; *The Negro's Struggle for Survival: A Study in Human Ecology.* Berkeley: University of California Press.

Hoppa, R. D. 1992; Evaluating Human Skeletal Growth: An Anglo-Saxon Example. *International Journal of Osteoarchaeology,* Vol. 2: 275-288.

Hoppa, R. D. and Fitzgerald, C. M. 1999; From head to toe: integrating studies from bones and teeth in biological anthropology. In. D. Hoppa and C. M. Fitzgerald (Eds.) *Human growth in the past: studies from bones and teeth*. Cambridge: Cambridge University Press: 1-32.

Huang, J. S., Lee, T. A., and Lu, M. C. 2007; Prenatal programming of childhood overweight and obesity. *Maternal and Child Health Journal,* Vol. 11 (5): 461–473.

Huber, M., Knottnerus, J. A., Gren, L., van der Horst, H., Jadad, A. R., Kromhout, D., Leonard, B., Lorig, K., Loureiro, M. I., van der Meer, J. W. M., Schnabel, P., Smith, R., van Weel, C. and Smid, H. 2011; How should we define health? *British Medical Journal,* Vol. 343: d4163.

Huda, T. F. J. and Bowman, J. E. 1995; Age Determination From Dental Microstructure in Juveniles. *American Journal of Physical Anthropology,* Vol. 97: 135-150.

Hughes, C., Heylings, D. J. A. and Power, C. 1996; Transverse (Harris) lines in Irish archaeological remains. *American Journal of Physical Anthropology,* Vol. 101: 115–131.

Hujoel, P. P., Masterson, E. E. and Bollen, A. M. 2017; Lower face asymmetry as a marker of developmental instability. *American Journal of Human Biology,* Vol. 29 [Early View].

Humphrey, L. 1998; Growth patterns in the modern human skeleton. *American Journal of Physical Anthropology,* Vol. 105: 57–72.

Humphrey, L. 2000a; Interpretations of the growth of past populations. In J. S. Derevenski (Ed.) *Children and Material Culture*. London: Routledge: 193–205.

Humphrey, L. 2000b; Growth Studies of Past Population: An Overview and an Example. In M. Cox and S. Mays (Eds.) *Human Osteology in Archaeology and Forensic Science*. London: Greenwich Medical Media Ltd: 23-38.

Hunt, D. 1990; Sex determination in the subadult ilia: an indirect test of Weaver's non-metric sexing method. *Journal of Forensic Sciences,* Vol. 35: 881–885.

Hunt, D. [Personal Communication: October 2015]; *Brief background to the fetal collections housed at the NMNH* (Unpublished).

Hunt, E. E. and Hatch, J. W. 1981; The estimation of age at death and ages of formation of transverse lines from measurements of human long bones. *American Journal of Physical Anthropology,* Vol. 54: 461-469.

Huxley, A. K. and Angevine, J. B. 1998; Determination of Gestational Age from Lunar Age Assessments in Human Fetal Remains. *Journal of Forensic Science,* Vol. 43 (6): 1254-1256.

Ingvarsson-Sundström, A. 2003; *Children lost and found: a bioarchaeological study of Middle Helladic children in Asine with a comparison to Lerna*. Ph.D. Thesis, Uppsala University, Uppsala, Sweden.

Innes, A. M., Seshia, M. M., Prasad, C., Al Saif, S., Friesen, F. R., Chudley, A. E., Reed, M., Dilling, L. A., Haworth, J. C. and Greenberg, C. R. 2002; Congenital rickets caused by maternal vitamin D deficiency. *Paediatric Child Health,* Vol. 7 (7): 455-458.

Issel, E. P. 1985; Ultrasonic measurement of the growth of fetal limb bones in normal pregnancy. *Journal of Perinatal Medicine,* Vol. 13: 305-313.

Ivanovski, A. 1923; Physical modification of the population of Russia under famine. *American Journal of Physical Anthropology,* Vol. 6: 331-353.

Ives, R. and Humphrey, L. 2017; Patterns of long bone growth in a mid-19th century documented sample of the urban poor from Bethnal Green, London, UK. *American Journal of Physical Anthropology*, Vol. 163 (1): 173-186.

Jackes, M. 2011; Representativeness and Bias in Archaeological Skeletal Samples. In S.C. Agarwal and B.A. Glencross (Eds.) *Social Bioarchaeology.* Oxford: Wiley-Blackwell: 385-311.

Jeanty, P., Cantraine, F., Romero, R., Cousaert, E. and Hobbins, J. C. 1984a; A Longitudinal Study of Fetal Weight Growth. *Journal of Ultrasound Medicine,* Vol. 3: 321-328.

Jeanty, P., Rodesch, F., Delbeke, D. and Dumont, J. E. 1984b; Estimation of Gestational Age from Measurements of Fetal Long Bones. *Journal of Ultrasound Medicine,* Vol. 3: 75-79.

Jeanty, P. and Romero, R. 1984; Estimation of Gestational Age. *Seminars in Ultrasound, CT and MRI,* Vol. 5: 121-129.

Johnston F. E. 1962; Growth of the long bones of infants and young children at Indian Knoll. *American Journal of Physical Anthropology,* Vol. 20:249–254.

Johnston, F. E., Wainer, H., Thissen, D., and MacVean, R. 1976; Hereditary and Environmental Determinants of Growth in Height in a Longitudinal Sample of Children and Youth of Guatemala and European Ancestry. *American Journal of Physical Anthropology,* Vol. 44 (3): 469-476.

Johnston F. E. and Zimmer, L. O. 1989; Assessment of Growth and Age in the Immature Skeleton. In M. Y. Işcan and K. A. R. Kennedy (Eds.) *Reconstruction of Life From the Skeleton.* New York: Alan R. Liss:11-21

Jones H. 1991; *Preliminary Report of Archaeological Excavations at New London Bridge House, London Bridge Street, S.E.1.* Museum of London, Department of Great London Archaeology: London. (Unpublished)

Joseph, K. S. and Kramer, M. S. 1996; Review of the Evidence on Fetal and Early Childhood Antecedents of Adult Chronic Disease. *Epidemiologic Reviews,* Vol. 18 (2): 158-174.

Jukic, A. M., Baird, D. D., Weinberg, C. R., McConnaughey, D. R. and Wilcox, A. J. 2013; Length of human Pregnancy and Contributors to Its Natural Variation. *Reproductive Epidemiology,* Vol. 28 (10): 2848-2855.

Kamp, K. A. 2001; Where have all the children gone?: The archaeology of childhood. *Journal of Archaeological Method and Theory,* Vol. 8: 1-34.

Kamp, K. A. 2015; Children and their Childhoods: Retrospectives and Prospectives. *Childhood in the Past,* Vol. 8 (2): 161-169.

Kaplan, B. A. 1954; Environment and Human Plasticity. *American Anthropologist,* Vol. 56 (5): 780-800.

Karlberg, J. 1987; On the modelling of human growth. *Statistics in Medicine,* Vol. 6: 185-192.

Karsenty, G. and Kronenberg, H. M. 2003; Postnatal Bone Growth: Growth Plate Biology, Modelling, and Remodeling. In F. H. Glorieux, J. M. Pettifor and H. Jüppner (Eds.) *Pediatric Bone: Biology and Diseases.* London: Academic Press: 119-133.

Katzenberg, A. 2000; Stable isotope analysis: a tool for studying past diet, demography, and life history. In A. Katzenberg and S. R. Saunders (eds.) *Biological Anthropology of the Human Skeleton*. New York: Wiley-Liss: 305–327.

Kausmally, T. 2007 *East Smithfield Black Death cemetery summary* [Online] [Accessed: November 2016] Available from: https://www.museumoflondon.org.uk/collections/other-collection-databases-and-libraries/centre-human-bioarchaeology/osteological-database/medieval-cemeteries/east-smithfield-black-death-medieval

Kausmally, T. 2008 *St Bride's Lower churchyard cemetery summary* [Online] [Accessed: November 2016] Available from: https://www.museumoflondon.org.uk/collections/other-collection-databases-and-libraries/centre-human-bioarchaeology/osteological-database/post-medieval-cemeteries/st-brides-lower-post-medieval

Keinan-Boker, L. 2014; "The mothers have eaten unripe grapes and children's teeth are set on edge": the potential inter-generational effects of the Holocaust on chronic morbidity in Holocaust survivors' offspring. *Israel Journal of Health Policy Research,* Vol. 3 (11): 1-7.

Kelnar, C. J. H., Harvey, D. and Simpson, C. 1995; *The Sick Newborn Baby.* London: Baillière Tindall.

Key, C. 2000; The evolution of human life history. *World Archaeology,* Vol. 31: 329–350.

Khashan, A.S., Abel, K.M., McNamee, R., Pedersen, M. G., Webb, R. T., Baker, P.N., Kenny, L. C., and Mortensen, P. B. 2008; Higher risk of offspring schizophrenia following antenatal maternal exposure to severe adverse life events. *Archives of General Psychiatry*, Vol. 65: 146–152.

King, S. E. and Ulijaszek, S. J 1999; Invisible insults during growth and development: contemporary theories and past populations. In R. D. Hoppa and C. M. Fitzgerald (Eds.) *Human growth in the past: studies from bones and teeth*. Cambridge: Cambridge University Press: 161- 182.

Kini, U. and Nandeesh, B. N. 2012; Physiology of Bone Formation, Remodeling, and Metabolism. In I. Fogelman, G. Gnanasegaran, and H. van der Wall (Eds.) *Radionuclide and Hybrid Bone Imaging.* Verlag Berlin Heidelberg: Springer.

Kiserud, T., Piaggio, G., Carroli, G., Widmer, M., Carvalho, J., Neerup Jensen, L., Giordano, D., Guilherme Cecatti, J., Abdel Aleem, H., Talegawkar, S. A., Benachi, A., Diemert, A., Tshefu Kitoto, A., Thinkhamrop, J., Lumbiganon, P., Tabor, A., Kriplani, A., Gonzalez Perez, R., Hecher, M. A., Gülmezoglu, A. M. and Platt, L. D. 2017; The World Health Organization Fetal Growth Charts: A Multinational Longitudinal Study of Ultrasound

Biometric Measurements and Estimated Fetal Weight. *PLoS Medicine,* Vol. 14 (1): e1002220 (1-36).

Klaus, H. D. 2012; The Bioarchaeology of Structural Violence. In D. L. Martin, R. P. Harrod, and V. R. Perez (Eds.) *The Bioarchaeology of Violence. Bioarchaeological Interpretations of the Human Past: Local, Regional, and Global.* Gainesville: University Press of Florida: 29–62.

Klaus, H. D. 2014; Frontiers in the Bioarchaeology of Stress and Disease: Cross-Disciplinary Perspectives From Pathophysiology, Human Biology, and Epidemiology. *American Journal of Physical Anthropology,* Vol. 155: 294-308.

Kliegman, R. M. 2011; Intrauterine Growth Restriction. In R. J. Martin, A. A. Fanaroff and M. C. Walsh 2011; *Fanaroff and Martin's Neonatal-Perinatal Medicine.* Missouri: Elsevier: 245-275.

Kósa, F. 2002; Anthropological study for the determination of the Europid and Negroid characteristics on facial bones of human fetuses. *Acta Biologica Szegediensis,* Vol. 46 (1-2): 83-90.

Kramer M. S. 1998; Maternal nutrition, pregnancy outcome and public health policy. *Canadian Medical Association Journal*, Vol. 159: 663–665.

Kramer, M. S. and Joseph, K. S. 1996; Enigma of fetal/infant-origins hypothesis. *Lancet,* Vol. 348: 1269–1273.

Krenz-Niedbala, M. and Lukasik, S. 2017; Skeletal Evidence for Otitis Media in Mediaeval and Post-Mediaeval Children from Poland, Central Europe. *International Journal of Osteoarchaeology,* Vol. 27: 375-386.

Kuzawa, C. W. 2012; Early environments, developmental plasticity, and chronic degenerative disease. In N. Cameron and B. Bogin (Eds.) *Human Growth and Development* (2nd Edition). London: Elsevier: 325-341.

Kuzawa, C. W., Chugani, H. T., Grossman, L. L., Lipovich, L., Muzik, O., Hof, P. R., Wildman, D. E., Sherwood, C. C., Leonard, W. R. and Lange, N. 2014; Metabolic costs and evolutionary implications of human brain development. *PNAS,* Vol. 111 (36): 13010-13015.

Kuzawa, C. W. and Quinn, E. A. 2009; Developmental Origins of Adult Function and Health: Evolutionary Hypotheses. *Annual Review of Anthropology,* Vol. 38: 131-147.

Kuzawa, C. W. and Sweet, E. 2009: Epigenetics and the embodiment of race: developmental origins of US racial disparities in cardiovascular health. *American Journal of Human Biology,* Vol. 21: 2–15.

Kwon, D. S., Spevak, M. R., Fletcher, K. and Kleinman, P. K. 2002; Physiologic Subperiosteal New Bone Formation: Prevalence, Distribution, and Thickness in Neonates and Infants. *American Journal of Radiology,* Vol. 179: 985-988.

Kyselicová, K., Šebest, L., Beňus, R., Bognár, C., Šarkan, M. and Dörnhöferová, M. 2015; Skeletal manifestation of tuberculosis in the Medieval population of Borovce (8^{th}-12^{th} century AD, Slovakia) in relationship to the occurrence of long bone changes and cribra orbitalia. *Česká Antropologie,* Vol. 65 (2): 16-22.

Lallo, J. W., Armelagos, J. G. and Mensforth, R. P. 1977; The role of diet, disease and physiology in the origin of porotic hyperostosis. *Human Biology,* Vol. 49:471-483.

Lampl, M. and Jeanty, P. 2003; Timing is everything: a reconsideration of fetal growth velocity patterns identifies the importance of individual and sex differences. *American Journal of Human Biology,* Vol. 15: 667–680.

Lampl, M. and Johnston, F. E. 1996; Problems in the ageing of skeletal juveniles: Perspectives from maturation assessments of living children. *American Journal of Physical Anthropology,* Vol. 101 (3): 345-355.

Lampl, M., Veldhuis, J. D. and Johnston, M. L. 1992; Salutation and stasis: a model of human growth. *Science,* Vol. 258: 801–803.

Langley, K., Heron, J., Smith, G. D., and Thapar, A. 2012; Maternal and paternal smoking during pregnancy and risk of ADHD symptoms in offspring: Testing for intrauterine effects. *American Journal of Epidemiology*, Vol. 176: 261-268.

Larsen, C. S. 2015; *Bioarchaeology: Interpreting Behaviour from the Human Skeleton* (Second Edition). Cambridge: Cambridge University Press.

Leach, S., Lewis, M., Chenery, C., Müldner, G. and Eckardt, H. 2009; Migration and Diversity in Roman Britain: A Multidisciplinary Approach to the Identification of Immigrants in Roman York, England. *American Journal of Physical Anthropology,* Vol. 140: 546-561.

Lejarraga, H. 2012; Growth in Infancy and Childhood: A Pediatric Approach. In N. Cameron & B. Bogin (Eds.) *Human Growth and Development.* London: Elsevier: 23-56.

Lewis M. E. 2000; Non-adult palaeopathology: current status and future potential. In M. Cox and S. Mays (Eds.) *Human Osteology in Archaeology and Forensic Science*. London: Greenwich Medical Media Ltd: 39–57.

Lewis, M. E. 2002a; The impact of industrialisation: comparative study of child health in four sites from medieval and post-medieval England (AD 850–1859). *American Journal of Physical Anthropology*, Vol. 119: 211–223.

Lewis, M. E. 2002b; *Urbanisation and Child Health in Medieval and Post-Medieval England.* British Archaeological Reports British Series 229. Oxford: Archaeopress.

Lewis, M. E. 2004; Endocranial Lesions in Non-Adult Skeletons: Understanding their Aetiology. *International Journal of Osteoarchaeology,* Vol. 14: 82-97.

Lewis, M. E. 2007; *The Bioarchaeology of Children: Perspectives from Biological and Forensic Anthropology.* Cambridge: Cambridge University Press.

Lewis, M. E. 2010; Life and Death in a Civitas Capital: Metabolic Disease and Trauma in the Children from Late Roman Dorchester, Dorset. *American Journal of Physical Anthropology,* Vol. 142: 405-416.

Lewis, M. E. 2011; Tuberculosis in the non-adults from Romano-British Poundbury Camp, Dorset, England. *International Journal of Palaeopathology,* Vol. 1: 12-23.

Lewis, M. E. 2012; Thalassaemia: Its diagnosis and interpretation in past skeletal populations. *International Journal of Osteoarchaeology,* Vol. 22: 685-693.

Lewis, M. E. 2017a; *Paleopathology of Children: Identification of Pathological Conditions in the Human Skeletal Remains of Non-Adults.* London: Academic Press.

Lewis, M. E. 2017b; Childcare in the Past: The Contribution of Palaeopathology. In L. Powell, W. Southwell-Wright, and R. Gowland (Eds.) *Care in the Past: Archaeological and Interdisciplinary Perspectives.* Oxford: Oxbow Books: 23-37.

Lewis, M. E. 2018; Fetal Paleopathology: An Impossible Discipline? In S. Han, T. K. Betsinger, and A. B. Scott (Eds.) *The Fetus: Biology, Culture, and Society.* New York: Berghahn Books: 112-131.

Lewis, M. E. and Gowland, R. L. 2007; Brief and Precarious Lives: Infant Mortality in Contrasting Sites from Medieval and Post-Medieval England (AD 850-1859). *American Journal of Physical Anthropology,* Vol. 134: 117-129.

Lewis, M. E. and Roberts, C. 1997; Growing Pains: the interpretation of Stress Indicators. *International Journal of Osteoarchaeology,* Vol. 7: 581-586.

Lillehammer, G. 1989; A child is born: the child's world in an archaeological perspective. *Norwegian Archaeological Review,* Vol. 22: 89-105.

Lillehammer, G. 2000; The world of Children. In J. R. Sofaer Derevenski (Ed.) *Children and Material Culture*. London: Routledge: 17-26.

Lillehammer, G. 2015; 25 Years with the 'Child' and the Archaeology of Childhood. *Childhood in the Past*, Vol.8 (2): 78-86.

Lindert, P. 1994; Unequal living standards. In R. Floud and D. McCloskey (Eds.) *The economic history of Britain since 1700*. Cambridge: Cambridge University Press: 357–386.

Liversidge, H. M. and Molleson, T. 2004; Variation in Crown and Root Formation and Eruption of Human Deciduous Teeth. *American Journal of Physical Anthropology*, Vol. 123: 172-180.

Lock, M. M. 1993; Cultivating the Body: Anthropology and Epistemologies of Bodily Practice and Knowledge. *Annual Review of Anthropology*, Vol. 22: 133-155.

Looney, C. B., Smith, J. K., Merck, L. H., Wolfe, H. M., Chescheir, N. C., Hamer, R. M. and Gilmore, J. H. 2007; Intracranial hemorrhage in asymptomatic neonates: prevalence on MR images and relationship to obstetric and neonatal risk factors. *Radiology*, Vol. 242 (2): 535-541.

Loth, S. R. 1996; Sexual Dimorphism in the human mandible: an evolutionary and developmental perspective. Ph.D. Thesis, University of Witwatersrand.

Loth, S. R. and Henneberg, M. 2001; Sexually Dimorphic Mandibular Morphology in the First Few Years of Life. *American Journal of Physical Anthropology*, Vol. 115: 179-186.

Lubchenco, L. O., Hansman, C. and Boyd, E. 1966; Intrauterine growth in length and head circumference as estimated from live births at gestational ages from 26 to 42 weeks. *Pediatrics*, Vol. 37 (3): 403-408.

Lucy, S. 2005; The archaeology of age. In M. Díaz-Andreu, S. Lucy, S. Babić and D. N. Edwards (Eds.) *The Archaeology of Identity. Approaches to Gender, Age, Status, Ethnicity and Religion*. Oxford: Routledge: 43-66.

Luo, Z. C., Fraser, W. D., Julien, P., Deal, C. L., Audibert, F., Smith, G. N., Xiong, X. and Walker, M. 2006; Tracing the origins of 'fetal origins' of adult diseases: Programming by oxidative stress? *Medical Hypotheses,* Vol. 66: 38-44.

Luo, Z. C., Xiao, L. and Nuyt, A. M. 2010; Mechanisms of developmental programming of the metabolic syndrome and related disorders. *World Journal of Diabetes,* Vol. 1 (3): 89-98.

Maat, G. J. R. 1984; Dating and rating of Harris lines. *American Journal of Physical Anthropology,* Vol. 63**:** 291-299.

Macchiarelli, R., Bondioli, L., Censi, L., Kristoff Hernaez, M., Salvadei, L. and Sperduti, A. 1994; Intra- and inter-observer concordance in scoring Harris lines: a test on bone sections and radiographs. *American Journal of Physical Anthropology,* Vol. 95: 77-83.

Magennis, A. L. 1990; Growth velocity as a factor influencing the formation of transverse lines. *American Journal of Physical Anthropology,* Vol. 81: 262.

Mahon, P., Harvey, N., Crozier, S., Inskip, H., Robinson, S., Arden, N., Swaminathan, R., Cooper, C., The SWS Study Group, and Godfrey, K. 2010; Low maternal vitamin D status and fetal bone development: cohort study. *Journal of Bone and Mineral Research,* Vol. 25 (1): 14-19.

Malaspina, D., Corcoran, C., Kleinhaus, K., Perrin, M., Fennig, S., Nahon, D., Friedlander, Y., and Harlap, S. 2008; Acute maternal stress in pregnancy and schizophrenia in offspring: a cohort prospective study. *BMC Psychiatry,* Vol. 8: 71.

Malina, R. M., Brown, K. H. and Zavaleta, A. N. 1987; Relative Lower Extremity Length in Mexican American and in American Black and White Youth. *American Journal of Physical Anthropology,* Vol. 72: 89-94.

Mamluk, L., Edwards, H.B., Savović, J., Leach, V., Jones, T., Moore, T. H. M., Ijaz, S., Lewis, S. J., Donovan, J. L., Lawlor, D., Davey Smith, G., Fraser, A., and Zuccolo, L. 2017; Low alcohol consumption and pregnancy and childhood outcomes: time to change guidelines

indicating apparently ‘safe’ levels of alcohol during pregnancy? A systematic review and meta-analyses. *BMJ Open,* Vol. 7 (7) [Early View].

Maresh, M. M. 1943; Growth of major long bones in healthy children. *American Journal of Diseases of Children,* Vol. 66: 227-257.

Maresh, M. M. 1955; Linear growth of long bones of extremities from infancy through adolescence. *American Journal of Diseases of Children,* Vol. 89: 725-742.

Maresh, M. M. 1970; Measurements from roentgenograms. In R. W. McCammon (Ed.) *Human Growth and Development.* Springfield: C. C. Thomas: 157-200.

Maresh, M. M. and Deming, J. 1939; The Growth of Long Bones in 80 Infants: Roentgenograms Versus Anthropometry. *Child Development,* Vol. 10 (2): 91-106.

Marmot, M. 2005; Social determinants of health inequalities. *Lancet,* Vol. 365: 1099-1104.

Márquez-Grant and Errickson 2017; Ethical Considerations: An Added Dimension. In D. Errickson and T. Thompson (Eds.) *Human Remains: Another Dimension: The Application of Imaging to the Study of Human Remains.* London: Elsevier: 193-204.

Martin, D. L., Harrod, R. P. and Pérez, V. R. 2013; *Bioarchaeology: An Integrated Approach to Working with Human Remains*. New York: Springer.

Martorell, R. and Habicht. J. P. 1986; Growth in early childhood in developing countries. In F. Falkner and J. Tanner (Eds.) *Human Growth: methodology ecological, genetic, and nutritional effects on growth*. New York: Plenum Press: 241-262.

Martorell, R., Malina, R. M., Castillo, R. O., Mendoza, F. S. and Pawson, I. G. 1988; Body Proportions in Three Ethnic Groups: Children and Youths 2-17 Years in NHANES II and HHANES. *Human Biology,* Vol. 60 (2): 205-222.

Massler, M., Schour, I. and Poncher, H. G. 1941; Developmental Pattern of the Child as Reflected in the Calcification Pattern of the Teeth. *American Journal of Diseases of Children,* Vol. 62: 33-67.

Mattingly, D. 2006; *An imperial possession: Britain in the Roman Empire*. London: Penguin Books Ltd.

Mayhew, H. 1985; *London labour and the London poor. Selections made and introduced by Neuberg, V.* London: Penguin.

Mays, S. 1993; Infanticide in Roman Britain. *Antiquity* 67: 883–8.

Mays, S. 1995; The Relationship between Harris Lines and other Aspects of Skeletal Development in Adults and Juveniles. *Journal of Archaeological Science,* Vol. 22: 511-520.

Mays, S. 1998; *The Archaeology of Human Bones*. London: Routledge.

Mays, S. 1999; Linear and appositional long bone growth in earlier human populations: a case study from Mediaeval England. In D. Hoppa and C. M. Fitzgerald (Eds.) *Human growth in the past: studies from bones and teeth*. Cambridge: Cambridge University Press: 290-312.

Mays, S. 2014; The palaeopathology of scurvy in Europe. *International Journal of Paleopathology,* Vol. 5: 55-62.

Mays, S., Brickley, M. and Ives, R. 2007; Skeletal Evidence for Hyperparathyroidism in a 19th Century Child with Rickets. *International Journal of Osteoarchaeology,* Vol. 17: 73-81.

Mays, S. and Eyers, J. 2011. Perinatal infant death at the Roman villa site at Hambleden, Buckinghamshire, England. *Journal of Archaeological Science* 38, 1931-38.

Mays, S. & Faerman, M. 2001; Sex identification of some putative infanticide victims from Roman Britain using ancient DNA. *Journal of Archaeological Science,* Vol. 28: 555-559.

Mays, S., Gowland, R., Halcrow, S. and Murphy, E. 2017; Child Bioarchaeology: Perspectives on the Past 10 Years. *Childhood in the Past*, Vol. 10 (1): 38-56.

Mays, S., Ives, R. and Brickley, M. 2009; The Effects of Socioeconomic Status on Endochondral and Appositional Bone Growth, and Acquisition of Cortical Bone in Children from 19th Century Birmingham, England. *American Journal of Physical Anthropology*, Vol. 140: 410-416.

MBRRACE-UK Maternal Death 2016 *Lay Summary* [Online] [Accessed: July 2017] Available from: https://www.npeu.ox.ac.uk/downloads/files/mbrrace-uk/reports/MBRRACE-UK%20Intrapartum%20Confidential%20Enquiry%202017%20Lay%20summary%20FINAL.pdf

McDade, T. W. 2005; Life History, Maintenance, and the Early Origins of immune Function. *American Journal of Human Biology*, Vol. 17: 81-94.

Melby, M. K., Yamada, G. and Surkan, P. J. 2016; Inadequate Gestational Weight Gain Increases Risk of Small-for-Gestational-Age Term Births in Girls in Japan: A Population-Based Cohort Study. *American Journal of Human Biology*, Vol. 28: 714-720.

Mensforth, R. P., Lovejoy, O. C., Lallo, J.W. and Armelagos, G. J. 1978; The role of constitutional factors, diet and infectious disease in the etiology of porotic hyperostosis and periosteal reactions in prehistoric infants and children. *Medical Anthropology*, Vol. 2:1–59.

Meskell, L. 2001; Archaeologies of Identity. In I. Hodder (Ed.) *Archaeological Theory Today*. Cambridge: Polity: 187-213.

Messer, L. B. and Till, M. J. 2013; A landmark report on understanding the human dentition. *Journal of the American Dental Association*, Vol.144: 357–361.

Mikulski, R. 2007 *Cross Bones burial ground summary* [Online] [Accessed: November 2016] Available from:

https://www.museumoflondon.org.uk/collections/other-collection-databases-and-libraries/centre-human-bioarchaeology/osteological-database/post-medieval-cemeteries/cross-bones-post-medieval

Miles, A. and Conheeney, J. 2005; *A Post-medieval population from London: Excavations in the St Bride's Lower Churchyard 75-82 Farringdon Street, City of London*. London (Unpublished).

Miles, A. White, W. and Tankard, D. 2008; *Burial at the site of the parish church of St Benet Sherehog before and after the Great Fire: Excavations at 1 Poultry, City of London* (MOLAS Monograph 39). London: Museum of London Archaeology Service.

Miles, D. 1986; *Archaeology at Barton Court Farm, Abingdon, Oxfordshire*. Oxford Archaeological Unit Report 3, CBA Research Report 50. Oxford: Oxford Archaeological Unit.

Millard, A. R. 2001; The deterioration of bone. In D. R. Brothwell and A. M. Pollard (Eds.) *Handbook of Archaeological Sciences.* Chichester, New York: Wiley: 637-645.

Miller, E. M. 2018; The first Seriatum study of growth by R. E. Scammon. *American Journal of Physical Anthropology,* Vol. 165 (3): 415-420.

Miller, Z. E. S. 2010; The Infant Burials from Piddington. In R. M. Friendship-Taylor & D. E. Friendship-Taylor (Eds.), *Iron Age and Roman Piddington: Iron Age, Roman and Anglo Saxon Human Burials and Recent Research 1979-2010.* The Upper Nene Archaeological Society 2010, Fascicule 7: 1-31.

Millet, M & Gowland, R. 2015; Infant and Child Burial Rites in Roman Britain: A Study from East Yorkshire. *Britannia,* Vol. 46: 171-189.

Mittler, D. and Sheridan, S. 1992; Sex determination in subadults using auricular surface morphology: a forensic science perspective. *Journal of Forensic Sciences,* Vol. 37:1068-1075.

Molleson, T. 1989; Social implications of mortality patterns of juveniles from Poundbury Camp, Romano-British cemetery. *Anthropologischer Anzeiger,* Vol. 47 (1): 27-38.

Molleson, T. I. 1990; The children from Christ Church crypt, Spitalfields. *American Journal of Physical Anthropology,* Vol. 81 (2): 271.

Molleson, T. 1992; The anthropological evidence for change through Romanisation of the Poundbury population. *Anthropologischer Anzeiger,* Vol. 50 (3): 179-189.

Molleson, T. I. and Cox, M. 1993; *The Spitalfields Project, Vol. 2 The Anthropology: The Middling Sort*. CBA Research Report 86. Council for British Archaeology.

Moore, A. 2009. Hearth and home: the burial of infants within Romano-British domestic contexts. *Childhood in the Past,* Vol. 2: 33-54.

Moon, R. Y. and Fu, L. 2012; Sudden Infant Death Syndrome: An Update. *Pediatrics in Review,* Vol. 33: 314-320.

Moore, J. and Scott, E. C. 1997; *Invisible People and Processes: Writing Gender and Childhood into European Archaeology*. London: Leicester University Press.

Moorrees, C. F. A. Fanning, E. A. & Hunt, E. E. 1963a; Formation and Resorption of Three Deciduous Teeth in Children. *American Journal of Physical Anthropology,* Vol. 21: 205-213.

Moorrees, C. F. A. Fanning, E. A. and Hunt, E. E. 1963b; Age Variation of Formation Stages for Ten Permanent Teeth. *Journal of Dental Research,* Vol. 42: 1490-1502.

Mortier, G. R. and Vanden Berghe, W. 2012; Genomics, Epigenetics and Growth. In N. Cameron and B. Bogin (Eds.) *Human Growth and Development* (2nd Edition). London: Elsevier: 153-172.

Murgatroyd, C. and Spengler, D. 2011; Epigenetics of early child development. *Frontiers in Psychiatry,* Vol. 2 (16): 1-15.

Murphy, E. M. 1996; A Possible Case of Hydrocephalus in a Medieval Child from Doonbought Fort, Co. Antrim, Northern Ireland. *International Journal of Osteoarchaeology,* Vol.6: 435-442.

Murphy, E. M. 2017; Ten years of '*Childhood in the Past'. Childhood in the Past,* Vol. 10 (1): 1-9.

Museum of London 2009 *Chelsea Old Church (Post-Medieval) cemetery summary* [Online] [Accessed: November 2016] Available from: https://www.museumoflondon.org.uk/collections/other-collection-databases-and-libraries/centre-human-bioarchaeology/osteological-database/post-medieval-cemeteries/chelsea-old-church-post-medieval

Museum of London 2015 *Broadgate (Post-Medieval) cemetery summary* [Online] [Accessed: November 2016] Available from: https://www.museumoflondon.org.uk/collections/other-collection-databases-and-libraries/centre-human-bioarchaeology/osteological-database/post-medieval-cemeteries/broadgate-post-medieval

Museum of London *Wellcome Osteological Research Database* [Online] [Accessed July 2017] Available from: https://www.museumoflondon.org.uk/collections/other-collection-databases-and-libraries/centre-human-bioarchaeology/osteological-database

Nade, S. 1983; Acute Haematogenous Osteomyelitis in Infancy and Childhood. *Journal of Bone and Joint Surgery,* Vol. 65B (2): 109-119.

NHS *Sudden Infant Death Syndrome (SIDS)* [Online] [Accessed March 2018] Available from: https://www.nhs.uk/conditions/sudden-infant-death-syndrome-sids/

Niederhofer, H., and Reiter, A. 2004; Prenatal maternal stress, prenatal fetal movements and perinatal temperament factors influence behavior and school marks at the age of 6 years. *Fetal Diagnosis and Therapy*, Vol. 19 (2): 160–162.

Newman, S. L. and Gowland, R. L. 2015; Brief Communication: The Use of Non-Adult Vertebral Dimensions as Indicators of Growth Disruption and Non-Specific Health Stress in Skeletal Populations. *American Journal of Physical Anthropology,* Vol. 158: 155-164.

Newman, S. L. and Gowland, R. L. 2017; Dedicated Followers of Fashion? Bioarchaeological Perspectives on Socio-Economic Status, Inequality, and Health in Urban Children from the Industrial Revolution (18th-19th Century) England. *International Journal of Osteoarchaeology,* Vol. 27 (2): 217-229.

Nicholas, S. and Steckel, R. H. 1991; Heights and living standards of English workers during the early years of industrialization, 1770–1815. *Journal of Economic History*, Vol. 51: 937–957.

Nilsson Stutz, L. N. and Tarlow, S. 2013; Beautiful Things and Bones of Desire: Emerging issues in the archaeology of death and burial. In S. Tarlow and L. Nilsson Stutz (Eds.) *The Oxford Handbook of the Archaeology of Death and Burial.* Oxford: Oxford University Press.

Nitsch, E.K., Humphrey, L.T. and Hedges, R. E. M. 2011: Using stable isotope analysis to examine the effect of economic change on breastfeeding practices in Spitalfields, London, UK. *American Journal Physical Anthropology*, Vol. 146: 619-628.

Non, A. L., Hollister, B. M., Humphreys, K. L., Childebayeva, A., Esteves, K., Zeanah, C. H., Fox, N. A., Nelson, C. A. and Drury, S. S. 2016; DNA Methylation at Stress-Related Genes in Associated with Exposure to Early Life Institutionalization. *American Journal of Physical Anthropology,* Vol. 161: 84-93.

Nyati, L. H., Norris, S. A., Cameron, N. and Pettifor, J. M. 2006; Effect of Ethnicity and Sex on the Growth of the Axial and Appendicular Skeleton of Children Living in a Developing Country. *American Journal of Physical Anthropology,* Vol. 130: 135-141.

Nystrom, P. and Mahoney Swales, D. [No Date]; *Report on the Immature Inhumations at Owslebury.* Unpublished.

O'Brien, G. D. and Queenan, J. T. 1981; Growth of the ultrasound fetal femur length during normal pregnancy. *American Journal of Obstetrics and Gynaecology*: 833-837.

O'Driscoll, K., Meagher, D., Macdonald, D., and Geoghegan, F. 1981; Traumatic intracranial haemorrhage in firstborn infants and delivery with obstetric forceps. *BJOG: International Journal of Obstetrics and Gynaecology*, Vol. 88 (6): 577-581.

Oberlander, T. F., Weinberg, J., Papsdorf, M., Grunau, R., Misri, S. and Devlin, A. M. 2008; Prenatal exposure to maternal depression, neonatal methylation of human glucocorticoid receptor gene (NR3C1) and infant cortisol stress responses. *Epigenetics*, Vol. 3 (2): 97-106.

Oestreich, A. E. 2008; *Growth of the Pediatric Skeleton: A Primer for Radiologists.* New York: Springer.

Ogden, A. R., Pinhasi, R. and White, W. J. 2007; Gross enamel hypoplasia in molars from subadults in a 16th-18th century London graveyard. *American Journal of Physical Anthropology*, Vol. 133: 957-966.

Ojha, S. and Budge, H. 2017; Early Origins of Obesity and Developmental Regulation of Adiposity. In M. Symonds (Ed.) *Adipose Tissue Biology.* New York: Springer: 379-408.

Ong, K. K., Kennedy, K., Castañeda-Gutiérrez, E., Forsyth, S., Godfrey, K. M., Koletzko, B., Latulippe, M. E., Ozanne, S. E., Rueda, R., Schoemaker, M. H., van der Beek, E. M., van Buuren, S. and Fewtrell, M. 2015; Postnatal growth in preterm infants and later health outcomes: a systemic review. *Acta Paediatrica*, Vol. 104: 974-986.

Ortner, D. J. 2003; Identification of Pathological Conditions in Human Skeleton Remains. San Diego: Elsevier.

Ortner, D. J. 2006; Foreword. In J. E. Buikstra and L. A. Beck (Eds.) *Bioarchaeology: The Contextual Analysis of Human Remains*. London: Elsevier Inc.

Ortner, D. J. 2008; Differential diagnosis of skeletal lesions in infectious disease. In R. Pinhasi and S. Mays (Eds.) *Advances in Human Palaeopathology.* Chichester: Wiley and Sons: 191-214.

Ortner, D. J. and Ericksen, M. F. 1997; Bone Changes in the Human Skull Probably Resulting from Scurvy in Infancy and Childhood. *International Journal of Osteoarchaeology,* Vol. 7: 212-220.

Ortner, D. J. Kimmerle, E. H. & Diez, M. 1999; Probable Evidence of Scurvy in Subadults from Archaeological Sites in Peru. *American Journal of Physical Anthropology,* Vol. 108: 321-331.

Ortner, D. J. and Mays, S. 1998; Dry-bone Manifestations of Rickets in Infancy and Early Childhood. *International Journal of Osteoarchaeology,* Vol. 8: 45-55.

Oxenham, M.F. and Cavill, I 2010; Porotic hyperostosis and *cribra orbitalia*: the erythropoietic response to iron-deficiency anemia. *Anthropological Science,* Vol. 118: 199-200.

Oxford English Dictionary Online 2017: 'well-being, noun.' [Online] [Accessed August 2017] Available from: http://www.oed.com.ezphost.dur.ac.uk/view/Entry/227050?redirectedFrom=well+being&

Owsley, D. W. and Jantz, R. L. 1985; Long Bone Lengths and Gestational Age Distributions of Post-Contact Period Arikara Indian Perinatal Infant Skeletons. *American Journal of Pysical Anthropology,* Vol. 68 (3): 321-328.

Paine, R. R. and Harpending, H. C. 1996; Assessing the reliability of paleodemographic fertility estimators using simulated skeletal distributions. *American Journal of Physical Anthropology,* Vol. 101 (2): 151-159.

Paine, R. R. and Harpending, H. C. 1998; Effect of Sample Bias on Paleodemographic Fertility Estimates. *American Journal of Physical Anthropology,* Vol. 105: 231-240.

Papageorgopoulou, C., Sutter, S. K., Rühli, F. J. and Siegmund, F. 2011; Harris lines revisited: prevalence, comorbidities, and possible etiologies. *American Journal of Human Biology*, Vol. 23: 381-391.

Pearce, J. 2001; Infants, cemeteries and communities in the Roman provinces. In D. Davis, A. Gardner and K. Lockyear (Eds.) *TRAC 2000.* Oxford: Oxbow: 125-142.

Perini, T. A., de Oliveira, G. L., dos Santos Ornellas J., and de Oliveira, F. P. 2005; Technical error of measurement in anthropometry. *Review of Brasileira de Medicina do Esporte,* Vol. 11 (1): 86-90.

Perry, M. A. 2006; Redefining childhood through bioarchaeology: Toward an archaeological and biological understanding of children in Antiquity. *Archaeological Papers of the American Anthropological Association,* Vol. 15 (1): 89-111.

Phelan, J. C., Link, B. G. and Tehranifar, P. 2010; Social conditions as fundamental causes of health inequalities: theory, evidence, and policy implications. *Journal of Health and Social Behaviour*, Vol. 51: S28–S40.

Phillips, T. 2008; The Role of Methylation in Gene Expression. *Nature Education*, Vol. 1 (1): 116.

Picard, L. 2005; Victorian London. London: Orion Books.

Pinhasi, R., Shaw, P., White, B. and Ogden, A. R. 2006; Morbidity, rickets and long-bone growth in post-medieval Britain - a cross-population analysis. *Annals of Human Biology,* Vol. 33: 372-398.

Pitts, M. 2008; Globalizing the local in Roman Britain: An anthropological approach to social change. *Journal of Anthropological Archaeology,* Vol. 27: 493-506.

Pitts, M. and Griffin, R. 2012; Exploring Health and Social Well-Being in Late Roman Britain: An Intercemetery Approach. *American Journal of Archaeology,* Vol. 116 (No. 2): 253-276.

Plana-Ripoll, O., Li, J., Kesmodel, U. S., Olsen, J., Parner, E. and Basso, O. 2016; Maternal stress before and during pregnancy and subsequent infertility in daughters: a nationwide population-based cohort study. *Human Reproduction,* Vol. 31 (2): 454-462.

Pollock, l. A. 1983; *Forgotten Children: Parent-Child Relations from 1500 to 1900.* Cambridge: Cambridge University Press.

Pomeroy, E., Stock, J. T., Stanojevic, S., Miranda, J. J., Cole, T. J., and Wells, J. C. K. 2012; Trade-Offs in Relative Limb Length among Peruvian Children: Extending the Thrifty Phenotype Hypothesis to Limb Proportions. *Plos One¸* Vol. 7 (12): 1-10.

Prentice, A. 2003; Pregnancy and Lactation. In F. H. Glorieux, J. M. Pettifor and H. Jüppner (Eds.) *Pediatric Bone: Biology and Diseases*. London: Academic Press: 249-269.

Prout, A. 2005; *The Future of Childhood: Towards the Interdisciplinary Study of Children*. Oxon: RoutledgeFalmer.

Ramji, S. 2009; Impact of infant and young child feeding and caring practices on nutritional status and health. Indian Journal of Medical Research, Vol. 130: 624-626.

Rana, R. S., Wu, J. S. and Eisenberg, R. L. 2009; Periosteal Reaction. *American Journal of Radiology,* Vol. 193: 259-272.

Rasool, M. 2001; Primary subacute haematogenous osteomyelitis in children. *The Journal of Bone and Joint Surgery,* Vol. 83 (1): 93-98.

Rawson, B. 1986; Children in the Roman *Familia.* In B. Rawson (Ed.) *The Family in Ancient Rome: New Perspectives.* New York: Cornell University Press: 170-200.

Rawson, B. 2003; *Children and Childhood in Roman Italy.* Oxford: Oxford university Press.

Redfern, R. 2003; Sex and the City: A biocultural investigation into female health in Roman Britain. In G. Carr, E. Swift and J Weekes (Eds.) *TRAC 2002 Proceedings of the Twelfth Annual Theoretical Roman Archaeology Conference.* Oxford: Oxbow Books.

Redfern, R. 2007; The influence of culture upon childhood: an osteological study of iron Age and Romano-British Dorset. In M. Harlow and R. Laurence (Eds.) *Age and Ageing in the Roman Empire.* Portsmouth, Rhode Island: Journal of Roman Archaeology: 171-194.

Redfern, R. 2008; A Bioarchaeological investigation of Cultural Change in Dorset, England (Mid-to-Late Fourth Century B.C. to the End of the Fourth Century A.D.). *Britannia,* Vol. 39: 161-192.

Redfern, R. C. and DeWitte, S. N. 2011; A New Approach to the Study of Romanization in Britain: A Regional Perspective of Cultural Change in Late Iron Age and Roman Dorset Using the Siler and Gompertz-Makeham Models of Mortality. *American Journal of Physical Anthropology,* Vol. 144: 269-285.

Redfern, R. C., DeWitte, S. N., Pearce, J., Hamlin, C. and Egging Dinwiddy, K. 2015; Urban-Rural Difference in Roman Dorset, England: A Bioarchaeological Perspective on Roman Settlements. *American Journal of Physical Anthropology,* Vol. 157: 107-120.

Redfern, R. C. Millard, A. R. and Hamlin, C. 2012; A regional investigation of subadult dietary patterns and health in late Iron Age and Roman Dorset, England. *Journal of Archaeological Science,* Vol. 39: 1249-1259.

Redfern, R. C. and Roberts, C. A. 2005; Health in Romano-British urban communities: Reflections from the cemeteries. In D. N. Smith, M. B. Brickley and W. Smith (Eds.) *Fertile ground: Papers in honour of Susan Limbey.* Oxford: Oxbow Books: 115-129.

Redfield, A. 1970; A New Aid to Aging Immature Skeletons: Development of the Occipital Bone. *American Journal of Physical Anthropology,* Vol. 33: 207-220.

Reid, A. 1990; Death before Birth: Fetal Health and Mortality in Historical Perspective (Review). *Journal of Interdisciplinary History,* Vol. 41 (4): 621-623.

Reitsema, L.J. and McIlvaine, B. K. 2014; Reconciling “stress” and “health” in physical anthropology: What can bioarchaeologists learn from the other subdisciplines? *American Journal of Physical Anthropology,* Vol. 155: 181-185.

Resnick, P. J. 1970; Murder of the Newborn: A Psychiatric Review of Neonaticide. *American Journal of Psychiatry,* Vol. 126, No. 10: 1414- 1421.

Ribot, I. and Roberts, C. A. 1996; A Study of Non-Specific Stress Indicators and Skeletal Growth in Two Mediaeval Subadult Populations. *Journal of Archaeological Science,* Vol. 23 (1): 67-79.

Richards, G. D. and Anton, S. C. 1991; Craniofacial Configuration and Postcranial Development of a Hydrocephalic Child (ca. 2500 B. C. – 500 A. D.): With a Review of Cases and Comment on Diagnostic Criteria. *American Journal of Physical Anthropology,* Vol. 85: 185-200.

Rivera, A. C. and Miller, E. M. 2017; Pregnancy and immune stimulation: re-imagining the fetus as a parasite to understand age-related immune system changes in US women. *American Journal of Human Biology* (Early View): 1-6.

Rivera, F. and Lahr, M. M. 2017; New evidence suggesting a dissociated etiology for *cribra orbitalia* and porotic hyperostosis. *American Journal of Physical Anthropology* (Early View): 1-21.

Robb, J. 2002; Time and biography: Osteobiography of the Italian Neolithic lifespan. In Y. Hamilakis, M. Pluciennik, and S. Tarlow (Eds.) *Thinking Through the Body: Archaeologies of Corporeality*. London: Kluwer Academic/Plenum:153-171.

Robb, J., Bigazzi, R., Lazzarini, L., Scarsini, C. and Sonego, F. 2001; Social ‘status’ and biological ‘status’: a comparison of grave goods and skeletal indicators from Pontecagnano. *American Journal of Physical Anthropology,* Vol. 115: 213–222.

Robbins, G. 2011; Don't Throw Out the Baby with the Bathwater: Estimating Fertility from Subadult Skeletons. *International Journal of Osteoarchaeology,* Vol. 21: 717-722.

Roberts, C. A. 2000; Infectious disease in biocultural perspective: Past, present, and future work in Britain. In: M. Cox and S. Mays (Eds.) *Human Osteology in Archaeology and Forensic Science*. New York: Greenwich Medical Media:145-162.

Roberts, C. A. 2006; A View from Afar: Bioarchaeology in Britain. In J. E. Buikstra and L. A. Beck (Eds.) *Bioarchaeology: The Contextual Analysis of Human Remains*. Amsterdam, Boston: Academic Press: 417-439.

Roberts, C. A. 2009; *Human Remains in Archaeology*. York: Council for British Archaeology.

Roberts, C. 2017; Navigating Approaches to Impairment, "Disability" and Care in the Past: The Need for Reflection. In L. Powell, W. Southwell-Wright, and R. Gowland (Eds.) *Care in the Past: Archaeological and Interdisciplinary Perspectives.* Oxford: Oxbow Books: xi-xviii.

Roberts, C.A. and Cox, M. 2003; *Health & disease in Britain: from prehistory to the present day*. Gloucester: Sutton Publishing.

Roberts, C. and Manchester, K. 2010; *The Archaeology of Disease.* Stroud: The History Press.

Robertson, T., Batty, G. D., Der, G., Fenton, C., Shiels, P. G. and Benzeval, M. 2013; Is socioeconomic status associated with biological aging as measured by Telomere Length? *Epidemiological Review*, Vol. 35: 98–111.

Rogers, A. 1997; Vulnerability, health and healthcare. *Journal of Advanced Nursing,* Vol. 26: 65-72.

Rogers, T. L. 2009; Sex Determination of Adolescent Skeletons Using the Distal Humerus. *American Journal of Physical Anthropology,* Vol. 140: 143-148.

Rogne, T., Tielemans, M. J., Foong-Fong Chong, M., Yajnik, C. S., Krishnaveni, G. V., Poston, L., Jaddoe, V. W. V., Steegers, E. A. P., Joshi, S., Chong, Y-S., Godfrey, K. M., Yap, F., Yahyaoui, R., Thomas, T., Hay, G., Hogeveen, M., Demir, A., Saravanan, P., Skovlund, E., Martinussen, M. P., Jacobsen, G. W., Franco, O. H., Bracken, M. B., and Risnes, K. R. 2017; Associations of Maternal Vitamin B12 Concentration in Pregnancy With the Risks of Preterm Birth and Low Birth Weight: A Systematic Review and Meta-Analysis of Individual Participant Data, *American Journal of Epidemiology*, Vol. 185 (3): 212–223.

Rohnbogner, A. and Lewis, M. E. 2017; Poundbury Camp in Context – a new Perspective on the Lives of Children from urban and rural Roman England. *American Journal of Physical Anthropology,* Vol. 162 (2): 208-228.

Roseboom, T., Meulen, J. V. D., Osmond, C., Barker, D. J. P., Ravelli, A., Schroeder-Tanka, J., Montfrans, G., Michels, R., and Bleker, O. 2000; Coronary heart disease after prenatal exposure to the Dutch famine, 1944–45. *Heart*, Vol. 84: 595–598.

Roseboom, T. J., van der Meulen, J. H. P. and Ravelli, A. C. J. 2001; Effects of prenatal exposure to the Dutch famine on adult disease in later life: an overview. *Molecular and Cellular Endocrinology*, Vol. 185: 93–98.

Roth, E. A. 1992; Applications of demography models to paleodemography. In S. R. Saunders and M. A. Katzenberg (Eds.) *Skeletal Biology of Past Peoples: Research Methods*. New York: Wiley-Liss: 175–188.

Royal College of Obstetricians & Gynaecologists 2014; *The Investigation and Management of the Small-for-Gestational-Age Fetus,* Green-top Guideline No. 31 (2nd Edition). London: Royal College of Obstetricians & Gynaecologists.

Ruff, C. B. Garofalo, E. and Holmes, M. A. 2013: Interpreting Skeletal Growth in the Past From a Functional and Physiological Perspective. *American Journal of Physical Anthropology,* Vol 150: 29-37.

Rumbaugh, C. L. and Potts, D. G. 1966; Skull changes associated with intracranial arteriovenous malformations. *The American Journal of Roentgenology, Radium Therapy and Nuclear Medicine,* Vol. 98 (3): 525-534.

Russett, A. and Pocock, T. 2004; *A History of Chelsea Old Church – The Church that refused to die*. London: Historical Publication Ltd.

Sadler, T. W. 2006; *Langman's Medical Embryology* (10th Edition). Baltimore: Lippincott Williams & Wilkins.

Said-Mohamed, R., Pettifor, J. M. and Norris, S. A. 2018; Life History theory hypotheses on child growth: Potential implications for short and long-term child growth, development and health. *American Journal of Physical Anthropology,* Vol. 165: 4-19.

Sánchez Romero, M. 2017; Landscapes of Childhood: Bodies, Places and Material Culture. *Childhood in the Past,* Vol 10. (1): 16-37.

Sandman, C. A., Glynn, L. M. and Davis, E. P. 2016; Neurobehavioral Consequences of Fetal Exposure to Gestational Stress. In N. Reissland and B. S. Kisilevsky (Eds.) *Fetal Development.* Switzerland: Springer International Publishing: 229-265.

Satterlee Blake, K. A. 2018; The Biology of the Fetal Period: Interpreting Life from Fetal Skeletal Remains. In S. Han, T. K. Betsinger, and A. B. Scott (Eds.) *The Fetus: Biology, Culture, and Society.* New York: Berghahn Books: 34-58.

Saunders, S. R. 1992; Subadult skeletons and growth-related studies. In S.R. Saunders and M.A. Katzenberg (Eds.). *Skeletal Biology of Past Populations: Advances in Research Methods*. New York: Wiley-Liss: 1-20.

Saunders, S. R. 2000; Subadult skeletons and growth-related studies. In M.A. Katzenberg and S.R. Saunders (Eds.). *Biological Anthropology of the Human Skeleton*. New York: Wiley-Liss: 135-162.

Saunders, S. R. and Barrans, L. 1999; What can be done about the infant category in skeletal samples? In R. D. Hoppa and C. M. Fitzgerald (Eds.) *Human growth in the past: studies from bones and teeth*. Cambridge: Cambridge University Press: 183- 209.

Saunders, S. R. and Hoppa, R. D. 1993; Growth Deficit in Survivors and Non-Survivors: Biological Mortality Bias in Subadult Skeletal Samples. *Yearbook of Physical Anthropology,* Vol. 36: 127-151.

Saunders, S., Hoppa, R. and Southern, R. 1993; Diaphyseal Growth in a Nineteenth Century Skeletal Sample of Subadults from St Thomas' Church, Belleville, Ontario. *International Journal of Osteoarchaeology,* Vol. 3: 265-281.

Sayer, D. 2010; Ethics and Burial Archaeology. London: Duckworth & Co. Ltd.

Schaefer, M. Black, S. & Scheuer, L. 2009; *Juvenile Osteology: A Laboratory and Field Manual.* London: Elsevier Inc.

Schell L. M. 1981; Environmental noise and human prenatal growth. *American Journal of Physical Anthropology,* Vol. 56: 63–70.

Scheuer, L. 2002; Brief communication: a blind test of mandibular morphology for sexing mandibles in the first few years of life. *American Journal of Physical Anthropology,* Vol. 119: 189–191.

Scheuer, L. 1998; Age at death and cause of death of the people buried in St Bride's Church, Fleet Street, London. In M. Cox (Ed.) *Grave Concerns: Death and Burial in England 1700-1850.* York: Council for British Archaeology: 100-111.

Scheuer, L. and Black, S. 1995; *The St Bride's Documented Skeletal Collection*. London: Research Report St Bride's Church.

Scheuer, L. and Black, S. 2000a; *Developmental Juvenile Osteology*. London: Academic Press.

Scheuer, L. and Black, S. 2000b; Development and Ageing of the Juvenile Skeleton. In M. Cox and S. Mays (Eds.) *Human Osteology in Archaeology and Forensic Science*. London: Greenwich Medical Media Ltd: 9-21.

Scheuer, L. and Maclaughlin-Black, S. 1994; Age Estimation from the Pars Basilaris of the Fetal and Juvenile Occipital Bone. *International Journal of Osteoarchaeology,* Vol. 4: 377-380.

Scheuer, L. Musgrave, J. H. & Evans, S. P. 1980; The estimation of late fetal and perinatal age from limb bone length by linear and logarithmic regression. *Annals of Human Biology,* 7 (3): 257-265.

Schillaci, M. A., Sachdev, H. P. S. and Bhargava, S. K. 2012; Technical Note: Comparison of the Maresh Reference Data With the WHO International Standard for Normal Growth in Healthy Children. *American Journal of Physical Anthropology,* Vol. 147: 493-498.

Schmit, P. and Glorion, C. 2004; Osteomyelitis in infants and children. *European Radiology,* Vol. 14: 44-54.

Schoenwolf, G. C., Bleyl, S. B., Brauer, P. R. and Francis-West, P. H. 2014; Larsen's Human Embryology (5th Edition). Philadelphia: Elsevier.

Schofield, J. and Maloney, C. 1998; *Archaeology in the City of London, 1907-1991: A guide to records of excavations by the Museum of London and its predecessors***.** London: Museum of London.

Schour, I. and Massler, M. 1941a; The development of the human dentition. *Journal of the American Dental Association*, Vol. 28:1153–1160.

Schour, I. and Massler, M. 1941b; *Development of human dentition chart* (2nd edition). Chicago: American Dental Association.

Schultz, M. 1984; The diseases in a series of children's skeletons from Ikiz Tepe, Turkey. In V. Capecchi and E. Rabino Massa (Eds.) *Proceedings of the 5th European Meeting of the Paleopathology Association, Siena, Italy*. Siena: Tipografia Sienese: 321–325.

Schultz, M. 1989; Causes and frequency of diseases during early childhood in Bronze Age populations. In L. Capasso (Ed.) *Advances in Palaeopathology*. Italy: Marino Solfanelli Editore: 175–179.

Schultz, M. 2001; Paleohistopathology of Bone: A New Approach to the Study of Ancient Diseases. *Yearbook of Physical Anthropology,* Vol. 44: 106-147.

Schutkowski, H. 1993; Sex determination of infant and juvenile skeletons: I. Morphognostic features. *American Journal of Physical Anthropology* 90: 199-205.

Schwartzman, H. B. 2001; Children and Anthropology: A Century of Studies. In H. B. Schwartzman (Ed.) *Children and Anthropology: Perspectives for the 21st Century*. London: Bergin & Garvey: 15-38.

Scientific Advisory Committee on Nutrition 2007. *Update on vitamin D. Position statement by the Scientific Advisory Committee on Nutrition.* The Stationery Office; London: 2007.

Scobie, A. 1986; Slums, Sanitation, and Mortality in the Roman World. *KLIO,* Vol. 68 (2): 390-433.

Scott, E. 1989; Animal and infant Burials in Romano-British Villas: A Revitalization Movement. In P. Garwood, D. Jennings, R. Skeates and J. Toms (Eds.) *Sacred and Profane: Proceedings of a Conference on Archaeology, Ritual and Religion. Oxford 1989.* Oxford: Oxbow Books: 115-121.

Scott, E. C. 1997; On the incompleteness of archaeological narratives. In J. Moore and E. C. Scott (Eds.) *Invisible People and Processes: Writing Gender and Childhood into European Archaeology*. London: Leicester University Press: 1-12.

Scott E. 1999; *The Archaeology of Infancy and Infant Death*. BAR (International Series) 819. Oxford, UK: Archaeopress.

Selkirk, A. 1996; Piddington. *Current Archaeology*, No. 146, Vol. XIII (2): 57-64.

Selye, H. 1973; The evolution of the stress concept. *American Scientist*, Vol. 61: 692-699.

Šešelj, M. 2013; Relationship Between Dental Development and Skeletal Growth in Modern Humans and Its implications for Interpreting Ontogeny in Fossil Hominins. *American Journal of Physical Anthropology*, Vol. 150: 38-47.

Sherwood, R. J., Meindl, R. S., Robinson, H. B. and May, R. L. 2000; Fetal Age: Methods of Estimation and Effects of Pathology. *American Journal of Physical Anthropology*, Vol. 113: 305-315.

Shilling, C. 2003; *The Body and Social Theory*. London: Sage.

Shopfner, C. E. 1966; Periosteal bone growth in normal infants: A preliminary report. *American Journal of Roentgenology*, Vol. 97: 154-163.

Sinclair, D. 1985; *Human Growth After Birth* (4th Edition). Oxford: Oxford University Press.

Slack, J. M. W. 1991; *From Egg to Embryon: Regional Specification in Early Development* (2nd Edition). Cambridge: Cambridge University Press.

Smith, P. and Kahila, G. 1992; Identification of Infanticide in Archaeological Sites: A Case Study from the Late Roman-Early Byzantine Periods at Ashkelon, Israel. *Journal of Archaeological Science*, Vol. 19: 667-675.

Sofaer Derevenski, J. 1994a; Where are the children? Accessing children in the past. *Archaeological Review from Cambridge*, Vol. 13: 7-20.

Sofaer Derevenski, J. 1994b; *Perspectives on Children and Childhood. Archaeological Review from Cambridge.* Cambridge: Cambridge University Press.

Sofaer Derevenski, J. 1997; Engendering children, Engendering Archaeology. In J. Moore and E. C. Scott (Eds.) *Invisible People and Processes: Writing Gender and Childhood into European Archaeology*. London: Leicester University Press: 192-202.

Sofaer, J. 2006; *The Body as Material Culture: A Theoretical Osteoarchaeology*. Cambridge: Cambridge University Press.

Sofaer, J. 2011; Towards a Social Bioarchaeology of Age. In S.C. Agarwal and B.A. Glencross (Eds.) *Social Bioarchaeology*. Oxford: Wiley-Blackwell: 385-311.

St. Jacques, B. and Helms, J. A. 2003; Prenatal Bone Development: Ontogeny and Regulation. F. H. Glorieux, J. M. Pettifor and H. Jüppner (Eds.) *Pediatric Bone: Biology and Diseases.* London: Academic Press: 77-117.

Steckel, R. H. 2009; Heights and human welfare: recent developments and new directions. *Explorations in Economic History*, Vol. 46: 1-23.

Steckel, R. H. and Rose, J. C. 2002; Introduction. In R. H. Steckel and J. C. Rose (Eds.) *The Backbone of History; Health and Nutrition in the Western Hemisphere.* Cambridge: Cambridge University Press: 3-8.

Stevens, D. A. and Williams, G. R. 1999; Hormone regulation of chondrocyte differentiation and endochondral bone formation. *Molecular and Cellular Endocrinology,* Vol. 151: 195-204.

Stinson, S. 2000; Growth variation: biological and cultural factors. In S. Stinson, B. Bogin, R. Huss-Ashmore and D. H. O'Rourke (Eds.) *Human biology: an evolutionary and biocultural perspective*. New York: Wiley-Liss: 434-438.

Stodder, A. L. W. 2008; Taphonomy and the nature of archaeological assemblages. In M. A. Katzenburg and S. R. Saunders (Eds.) *Biological Anthropology of the Human Skeleton*. New York: Wiley-Liss: 71-114.

Stöger, R. 2008; The thrifty epigenotype: An acquired and heritable predisposition for obesity and diabetes? *Bioessays,* Vol. 30 (2): 156-166.

Stoodley, N. 2000; From the Cradle to the Grave: Age Organization and the Early Anglo-Saxon Burial Rite. *World Archaeology,* Vol. 31 (3): 456-472.

Storey, R. 1992; Preindustrial urban lifestyle and health. *MASCA Research Papers in Science and Archaeology,* Vol. 9:33–41.

Strauss, R. S. and Dietz, W. H. 1997; Effects of intrauterine growth retardation in premature infants on early childhood growth. *The Journal of Pediatrics,* Vol. 130 (1): 95-102.

Stuart-Macadam P. L. 1988; Rickets as an interpretative tool. *Journal of Paleopathology,* Vol. 2: 33-42.

Stull, K. E. and Godde, K. 2013; Sex estimation of infants between birth and one year through discriminant analysis of the humerus and femur. *Journal of Forensic Sciences,* Vol. 58 (1): 13-20.

Sundelin-Wahlsten, V., Hallberg, G., and Helander, A. 2017; Higher alcohol consumption in early pregnancy or low-to-moderate drinking during pregnancy may affect children's behaviour and development at one year and six months. *Acta Paediatrica,* Vol. 106 (3): 446-453.

Swain, H. 2002; The ethics of displaying human remains from British archaeological sites. *Public Archaeology,* Vol. 2: 95-100.

Syddall, H. E., Sayer, A. A., Simmonds, S. J., Osmond, C., Cox, V., Dennison, E. M., Barker, D. J. P., and Cooper, C. 2005; Birth weight, infant weight gain, and cause-specific mortality: The Hertfordshire Cohort Study. *American Journal of Epidemiology,* Vol. 161: 1074–1080.

Synnes, A. R., Ling, E. W. and Whitfield, M. F. 1994; Perinatal outcomes of a large cohort of extremely low gestational age infants (twenty-three to twenty-eight weeks of gestation). *Journal of Pediatrics,* Vol. 125: 952–960.

Talge, N. M., Neal, C., and Glover, V. 2007; Antenatal maternal stress and long-term effects on child neurodevelopment: How and why? *Journal of Child Psychology and Psychiatry*, Vol. 48 (3–4): 245-261.

Tanner, J. M. 1963; The regulation of human growth. *Child Development,* Vol. 34: 817-847.

Tanner, J. M. 1978; *Foetus Into Man: Physical Growth from Conception to Maturity*. London: Open Books Publishing Ltd.

Tanner, J. M. 1981; *A History of the Study of Human Growth.* Cambridge: Cambridge University Press.

Tanner, J. 1994; Growth in height as a mirror of standard of living. In J. Komlos (Ed.) *Stature, living standards, and economic development: essays in anthropometric history*. Chicago, IL: University of Chicago Press: 1-9.

Tanner, J. M. and Whitehouse, R. H. 1975; Revised standards for triceps and subscapular skinfolds in British children. *Archives of Disease in Childhood,* Vol. 50: 142-145.

Tanner, J. M. and Whitehouse, R. H. 1976; Clinical longitudinal standards for height, weight, height velocity, weight velocity and stages of puberty. *Archives of Disease in Childhood,* Vol. 51: 170-179.

Temple, D. H. and Goodman, A. H. 2014; Bioarchaeology has a "health" problem: Conceptualizing "stress" and "health" in bioarchaeological research. *American Journal of Physical Anthropology*, Vol. 155: 186-191.

Thomas, C. Sloane, B. and Phillpotts, C. 1997; *Excavations at the Priory and Hospital of St Mary Spital, London* (Medieval Monasteries Series: MOLAS Monograph 1). London: Museum of London Archaeology Service.

Thorsell, A. and Nätt, D. 2016; Maternal stress and diet may influence affective behaviour and stress-response in offspring via epigenetic regulation of central peptidergic function. *Environmental Epigenetics,* Vol. 2 (3): 1-10.

Tocheri, M. W., Dupras, T. L., Sheldrick, P. and Molto, J. E. 2005; Roman Period Fetal Skeletons from the East Cemetery (Kellis 2) of Kellis, Egypt. *International Journal of Osteoarchaeology,* Vol. 15 (5): 326-341.

Ubelaker, D. H. 1978; *Human skeletal remains: excavation, analysis, interpretation*. Chicago: Aldine Publishing Co. Inc.

Ulijaszek, S. J. and Henry, C. J. K. 1996; Introduction: Growth, development and the lifespan developmental perspective. In C. J. K. Henry and S. J. Ulijaszek (Eds.) *Long-term Consequences of Early Environment Growth, Development and the Lifespan Developmental Perspective,* Society for the Study of Human Biology Symposium 37. Cambridge: Cambridge University Press: 1-6.

Ulizzi, L. and Zonta, L. A. 2002; Sex Differential Pattern in Perinatal Deaths in Italy. *Human Biology*, Vol. 74 (6): 879-888.

Upper Nene Archaeological Society 2009 *Briefing Notes* [Online] [Accessed: November 2016] Available from: http://www.unas.org.uk/user/magdirs/8images/brief2012.pdf

Upper Nene Archaeological Society 2009 *Interim Report* [Online] [Accessed: November 2016] Available from: http://www.unas.org.uk/magazine/magview.php?ID=8&date=0910&secshun=2

Upper Nene Archaeological Society 2009 *Phase Descriptions* [Online] [Accessed: November 2016] Available from: http://www.unas.org.uk/magazine/magview.php?ID=8&date=0910&secshun=2

Utczas, K., Muzsnai, A., Cameron, N., Zsakai, A. and Bodzsar, E. B. 2017; A comparison of skeletal maturity assessed by radiological and ultrasonic methods. *American Journal of Human Biology,* Vol. 29 (Early View): 1-7.

Van den Bergh, B. R., Mulder, E. J., Mennes, M., and Glover, V. 2005; Antenatal maternal anxiety and stress and the neurobehavioural development of the fetus and child: Links and possible mechanisms. A review. *Neuroscience & Biobehavioral Reviews,* Vol. 29 (2): 237–258.

Vattoth, S., DeLappe, R. S., and Chapman, P. R. 2013; Endocranial Lesions. *Seminars in Ultrasound, CT and MRI,* Vol. 34: 393-411.

Vickers, M. H., Breier, B. H., Cutfied, W. S., Hofman, P. L., and Gluckman, P. D. 2000; Fetal origins of hyperphagia, obesity, and hypertension and postnatal amplification by hypercaloric nutrition. *American Journal of Physiology – Endocrinology and Metabolism*, Vol. 279: 83–87.

Vlak, D., Roksandic, M. and Schillaci, M. A. 2008; Greater Sciatic Notch as a Sex Indicator in Juveniles. *American Journal of Physical Anthropology,* Vol. 137: 309-315.

Volk, A. A. and Atkinson, J. 2008; Is Child Death the Crucible of Human Evolution? *Journal of Social, Evolutionary and Cultural Psychology*: 103-116.

Wadhwa, P. D., Entringer, S., Buss, C. and Lu, M. C. 2011; The Contribution of Maternal Stress to Preterm Birth: Issues and Considerations. *Clinics in Perinatology,* Vol. 38 (3): 351-384.

Waldron, T. 2007; *Palaeoepidemiology. The Epidemiology of Human Remains*. Walnut Creek, California: Left Coast Press.

Waldron, T. 2009; *Palaeopathology*. Cambridge; Cambridge University Press.

Waldron, T., Taylor, G. and Rudling, D. 1999; Sexing of Romano-British baby burials from the Beddingham and Bignor villas. *Sussex Archaeological Collections,* Vol. 137: 71–79.

Walker, P. L. 2000; Bioarchaeological Ethics: A Historical Perspective on the Value of Human Remains. In M. A. Katzenberg and S. R. Saunders (Eds.) *Biological Anthropology of the Human Skeleton.* New York: Wiley-Liss: 3-39.

Walker, P.L., Bathurst, R. R., Richman, R., Gjerdrum, T., and Andrushko, V. A. 2009; The causes of porotic hyperostosis and *cribra orbitalia*: a reappraisal of the iron-deficiency anemia hypothesis. *American Journal of Physical Anthropology,* Vol. 139: 109-125.

Wapler, U., Crubézy, E. and Schultz, M. 2004; In Cribra Orbitalia Synonymous With Anemia? Analysis and Interpretation of Cranial Pathology in Sudan. *American Journal of Physical Anthropology,* Vol 123: 333-339.

Warren M. W. 1999; Radiographic determination of developmental age in fetuses and stillborns. *Journal of Forensic Sciences,* Vol. 44: 708–712.

Watts, D. J. 1989; Infant burials and Romano-British Christianity. *Archaeological Journal*, Vol.146: 372–383.

Watts, R. 2013; Lumbar vertebral canal size in adults and children: Observations from a skeletal sample from London, England. *HOMO Journal of Comparative Human Biology*, Vol. 64: 120-128.

Weaver, D. S. 1980; Sex differences in the ilia of a known sex and age sample of fetal and infant skeletons. *American Journal of Physical Anthropology* 52: 191-195.

Webster, J. 2001; Creolizing the provinces. *American Journal of Archaeology*, Vol. 105:209–225.

Webster, J. 2005. Archaeologies of slavery and servitude: bringing 'New World' perspectives to Roman Britain. *Journal of Roman Archaeology* 18:161-179.

Wells, C. and Collis, J. R. [No Date]; *The Burials.* Owslebury Site Report (Unpublished).

Weston, D. A. 2008; Investigating the Specificity of Periosteal Reactions in Pathology Museum Specimens. *American Journal of Physical Anthropology*, Vol. 137: 48-59.

Weston, D. A. 2012; Nonspecific infection in palaeopathology: Interpreting periosteal reactions. In A. L. Grauer (Ed.). *A Companion to Paleopathology*. Chichester: Wiley-Blackwell: 492-512.

White, T. D., Black, M. T. and Folkens, P. A. 2012; Human Osteology (3rd Edition). Oxford: Academic Press.

White, L. and Booth, T. J. 2014; The origin of bacteria responsible for bioerosion to the internal bone microstructure: results from experimentally-deposited pig carcasses. *Forensic Science International,* Vol. 239: 92-102.

WHO (World Health Organization) 1946: Constitution of WHO: Principles [Online] [Accessed: August 2017] Available from: http://www.who.int/about/mission/en/

WHO (World Health Organisation) *Global Health Observatory* [Online] [Accessed July 2017] Available from: http://www.who.int/gho/child_health/mortality/neonatal_infant_text/en/

WHO (World Health Organisation) *Maternal and Perinatal Health* [Online] [Accessed July 2017] Available from: http://www.who.int/maternal_child_adolescent/topics/maternal/maternal_perinatal/en/

WHO (World Health Organisation) *Newborns: Reducing Mortality* [Online] [Accessed July 2017] Available from: http://www.who.int/news-room/fact-sheets/detail/newborns-reducing-mortality

WHO (World Health Organisation) *Preterm Birth* [Online] [Accessed July 2017] Available from: http://www.who.int/en/news-room/fact-sheets/detail/preterm-birth

WHO (World Health Organisation) *Sexual and Reproductive Health* [Online] [Accessed July 2017] Available from: http://www.who.int/reproductivehealth/topics/maternal_perinatal/stillbirth/en/

Wiedemann, T. 1989; Adults and Children in the Roman Empire. New Haven & London: Yale University Press.

Wilcox, A. J. 2010; *Fertility and Pregnancy; An Epidemiologic Perspective.* Oxford: Oxford University Press.

Wiley, A. S. and Pike, I. L. 1998; An Alternative Method for Assessing Early Mortality in Contemporary Populations. *American Journal of Physical Anthropology*¸ Vol. 107: 315-330.

Wilkie, L. A. 2000; Not merely child's play: creating a historical archaeology of children and childhood. In J. Sofaer Derevenski (Ed.) *Children and Material Culture*. London: Routledge: 100–113.

Wilkie, L. A. 2013; Expelling frogs and binding babies: conception, gestation and birth in nineteenth-century African-American midwifery. *World Archaeology,* Vol. 45 (2): 272-284.

Wilson, L. A., MacLeod, N. and Humphrey, L. T. 2008; Morphometric Criteria for Sexing Juvenile Human Skeletons Using the Ilium. *Journal of Forensic Sciences,* Vol. 53 (2): 269-278.

Winick, M., Brasel, J. A. and Rosso, P. 1972; Nutrition and Cell Growth. In M. Winick (Ed.) *Nutrition and Development.* London: John Wiley & Sons Inc.: 49-98.

Wood J. W., Milner, G. R., Harpending, H. C. and Weiss, K. M. 1992; The osteological paradox: problems of inferring prehistoric health from skeletal samples. *Current Anthropology,* Vol. 33: 343–370.

Woods, R. 2008; Late-Fetal Mortality: Historical Perspectives on Continuing Problems of Estimation and Interpretation. *Population,* Vol. 63 (4): 591-614.

Wrigley, E. A. and Schofield, R. S. 1989; The Population History of England 1541-1871. Cambridge: Cambridge University Press.

Wrigley, E. A., Davies, R. S., Oeppen, J. E. and Schofield, R. S. 1997; *English Population History from Family Reconstitution 1580-1837*. Cambridge: Cambridge University Press.

Wu, G. Imhoff-Kunsch, B. and Webb Girard, A. 2012; Biological Mechanisms for Nutritional Regulation of Maternal Health and Fetal Development. *Paediatric and Perinatal Epidemiology,* Vol 26, Suppl.1: 4-26.

Wyness, M.G. 2006; *Childhood and Society: An Introduction to the Sociology of Childhood*. Houndmills: Palgrave Macmillan.

Yongen, G. 1980; Neonatal Intracranial Hemorrhage. *Tianjin Medical Journal,* Vol. 6.

Zemel, B. S. 2017; Influence of complex childhood disease on variation in growth and skeletal development. *American Journal of Human Biology,* [Early View].

Zhu, P., Tao, F., Hao, J., Sun, Y. and Jiang, X. 2010; Prenatal life events stress: implications for preterm birth and infant birthweight. *American Journal of Obstetrics and Gynecology,* Vol. 203 (34): 1-8.

Zuckerman, M. K., Turner, B. L. and Armelagos, G. J. 2012; Evolutionary thought in palaeopathology and the rise of the biocultural approach. In A. L. Grauer (Ed.) *A Companion to Paleopathology*. Chichester; Malden, MA: Wiley-Blackwell: 34-57.