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The Growth of a Nation: Child health and development in the Industrial Revolution in England, c. AD 1750-1850.

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Thesis submitted for the Degree of Doctor of Philosophy

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The Growth of a Nation: Child health and development in the Industrial Revolution in England, c. AD 1750-1850

Sophie Louise Newman

ABSTRACT – The Industrial Revolution of 18th-19th century England was a period of marked social, economic, and political change through which urban landscapes were irrevocably transformed. Increasing industrialisation brought with it issues of overcrowding, deteriorating sanitary conditions, and rising air pollution. Disease was rife, and life on the epidemic streets brought significant risks to child mortality and morbidity.

This study undertook a comprehensive analysis of health in urban children from the 18th-19th centuries. Six skeletal collections were selected from urban-based sites to attempt to broaden existing knowledge on the impact of industrialisation on child health from a range of geographical and social contexts. Growth parameters (long bone length, cortical thickness, and vertebral dimensions) and non-specific indicators of stress (dental enamel hypoplasia, metabolic disease, cribra orbitalia, and periosteal new bone formation) were selected to assess health status in both children and adults, to identify differing patterns in health stress and longevity.

No significant differences were identified between northern and southern-based sites, with social status being the primary determinant of child health. Lower status groups demonstrated the highest perinatal mortality rates, lowest growth values, some of the highest rates of pathology, and intrauterine onset of deficiency diseases, indicating a heightened exposure to poor maternal health and detrimental exogenous influences associated with poverty. However, the high status group from Chelsea Old Church, London, also showed significant deficiencies in growth values and a high rate of metabolic disease, suggestive of "fashionable" child-care practices. A potential association between the presence of non-specific indicators of stress and an earlier age-at-death was identified in adults, suggestive of a reduction in longevity associated with early life stress. Life in the city came with significant health risks for children, and the use of multiple growth parameters and indicators of stress proved an effective means to increase the osteobiographical understanding of past populations.

CONTENTS LIST

List of Figures

Image on front page – "Substance and Shadow", drawn by John Leech for Punch, 1843. Adapted from Allingham (2014).

List of Tables

MANUSCRIPT ONE

MANUSCRIPT TWO

MANUSCRIPT THREE

MANUSCRIPT FOUR

List of Abbreviations

ANCOVA – Analysis of co-variance
ANOVA – Analysis of variance
AP diameter– Anteroposterior diameter
BB – Bow Baptist
BH – Body height
CB – Cross Bones
CL – Coach Lane
CO – Cribra orbitalia
COC – Chelsea Old Church
CPR – Crude prevalence rate
CS – Coronation Street
CT – Cortical thickness
DEH – Dental enamel hypoplasia
F - Female
F? – Possible female
GH – Growth hormone
HL – Harris' lines
I - Indeterminate
L - Left
M - Male
M? – Possible male
MoL – Museum of London
MoLA – Museum of London Archaeology
mths - Months
PNBF – Periosteal new bone formation
R - Right
SBS – St Benet Sherehog
SES – Socio-economic status
SGA – Small-for-gestational age
TEM – Technical error of measurement
TR diameter – Transverse diameter
U – Unclassified
VBH – Vertebral body height
VNC – Vertebral neural canal
wiu – Weeks *in utero*
WORD – Wellcome Osteological Research Database
yrs - Years

Statement of Copyright

The copyright of this thesis rests with the author. No quotation from it should be published without the author's prior written consent and information derived from it should be acknowledged.

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CHAPTER ONE
Introduction

1.1 Children and childhood in context

The study of child health in the past can provide a wealth of information regarding past communities, including health, behaviour, and living environment (Ruff *et al*., 2013). Children are most sensitive to fluctuations in climate, economic circumstance, diet, exposure to disease, and episodes of psychosocial stress, and will in turn grow up to shape the health of the adult populace (Lewis, 2007; Cameron and Bogin, 2012). During the vital developmental period of childhood, a strong biological foundation is laid down that will determine future adult health, and potentially longevity (Barker, 1991,1992,1994; Kamp, 2001). Early life experiences can influence stature, exposure to disease and subsequent immune resistance or immune deficiency, our susceptibilities to future health issues, and even cognitive developmental ability (Barker and Osmond, 1986; Barker, 1992,1994; Chavez *et al*., 2000; Cameron and Bogin, 2012; Aizer and Currie, 2014; Noble *et al*., 2015; Gowland, 2015).

However, before the interactions between child health, community, and environment can begin to be understood, it is important to establish some definitions regarding how children and childhood will be considered within this thesis. Cunningham stated "we need to distinguish between children as human beings and childhood as a shifting set of ideas" (2005: 1). In this sense, and within the scope of this thesis, "children" can be viewed as biological entities, individuals who have not yet reached maturity, and are continuing to learn and develop in relation to the world around them. Whereas "childhood" is more reflective of concepts regarding the treatment of children within discrete communities, and the behaviour expected of children growing up within them. Experiences of "childhood" are therefore expected to differ over time and space, as these concepts will alter access to resources, the types of activities undertaken by children, exposure to disease agents and occupational hazards, and may influence health care strategies (Cunningham, 2005; Gowland, 2006; Halcrow and Tayles, 2008). Perceptions of when childhood begins and ends have varied throughout history, and may have been influenced by geographic location, social status, and gender (Kamp,

2001; Cunningham, 2005; Halcrow and Tayles, 2008). Therefore, while this thesis is founded on data relating to the physical ageing of children, "as the body develops it is, to an extent, taken up and transformed by social factors: biology and culture are largely inter-twined" (Gowland, 2006: 144). Thus, it is vital to appreciate that social identity will have a substantial impact on the course of development when interpreting bioarchaeological data. The "childhood" of the present day is a period in the life course of dependency and education, where children are to be protected throughout their early interactions with the world around them as they continue to develop towards adulthood (Kamp, 2001; Cunningham, 2005). Whereas the "childhood" of past communities, prior to the onset of compulsory education, was one where commencement of work within the family sphere would have been expected from an early age (although this too would have been dependent on social status) (Kamp, 2001; Cunningham, 2005). Therefore, depending on the community and time period in question, the "role" of children within society may have differed, and this alongside household economic strategies, would have varied the types of health patterns we may expect to see in past populations (Kamp, 2001). It is therefore essential to maintain an appreciation for the varieties of childhood that existed over time and space, and avoid discussing topics of child health in the past in reference to modern day Western ideals of "childhood" (Sofaer Derevenski, 1997; Gowland, 2001,2002,2006; Sofaer, 2006; Halcrow and Tayles, 2008). Thus, while "childhood" is a somewhat of a loaded term, within this thesis it will serve to describe the experiences of children throughout the period of growth and development from birth to 17 years of age, with due consideration to the roles expected of children within $18^{th}/19^{th}$ century society.

1.2 Accessing child heath in the past: bioarchaeological perspectives

When considering "age" in bioarchaeology, it can be approached from three different viewpoints, with origins in both biological and social ideas of the life course (Sofaer Derevenski, 1997; Gowland, 2001,2002,2006; Sofaer, 2006; Halcrow and Tayles, 2008). Physiological age refers to the physical ageing of the body. For children, interchangeably described as "non-adults" or "sub-adults" within bioarchaeological literature, this relates to those between 0-17 years of age (Lewis, 2007; Halcrow and Tayles, 2008). Physiological age is assigned based on

the progression of developmental stages from parameters such as dental calcification and eruption (Moorrees *et al*., 1963a,b; Van Beek, 1983; Smith, 1991), and growth and fusion of the long bones (Scheuer and Black, 2000). Chronological age corresponds to the amount of time that has passed from the moment of birth (Gowland, 2006; Sofaer, 2006; Halcrow and Tayles, 2008). Lastly, social age relates to socially constructed norms concerning appropriate behaviour and attitudes for an age group, not dissimilar to that seen in the construction of ideals for childhood seen throughout time (Gowland, 2006; Sofaer, 2006; Halcrow and Tayles, 2008). Through the interaction of these three perspectives of age, the life-course is often categorised roughly into "infant", "child", "adolescent", "young adult", "prime adult", "mature adult", and "older adult". Into these stages skeletal data are frequently categorised, and interpreted in relation to the "norms" for each stage in the life course. With an appropriate consideration for the historical background, and social norms for the skeletal collection under study, this life-course approach can provide an effective means by which data collected from skeletal populations can be assessed. Table 1.1 describes the terminology to be used within this thesis, and the corresponding age range to which these relate.

Table 1.1 – *Breakdown of age terminology. Adapted from Lewis (2007), Raynor et al. (2011), and Powers (2012). These categorisations are made purely to ease discussion regarding the data collected in this thesis.*

Terminology	Age range
Foetus/Prenatal	8 weeks *in utero* (wiu) to birth
Perinate	Period relating to around the time of birth, 36 wiu to 4 weeks post-partum
Infant	Relating from birth to 1 year of age
Child	Approximately 1 to 12 years of age
Early childhood	Approximately 1 to 6 years of age
Late childhood	Approximately 6 to 12 years of age
Adolescent	Approximately 12 to 17 years of age
Childhood	For this thesis this will be used to discuss the entirety of the growth period, from birth to 17 years of age
Non-adult	0-17 years of age
Adult	>17 years of age
Young adult	18-25 years of age
Prime adult	26-35 years of age
Mature adult	36-45 years of age
Older adult	>46 years of age

Child health within bioarchaeological study is primarily accessed through the construction of mortality distributions, analysis of growth, and the presence of a multitude of non-specific indicators of stress. As will be described later in the thesis (Chapter Two), growth is genetically determined, but highly influenced by environmental factors such as access to adequate nutrition and exposure to disease (Duren *et al*., 2013; Stulp and Barrett, 2014). Consequently, evidence of growth delay in past populations has classically been used as an indicator of suboptimal environment in childhood (Hummert, 1983; Goodman *et al*., 1984; Ribot and Roberts, 1996; Hoppa and Fitzgerald, 1999; Pinhasi *et al*., 2006; Saunders, 2008; Mays *et al*., 2008, Mays *et al*. 2009a,b; Lewis, 2002a,b; Cardoso and Garcia, 2009; Pinhasi *et al*., 2014). However, studies of skeletal growth in bioarchaeology have often solely relied on the analysis of long bone length. With emerging methodologies, such as measurement of cortical thickness (Mays *et al*., 2009a), it is of interest to seek new avenues for the detection of growth disruption in past populations. Stress, within the context of bioarchaeological study, refers to the myriad of factors (i.e. poor health, malnutrition, and/or psychosocial stress) that may result in physiological disruption within the developing body. As will be described in Chapter Two, there are a limited number of ways that the body can respond to stress, and the skeletal response to such disruption is further restricted; therefore evidence of stress in past populations can rarely be attributed to a distinct aetiology (Huss-Ashmore *et al*., 1982; Sapolsky, 2004). The most commonly utilised non-specific indicators of stress are dental enamel hypoplasia, periosteal new bone formation, cribra orbitalia, and Harris' lines, and these will be described further in Chapter Four.

Therefore, indicators of growth disruption can be used within palaeopathological study to provide a commentary on environmental pressures and social experiences of children in past communities.

1.3 Children and childhood in the 18th and 19th centuries

While bioarchaeological data enables the detection and interpretation of biological variability in the experience of children in past communities, caution must be applied when discussing these in relation to the social experiences of childhood in the 18th/19th centuries. Concepts of childhood would have differed

throughout the social strata at this time. In the middle to upper classes children may have been more protected from the more severe aspects of industrialisation of the cities, whilst within lower class families the sending of children to work from an early age to support the family economy was commonplace and indispensable (Pike, 1966; Pinchbeck and Hewitt, 1973; Earle, 1989; Cunningham, 2005; Humphries, 2010). These socially determined expectations of the roles of children at this time means that concepts of age, experiences of childhood, and consequently child health would have differed markedly at this time. For example, the poignant description of Henry Mayhew's "little watercress girl" in the mid-18th century reveals the effect of growing up in deprivation during this time on not only the corporeal identity of the young girl, but also her self-constructed social identity in terms of age -

"The little watercress girl who gave me the following statement, although only eight years of age, had entirely lost all childish ways, and was, indeed, in thoughts and manner, a woman….Her little face, pale and thin with privation, was wrinkled where the dimples ought to have been, and she would sigh frequently…'I ain't a child, and I shan't be a woman till I'm twenty, but I'm past eight, I am.'" – Mayhew (1851:151-152).

The impact of social constructions of identity, experiences of childhood, and even whether an individual had entered the labour market, cannot always be directly ascertained through the analysis of skeletal remains, however, such considerations of the interplay of social status, external environment, and health must be kept in mind when interpreting bioarchaeological data.

While the field of bioarchaeology continues to make strides in how we access child health in the past, historical research into the role of children in past societies has developed rapidly (Kirby, 1995,1997; Crawford, 2010; Humphries, 2010; Levene, 2012; Kirby, 2013; Goose and Honeyman, 2013). The topic of children in the industrial revolution is one of the most widely researched areas of childhood in the past, with a vast body of work dedicated to the plight of the child labourer. The industrial centres of this time are notorious for their injurious living conditions, pervasive poverty, and merciless child labour practices (Humphries, 2010; Sharpe, 2012; Kirby, 2013; Goose and Honeymen, 2013). However economic and social historians have recently begun to view childhood during this time in a

new light, with a movement away from the perceptions of child labourers as infant slaves, to those able to exert their own agency (Goose and Honeyman, 2013). There has also been a multitude of studies that have recognised the considerable regional variation in experiences of industrialisation at this time (Kirby, 1995, 1997; Kirby, 2013; Goose, 2013; Humphries, 2013). With this in mind, it is time to re-evaluate the way in which we approach the topic within bioarchaeological study, in order to reveal a more comprehensive and broader view of child health during this time.

1.4 Research aims and objectives

This study will undertake a comprehensive analysis of childhood health in Post-medieval England through the analysis of six skeletal collections of varying geographic location and socioeconomic status. This research will thus provide a comparison of the morbidity and levels of stress experienced by children growing up in urban (i.e. industrial) environments in the North and South of England. It will achieve this through the analysis of a variety of growth parameters (long bone length, cortical thickness, and vertebral dimensions) and non-specific indicators of stress within the non-adult (0-17 years) sample from each site. Adult comparative data (relating to vertebral dimensions, and non-specific indicators of stress) will also be included from each site to discuss the impact of industrialisation on longevity. It will also utilise and evaluate emerging methodologies for the detection of poor health within skeletal remains, which will further our understanding regarding the nature of stressors affecting these populations, and could prove to be valuable to future population studies.

1.5 Research questions

1. Are there observable differences in the skeletal indicators of poor health in non-adults between northern and southern populations of seemingly similar environmental background?
2. Are there observable differences in the skeletal indicators of poor health in non-adults from differing socio-economic backgrounds?
3. At what stage of the life course do these skeletal indicators of poor health become apparent, and how might they relate to child-care and child labour practices?

4. Do vertebral indicators of stress and measurements of cortical thickness provide a complementary understanding of the living environments and health status of non-adults within a population when combined with more commonly utilised techniques (e.g. long bone length, enamel hypoplasia, periosteal new bone formation, cribra orbitalia, and Harris' lines)?
5. Is there an association between evidence of growth disruption in past populations and the presence of indicators of stress and/or metabolic disease?

1.6 Thesis layout

This thesis will be structured into the following chapters to achieve the research aims and questions outlined above. As this is a PhD thesis that incorporates published works, the main body of the results and discussion are presented via four separate manuscripts that address the key questions within this study.

Chapter Two – The impact of environment on growth and development: This chapter discusses the fundamental aspects of the growth process, focusing on the development of the long bones and the vertebral column. It introduces the existing evidence for the impact that environmental stressors can have on these growth processes, and how this disruption can be detected within bioarchaeological study. This chapter will also outline the importance of the development of a new methodology for the detection of growth disruption within the vertebral column. It also includes background information on the Developmental Origins of Health and Disease hypothesis (DOHaD hypothesis) to discuss the impact that environmental stressors, such as recurrent infection and malnutrition in early life, can have on development and future adult health.

Chapter Three – The Industrial Revolution, c.1750-1850: This chapter represents the historical context section of the thesis. It considers the time period as a whole, with an emphasis on the effects of industrialisation on the health of urban populations. It particularly focuses on child health, including descriptions of the types of infectious diseases that posed high risks to the survival of young children. It also includes important contextual information on the vitamin deficiency

conditions rickets and scurvy, as these will become major areas of focus in the later sections within this thesis.

Chapter Four – Materials: This chapter will provide the essential background information for each site used within this project. It will begin by providing inclusion criteria for pathological and metric analysis, and then will provide summary contextual information for each site.

Chapter Five – Methods: This chapter will provide descriptions of the methods used for the age estimation of non-adult individuals, methods of measurement of the long bones and vertebrae, and standards for the recording of pathology within the collections. It will also include the descriptions of the statistical analysis used within the four manuscripts.

Manuscript One - Brief Communication: the use of non-adult vertebral dimensions as indicators of growth disruption and non- specific health stress in skeletal populations: This published paper introduces a new method through which growth disruption can be detected in the non-adult populations of skeletal collections. It uses measurements of vertebral body height and transverse diameter of 96 non-adult skeletons and 40 adult skeletons from two post-medieval sites (Bow Baptist, London and Coronation Street, South Shields). Non-adult measurements were plotted against dental age to construct vertebral growth profiles through which inter-population comparisons could be made.

Manuscript Two – Dedicated Followers of Fashion? Bioarchaeological perspectives on socio-economic status, inequality, and health in urban children from the Industrial Revolution (18th-19th C), England: This paper investigates the impact of the widening social inequality of the 18th and 19th centuries in England on child health. It uses measurements of diaphyseal length, cortical thickness, vertebral measurements, and prevalence of pathology. This data was collected from 403 non-adults (0-17yrs) from four skeletal populations from London of varying socio-economic status. The results of this study allowed for a discussion of the child-care practices in use during this time, and the impact of social inequality on health in the past.

Manuscript Three – North and South: a comprehensive analysis of non-adult growth in the Industrial Revolution (18th-19th C), England: This paper compares health and growth data collected from skeletal collections of urban sites from both northern and southern geographic locations, amounting to 575 non-adults (0-17 yrs) from six urban sites (c. AD 1711-1856). Most bioarchaeological studies related to the industrial revolution have focused on London based populations, giving a southern bias in our interpretation of child health in this time. This study will therefore help develop an appreciation of the broader experience of child health in the post-medieval period.

Manuscript Four – Town-made articles of small stature: unravelling growth disruption and pathological indicators of stress in skeletal populations: The aim of this paper will be to identify the key disease processes/stresses that result in the growth stunting (diaphyseal length, appositional, and vertebral) often identified in archaeological populations. This paper used growth and health data from 284 non-adults (0-17 yrs) from two sites (Bow Baptist, London and Coach Lane, North Shields) to attempt to ascertain a more directed explanation for why these specific populations experienced growth disruption.

Chapter Six – Discussion: This chapter will synthesise the content of the four manuscripts, and discuss the results in relation to the wider context of the time period. It will use these results to discuss the environmental conditions that impacted health, and also the social conditions (such as the employment of women) that may have led to a decline in both maternal and child health. It will connect the subject of poor maternal health with the Developmental Origins of Health and Disease (DOHaD) hypothesis, and discuss what this may have meant for overall population health, and the health of future generations. It will also allow for a critique of the types of techniques used in this study (and within wider bioarchaeological research) to access evidence of "stress" in the past.

Chapter Seven – Conclusion: This final chapter will summarise the main findings of this thesis, and suggest avenues for further research.

CHAPTER TWO

The impact of environment on growth and development

"The three dualities of genotype and phenotype, genes and development, and organism and environment are coupled beautifully and integrated mechanistically" (Hall, 2005; 191).

2.1 Introduction

This chapter will address the intrinsic and extrinsic factors that govern the processes of growth, and more specifically the effects that the external environment of an individual/population may have on the course of development. It will begin by outlining the fundamental principles of the process of growth and development, and then will consider the specific growth patterns of the skeletal elements relevant to this study (the long bones and vertebrae). This will establish the "normal" growth of an individual, so that deviations can be identified and understood in relation to environmental conditions. This will allow for the identification of potential indicators of poor growth within a population, and the application of such indicators in palaeopathological study will be discussed.

2.2 Developmental homeostasis

The process of growth and development of an organism from conception to maturity is complex and highly regulated. The growth period is characterised by progressive changes in size and developmental maturity that accompany the transition from the earliest stages of life to adulthood. According to Hallgrímsson *et al*. (2002; p.133), "for organisms to develop as functionally integrated systems, structures have to develop in highly predictable ways". To achieve this, a highly regulated developmental system is required to reduce variation from the intended phenotype (e.g. morphology) (Hallgrímsson *et al*., 2002). Homeostasis is an expression used to describe the maintenance of the internal environment of an organism so that properties required for efficient functioning, such as body temperature and blood pressure, are regulated appropriately (Martini, 2006). In the same way that processes crucial for everyday life are maintained, the processes of growth and development must also be carefully controlled. This regulation, via a myriad of physiological processes, is essential for the stable development of an

organism, and acts to ensure that any deviation from the desired developmental pathways are either avoided or rectified.

Developmental homeostasis therefore refers to the processes implemented to regulate and stabilise growth patterns in all systems of the body, whether skeletal, muscular, or in the development of the organs. (Debat and David, 2001). Developmental homeostasis is usually divided into two aspects of developmental control, developmental stability and canalisation (Møller and Swaddle, 1997; Debat and David, 2001). These are used to describe as an organism's ability to produce a desired phenotype despite the range of genetic and environmental influences that may perturb development (Albert and Greene, 1999; Debat and David, 2001; Hallgrímsson *et al.,* 2002). This mechanism has been best depicted by Waddington (1940) in his description of the 'epigenetic landscape'. In this model Waddington imagines the developmental course as that of a system of valleys on an inclined plane (Waddington, 1940). The rolling of a ball down this inclined network of valleys represents the passage of an organism down its developmental pathway to maturity. Steep sided valleys represent a high capability for canalisation, as any deviation away from the developmental path is quickly righted. In this case a significant environmental stressor would be required to exceed the threshold to push an organism capable of high canalisation away from its developmental trajectory. Shallow valleys therefore represent a reduced capacity for canalisation whereby perturbations in development can easily push an organism away from the desired phenotype (Møller and Swaddle, 1997). Over the course of growth and development of an organism's capacity for canalisation is thought to fluctuate. As an individual nears maturity the "valleys" steepen so that any earlier movement away from the desired developmental trajectory becomes increasingly difficult to recover from, and the effects of perturbations in growth and development may become permanent. In this way, developmental stability works to resist changes to the developmental pathway under times of stress, and canalisation ensures that any deviation that does occur is righted and the pathway restored. It is, in short, an organism's buffering capacity against stress (Storm, 2009). Thus, developmental instability describes a state where the organism is unable to buffer stress, so succumbs to its disruptive effects on development and growth (Storm, 2009; Barrett *et al.,* 2012).

Factors that act negatively on developmental homeostasis are classed as 'developmental stressors' (Møller and Swaddle, 1997). Importantly to this study, developmental stressors can include environmental factors such as pollution, population density, temperature, and nutritional stress (Møller and Swaddle, 1997). The effect of the external environment that an individual lives and develops in is thought to be far-reaching at both the genotypic and phenotypic level. A study of monozygotic twin pairs revealed differences in epigenetic modifications of the genotype, which increased in frequency with age due to differences in lifestyle (Fraga *et al.,* 2005). The authors felt that this underlined "the significant role of environmental factors in translating a common genotype into a different phenotype" (Fraga *et al.,* 2005; 10609).

When considering the more direct effects of environment on growth, it has long been recognised that the environment heavily influences the regulatory processes that control growth, with those living in poorer conditions often suffering periods of growth disruption at great detriment to their immediate and future health. Exposure to stressful environmental conditions will divert energy into the maintenance of key survival processes, which does not include the maintenance of developmental stability (Møller and Swaddle, 1997). As a result, the individual is unable to efficiently buffer growth processes from deleterious effects. It is clear, that despite the mechanisms in place to maintain the processes of growth and development, the external environment remains a notable influence on the ability of an organism to stay on its set developmental pathway. The buffering capacity against stress will always vary between populations (Graham *et al.,* 1998; Barrett *et al.,* 2012). Some may have much higher thresholds and can therefore buffer the effects of a negative environment more efficiently, and with little to no disturbance to the normal growth trajectory. However, some populations may have a reduced threshold to stress and will suffer growth disturbance more readily (Barrett *et al.,* 2012).

The growth and development of the skeletal elements of interest in this study will be discussed in the following sections, including the direct mechanisms by which the environment may perturb these processes.

2.3 The process of growth

As shown in Figure 2.1, as an individual ages, they pass through the growth stages of infancy, childhood, and adolescence before reaching adulthood (maturity) (Nicoletti *et al.,* 2004). Each of these stages varies in duration, and also rate of growth.

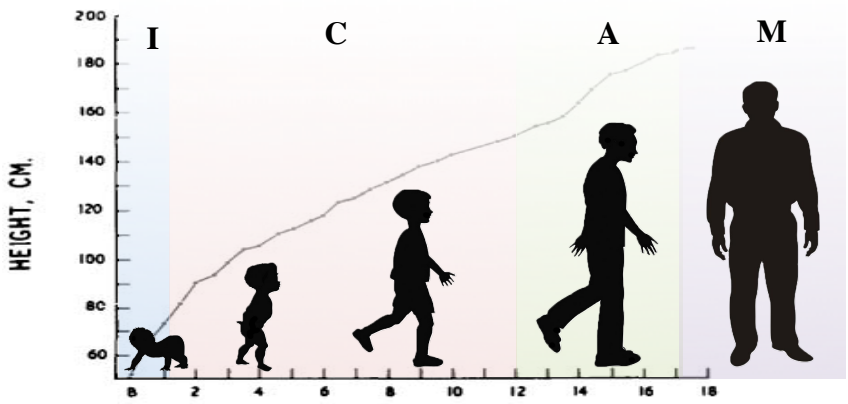

Fig.2.1 – *The stages of growth from birth to maturity. I= Infancy; C= Childhood; A= Adolescence; M= Maturity. Adapted from Tanner (1989; 7).*

"Infancy" encompasses the period from birth to approximately one year of age (although some may include individuals of up to three years of age) (Bogin, 1999; Lewis, 2002). This stage is associated with a very high growth velocity compared to all the other stages of postnatal development, and an individual may double their length at birth in the first year alone (Bogin, 1999). The *in utero* growth of the foetus is associated with a rapid increase in body length, therefore at birth the growth rate is already high. From approximately four months *in utero* growth velocity begins to decrease, therefore despite demonstrating a very high increase in body length, the growth period during infancy actually represents a deceleration in growth (Tanner, 1989). Following this stage, growth rate throughout "childhood" is relatively steady, albeit still slowly decelerating in velocity (Fig.2.1) (Bogin, 1999). The period of childhood growth ends with the beginning of puberty, which generally occurs earlier in girls than boys (approximately age 10 and 12 respectively) (Fig.2.1) (Bogin and Smith, 1966; Bogin, 1999; Stulp and Barrett, 2014). This indicates the start of the adolescent stage of growth, and thus the rapid acceleration in growth velocity associated with the pubertal growth spurt, which makes a significant contribution to final adult height (Bogin, 1999; Nicoletti *et al.*,

2004; Lewis *et al.,* 2015). Prior to the adolescent growth spurt an individual has usually only fulfilled 77% of their skeletal growth (Bogin and Smith, 1966). After the peak velocity of the growth spurt is reached, growth then decelerates until cessation when the individual reaches "maturity" (Ulijaszek *et al.,* 1998).

While the process of postnatal growth generally follows the same pattern for all individuals (see Fig.2.1), a degree of variation is expected as some may develop in size and maturity at a faster rate than others, particularly between the sexes and when comparing populations (Tanner, 1981; Lewis *et al.,* 2015). However, according to Tanner (1989) "growth is in general a very regular process" (p.6), therefore variations in growth may be indicative of disruption due to external influences. This is the basis for the use of measurements of stature as an indicator of population fitness in modern growth studies. However, when considering stature, it is important to remember that it is a result of a combination of both standing height (i.e. leg length) and sitting height (i.e. contributions from both the head and the vertebral column). Therefore, to understand how the environment could potentially disrupt the normal growth pattern in past populations, it is necessary to understand the processes by which the relevant skeletal elements develop (the long bones and the vertebrae). Unfortunately, despite sitting height being a major contributor to an individual's stature, longitudinal growth of the vertebral column is often overlooked in growth studies in favour of long bone length.

2.3.1 Long bone growth

Long bone development begins with the formation of a cartilage template by approximately 49-52 days *in utero* (Fig.2.2a). From this template, ossification can commence (Fig.2.2b), with the appearance of the primary centre of ossification in the centre of the shaft at approximately 7-8 weeks *in utero* (Fig.2.2c) (Price *et al.*, 1994; Scheuer and Black, 2000). Following birth, secondary centres of ossification, called the epiphyses, begin to appear at both ends of the long bones (Fig.2.2d) (Tanner, 1989). The growth plate (or epiphyseal plate) sits between the epiphysis and the metaphysis of the long bone, and it is within here that chrondroblasts divide, and undergo proliferation, differentiation, and finally resorption (Tanner, 1989). This process, by which cartilage is progressively replaced by new bone, leads to the

increase in length of the long bones at both ends necessary for longitudinal growth (Fig.2.2d,e) (Scheuer and Black, 2000).

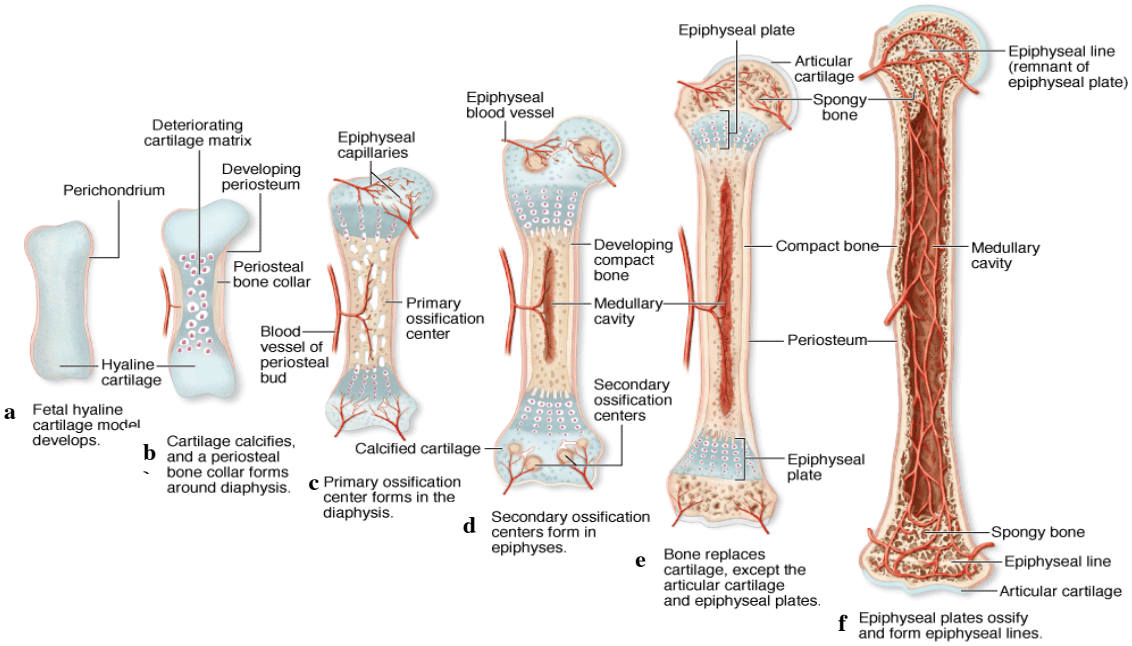

Fig.2.2 – *The process of longitudinal growth. a) Development commences with the formation of the cartilage template. b) Ossification is initiated by the laying down of a collar of bone at the midshaft. This forms the initial periosteum via intramembranous ossification. c) Endochondral ossification at primary centre. d) Formation of secondary centres and growth at the epiphyseal plate. e) Cartilage replaced by new bone at the growth plate. 6) Fusion of epiphyses to diaphysis marking end of growth. (Price et al, 1994; Scheuer and Black, 2004). Image adapted from Mescher (2009).*

Longitudinal growth is accompanied by appositional growth, which refers to the increase in diameter (width) of the long bone. Appositional growth is classed as intramembranous growth, differing from the endochondral process of longitudinal growth (Price *et al.*, 1994). Intramembranous growth of the long bone diaphysis requires the addition of new bone to the periosteal (outer) surface of the bone, which sits beneath the fibrous covering of the periosteum (Mays, 1999; Prentice *et al.*, 2006; Gosman *et al.*, 2011) (see Fig.2.3). Concomitant to the deposition of new bone on the periosteal surface, resorption of bone occurs on the endosteal (inner) surface (Fig.2.3) (Price *et al.*, 1994; Prentice *et al.*, 2006). These two processes work together to create a proportional increase in medullary width, while the total bone width continues to increase (Price *et al.*, 1994; Prentice *et al.*, 2006). This balanced process of outer bone formation and inner bone removal continues throughout infancy and childhood, until adolescence when the addition of cortical bone occurs at both the periosteal and endosteal surfaces (Mays, 1999).

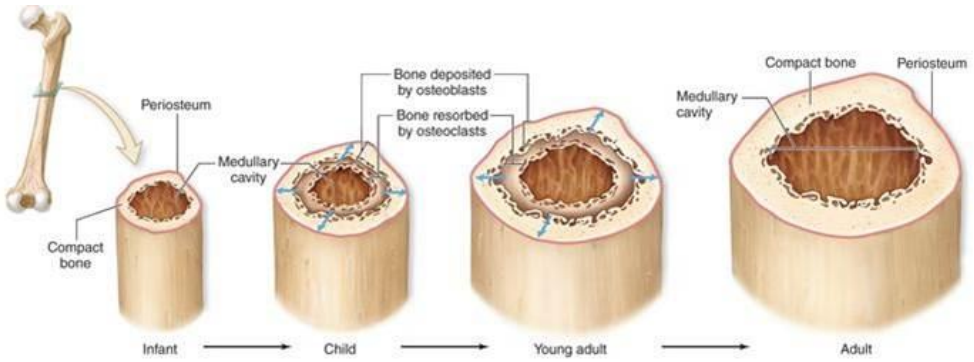

Fig. 2.3 – *The process of appositional growth. Bone resorption by osteoclasts increases medullary width. Bone addition by osteoblasts increases total bone width. Image taken from Alfa Image (2015).*

Eventually, the rate of cartilage proliferation within the growth plate becomes exceeded by the rate of new bone deposition at the metaphysis, following which the growth plate thins and is bridged by new bone formation between the epiphysis and the diaphysis (Fig.2.2f) (Tanner, 1989; Scheuer and Black, 2000). These eventually fuse together, signalling the end of the longitudinal growth process (Tanner, 1989). Growth plate closure is determined by the sex hormones (androgens and oestrogens) during puberty, and due to the desensitisation of the cartilage cells to growth promoting hormones (Tanner, 1989; Bogin, 1999). As depicted in Figure 2.1, once the growth of an individual has ceased, they have attained adult stature and are considered to have reached "maturity" (Bogin, 1999). In modern populations young men and women are expected to reach adult height at 21 years and 18 years of age respectively, however there may be variation between individuals and populations, and some may take longer to reach maturity (Bogin, 1999; Duren *et al*., 2013; Lewis *et al*., 2015). Whilst longitudinal growth of the long bones concludes with the onset of maturity, cortical width may still fluctuate throughout adulthood via remodelling and may be affected by pregnancy, lactation, menopause, and activity (Prentice *et al*., 2006; Duren *et al*., 2013).

2.3.2 Vertebral growth

Vertebral growth is a complex process. Vertebral formation not only requires the ossification and fusion of three separate centres to form the ring-like structure of the neural canal, but this development must also progress around another essential developing structure, the spinal cord. This means that the

appropriate development of the vertebral column is vital to the future functioning of the growing individual, and when perturbations in normal development arise in this period, they can often have serious consequences.

Formation of the vertebral column begins very early in development, with the midline of the embryo established by the notochord by approximately 28 days (Scheuer and Black, 2000; Bogduk, 2005). Masses of paraxial mesoderm form bilaterally to the notochord and eventually segment to become the somites, from which cells will migrate medially and posteriorly around the fourth week of gestation to form the mesenchymal precursors of the vertebral bodies and neural arches (Jinkins, 2000; Scheuer and Black, 2000; Bogduk, 2005). Following resegmentation (see Jinkins, 2000; Scheuer and Black, 2000), the mesenchymal template of each "primitive" vertebra is complete. At approximately six prenatal weeks chondrification of these templates commences, and by the fourth prenatal month a complete cartilaginous template exists for each future vertebra (Scheuer and Black, 2000).

Once the cartilaginous template has been laid down, ossification of each vertebral element can commence, usually at around 10 weeks *in utero* (Scheuer and Black, 2000). There are three primary centres of ossification in each vertebral element, the centra (vertebral body), and two halves of the neural arch. These centres are usually all present for each of the 24 presacral vertebrae (seven cervical, 12 thoracic, and five lumbar) by the end of the fourth month *in utero* (Scheuer and Black, 2000). Ossification of the growing vertebral element is achieved by both endochondral and intramembranous bone formation, and after birth allows an increase in size to eventually produce the adult form (Brandner, 1970; Reichmann and Lewin, 1971; Bogduk, 2005).

Horizontal growth of the centra is via intramembranous ossification, and enables the increase in the antero-posterior and transverse diameter of the vertebral body (Bogduk, 2005). However, it is the longitudinal growth of the vertebral body that is of most interest to this research, as it contributes to an individual's stature through an increase in sitting height. Longitudinal growth refers to the vertical increase in size of the vertebral body. This occurs by endochondral ossification at cartilaginous vertebral growth plates located at the superior and inferior surfaces of the body (Fig.2.4) (Roaf, 1960; Wang *et al.,* 2007; Day *et al.,* 2008) (see Fig.2.4).

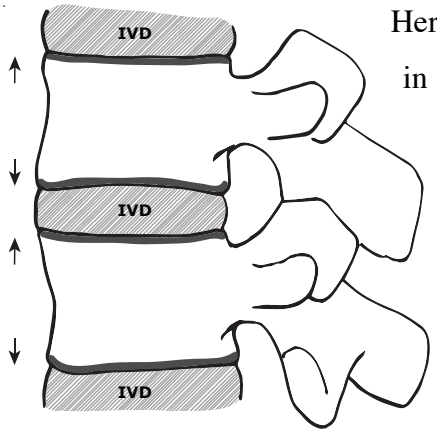

Figure 2.4 – *Process of longitudinal vertebral body growth, depicting the growth plates (in dark grey) superior and inferior to the vertebral body, and the direction of growth (arrows). IVD = Intervertebral disc. Adapted from Bogduk (2005).*

Here growth occurs in a similar fashion to that seen in long bone growth. The chondroblasts of these growth plates undergo the same process of columnar proliferation, differentiation, and mineralisation so that new bone is incorporated onto the superior and inferior faces of the vertebral bodies (Stevens and Williams, 1999; Wang *et al*., 2007). The addition of new bone at the superior and inferior surfaces usually occurs at an equal rate (Bogduk, 2005). This results in the overall increase in vertical height of each vertebral body. A lumbar vertebra would be expected to increase in height from around 5mm to around 15mm between birth and 5 years of age, and will reach up to 25mm by adulthood (Bogduk, 2005). Longitudinal growth of the vertebral bodies is a process that continues throughout childhood and adolescence, but slows in rate throughout the growth period (Bogduk, 2005). As growth slows, the superior and inferior growth plates begin to thin as the ossification process completes (Bogduk, 2005). The growth plate is eventually sealed off by the formation of subchondral bone plates (known as vertebral end-plates) at the superior and inferior surfaces of the vertebral bodies, this completes the longitudinal growth of the vertebral bodies between 18-25 years of age (Bogduk, 2005) However, as with the growth of other skeletal elements, such as long bone length, timing of growth (and age of completion of growth) in vertebral height can be variable from person to person.

Similar to that seen in the growth of long bones, secondary centres of ossification also appear in vertebral body development, however, their contribution to the longitudinal growth of the centrum is debatable. At approximately 12-16 years of age annular rings form at the superior and inferior surfaces of the vertebral body (Scheuer and Black, 2000). These "rings" ossify separately around the rims of the superior and inferior vertebral body margins (Bick and Copel, 1950; Scheuer and Black, 2000). Contrary to past theories regarding the epiphysis-like role of the annular rings, according to Bick and Copel (1950), unlike the secondary centres involved in long bone growth, these rings are not in any way involved in the growth

of the vertebral body. It is only once vertebral longitudinal growth has completely ceased that these secondary centres can fuse with the vertebral body, usually around 18-24 years of age (Bick and Copel, 1950; Scheuer and Black, 2000).

Development of the neural arch differs from that of the vertebral body. Where longitudinal vertebral growth proceeds throughout the entire growth period of an individual, the final size of the neural arch, and in particular the neural canal, is relatively complete early in postnatal growth. Once the three primary centres of ossification of the vertebra have formed, cartilaginous growth plates remain between the two halves of the neural arch (the future spinous process), and between the ventral neural arches and the posterolateral centrum (the neuro-central synchondroses) (see Fig.2.5) (Rajwani *et al.,* 2002; Chen *et al.,* 2006). These growth plates allow for the appropriate increase in size of the vertebral foramen to accommodate growth of the enclosed spinal cord (Chen *et al.,* 2006). The neural arches ossify and grow via intramembranous bone formation on the outer surfaces, whilst bone resorption occurs on the inner surfaces to contribute to growth of the neural canal (Fig.2.5) (Roaf, 1960; Reichmann and Lewin, 1971).

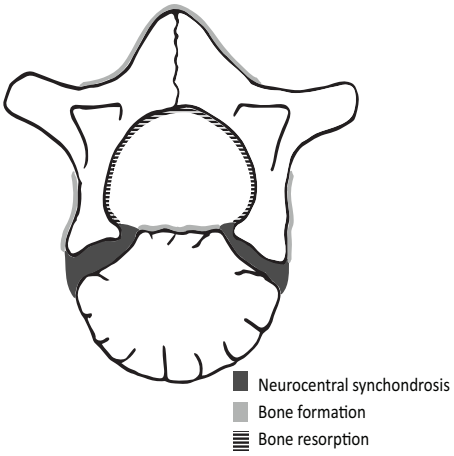

Figure 2.5 – *Process of neural arch growth in a developing vertebra, with the neuro-central synchondrosis (dark grey) and areas of bone formation (light grey) and resorption (black lines). Adapted from Reichmann and Lewin (1971).*

Near completion of growth is marked by the closure of these growth plates by fusion of the primary neural arch centres at the spinous process, and the fusion of the complete neural arch to the vertebral body (Jinkins, 2000). Fusion of the neural arches occurs at various times depending on the location of the vertebrae within the column. In general fusion at the spinous process begins between T10-T12 in the first year of life, and spreads up the column with the cervical region completing fusion between 2-3 years (Scheuer and Black, 2000). The lumbar region is the last to undergo fusion at the spinous process (Scheuer and Black, 2000). However, fusion at the neurocentral synchondrosis occurs primarily in the lumbar region between 2-4 years of age, then the cervical and upper thoracic region between 3-4 years, and lastly in the lower thoracic region between 4-5 years (Scheuer and Black,

2000). Thus, in general, fusion of the neural arches completes growth between approximately 1-2 years of age, and the complete neural arch fuses to the vertebral body at around 2-5 years of age (Scheuer and Black, 2000). The resultant growth pattern of the neural canal is therefore characterised by a rapid increase in the antero-posterior and transverse diameter up until approximately 5 years of age when up to 80% of final adult size has been attained (Hinck *et al.,* 1966). After this point there is little change in size until completion of the neural arch growth period at approximately 9-10 years of age (Hinck *et al.,* 1966; Reichmann and Lewin, 1971; Watts, 2013a,b). Therefore growth of the neural arch and vertebral foramen is complete in early childhood.

2.4 Disruption to the growth process

The influence of the environment on each of the growth processes described above for the long bones and vertebral elements will be discussed in the following sections.

2.4.1 Longitudinal growth disruption

Our final adult height has a strong genetic component, whereby tall parents are more likely to produce tall offspring (Camanni and Ghigo, 2004). However, growth is a process that is heavily influenced not only by genetic factors, but also the physical, socio-economic, climatic, and psychological environment of the growing individual (Duren *et al*., 2013). Thus, due to the ability of the environment to overcome the expression of hereditary characteristics, in adverse conditions an individual may fail to meet their genetic potential in terms of maximum adult stature (Bogin, 1999; Eveleth and Tanner, 1990). As established above, "normal" longitudinal growth depends on the proper functioning of the growth plate, thus any disturbance to the processes that govern the increase in bone length and/or width (e.g. cellular division, differentiation, and maturation) would be subsequently result in growth retardation (Price *et al.,* 1994).

The cellular processes at the growth plate are under the control of the endocrine system (Malina, 1998). Growth Hormone (GH), growth factors such as IGF-1, and Thyroid hormone each target different cells within the growth plate to stimulate their division, proliferation, and maturation (Tanner, 1989; Nilsson *et al.,* 1994; Malina, 1998). It has been stated that "the endocrine system serves as a

mechanism that unifies the genes we inherit and the environments in which we live to shape the pattern of growth of every human being", meaning that hormones and growth factors such as these respond to both genetic and environmental cues (Bogin, 1999; 330).

There are many factors that can contribute to a poor living environment, as well as those that may affect psychological well-being, including malnutrition, episodes of disease, crowded/unfavourable living conditions, and low socioeconomic status (SES) (Bogin, 1999). Adverse living conditions such as these can place stress on an individual/population, which may result in the stunting of growth. Under conditions of acute stress (whether physical or psychological) the stress response is triggered, in which the sympathetic nervous system stimulates the release of adrenaline, noradrenaline, and cortisol (McEwen, 1998; Sapolsky, 2004). This response is designed to bring about the "fight or flight" response, and thus to reinstate homeostasis (Sapolsky, 2004). However, this response also requires the inhibition of the release of GH, which allows for the delay of growth necessary to transfer resources to meet the demands of the stress response (Sapolsky, 2004). For episodes of acute stress this inhibition would not be expected to have a substantial effect on growth, however, when considering the chronic stressors associated with growing up in a detrimental living environment this aspect of the stress response becomes highly significant. Significantly higher levels of salivary cortisol were detected in children of low SES when compared to their counterparts from high SES backgrounds (Lupien *et al.,* 2000). Thus, factors such as low SES can impart heightened levels of stress to individuals for lengthy durations (Lupien *et al.,* 2000). Frequent activation of this stress response, or chronic exposure to stress hormones, can become damaging to an individual's health as important regulatory and developmental functions are deferred and energy is constantly being mobilised rather than stored (McEwen, 1998; Sapolsky, 2004). Therefore, chronic environmental stressors, such as SES and psychosocial factors, not only impair health status, but can also result in prolonged periods of growth disruption until such conditions are alleviated.

Malnutrition/undernutrition is one of the most common causes of growth disruption, as nutritional demands are at their highest throughout the growth period from birth to maturity, particularly during infancy (Martorell *et al.,* 1994; Eckhardt

et al., 2005). A developing individual requires enough energy so that essential processes such as growth, maintenance (basal metabolism), and repair can be undertaken in addition to the energetic demands of day-to-day activity (Bogin, 1999). Under conditions of undernutrition, energy resources become sparse, therefore activities such as physical exertion, maintenance, and repair take priority over growth, and growth will be delayed until nutritional availability is improved. It is also thought that nutrition affects the transcription of GH receptor (GHR) genes, therefore impaired nutritional status would reduce the effect of GH on target tissues, diminish the production of IGF-1, and ultimately disrupt long bone growth (Ulijaszek, 1998; Böhles, 2004). Starvation in rats results in the narrowing of the epiphyseal plate and consequently impairment of longitudinal bone growth. This would suggest that the regulatory functions of GH and IGF-1 on cellular activity at the growth plate were reduced as a result of malnutrition (MacCord, 2009). In addition to the direct impact of the endocrine system on the regulation of the growth process, reduction in nutrient supply may also reduce the number of cells in the growth plate, thus limiting the capacity for growth and consequently slowing the progress of increases in long bone length (Gosman *et al*., 2011). In this way the body may be "adapting" to conditions of present and future undernutrition; slower growth lessens nutritional demands throughout development, and smaller adult heights require less maintenance for the remainder of the life-course (Gosman *et al*., 2011; Stulp and Barrett, 2014).

Studies in modern day populations from developing countries have shown that longitudinal growth retardation can be experienced by more than 30% of children (Eckhardt *et al*., 2005). For example, a study of 114 Otomi Indian infants from Capulhuac, Mexico between 1986-1989 found that between four and six months of age, the diet of infants demonstrated deficiencies in energy and protein intake, leading to a significant decline in growth velocity (Hernandez-Beltran *et al*., 1996). By six months of age the increase in length of the Otomi infants was below approximately 70% of that of the reference sample (Hernandez-Beltran *et al*., 1996). It is also thought that an insufficient maternal diet during gestation, along with episodes of maternal infection, may lead to a restriction of intrauterine growth so that at birth the infant is already classed as small-for-gestational age (SGA) (Hernandez-Beltran *et al*., 1996; Eveleth and Tanner, 1990). These infants are more

likely to fail to meet their genetic potential as intrauterine growth retardation is strongly associated with shorter height in childhood and adolescence, and mothers who do not meet their own genetic potential are far more likely to produce SGA infants (Haeffner *et al*., 2002; Prentice *et al*., 2006). Therefore, the effects of a poor environment on birth size and consequent stunted growth may span multiple generations before it is fully reversed despite alleviation of environmental stressors (Hernandez-Beltran *et al*., 1996; Uauy *et al*., 2011; Sletner *et al*., 2014; Chung and Kuzawa, 2014; Gowland, 2015). This may be evident in populations that demonstrate a smaller mean adult stature.

Acute and chronic episodes of infection can also lead to the stunting of growth, for example children living in areas that suffer from a high prevalence of diarrhea have been shown to demonstrate lower growth rates (Stephenson, 1999). The immune response associated with episodes of disease, and the process of repair following recovery from poor health, requires an elevation in metabolic requirements that necessitates the direction of resources away from the maintenance of growth (McDade, 2003; McDade *et al*., 2008). A study by McDade *et al*. (2008) found that Tsimane' children from Amazonian Bolivia suffering from symptoms of diarrhea or respiratory infection experienced smaller gains in height, particularly affected were those between 2-4 years of age, and those with low energy stores. A synergistic relationship exists between infection, nutritional status, and growth as many infections may lead to a decrease in food intake or impairment of nutrient absorption, and undernutrition can in turn decrease resistance to infectious disease (Martorell *et al*., 1994; Stephenson, 1999; Hoppa and Fitzgerald, 1999). Frequent and severe infection caused by unsanitary environments is a significant cause of the malnutrition faced by children from developing countries (Martorell *et al*., 1980). In addition, there are many diseases that are known to impact nutritional status (e.g. gastroenteritis, pneumonia, measles, upper respiratory tract infections, and intestinal parasites), and in turn there are many diseases that are influenced by nutritional status (e.g. tuberculosis and cholera) (Hoppa and Fitzgerald, 1999). For example, conditions such as diarrhea and respiratory infection have been shown to decrease calorie and protein intake by 19% and 18%, respectively, in children from Guatemala (Martorell *et al*., 1980). It is important to note that both nutritional status and infection are equally linked to other environmental factors such as SES,

demonstrating the complexity of the role that living environment plays in growth.

If there is an improvement in environmental conditions, and if enough of the growth period remains to achieve sufficient catch-up growth, an individual still may be able to meet their genetic potential in terms of height. This requires a substantial increase in the normal growth velocity, therefore for optimal catch-up growth there must also be an increase in energy intake to support it (Eveleth and Tanner, 1990). Large increases in height have been noted in children of low SES when energy intake has been increased, however when nutritional sources are sparse and such improvements unattainable, growth will remain permanently stunted (Eckhardt *et al.,* 2005). The ability of an individual to recover from episodes of growth stunting will also depend on the duration, severity, and recurrence of the stressor. The age at which a child experiences growth stunting is also an important factor, as full recovery following periods of malnutrition may take up to three or four years before the normal growth trajectory is regained (Briers *et al.,* 1975; Alvear *et al.,* 1986).

A wide range of bioarchaeological studies have used measurements of diaphyseal length (acting as a proxy for stature) to identify evidence of growth disruption within past populations (see Saunders, 2008 for an extensive list). In the context of this study, there have been some valuable contributions to existing knowledge of 18th-19th century child health through the analysis of diaphyseal length within skeletal collections.

Lewis (2002a,b) found that non-adults from the post-medieval site of Christ Church, Spitalfields demonstrated stunted growth when compared to those from St Helen-on-the-Walls, York, from the late-medieval period. This study emphasised the significant impact that industrialisation had on child health during this time. A study by Pinhasi *et al*. (2006) including growth data from Anglo-Saxon, late-medieval, and post-medieval sites identified socioeconomic status as the significant influence on the growth capacity of non-adults of this time period, rather than industrialisation/urbanisation itself. Mays *et al*. (2008) investigated the growth of non-adults from Wharram Percy, North Yorkshire (late-medieval rural) and St Martin's Churchyard, Birmingham (post-medieval urban), but found no significant differences in diaphyseal length (Mays *et al.,* 2008). Hughes-Morey (2015) has also recently used adult femoral and tibial lengths from the 18th-19th century skeletal collections of St Bride's lower (low status) and Chelsea Old Church (high status) to

identify status-related differentials in health between the sites. Females from Chelsea Old Church had longer femoral and tibial lengths than females from St Bride's lower, possibly indicative of the harsher environmental conditions, and risks of recurrent poor health and malnutrition, experienced by women living in poverty at this time (Hughes-Morey, 2015).

The results of these studies are clear; industrialisation did indeed impact on growth and, by proxy, child health at this time. However, the root causes are complex, highlighting the importance of social status on growth and development. There is still much to be learnt through the analysis of growth from skeletal collections from the 18th-19th centuries.

2.4.2 Reduced cortical thickness

The analysis of appositional growth (i.e. the acquisition of cortical thickness) has recently been implemented in the study of growth disruption in past populations by Mays *et al*. (2009a). Appositional growth may be more sensitive to the effects of environmental stress, as long bone growth may be maintained through catch-up growth, but at the expense of cortical thickness of the diaphysis (Ribot and Roberts, 1996; Saunders, 2008). This means that disruption to appositional growth is less likely to be veiled by catch-up growth than endochondral growth (Mays *et al*., 2008). Within animal studies it was observed that endosteal resorption was increased under conditions of dietary insufficiency, leading to an increased medullary diameter, and subsequently a reduction in cortical thickness of the long bone shaft (Huss-Ashmore *et al*., 1982). Brandt and Siegel (1978) also found that rodents exposed to cold and heat stress showed a reduction in femoral cortical thickness when compared to control groups. Therefore these studies in animal models show the potential for using measurements of cortical thickness to detect stress in human archaeological populations.

Reduced cortical thickness has been associated in archaeological studies with nutritional stress, and cortical bone loss has been identified in studies of protein-calorie malnutrition in living subjects (Garn *et al*., 1964; Garn *et al*., 1969; Hummert, 1983; Van Gerven *et al*., 1985). In addition, maternal smoking, poor maternal nutrition at 18-weeks gestation, maternal vitamin D deficiency, and high maternal activity have also been linked to deficiencies in neonatal bone density

(Gosman *et al*., 2011). During the period of peak growth velocity in height from approximately 11-14 years of age, the increase in cortical strength involved in appositional growth diminishes (Gosman *et al*., 2011). This cortical thinning has been associated with an increased risk of forearm fracture during puberty (Gosman *et al*., 2011). This presents a period of potential susceptibility to disruption of appositional growth, as resources may be preferentially directed toward an increase in long bone length during this time. Following the period of peak growth velocity, cortical density continues to increase through to skeletal maturity (Gosman *et al*., 2011).

The study of the tibial growth of non-adults within two Medieval cemeteries from Kulubnarti, Sudanese Nubia by Hummert (1983) revealed a reduction in percentage of cortical area where other measurements of length appeared normal. It was suggested that the seasonality of subsistence agriculture lead to a diet that was deficient in quality and quantity, and the interplay of malnutrition and infection lead to this reduction in cortical area (Hummert, 1983). Mays *et al*. (2009a) examined the effect of SES on the longitudinal and appositional growth of non-adults from the 19th century site of St Martin's Churchyard, Birmingham. Interestingly, despite revealing no differences in femoral diaphyseal length between the low and high status groups, deficiencies in cortical thickness were identified within the lower status individuals when compared to the high status individuals (Mays *et al*., 2009a). The results of these studies therefore encourage the inclusion of cortical thickness as a sensitive indicator of stress within bioarchaeological studies.

2.4.3 Potential for disruption of vertebral growth

It is clear that vertebral growth proceeds in a manner similar to that seen in long bone growth, where there is an increase in vertebral body height (VBH) via endochondral ossification (akin to the increase in long bone diaphyseal length) and a system of intramembranous bone formation accompanied by bone resorption in the formation of the neural arch and vertebral neural canal (VNC) (showing similarities to the appositional growth necessary for the increase in cortical width). It is therefore reasonable to expect that the growth processes described previously for the vertebral column would experience similar disruption as that seen in the stunting of long bone growth under conditions of environmental stress. Due to the complex structure of the developing vertebra, where there are three separate

primary centres of ossification that each need to form and grow appropriately, the potential for extrinsic disruption is great. Asymmetric growth at the superior and inferior growth plates has even been a suggested aetiology for the development of scoliosis (Wang *et al.,* 2007; Day *et al.,* 2008). Perturbation to the proper functioning of the vertebral growth plates, for example due to the physiological effects of stress induced by infection and/or malnutrition, could result in the stunting of longitudinal growth of the vertebral body or the disruption of growth in the vertebral canal. This would be expected to be evident as a reduction in vertebral body height and "constricted" neural canal size.

The use of these vertebral measurements in bioarchaeological study is an attractive prospect. As previously described, growth in the VNC is completed early in postnatal growth, with fusion of the neural arch elements by five years of age, and the attainment of adult size by approximately 10 years of age. Consequently, any stress events experienced prior to this completion should be evident as a reduction in antero-posterior and transverse diameters of the neural canal. What makes these measurements potentially very valuable to the study of stress in past populations is that, due to the early completion of growth, there is no potential for the catch-up growth that so often masks disruption to growth in the long bones (Watts, 2011; Watts, 2013a,b). Therefore, evidence of episodes of stress occurring in the vulnerable life stages of infancy and early childhood should be preserved in the dimensions of the neural canal. Measurement of vertebral body height is equally useful. Vertebral elements continue to increase in height throughout the entire growth period until maturity is reached (Bogduk, 2005). Therefore, they may provide a record of stress experienced in late childhood and adolescence, but are also susceptible to the veiling effects of catch-up growth (Clark, 1988). The differing growth patterns of the vertebral neural canal and the vertebral bodies provide a unique opportunity to combine measurements of these vertebral elements to reveal a greater understanding of the stressors experienced by non-adults (Clark *et al.,* 1986). For example, an individual who demonstrates reduced VNC dimensions but not stunting of VBH was likely to have experienced stress during the period of intensive growth of the neural canal (i.e. before around five years of age). The absence of VBH stunting would also suggest that the stress experienced by the individual was either alleviated or they were able to adapt so that catch-up

growth was enabled. However, when both VNC size and VBH are stunted, it suggests that the stress experienced by the individual was more chronic in nature, and continued to affect growth throughout the entire growth period (Clark, 1988; Larsen, 1997).

Unfortunately, studies that consider the effect of the environment on the growth of the vertebral column are scarce. Most growth studies focus on the overall stature or leg length within in a population, and many of these studies do not even mention the contributory factor of sitting height in stature. A study by Tanner *et al*. (1982) set out to investigate whether a secular trend of increase in height in Japanese children and adults between 1957 and 1977 was due to an increase in sitting height or an increase in leg length. They found that in this case sitting height had shown no significant change, and that the increase in height seen over this period of time was due to leg length (Tanner *et al*., 1982). However, more recently, improvements in health, nutrition and living conditions were found to have led to secular improvements in sitting height of Urban school children from Oaxaca, Mexico between 1972 and 2000 (Malina *et al*., 2009). It is therefore hopeful that the analysis of growth in vertebral body height may reveal evidence of growth disruption in past populations, and further study into the use of this indicator in bioarchaeology is certainly warranted.

Studies by Clark *et al*. (1985; 1986) have revealed evidence for the association of reduced VNC size with nutritional and health stress during development, and the lasting effects this stunting may have on neural and immune development and function. Clark *et al*. (1985) previously identified deficiencies in adult neural canal size (stenosis) associated with protein deficient diet in a prehistoric American Indian population. Spinal stenosis is the narrowing of the spinal canal that results in the compression of spinal nerve roots (Botwin and Gruber, 2003). Stenosis of the spinal canal can potentially affect neurological functioning due to the compression of the nerve roots (Botwin and Gruber, 2003). Therefore the fact that this condition could develop as a result of growth disruption in the early growth period is of great interest when considering the effects of the environment on growth and future adult health. Further study by Clark *et al*. (1986) found an association between small VNC size and a decreased life span, which led them to suggest that VNC size could be a useful predictor of adult health due to the

potential effects on neural and immune development (which occurs concomitant to vertebral growth). They therefore concluded that vertebral growth of the neural canal was associated with future adult health and life span (Clark *et al*., 1986). The use of vertebral growth to assess the effects of early growth disruption on adult health and life span has been implemented more recently by Watts (2011; 2013a,b). She also found an association between reduced transverse diameter of the VNC and early adult mortality, which further reinforces the importance of the influence that early growth disruption might have on future adult health and longevity (Watts, 2011; 2013a,b).

Interestingly, growth in vertebral body height and tibial length in the study by Clark *et al*. (1986) was not found to be associated with reduced life span, which highlights the importance of the analysis of skeletal elements that complete their growth early in development (e.g. VNC size) as these are most likely to have a lasting effect on health status and cannot undergo catch-up growth (Clark *et al*., 1986).

2.5 Developmental Origins of Health and Disease Hypothesis

It is clear that when placed under adverse conditions (such as high pathogen load, malnutrition, psychosocial stress, and low socio-economic status) growth may be delayed to ensure that vital bodily processes can continue (Bogin *et al*., 2007). While such reactions are necessary to ensure immediate survival, termed biological trade-offs, they can be costly in the long term. Trade-offs can be defined as the competition between two processes for resources during the development of an individual. When one process is preferentially selected (e.g. those vital for survival) it must therefore come at the expense of the other (Bogin *et al*., 2007). Sacrifices such as these can have lasting effects on the remainder of the life course.

It has already been stated that insufficiencies in maternal diet, or episodes maternal infection, may lead to restrictions in growth *in* utero (Hernandez-Beltran *et al*., 2996; Eveleth and Tanner, 1990). This growth stunting so early in development may hamper the remainder of the growth period, so that many individuals never recover to meet their genetic potential in terms of height (Prentice *et al*., 2006). Individuals of smaller height are also more likely to die from cardiovascular and non-cancerous disease (Stulp and Barrett, 2014). Clearly, in this situation, a

biological trade-off has been made under conditions of maternal health stress to direct resources toward survival and maintenance. However, the excessive growth velocities required for catch-up growth following alleviation of stressful conditions has also been described as a costly process in terms of future health outcomes (Stulp and Barrett, 2014). While height is deemed a selective advantage, those who are taller have higher risks of developing cancer (Stulp and Barrett, 2014). Therefore, the balances between costs and benefits when trade-offs are made will not always result in preferential outcomes in the long run.

The impact that early life stressors, many originating as early as foetal development, can have on future adult health and propensity towards disease has been an area of prolific research in the past 40 years (Barker and Osmond, 1986; Barker, 1991,1992,1994; Ravelli *et al.,* 1998; Roseboom *et al*., 2000; Blackwell *et al*., 2001; Worthman and Kuzara, 2005; Armelagos *et al*., 2009; Bogin and Baker, 2012). Results have lead to the formation of the Barker hypothesis, now known as the Developmental Origins of Health and Disease (DOHaD) hypothesis (Armelagos *et al*., 2009). The development of this hypothesis began with the identification of a correlation between regions that had suffered high infant mortality rates in 1921-1925, with a high prevalence of cardiovascular disease in 1968-1978 (Barker and Osmond, 1986). Since then, the association between high infant mortality has also been connected to a greater risk of ischemic heart disease, strokes, respiratory disease, type 2 diabetes, and hypertension (Barker, 1991,1992,1994). Ravelli *et al*. (1998) revealed that those in their first trimester of gestation during the Dutch Hunger Winter of 1944-45 had an elevated risk of obesity in adulthood. Later studies on this cohort also revealed a decrease in glucose tolerance following undernutrition in late gestation (Ravelli *et al*., 1998), and an increased risk of developing coronary heart disease later in life due to undernutrition in early gestation (Roseboom *et al*., 2000). Blackwell *et al*. (2001) have also identified associations between poor childhood health and cancer, lung disease, cardiovascular conditions, and arthritis. Interestingly, infectious childhood diseases were more associated with heightened risk of cancer and arthritis, while non-infectious diseases correlated more with respiratory conditions (Blackwell *et al*., 2001). Therefore, there is a clear link between poor maternal health during pregnancy, and exogenous stressors in infancy, on future adult morbidity and mortality.

The thrifty phenotype hypothesis has been posited as a potential route through which these trade-offs in future health and longevity occur. Under this hypothesis developmental processes critical to survival and functional outcome are preferentially preserved at the expense of less vital growth processes (Pomeroy *et al*., 2012). For example, under adverse conditions growth processes related to the brain are protected, whereas growth in limb length (particularly the tibia and ulna) is sacrificed (Pomeroy et al., 2012). Other regions with essential functional attributes are the hands and feet (Pomeroy *et al*., 2012). Thus, under stress *in utero* these infants are likely to be born SGA, and shorter lower limbs have shown association with heightened risks of chronic disease in adulthood (Pomeroy *et al*., 2012). Early growth environment not only influences future health outcomes through alterations in body proportions, but also through changes in metabolism (Pomeroy *et al*., 2012). The thrifty phenotype hypothesis also suggests that when nutrient supplies are reduced, permanent alterations in metabolic processes may occur to aid immediate survival in early life, at the expense of future adult health (Worthman and Kuzara, 2005; Armelagos *et al*., 2009). An injurious gestational environment (e.g. under conditions of maternal infection or malnutrition) is thought to not only result in growth disruption *in utero,* but also triggers a response in which physiological and metabolic processes in the developing foetus are "re-programmed" to ensure that they are well equipped for survival in an adverse environment (Worthman and Kuzara, 2005). However, when the post-natal environment is not as adverse as the *in utero* environment, there appears a mismatch in programming which can be detrimental to future health (Kuzawa, 2007; Gowland, 2015). Energy-sparing adaptations in foetal development under conditions of reduced nutrient supply promotes weight gain and rapid growth, which may lead to issues with obesity, coronary heart disease, diabetes, and hypertension later in life when faced with an environment where resources are plentiful (Worthman and Kuzara, 2005). However, responses to stressors are variable on the individual level and as such early deprivation may be overcome if environments later in the developmental period are more favourable (Worthman and Kuzara, 2005; Armelagos *et al*., 2009). While the *in utero* environment has been identified as a key period of phenotypic plasticity in the life course for influencing future health and development, and forms the basis of the DOHaD hypothesis, it is

important to emphasise that the accumulation of stressful conditions throughout the life course is also of significance to determining longevity (Blackwell *et al*., 2001). Exposure to adversity in the early post-natal environment can also impact on future adult health (Gowland, 2015). Exposure to pathogens, environmental toxins, poor nutrition, and psychosocial stress are not experiences unique to gestation, infancy, or even childhood, but in adverse environments will continue to impact on the entirety of the life course (Blackwell *et al*., 2001). As will be described below, this can make the application of the founding principles of the DOHaD hypothesis problematic within the bioarchaeological context.

When the growth period is viewed within the life course perspective, it becomes an essential developmental stage through which an individual will not only increase in size, but will also continue to further social development within their community, strengthen immunoregulatory systems, and importantly progress through puberty and maturation so that they too can reproduce and contribute to future generations (Worthman and Kuzara, 2005; Bogin *et al*., 2007). The key components to the life course are therefore primarily to survive, to reproduce, and to raise off-spring (Worthman and Kuzara, 2005; Bogin *et al*., 2007). Under conditions of stress, survival is threatened, and thus the capability of that individual living long enough to reproduce becomes uncertain. In terms of the developing foetus, in infants, and in children, the primary concern is immediate survival, which comes at cost to growth and maintenance (Bogin *et al*., 2007). However, for adolescents in an environment where there is a high risk of premature mortality, it is beneficial to undergo acceleration in maturity, and therefore reproductive capacity, in order to continue genetic lineage (Worthman and Kuzara, 2005). As limited resources must be stretched between growth, maintenance, and reproduction, trade-offs must occur (Worthman and Kuzara, 2005; Kuzawa, 2007). Onset of puberty and maturity, and therefore reproductive capacity, may be brought forward in the life-course to increase chances of producing off-spring when survival chances are low (Stulp and Barrett, 2014; Sheppard *et al*., 2015). However, this may come at the cost of growth and maintenance, which may have future implications for health (Worthman and Kuzara, 2005; Bogin *et al*., 2007). As these hypothetical individuals live in such risky environments, it is unlikely that they will survive long enough for these health deficits to manifest, which demonstrates a balancing of cost-benefit trade-offs

(Worthman and Kuzara, 2005). However, it is in those robust enough to survive to adulthood, and even older age, that these chronic health problems may begin to arise as a result of these trade-offs.

While it would be of great benefit to the study of the life course within bioarchaeological contexts to assess the impact of early life stress on future adult mortality and morbidity, there are some limitations. The chronic conditions of adulthood associated with the DOHaD hypothesis are not readily identifiable on skeletal remains, if not obscured from the bioarchaeological record entirely (Steckel, 2005). In addition, while the key conditions associated with adult health today are related to cancers, cardiovascular health, diabetes, and respiratory illness, this was not necessarily the case for historical populations (Gowland, 2015). Prior to the late-19th to early-20th centuries, infectious diseases were the most prominent causes of ill health and premature mortality; this may change the selection pressures experienced by these populations (Steckel, 2005; Armelagos *et al*., 2009; Gowland, 2015). Fewer adults would have been expected to survive long enough for the costly effects of biological trade-offs to manifest, as life expectancies were typically much shorter than they are today (Gowland, 2015). However, it may be possible to access evidence of reductions in longevity through the association of earlier age-at-death with presence of indicators of childhood stress. Evidence of early life stress in bioarchaeological study comes from the analysis of "non-specific indicators of stress", such as dental enamel hypoplasias, evidence of growth disruption, reduction in VNC diameter, and cribra orbitalia. The presence of these indicators not only demonstrates that some degree of physiological disruption occurred during childhood, but they are also often preserved in the adult skeleton as indicators of past episodes of stress. It is therefore possible that through the correlation of these indicators of stress with early age-at-death within skeletal collections, connections between early life stress in past environments and social structures can be tied to a reduction in life-span, and by proxy poor adult health. Armelagos *et al*. (2009) identified dental enamel hypoplasia in the permanent dentition, indicative of insults to health from birth to adolescence, as being a particularly promising method by which we can test the DOHaD hypothesis within bioarchaeological contexts. Various studies have highlighted the association between the presence of this indicator of stress with shorter life-spans in past populations (Rose *et al*., 1978;

Cook and Buikstra, 1979, Duray, 1996; Steckel, 2005, Miszkiewicz, 2015). In addition, Steckel (2005) found that cribra orbitalia also showed some association with reduction in life span, as well as those stunted by 20cm in height. Evidence of such a reduction in longevity associated with indicators of early life stress may be indicative of a susceptibility to future health insults, and consequently earlier age-at-death, due to their exposure to physiological disruption in infancy and early childhood (Armelagos *et al*., 2009). However, it cannot be discounted that these individuals simply suffered from an inherent frailty that left them prone to suffering insults to health from infancy to adulthood, therefore this correlation may not necessarily relate to the DOHaD hypothesis (Steckel, 2005; Armelagos *et al*., 2009).

Vertebral dimensions have also been used to access evidence of reduction in longevity as a response to stress in infancy. As discussed in section 2.4.3, the neural canal completes growth early in life, with TR diameter being "locked-in" by approximately 1-2 years of age, and AP diameter by approximately 5 years of age (Jinkins, 2000; Scheuer and Black, 2000). Any disruption to growth in VNC dimensions (resulting in spinal stenosis) also reflects on immune and neural function and development, as the growth of the thymus and lymphatic tissues are also most susceptible to disruption from the prenatal period through to early childhood (Clark *et al*., 1986). As the thymus is responsible for directing the maturation of T cells, vital for the adaptive immune system, episodes of stress suffered during this period of development may therefore impact on immunocompetance, and future immune resistance (Clark *et al*., 1986; Cameron and Bogin, 2012). Thus evidence of deficiencies in VNC dimensions may also reflect impairments to both neural and immune development (Clark *et al*., 1986). Studies by Clark *et al*. (1986) and Watts (2011, 2013a,b) have found an association with reduction in longevity in adults with deficiencies in VNC dimensions (see section 2.4.3).

2.6 Summary

When developmental homeostasis is disrupted it may result in disruption of the mechanisms of growth and development. Thus growth in children is used as a proxy for overall health and well-being within a population, and evidence of disruption may be indicative of suboptimal health-care and child-care strategies,

social and economic conditions, poor environment, poor maternal health, and/or malnutrition. There are many ways by which the growth process can be affected by environmental stimuli, and those environments that negatively impact the health status of an individual/population leave behind these markers of disrupted growth that can potentially be detected in the skeleton. This is valuable to the study of bioarchaeology as detection of these markers within a skeletal collection can help to reconstruct the type of environments that past populations may have experienced. However, this must only be done with due consideration of the cultural constructs of childhood that may have existed within a particular time period and/or geographic region.

While measurement of long bone length has been used extensively in the study of past populations, the other techniques described in this chapter have not been explored sufficiently. The implementation of measurements of cortical thickness and vertebral dimensions within non-adult samples may prove to be valuable additions to the methods currently used to explore episodes of stress in the past. In terms of the life history perspective, the body may have differential reactions to stressors, depending on their timing within the life course (Gosman *et al*., 2011; Stulp and Barrett, 2014). While final adult heights, and formation of the neural canal, are most influenced by early life stressors (those occurring from gestation through to 2-3 years of age) (Kuzawa, 2007; Stulp and Barrett, 2014; Gowland, 2015), cortical thickness and increases in vertebral body height may be more vulnerable to health insults during adolescence. In addition, research relating to the DOHaD hypothesis has revealed that early development within adverse environments are subject to trade-offs for survival and productivity, and this may come at the expense of growth and future adult health. The application of such research to bioarchaeological analyses of childhood health is essential to elucidate whether the prolific evidence of early life stress frequently seen in past populations had repercussions for future adult health. Therefore further study into the use of these alternative indicators is necessary to validate their use in future population studies, and to assess the interaction of growth and the environment within the life course perspective.

CHAPTER THREE

The Industrial Revolution, AD 1750-1850

"More changed in Britain than just the way in which goods and services were produced. The nature of the family and household, the status of women and children, the role of the church, how people chose their rulers and supported their poor, what they knew about the world and what they wanted to know – all of these were transformed." – Mokyr (1985) cited in Hudson (1992: 4-5).

3.1 Introduction

This chapter aims to put the historical background of this thesis in context, so that the data can be analysed in reference to the appropriate social and economic frameworks for the 18th-19th centuries. The condition of urban centres of industry will be described, along with the primary health risks to children at this time. As nutrition has a fundamental influence on the process of growth, the norms for diet throughout the social strata will also be addressed. While this thesis is centred on urban locations within the 18th-19th centuries, the rural hinterlands of this time will also be briefly considered, as some of the sites within this study would have been more sub-urban in nature, and therefore may have had more in common with rural environments than their urban counterparts.

3.2 A Nation of Change

The industrial revolution of the 18th and 19th centuries has been branded as one of the most dynamic periods in British history. The primary change associated with this era was the shift from a predominantly small-scale agricultural population to one of the urban dweller employed chiefly in the manufacturing and service industries (Hudson, 1992; O'Brien and Quinault, 1993). From 1800 to 1850 many of the large industrial centres of England quadrupled in size (Kirby, 2013). It was this transformation in economy, along with the subsequent urbanisation of small towns to large industrialised cities to support the growing population and expanding industry, which spurred on a change in society for both urban and rural populations (Burnett, 1989; Hudson, 1992; O'Brien and Quinault, 1993). Britain's "industrial revolution" was characterised by fundamental changes in agriculture (which allowed for the fuelling of a growing workforce), and rapid population growth,

which in turn increased the demand on industries to produce and support a new consumer lifestyle (Hudson, 1992).

While these factors were all undoubtedly essential for the progression of this changing society, it is the rapid advancement in technology that is distinctive of this era. The introduction of novel machinery increased both the quality and quantity of materials that could be manufactured in a small time scale. The development of the steam engine and its application to a multitude of types of machinery powered the progression of textile manufacture (such as the spinning jenny in 1764, Kay's flying shuttle, Arkwright's water-frame, and James Watt's steam engine), and the use of coal fuelled the iron industry (Engels, 1950; O'Brien and Quinault, 1993). Such innovation ricocheted through the developing industries, increasing efficiency of manufacturing processes, and different regions came to be identified with specific types of manufacture (such as Lancashire, Yorkshire, and Manchester for textiles, and the North-East for coal, engineering and shipbuilding) (Hudson, 1992; Green, 2010a). Consequently, Lancashire, the west riding of Yorkshire, Staffordshire, Warwickshire, and the North-East became some of the most industrialised regions (O'Brien and Quinault, 1993; Green, 2010). London in particular became a centre of industry, employing almost 14.9% of the workers involved in the manufacturing industry by 1861 (O'Brien and Quinault, 1993).

The increasing size of machinery required for manufacture meant that industry moved away from the domestic sphere and into the large, urban-based, factory system that became the prevailing image of the industrial revolution (see Fig.3.1) (Gaskell, 1833; Pike, 1966; Hudson, 1992; Mathias, 2001). With this shift came the migration of the labour force away from the land, and toward the rapidly expanding cities with their promise of new work opportunities in the factories, coal mines, and iron works (Burnett, 1989; O'Brien and Quinault, 1993). Even the working day itself was mechanised in terms of time, no longer were

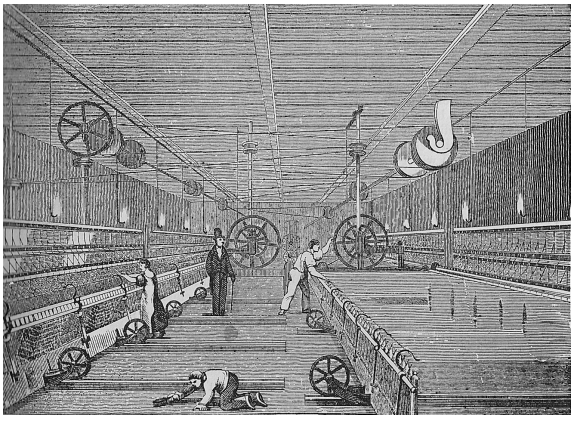

Figure 3.1 – *Image of a Mule Spinning Room from the 1860s, featuring child piecers and scavengers. Image taken from Wilkes (2011:51), originally from Charles Knight's Pictorial Gallery of Arts Vol. I (c.1862).*

working hours determined by daylight, but put in the hands of the factory clock (O'Brien and Quinault, 1993).

However, this new breed of factory work did not guarantee profit for all. New machinery could perform the work of many men, such as the spinning jenny that only required one person for its operation, thus leaving five out of work (Engels, 1950). Therefore the lower classes suffered from seasonal, and sometimes chronic, underemployment or unemployment (Hudson, 1992). Workers were employed purely for man-power, leading to the de-skilling of a large proportion of the population. Children in particular became desirable employees. Mechanisation meant that children could operate machinery to achieve tasks originally performed by men, and women and children could be paid lower wages than adult males (Pike, 1966). With this new economic structure came issues of worker's rights, welfare, and occupational and environmental health concerns, which began to impact on both the political and social systems that governed the country. The nation also had to contend with involvement in recurrent episodes of war (e.g. The Seven Year's War in 1756-63, The War of American Independence in 1775-84, and the long French Wars in 1793-1815) (Mathias, 2001). In terms of the family unit this may have meant the loss of the patriarchal head of the family, and therefore the loss of the main breadwinner of the family (Humphries, 2010).

Whether the industrial revolution (which can be typically classed as occurring between 1750-1850) did indeed see such rapid and unique transformation however, has been hotly contested, with preference for it to be viewed as a more gradual process with origins much earlier than the 18th century (Humphries, 2010). Factories still remained the minority in terms of the numbers of the population employed within them, with many still occupied within agriculture and small 'cottage industries' (O'Brien and Quinault, 1993). For these labourers the steam and coal fuelled industry more affiliated with the mid-19th century was not the reality of everyday life. Nevertheless, the changes wrought in these two centuries undeniably produced a massive shift in society and the condition of the nation, and this inevitably had major repercussions for its littlest members, the children.

The most emotive, and therefore most intensely studied, aspect of childhood in the 18th and 19th centuries is that of the plight of the child labourers. These 'infant slaves' have traditionally been the mascot for the exploitation of the workforce by

factory masters of this time (Goose and Honeyman, 2013). However, recent reconsiderations of how we view these child workers has attributed them a fundamental position within the family economy, within industrial economy and capitalism, and within academic research (Humphries, 2010; Goose and Honeyman, 2013; Kirby, 2013) In this new light they are not to be represented as passive victims, but in the words of Goose and Honeyman "as active in the construction of their own lives" (2013: 3).

Child labour was not unique to industrialisation. Children had always worked as part of the family unit throughout history; in the case of industrialisation it was just the type and extent of work that changed (Pike, 1966; Goose and Honeyman, 2013). The booming population of the 18th and 19th centuries meant that a large proportion of the population were young, with 39% under the age of 14, and 25% between the age of 5-14 years in 1831 (Goose and Honeyman, 2013). This, combined with the high rates of families living in poverty, meant that the widespread employment of children in this era would have been considered normal, practical, and essential to both the survival of poor families (particularly in families that had experienced a loss of one or both of the parents, a commonplace occurrence in an age of high mortality rates) and the economy in general (Humphries, 2010; Humphries, 2013; Goose and Honeyman, 2013). The employment of children did not just begin at the factory doors. Children were engaged in a multitude of occupations, largely in agriculture and service, and their experiences within the world of work were diverse (Humphries, 2010; Goose, 2013; Honeyman, 2013). Exploitation of these young workers was certainly a reality, employers could enforce long hours of work for very little pay, and instances of both physical and emotional abuse were widely reported (Pike, 1966; Honeyman, 2013; Humphries, 2013).

"…cruelties the most heart-rending were practiced upon the unoffending and friendless creatures who were thus consigned to the charge of master-manufacturers; that they were harassed to the brink of death by excess of labour, that they were flogged, fettered, and tortured in the most exquisite refinement of cruelty; that they were, in many cases, starved to the bone while flogged to their work…" John Fielden, The Curse of the Factory System (1836:5-6) – cited in Pike (1966: 78).

However, this was not the experience for all. Many institutions and employers provided a positive working environment whereby children could contribute to the family earnings, or those of the truly destitute (the abandoned and orphaned) could find a life away from the streets and gain some form of education (Honeyman, 2013). While the term "infant slaves" (Pike, 1966: 186) may be misleading in just how young children were beginning their employment, their occupational involvement did start early with some as young as seven or eight years of age (Pinchbeck and Hewitt, 1973; Humphries, 2010; Goose and Honeyman, 2013). In 1819 and 1833 Factory Acts were passed to prohibit the employment of those less than nine years of age within cotton mills, and from 1847 children under nine years of age could no longer be apprenticed (Pike, 1966; Pinchbeck and Hewitt, 1973; Burnett, 1984). The very youngest workers were perhaps those situated in more precarious positions in life, such as the orphaned and abandoned who were reliant on the parish for support and placement within occupations (Humphries, 2010). Placement within a reputable apprenticeship was the most desirable and successful pathway to a steady and employable future (Humphries, 2010). In this system masters and apprentices were bound together with the obligation for the former to provide adequate housing and training within a skilled trade, and the latter to provide diligent work throughout the 5-7 years which was the norm for serving out indentures (Humphries, 2010). Towards the end of the period of the industrial revolution (from the mid-19th century onwards), the employment of children began to lose favour for a multitude of reasons relating to parliamentary reform and the ever-changing economy. Society came to see childhood as an essential period of development that required protection, and education gained prominence with the hope that it would be the key to the improvement of future generations (Cunningham, 2005).

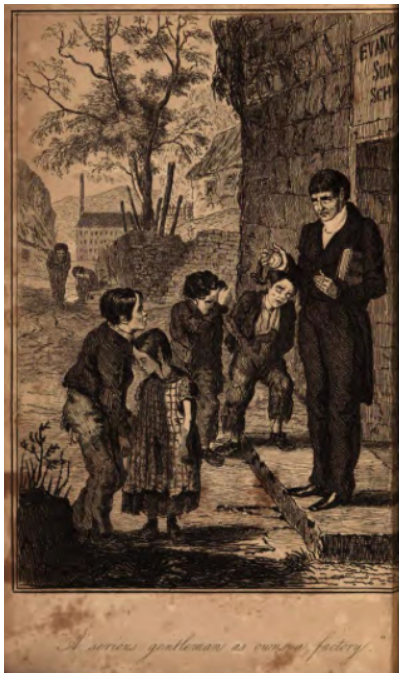

Figure 3.2 – *Image of child labourers entitled "A serious gentleman as keeps a factory". Taken from Trollope (1840: frontispiece).*

Despite the topic of child labour clearly being a key aspect of childhood in the industrial revolution, this thesis will not focus on the intricacies of its effect on child health. The principle reason being that despite the indisputable effect of hard labour on the physical condition (considering the increase in energetic demands placed on the developing body at the onset of working age, and the detrimental exposure to occupational hazards), it would be difficult within skeletal populations to identify those who would have been involved in such activities with certainty. Perhaps most importantly, in terms of the overall growth potential and wellbeing of children who lived during the industrial revolution, there was a far greater influence on the health of the nation's developing inhabitants, whose effects were ubiquitous and habitually devastating, the industrial environment.

3.3 Concern for the "condition of England"

"Fog everywhere. Fog up the river, where it flows among green aits and meadows; fog down the river, where it rolls defiled among the tiers of shipping, and the waterside pollutions of a great (and dirty) city." – Charles Dickens, Bleak House (1853: 1)

As stated previously, the industrial revolution was characterised, and in part fuelled, by its expanding population. Beyond 1750 the population had tripled, and while London had been the only town with more than 100,000 inhabitants in 1801, by 1851 there were 10 towns with populaces exceeding this number (Floud and Harris, 1997; Kirby, 2013). By this time there were over 2.5 million people living within London (Fig.3.3) (Floud and Harris, 1997).

Not only was the population growing, it was also highly mobile. The attraction of the new employment opportunities of the swelling urban centres, combined with changes within agriculture which released a large number of the workforce in the job market (see section 3.5), was enough to pull large proportions of

Figure 3.3 – *Image of a crowded street market, drawn by George Cruikshank for The Comic Almanack, 1838. Image taken from Spitalfields Life (2013).*

the population away from the country and into the towns. This population shift was ever increasing, with approximately 13.5% of people living in towns in 1670, 21% in 1770, and as much as 27.5% in 1801 (Hudson, 1992). However, despite the increased employment opportunities afforded by these large industrial cities, the sheer amount of people wanting to live and work in urban districts meant that they quickly became areas of severe overcrowding, poor sanitation, and inadequate ventilation (Kay, 1832; Engels, 1950; Report of the Commissioners, 1845a). This not only had a detrimental effect on the health of the cities' inhabitants, but also had a significant strain on the amenities available to them, particularly that of poor relief and healthcare.

"Day after day, such travellers crept past, but always, as she thought, in one direction – always towards the town. Swallowed up in one phase or other of its immensity, towards which they seemed impelled by a desperate fascination, they never returned. Food for the hospitals, the churchyards, the prisons, the river, fever, madness, vice, and death – they passed on to the monster, roaring in the distance, and were lost." – Charles Dickens, Dombey and Son (1896: 386).

Housing to support the growing number of families requiring accommodation was deficient both in availability and condition (Oxley, 2003; Broad, 2010; McEwan and Sharpe, 2010). Large families were required to inhabit extremely small residences, often only consisting of one or two rooms (Gaskell, 1833; McEwan, 2010). "One wretched garret" could contain up to 10 people (Archer, 1865: 13). Engels stated that "…in this overcrowding it is nothing unusual to find a man, his wife, four or five children, and, sometimes both grandparents, all in one single room, where they eat, sleep, and work" (1950: 29). The houses themselves were frequently in a very deprived state, and were described by Thomas Archer in 1865 as "…ruininous tenements, reeking with abominations…" (Archer, 1865: 9). The poorest families were required to live in squalor within "…cottages separated by narrow, unpaved, and almost pestilential streets…" (Kay, 1832: 10), where "Foul channels, huge dust-heaps, and a variety of other unsightly objects, occupy every space, and dabbling among these are crowds of ragged dirty children who grub and wallow, as if in their native element" (Mayhew, 1861; 140). A committee appointed by the Special Board of Health in Manchester found that of 687 streets inspected, 112 were ill-ventilated, and 352 contained heaps of refuse and

stagnant pools (Kay, 1832). It was also found that out of 6,951 houses inspected, approximately 20% were damp, and approximately 32% were without privies (Kay, 1832). Dwellings continued to deteriorate as the repair of the "…dark, steep, broken, and filthy stairs, the black and crumbling ceilings, the bare and broken walls…" (Archer, 1865: 12) was not high on the agenda of the landlords, and the dilemma of how to dispose of waste materials was yet to be solved (Fitzgerald, 2011). Access to water for drinking, cooking, and cleaning was usually via a communal pump located on the street (Fitzgerald, 2011). Contamination of a water source could therefore be of dire consequences for an entire community. The lack of drainage meant that the fate of waste products such as rubbish and excrement (both human and animal) was disposal in a cesspit or often on the street (Gaskell, 1833; Engels, 1950).

> *"The whole of the washings and filth from these consequently are thrown into the front or back street, which being often unpaved and cut up into deep ruts, allows them to collect into stinking and stagnant pools…it is in a very short time completely choked up with excrementitious matter." - Gaskell (1833, 135).*

Consequently, the city streets were not any more hygienic than the housing with "…their dirty pavements…" and "…poisonous exhalations from choked drains, and reeking cesspools and decaying filth…" (Archer, 1865: 25).

In addition to the severe overcrowding and poor sanitation, the population also had to contend with engulfing levels of air pollution generated by the coal-fuelled industry that so often dominated cities such as London, a city dubbed the "epitome of evils of the urban environment" (O'Brien and Quinault, 1993: 236). The transition to coal as a primary fuel source resulted in extremely high levels of atmospheric pollution (see Fig.3.4) (Brimblecombe, 1978; Green, 2010a). Mayhew stated that in the few years preceding his publication (in 1861) London had imported on average 3,500,000 tons of coal per year (1861: 81). This resource was devoured in both industry and within the home, and subsequently its emissions filled the air, leading to "sooty showers", "dreary fogs", and "suffocating mists" with vast effects on health (Brimblecombe, 1978: 116). Even at the time it was recognised that "the air of cities is not so friendly to the lungs as that of the country, for it is replete with sulphureous steams of fuel", and that "the air of cites is

unfriendly to infants and children" (Arbuthnot, 1733; 208). This dense pall of pollution also prevented sunlight from piercing through to the streets below, which had severe effects on the developing inhabitants and will be explored further in section 3.3.

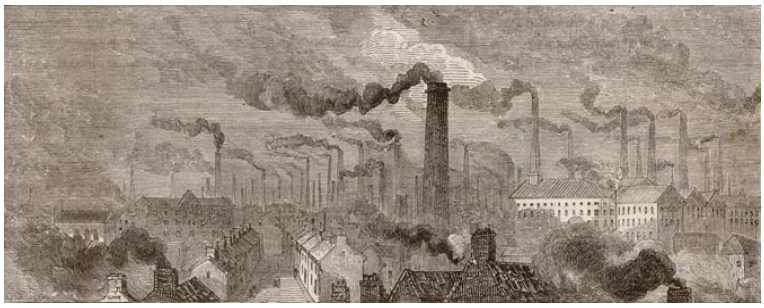

Figure 3.4 – *The polluted skies of Manchester c.1870. Image taken from BBC (2014).*

All in all, this combination of detrimental environmental factors produced what must have been a cacophonic setting, unfavourable for both health and development of the children.

"…the clustered roofs, and piles of buildings, trembling with the working of engines, and dimly resounding with their shrieks and throbbings; the tall chimneys vomiting forth a black vapour, which hung in a dense ill-favoured cloud above the housetops and filled the air with gloom; the clank of hammers beating upon iron, the roar of busy streets and noisy crowds, gradually augmenting until all the various sounds blended into one and none was distinguishable for itself…" - Charles Dickens, The Old Curiosity Shop (1853: 198).

These streets were indeed "teeming with disease and death" (Archer, 1865: 13). It has been frequently noted that death rates were much higher in urban areas than in their rural counterparts (Hudson, 1992; O'Brien and Quinault, 1993; Woods, 2003), and in 1801 the life expectancy of town dwellers was 31 years of age, compared to 41 years of age in rural environs (Hudson, 1992). Infant mortality was high and commonplace in this time, and the loss of a sibling was an unexceptional experience for most children – "Families were built up in this fashion, with so many defunct and so many living" (Burnett, 1984: 35). The London Bills of Mortality have revealed that around half of the deaths between 1730-1779 were those less than five years of age (Pinchbeck and Hewitt, 1969), and throughout the 19th century averaged around 149 deaths per 1,000 live births (Dyhouse, 1978).

The high mortality rates of both the adults and children, and the serious matter of the sanitary conditions became the concern of social reformers of the time, who fought for the improvement of the country. Edwin Chadwick was a major supporter of the campaign to improve the urban environment, reporting on the sanitary conditions of the labouring population of Great Britain in 1842 (Chadwick, 1842). Chadwick believed that improvements to water supplies, drainage systems, and overcrowding of burial grounds would primarily improve public health, and subsequently relieve some of the pressures of those living in poverty (Porter, 1995). He felt that healthy, happy workers would mean greater efficiency in the workplace, more profit to the employer, and overall a decrease in poor rates required to support the destitute (Chadwick, 1842). While sanitary acts were gradually passed to improve drainage and general hygiene of the streets, progress was slow and problematic, with Engels stating in 1892 that "Whole districts which in 1844 I could describe as almost idyllic, have now, with the growth of the town fallen into the same state of dilapidation, discomfort, and misery" (1850: viii).

3.3.1 "We must begin with the children"

(Pinchbeck and Hewitt, 1973: 634)

This growing social awareness of the harmful living conditions of the labouring classes spread to the plight of the working children, which resulted in a series of growth studies by the Parliamentary Commission (Tanner, 1981). The practice of using data related to the growth of children to identify populations that may be suffering from suboptimal health is termed auxological epidemiology, and in this period was used by reformers such as Chadwick to inform the parliament on matters concerning public health and sanitary reform (Tanner, 1981; Floud, 1990). In the 1830s the mean height of boys working in factories was smaller than that of modern day third-world populations (Ulijaszek *et al.,* 1998). There were a multitude of studies which found deficiencies in the heights of urban factory children compared to those of differing occupation, particularly in agriculture (Kirby, 2013). An investigation in 1873 found that urban children from the North of England between 8-14 years of age demonstrated deficits in height from 11 years of age when compared to their rural counterparts (Kirby, 2013). Significantly, these children were not yet employed in factory work, emphasising that the height

deficiencies seen amongst urban factory children were more likely a product of the urban environment rather than child labour practices per se (Kirby, 2013). As well as general deficiencies in height, urban factory children were also prone to delays of up to a year to eighteen months in the onset of the adolescent growth spurt. This persistently poor growth seen in industrial populations, particularly those of lower socioeconomic status, has been unanimously attributed to chronic undernutrition (and malnutrition) and episodes of disease resulting from the unsanitary living conditions, as well as socioeconomic and occupational pressures (Sharpe, 2012).

This established link between nutrition, infection, socioeconomic status, and growth has proven to be a popular route through which historians and economists have attempted to discuss topics such as the "standard of living" during this time of significant political and economic upheaval. Large collections of stature data from sources such as the Marine Society recruitment records, prison records, and school records have been utilised in an attempt to access evidence of the effects of urbanisation, diet, disease, occupation, and inequality on heights of children and adults to map "trends in biological wellbeing" in the 18th and 19th centuries (Nicholas and Steckel, 1991; Voth and Leunig, 1996; Oxley, 2003:623; Humphries and Leunig, 2009). This is known as anthropometric history. Floud *et al*. (1990) assessed records from the Marine Society of London where heights were recorded on the admission of its boys from 1770. They commented that the boys of the Marine Society, (a charity set up to recruit poor boys to the navy) were "extraordinarily short", with 13 year olds born between 1753-1780 being on average 25.4cm shorter than those measured in the 1960s (Floud *et al*., 1990). These data were also compared against stature data collected from sons of the upper classes admitted to the Royal Military Academy at Sandhurst in the 19th century. They were found to be on average taller than the boys of the Marine Society, intimating an inequality in height, and therefore living standard between the classes at this time (Floud *et al*., 1990). However, it is important to note, that despite this advantage in height of the upper class boys, they were still deficient in height by today's standards. Following the results from these studies, and further results from Floud *et al*. in this volume regarding regional differences in heights between soldiers and marines in the 18th-19th centuries, it was concluded that urbanisation was a strong contributor to falling stature values in the 19th century, and a

considerable social inequality existed due to the heightened exposure of the lower classes to the most adverse aspects of life associated with urban living (1990). Stature data from English and Irish convicts transported to Australia between 1817-1840 also revealed a drop in heights of both English rural and urban males from 1780, delays to the growth-spurt in adolescents, and a prolonged growth period, indicating falling standards in living for English workers in this time (with a more pronounced decline for urban workers) (Nicholas and Steckel, 1991).

Following the investigations of the commissioners, new legislation lead to improvements in public health through various Factory Acts, and Sanitation and Housing Acts (Tanner, 1981). The use of measurements of height have since then been used to form standards of "normal" growth to inform public health officials, and increase the understanding of the effects of poor nutrition and disease upon child health and development within developing countries (for example the growth standards set by WHO; Eveleth and Tanner, 1990).

3.4 Diet

The ever-expanding industrial centres of the later half of the 18th century and early 19th century required adequate food sources to fuel their swelling workforces, placing heavy demands on food production, and resulting in the doubling of agricultural output from 1750-1850 (Drummond and Wilbraham, 1994; Horrell and Oxley, 2012). For London, a nucleus of industry and manufacture, the demand for such provisions was enormous, and a large proportion of the agricultural production in the South of England worked to supply the metropolis (O'Brien and Quinault, 1993). This reliance of the urban workers on the surrounding agricultural regions for the supply of food came with some significant issues. The transportation of foodstuffs such as meat and vegetables was a slow process when compared to today's standards. Until the establishment of the canals in 1755, and railway systems in 1830, food arrived in the city by carts via the roads, "few of them were better than deeply rutted tracks, ankle deep in dust in summer and veritable bogs in winter" (Engels, 1950; Drummond and Wilbraham, 1994: 185). In the absence of methods of refrigeration, by the time these supplies reached the urban consumers the quality was frequently poor (Fitzgerald, 2011).

"The potatoes which the workers buy are usually poor, the vegetables wilted, the cheese old and of poor quality, the bacon rancid, the meat lean, tough, taken from old, often diseased, cattle, or such as have died a natural death, and not fresh even then, often half decayed."- Engels (1950: 68).

While the wealthier classes may have been able to afford access to fresher produce, the poorer residents could not afford this luxury, therefore their diet would have been suboptimal in nutritional value, and in the case of meat supplies, fetid and foul smelling (Harrison and Gibson, 1976; Fitzgerald, 2011; Horrell and Oxley, 2012). Market gardens were also often cultivated in the surrounding areas of London to supply the city's markets with fruit and vegetables to sell to the urban dweller (Drummond and Wilbraham, 1994). However, the quality of this produce was not highly regarded, and the cleanliness of the handling and transport of the produce prior to sale came with certain misgivings from some -

"It was but yesterday that I saw a dirty barrow-bunter in the street, cleaning her dusty fruit with her own spittle; and who knows but some fine lady of St. James's parish might admit into her delicate mouth those very cherries which had been rolled and moistened between the filthy, and perhaps ulcerated, chops of a St. Giles's huckster?" – from The Expedition of Humphrey Clinker, T. Smollett, 1771, cited in Drummond and Wilbraham (1994: 192).

The seasonality of the food supplies provided by the farms, and rising prices of staple foodstuffs (due to a series of bad harvests throughout the period, the continuing wars, and the much maligned Corn Laws of 1815), meant that the more readily available cheap, filling foods became popular with the working classes, and bread, potatoes, tea, and sugar became staples of the diet (Gaskell, 1833; Drummond and Wilbraham, 1994; Horrell and Oxley, 2012). Kay stated that "The tea is almost always of a bad, and sometimes of a deleterious quality, the infusion is weak, and little or no milk is added" (1832: 8), and between 1814-1832 the quantity of sugar consumed increased by approximately 83% (Gaskell, 1833). In the poorest families, meals were frequently meagre, monotonous, and far from nutritionally balanced (Burnett, 1984). Some families could occasionally afford meat, but this was usually a rare event, often limited to once a week at most and frequently not at all (Kay, 1832; Gaskell, 1833; Engels, 1950; Horrell and Oxley, 2012). This type of diet may have provided a quick fix in terms of hunger suppression, but in the long

term would have led to some significant deficiencies in other vital aspects of nutrition, as well as leading to considerable issues with dental diseases such as caries due to the high sugar diet.

"Breakfast is generally porridge, bread and milk, lined with flour or oatmeal. On Sunday, a cup of tea and bread and butter. – Dinner, on week days, potatoes and bacon, and bread, which is generally white. On a Sunday, a little flesh meat; no butter, egg, or pudding. – Tea-time every day, tea, and bread and butter; nothing extra on Sunday at tea. – Supper, oatmeal porridge and milk; sometimes potatoes and milk. Sunday, sometimes a little bread and cheese for supper; never have this on week days. Now and then buys eggs when they are as low as a halfpenny apiece, and fries them to bacon. They never taste any other vegetable than potatoes…" - The diet of the family of a spinner in Manchester with five children, 1844 (Pike, 1966: 52-53).

While this description of the diet of a labouring family in Manchester evidently lacks in the fruit, vegetables, and fresh meat necessary for a well-balanced and nutritious diet, the presence of dairy products such as cheese and milk may have staved off the more serious consequences of nutritional deficiency. This diet was more typical of the inhabitants of the northern counties, which as can be seen in Figure 3.5 maintained a better quality of nutrition than their southern counterparts, particularly in London (Horrell and Oxley, 2012). Regional variations in diet inevitably occurred. Potatoes, oatmeal, and milk were most popular and widely available in the North, but inhabitants of the southern counties tended to be more reliant on bread (often omitting milk entirely), whether due to lack of availability of other staple food sources or general preference (Shammas, 1984).

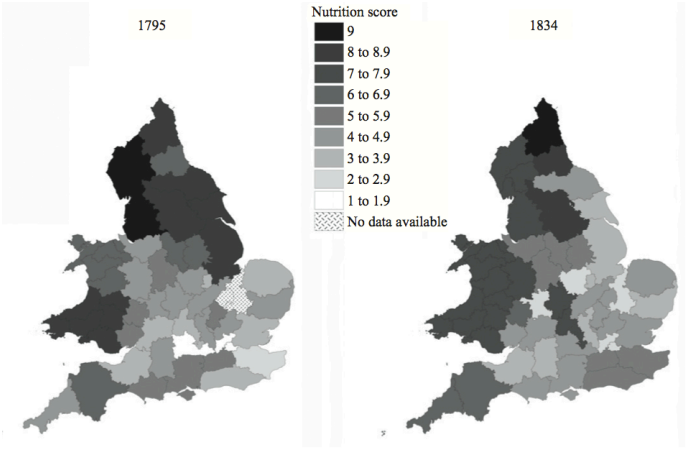

Figure 3.5 – *Nutritional quality of regional diets in England and Wales 1795-1834. Taken from Horrell and Oxley (2012: 1363).*

Allocation of food resources within a struggling family was often weighted towards the "breadwinners", the wage earners contributing to the family income. This was usually the father, who required priority in the allocation of resources within the household to allow him to continue to work and support the family (Burnett, 1984; Horrell and Oxley, 1999; Horrell and Oxley, 2012). While access to niceties such as cheese and occasional supplies of meat may have been within the means of some families, priority of these dietary supplements may have gone to the father, meaning the children and mother would have subsisted on a similar diet of tea, sugar, bread, and potatoes (Horrell and Oxley, 2012). This meant that the children of the poor were more than likely chronically malnourished, and hunger has been described as an enduring experience of childhood in working-class autobiographies (Burnett, 1984; Humphries, 2010).

The comparative wealth of the middling and higher classes meant that they could afford improvements in their dietary arrangements. Fruit and vegetables were widely available to these classes (although some aversion to these existed amongst the upper classes), as well as dairy products, and meat including both beef and poultry (Drummond and Wilbraham, 1994). However, such comforts were not always extended to their children due to unfortunate beliefs at the time in the differing dietary requirements of children and adults. It was a common belief that fruit and vegetables were bad for children, leading to a susceptibility to vitamin C deficiency (described further in section 3.4.3) (Bayne-Powell, 1939). While older beliefs that vegetables were a potential cause of disease and disorders of the bowel were beginning to be dispelled, it was still thought that excesses of certain vegetables and fruits could produce a bodily imbalance (following on from the humoral theory still in place within medicine) (Drummond and Wilbraham, 1994). For example, some believed that cucumbers and plums in excess might lead to cholera (Drummond and Wilbraham, 1994).

Infants in particular were vulnerable to the effects of the poor urban diet (within all levels of society) due to their reliance on maternal nutritional status, fashionable child-care practices, and inadequate weaning diets. Infants are reliant on their mother's breastmilk for passive immunity against the pathogens in the external environment (Cunningham, 1995; Katzenberg *et al.*, 1996; Lewis, 2002a). The process of weaning exposes the infant's immune system to infectious agents such as

bacteria and parasites, and those who suffer from malnourishment from an improper weaning diet are more likely to succumb to infection (Stuart-Macadam and Dettwyler, 1995; Lewis, 2002a). In the 18th and 19th centuries, employment of women in factories meant that weaning ages were reduced, and among the higher classes breastfeeding was considered inconvenient (Burnett, 1984; Perkin, 1993; Stevens *et al*., 2009). Breast milk was often substituted with "pap", usually made by mixing flour/bread with cow's milk or water (Wickes, 1953; Hardy, 2003). This insufficient diet would have led to malnourishment and, with the addition of the high levels of infectious disease associated with the industrial environment and from bacteria thriving on improperly cleaned feeding utensils, would have contributed greatly to the gross morbidity and mortality of infants. The quality of the constituents of the substituted infant diet is also an important consideration. Cow's milk was a significant source of infection. Milk transported into the city often arrived in a soured and impure state, and cows kept within towns were housed in unhygienic conditions propagating disease amongst the animals (Atkins, 1992; Drummond and Wilbraham, 1994). Cow's milk also provided a particularly good mode of transmission for diseases such as bovine tuberculosis, typhoid, and dysentry (Atkins, 1992; Drummond and Wilbraham, 1994).

Adulteration of food was also a regular occurrence, with many incidences of toxic substances being added to foodstuffs to either increase their quantity and size, or give them a more appealing colour. For example, copper and lead were used as colourings for sweets, copper to enhance the colour of green vegetables, pepper was mixed with floor sweepings, waste products of soap-boiling mixed with sugar, cocoa adulterated with fine brown earth, and the addition of alum to flour in the production of bread (see Fig.3.6) (Engels, 1950; Drummond and Wilbraham,

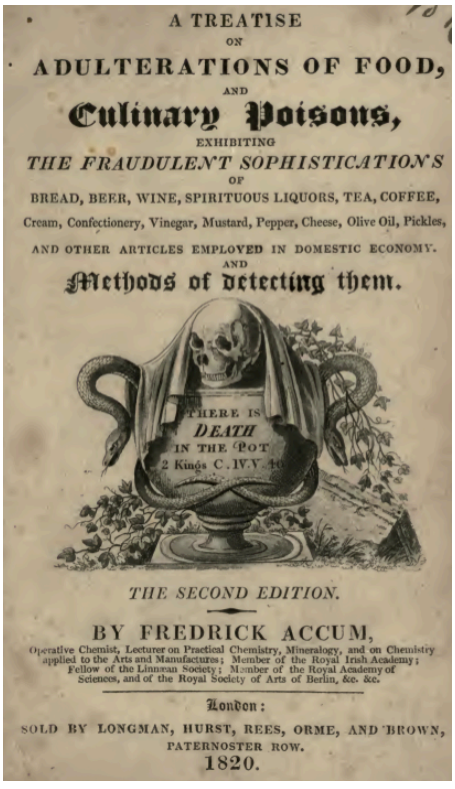
A TREATISE
ON
ADULTERATIONS OF FOOD,
AND
Culinary Poisons,
EXHIBITING
THE FRAUDULENT SOPHISTICATIONS
OF
BREAD, BEER, WINE, SPIRITUOUS LIQUORS, TEA, COFFEE,
Cream, Confectionery, Vinegar, Mustard, Pepper, Cheese, Olive Oil, Pickles,
AND OTHER ARTICLES EMPLOYED IN DOMESTIC ECONOMY.
AND
Methods of detecting them.
THERE IS
DEATH
IN THE POT.
2 Kings C. IV.V. 40
THE SECOND EDITION.
BY FREDRICK ACCUM,
Operative Chemist, Lecturer on Practical Chemistry, Mineralogy, and on Chemistry applied to the Arts and Manufactures; Member of the Royal Irish Academy; Fellow of the Linnæan Society; Member of the Royal Academy of Sciences, and of the Royal Society of Arts of Berlin, &c. &c.
London:
SOLD BY LONGMAN, HURST, REES, ORME, AND BROWN,
PATERNOSTER ROW.
1820.

Figure 3.6 – *Front page of the publication by Frederick Accum in 1820, demonstrating the widespread reported adulteration of numerous consumables at this time. Image taken from Accum (1820: frontispiece).*

1994; Snow, 2003; Fitzgerald, 2011; Horrell and Oxley, 2012). As bread and flour was such a major constituent of not only the pap fed to infants, but also the general diet of older children, this adulteration may have significantly risked the health of these growing individuals.

Therefore, it is clear that the diets available to and utilised by families from all levels of the social strata were more than likely deficient to some degree. The diet of the urban family therefore frequently would not have been able to provide a sufficient level of nutrition to their children. It is likely that many children were both malnourished and undernourished (diets suffering in both quality and quantity), leading to deficiencies in protein and essential micronutrients. Shammas (1984) calculated that rural calorie intake was approximately 2100-2500 calories/day in the South of England, and 2800-3200 in the North. Though representative of the rural worker, it is expected that these intakes should be similar, if not preferable to that of urban workers (see section 3.5). Energetic expenditure amongst working class families would be expected to be high, due to the requirements of physical labour. The accumulation of physical activities from moderate to heavy physical labour, long days spent on the feet from occupations as domestic servants, shop-workers, or within factories, alongside the addition of housework meant that men and women spent approximately 50-60 hours per week engaged in physical activity (Clayton and Rowbotham, 2008). Clayton and Rowbotham (2008) calculated that calorific requirements for this level of energy expenditure were 3000-4500 calories/day for men, and 2400-3500 calories/day for women. Required caloric intakes may have also been heightened in women who were pregnant or lactating, and growing children also had high-energy requirements that the average working-class diet may not have been sufficient to support (Shammas, 1984). Shields (2015) calculated that workhouse diets (consisting primarily of beer, bread, butter, and cheese) only provided 82% of the daily-recommended calorie intake for inmates when accounting for physical activity requirements. While the average working-class household would not have necessarily subsisted on "starvation diets" such as these (Shields, 2015), it is likely that the physical requirements from occupational and household activities outweighed net nutritional income. Poor diet can lead to disorders related to specific vitamin deficiencies, stunting of growth as diminished resources are directed

towards survival, and also a suppression of the immune system leading to weaknesses in both defence against infectious agents and recovery from episodes of disease.

As improvements to agricultural processes and transportation continued throughout the period, and with the cessation of trade restrictions following the end of the French wars, the availability of fresh produce grew (Engels, 1950; Bayne-Powell, 1939). Cattle could be fed during the winter months with the implementation of winter feeds, such as turnips, enabling the availability of meat through all seasons (and negating the reliance of salting meat for long term preservation), and the importation of new types of fruit and vegetable brought great benefits to the diet of the English (Bayne-Powell, 1939; Fitzgerald, 2011; Drummond and Wilbraham, 1994).

The industrial environment was clearly one of great detriment to its inhabitants. The conditions of overpopulation, malnutrition, lack of sanitation, and pollution exposed all those who resided in urban areas to a high risk of disease and death. It was the children of these populations who felt the harshest effects of these industrial conditions. Not only did they have to contend with the spread of the more commonplace diseases of childhood, but also faced an assault from those which thrived in such poor conditions. It is the plight of the children, and the health insults they faced throughout their precarious (and sometimes short) journey through childhood to maturity, which will be considered in the next section.

3.5 Childhood diseases and the epidemic streets

The detrimental industrial environment inevitably had a significant effect on the health of its inhabitants, and the Report of the Commissioners in 1845 stated that amongst the poorer classes "…their vigour and health are undermined, and their lives shortened by the deleterious external influences…" (1845a: p4). The effects on infants from such an environment were said to be "peculiarly severe", as evidenced by the high infant mortality rates seen within urban centres at this time (Report of the Commissioners, 1845a: p5; Hardy, 1992). It was reported that -

> *"…more than one-half of the offspring of the poor…die before they have completed their fifth year. The strongest survive; but the same causes which destroy the weakest, impair the vigour of the more robust; and hence the children of our*

manufacturing population are proverbially pale and sallow…" – Kay (1832: 42-43).

In Manchester, more than 57% of working-class children died within the first five years of life, compared to approximately 20% in the higher classes (Engels, 1950). Children of the 18th and 19th centuries, like today, had to face a multitude of illnesses as they passed through the development of both their physical form and their immune system. However, due to the fact that their environmental circumstances were more often than not far from nurturing, many children would have found themselves facing these threats in an already weakened state.

Whether due to malnutrition, a history of chronic illness, or more likely a combination of both, children were at an extremely high risk of mortality from diseases which today are seen as unexceptional, and part of the experience of growing up. For example, chicken pox (seen today as almost a rite of passage of childhood, with adults often choosing to "share" this condition amongst their children to confer later immunity), measles (today regularly vaccinated against in early life), and even the process of teething were experiences that were of great anxiety to the parents of afflicted infants. The fear surrounding childhood diseases at this time were not reserved for the poor, ill health struck regardless of class, and in the pre-antibiotic era its course was unpredictable and indiscriminate.

3.5.1 Infectious disease

"As season follows season, so does disease follow disease in the quarters that may be more literally than metaphorically styled the plague-spots of London" – Mayhew (1849: 4).

The poor sanitation, damp housing, and severe overcrowding of the cities unsurprisingly favoured the spread of a range of infectious "filth diseases" such as typhoid, typhus, and cholera (see Fig.3.7) (Hardy, 1993; Crawford, 2010). In the fifth report of the Poor Law Commissioners from 1839, the spread of "fever" was a problematic epidemic that caused great suffering to the population, and was attributed to the "…filthy, close and crowded state of the houses…" and "…the total want of drainage…" (Young and Handcock, 1956; 769). Children were continually exposed to the endemic (often epidemic) diseases of childhood including whooping cough, scarlet fever, measles, and diphtheria, and not forgetting

the ever-prevalent risk of infantile diarrhoea (Bayne-Powell, 1939; Hardy, 1992; Hardy, 1993; Lewis, 2002a). Fatalities from these diseases occurred up to four times more frequently within the towns than in the country (Engels, 1950). The extensive air pollution from the smoke and chemicals pouring into the atmosphere (by-products of the coal burning and manufacturing processes) promoted respiratory infections such as bronchitis and pneumonia, and tuberculosis was a constant risk which brought with it chronic poor health (Hardy, 1993; Kirby, 2013). These deficient living conditions were extremely hazardous to the growing child, and alongside paucities in nutrition (see section 3.4) they could result in serious failings in immune status, which left these young individuals continually susceptible to the next insult to health if they were so lucky to survive. There was an element of seasonality to the mortality of children living in London. While winter proved to be the most hazardous season to health for the entire population, summer was particularly "hostile to infants and children", with child mortality peaking in the month of August (Reid, 1845: 238; Crawford, 2010). Winter brought with it the risk of respiratory disease and summer potentially fatal bacterial infections, particularly infantile diarrhoea, so that at no point within the year were the very young safe from the myriad of health insults bred by the harmful urban environment (Crawford, 2010). In 1741-99 the diseases which proved most fatal to children aged 1-5 years in the foundling hospital were smallpox, measles, consumption (tuberculosis), and whooping cough, along with convulsions and fits (Levene, 2012). Reported mortality from selected diseases from 1844-1853 (in order of highest frequency first) revealed that phthisis, pneumonia, bronchitis, typhus, convulsions, scarlatina, and whooping cough were the most frequent causes of death in London (Smith, 1854). Some of these infectious diseases, and how they flourished in the industrial environment, will be explored further.

Figure 3.7 – *Punch cartoon depicting the spread of epidemic disease in London in 1858. The three diseases depicted are diphtheria, scrofula, and cholera. Taken from the British Library (2015b).*

Typhoid is transmitted via food or water that has been contaminated by the faeces of a carrier of the typhoid bacterium *Salmonella typhi* (Waddington *et al.,* 2014) Those afflicted with typhoid all experience fever, diarrhoea, headaches, nausea, rashes, and abdominal pains (Waddington *et al.,* 2014). One of the primary sources of this disease in the 18th and 19th centuries was through contaminated water sources. Due to the limited access to fresh water in most communities, many streets would be reliant on one water source, often a water pump, therefore the potential for widespread infection of this bacterial infection was high. While typhoid was prevalent amongst poorer communities and closely associated with detrimental living conditions, this did not necessarily mean that the higher classes were protected from it. In this time of appalling sanitation contamination of a water supply was a threat faced by all (Hardy, 1993; Lane, 2001; Steere-Williams, 2010). For the inhabitants of London, a large proportion would have been reliant on the Thames for both their water supply, and a means for disposal of sewage (Hardy, 1993). As filtration systems were not sufficiently implemented by water companies before 1840, it is not surprising that the spread of typhoid fever was endemic within local communities in the 19th century (Hardy, 1993). Infection could also occur through food, and the significance of milk as a particularly good source of transmission was recognised in the 1870s for not only typhoid, but also scarlet fever, diphtheria, and tuberculosis (Atkins, 1992; Steere-Williams, 2010). This form of transmission made it a significant risk to children, and also provided another route by which the wealthier classes may contract this illness (Steere-Williams, 2010). Typhoid began to decrease in prevalence by the 20th century due to improvements to sanitation brought about by sanitary reform. The clearing of cesspools, repairs to drains and improvements to sewerage, and general improvement in water supplies restricted the type of environment in which this disease flourished (Hardy, 1993; Lane, 2001).

Cholera was similar in origin to typhoid but far more contagious, causing approximately 21,882 deaths within England and Wales in its epidemic of 1831, and approximately 53,293 deaths in its 1848 epidemic (Lane, 2001). Cholera is a highly infectious diarrhoeal disease caused by the water-borne bacterium *Vibrio cholera* (Chan *et al.,* 2013). Thriving in high temperatures and spread through faeces, primarily via water contamination, it can reach endemic, epidemic and even

pandemic levels (Lane, 2001; Chan *et al*., 2013). Speaking of the inhabitants of Jacob's Island, London (dubbed the capital of cholera) Mayhew commented that –

> *"either their skins are white, like parchment, telling of the impaired digestion, the languid circulation, and the coldness of the skin peculiar to persons suffering from chronic poisoning, or else the cheeks are flushed hectically, and their eyes glassy, showing the wasting fever and general decline of bodily functions." (1849; 4).*

Once contracted, the disease rapidly progressed and fatalities usually occurred from as little as 2-6 days after onset (Lane, 2001). Cholera first reached England in the second cholera pandemic of 1831, originating in India in 1826 and following trade and military routes until it reached the north-east coast of the country, and beyond (Chan *et al*., 2013). It was not until the epidemic of 1853-4 that the link between cholera and contaminated water supplies was made by John Snow when he identified the Broad Street pump in Soho, London, as the root of 344 deaths in its locality in the short space of four days (Lane, 2001; Chan *et al*., 2013). With the growing awareness of the detrimental effects of faulty sewerage systems and poor water supplies, improvements to public health systems meant that cholera could be controlled more efficiently in the later outbreak in 1865.

Frequently associated with typhoid due to their similarities in symptoms, but distinctive in cause, is typhus. Like many of the infectious diseases of the 18th and 19th centuries, typhus thrived in conditions of uncleanliness, and poor ventilation and personal hygiene within the unsanitary housing of the city dweller. Engels stated "It is to be found in the working-people's quarters of all great towns and cities, and in single ill-built, ill-kept streets of smaller places, though it naturally seeks out single victims in better districts also..." (1950: 99). Although it is likely that many of these described cases of the time were just as likely to have been typhoid. Also known as putrid fever, factory fever, and gaol fever, typhus was caused by the parasitic bacterium *Rickettsia prowazeki* carried by lice and fleas, and was most prevalent in the winter months (Hardy, 1988) The bacterium was transmitted via the scratching of the skin or inhalation of dust infected with louse faeces (Hardy, 1988). Unfortunately a common treatment for typhus was the confinement of the afflicted individual in a hot dark room with no ventilation (Bayne-Powell, 1939). Typhus was staunchly a disease of poverty, and was responsible for high death rates and both epidemic and endemic outbreaks in the

18th and 19th centuries (Hardy, 1988; Lane, 2001). Similar to that seen in typhoid and cholera, the changes brought about by sanitary reform from the mid-19th century greatly diminished the spread of typhus (Hardy, 1988; Lane, 2001).

Typhoid, cholera, and typhus are as stated previously, the "filth diseases", they thrived in and were spread in the unsanitary conditions of the environment in this time. However, the next diseases to be considered are those that are common childhood risks, but it was the overcrowded conditions of both the streets and within lodgings that stimulated their spread amongst the children of this time in epidemic proportions. Whooping cough was a significant cause of mortality amongst infants in the 19th century, with one source reporting from 60 to 80 deaths per week in London in the winter and spring months of 1844-1853 (Smith, 1854). Children of all classes were prey to this disease, and two thirds of those affected were less than two years of age, and nearly all under the age of five years (Smith, 1854). Whooping cough, also known as pertussis, is caused by the bacterium *Bordetella pertussis*, and even today remains a life threatening disease to infants under the age of three months (Guiso, 2014). It is named after its characteristic "…fits of violent coughing, with short expirations – a volley of them, and then one deep, long, hooping, crowing inspiration…" (Elliotson, 1833: 193). These convulsive coughing fits, often accompanied by vomiting, can last up to six weeks (Guiso, 2014). The whole disease process from infection to convalescence can last up to three months, and is a large drain on the body's resources. Although this disease was not class specific, it did occur at a higher frequency within the poorer classes due to the severe overcrowding and poor ventilation within households and the inability to keep afflicted siblings apart from the rest of the family sphere (Hardy, 1993). In modern day studies of infants hospitalised with pertussis, it has been shown that transmission occurred from older siblings in 33% of cases, and from the mother in 28% (De Greeff *et al.,* 2010). Complications included pneumonia and bronchitis, which often increased the fatality rates associated with whooping cough, and the damage to the lungs and potential broken ribs from the persistent convulsive coughing would have had a considerable impact on health.

Scarlet fever (caused by the bacterial infection group A *Streptococcus* – CDC, 2015a) was also a high risk to those between 1-5 years of age, and struck children irrespective of class. Like whooping cough, this disease could have severe

complications such as anaemia, meningitis, kidney disease, and rheumatic fever (Hardy, 1993; CDC, 2015a). Like scarlet fever, measles was also responsible for a large number of fatalities in those less than five years of age, particularly those of 1-2 years of age (Hardy, 1992; Hardy, 1993). Measles is a viral disease, transmitted via airborne droplets, and appearing 10-12 days after infection (WHO, 2015). Symptoms include a high fever, a runny nose, and a rash starting on the face which then spreads (WHO, 2015). In the 18th-19th centuries measles was most prevalent in early winter and spring (Hardy, 1993). While infants were protected up until six months of age via maternal antibodies, when no longer protected via passive immunity they were most vulnerable to contraction of measles, particularly in the second year of life (Hardy, 1993). Malnutrition is one of the key factors when considering the mortality of this disease. In the very young when the immune status is not developed sufficiently, or when health status is already compromised by prior infection and/or poor nutrition, contraction of this disease would be extremely precarious to survival (Ashby and Wright, 1896; Hardy, 1993; WHO, 2015). As in the case of whooping cough, potentially fatal secondary bacterial infections often accompanied measles, and the disease itself was not only aggravated by poor nutritional status, but also itself depleted the child's energetic stores leaving them vulnerable to severe malnutrition and/or further contraction of infectious disease (Ashby and Wright, 1896; Hardy, 1993; CDC, 2015b; WHO, 2015).

Whooping cough, measles, scarlet fever, and diseases of the respiratory system are all passed from person to person via airborne droplet infection (e.g. saliva expelled from coughing and sneezing). The efficient spread of these diseases would require close contact, and the severe overcrowding and poor ventilation within households of towns and industrial cities incited their rapid spread amongst urban populations. It has already been shown that one childhood disease may have been closely followed by another, for example, whooping cough outbreaks often followed measles epidemics in the 19th century (Hardy, 1993). An episode of disease may have reduced the nutritional status of an infant or child, therefore depleting the energetic resources needed for recovery and immune resistance (Hardy, 1992) This would have left them vulnerable to contraction of another infectious disease, subsequently weakening the individual even further and increasing the risk of mortality (Hardy, 1992; Horrell *et al*., 2001).

"Yet, in spite of his early promise, all this vigilance and care could not make little Paul a thriving boy… This dangerous ground in his steeple-chase towards manhood passed, he found it very rough riding, and was grievously beset by all the obstacles in his course. Every tooth was a break-neck fence, and every pimple in the measles a stone wall to him. He was down in every fit of the hooping-cough, and rolled upon and crushed by a whole field of small diseases that came trooping on each other's heels to prevent his getting up again." – Charles Dickens, Dombey and Son (1896: 73).

Not confined to childhood, tuberculosis (also referred to as phthisis, "wasting", and "consumption") was a chronic infectious disease widespread in the 18th and 19th centuries (peaking between 1780-1830), and was also promoted by the overcrowded and poorly ventilated conditions (Lane, 2001). There are two avenues through which tuberculosis may be contracted. There are a multitude of strains of mycobacteria that can cause tuberculosis, and these make up the *Mycobacterium tuberculosis* complex (MTBC) (Galagan, 2014). The primary cause of human tuberculosis is *Mycobacterium tuberculosis*, and is spread by droplet infection. This was the cause of approximately 98% of all pulmonary cases of tuberculosis in the 18th and 19th centuries (Hardy, 1993). It was propagated by the close conditions described previously, and also through occupational exposure, poor hygienic conditions, and insufficient diet (Lane, 2001). Another form, bovine tuberculosis (*Mycobacterium bovis*), was transmitted through infected cows milk. This represented a higher risk for infants, as they were more likely to consume it during the weaning process (Atkins, 1992). Scrofula was also a common form of tuberculosis, referring to a tubercular infection of the lymph nodes of the neck causing them to swell, however it was the pulmonary manifestation of this disease that caused the highest fatality rates averaging 60,000 deaths a year between 1839-43 in the industrial centres of this time (Lane, 2001).

Another infectious disease of this period, which, like that seen in tuberculosis, plagued society regardless of age and social status, was the notorious smallpox. The highly infectious strain of smallpox (Variola major) had been present within English society from as early as the 10th century AD and this disease, spread through droplet infection and close contact, thrived at both the endemic and epidemic level within the congested urban conditions of the 18th and 19th centuries

(Oxley, 2003). Those most vulnerable to severe effects of infection were infants and children, pregnant women, and the elderly (Oxley, 2003). Small pox is characterised by the rash that spread over the face and the rest of the body, which developed into weeping pustules before scabbing and falling off over the course of 2-3 weeks (signalling the end of the infectious period) (Oxley, 2003). Not only was this disease visually scarring, the pockmarked face being a classical marker of the smallpox victim and evoking social stigma, but there were also a multitude of debilitating consequences of smallpox on the sufferer that may have become permanent afflictions. Partial or full loss of sight was common, limb deformities were rarer after effects, and many were left vulnerable to consumption and general weakening of the condition to further ill health (they became "sickly") (Oxley, 2003). Possible cases of smallpox have been detected in the skeletal record, including a four-year-old child from the St Marylebone site in London, through identification of smallpox osteomyelitis at the distal humerus (Walker, 2012: 88).

A large proportion of those who contracted smallpox would not survive, succumbing to severe toxaemia, with 755 fatalities per million in London alone in 1838-1842 (Oxley, 2003). With the introduction of smallpox inoculation in the 1720s by Lady Mary Montagu (who had witnessed a method of inoculation used in Turkey), the outlook for bringing this disease under control looked promising (Porter, 1995; Fine, 2014). However, due to the necessity of infecting the child with smallpox (via the pus from a smallpox pustule from a sufferer of a milder strain of the disease) to bring immunity, and the high risk of disfigurement or fatality that accompanied this procedure, many parents were suspicious of this practice particularly among the lower classes, or preferred to follow their own methods of amateur home inoculation (Bayne-Powell, 1939; Burnett, 1984; Lane, 2001). It was not until the introduction of the smallpox vaccination by Dr Jenner in 1797, and the initiation of compulsory vaccination of infants in 1853, that the eventual path towards elimination of smallpox began to move forward, and between 1891-1900 fatalities had dropped to 10.54 per million in London (Bayne-Powell, 1939; Burnett, 1984; Oxley, 2003; Fine, 2014).

The infectious diseases described above were all propagated and extensively spread through the detrimental urban environment. Their inhabitants were constantly exposed to the contagion of an unremitting multitude of diseases through

close contact with family, neighbours, and the wider community. Living conditions were poor, polluted, and food sources restricted by both income and preservation so that "Hour after hour, day after day, year after year, the negative effects of these disamenities accumulated, carving their influence on the human body" (Oxley, 2003; 9).

"When one remembers under what conditions the working-people live, when one thinks how crowded their dwellings are, how every nook and corner swarms with human beings, how sick and well sleep in the same room, in the same bed, the only wonder is that a contagious disease like this fever does not spread yet farther." - Engels (1950: 100).

However, these infectious diseases are not detectable on the skeleton to any reliable degree (with the exception of tuberculosis), so unfortunately their impact on the skeletal record and therefore within post-medieval skeletal collections are not easily assessed. Infectious disease, particularly in the very young who lack a robust immune system, is often acute in nature leaving little time to show any effect on the skeleton, and even in the event of the skeletal system reacting to such stress it would be virtually impossible to differentiate between the multitude of infectious diseases that the individual may have suffered from. While undoubtedly some of the individuals within this study must have been expected to suffer from infectious disease, it is not within the bounds of this project to be able to directly comment on the impact of it within these past populations. However, there are some conditions notoriously associated with the industrial environment that can be detected skeletally and can reveal valuable information regarding the environmental and osteobiographical context of these populations.

3.5.2 Rickets

Rickets was classed as one of the "new" diseases of industrialisation, alongside tuberculosis and typhus, however it had been recognised within England as a discrete condition from as early as the 17th century (Gibbs, 1994; Porter, 1995). A recent study has also identified the earliest known case of residual rickets in a young adult woman from the Neolithic period (Armit *et al*., 2015). One of the first documented accounts of rickets came from Francis Glisson in 1650 (translated into English in 1651 by Philip Armin), who described its high prevalence in South-

Western England (being particularly epidemic in Dorset and Somerset) around the 1620s and then later in London, Oxford, and Cambridge (Glisson, 1651; Gibbs, 1994). To understand why rickets was making an appearance in particular communities in the 17th century, and later became such a dominant child health concern in the 18th and 19th centuries, it is essential to understand the pathogenesis of this condition.

Vitamin D is a pro-hormone required for the absorption of calcium and phosphorus, necessary for ossification of the developing bones (Holick, 2006; Brickley and Ives, 2008; Shin *et al*., 2010). Despite our ability to synthesise vitamin D through ultraviolet light from sun exposure, there is still a relatively high prevalence of vitamin D deficiency in many countries, mainly due to a lack of exposure to sunlight as a consequence of latitude, lifestyle, cultural dress, or atmospheric pollution, and also through dietary deficiency (Prentice *et al*., 2006; Brickley *et al*., 2007). While vitamin D can be gained through dietary means, sunlight remains the principal source, and without sufficient exposure to it vitamin D deficiencies will develop whether mild or severe in nature.

Rickets predominately affects areas of rapid bone growth, such as the long bone epiphyses (Holick, 2006). Typical manifestations of rickets include flaring of the metaphyses of long bones, swelling of the costal rib ends, and bowing of the long bones (Pettifor, 2003; Holick, 2006). Bowing occurs as the affected child begins to stand and walk, and their weight acts on the softened bone (Pettifor, 2003; Holick, 2006). Descriptions of children with knock-knees, "bandy legs", deformed pelves, and crooked spines (scoliosis) are rife within parliamentary and popular literature of the time. These skeletal deformities are suggestive of chronic periods of

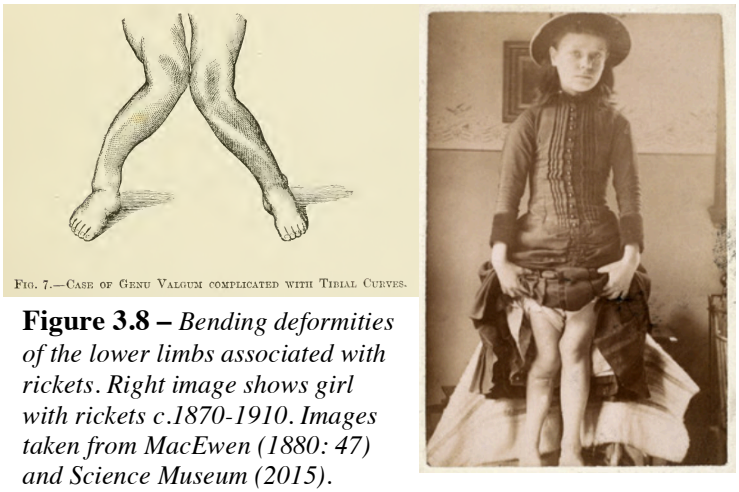

Figure 3.8 – *Bending deformities of the lower limbs associated with rickets. Right image shows girl with rickets c.1870-1910. Images taken from MacEwen (1880: 47) and Science Museum (2015).*

vitamin D deficiency, and were also recognised as indicators of rickets by Glisson in the 17th century. He described such symptoms as metaphyseal flaring, "About the joynts, especially in the wrests and ankles certain swellings are conspicuous…" (Glisson, 1651: 11). "The top of the ribs to which the stern is conjoyned with gristles, are knotty, like unto the joynts of the Wrest and Ankles…" demonstrates his recognition of the "bead-like" appearance of the flared rib ends, as well as describing the lateral straightening of the rib shafts often seen in osteological collections, and cases of pectus carinatum (pigeon chest) which can be associated with rickets (Glisson, 1651: 12; Pettifor, 2003; Holick, 2006). The epiphyses of the bones of the wrists and ankles are also described as being "…more soft and spongy than in other parts of the same Bones" referring to the insufficient deposition of bone at the metaphyses (Glisson, 1651: 117-118). Furthermore, the classic bending deformities of this visually striking condition are recognised as being "…frequently somewhat crooked, especially the Shin Bones, and the lesser Bones of the Legs…somtimes the Bones of the Sholder and Thigh: som Joynts also becom crooked, somtimes inward, somtimes outwards: the whole Spine is likewise many times bended…" (see Fig.3.8) (Glisson, 1651: 120).

The appearance of rickets in 17th century South-Western England therefore would have resulted from a lack of exposure to sunlight. This is most likely to have been a social consequence of the requirement of families to work indoors from morning to evening, thus preventing children from spending time outdoors in daylight hours (Gibbs, 1994). Speculated causes of the time ranged from inadequate nutrition and incompetent nursing, and while these certainly would have aggravated the condition, without sufficient exposure to ultraviolet light children still would have developed deficiencies. Glisson commented that "We see the Children of the Poor People are ordered and handled with less care, and sooner committed to their feet then the Children of the rich, yet the Children of Poor People are more rarely afflicted with this infirmity…" (1651: 121). It was the children of the poorest status, who were reliant on employment within agriculture or other outdoor pursuits, that would have received adequate sunlight exposure to avoid the severe effects to health that came with vitamin D deficiency (Gibbs, 1994).

Rickets was considered a major problem in the post-medieval period (Mankin, 1974). It was found to be particularly abundant in the industrial regions of

London, Newcastle and Gateshead, Lancashire, Yorkshire, Cheshire, Derbyshire, and Nottingham (Owen, 1889) (see Fig.3.9). Its high incidence among urban children is attributed to the thick coal smoke that diminished sunlight in the cities, the overcrowding, improper diets, working long hours indoors, and social practices of the time that restricted time spent outdoors (Hardy, 2003; Roberts and Cox, 2003). Although rickets could occur in children of all ages, it was most common within infants particularly between six months to two years of age (weanlings) (Crawford, 2010). Cases of rickets varied from mild to severe, so it is possible that while not all individuals demonstrated a severe enough deficiency to present the bowing deformities, they still may have suffered from some of the less visual effects. It has been suggested that vitamin D plays an important immunoregulatory role, with reports that children affected by rickets often experience repeated episodes of infectious disease, including an increased susceptibility to the common cold virus (Holick, 2006; Shin *et al*., 2010). In 1868 it was found that of children under two years of age admitted to Great Ormond Street, London, approximately a third demonstrated symptoms associated with rickets (Wickes, 1953a; Gibbs, 1994), therefore a large proportion of the population may have had an increased susceptibility to numerous sources of poor health that accompanied urban life. Rachitic children would have been less resistant to the common childhood diseases and conditions such as tuberculosis (Hardy, 1993). Rickets and whooping cough were reported to frequently co-occur due to the common origin of dark crowded housing that instigated both conditions (Hardy, 1993). Co-morbidity of diseases could be deadly, with the reduction in immune resistance associated with rickets inciting vulnerability to catching whooping cough, and the convulsive coughing that

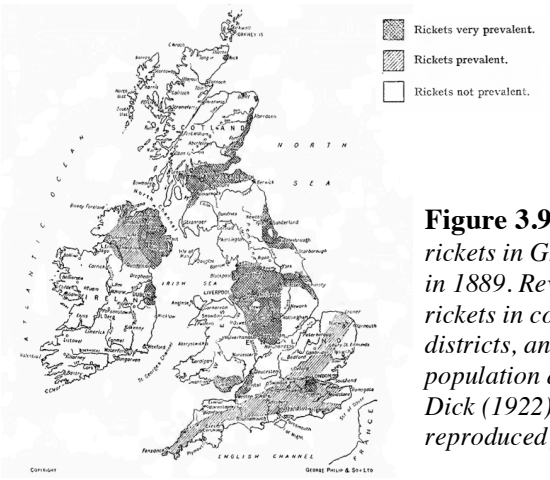

Figure 3.9 – *Map of prevalence of rickets in Great Britain and Ireland in 1889. Reveals high prevalence of rickets in coal-mining and industrial districts, and in areas of high population density. Adapted from Dick (1922), where it was reproduced from Owen (1889).*

came with this disease causing gross morbidity to the weakened body associated with vitamin D deficiency (Hardy, 1993). Those who demonstrated both rickets and whooping cough were less likely to survive the course.

Adults were not protected from the ill-effects of vitamin D deficiency despite the cessation of their growth period. Vitamin D deficiency following the completion of endochondral growth is termed osteomalacia, and is characterised by appearance of pseudofractures (primarily on the ribs, scapulae, pelves, and long bones), buckling of the vertebral bodies, pelvic deformities, and long bone bending deformities (Ives and Brickley, 2014). Due to the long working hours indoors required of the urban labourer, it is highly likely that many adults were also severely vitamin D deficient. This is significant when considering maternal health, as this type of deficiency during pregnancy can translate to the developing foetus, so by the time of birth the infant would already be vitamin D deficient (Abrams, 2007; Shin *et al.,* 2010; Hanieh *et al*., 2014). Unfortunately, it was not until 1919 that the association between sunlight exposure and rickets was conclusively proven, although the acknowledgement of the association of rickets with industrial centres and the beneficial effect of sunlight on overall health and had begun to take root from the mid-19th century (Gibbs, 1994).

Rickets falls into the category of "metabolic disease". Within bioarchaeology this refers to conditions that affect the normal processes of bone modelling and remodelling (and in non-adults, the formation of bone) (Brickley and Ives, 2008). The considerable effect that vitamin D deficiency can have on the developing skeleton means that this type of pathology is identifiable within skeletal remains; therefore its prevalence and impact on past populations can be assessed (see Chapter Five for the identification of rickets within skeletal remains). Vitamin D deficiency in non-adults may reduce long bone length as a result of the bowing associated with rickets, and may also cause retardation of endochondral growth, thereby consequently reducing stature (Mays *et al.,* 2009a). The stunting effect of rickets on growth has been demonstrated in modern clinical studies, therefore its disruption to growth could potentially be detected in skeletal remains (Kreiter *et al.,* 2000). Mays *et al*. (2009b) found that non-adults from St Martin's Churchyard, Birmingham exhibiting signs of rickets were also deficient in long bone growth between the ages of 2-6 years. However, in a study by Pinhasi *et al*. (2006) using

two skeletal collections from London (Christ Church, Spitalfields and Broadgate) there was no evident difference in growth between non-adults with rickets and those without signs of the disease. Due to the high percentage of cases of healed rickets within the sample of this particular study it was suggested that the lack of difference was a result of post-vitamin D deficiency catch up growth (Pinhasi *et al.,* 2006). While the detection of growth disruption resulting from rickets has proven to be problematic due to the veiling effects of catch-up growth, the prevalence of this condition within skeletal collections can potentially provide us with valuable information regarding environmental conditions and social practices of the time.

3.5.3 Scurvy

Scurvy, also a form of "metabolic disease", refers to the condition that results from vitamin C deficiency. Humans, alongside guinea pigs, cannot synthesise their own vitamin C (ascorbic acid) therefore must obtain it from dietary sources, e.g. primarily fruits and vegetables, and minimally from milk, meat and fish (Brickley and Ives, 2008; Armelagos *et al.,* 2014; Mays, 2014). These dietary sources are essential to human health, as vitamin C is principally required for the maintenance of collagen formation, and also has roles in blood formation, iron and folate metabolism, and maintenance in immune function (Ortner and Erickson, 1997; Brickley and Ives, 2008; Popovitch *et al.,* 2009). It is the disruption to collagen maintenance resulting from vitamin C deficiency that causes the distinctive skeletal changes seen in scurvy. Collagen is a vital component of soft tissues such as muscles, tendons, and blood vessels, as well as contributing to bone formation (Brickley and Ives, 2006; Brickley and Ives, 2008; Popovich *et al.,* 2009; Armelagos *et al.,* 2014; Stark, 2014). When collagen formation is disrupted, this weakens structures reliant on it for mechanical integrity and repair. Therefore typical symptoms of scurvy include haemorrhaging of weakened blood vessels (often those supplying musculature of areas of frequent movement, such as the mandible when chewing, in the eye orbits relating to the movement of the eyes, and bleeding into the joints), deficient bone formation in growing children leading to osteopenia, sub-periosteal haemorrhages leading to damage to the periosteum of the long bones, irritation of the gums and loosening of the connective tissue adhering them to the dentition potentially leading to ante-mortem tooth loss, and delays to

wound healing (see Chapter Five for the identification of scurvy in skeletal remains) (Ortner and Erickson, 1997; Brickley and Ives, 2006; Brickley and Ives, 2008; Armelagos *et al*., 2014; Stark, 2014).

"The patient is usually anaemic, though he may be well nourished as far as subcutaneous fat is concerned; the gums are spongy and offensive, they bleed with the slightest injury, the teeth are loose and may fall out; haemorrhage is apt to occur from the nose, kidneys, and bowels purpuric spots are common, and bruising occurs after the slightest injuries. The majority of the cases which come under our notice in hospital quickly improve with proper dieting and careful nursing." - Ashby and Wright (1896: 363).

Individuals suffering from this deficiency may also experience tiredness and lethargy, as well as pain and weakness of the limbs (Brickley and Ives, 2008; Geber and Murphy, 2012).

Scurvy represented a significant health risk to children in the post-medieval period (particularly the infants), and was not just a problem faced by the lower classes, but also regularly affected the wealthier families (Hardy, 1993). As discussed in section 3.3, there were some substantial difficulties in access to fresh produce in the 18th and 19th century urban centres. Particularly in the 18th century, fresh meat in winter was scarce, and fresh vegetables were also difficult to come by (Bayne-Powell, 1939). As described previously, the fresher the produce, the higher the levels of vitamin C present to be absorbed into the body. In addition, there was also an aversion to fruit and vegetables in general, with the unfortunate belief that they were bad for children and indigestible (Bayne-Powell, 1939). The potato represented a large proportion of the diet of the labouring population, therefore this may have provided a potential source of vitamin C depending on the extent of the cooking (as heat can greatly diminish vitamin C levels within food sources). Therefore, the simple aversion to the ingestion of fruit and vegetables in the higher ranks of society, and the restricted access to a variety of food sources to the poorer classes, meant that cases of scurvy (ranging from mild to severe in manifestation) would have been expected to be rife amongst families during this time. A particularly high rate of scurvy was identified within the skeletal collection from the Kilkenny Union Workhouse (c.1847-1852), and was found to be a significant influence on mortality risks, with highest rates occurring in young children, older

children, and adolescents (Geber and Murphy, 2012). The deficiencies seen within this population are thought to be a result of a complete absence of vitamin C in the diet resulting from potato blight during The Great Famine (1845-1852), followed by a reintroduction in the workhouse diet (Geber and Murphy, 2012). It can take up to 29 to 90 days for symptoms of scurvy to manifest, but recovery is quick following the re-introduction of vitamin C to the diet with physical symptoms usually subsiding within two weeks (Brickley and Ives, 2008; Popovich *et al*., 2009). Therefore it is likely that the vitamin C levels of many were constantly in flux, with individuals recurrently developing scorbutic symptoms.

Infantile scurvy was a significant issue. In the 19th century there was an increase in manufactured "infant feeds" available to mothers who were unable to breastfeed (whether due to occupational responsibilities, or the inability to produce breast milk due to nutritional deficiencies) or simply did not wish to (Wickes, 1953; Fildes, 1995; Nitsch *et al*., 2011). This meant that more and more infants were being denied the passive immunity and nutritional support provided by the mother's breast milk. Infants who are breast fed by healthy and well-nourished mothers rarely develop scurvy as breast milk contains sufficiently high levels vitamin C (Cheadle, 1889; Brickley and Ives, 2008). Daily requirements of vitamin C increase for women during pregnancy and lactation, therefore mothers whose diet provides a sub-optimal source of vitamin C can potentially become severely deficient in this essential vitamin, and in turn pass on this deficiency to their developing child, leading to congenital scurvy (Cheadle, 1889; Brickley and Ives, 2008). Evidence of scurvy in the first year of life may suggest an early cessation of breastfeeding, or complete lack of this practice, and its manifestation in the 2-3 years of life may also suggest an insufficient weaning diet. Therefore evidence of this condition in infants can be a valuable indicator of breastfeeding and weaning practices, as well as diet, in past populations (Lewis, 2002a).

An important feature of this condition is that due to the characteristics of its onset, and the types of environment that is was likely to feature in, scurvy was likely to occur alongside a multitude of other ailments. For example, scurvy and rickets are frequently found to co-occur, an association also noted by Glisson in 1650, making their osteological differentiation problematic and leading it to often be categorised as "acute rickets" in the medical literature until the late 19th century

(Rajakumar, 2001; Stark, 2014). Considering the role of vitamin C in iron metabolism, scurvy is also likely to develop alongside anaemia (Brickley and Ives, 2008; Baker *et al*., 2010). Like rickets, scurvy can influence immune function leaving the individual vulnerable to additional health insults such as infectious disease, while slowing their recovery from these episodes of disease (Geber and Murphy, 2012). Therefore, the risk of children of this time being "trapped" in a cycle of ill health was high.

3.5.4 Occupational hazards

"'Fluff,' repeated Bessy. 'Little bits, as fly off fro' the cotton, when they're carding it, and fill the air till it looks all fine white dust. They say it winds round the lungs, and tightens them up. Anyhow, there's many a one as works in a carding-room that falls into a waste, coughing and spitting blood, because they're just poisoned by the fluff." - North and South, Gaskell (1855: 41).

In section 3.1 the employment of children was identified as one of the key features of the industrial revolution that has dominated historical inquiry. This fascination stems from both the fact that this practice represents an ideology far from that which exists today in western thought, but also from the mass of parliamentary evidence and social literature of the time. Such sources documented the severe effects of industrial occupation on these vulnerable members of society, with stories of cruelty and exploitation that lie beyond imagination. While it has already been noted that this was not the experience for all, there were certainly still instances where specific occupations had dire consequences for growing children. The heightened energetic requirements such intensive work entailed, combined with poor diet, lack of sunlight, and weakened growing bones meant that the repercussions for some of these children were dramatic and enduring.

"Well I can recollect in the earlier periods of the factory movement, waiting at the factory gate to see the children come out, and a set of sad, dejected, cadaverous creatures they were. In Bradford especially, the proofs of long and cruel toil were most remarkable…A friend of mine collected a vast number together for me; the sight was most piteous, the deformities incredible. They seemed to me, such were their crooked shapes, like a mass of crooked alphabets." Lord Shaftesbury – cited in Pinchbeck and Hewitt (1973: 403).

The physical deformities so described by Lord Shaftesbury undoubtedly refer to the resultant bowing and twisting of the limbs and distortion of the spine of vitamin D deficient children required to stand for exceedingly long periods of time while undertaking repetitive factory work. Working days could last up to 12-14 hours and the stress that would be placed on the developing growth plates, alongside severe vitamin D deficiencies, would have been highly damaging and may have led to this type of distortion. Considering that these children would not have been exposed to sufficient sunlight during the hours of work, it is likely that they were rachitic, thereby adding to the potential for gross distortion of the malleable bones. Such work was said to be responsible for flat footedness, knock-knees (genu valgum), and stunted growth amongst factory workers (Kirby, 2013). Gaskell describes an inspection of 2,000 factory children –

"The children were stunted, pale, flesh soft and flabby; many with limbs bent, in most the arch of the foot flattened; several pigeon chested, and with curvatures in the spinal column; one hundred and forty had tender eyes, in a great majority the bowels were said to be irregular, diarrhoea often existing, and ninety shewed decided marks of having survived severe rachitic affections" (1833: 208).

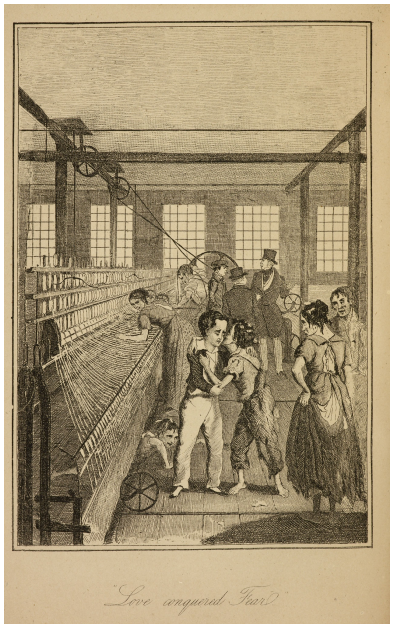

Figure 3.10 - *Image entitled "Love conquers fear", from the series by Francis Trollope – The life and adventures of Michael Armstrong, the factory boy. Shows some of the hazardous jobs undertaken within cotton mills by children. Taken from British Library (2015a).*

Very young child workers were often employed in cotton mills as "piecers", which required them to crawl under the looms to tie broken threads together (see Fig.3.10) (Lane, 2001). Not only was this exposure to the new industrial machinery extremely hazardous, putting them at risk of serious injury if caught by the moving components, but also meant that the bones of the arms may have been subject to unilateral deformity from the repetitive movement required of this task, resulting in a crooked appearance (Lane, 2001; Kirby, 2013). Accidents relating to machinery were a perpetual risk, with many children coming to harm due to a stray

piece of clothing or hair becoming caught, and the results could be brutal, disabling, and fatal (Pike, 1966).

Particular occupations often came with their own unique set of health complications. The quote that heads this section relates to the "fluff" or dust that filled the air of cotton mills generated from the processing of raw cotton, and thus was inhaled by the workers (Thackrah, 1831; Kirby, 2013). This constant exposure to the damaging effects of the "fuz" on the lungs led to pulmonary disease, and often culminated in chronic respiratory disorder or even tuberculosis (Thackrah, 1831; Pike, 1966; Kirby, 2013).

"In addition to the noxious effluvia of the gas, mixed with the steam, there are the dust, and what is called cotton-flyings or fuz, which the unfortunate creatures have to inhale; and the fact is, the notorious fact is, that well constituted men are rendered old and past labour at forty years of age, and that children are rendered decrepit and deformed, and thousands upon thousands of them slaughtered by consumptions, before they arrive at the age of sixteen…" - William Cobbett, Political Register, vol. LII, 1824 – reproduced in Pike (1966: 61).

The "fuz", when accidentally ingested, could also lead to stomach disorders resulting from bacteria within the raw cotton (Wilkes, 2011). Weavers were also exposed to diseases of the eye from working in dark conditions for prolonged periods of time, as well as pulmonary diseases and consumption from ill-ventilated work spaces (Pinchbeck and Hewitt, 1973). Diseases of the stomach have also been described for both children and adults in this occupation, resulting from the pressure of the body against the beam of the loom (Pinchbeck and Hewitt, 1973).

New raw materials were also beginning to gain popularity in the developing industries that were extremely hazardous to health (Lane, 2001). Lead was used widely in the glass and paint producing industries, and within potteries as a glaze, exposure to which could cause paralysis and lead poisoning (or plumbism) (Thackrah, 1831; Kirby, 2013; Millard *et al*., 2014). Long-term exposure to lead has a multitude of severe implications for health, including anaemia, gastro-intestinal irritation, convulsions, short-term memory loss, and impairment of motor functioning (see Millard *et al*., 2014). Constant contact with soot also resulted in scrotal and lip cancer among chimney sweeps, in addition to the risks of suffocation and burning associated with this occupation (Thackrah, 1831; Lane, 2001).

Work by Kirby has offered insight into the effects of mining on the growth of children. His evidence of deficient heights of coal-mining children compared to those from other occupations such as agriculture has prompted an interesting debate into the source of this stunted growth (see Kirby, 1995; Humphries, 1997; Kirby, 1997). What is undeniable in the case of the occupational requirements of coal-mining children is the significant deprivation of sunlight that these workers would have experienced during their 10-12 hour working days underground, and the subsequent effects that this deprivation would have had on growth through their susceptibility to rickets (Kirby, 1995). This deprivation of sunlight, combined with the constricted working conditions, e.g. movement through narrow coal seams, resulted in the bowing of the legs and curvature of the spine of the young employees of the mining industry (Kirby, 1995).

While both children and adults were exposed to these work-place hazards, the results were more likely to be far more dramatic in the young than the mature. At the age in which children typically entered the workforce (around 9-10 years of age) they were also entering a particularly rapid stage of growth, thus their heightened metabolic rate would have promoted an increased susceptibility to the absorption of noxious agents (Kirby, 2013; Millard *et al*., 2014). Therefore while adults would have eventually succumbed to declining health over the course of many years of employment, the effects in children would have been more immediate and detrimental to survival (Kirby, 2013). Unfortunately, within skeletal collections identifying pathologies directly related to child labour is in most cases unfeasible. Not only were labouring children exposed to poor working conditions, but also their home conditions were equally detrimental, making it difficult to separate out the effects of poor environment and occupational influences (Kirby, 2013). There are some cases however where pathology within archaeological assemblages may be suggestive of the participation of a community's children within occupational pursuits, such as the case of a 12-14 year old child from the Coach Lane collection from North Shields who demonstrates skeletal changes suggestive of phossy jaw (Roberts *et al*., 2014). Phossy jaw refers to the painful and disfiguring necrosis of the mandible, resulting from exposure to white phosphorus (McLeod *et al*., 2012). In the 19th century, this condition was notoriously associated with the match making industry (Harrison, 1995; McLeod *et al*., 2012).

3.6 The urban-rural dichotomy

"…windmills, rick-yards, milestones, farmers' wagons, scents of old hay, swinging signs and horse troughs: trees, fields, and hedgerows. It was delightful to see the green landscape before us, and the immense metropolis behind…." – Bleak House, Charles Dickens (1853: 42).

Rural life during this period is often described in literature as being far superior to that of their urban counterparts. It is certainly true that country dwellers would not have suffered from the effects severe overcrowding and air pollution typical of the towns and cities. Highly descriptive accounts of living conditions during the 18th and 19th centuries portray a stark contrast between life in the smoggy, disease-ridden cities, and life within quaint rural idylls (for examples see the above quote by Dickens, the quote below by Reid, and Fig.3.11). However, it is important to consider that the contemporaneous accounts of life at this time were often also concerned by the political issues regarding the rapidly expanding cities (Harrison and Gibson, 1976). The epidemiologist William Farr felt that the fixation of writers on the declining conditions of urban centres was often "fanatical" (Young and Handcock, 1956), thus it is possible that rural life was greatly romanticised as a result.

"The agricultural class, though in a declining condition, is more near to what nature made it; the other [referring to the urban labouring population] presents us with a rapid succession of generations of stunted and debilitated families, reared up in moral and physical suffering…" - Reid (1845: 113).

The rural hinterlands did not escape the rapid transformations of this era. The ever-expanding population within towns required a concomitant increase in production from the land. This increasing demand combined with technological innovation in agricultural machinery fuelled the agricultural revolution, and transformed rural life from one of small-scale subsistence

Figure 3.11 – *The Hay Wain by John Constable, 1821. Depiction of the typical "rural idyll" of the 19th century. Image taken from The National Gallery (2015).*

cultivation, to large commercial farms (Broad, 2010). This movement away from a system that enabled rural dwellers to eke out a living on common land through the keeping of livestock or cultivation of small amounts of grain, to one where they were employed as landless day labourers by large landowners had dramatic consequences for rural life and health.

This fundamental change in agricultural society was driven by the Enclosure Acts, where common land became partitioned into plots of land, which could be bought and subsequently rented out to tenant farmers (Mathias, 2001; Hudson, 1992; O'Brien and Quinault, 1993; Green, 2010a). By 1834 approximately 1.9 million acres of common land had been enclosed (Horrell and Oxley, 2012). This meant that those previously reliant on the open field system, by necessity became the mobile workforce of the agricultural industry. The seasonal nature of farming meant that employment was irregular, there would be a high demand for labourers during harvest times, but underemployment or unemployment for up to eight months in the year especially during winter (Burnett, 1989; Hudson, 1992). The rising competition for agricultural employment, low wages, and high levels of unemployment (particularly for women and children) meant that more and more of the rural inhabitants sought work within the manufacturing towns and cities, leading to a large migration from country to town (Hudson, 1992). However, agricultural labourers still remained an important contributor to the workforce, with around 961,000 families still employed in agriculture by the 1831 census, and making up approximately 22% of the nation's workforce towards the end of the period (Pinchbeck and Hewitt, 1973; O'Brien and Quinault, 1993).

As in urban areas, child labour was an important feature of the agricultural economy, with children working for as long as daylight persisted in the winter periods, and from as long as 5am to 9pm during the harvesting season (Pinchbeck and Hewitt, 1973). While employment out of doors in the sunlight may have been more beneficial to rural children when compared to the conditions experienced by the young urban labourers, hours were still long and nutrition often insufficient to support the energetic demands of manual labour. Not all of the industries performed by children within rural areas were confined to agriculture, many were also employed in manufacturing activities from young ages and for long hours within cottages (Pinchbeck and Hewitt, 1969). While protective measures were gradually

being imposed on the legislation of employment of children within factories, those working within agriculture and rural cottage industries were overlooked until the Education Act of 1876 which meant that children under 10 years of age could not be employed in any agricultural work (Burnett, 1984; Pinchbeck and Hewitt, 1973).

Rural populations were also highly susceptible to periods of food shortages and crop failures, and a series of poor harvests could result in poverty and famine (Bogin, 1999).

"Village life during this period was a time of trial and difficulty. The agricultural workers had long hours, they pay barely enough to keep body and soul together. The condition of the children in many cases was pitiable. Rough food and clothes; everything depended on the skill and character of the mother." – Alfred Ireson, born 1856 (Burnett, 1984: 83).

The lower population density, and improvements to sanitation and housing, may have brought some benefits to rural life. The likelihood of surviving to childhood was twice as high when compared to urban areas during this period, and it had been stated, "death amongst children is fourfold by epidemics, and nearly ten-fold be convulsions in towns, as compared to rural districts" (Reid, 1845: 113; Hardy, 1992; Woods, 2003). However, this does not necessarily mean that those residing in rural areas were healthier than those in urban areas, but that they may have experienced different health risks (Woods, 2003). William Farr stated that 'epidemics desolated the country as well as towns, though to less extent…' (Young and Handcock, 1956; 771). John Dickinson (a man from Fewston, North Yorkshire who kept diaries between 1878-1912) refers to fatal outbreaks of disease within this small community, particularly typhoid from contaminated water sources (Harker, 1988). He also makes reference to the effects of depopulation of rural areas due to the migration of the workforce into towns -

'As regards this immediate neighbourhood the state of the community is very dark and depressing. Farming is very unprofitable, the population is decreasing, the best families and the best men and women migrate to the towns.' - Harker (1988; 84).

While rural life in general may have been greatly idealised, the rural diet may have been of better quality when compared to that of the urban worker. Rural

inhabitants may have had more access to grains, milk, and fresh green vegetables, whereas urban inhabitants would have consumed more store bought goods (such as bread from bakeries) that came with a higher risk of adulteration (Snow, 2003), and had an overall lower caloric and protein intake (Clark *et al.,* 1995; Horrell *et al.,* 1998). However, studies focusing on past accounts of rural diet at this time have indicated that the rural fare was not too dissimilar to that seen in the urban labouring populations, consisting mainly of potatoes, bread, cheese, and bacon (Shammas, 1984; Drummond and Wilbraham, 1994; Horrell and Oxley, 2012). George Mockford, a son of a poor shepherd born in 1826 stated that "I was always rather delicate in health, and had no stamina about me for outdoor exposure; the food for us young ones consisting of little else than potatoes with a little bacon fat on them" (Burnett, 1984: 73). The implementation of the enclosures and the subsequent reduction in common land available for the raising of livestock would have reduced the opportunities for families to keep cows, therefore access to dairy products such as milk, cheese, and butter may have been problematic for some rural communities. Meat was also often absent or scarce in the diet. In one survey of 193 rural families only one-third had access to gardens, and only two kept cows (Shammas, 1984). In areas where common land still remained, or families were able to keep gardens, rural diet may have been improved.

Therefore, rural inhabitants also likely experienced the debilitating effects of infectious disease and malnutrition, so growth in these populations may not necessarily show improvement in comparison to urban populations. While this is a study of urban health, two of the sites analysed within this thesis were more suburban in nature, before being engulfed by the expanding city of London. Therefore, they may have retained more of a rural status prior to this industrialisation. Thus it is important to contextualise the quality of life away from the city centre at this time.

3.7 Summary

What is certain is that the economic, social, and physical environment of this time (whether urban or rural) was indeed precarious for the growing child, making it unsurprising that morbidity amongst children at this time was rife, and infant mortality so high. The aim of this thesis is to assess the impact of the historical

factors discussed in this chapter in relation to indicators of health and growth disruption seen within the skeletal populations selected for this study. It has already been noted previously that despite the predominant effect of infectious disease on child health and survival at this time, we unfortunately are currently unable to assess the prevalence of such diseases within skeletal populations at this time. However, using measurements of long bone lengths and vertebral dimensions, evidence of growth disruption can be detected and, in combination with region specific historical documentation, can provide us with a wealth of information regarding the effects of living environment and social status on child health. Conditions such as scurvy, rickets, and anaemia are also potentially detectable on the skeleton (with varying degrees of reliability to be discussed later) and can also provide evidence of deficiencies that have origins in dietary, social, and child-care practices that can provide an added perspective of childhood and child health for the littlest members of these skeletal populations.

CHAPTER FOUR

Materials

4.1 Introduction

This chapter outlines the materials used in this study, including the skeletal collections analysed, the selection criteria for individuals from which measurements were taken, and the historical context for each of the samples.

4.2 Selection criteria

The primary aim of this study was to assess the patterns of growth disruption and prevalence of indicators of stress within non-adults from post-medieval urban centres. To appropriately assess child "health" and "stress", individuals up to 17 years of age were selected from each site. This enabled the analysis of skeletal parameters throughout the growth period, as well as visualisation of the prevalence of pathological conditions throughout infancy, childhood, and adolescence/puberty. As there is currently no reliable method for the determination of sex in non-adults (Mays and Cox, 2000; Saunders, 2008), all non-adult data in this study was pooled. Individuals aged between 0-17 years of age were selected for metric analysis based on the presence of the dentition, long bones (femur, tibia, and/or humerus), and/or vertebrae. All non-adult individuals from the Coach Lane and Coronation Street collections were assessed for the prevalence of select pathological conditions (see section 5.7), and pathological data for the collections curated by the Museum of London (MoL) and Museum of London Archaeology (MoLA) were gathered from the Wellcome Osteological Research Database (WORD database) (WORD database, 2012a; WORD database, 2012b; WORD database, 2012c).

Adults aged between 18-25 years and 26-35 years from each site were also selected for metric analysis to provide comparative data. Adult metric data were pooled for the sexes, to ensure compatibility with the non-adult data sets.

4.3 The sites

Figure 4.1 – *Map of England showing location of sites within a) the north-east (Tyneside), and b) London.*

In total, six skeletal collections were identified to represent urban populations from the post-medieval period, from a diverse range of backgrounds. Two sites were selected from the North-East of England, and four from London (see Fig.4.1). A summary of the context of each site can be found in Table 4.1. The historical context, and excavation background, for each sample will be described below.

Table 4.1 - *Summary of the skeletal collections used in this study (Brickley and Miles, 1999; Cowie et al., 2008; Miles et al., 2008; Raynor et al., 2011; Pre-construct Archaeology Ltd, 2012; Henderson et al., 2013).*

Site	Period	Context	Status	No. individuals	No. non-adults
NORTH					
Coach Lane, N.Shields	c.1711-1857	Urban	Low/Middle	236	82
Coronation Street, S.Shields	c.1816-55	Urban	Low	204	90
LONDON					
Chelsea Old Church, Chelsea	c.1712-1842	Suburban	High	198	33
St Benet Sherehog, City of London	<1853	Urban	Middle	230	64
Bow Baptist, Bow	c.1816-1856	Suburban	Middle	416	202
Cross Bones, Southwark	c.1800-1853	Urban	Low	148	104

4.3.1 Coach Lane, North Shields

The Coach Lane skeletal collection is curated by the Department of Archaeology, Durham University. It consists of 236 individuals, 82 of whom are non-adults. The site is located within the borough of North Tyneside, to the east of Newcastle-upon-Tyne, and to the north of the river Tyne (Pre-construct Archaeology Ltd, 2012). Excavation was undertaken from April-September 2010, in response to plans for residential development on the site (Pre-construct Archaeology Ltd, 2012).

North Shields in the early 19th century was a populous port and market town, undergoing rapid development (see Fig.4.2) (Gibson, 1849; Gould and Chappel, 2000). The principal industries of this town in the 18th and 19th centuries were fishing, shipbuilding, shipping of coal and lime, iron-foundries, salt works, and various manufactories (Gibson, 1849; Tyne & Wear Historic Towns Survey, 2004). Due to its prime location for trade via the port in the estuary of the River Tyne, North Shields often faced rivalry from the neighbouring port town of Newcastle, opposition from which they faced throughout the founding and development of the town (Tyne & Wear Historic Towns Survey, 2004). The first indication of the existence of a settlement on the site that became North Shields was in reference to a cluster of fishermans' huts, or "sheels" associated with the Priors of Tynemouth Abbey in 1259 (Gibson, 1849; Haswell, 1895). From as early as this small fishing settlement, the burgesses of Newcastle consistently suppressed the development of North Shields, through restrictions to trade and prohibitions on the unloading and loading of ships (Gibson, 1849; Haswell, 1895). This led to the town of North Shields, and South Shields, which developed simultaneously directly across the River Tyne, being referred to as "…a town where no town should be…" (Haswell, 1895: 16). After the dissolution of the Tynemouth monastery in 1539, North Shields lost its protection from the Prior, severely affecting trade, and it became sparsely populated (Haswell, 1895; Tyne & Wear Historic Towns Survey, 2004). However, major developments in the 18th and

Figure 4.2 – *Depiction of the port-town of North Shields in the 19th century. Image taken from Haswell (1895).*

19th centuries led to the establishment of shipping, shipbuilding, manufacturing, and coal mining industries in North Shields, and its consequent rapid expansion (Tyne & Wear Historic Towns Survey, 2004).

Coach Lane was a former Society of Friends burial ground (c.1711-1857 AD), and excavations encompassed the entire area of the cemetery, therefore all burials within this site were exhumed (Pre-construct Archaeology Ltd, 2012). Following the start of the Quaker movement in the mid-17th century, meetings began in North Shields after a visit from George Fox (the founder of Quakerism) to Newcastle in 1653 (Pre-construct Archaeology Ltd, 2012). There is unfortunately limited biographical information for those interred at this site, and due to the simplicity and plainness of the Quaker lifestyle there are limited grave goods from which social status can be inferred (PCA-North, 2011; Pre-construct Archaeology Ltd, 2012). A previous study of the Coach Lane population suggested that this Quaker community was relatively prosperous in the 18th and 19th centuries, and prior to the establishment of a new burial ground in Stephenson Street in 1811, the majority of Quakers within North Shields were likely to have been buried here (Grybowska, 2011; Pre-construct Archaeology Ltd, 2012). Diaries and records kept by Quaker families are suggestive of the existence of relatively affluent members of the Society of Friends within this region (Watson, 1864; Boyce, 1889). The higher mean and median ages of individuals interred at Stephenson Street (another Quaker burial ground of North Shields) compared to that of those within Tynemouth General Cemetery implied that the Quaker community of this area may have been relatively well-off (Gould and Chappel, 2000; Grybowska, 2011). Therefore, when considering the data from the Coach Lane population, it is possible that they represent individuals of the 'middling sort'. However, this assumption should be approached with caution, as North Shields was heavily industrialised at the time and the effects of a heavily polluted environment would have been detrimental to all social classes. In 1845, the Second Report of The Commissioners described the towns and villages of the northern districts (North and South Shields included) as being destitute of "…those conveniences which common decency requires…" and there being an "…absence of a sufficient and easily accessible supply of wholesome water, and the utter inefficiency of the means resorted to for cleansing, drainage,

and sewerage…" (Report of the Commissioners, 1845b: 10). Therefore is it more likely that this group represents a mix of low and middle status individuals.

4.3.2 St Hilda's Parish Church, Coronation Street, South Shields

Excavations at the burial ground of St Hilda's Parish Church (c. 1816-55 AD), South Shields, located south of the River Tyne, have provided skeletal remains from a working class northern population (referred to hereafter as the Coronation Street skeletal collection). This skeletal collection consists of 204 individuals, 90 of whom are non-adult. Excavation on this site began in 2006 by Oxford Archaeology North, and was completed in 2007 (Raynor *et al*., 2011). While this cemetery has been in use since c. 1402, the excavation area relates to the southern section of the burial ground, and is split into three burial horizons that date to the 18th and 19th centuries (Raynor *et al*., 2011). Interestingly, the western half of the lower burial horizon lay outside of the original boundaries of the cemetery; this may have been used for unconsecrated burials, such as unbaptised infants (Raynor *et al*., 2011). The majority of the burials in this western section of the excavation site were indeed pre-term or perinatal infants (Raynor *et al*., 2011). The human remains from this site were analysed in 2012, and the skeletal collection is now curated by the Department of Archaeology, University of Sheffield.

South Shields was a manufacturing and shipbuilding town that developed in parallel to North Shields across the River Tyne (see Fig.4.3) (Hodgson, 1903). The individuals of this population are said to have been employed in local industries, such as the shipyards and port, gas works, salt-works, a glass factory, chemical works, and in nearby collieries (Raynor *et al*., 2011; Green, 2010b). While generally regarded as being of "working class", the majority of the population would have had sufficient means to buy food and shelter as the wages paid in many of these industries were comparably good. While the "working-class" usually accommodates a large spectrum of family circumstance, from the "comfortably working-class" to those of more of a pauper

Figure 4.3 – *Joseph Mallord William Turner's painting "Shields, on the River Tyne", 1823. Depicting the vital role in the coal trade played by the shipping industries within North and South Shields. Image taken from Tate (2014).*

status, those interred at St Hilda's appear to have afforded superior funerary arrangements to that available to paupers reliant on parish or workhouse burials (Raynor *et al*., 2011).

However, like many industrial centres of this time, South Shields also suffered from the insanitary and polluted environments promoted by increasing population density, and the effluence from the surrounding industries (Green, 2010b). As already described in the above section regarding North Shields, Commissioners in 1845 reported on the noxious conditions of industrial towns of the northern districts, and referred to South Shields as having "…abundant evidence of the causes of that amount of disease which led the towns in which they occur to be selected as objects of particular attention." (Report of the Commissioners, 1845b). The houses of the poor were reported to be inadequately ventilated, as well as having defective water supplies and drainage provisions (Hodgson, 1903). In addition, prior to its closure in 1855 St Hila's churchyard had become extremely overcrowded, thus contributing to the insanitary conditions that prevailed in this town (Report of the Commissioners, 1845; Hodgson, 1903).

4.3.3 All Saints, Chelsea Old Church, Chelsea, London

The skeletal collection of All Saints, Chelsea Old Church (c.1712-1842), hereafter referred to as Chelsea Old Church, is curated by the Museum of London, Centre for Human Bioarchaeology. Chelsea today is classed as being part of central London in the borough of Kensington, located to the west of the city centre. However, in the 18th century Chelsea was located on the outskirts of the city, still retaining a relatively rural setting despite its gradual transition to a London suburb (Davies, 1904). After the original church (from the 12th century) (see Fig.4.4) was mostly destroyed in a bombing raid in 1941, a new church was rebuilt on this site in the 1950s, followed by a new vicarage and church hall in the early 1960s (Cowie *et al*., 2008). The vicarage and church hall covered the section of the cemetery that had been in use during the 18th and 19th centuries, and prior to the building work, permission was granted for the clearance of these burials (Cowie *et al*., 2008). Excavation of this area of the cemetery following demolition of these buildings began in 2000, and by the end of excavation had yielded 290 interments dating to the 18th and 19th centuries (Cowie *et al*., 2008). Of the 290 individuals, 198 were

selected for osteological analysis within the site report, and biographical information is available for 25 of the individuals within this population (Cowie *et al*., 2008). The Chelsea Old Church skeletal collection is therefore comprised of 198 individuals, 33 of whom are non-adult.

The population of Chelsea Old Church represents those of a higher status who resided in the suburbs of the city. It has been described as "a relatively healthy and prosperous place" and "a fashionable resort for Londoners" (Davies, 1904; Cowie *et al*., 2008: 13). It has been stated, "There is scarcely another parish within the environs of London which can boast of so long a roll of illustrious or memorable inhabitants…" (Davies, 1904: xii), and was "…celebrated for the residence of many of the nobility and gentry…" (Davies, 1904: 36). The surrounding rural land meant that many of the market gardens that supplied the city were established here, and towards the end of the 18th century was thought to include around 130 acres of pasture meadow, and 150 acres of arable land (Lysons, 1795). However, as London continued to grow, by the mid-19th century Chelsea became amalgamated with the expanding city, becoming increasingly urban in character (Cowie *et al*., 2008). While in 1801 the population of Chelsea was believed to be approximately 12,079, by 1902 this had dramatically increased to around 73,842 (Mitton, 1902).

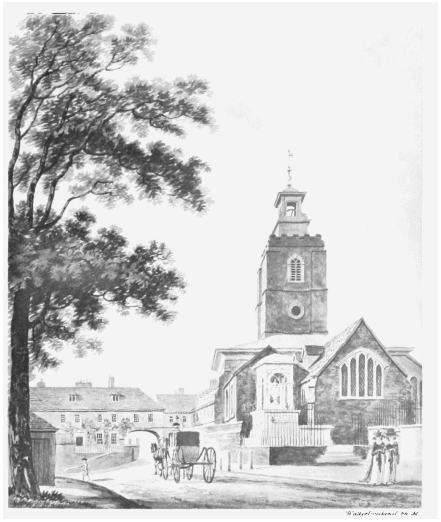

Figure 4.4 – *Image of Chelsea Old Church. Taken from Godfrey (1921: xvi).*

4.3.4 St Benet Sherehog, City of London

Excavations at 1 Poultry, in the City of London, from 1994 to 1996 revealed 280 burials related to the parish church of St Benet Sherehog before and after the Great Fire of 1666 (Miles *et al*., 2008a). The burials from this site are representative of both medieval and post-medieval populations, with 56 individuals believed to date to before the Great Fire, and the remaining 224 being more likely associated with the later burial ground (post-Fire). The excavated burial ground of post-medieval St Benet Sherehog is thought to have been in use from the 16th-19th

century, therefore this sample will represent a population from a wide temporal range within the post-medieval period during which this district saw great changes in the surrounding industrialised city (Miles *et al*., 2008a). After the Great Fire destroyed the church it was not rebuilt, and the land was instead used solely for new burials for the newly combined parishes of St Benet and St Stephen Walbrook (united in 1670), until its closure in 1853 (Miles *et al*., 2008a). A sample of 267 individuals (both pre-Fire and post-Fire) were retained for osteological analysis. The post-medieval group of the St Benet Sherehog skeletal collection (curated by the Museum of London, Centre for Human Bioarchaeology) comprises of 230 individuals, 64 of whom are non-adults.

The post-medieval St Benet Sherehog population represents individuals of the middling class. The parish of St Benet Sherehog, located within the City of London, was affluent and small, and in the 14th century was considered be one of the wealthiest areas of the city (Miles *et al*., 2008a). This affluence continued through to the 16th and 17th centuries, with the predominant trade in this parish being that of a grocer (Miles *et al*., 2008a). Following the rebuilding of the parish after the fire, St Benet still retained its wealthy status, however by the 18th-19th centuries it represented a reasonably prosperous but declining population, as more and more businesses relocated to the outer regions of the city (Knight, 1851; Miles *et al*., 2008a). The parish of St Benet Sherehog was also a major contributor to poor relief and, as in every part of London at this time, some of those living within this parish would have been living in conditions of poverty. Therefore it is possible that individuals of a lower class may have also been interred within this burial ground alongside the wealthy.

Figure 4.5 – *Image depicting the environs of Cheapside, London, in 1837 close to the location of the former St Benet Sherehog church. Image taken from Ancestry Images (2015).*

The location of this parish in the centre of the City of London meant that it was placed within the epicentre for industrial growth (see Fig.4.5). The developing industries and resultant employment opportunities attracted more and more people into the city, thus it is likely that life within the city centre still exposed its inhabitants to increased population density, and the pollution and poor sanitation that was ubiquitous throughout the metropolis.

4.3.5 Baptist Congregational burial ground, Bow Baptist Church, London

Excavations in the Borough of Tower Hamlets, East London, between 2005-2008 resulted in the uncovering of three burial grounds dating to the post-medieval period (Henderson *et al*., 2013). Of the 1,375 burials recovered during this excavation, 416 individuals belonged to the Bow Baptist Church site, and are curated by the Museum of London Archaeology, and housed at the Museum of London, Centre for Human Bioarchaeology. In addition, 100 of these individuals have been identified via coffin plates and cross-referencing of burial register locations with osteological evidence (Henderson *et al*., 2013). The Bow Baptist skeletal collection of Payne Road, London represents a relatively prosperous post-medieval population (see Fig.4.6) (c. AD 1816-56), with a large proportion of merchants and tradesmen interred in the burial ground (Henderson *et al*., 2013). Of the 416 individuals, 202 are classed as non-adult (Henderson *et al*., 2013). The burial ground was excavated in two stages, so this collection is comprised of individuals from the PAY05 site, and the BBP07 site.

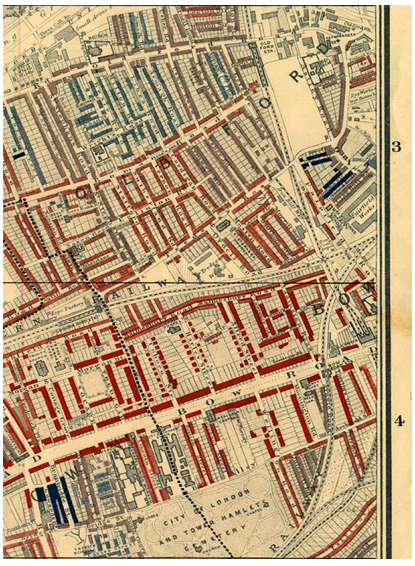

Figure 4.6 – *Section of Charles Booth's 1889 descriptive map of London poverty representative of Bow and it's immediate surroundings. Bow continues beyond the eastern limits of this map, however a high proportion of middle class, "well-to-do", households were in this area. Image taken from Ahmad (1999).*

At the beginning of 1816, Bow existed as a village on the eastern outskirts of London, with a small population of around 2,000 at the start of the 19th century (Henderson *et al*., 2013). However, the second half of the 19th century saw the rapid industrialisation of this area, with the opening of factories producing matches, rubber,

soap, and hemp cloth (Henderson *et al*., 2013). Bow was eventually incorporated into the growing city as the outer limits of London continued to push further afield, and the population of this area grew alongside the increasing urbanisation (Henderson *et al*., 2013). The Bow Baptist church belonged to the parish of St Mary Stratford, and represents a "nonconformist" population (i.e. non-Church of England). By the end of the 18th century, there were over 90 registered within the congregation of the Bow Baptist Church, and this expanding Baptist community necessitated the construction of a new chapel in 1801, and plots for a burial ground purchased in 1814 (Henderson *et al*., 2013). In the early 19th century, this congregation continued to grow, and despite the new church being able to accommodate 600, within 10 years of its opening it was once again too small for the rising numbers (Henderson *et al*., 2013). However, by the mid-19th century the increasing industrialisation of the area began to deter members of the congregation, and numbers fell as families moved away from the city, which is indicative of the deterioration of this area associated with its amalgamation with the polluted metropolis (Henderson *et al*., 2013).

4.3.6 Cross Bones burial ground, Redcross Way, Southwark, London

A multitude of archaeological excavations were carried out between 1991-1998 on behalf of the London Underground Jubilee Line Extension Project, one site of which was located in the London Borough of Southwark, to the south of the River Thames (Brickley and Miles, 1999). Redcross Way had been identified via documentary evidence as the site of a post-medieval burial ground, named Cross Bones, and following confirmation the main phase of excavation occurred in 1993 (Brickley and Miles, 1999). The Cross Bones collection consists of 148 individuals, 104 are non-adult, and thus represent a large proportion of this skeletal collection. Such a high representation of non-adults may be an artefact of the excavation process, which unfortunately only recovered 1% of the number interred at Redcross Way (Brickley and Miles, 1999). It is possible that this area of the cemetery was more frequently utilised for the burial of the very young, skewing the demography of this skeletal sample. It was not unusual for the burials of children to be confined to distinct areas of burial grounds, a practice identified in other burial sites dating to the 19th century (Brickley and Miles, 1999; Henderson *et al*., 2013). The Cross

Bones skeletal collection is curated by the Museum of London, Centre for Human Bioarchaeology.

The Cross Bones burial ground is located within the parish of St Saviour's, Southwark. According to Booth, in 1891 St. Saviour's Southwark (parish of the Cross Bones burial ground) sat on a "wretched throne" of poverty in South London (1891: 395). This unconsecrated burial ground is renowned for the great poverty experienced by those interred there, and was reserved for the very poorest of society, including those who received pauper burials (Brickley and Miles, 1999). This population represents those who would have likely experienced the worst of the detrimental urban sanitary conditions, with areas of the parish being described as "a ruinous and filthy slum" (see Fig.4.7) (Brickley and Miles, 1999: 20). It is most notorious for its use as a burial ground for "single women" (prostitutes), however later was heavily used as a parish poor ground (Brickley and Miles, 1999).

Figure 4.7 – *Image depicting the conditions of poverty experienced by many living within London in the 18th/19th centuries. Taken from Price (2014).*

4.4 Summary

The six urban-based sites selected for analysis within this study are demonstrative of the spectrum of wealth that existed within the cities of the 18th-19th centuries, with Chelsea Old Church being representative of the upper classes, St Benet Sherehog and the Bow Baptists of the middle classes, and Cross Bones and Coronation Street of the lower classes. Coach Lane is tentatively classed as containing individuals of low/middle status. These sites have also been selected to allow for a comparison of child health within northern and southern centres of industry. With the study sites identified, the methods by which the skeletal samples were assessed for variances in health status will be now be described.

CHAPTER FIVE

Methods

5.1 Introduction

This chapter will outline the methods used throughout this study. These relate to the ageing of both non-adult and adult individuals from each site, procedures for metric analysis, standards for the recording of specific pathologies, and finally statistical techniques implemented during data analysis. An example recording form for non-adult individuals can be found in Appendix 1, and for adult individuals in Appendix 2. A summary table for non-adult metric data for each site can be found in Appendix 8. Overall, 575 non-adult skeletons were assessed for evidence of pathology, and measurements of growth taken where preservation allowed.

5.2 Age estimation in non-adults

The most accurate approximation of chronological age in non-adults is through the estimation of dental age (Saunders, 2008). Dental age was primarily assessed according to dental development using standards for calcification (Moorrees *et al.,* 1963a,b; Smith, 1991). These dental age estimations were cross-checked with additional standards for dental development and eruption (Ubelaker, 1978; Van Beek, 1983) to aid the placement of individuals bordering between age categories. Calcification stages were determined by examination of the dentition radiographically (see Fig.5.1), or macroscopically when loose teeth were present. These stages were used to assign a dental age to each individual, based on the mid-point of the age category that they fell into. For example, those between 0.5-1.49 years of age were classed as 1 year of age (Mays *et al.,* 2008). Individuals within the samples that had no dentition due to post-mortem loss were omitted from further metric analysis, as the dental age could not be assessed. Ageing of the individuals within the metric sample using other indicators of skeletal maturation (e.g. epiphyseal fusion and diaphyseal length) would not be suitable alternatives to the use of dental criteria when creating growth profiles, as they are more susceptible to disruption from environmental factors (Cardoso, 2007; Saunders, 2008).

For the remaining non-adult individuals that had no dentition present, and

who were not selected for metric analysis, age estimates were made based on standards for skeletal maturation (epiphyseal fusion and diaphyseal length) (Scheuer and Black, 2000). All individuals were placed into one of the following age categories when possible: "Foetal and perinatal" (see Table 1.1 for age boundaries for these categories), "1-11 months", "1-5 years", "6-11 years", and "12-17 years". This allowed for the analysis of age-at-death distribution for each site, and for a breakdown of pathology rates in each population by age.

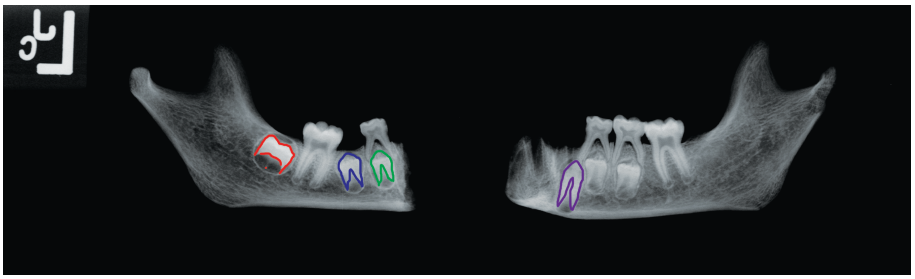

Figure 5.1 – *Radiographic analysis of the mandibular dentition for non-adult dental ageing using calcification stages by Moorrees et al (1963a,b). ONE94 SK294. Radiograph: Gerald Conlogue.*

5.3 Age estimation in adults

As this study includes the metric analysis of adult individuals to provide comparative samples, it was necessary to provide accurate age estimates of the adult populations within each site so that a suitable sample could be selected. Information relating to the age and sex of the adult individuals was taken from each site report/MoL online databases (Brickley and Miles, 1999; Cowie *et al.*, 2008; Miles *et al.*, 2008a; Raynor *et al.*, 2011; WORD database, 2012a; WORD database, 2012b; WORD database, 2012c; Henderson *et al.*, 2013), and recorded personally from the Coach Lane skeletal collection. Using these data, adults aged between 18-35 years of age, or classed as "young adult" or "mature adult", were selected for metric analysis. The age categories 18-25, and 26-35 years were then corroborated independently for the purposes of this study using standards for age estimation of the pubic symphysis (Brooks and Suchey, 1990), the auricular surface (Lovejoy *et al.*, 1985), and late-fusing epiphyses (Scheuer and Black, 2000). Individuals that could not be reliably placed into one of these two age categories were excluded from any further analysis. Overall, 224 adults were selected from the six sites for metric analysis; see Appendix 3 for sample sizes for adult metric data.

5.4 Diaphyseal length

For assessment of diaphyseal length, long bone length for each non-adult individual was plotted against dental age for each of the populations to form a growth profile. The maximum diaphyseal length was measured for the left femur, tibia, and humerus using a standard osteometric board (Buikstra and Ubelaker, 1994). When the left long bone was absent it was substituted with the right, as differences in size between them would not be expected to be significant (Ives and Brickley, 2004). However, when both left and right long bones were absent, the individual was discounted from the sample. This was also necessary for long bones that had undergone post-mortem damage, or demonstrated extensive fraying of the bone ends due to rickets. The tibia was selected for data comparisons between the six sites throughout the four manuscripts, as it undergoes rapid growth during development, so is thought to be more sensitive than the other long bones to growth disruption (Holliday and Ruff, 2001; Bogin *et al*., 2002; Pomeroy *et al*., 2012). For the Chelsea Old Church, St Benet Sherehog, Bow Baptist, and Cross Bones collections tibial diaphyseal length had previously been recorded according to the above standards for measurement; therefore these data were taken from the WORD database (WORD database, 2012a; WORD database, 2012b; WORD database, 2012c; Bow Baptist database retrieved from MoLA but is not publicly available online). Measurements for long bone length for each site can be found in Appendix 8, overall sample sizes for each site for tibial diaphyseal length in Table 5.1, and a breakdown of sample sizes by age for tibial diaphyseal length in Appendix 4.1.

Modern comparative data exists for individuals 0-18 years of age from the study by Maresh (1955). This sample represents a healthy modern data-set from Colorado, US, and is based on a study of 175 individuals, and includes measurements of long bone lengths for the femur, tibia, and humerus (Maresh, 1955). Data taken from this study was pooled for males and females, to ensure comparability with the mixed sex archaeological non-adult data sets. Measurements were taken from the 50% percentile group, and reduced by 1.5% to account for radiographic enlargement (in accordance with MacCord, 2009).

5.5 Cortical thickness

To measure cortical thickness (CT), the left femur, tibia, and humerus were selected from each non-adult for radiographic analysis. Antero-posterior

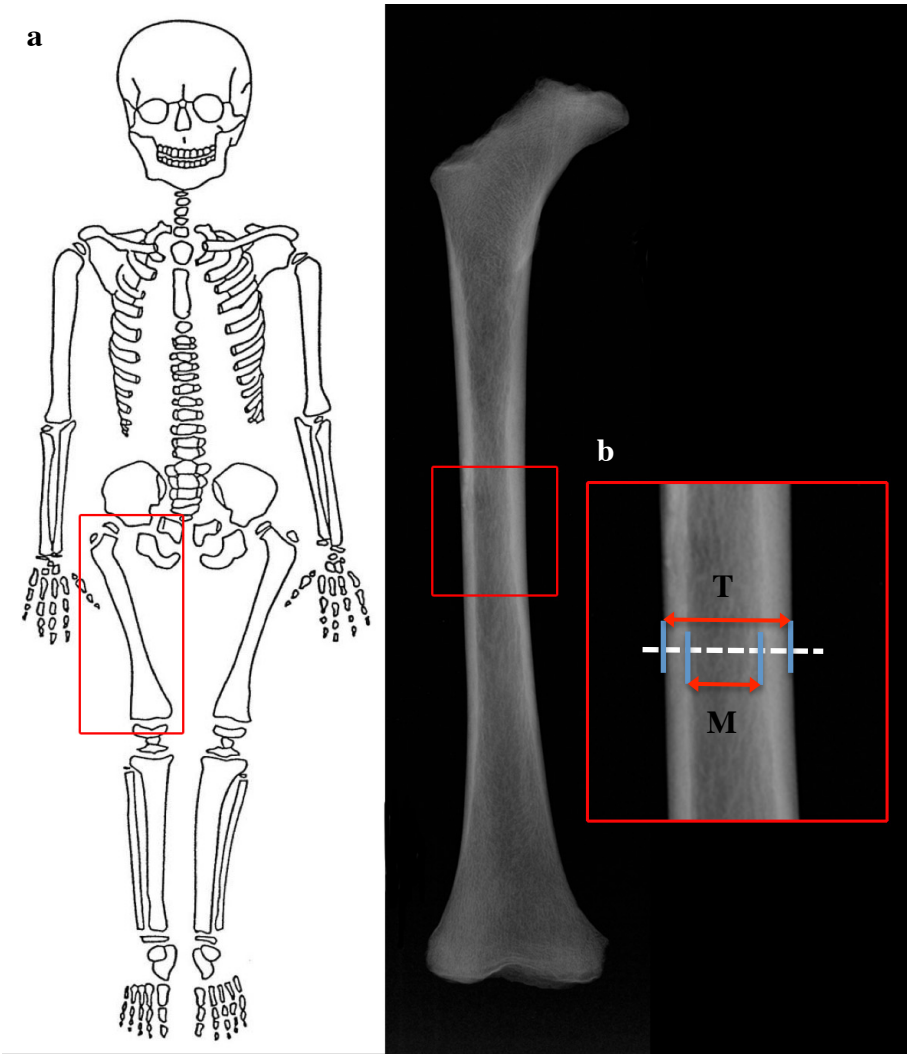

Figure 5.2 – *Measurement of cortical thickness, a) locations of measurements taken from femur, and b) method for the measurement of T and M from the radiographs. Tibial measurements taken following the same protocol. PAY05 SK559 Radiograph: Gerald Conlogue.*

radiographs were taken at between 65-80kVp and 4-6mAs from the Coach Lane sample using a Portable GE Medical MPX X-ray unit and Kodak point of care CR System, from the London based samples using a Kubtec Xtend 100HF x-ray source and Kubtec 3600 CR reader (by Professor Gerald Conlogue, personal communication, 20th April 2015), and from the Coronation Street sample using a NOMAD Pro handheld x-ray system (with the assistance of Dr Diana Swales).

Measurements of the total bone width (T) and the medullary width (M) were taken from the mid-shaft of the femur, in accordance with the criteria followed by Mays *et al*. (2009a), and also the tibia due to its sensitivity to environmental disruption (see Fig.5.2) (Holliday and Ruff, 2001; Bogin *et al*., 2002; Ives and Brickley, 2004; Pomeroy *et al*., 2012). Cortical thickness was determined as T-M,

and plotted against the dental age to form a cortical thickness growth profile for each population using the mean CT for each age category (Mays *et al*., 2009a).

The sample size for analysis of cortical thickness is larger than that for diaphyseal length, as long bones with slight post-mortem damage could still be used as long as the site of measurement (mid-shaft) could be determined reliably. Measurements for M, T, and CT for each site can be found in Appendix 8, overall sample sizes for each site for the femur and tibia in Table 5.1, and a breakdown of sample size for the femur and tibia by age in Appendix 4.2 and 4.3.

Modern comparative data for CT exists for individuals 0-18 years of age from the study by Virtama and Helëla (1969). This study was undertaken on a healthy modern Finnish population, and includes CT measurements for 164 femora, 227 tibiae, and 150 humerii (Virtama and Helëla, 1969). Cortical thickness data was taken for the femur and tibia for comparison against the archaeological data sets. As with the Maresh (1955) data, measurements were pooled for the sexes. However, as the archaeological data was also recorded via radiographs, there was no need to account for any radiographic enlargement.

5.6 Vertebral growth

For measurement of the vertebral dimensions used in this study, individuals between 0-17 years of age were selected based on the presence of vertebrae whose position within the vertebral column could be reliably determined. In infants the morphology of the vertebral body is more ambiguous than in later ages, therefore this made accurate identification of vertebral position problematic when only a few vertebral elements were preserved. Therefore, for those aged around 1-2 years, and under, the majority of the vertebral elements had to be present for inclusion of the individual in the study. In addition, any vertebrae that displayed signs of pathology such as Schmorl's nodes were omitted from the study. Schmorl's nodes are indentations that occur on the superior and/or inferior surfaces of the vertebral body in response to herniation of the nucleus pulposus of the intervertebral disc (Plomp *et al*., 2012). Their aetiology is uncertain, with spinal trauma, and stress from physical activity being suggested influences in their formation (Plomp *et al*., 2012). However, there appears to be a correlation between their appearance and the shape of the posterior vertebral body and the pedicles, therefore there may be an element of developmental predisposition to the formation of Schmorl's nodes (Plomp *et al*.,

2012). Individuals with border shifts were also excluded from further vertebral metric analysis. Border shifts occur when vertebrae develop morphological features associated with the neighbouring section of the vertebral column, e.g. in the lumbarisation of T12 (Barnes, 1994). It was necessary to exclude these individuals from further analysis as it is yet to be established whether these anomalies would have affected the course of vertebral development (Barnes, 1994; Watts 2011; 2013a,b). Measurements of vertebral body height and vertebral neural canal (VNC) dimensions were taken from the non-adult and adult individuals from each skeletal collection that met the above criteria.

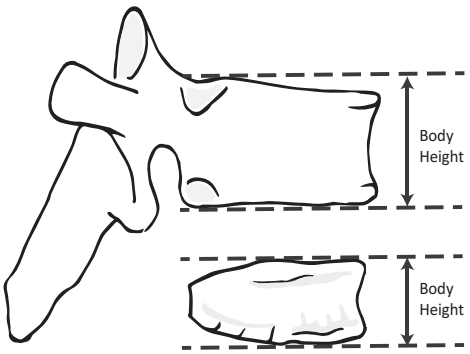

Figure 5.3 – *Measurement locations for vertebral body height in fused and unfused vertebrae. Taken from the midline of the centrum.*

Measurements of vertebral body height were taken from the midline of each centra (C3-L5) at the point of maximum height. To achieve this, the inferior surface of the body was positioned horizontally on the sliding calipers and the mobile component moved until it touched the superior surface, thereby marking the point of maximum body height (Fig. 5.3). Measurements were taken to the nearest 0.01mm. Due to the linear increase in vertebral body height with age, it was possible to calculate measurements for missing vertebrae using the average value from the two adjacent vertebrae, when present (Auerbach, 2011).

To assess VNC size, measurements of TR diameter of the neural canal of the cervical, thoracic, and lumbar vertebrae (C1-L5) were taken using sliding calipers (to the nearest 0.01mm). These measurements represented the furthest distance between the medial surfaces of the left and right pedicles (Watts, 2011; 2013a,b) and could only be taken when the neural arches had fused at the spinous process, though fusion of the neural arch to the vertebral body was not essential (Fig.5.4). The AP diameter (normally

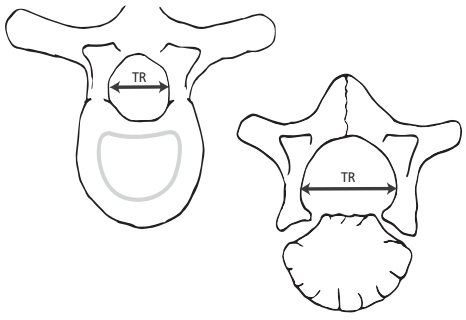

Figure 5.4 – *Measurement location of the transverse diameter of the neural canal in fused and unfused vertebrae.*

taken from the posterior surface of the vertebral body to the furthest opposite point of the neural canal, anterior to the spinous process) (Watts, 2011; 2013a,b) was excluded from this study as these measurements could only be taken in individuals that had begun fusion of the neurocentral synchondrosis. Although this measurement is of great value in adult individuals, its use within non-adult samples is restricted to those individuals whose neural arches and vertebral bodies have fused (i.e. older than approximately five years of age). This limitation means that sample sizes for AP diameter tend to be small, as older children and adolescents appear less frequently in skeletal collections (Lewis, 2007).

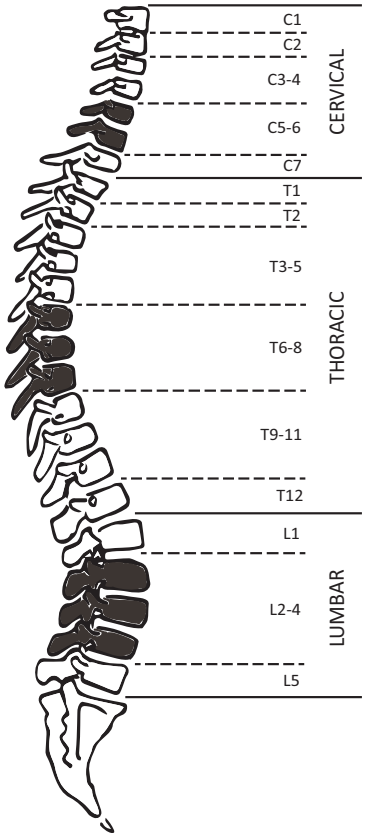

Figure 5.5 – *Vertebral groupings used for analysis of measurements. Shaded elements reflect the vertebral groups for which data will be presented within this study.*

Once measurements had been taken, vertebrae were categorised into groups to maximize sample size (Fig.5.5). The groups were delimited based on similarities in morphology of vertebrae, and averages of the vertebral measurements for each individual were calculated for each vertebral group. Transitional vertebrae (C7, T1, T12, L1, and L5) were considered separately. Vertebral groups C5-6, T6-8, L2-4 were chosen for further analysis due to their higher rate of preservation, and therefore larger sample sizes. These grouped measurements were then plotted against dental age to form vertebral growth profiles for C5-6, T6-8, and L2-4. Measurements for vertebral dimensions for each site can be found in Appendix 8, overall sample sizes for each site in Table 5.1, and a breakdown of sample size by age for C5-6, T6-8, and L2-4 in Appendix 4.4, 4.5, and 4.6 for vertebral body height, and Appendix 4.7, 4.8, and 4.9 for TR diameter.

Measurements of body height and TR diameter were also taken from adults aged 18-35 years in both collections to provide comparative data, and to assess the impact of early life stress on adult longevity (Clark *et al.,* 1986; Clark, 1988; Watts, 2011, 2013a,b). Adult vertebral measurements can be found in Appendix 10, and

overall sample sizes for each site in Appendix 3. While there is unfortunately no modern comparative data set for vertebral body height, a study by Hinck *et al*. (1966) provides average transverse diameter measurements for 353 children from Oregon, US, up to 18 years of age (data averaged into ages 4, 7, 9, 12, 14, and 16), and also averages for measurements from 121 adults (aged 18 years and above).

Table 5.1 – *Number of individuals from each site available for non-adult metric analysis (percentage of total non-adult sample for each site in brackets). BH= vertebral body height; TR=transverse diameter.*

SITE	Tibial length	Femoral CT	Tibial CT	BH C5-6	BH T6-8	BH L2-4	TR C5-6	TR T6-8	TR L2-4
Coach Lane	22 *(26.8)*	20 *(24.4)*	26 *(31.7)*	31 *(37.8)*	23 *(28)*	19 *(23.2)*	12 *(14.6)*	5 *(6.1)*	12 *(14.6)*
Coronation Street	19 *(21.1)*	31 *(34.4)*	23 *(25.6)*	20 *(22.2)*	20 *(22.2)*	19 *(21.1)*	10 *(11.1)*	10 *(11.1)*	10 *(11.1)*
Chelsea Old Church	10 *(30.3)*	10 *(30.3)*	9 *(27.3)*	7 *(21.2)*	10 *(30.3)*	10 *(30.3)*	5 *(15.2)*	10 *(30.3)*	8 *(24.2)*
St Benet Sherehog	9 *(14.1)*	13 *(20.3)*	11 *(17.2)*	18 *(28.1)*	16 *(25)*	14 *(21.9)*	13 *(20.3)*	8 *(12.5)*	8 *(12.5)*
Bow Baptist	70 *(34.7)*	48 *(23.8)*	36 *(17.8)*	49 *(24.3)*	42 *(20.8)*	40 *(19.8)*	42 *(20.8)*	46 *(22.8)*	43 *(21.3)*
Cross Bones	36 *(34.6)*	37 *(35.6)*	35 *(33.7)*	37 *(35.6)*	38 *(36.5)*	36 *(34.6)*	14 *(13.5)*	22 *(21.2)*	16 *(15.4)*
Total	166	159	140	162	149	138	96	101	97

5.7 Palaeopathological methods

All non-adult individuals within each skeletal collection were also assessed for the presence/absence of six pathological conditions. This assessment was either carried out by the author (for Coach Lane and Coronation Street), or taken from pathological data from the WORD database (WORD database, 2012a; WORD database, 2012b; WORD database, 2012c; Bow Baptist database retrieved from MoLA but is not publicly available online). Each individual was assessed according to the criteria outlined below for skeletal indicators of rickets, scurvy, periosteal new bone formation, cribra orbitalia, and dental enamel hypoplasia (DEH). Adult individuals were assessed for presence/absence of residual rickets, periosteal new bone formation, cribra orbitalia, and DEH. A summary table for presence/absence of pathology for each non-adult individual within the six sites can be found in Appendix 9. Crude prevalence rates for adult health data can be found in Appendices 5 (Overall), 6 (Males), and 7 (Females).

Table 5.2 – *Diagnostic criteria for the pathology categories "Scurvy", "Possible scurvy", "Rickets", "Possible rickets", and "Metabolic disease". Placement into category dependent on presence of multiple features from each column in accordance with – Ortner and Erickson (1997), Ortner and Mays (1998), Brickley and Ives (2006), Mays et al. (2006), Pinhasi et al. (2006), Brickley and Ives (2008), Armelagos et al. (2014), Klaus (2014a), and Stark (2014). NBF = New bone formation.*

Region	Feature	Diagnostic criteria for "Scurvy"	Criteria for "Possible scurvy"	Diagnostic criteria for "Rickets"	Criteria for "Possible rickets"	Criteria for "Metabolic disease"
Cranium	Porosity/NBF on sphenoid	✔				
	Porosity/NBF on maxillae	✔				
	Porosity/NBF on mandible	✔				
	Porosity/NBF in orbits	✔				
	Porosity/NBF on cranial vault		✔		✔	✔
	Medial angulation of mandibular ramus			✔		
Ribs	Alteration of neck angle			✔		
	Lateral straightening			✔		
	Flaring/swelling of rib ends		✔		✔	
Dentition	DEH					✔
	Caries				✔	✔
Scapulae	Porosity/NBF on infra- and supra-spinous regions	✔				
Long bones	NBF		✔		✔	✔
	Flaring/swelling of metaphyses		✔	✔		
	Porosity of growth plate				✔	
	Cupping deformities		✔	✔		
	Bowing			✔		
	Thickening		✔		✔	✔
	Coxa vara			✔		
	Osteopenia (Radiograph)		✔		✔	✔
	Irregularity and thinning of cortex (Radiograph)		✔		✔	✔

5.7.1 Rickets

The classic indicators of rickets in the skeleton (and implemented in this study) include bowing of the long bones, flaring of the metaphyses, coxa vara (where the angle of the femoral neck is reduced), and thickening of the diaphysis (Mays *et al*., 2006; Pinhasi *et al*., 2006) (Fig.5.6). Other indicators include porosity of the growth plate, new bone formation, flaring of the costo-chondral rib ends, and porosity of the cranial bones (Ortner and Mays, 1998; Brickley and Ives, 2008) (Fig.5.7). Those demonstrating diagnostic indicators of this condition were classed as having "rickets"; whereas those demonstrating non-diagnostic skeletal indicators were placed in the category "possible rickets", see Table 5.2. Cases of both active and healed rickets were considered together in this study, as a previous study by Mays *et al*. (2009b) suggested that the effects on growth between the two was not statistically significant.

5.7.2 Scurvy

This condition can be detected skeletally through evidence of bone formation and porosity resulting from haemorrhaging of the blood vessels in areas where movement frequently occurs (Ortner and Erickson, 1997). For example, in the orbits, on the long bones (in association with the joints), and on the mandible and maxillae (Ortner and Erickson, 1997; Brickley and Ives, 2008; Armelagos *et al*., 2014; Stark, 2014; Klaus, 2014a) (Fig.5.8). Other indicators include antemortem tooth loss, and new bone formation and porosity on the cranial bones (particularly the sphenoid bone) and scapulae (Ortner and Erickson, 1997; Brickley and Ives, 2006; Armelagos *et al*., 2014; Klaus, 2014a; Stark, 2014) (Fig.5.9). In those whose long bones had been radiographed for CT analysis, radiographic indicators of metabolic disease (such as osteopenia and irregularity of the cortex) could also be assessed. Those demonstrating multiple diagnostic indicators of this condition were classed as having "scurvy", whereas those demonstrating non-diagnostic skeletal indicators were placed in the category "possible scurvy", see Table 5.2.

Figure 5.6 – *Skeletal indicators of rickets; a) Bowing of the long bones. Left and right femora demonstrating coxa vara, and left tibia with mediolateral bowing of the diaphysis (COL10 Sk75); b) mediolaterial view of the left and right femora, demonstrating anteroposterior bowing of the diaphyses (COL10 Sk75; c) thickening of the proxminal end of the right humeral diaphysis (COL10 Sk108); d) anteroposterior and mediolateral views of bowing of the right tibia, and new bone formation along the medial surface of the tibial diaphysis (COL10 Sk39); e) flaring of the distal metaphyses of the right femur and tibia (COL10 Sk104); f) coxa vara deformity of the femoral head (left – COL10 Sk156) vs normal angulation of the femoral head (right – COL10 Sk157). Photos taken by author.*

Figure 5.7 – *More skeletal indicators of rickets; a) Flaring of the rib ends (COL10 Sk39); b) Porosity of the cranial bones as seen in the left temporal bone above (COL10 Sk100); c) Porosity of the growth plate on the distal end of a right tibia (COL10 Sk100). Photos taken by author.*

Diagnosis of scurvy within perinatal individuals is problematic, as normal bone growth and pathological skeletal changes can be easily confused at this age. The development of scurvy is most commonly associated with those aged 6 months-2 years of age (Crawford, 2010), therefore the potential for misdiagnosis is usually not a significant issue. However, the Cross Bones site is reported to have a particularly high rate of scurvy within its perinatal sample (Brickley and Miles, 1999; WORD database, 2012c). The diagnostic criteria for scurvy have continued to evolve over recent years (Ortner and Erickson, 1997; Brickley and Ives, 2008; Armelagos *et al.*, 2014; Stark, 2014; Klaus, 2014a). Therefore individuals were re-categorised into the groups "scurvy", "possible scurvy", and "metabolic disease" (see below) based on detailed descriptions of skeletal changes from the WORD

database (WORD database, 2012c) in accordance with criteria from more recent publications (Brickley and Ives, 2008; Armelagos *et al*., 2014; Stark, 2014).

Figure 5.8 – *Skeletal indicators of scurvy; a) New bone formation (endocranial) and porosity (ectocranial) on the left greater wing of the sphenoid (COL10 Sk212); b) Porosity and new bone formation on the supra- and infraspinous surfaces of the left and right scapulae (CS06 Sk401); c) New bone formation in the left orbit (COL10 Sk212); d) Porosity of the right maxilla (COL10 Sk212). Photos taken by author.*

5.7.3 Metabolic disease

Scurvy and rickets have many skeletal changes in common (see Table 5.2), and also frequently co-occur, which can lead to issues regarding their identification in past populations (Ortner and Mays, 1998; Stark, 2014). To account for any potential misidentification of these two conditions, individuals with rickets and scurvy were also considered together under the separate category "metabolic disease". This category encompasses all individuals demonstrating diagnostic indicators of "rickets" and "scurvy", but also those with "possible rickets", "possible scurvy", and those that could not reliably be separated out into either of the two discrete conditions (see Table 5.2). Prevalence rates for rickets, scurvy, and metabolic disease are based on the total number of non-adults within each collection, regardless of preservation.

5.7.4 Periosteal new bone formation

Periosteal new bone formation refers to areas of bone formation or porosity on the skeleton that cannot be attributed to a specific cause, but may indicate inflammatory processes associated with infection, trauma, and malnutrition (Ribot and Roberts, 1996; Larsen, 1997; Weston, 2008; Weston, 2012; Klaus, 2014b). These are often found on the long bones and the ectocranial surface of the bones of the cranial vault (Ribot and Roberts, 1996). Evidence for periosteal new bone formation includes the formation of woven bone, immature bone laid down on the periosteum suggestive of active infection/inflammation, and lamellar bone, which represents remodelling bone suggestive of healing (see Fig.5.10) (Larsen, 1997; Weston, 2012) (see Figure 5.10). Periosteal new bone formation has been used in recent research to comment on "frailty" in the past. Frailty being an indicator of an individual's increased risk of death when compared to others within the skeletal population, potentially due to a heightened susceptibility to disease (DeWitte, 2010; Klaus, 2014b). The presence of periosteal new bone formation has been linked to earlier age-at-death within particular groups in the past, such as a heightened risk of mortality in men during the Black Death (DeWitte, 2010), and within lower status children within the 18th-19th centuries (DeWitte *et al.,* 2015). While such studies have suggested that it may be beneficial to distinguish between those with active (woven) and healed (lamellar) lesions in terms of frailty and early mortality (DeWitte, 2014), this was beyond the scope of this present study, and may be an avenue for future research.

Figure 5.9 – *Skeletal indicators of non-specific infection; a) Woven bone formation on the left femur and tibia (COL10 Sk237); b) Lamellar bone formation on the right tibia (COL10 Sk127). Photos taken by author.*

5.7.5 Cribra orbitalia

Cribra orbitalia refers to the marrow hypertrophy in the diploe of the cranial vault bones, centres for red blood cell production in childhood and adolescence, and is evident on the orbital plate of the frontal bone (see Fig.5.11) (Walker *et al*., 2009). The classically reported origin of these orbital lesions is the response to iron-deficiency anaemia (Stuart-Macadam, 1991; Lewis, 2002a). Iron deficiency anaemia can result from chronic blood loss, parasitic infection, and malnutrition (Stuart-Macadam, 1991; Lewis, 2002a). However, Walker *et al*. (2009) have contested this aetiology, suggesting that the most likely origin of this condition is through acquired megaloblastic anaemia rather than dietary iron-deficiency. They suggest that megaloblastic anaemia is acquired through a combination of vitamin B12 deficiency and nutrient loss through gastrointestinal infections promoted by unhygienic living environments (Walker *et al*., 2009). This movement away from the involvement of iron deficiency anaemia has been likewise disputed, and it is likely that this condition can arise from a multitude of aetiologies and can reflect on poor health, unhygienic environment, and dietary deficiencies in past populations (Oxenham and Cavill, 2010). Cribra orbitalia was recorded using the five stage scoring system of Stuart-Macadam (1991). This system scores cribra orbitalia according to the following stages – 0= normal bone surface, 1= capillary-like impressions on the bone, 2= scattered fine foramina, 3= large and small isolated formina, 4= foramina linking into a trabecular structure, and 5= outgrowth in trabecular form from the outer table surface (Stuart-Macadam, 1991).

Figure 5.10 – *Example of stage 3/4 cribra orbitalia in the right orbit (COLIO Sk230). Photo taken by author.*

5.7.6 Dental enamel hypoplasia

Dental enamel hypoplasia (DEH) is a defect that arises due to the disturbance of enamel formation in the developing teeth (Ribot and Roberts, 1996; King *et al.,* 2005). Malnutrition and episodes of disease have been identified as major influences on the formation of DEH (Ogden *et al.,* 2007; Hillson, 2008). Children of very low birth weight have also been found to demonstrate a greater prevalence of enamel defects (Corrêa-Faria *et al.,* 2013). Evidence of enamel hypoplasias in past populations has therefore been classically used as an indicator of systemic physiological stress in early life (Goodman and Rose, 1990; King *et al.,* 2005; Ogden *et al.,* 2007; Hillson, 2008). Defects in enamel thickness can manifest as furrows, pits, or steps on the crown surface, and a more recently described occurrence of "cuspal enamel hypoplasia" (CEH) on the crowns of molars (Lewis and Roberts, 1997; Ogden *et al.,* 2007; Hillson, 2008) (see Fig.5.12 for some examples). Due to the inability of enamel to remodel, evidence of DEH serves as a permanent marker of childhood stress, and can thus be assessed within adult individuals (King *et al.,* 2005; Miszkiewicz, 2015). As the dentition begins development *in utero* with the deciduous dentition, and ends with the completion of crown development of the permanent dentition at approximately eight years of age, enamel defects such as these provide an invaluable source of chronological evidence of both early life and childhood stress (Goodman and Rose, 1990). Which teeth are affected depends on the age at which the episode of stress occurs, as timing of crown formation varies between tooth types (Goodman *et al.,* 1980; Smith, 1991; King *et al.,* 2005). This makes the detection of DEH in an individual

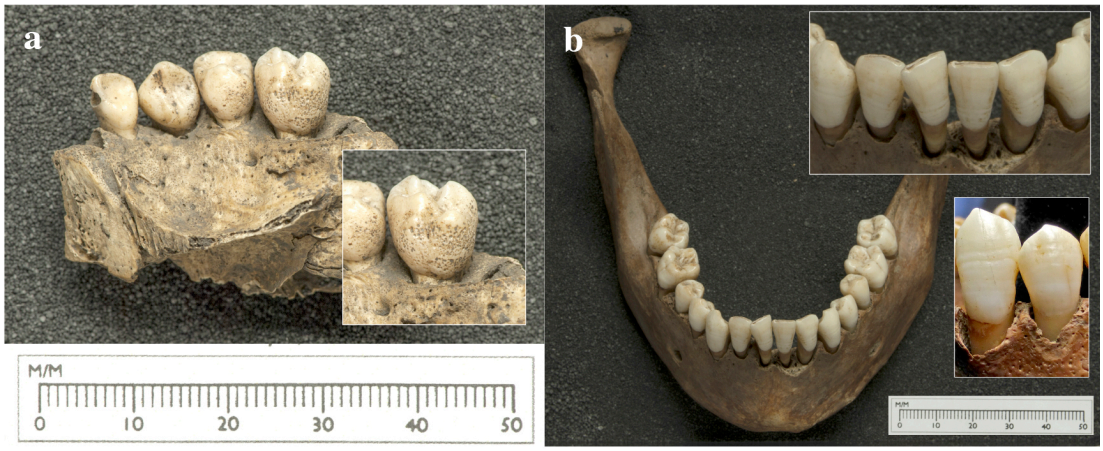

Figure 5.11 – *Examples of a) pitted (COL10 Sk14), and b) linear (COL10 Sk122) dental enamel hypoplasia (DEH). Photos taken by author, with the assistance of Jeff Veitch.*

particularly useful, as it provides an approximate chronological indicator of when they underwent periods of illness or non-specific stress during development (Goodman *et al.*, 1980; King *et al.*, 2005). When an individual demonstrated evidence of either of the above types of defects on any of the teeth, DEH was recorded as present for that individual. From this a crude prevalence rate for DEH was calculated for each site. No attempt was made to record severity or chronology of these defects.

5.7.7 Harris' lines

Whilst recording measurements of CT, each individual was also assessed for the presence/absence of Harris' lines on the femora and tibiae. Harris' lines are observable radiographically as dense transverse lines (Alfonso-Durruty, 2011). They are most commonly found on the metaphyses of the long bones such as the femur and tibia (see Fig.5.13) (Mays, 1985; 1995). These lines are said to depict periods of delayed growth, and have been associated with disease, malnutrition, and psychological stress (Mays, 1995; Alfonso-Durruty, 2011). They are formed when growth arrest occurs in the growth plate, followed by the recovery of normal growth (Mays, 1995). However, there is controversy over the use of Harris' lines, as their appearance does not always show a consistent correlation with stressful events. Correlation with illness is often low, and they have also been observed in the absence of stress (Nowak and Piontek, 2002; Alfonso *et al.*, 2005; Papageorgopoulou *et al.*, 2011; Alfonso-Durruty, 2011). It has been suggested that while Harris' lines may indeed form during episodes of stress due to growth retardation, they may also result from normal periods of growth acceleration, such as that seen in the rapid growth in infancy and the pubertal growth spurt

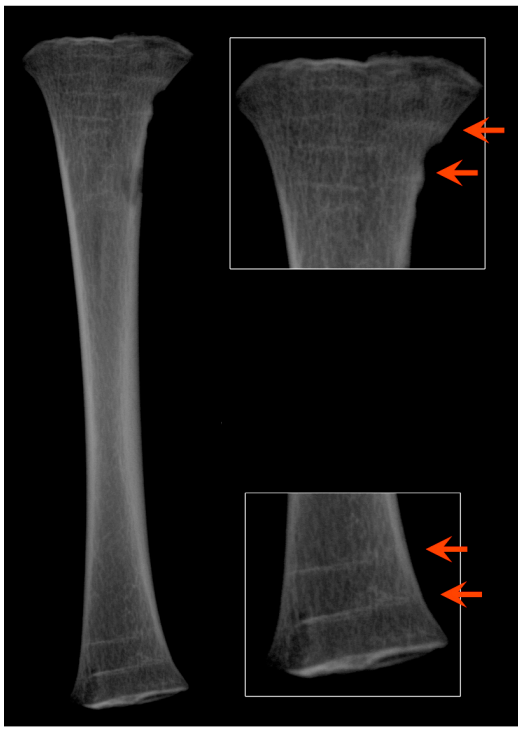

Figure 5.12 – *Example of Harris' lines on the proximal and distal ends of a right tibia (BBP07 Sk4). Radiograph: Gerald Conlogue.*

(Alfonso-Durruty, 2011). However, Geber *et al*. (2014) have argued that Harris' lines were an effective indicator of stress within the famine context in their study of children from the Great Irish Famine (1845-1852). Their study found evidence of intense periods of catch-up growth (as indicated by the presence of Harris' lines) in those suffering from malnutrition.

Due to limitations in terms of access to radiographic equipment, only individuals that had undergone radiographic analysis for measurement of CT could be assessed for the presence of Harris' lines. Lines were scored as present if they crossed at least half of the diameter of the bone (Ribot and Roberts, 1996; McEwan *et al*., 2005).

5.8 Statistical analysis

The results for tibial diaphyseal length, femoral and tibial CT, vertebral body height and TR diameter were statistically assessed via analysis of covariance (ANCOVA) throughout the manuscripts to detect any potential differences in growth between the populations under study. This statistical method allows for the detection of differences between the regression slopes of two datasets, while acknowledging the influence of dental age as a covariate (Pinhasi *et al*., 2006; Field, 2013). Before this statistical test was applied, homogeneity of the regression slopes was first confirmed by plotting the data for the above growth parameters against dental age on a scatter graph (Field, 2013). Accordingly, this method was only applied to individuals between 0-12 years of age to avoid the complications of the sex-differentiated pubertal growth spurt (Lewis *et al*., 2015). Inability to differentiate between the sexes prior to 12 years of age is unlikely to skew results, as it has been stated that sexual dimorphism in CT and long bone length is not evident until shortly after the pubertal growth spurt (Duren *et al*., 2013).

An ANOVA test was also applied to the vertebral groups for the adult measurements of vertebral body height and TR diameter to establish whether any inter-population differences identified in the non-adult samples persisted to the end of the growth period. This also allowed for the assessment of the impact of early life stress on future adult health.

Within Manuscript Four, overall prevalence of pathology was compared between the two sites via a Chi-squared test using a significance level of p=0.001.

Lastly, age-at-death distributions for males and females were compared for each site via a Kolmogorov-Smirnov test in section 6.4.2.

5.9 Intra-observer error

To assess the effects of intra-observer error on the data collected within this study, a small sample of skeletal elements was selected for remeasurement to determine the accuracy of the measurements taken for metric analysis. To test intra-observer error, 10 tibiae were selected for measurement of maximum diaphyseal length, 10 radiographs for measurement of femoral CT, and 10 vertebrae for vertebral body height and transverse diameter. Each measurement was taken twice by the author.

The technical error of measurement (TEM) was calculated using the following formula (Goto and Mascie-Taylor, 2007) –

$$\mathbf{TEM=\sqrt{(\sum D^2/2N)}}$$

(D= the difference between the measurements, and N= the number of bones measured).

Once the TEM was determined, the coefficient of reliability (R) could be then calculated as follows –

$$\mathbf{R=1-(TEM^2/SD^2)}$$

(SD= the standard deviation of all the measurements taken).

The output from this formula lies between the values 0-1, where an output value of 0 = not reliable, and 1 = completely reliable. Accordingly, a high reliability indicates that any variation seen in the data set is unlikely to be caused by measurement error (Goto and Mascie-Taylor, 2007).

5.10 Summary

The data collected, following the above methodology, will be presented within four manuscripts. Each manuscript aims to address differing aspects of the impact of industrialisation on child health, in accordance with the research questions outlined in the introduction of this thesis. Manuscript One is a

methodology paper, through which the efficacy of using non-adult vertebral dimensions as an indicator of stress is tested and discussed. Manuscript Two explores the influence of social status on health during this time, while Manuscript Three focuses on variances on child health in relation to geographical location. Finally, Manuscript Four investigates the relationship between growth disruption and indicators of stress/pathology within skeletal collections. The results of these four manuscripts will then be summarised and discussed as a whole in Chapter Six.

MANUSCRIPTS

MANUSCRIPT ONE - Brief Communication: the use of non-adult vertebral dimensions as indicators of growth disruption and non-specific health stress in skeletal populations

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ABSTRACT

Objective: Traditional methods of detecting growth disruption have focused on deficiencies in the diaphyseal length of the long bones. This study proposes the implementation of vertebral measurements (body height and transverse diameter of the neural canal) from non-adults (0-17 years) as a new methodology for the identification of growth disruption.

Methods: Measurements of vertebral body height and transverse diameter were taken from 96 non-adult skeletons and 40 adult skeletons from two post-medieval sites in England (Bow Baptist, London and Coronation Street, South Shields). Non-adult measurements were plotted against dental age to construct vertebral growth profiles through which inter-population comparisons could be made.

Results: Results demonstrated that both sites experienced some growth retardation in infancy, evident as deficiencies in transverse diameter. However, analysis of vertebral body height revealed different chronologies of growth disruption between the sites, with a later age of attainment of skeletal maturity recorded in the Bow Baptist sample.

Discussion: These vertebral dimensions undergo cessation of growth at different ages, with transverse diameter being 'locked-in' by approximately 1-2 years of age, while vertebral body height may continue to grow into early adulthood. These measurements can therefore provide complementary information regarding the timing of growth disruption within archaeological populations. Non-adult vertebral measurements can increase our osteobiographical understanding of the timings of episodes of health stress, and allow for the analysis of growth when other skeletal elements are fragmentary.

INTRODUCTION

The detrimental impact of chronic nutritional and health stress on the growth of children has long been recognised, and consequently adult stature and non-adult growth are considered to be robust indicators of population health (Eveleth and Tanner, 1990; Saunders and Hoppa, 1993; Larsen, 1997; Lewis, 2007). The use of multiple indicators of stress has been an integral feature within bioarchaeology for many years (e.g. Goodman et al., 1984). However, a recent call to revitalize the way in which we interpret "health" and "stress" suggests that we should seek to implement a more comprehensive approach, and potentially pursue new avenues of research (Klaus, 2014; Temple and Goodman, 2014). While previous growth studies have relied on long bone length, more recently, other skeletal parameters such as cortical thickness, or compact bone geometry and histology, have provided fruitful new avenues for investigation (Mays et al., 2009; Robbins Schug and Goldman, 2014). This study aims to add to this developing corpus by introducing non-adult vertebral dimensions as a new method of detecting growth disruption in past populations.

Research rationale

The importance of the vertebrae in terms of non-adult growth is best demonstrated by the renewed interest in the "anatomical" method of stature reconstruction from skeletal remains. This method does not rely on calculating stature from a single long bone using regression formulae (the 'mathematical method'), but instead produces an estimate based upon the measurement of all of those bones that contribute to height (Raxter et al., 2006; Raxter et al., 2007; Maijanen, 2009; Auerbach and Ruff, 2010; Auerbach, 2011; Vercellotti 2014). This provides a more accurate method of calculating living stature, as it is not biased by differences in bodily proportions (Maijanen, 2009; Auerbach and Ruff, 2010; Vercellotti 2014). As studies of stature are increasingly beginning to incorporate vertebral components, it is also now prudent to consider the growth of these skeletal elements.

The process of vertebral growth

Vertebral growth, while complex due to development around the spinal cord, shares similarities with that of long bone growth, in that both endochondral and intramembranous ossification occurs in order to create the adult form (Brandner, 1970; Reichmann and Lewin, 1971; Bogduk, 2005). Each vertebral component is formed from three primary centres of ossification, the centra (the body) and two halves of the neural arch (Scheuer and Black, 2000). It is the increase in vertebral body height and the development of the neural canal that is of interest to this study.

An increase in vertebral body height is achieved via the process of columnar proliferation, differentiation and mineralization of chondroblasts on the superior and inferior faces of each of the centra (Stevens and Williams, 1999; Wang et al., 2007). As new bone is laid down on these surfaces the vertebral column increases in length, therefore contributing to an increase in sitting height (Fig. 1a). This process displays three stages of growth throughout childhood and adolescence. There is a rapid increase in height from birth to 5 years of age, a period of quiescence between 5-10 years, and finally a pubertal growth spurt between approximately 10.5-13.5 years of age in girls and 12.5-15.5 years in boys (Hefti and McMaster, 1983; Diméglio and Canavese, 2012). The formation of vertebral end plates (subchondral bone plates, not to be confused with the annular rings) at both the superior and inferior surfaces of the vertebral body marks the end of the growth period between 18-25 years of age (Bogduk, 2005).

While longitudinal growth of the vertebral body progresses throughout the growth period of an individual until maturity, the majority of neural arch growth is completed relatively early in postnatal growth. Cartilaginous growth plates that lie between the two halves of the neural arch (the spinous process), and between the neural arches and the centrum (the neuro-central synchondroses), allow for the continued growth of the vertebral foramen around the developing spinal cord. This occurs via intramembranous bone formation and complementary bone resorption on the inner and outer surfaces of the neural canal (Fig. 1b) (Roaf, 1960; Reichmann and Lewin, 1971; Rajwani et al., 2002; Chen et al., 2006). Fusion of the spinous process occurs at approximately 1-2 years of age (Jinkins, 2000; Scheuer and Black, 2000). The completed neural arch then fuses to the vertebral body at approximately

3-5 years of age, completing the neural canal (Jinkins, 2000; Scheuer and Black, 2000). The formation of the anteroposterior (AP) and transverse diameters (TR) of the neural canal is predominantly complete in early childhood, reaching approximately 95% of its final size by 5 years of age (Diméglio, 1993). Consequently evidence of growth disturbance during early postnatal life may become 'locked into' these dimensions (Clark et al., 1986; Clark, 1988; Diméglio, 1993; Larsen, 1997; Watts, 2011; 2013a,b). The value of the AP and TR dimensions of the vertebrae has been previously recognised and implemented in both adult (Clark et al., 1986; Clark, 1988; Watts, 2011; 2013a,b) and non-adult (Watts, 2013b) skeletal collections. However, these measurements, as well as that of vertebral body height, have yet to be explored as a potential method to map the growth of non-adults. Evidence suggests that vertebral growth proceeds in a manner analogous to long bone growth (Bick and Copel, 1950); therefore it is reasonable to expect disruptions to the development of both these elements under conditions of environmental stress. This study will assess the feasibility of the construction of vertebral growth profiles to detect episodes of "stress" within skeletal samples.

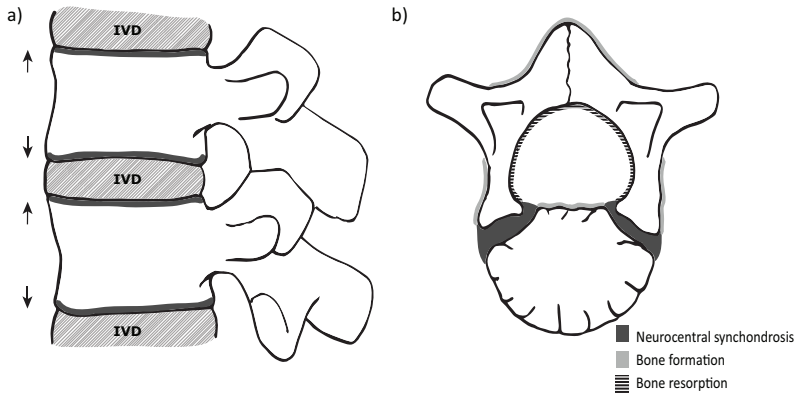

Figure 1 - *The process of vertebral growth. (a) Process of longitudinal vertebral body growth, depicting the growth plates (in dark gray) superior and inferior to the vertebral body, and the direction of growth (arrows). IVD = intervertebral disc. (b) Process of neural arch growth in a developing vertebra, with the neuro-central synchondrosis (dark gray) and areas of bone formation (light gray) and resorption (black lines). (Adapted from Bogduk, 2005; Reichmann and Lewin, 1971).*

MATERIALS AND METHODS

Two skeletal samples were selected for analysis so that the inter-population comparability of the vertebral growth profiles could be assessed. The Bow Baptist

skeletal collection of Payne Road, London represents a relatively prosperous post-medieval population (c. AD 1816-56) of 416 individuals, 202 of whom are classed as 'non-adult' (0-17 years of age). The small village of Bow once existed as a separate entity to London, located on the eastern outskirts of the city. However, the second half of the 19th century saw a rapid industrialisation of this area and its eventual incorporation into the expanding metropolis (Henderson et al., 2013).

The skeletal collection from Coronation Street, South Shields (c. AD 1816-55), consists of 204 individuals, 90 of whom are non-adults. The site is located south of Newcastle-upon-Tyne and during this period was centred on local industries such as shipyards and collieries (Raynor et al., 2011). The individuals buried here are generally regarded as representative of a working-class population.

Individuals between 0-17 years of age were selected from each collection, based on the presence of vertebrae whose position within the column could be reliably determined. Due to the ambiguous nature of vertebral body morphology in those aged less than one year, accurate identification was reliant on the majority of the vertebral elements being present. Any vertebrae that demonstrated signs of pathology (e.g. Schmorl's nodes) were omitted from this study, as were individuals with border shifts (when vertebrae develop features associated with the neighbouring section, e.g. lumbarisation of T12). These were removed as it is yet to be established whether these anomalies would have affected the course of vertebral development (Barnes, 1994; Watts 2011; 2013a,b).

In individuals where the necessary vertebral elements were present (68 in the Bow Baptist sample, and 28 in the Coronation Street sample) dental age was assessed using standards for dental formation of the deciduous and permanent dentition (Smith, 1991). Tooth formation stages were determined by examination of the dentition radiographically, or macroscopically when loose teeth were present (Pinhasi et al., 2005). These stages were used to assign a dental age to each individual, based on the mid-point of the relevant age-category. For example, those between 0.5-1.49 years of age were classed as 1 year of age, those between 1.5-2.45 as 2 years of age, and so on until 17 years of age. This methodology was chosen to ensure comparability of the data with previous growth studies (Mays et al., 2008). Individuals with no dentition preserved were omitted from the study.

Vertebral measurements

Measurements of vertebral body height were taken from the midline of each centra (C3-L5) at the point of maximum height. To achieve this, the inferior surface of the body was positioned horizontally on the sliding calipers and the mobile component moved until it touched the superior surface, thereby marking the point of maximum body height (Fig. 2a). Measurements were taken to the nearest 0.01mm.

To assess vertebral neural canal size (VNC), measurements of TR diameter of the neural canal of cervical, thoracic, and lumbar vertebrae (C1-L5) were taken using sliding calipers (to the nearest 0.01mm). These measurements represented the furthest distance between the medial surfaces of the left and right pedicles (Watts, 2011; 2013a,b) and could only be taken when the neural arches had fused at the spinous process, though fusion of the neural arch to the vertebral body was not essential (Fig. 2b). The AP diameter (normally taken from the posterior surface of the vertebral body to the furthest opposite point of the neural canal, anterior to the spinous process) (Watts, 2011; 2013a,b) was excluded from this study as these measurements could only be taken in individuals that had begun fusion of the neurocentral synchondrosis. Although this measurement is of great value in adult individuals, its use within non-adult samples is restricted to those individuals whose neural arches and vertebral bodies have fused (i.e. older than approximately five years of age). This limitation means that sample sizes for AP diameter tend to be small, as older children and adolescents are often less common in skeletal collections (as can be seen in Table 2). Measurements of body height and TR diameter were also taken from adults aged 18-35 years in both collections to provide comparative data. A sample of 20 adults (10 males and 10 females) was selected from each site to enable comparability with the mixed-sex non-adult

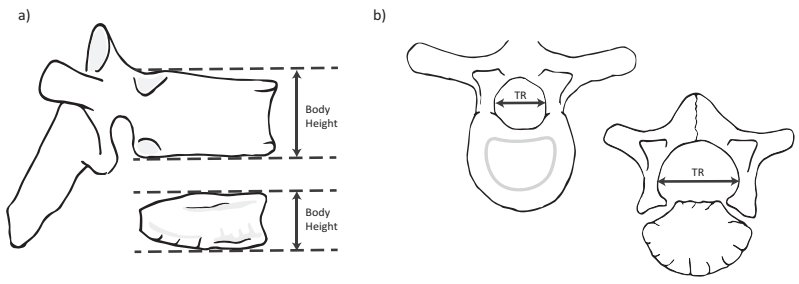

Figure 2 - *Measurements of vertebral growth. (a) Measurement points of vertebral body height in fused and unfused vertebrae. Taken from the midline of the centrum. (b) Measurement location of the transverse diameter of the neural canal in fused and unfused vertebrae.*

samples (see Table 1 for overall sample sizes for each measurement).

Due to the linear increase in vertebral body height with age, it was possible to calculate measurements for missing vertebrae using the average value from the two adjacent vertebrae, when present (Auerbach, 2011). Once measurements had been taken, vertebrae were categorised into groups to maximize sample size (Fig.3). The groups were delimited based on similarities in morphology of vertebrae, and transitional vertebrae (C7, T1, T12, L1, and L5) were considered separately. Averages of the vertebral measurements were taken for each age category and then plotted (C1-L5) for ages 3, 5, 9, and 16 years to allow for a primary assessment of growth in the vertebral column (Fig. 3). These ages were chosen as they had the best representation of vertebral elements present in both samples throughout the growth period. Vertebral groups C5-6, T6-8, L2-4 were chosen for further analysis due to their higher rate of preservation, and therefore larger sample sizes. The overall sample sizes for each vertebral group and measurement for both the non-adult and adults within the two samples can be seen in Table 1, and the breakdown of sample sizes by age category for the non-adult measurements in Table 2.

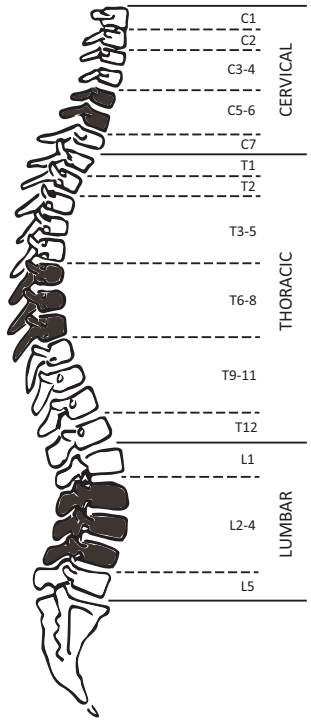

Figure 3 - *Vertebral groupings used for analysis of measurements. Shaded elements reflect the vertebral groups for which data will be presented within this study.*

These grouped measurements were then plotted on scatter graphs against dental age to form vertebral growth profiles for C5-6, T6-8, and L2-4.

The results for body height and TR diameter in these vertebral groups were statistically assessed via analysis of covariance (ANCOVA) to detect any potential differences in vertebral growth between the two samples. ANCOVA allows for the detection of differences between the regression slopes of two datasets, while acknowledging the influence of dental age as a covariate (Pinhasi et al, 2006). This method was only applied to individuals between 0-12 years of age to avoid the complications of the sex-differentiated pubertal growth spurt, and to allow for comparison with the data from a modern sample for TR diameter (see below). An ANOVA test was also applied to the vertebral groups for the adult measurements of

body height and TR diameter to establish whether any inter-population differences identified in the non-adult samples continued through to the end of the growth period.

Table 1 – *Sample sizes and number of individuals from which measurements of body height (BH) and transverse diameter (TR) were taken. Adult sample sizes in brackets.*

	C5-6		T6-8		L2-4	
Sites	BH	TR	BH	TR	BH	TR
Bow Baptist	49 (19)	42 (18)	42 (17)	46 (19)	40 (17)	43 (19)
Coronation Street	20 (11)	10 (15)	20 (11)	10 (13)	19 (10)	10 (14)

Table 2 – *Sample sizes for each non-adult measurement, broken down by age category.*

	Coronation Street						Bow Baptist					
	C5-6		T6-8		L2-4		C5-6		T6-8		L2-4	
Age group	BH	TR	BH	TR	BH	TR	BH	TR	BH	TR	BH	TR
0	9	-	11	-	9	1	2	-	-	-	-	-
1	-	-	-	-	-	-	8	5	7	5	6	4
2	5	4	2	3	2	2	12	9	12	12	11	10
3	1	1	1	1	1	-	2	2	2	4	4	4
4	-	-	-	-	-	-	3	3	3	3	2	3
5	1	1	1	1	1	1	4	4	4	3	3	2
6	1	1	1	1	1	1	2	2	1	2	-	2
7	-	-	-	-	-	-	2	3	3	4	4	4
8	-	-	-	-	-	-	-	-	-	-	-	-
9	1	1	1	1	1	1	1	1	1	1	1	1
10	-	-	-	-	-	-	3	3	2	3	2	3
11	1	1	-	1	1	1	4	4	3	4	3	4
12	-	-	1	1	1	1	1	1	1	1	1	1
13	-	-	-	-	-	-	-	-	-	-	-	-
14	-	-	-	-	-	-	-	-	-	-	-	-
15	-	-	1	-	1	1	3	3	1	2	1	2
16	1	1	1	1	1	1	1	1	1	1	1	2
17	-	-	-	-	-	-	1	1	1	1	1	1
Total	20	10	20	10	19	10	49	42	42	46	40	43

RESULTS

Figure 4 demonstrates the average size of each region in the vertebral column at ages 3, 5, 9, and 16 years, compared to the average adult measurements for each dimension. During infancy and early childhood, body height is similar between the Coronation Street and the Bow Baptist individuals (Figures 4a and 4b).

By 9 years of age differences in growth between the samples emerge, with Coronation Street demonstrating deficiencies in growth of the cervical, lumbar, and upper and lower thoracic vertebrae compared to the Bow Baptist individuals (Fig. 4c). This is most apparent in the lumbar portion of the column.

By 16 years of age the Coronation Street sample have, on average, attained 93% of the final adult body height throughout the vertebral column, while the Bow Baptist sample have only reached an average of 79% (Figs. 4d and 4e). The adult measurements themselves do not differ markedly, with the Bow Baptists

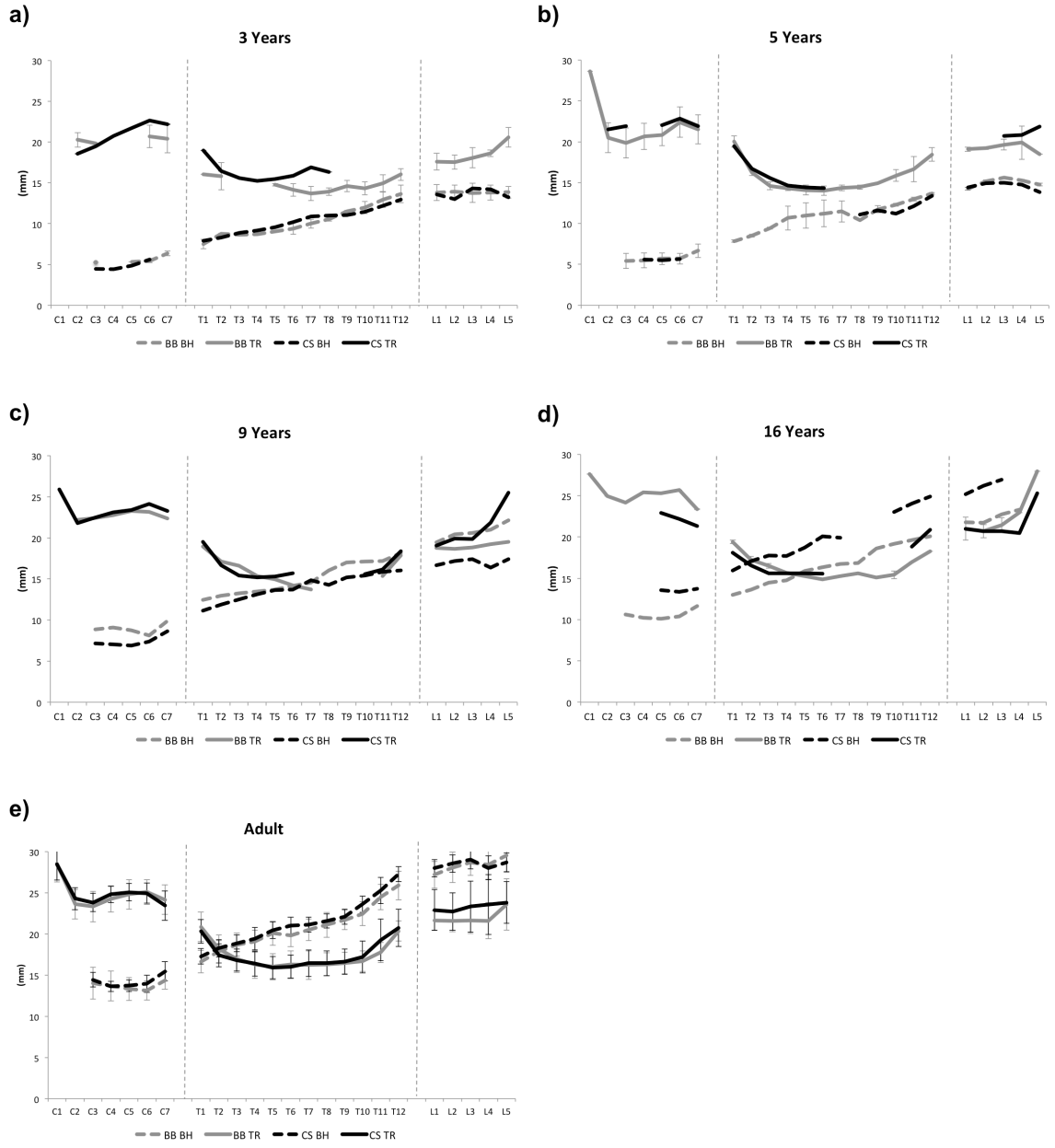

Figure 4 - *Summary graphs for the overall growth in body height and transverse diameter throughout the vertebral column at ages (a) 3, (b) 5, (c) 9, (d) 16, and (e) adult. BB = Bow Baptist; CS = Coronation Street.*

measurements only slightly lower than those from Coronation Street between C2-C4 and from T9-L4 (Fig. 4e).

For the measurements of TR diameter the non-adult and adult values were similar, as expected due to the early age of fusion of this element (Figs. 4a-e). By 3 years of age the non-adults of the Bow Baptist and Coronation Street samples had attained approximately 85% and 91% of the adult TR diameter respectively throughout the column. In contrast to the growth in body height, the TR diameter measurements do not vary greatly either within or between the skeletal samples. Likewise, the adult TR diameter measurements were similar between the two samples (Fig. 4e).

For vertebral groups C5-6, T6-8, and L2-4, measurements of body height and TR diameter were plotted against dental age as scatter graphs to form growth profiles. The growth profiles for body height also include data for the average body heights of the relevant adult vertebrae from each site. Figure 5 demonstrates that these data form a useable growth profile. Modern comparative data on vertebral body height is not currently available. Values for both the Bow Baptist and Coronation Street samples show statistically significant differences in the T6-8 and L2-4 vertebral groups (Table 4), with Coronation Street reaching on average only 42% of the adult sample average at 2 years of age for T6-8, and 60% at 9 years of age for L2-4. This is compared to 47% and 73% in the Bow Baptist population respectively. However, while the Coronation Street sample reaches the target adult proportions in all vertebral sections (Figs. 5a-c), this is only achieved by the Bow Baptist sample in L2-4 (Figs. 5c). The individual of 17 years of age reached only 80% of the average adult body height for C5-6, and approximately 83% for T6-8. Whereas an individual of 16 years of age from Coronation Street achieved values of approximately 98% and 94% of the adult sample average. There were no statistically significant differences between the adult body heights of the Bow Baptist and Coronation Street samples (see Table 3).

Modern TR diameter data are available for comparison to the archaeological samples. The study by Hinck et al. (1966) provides average TR diameter measurements for 353 children up to 18 years of age (data averaged into ages 4, 7, 9, 12, 14, and 16), and also averages for measurements from 121 adults (classed as above 18 years of age). These data were plotted against the archaeological data-sets

Table 3 - *ANOVA results for adult measurements of body height (BH) and transverse diameter (TR) from the Coronation Street and Bow Baptist samples (p=<0.05).*

	BH		TR	
	F	*p*	F	*p*
C5-6	1.093	0.305	0.073	0.789
T6-8	1.029	0.320	0.070	0.793
L2-4	0.003	0.959	2.267	0.142

Table 4 - *ANCOVA results for the Bow Baptist and Coronation Street populations, including comparison of both sites with the modern sample for measurement of transverse diameter(p= <0.05).*

	Body Height		Transverse diameter	
	F	*p*	F	*p*
C5-6	2.938	0.092	0.462	0.501
T6-8	16.189	0.000	3.690	0.061
L2-4	16.746	0.000	0.019	0.892
Modern				
C5-6	-	-	21.945	0.000
T6-8	-	-	9.339	0.000
L2-4	-	-	11.096	0.000

(Figs. 6a-c). Both the modern data for children, and the archaeological non-adult data, appear to have a slight upward trajectory. In the modern data, the trend line for the measurements of TR diameter in non-adults meets that of the adult average value between 15-17 years of age.

The majority of individuals from both archaeological samples fall below modern values, most notably in the cervical and thoracic regions (Figs. 6 a-b). This difference between the modern and archaeological samples was statistically significant for all three vertebral groups (Table 4). By 17 years of age most individuals from the archaeological samples have not reached the expected measurements of TR diameter for a modern day healthy adult. While the Bow Baptist sample showed some of the lowest growth values in all three groups (Figs. 6 a-c), there were no statistically significant differences in TR diameter between the two sites, unlike that seen for body height (Table 4).

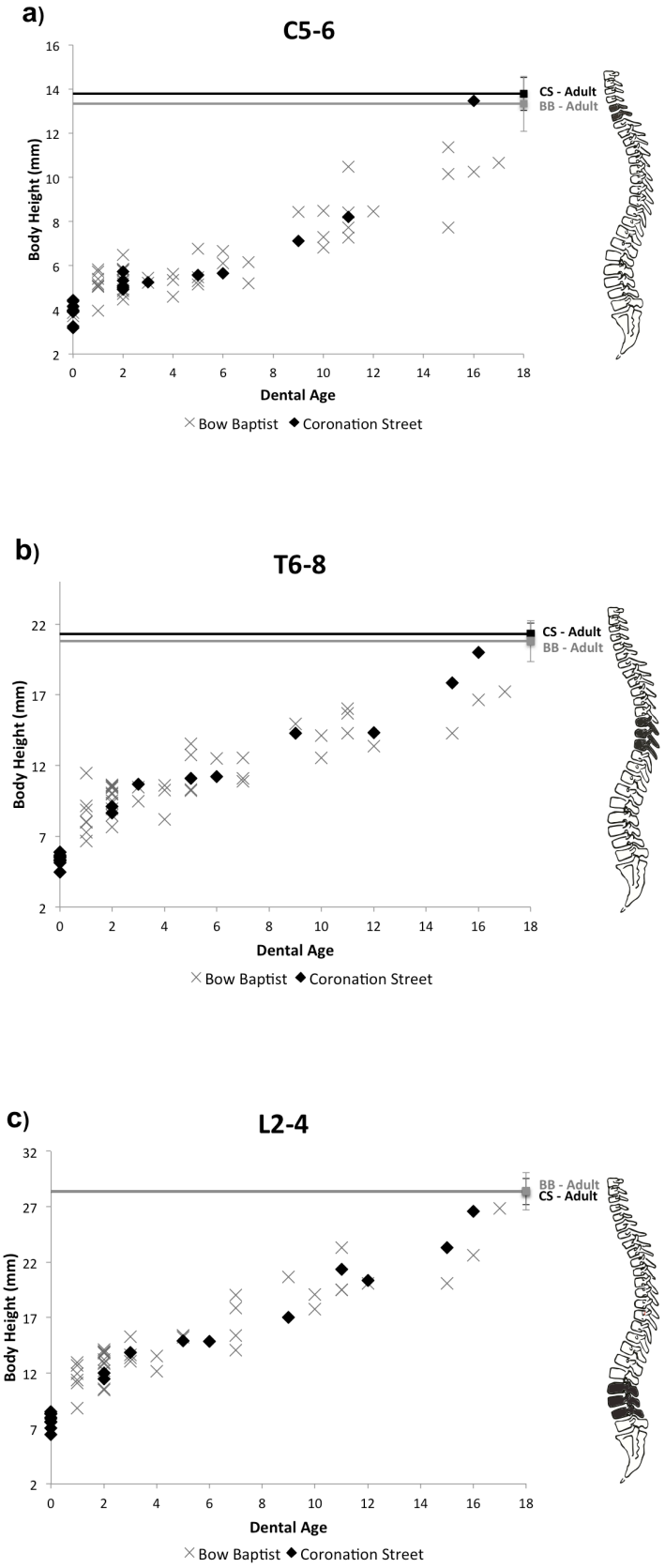

Figure 5 - *Vertebral growth profiles for body height for grouped vertebrae a) C5-6; b) T6-8; c) L2-4. Averages of adult body height for these groups shown as solid lines at the top of each graph.*

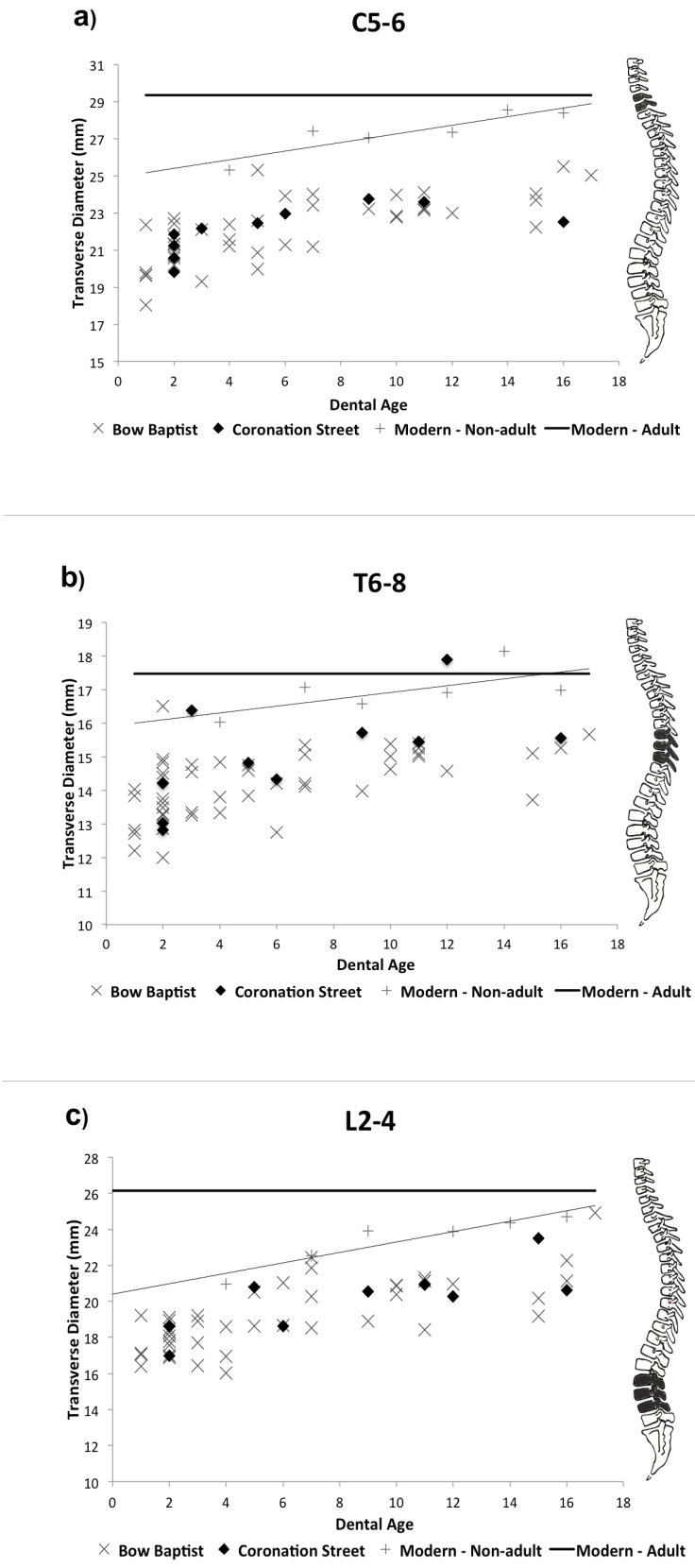

Figure 6 - *Vertebral growth profiles for transverse diameter for grouped vertebrae a) C5-6; b) T6-8; c) L2-4. Comparative modern adult data (solid black line) and modern non-adult data (solid grey line) taken from Hinck et al (1966).*

DISCUSSION

Earlier studies by Clark et al. (1986; 1988) and more recent studies by Watts (2011; 2013a,b) have successfully established the feasibility of detecting early episodes of stress within the AP and TR dimensions of the neural canal. However, techniques that incorporate vertebral dimensions into bioarchaeological research have yet to be fully explored in non-adult skeletons. The most significant outcome of this study, therefore, is that it is possible to construct usable growth profiles from vertebral measurements, akin to those produced using long bone length.

The results of this study confirm an increase in vertebral body height throughout the entirety of the growth period, and an almost complete TR diameter by early childhood. It is of note, however, that there remains a minor increase in TR diameter (presumably due to remodelling of the neural canal) until approximately 15-17 years of age. For example, at 2 years of age the Coronation Street and Bow Baptist samples have reached approximately 81% and 86% of their adult sample values within the vertebral group T6-8, respectively. By 16 years of age these values have both increased to 94%. This is indicative of a continuation of the growth period beyond the point of fusion of the vertebral arch to the vertebral body, contrary to the prior assumptions that this measurement is completely 'locked in' in infancy. While this result suggests a continuation in growth, it is also possible that the older children have larger TR diameters because they did not experience high levels of stress in infancy, thus represent "survivors". Whereas the younger individuals have smaller diameters as they were "non-survivors" and experienced heightened levels of stress in infancy, leading to an early age-at-death. The continuation of growth in the TR diameter of the neural canal has been noted by previous authors (Hinck et al., 1966; Reichmann and Lewin, 1971) and most recently by Watts (2013b). Nevertheless, the potential for catch-up growth in this structure is still limited and the increase in size is relatively small. Therefore, it is still reasonable to assert that measurements of neural canal size are "locked in" in early childhood and consequently deficiencies can represent early life episodes of stress. Growth in the lumbar portion of the vertebral column demonstrates the greatest variability in TR diameter when compared to other regions (Fig. 4e). This requires further analysis to elucidate the cause of such variability.

The Bow Baptist site shows potential growth retardation in body height at 16 years of age compared to the Coronation Street sample, yet still demonstrates similar adult average body height values. Body height within the vertebral column can continue to grow between 18-25 years of age (Bogduk, 2005), therefore this suggests that body height may also undergo catch-up growth and/or extended growth periods into early adulthood, analogous to that seen in long bone growth. This will need to be substantiated by further study due to the small sample size of the adolescent age group (Table 2). However, it does have implications for the study of stature and body proportions in past populations.

Both archaeological samples demonstrated deficiencies in TR diameter, with measurements falling significantly below that of modern "healthy" individuals. Some individuals within each data set are exceptionally underdeveloped, with the Bow Baptist individuals showing some of the lowest TR values. With regards to vertebral body height, comparison between the two archaeological samples reveals that the Bow Baptist non-adults lag in growth between 9-16 years of age, and Coronation Street between 5-9 years of age (appearing to "recover" by 16 years of age when they are nearing/have met the average adult measurement values).

Table 5 - *Crude prevalence rates (CPR) of skeletal indicators of stress in the Bow Baptist and Coronation non-adult samples (Bow Baptist N=202, Coronation Street N=90).*

	Bow Baptist	Coronation Street
DEH (%)	13.4	13.3
Rickets (%)	16.83	4.4
NSI (%)	11.88	4.4
Cribra Orbitalia (%)	13.37	10

Crude prevalence rates (CPR) for other metabolic and non-specific indicators of stress within each site were assessed to further elucidate these chronological patterns of vertebral growth disruption (see Table 5). Both the Bow Baptist and Coronation Street samples show very similar frequencies of dental enamel hypoplasia (DEH) (see Table 5). As tooth enamel of the deciduous and permanent dentition develops from the second trimester up until approximately 10 years of age, these rates reflect non-specific episodes of stress in infancy and childhood (Goodman and Rose, 1990). This evidence corroborates the deficiencies seen in the TR diameters of both archaeological populations. Overall crude prevalence rates for rickets, periosteal new bone formation and cribra orbitalia

within the non-adult sample from Coronation Street were lower than the Bow Baptist sample (Table 5). These indicators of poor health correlate with the greater degree of growth disruption recorded in vertebral body height in the Bow Baptist sample.

The Bow Baptist population were of a higher social status when compared to the Coronation Street population. However, while the latter were regarded as "working class", the wages paid in many of the local industries of South Shields would have provided sufficient means to buy food and shelter (Raynor et al., 2011). While child labour was common, the children of Coronation Street were more likely to have been employed in industries that involved outside work, such as ship-building (Raynor et al., 2011). Dietary differences in the North and South also existed and historical evidence suggests that London diets were particularly poor, with foodstuffs often subjected to adulteration (Horrell and Oxley, 2012). Therefore, the growth deficiencies recorded in both body height and TR within the Bow Baptist sample from London, together with the higher prevalence of skeletal pathologies, may be related to exposure to harsher environmental stressors in London, which affected the middle as well as the poorer classes, during this period of industrialisation.

This study highlights the potential of a combined approach to the measurement of both vertebral body height and TR diameter in non-adults (alongside the use of existing indicators of non-specific stress) to reveal further insight into the growth of individuals within skeletal samples. This combination of parameters allows growth to be analysed during different life course stages, thus providing complementary osteobiographical data. The early childhood "lock in" in TR diameter means that the masking effect of catch-up growth on this parameter is relatively minimal. These data have shown that while both of the archaeological samples faced episodes of stress in infancy and early childhood, the timing of later episodes of growth disruption differed. This suggests different timings of vulnerability throughout the growth period between the two sites, due to differences in environmental risk and/or age-related cultural practices.

The differences between sites in terms of attainment of final vertebral body height highlights the potential for catch-up growth within the vertebral column. Final height is produced from both the appendicular and axial skeleton and

therefore primarily depends on the differential timings of growth acceleration before and after puberty. In the pre-pubertal stage of adolescence, appendicular growth is more rapid than axial growth. In early puberty, while appendicular growth maintains a more constant rate of growth, axial growth undergoes acceleration (Bass et al., 1999; Bradney et al., 2000; Seeman et al., 2000). Through this tempo of growth, the bones of the limbs would be expected to reach their final size at an earlier age than those of the vertebral column (Nyati et al., 2006). Therefore, depending on the age of exposure to stress, the resulting growth disruption may have a differential effect on the developing skeletal elements, with those undergoing fastest growth being most affected (Bass et al., 1999). Shortly before puberty, growth disruption is most likely to be evident in the limbs; therefore the long bones would be expected to show deficiencies in length (Bass et al., 1999; Bradney et al., 2000). Whereas during puberty the axial skeleton is more likely to deviate from its developmental trajectory and vertebral body height is more likely to demonstrate stunted growth (Riggs et al., 1999; Bradney et al., 2000).

This has interesting applications to the study of the growth period and stature in bioarchaeology. The combination of both longitudinal growth and vertebral growth may prove to be fruitful in terms of better understanding episodes of stress during the growth period. However, one current limitation is that the relationship between puberty, growth and epiphyseal fusion is highly differentiated by sex. Girls undergo the onset of puberty, and therefore pubertal growth spurts, approximately two years earlier than males (girls approximately 10-11 years, and boys approximately 11-13 years, peaking at 12 and 14 years respectively) (Tanner, 1989; Diméglio, 2001). The onset of puberty, however, is influenced by environmental conditions, nutritional status, and episodes of disease (Shapland and Lewis, 2013). Historical evidence from 19th century England alludes to the impact of environment (in particular child labour conditions) on pubertal timing, referring to cases of both premature and delayed puberty in young factory workers (Engels, 1971).

Sexual dimorphism has long been recognised as a limitation in past studies of long bone growth. Male vertebral dimensions have been found to be larger than females during both adolescence and adulthood (Taylor and Twomey, 1984; Gilsanz et al., 1994; 1997; Bastir et al., 2014). However, Gilsanz et al. (1994, 1997)

found that while the cross-sectional area of the vertebral bodies was larger in boys than girls, measurements of vertebral body height were comparable at all ages (Gilsanz et al., 1994; 1997). Limited sexual dimorphism in the TR diameters of adult males and females has also been reported and this is an important advantage of studies of vertebral growth (Clark et al., 1986; Watts, 2011). However, advancement in the ability to assess pubertal development provides an additional avenue through which vertebral growth can be assessed in the future (Shapland and Lewis, 2013).

All bioarchaeological studies of non-adult growth must contend with preservation problems and small sample sizes. As can be seen in Table 2, many of the age categories are represented by only one individual, particularly within Coronation Street. Therefore it must be acknowledged that the results of this study are based on a very limited sample. Furthermore, the cross-sectional nature of archaeological data-sets and the use of dental age as a proxy for known age will introduce additional potential sources of bias. Some of these biases can be mitigated with the application of appropriate statistical techniques and the adoption of suitable caution when interpreting findings. Overall, however, the use of vertebral growth parameters has the potential to enrich our knowledge of growth in the past within a variety of social and environmental contexts.

CONCLUSION

This study has sought to introduce and evaluate the potential of vertebral parameters for the bioarchaeological examination of skeletal remains. It has demonstrated that both vertebral body height and the TR diameter of the neural canal have the potential to provide a commentary on growth lag, catch-up growth, and early life experiences in past populations. These data were also corroborated by the prevalence of skeletal pathologies within these two samples, indicating that this method can be used alongside existing indicators of stress. This is significant for providing osteobiographical data concerning the likely age at which stress events occurred. Further application to other bioarchaeological collections is now required in order to increase data-sets and to examine growth in relation to other non-specific skeletal indicators of poor health. The vertebral measurements discussed above provide another means by which we can access the growth profile of past children,

whether in conjunction with other growth parameters, such as long bone length, or as a substitute when long bones are fragmentary.

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MANUSCRIPT TWO - Dedicated followers of fashion? Bioarchaeological perspectives on socio-economic status, inequality, and health in urban children from the Industrial Revolution (18th-19th C), England.

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Abstract – The 18th and 19th centuries in England were characterised by a period of increasing industrialisation and urbanisation. It was also a period of widening social and health inequalities between the rich and the poor. Childhood is well-documented as being a stage in the life course during which the body is particularly sensitive to adverse socio-economic environments. This study therefore aims to examine the relationship between health and wealth through a comprehensive skeletal analysis of a sample of 403 children (0-17yrs), of varying socio-economic status, from four cemetery sites in London (c.1712-1854).

Measurements of long bone diaphyseal length, cortical thickness, vertebral neural canal size, and the prevalence of a range of pathological indicators of health stress were recorded from the Chelsea Old Church (high status), St Benet Sherehog (middle status), Bow Baptist (middle status), and Cross Bones (low status) skeletal collections.

Children from the low status Cross Bones site demonstrated deficient growth values, as expected. However, those from the high status site of Chelsea Old Church also demonstrated poor growth values during infancy. Fashionable child-care practices (e.g. the use of artificial infant feeds and keeping children indoors) may have contributed to poor infant health amongst high status groups. However, differing health risks in the lower status group revealed the existence of substantial health inequality in London at this time.

Introduction

The industrial cities of the 18th and 19th centuries in England were notorious for their unhealthy living conditions. Urban districts increasingly experienced severe overcrowding, poor sanitation and ventilation, and high levels of air pollution (Kay, 1832; Engels, 1950; Hudson, 1992). The most infamous city in terms of such detrimental environmental conditions was London. Known colloquially as 'the big smoke', London both reflected the spirit of the age, considered an "engine of growth" of the industrial revolution, and epitomised the "evils of the urban environment" (O'Brien and Quinault, 1993: 236).

The expanding cities of this period posed significant health risks, particularly for the youngest members of society. Children are vulnerable to adverse environments, due to their under-developed immune system and the physiological demands of growth (Lewis, 2007; Cameron and Bogin, 2012). Under conditions of poor nutrition or infection, resources are diverted away from growth towards those necessary for immediate survival, resulting in growth deficits (Bogin, 1999; Sapolsky, 2004). Historical records from the 18th and 19th centuries attest to the common-place stunting of growth, with children of the poorer classes most affected (Floud et al., 1990; Sharpe, 2012).

London was a highly stratified society during the 18th and 19th century: the rich and the poor lived in relatively close proximity (Booth 1891), but would have experienced markedly different lives. At the upper echelons were the "gentry" and the aristocracy, next came the "middling sort", which included merchants, manufacturers, and skilled craftsmen, who could afford a "comfortable" living for their families (Earle, 1989; Crawford, 2010). At the lowest levels of the social ladder were the labouring families and the truly destitute, who were often forced to seek poor relief in the workhouses (Hudson, 1992; Crawford, 2010). Opportunities for social mobility were limited and children of the poor were likely to remain in poverty throughout their lives, "…born to labour, they lived their lives in a cycle of poverty, which their children in turn inherited" (Crawford, 2010: 5).

This study aims to assess the growth and health of the skeletal remains of non-adults (0-17 years) of differing social statuses from four cemetery sites in London (c.1712-1856) to discern whether poor health was experienced in children throughout all levels of the social strata, or whether the children of middle to higher

classes were buffered through social privilege from the detrimental effects of urban life.

Social inequity is known to have a profound effect on the overall well-being of a population, producing a "social gradient" in health that deteriorates as income lessens (Marmot and Wilkinson, 2006; Wilkinson and Pickett, 2010; Pickett and Wilkinson, 2015). Social inequalities are associated with low birth-weight, higher rates of infant mortality, shorter height, poor health, and lower life expectancies (Marmot and Wilkinson, 2006; Wilkinson and Pickett, 2010; Elgar et al., 2015). Class inequalities and marginalisation of the poorest groups also lead to enhanced psychosocial stress, with increased chronic disease risk (Marmot and Wilkinson, 2006). Cultural practices such as child-care strategies will also have had implications for child health, and these would have been dictated by social status. However, it has been suggested that modern social gradients in health may not have existed prior to the late-19th to early-20th century, with adult mortality rates of the wealthy being as high as that of the lower status population (Razzell and Spence, 2006). A recent study by DeWitte et al. (2015) supports this, and it has been suggested that while adult differentials in health are a more recent phenomenon, social gradients in child health may have developed in the 18th-19th centuries alongside industrialisation (Razzell and Spence, 2006; DeWitte et al., 2015). It is therefore of great interest to determine whether evidence of social inequalities in non-adult health are evident within this study.

Materials and Methods

The four skeletal collections analysed in this study are summarised in Table 1. Data for the sexes were pooled, as there is currently no reliable method of determining sex prior to skeletal maturity (Saunders, 2008). Overall, 403 non-adult

Table 1 – *Summary of sites located in London.*

Site	Date	Status	No. Individuals	No. Non-adults
Chelsea Old Church	1712-1842	High	198	33
St Benet Sherehog	<1853	Middle	230	64
Bow Baptist	1816-1856	Middle	416	202
Cross Bones	1800-1853	Low	148	104

skeletons (0-17 years of age) were assessed for evidence of pathology, and measurements of growth were taken where preservation allowed.

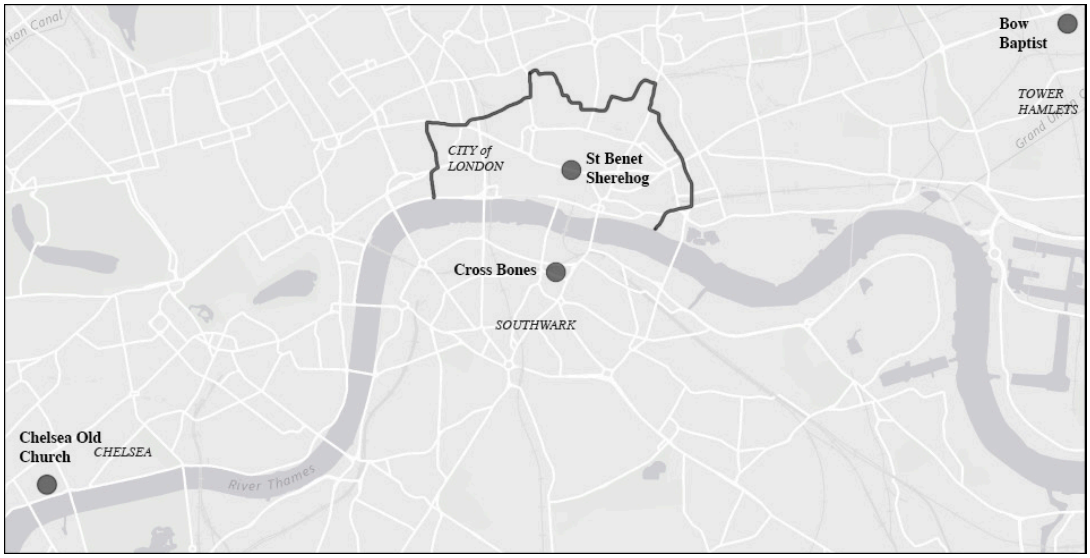

Figure 1 – *Locations of cemeteries within London. Grey line delimits old city boundaries established during Roman occupation, which continued to represent the centre of London throughout the expansion of the city.*

The sample of Chelsea Old Church represents those of a higher status who resided in the suburbs of the city (see Fig. 1). By the mid-18th century Chelsea still retained an almost rural status, and has been described as "a relatively healthy and prosperous place" and "a fashionable resort for Londoners" (Cowie et al., 2008: 13). However, as London continued to grow, by the mid-19th century it had become engulfed by the urban sprawl (Cowie et al., 2008).

The St Benet Sherehog and Bow Baptist skeletal samples represent individuals of the "middling" classes. St Benet Sherehog church was located within the inner city of London (see Fig.1), and this parish has been described as affluent and small (Miles et al., 2008a). Some of the individuals within this collection may date to an earlier period, as this site was in use from the 17th-19th centuries. The Bow Baptist group resided within the small village of Bow on the outskirts of the city at the beginning of 1816 (Henderson et al., 2013). However, the rapid industrialisation of London in the second half of the 19th century quickly subsumed this community into the expanding metropolis (Henderson et al., 2013).

Lastly, the Cross Bones cemetery was an unconsecrated burial ground located in the parish of St Saviour's, Southwark (Fig.1), and was reserved for the very poorest of society (Brickley and Miles, 1999). This skeletal collection

represents those who would have experienced the most squalid levels of sanitation, with areas of the parish being described as "a ruinous and filthy slum" (Brickley and Miles, 1999: 20).

Age assessment

Dental age was assessed using standards for dental calcification (Smith, 1991) of both the deciduous and permanent dentition, for comparability with previous growth studies (Pinhasi et al., 2006). Calcification stages were determined by examination of the dentition radiographically, or macroscopically when loose teeth were present.

Diaphyseal length

Tibial diaphyseal lengths for individuals within each sample were plotted against dental age to assess stunting of diaphyseal length. The tibia was selected because it undergoes rapid growth during development and is considered to be more sensitive than the other long bones to growth disruption (Bogin et al., 2002; Pomeroy et al., 2012). The maximum diaphyseal length of the left tibia (substituted with the right side when necessary) was measured using a standard osteometric board, according to the standards of Buikstra and Ubelaker (1994). Tibial diaphyseal length had previously been recorded for the four collections according to the above standards by the Museum of London (MoL), and was provided by the WORD database (WORD database, 2012a; WORD database, 2012b; WORD database, 2012c), and the Museum of London Archaeology (MoLA) for the Bow Baptist collection. Tibial diaphyseal lengths of "healthy" children aged 0-18 years of age were taken from the study by Maresh (1955), to provide a modern comparative data-set.

Cortical thickness

Appositional growth (growth in the width of long bones) was assessed through measurement of cortical thickness. Cortical thickness (CT) may be a more sensitive indicator of stress than diaphyseal length (Mays et al., 2009). The left femur was selected for CT measurements (substituted with the right side when necessary), to ensure comparability with the previous study by Mays et al. (2009).

Radiographs were taken using a Kubtec Xtend 100HF x-ray source and Kubtec 3600 CR reader at 54 kVp and 5 mAs, with a 120 cm source-image receptor-distance (SID) (Gerald Conlogue, personal communication 20th April 2015). Measurements of the total bone width (T) and the medullary width (M) were taken from the mid-shaft of the femur (Mays et al., 2009). Cortical thickness was determined as T-M, and plotted against the dental age. Modern comparative data for femoral cortical thickness was provided by the study by Virtama and Helëla (1969) of "healthy" Finnish children (0-18 years of age).

Vertebral growth

Vertebral neural canal (VNC) size is an effective indicator of stress in early life (Watts, 2013a,b). This skeletal feature completes the majority of growth during the first two years of age; therefore any disruption to growth occurring prior to this becomes "locked-in" (Scheuer and Black, 2000; Watts, 2013b). Measurements of transverse diameter in children can be used to form vertebral growth profiles, with the mid-thoracic region of the vertebral column demonstrating the least inherent variation (see Newman and Gowland, 2015 for methodology). Measurements of transverse diameter of the neural canal of vertebrae T6-8 were taken using sliding calipers (to the nearest 0.01mm). Averages of the vertebral measurements from T6-8 were calculated for each individual and then plotted against dental age to form growth profiles. The study by Hinck et al. (1966) provided modern comparative data for transverse diameter for both children (0-18 years) and adults (18+ years).

Statistical analysis

Scatterplots were constructed for tibial diaphyseal length, femoral CT, and TR diameter to determine the homogeneity of the regression slopes (Field, 2013). Once it had been confirmed that all assumptions had been met, the data were statistically assessed via analysis of covariance (ANCOVA). This statistical test allows for the comparison of the regression slopes of two datasets, while acknowledging the influence of dental age as a covariate (Pinhasi et al., 2006; Field, 2013). This method was only applied to individuals between 0-12 years of age, due to the influence of the sex-differentiated pubertal growth spurt in adolescence (Lewis et al, 2015).

Pathology

The presence or absence of four pathological categories was determined using data collected from the WORD database (WORD database, 2012a; WORD database, 2012b; WORD database, 2012c; data for the Bow Baptist collection provided by MoLA). The crude prevalence rate (CPR) for rickets, scurvy, periosteal new bone formation, and dental enamel hypoplasia within each site was calculated as a percentage of individuals demonstrating signs of each pathology within the sample.

Classic indicators of rickets in the skeleton include bowing of the long bones, flaring of the metaphyses, and thickening of the diaphyses (Mays et al., 2006; Pinhasi et al., 2006). Other indicators include porosity of the growth plate, new bone formation, flaring of the rib ends, and porosity of the cranial bones (Ortner and Mays, 1998; Brickley and Ives, 2008). The category "rickets" includes all cases indicative of rickets, and possible rickets (i.e. when more subtle manifestations of some of the above characteristics were observed).

Scurvy, resulting from vitamin C deficiency, can be detected skeletally through the presence of new bone formation and porosity resulting from haemorrhaging of the blood vessels in areas where movement frequently occurs; for example, in the orbits, on long bones (in association with the joints), and on the mandible and maxillae (Brickley and Ives, 2008; Armelagos et al., 2014). Other skeletal indicators include new bone formation and porosity on the cranial bones and scapulae (Brickley and Ives, 2006; Armelagos et al., 2014). The category "scurvy" includes all cases indicative of scurvy, and possible scurvy. Diagnosis of scurvy within perinatal individuals is problematic, as normal bone growth and pathological skeletal changes can be easily confused at this age. As the development of scurvy does not usually occur prior to six months of age, this is not a significant issue for the majority of skeletal collections (Crawford, 2010). However, Cross Bones is reported to have a particularly high rate of scurvy within its perinatal sample (Brickley and Miles, 1999). As the diagnostic criteria for scurvy have continued to evolve over recent years, all individuals were re-categorised according to more recent publications (Brickley and Ives, 2008; Armelagos *et al.*, 2014; Stark, 2014; Klaus, 2014a) into the groups "scurvy", "possible scurvy", and "metabolic

disease" based on descriptions of skeletal changes from the WORD database. The "metabolic disease" category includes all individuals demonstrating skeletal changes indicative of "rickets", "possible rickets", "scurvy", "possible scurvy", and those that could not be reliably placed into either of the aforementioned categories. This category ensured that prevalence of metabolic disease within each site could be assessed with due consideration to the issues associated with misdiagnosis of rickets and scurvy, and also their frequent co-morbidity (Stark, 2014).

Periosteal new bone formation refers to areas of bone formation, or porosity on the cranial bones and long bones that cannot be attributed to a specific cause (Weston, 2008; Klaus 2014b). This indicator was recorded as present when evidence of new bone formation (woven and/or lamellar) was identified on the long bones or ectocranial surface of the cranial bones. No attempt was made to assess coverage or severity, and no differentiation between healed/unhealed lesions was made.

Dental enamel hypoplasia (DEH), arising due to the disturbance of enamel formation in the developing teeth (Hillson, 2008), was recorded as present when one or more teeth demonstrated evidence of these defects. Malnutrition and episodes of disease have been identified as major influences in the occurrence of DEH (Ogden et al., 2007; Hillson, 2008).

The overall sample sizes for pathological analysis, diaphyseal length, cortical thickness, and vertebral growth are provided in Table 2.

Table 2 – *Sample sizes for pathological and metric analysis (adult sample size).*

Site	Pathology	Tibial length	Femoral CT	Vertebral BH (T6-8)	Vertebral TR (T6-8)
Chelsea Old Church	33	10	10	10 (9)	10
St Benet Sherehog	64	9	13	16 (11)	8
Bow Baptist	202	70	48	42 (27)	46
Cross Bones	104	36	37	38 (5)	22

Results

Age-at-death

Figure 2 shows the age-at-death distribution of the sample. The peak age-at-death for the Chelsea Old Church, St Benet Sherehog, and Bow Baptist samples is 1-5 years of age (at 39%, 27%, and 43%, respectively). However, for Cross Bones the perinatal age group represents nearly half of the non-adult sample (at 48%)

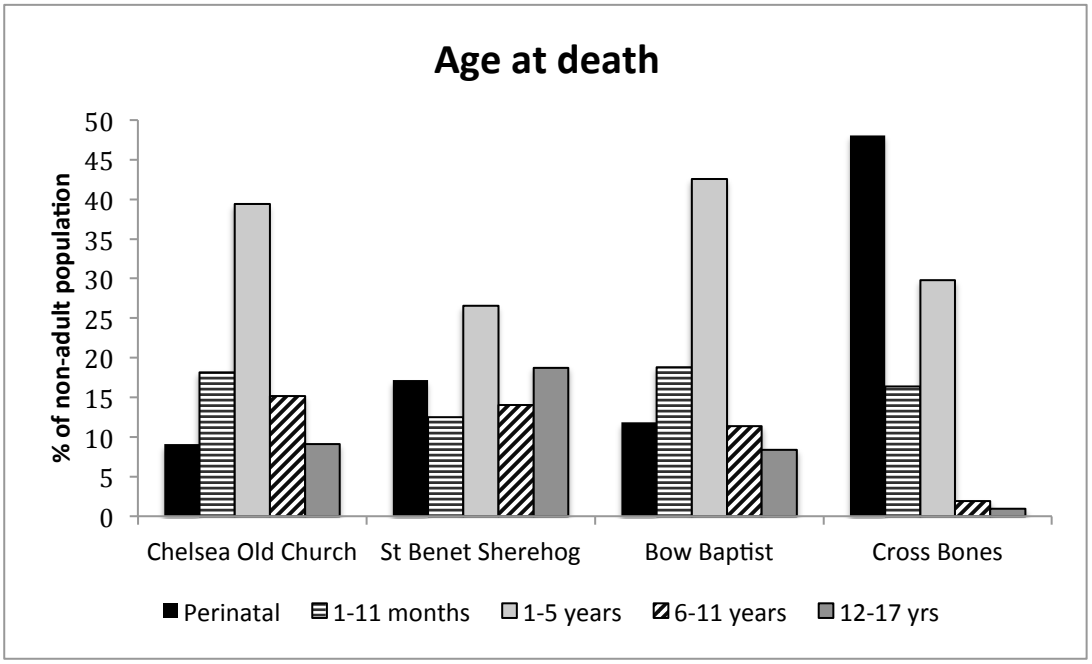

Figure 2 – *Proportion of non-adults within each group falling into the age categories perinatal (approximately 36 weeks in utero to 4 weeks post-partum), 1-11 months, 1-5 years, 6-11 years, and 12-17 years. Percentages for each sample based on the total number of non-adults for each site.*

Diaphyseal length

Tibial diaphyseal lengths were plotted against dental age, with each data point representing one individual (Fig.3a). All sites show growth comparable to the modern data for the first two years of life. However, from approximately 2-5 years of age all of the archaeological groups fall below the modern values. While St Benet Sherehog displays some of the highest values throughout the remainder of the growth period, Chelsea Old Church exhibits some of the lowest up until 10 years of age. Beyond 10 years this sample then appear to "catch-up", however, this may be an artefact of the small sample size of this site. It is of note that by 17 years of age, none of the Chelsea Old Church, St Benet Sherehog, and Bow Baptist samples have reached modern values for tibial diaphyseal length. At 16 years of age the St Benet

Sherehog and Bow Baptist samples have only reached approximately 84% and 70% of the modern values respectively. Unfortunately, the Cross Bones data-set for tibial diaphyseal length does not extend beyond 5 years of age due to preservation. There were no statistically significant differences between the tibial diaphyseal lengths of the four archaeological sites (Table 3). However all archaeological samples were significantly lower than the modern data-set (Table 3).

Table 3 – *ANCOVA results for measurements of tibial diaphyseal length, femoral CT, body height (BH) and transverse diameter (TR) from the four archaeological samples, and compared to modern data. p=<0.05, significant values in bold.*

		Tibial length		**Femoral CT**		**BH**		**TR**	
		F	***p***	**F**	***p***	**F**	***p***	**F**	***p***
Chelsea Old Church vs	St Benet Sherehog	0.106	0.750	0.334	0.571	0.018	0.894	4.444	0.061
	Bow Baptist	0.415	0.522	0.007	0.934	3.414	0.071	10.420	**0.002**
	Cross Bones	0.111	0.741	0.021	0.885	0.159	0.692	3.236	0.083
St Benet Sherehog vs	Chelsea Old Church	0.106	0.750	0.334	0.571	0.018	0.894	4.444	0.061
	Bow Baptist	0.000	0.999	0.527	0.471	4.858	**0.032**	0.455	0.504
	Cross Bones	0.010	0.919	0.470	0.497	0.000	0.985	0.132	0.720
Bow Baptist vs	Chelsea Old Church	0.415	0.522	0.007	0.934	3.414	0.071	10.420	**0.002**
	St Benet Sherehog	0.000	0.999	0.527	0.471	4.858	**0.032**	0.455	0.504
	Cross Bones	3.234	0.075	0.338	0.563	7.768	**0.007**	1.045	0.311
Cross Bones vs	Chelsea Old Church	0.111	0.741	0.021	0.885	0.159	0.692	3.236	0.083
	St Benet Sherehog	0.010	0.919	0.470	0.497	0.000	0.985	0.132	0.720
	Bow Baptist	3.234	0.075	0.338	0.563	7.768	**0.007**	1.045	0.311
Modern vs	Archaeological	5.313	**0.001**	11.957	**0.000**	-	-	6.453	**0.000**

Cortical thickness

Figure 3b shows the femoral measurements for CT plotted against dental age. The majority of individuals from all of the archaeological sites fall below the modern comparative data-set, revealing statistically significant deficiencies (Table 3). This deviation away from the modern data occurs from approximately 1-3 years onwards. Individuals from Chelsea Old Church demonstrate some notable deficiencies, particularly in the first two years of life, and at 10 years of age, before attaining higher values once again by 13 years. St Benet Sherehog and the Bow Baptists attain similar values to that seen in the modern population by 15 years of age, reaching approximately 100% and 94% of the modern data values. Individuals

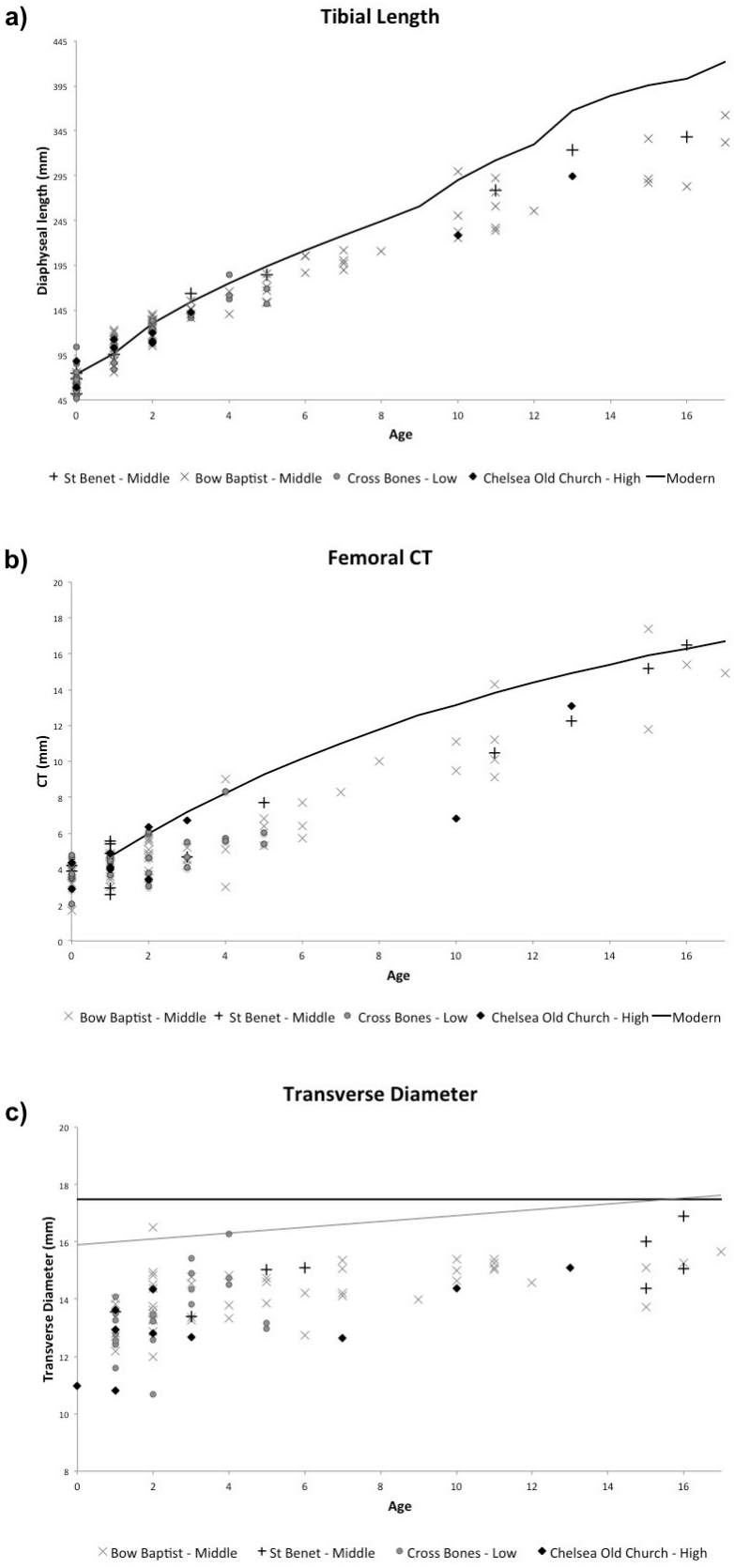

Figure 4 – *Growth profiles for skeletal parameters plotted against dental age: a) Tibial diaphyseal length – comparative modern data represented by solid black line, taken from Maresh (1955); b) Femoral CT – comparative modern data (black line) taken from Virtama and Helelä (1969); c) Transverse diameter for T6-8 – comparative modern adult data (solid black line) and modern non-adult data (solid grey line) taken from Hinck et al. (1966).*

from Cross Bones show a mix of higher and lower values for CT at birth and in infancy, however between 2-5 years of age these remain particularly low. Again no data for CT exists beyond five years of age for Cross Bones. There are no statistically significant differences between the four archaeological groups (Table 3).

Transverse diameter

Measurements of transverse diameter for each individual were plotted against dental age (Fig.3d). All of the archaeological data sets are severely deficient in comparison to the modern data, and this is statistically significant (Table 3). St Benet Sherehog is the closest to reaching modern values. Chelsea Old Church and Cross Bones have the lowest values for transverse diameter in the first two years, averaging approximately 72% and 73% of the modern adult values respectively. Chelsea Old Church continues to demonstrate deficient values for the remainder of the growth period, only achieving approximately 86% of the modern adult values by 13 years of age, and demonstrates significantly lower values than the Bow Baptists (Table 3).

Prevalence of pathology

All four groups have a high rate of rickets, between approximately 12%-17% (Fig. 4). The overall rate of metabolic disease is highest in Cross Bones, at

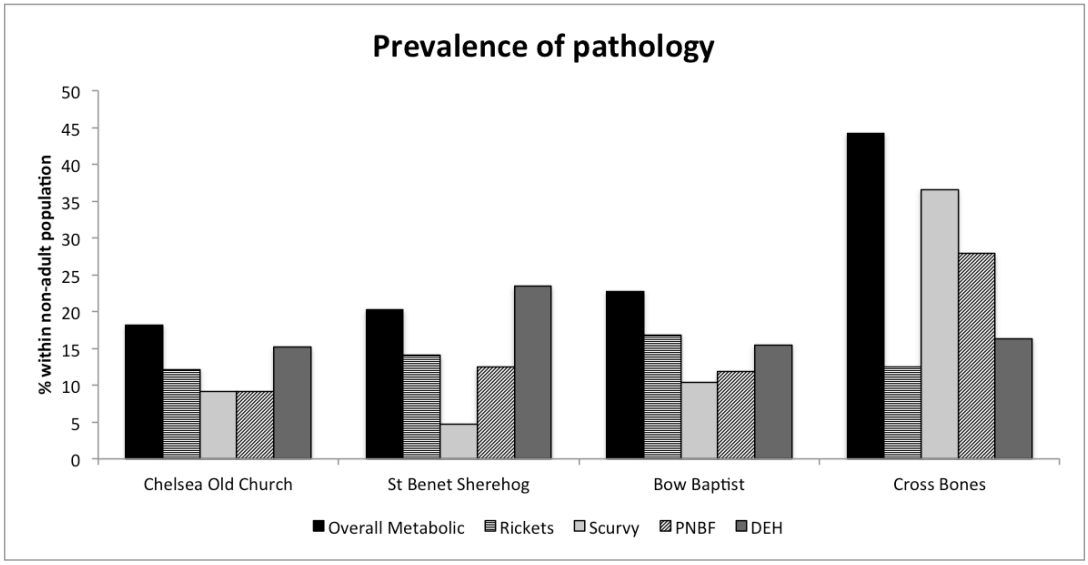

Figure 4 – *Crude prevalence rate (CPR) of pathology seen in each skeletal sample.*

approximately 44% compared to 18-23% at the other sites. This is due to the extremely high rate of scurvy in this group, affecting 37% of the non-adult sample. The rate of scurvy is much lower in the other sites, particularly St Benet Sherehog (at 5%). The prevalence rate of periosteal new bone formation is also much higher within Cross Bones (28%), compared to only 9%, 13%, and 11.88% of non-adult individuals in the Chelsea Old Church, St Benet Sherehog, and Bow Baptist samples respectively. The prevalence of DEH is similar between the Chelsea Old Church, Bow Baptist, and Cross Bones groups (15%, 13%, and 16%). However it is most prevalent in the St Benet Sherehog sample (23%). Overall, while prevalence of pathology is high in all of the archaeological groups, Cross Bones shows the highest rate of skeletal pathologies.

Discussion

Evidence for infant feeding practices

Kay stated in 1832 that "…more than one-half of the offspring of the poor…die before they have completed their fifth year" (p.42-43), and all four sites did show a preponderance of young children aged between 1-5 years. In addition, all four sites showed a high rate of DEH, and statistically significant deficiencies in growth when compared to modern data. This deviation from the modern data occurred in general between 1-5 years of age for tibial diaphyseal length and femoral CT (Figs. 4a and 4b). This is corroborated by the particularly deficient values for measurements of transverse diameter (which completes the majority of its growth by two years of age) in all the skeletal samples (Jinkins, 2000; Scheuer and Black, 2000). From these results, it can be inferred that individuals from each of the archaeological samples here suffered insults to health in infancy, regardless of class.

Overall, the non-adults of St Benet Sherehog showed the highest growth values for tibial diaphyseal length, femoral CT, and transverse diameter. However, this could reflect the more favourable environmental conditions prior to the 18th century, as some individuals may date to the 17th century. Cross Bones and Chelsea Old Church demonstrate some of the lowest values for all growth parameters indicating that individuals from these sites experienced particularly poor health in infancy. While these results were expected for the low status site of Cross Bones, the results for the wealthier population of Chelsea Old Church were surprising.

High rates of metabolic disease were evident in all of the archaeological samples in this study, with all four groups demonstrating similar rates of rickets. The occurrence of metabolic disease peaks between 1-5 years for Chelsea Old Church and St Benet Sherehog, and between 1-11mths and 1-5 years of age for the Bow Baptist sample (Table 4). The conditions of rickets and scurvy were at this time associated with "weanlings" (those aged 6 months-2 years) (Crawford, 2010). Therefore, while metabolic disease occurring in perinates at the Cross Bones sample may be related to maternal health, the later peaks in prevalence within the other sites may relate to an early onset of weaning and/or a deficient weaning diet.

Table 4 – *Breakdown of metabolic disease by age category. Metabolic disease referring to number of individuals within the skeletal sample demonstrating any sign of rickets, possible rickets, possible scurvy, and scurvy. Percentage of non-adult sample in brackets.*

Site	No. Non-adults	Perinatal	1-11mths	1-5 years	6-11 years	12-17 years	Unknown	Overall
Chelsea Old Church	33	0 (0)	0 (0)	3 (9.1)	1 (3)	2 (6.1)	0 (0)	6 (18.2)
St Benet Sherehog	64	2 (3.1)	1 (1.6)	6 (9.4)	1 (1.6)	0 (0)	3 (4.7)	13 (20.3)
Bow Baptist	202	4 (2)	18 (8.9)	21 (10.4)	2 (1)	1 (0.05)	0 (0)	46 (22.8)
Cross Bones	104	24 (23.1)	11 (10.6)	11 (10.6)	0 (0)	0 (0)	0 (0)	46 (44.2)

Weaning ages in the 18th and 19th centuries were often greatly reduced. Breastfeeding fluctuated in popularity, and upper class parents tended not to take a "hands-on" role in the rearing of children, who were instead left in the care of nursemaids (Burnett, 1984; Perkin, 1993). Amongst the higher classes, breastfeeding was viewed as unfashionable or inconvenient (Perkin, 1993; Stevens et al., 2009). Consequently, artificial feeding or wet nurses were popular alternatives (Wickes, 1953; Fildes, 1995; Nitsch et al., 2011). By contrast, in lower class families resources were often stretched due to inconsistent employment, low wages, and increasing family size (Burnett, 1984). Working-class mothers often prioritised allocation of food resources within the family to the "bread-winners"; frequently leading to malnourishment in mothers and younger children (Burnett,

1984; Humphries, 2010; Horrell and Oxley, 2012). Expectant mothers in employment would continue to work up to eighteen hours a day until birth, and the return to work following birth was usually quick (as little as three to four days) to ensure continued employment (Engels, 1950; Perkin, 1993). This often meant accelerating the process of weaning, and complete cessation of breastfeeding from an early age (Wickes, 1953; Fildes, 1995; Nitsch et al., 2011). The high levels of infection and malnutrition amongst the lower classes also meant that many mothers would have been unable to produce nutritionally adequate breast milk (Cheadle, 1889; Fildes, 1995). Conversely, poorer families in which the mother did not work, may have chosen to prolong the breastfeeding period, as it would have been a cheaper alternative to providing food to an additional member of the family (Crawford, 2010; Nitsch et al., 2011). However, beyond six months of age the nutritional constituents of breast milk alone are not sufficient to support the energetic needs of the growing infant (Haggerty and Rutstein, 1999).

The immature immune system of the newborn infant relies on the transmission of maternal antibodies via breast milk for protection from environmental pathogens, and development of their own immunocompetance (Cunningham, 1995; Katzenberg et al., 1996). Clinical studies have observed that infants fed food other than breastmilk suffer from more frequent bouts of acute illnesses (Stuart-Macadam and Dettwyler, 1995; Haggerty and Rutstein, 1999; Ip et al., 2007; Horta and Victoria, 2013). Therefore, it is likely that the status-driven breastfeeding strategies practiced in the 18th and 19th centuries had a significant impact on infant health.

A study by Nitsch et al. (2011) revealed a variety of feeding practices within Christ Church, Spitalfields, London (which was contemporaneous with the sites here) ranging from infants receiving little to no breastmilk, to those breastfed until around 1.5 years of age. Popular artificial infant feeds at this time included “paps” and “panadas” prepared from flour or bread mixed with water, or cow’s milk (Wickes, 1953; Drummond and Wilbraham, 1994; Rendall, 1990). Infants can only absorb around 10% of the iron available in cow’s milk and this, alongside the starchy food, could have caused irritation of the immature digestive tract and resulted in iron-deficiency anaemia (Cheadle, 1889; Stuart Macadam and Dettwyler, 1995; Lewis, 2002). Cow’s milk was also a source of bacterial diseases such as

scarlet fever and tuberculosis (Rendall, 1990; Atkins, 1992; Drummond and Wilbraham, 1994).

Additionally, infants of the higher classes were frequently swaddled: wrapped in strips of material to protect them from cold, and to help their limbs grow straight (Rousseau, 1889; Cadogan, 1748; Buchan, 1778; Levene, 2006). Ironically, this would have greatly reduced exposure to sunlight and may have exacerbated the formation of deformities associated with rickets. However, no associations between swaddling and the development of vitamin D deficiencies have been identified (Urnaa et al., 2006; van Sleuwen et al., 2007). Children of the middling and upper classes were also often kept indoors to protect them from "moral or physical contamination" (Burnett, 1984: 48), and direct sunlight was believed to be bad for babies' eyesight (Drummond and Wilbraham, 1994).

These child-care practices and infant feeding strategies meant that higher-class children would have been highly susceptible to developing deficiencies in vitamin D, especially in infancy. Increased rates of rickets within high-status families have also been noted in previous studies (Miles et al., 2008b; Giuffra et al., 2013). Considering that vitamin D is associated with immune response (Holick, 2003), the high rates of rickets that existed within the wealthier children of London in the 18th and 19th centuries, alongside diminished immunocompetance from the use of breastmilk substitutes, left these infants highly susceptible to the polluted urban environment.

Evidence for social inequalities in health

Exceptionally, the low status group of Cross Bones was dominated by perinates. Cross Bones was an unconsecrated burial ground and it is possible that it was therefore deemed an appropriate place to bury those infants who died before being baptised. The skeletal sample represents only a small proportion of the entire cemetery, which was not completely excavated, and so the predominance may also represent sample bias (Brickley and Miles, 1999). However, burial registers for the parish of St Saviour's, where the cemetery was located, reveal that of the 270 burials in 1845, approximately 45% were those aged 16 years and below, with the majority of deaths occurring between 0-1 years of age (Brickley and Miles, 1999). This high rate of perinatal death likely reflects the harsher environmental

conditions, heightened exposure to early life stressors, and poor maternal health associated with being born into poverty. This corroborates the results by DeWitte et al. (2015), which also identified heightened mortality risks for children of lower status from 18th/19th century London-based skeletal collections.

Overall, Cross Bones demonstrates the highest prevalence of pathology. Peaks in metabolic disease occurred from the perinatal stage, to 1-5 years of age (Table 4). This is suggestive of a range of breastfeeding practices, from dry nursing from birth, to prolonged breastfeeding to aid the family economy (Nitsch et al., 2011). Particularly notable is the high rate of scurvy, occurring primarily in perinates (Table 4). Young infants should not develop scurvy, as vitamin C is provided via maternal diet whilst *in utero*, or the breastmilk following birth (Cheadle, 1889; Brickley and Ives, 2008). In order for perinates to be affected, maternal health must have been severely compromised, demonstrating the inter-generational consequences of poverty on health. This finding is confirmed by the high rates of DEH in the deciduous dentition of Cross Bones (at 14% of the non-adult sample). The deciduous dentition commences development *in utero*, therefore these defects again reflect the health of the mother. Cross Bones likewise demonstrated some of the lowest growth values for tibial diaphyseal length and femoral CT in infancy, and some of the greatest deficiencies in transverse diameter (reflecting disruption in the first two years of life). Poor maternal health may lead to intrauterine growth restriction, resulting in infants that are born small-for-gestational age (SGA) (Hernandez-Beltran et al., 1996; Eveleth and Tanner, 1990). These early growth deficits may lead to permanent stunting, and mothers who were born SGA themselves are much more likely to produce SGA infants (Haeffner et al., 2002; Prentice et al., 2006). Therefore, the effects of poor environment on birth size may span multiple generations (Hernandez-Beltran et al., 1996).

Studies by Barker (1992, 1994; Barker and Osmond, 1986) have revealed the significant impact that infection and nutrition during pregnancy and infancy can have on future adult health, termed the Developmental Origins of Health and Disease (DOHaD) hypothesis. The *in utero* environment "programs" the developing foetus to ensure they are appropriately adapted for the external environment in which they will grow up (Barker, 1992, 1994). Maternal malnutrition limits the nutrition available to the foetus, thus will impact on the development of

physiological and metabolic responses (Barker, 1992,1994; Scott and Duncan, 2000). These changes in physiology increase chronic disease risk in later life, such as coronary heart disease, chronic bronchitis, stomach cancer, and diabetes (Barker and Osmond, 1986; Barker, 1992,1994; Wadsworth and Butterworth, 2006). A recent study by Hughes-Morey (2015) found that, when compared to high status females from Chelsea Old Church, low status females from St Bride's Lower, London, demonstrated a lower mean age-at-death, and significantly shorter femora (Hughes-Morey, 2015). It was suggested that the lower-status females were exposed to more frequent or severe stressors during the growth period than their wealthier counterparts. As shorter stature is associated with a higher risk of chronic disease, and early mortality, it is possible that disadvantages in early life in lower-status groups persisted to adulthood (Hughes-Morey, 2015). Inequalities in health are thus often rooted in early life and can continue to impact on health throughout the life course (Elgar et al., 2015). These initial health disadvantages can be exacerbated by postnatal care strategies due to the limited resources of families living in poverty, highlighting the importance of both prenatal (maternal) environment and postnatal exogenous influences (such as environmental conditions and social factors) on future health and well-being (Aizer and Currie, 2014).

All of the samples in this study were affected by high infant mortality and morbidity, but the lower status Cross Bones sample was most severely affected. While it is true that Chelsea Old Church demonstrated some of the lowest growth values alongside Cross Bones, it must be considered that wealthier families may have been able to provide an overall better quality of care than the lower classes (Hardy, 1992). This means those that enter the mortality sample for this site are more likely to demonstrate severe deficiencies in growth, as those exposed to more moderate stressors were more likely to survive the course. Mothers born and raised in poverty could pass on their health deficits to their children, and this combined with the detrimental external environment and disadvantageous family economy meant that life chances at birth for the children of the poor were limited to a high risk of premature death, or a lifetime of exertion (Gowland and Newman, in press; Gowland 2015).

Conclusion

Life in the city came with some significant health risks to all of its members. While undoubtedly the lower classes experienced a much higher risk of mortality and morbidity, some of these risks were also often felt by the upper classes. Poor infant health was aggravated by child-care practices, heavily influenced by a family's position within society, being dictated either by the desire to follow fashion amongst the upper echelons, or necessity amongst the poor. These promoted conditions such as rickets and scurvy, and led to high infant mortality that spanned the social strata. Amongst the poor, the inter-generational consequences of poverty were apparent in the high rates of perinatal death, but also in the intrauterine onset of deficiency diseases.

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MANUSCRIPT THREE - North and South: a comprehensive analysis of non-adult growth in the Industrial Revolution (18th-19th C), England.

To be submitted to the American Journal of Physical Anthropology.

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ABSTRACT

Objective: Health inequalities currently exist between the North and South of England, with people in the South, overall, experiencing better health across a range of parameters (e.g. life expectancy). Bioarchaeological studies of skeletal remains from cemeteries across this geographical divide have the ability to provide a temporal perspective on the aetiology, longevity and nature of this divide.

Methods: Children are most sensitive to the effects of environmental stress; therefore measurements of long bone length, cortical thickness, and vertebral dimensions were used alongside palaeopathological data to assess patterns in health stress between skeletal populations. In total 575 non-adults (0-17 yrs) from six urban sites (c. AD 1711-1856) were selected from the North and South of England.

Results: There were no significant differences in growth parameters between the six sites in relation to geographical location. However, the northern-based sample Coach Lane (North Shields) demonstrated some of the highest rates of pathology, with metabolic disease being particularly prevalent.

Discussion: Northern and southern populations suffered alike from the detrimental environmental conditions associated with urban centres of the 18th-19th centuries. However, the elevated prevalence of rickets seen within the Coach Lane sample is indicative of a regional specific risk of vitamin D deficiency that may be related to latitude, and/or the influence of differing industries in operation in the North-East.

INTRODUCTION

In the UK today a marked health inequality exists between the North and the South in relation to a number of physiological parameters (Hacking et al., 2011; Whitehead, 2014). For example, a study into healthy life expectancy (HLE) at birth between 2010-12 revealed a clear disparity between northern and southern regions, with the highest HLEs occurring in the South-East (at 65.8 and 67.1 years for males and females, respectively), and the lowest in the North-East (at 59.5 and 60.1 years) (ONS, 2014). Public Health England also highlighted the profound health inequalities experienced by those living in the North-East, citing the historical growth and decline of industry in the North as one of a multitude of causative factors for this inequity (Whitehead, 2014). While distinct "northern" and "southern" identities have existed within England throughout history, these have been fluid over time, mask sub-regional identities, and did not necessarily amount to presence of inequalities per se (Campbell, 2004). However, during the 18th and 19th centuries, different regions came to be associated with specific types of industry (e.g., the North-East with coal and shipping industries, the North-West with cotton mills, and London with manufactories) (Hudson, 1992). Therefore, it is possible that northern and southern regions may have experienced differentially expressed health inequalities aligned to occupation and class in the 18th-19th centuries. A generalised geographical approach to health obviously has the potential to mask intra-regional heterogeneity that relates to other social factors such as ethnicity, class, gender and religion (Whitehead 2014). For example, while longer life expectancies were identified within the wealthier areas of London, some of the shortest life expectancies also occurred within more deprived areas of the city (ONS, 2014).

Previous studies have explored the impact of social inequality on population health in the 18th-19th centuries, revealing heightened health risks for those living in poverty (DeWitte et al., 2015; Hughes-Morey, 2015; Newman and Gowland, accepted). However, this paper aims to explore whether health inequalities are also identifiable between contemporaneous skeletal samples from across the North/South geographical divide. Children are regarded as particularly sensitive to adverse environmental stressors such as poor diet, exposure to disease, climatic change, and fluctuations in economic circumstance (Bogin, 1999; Lewis, 2007). This study aims to analyse a variety of parameters relating to the growth period

(tibial diaphyseal length, femoral cortical thickness, and vertebral dimensions), including palaepathological data from non-adults (0-17 years) within skeletal collections from the Tyneside region in the North-East, and London in the South-East.

The "North" and "South" in context

Industrialisation in Britain was a major economic transition during the 18th to 19th centuries. The seismic shift from a rural, domestic, workforce to one that was primarily urban and factory/mining-oriented resulted in increased population density, inadequate housing and sanitation, air pollution, usually poor work conditions, and long working hours (Kay, 1832; Engels, 1950; Report of the Commissioners, 1845a; Hudson, 1992; O'Brien and Quinault, 1993; Gowland and Newman, in press). London in particular became a nucleus for industry, attracting increasing numbers of people into the expanding city in search of new employment opportunities. For example, approximately 330,000 migrants moved to London between 1841-1851 (Porter, 1994). In 1800 the total population in London was around a million, 2.5 million in 1851, and 4.5 million in 1881 (Porter, 1994; Floud and Harris, 1997). The detrimental living conditions of the industrial environment inevitably had an impact on health, and was said to be "peculiarly severe on infant life" in large towns and populous districts within the UK (Report of the Commissioners, 1845a: p5). This is evident in the high infant mortality rates seen in London at this time (Pinchbeck and Hewitt, 1969).

As the developing industries of the 18th and 19th centuries became increasingly reliant on steam power, the supply of coal from the northern regions became a vital commodity (Langton, 2004; Butler, 2012). This led to a rapid increase in population size within Newcastle ("Britain's metropolis of the North") and Northumberland in the early 19th century (Butler, 2012: 14). Consequently, this region suffered from similar issues as London, in both instances brought about by rapid development and increased population density. Crowded and insanitary living conditions inevitably led to a high incidence of infectious disease and respiratory conditions, and by the middle of the 19th century Newcastle had "the third lowest levels of life-expectancy at birth of all provincial cities in England", after Manchester and Liverpool (Barke, 2001; Butler, 2012: 61). These adverse

environmental conditions were also experienced by the towns surrounding Newcastle, with Gateshead, North Shields, and South Shields described as "destitute of effective arrangements for drainage, sewerage, or cleansing" contributing to "the production of fever and other diseases" (Report of the Commissioners, 1845b: 20). These industrial centers were also clustered within a valley, which meant that the atmospheric pollution was not easily dissipated.

"Dense black smoke from manufactories prevails to a great extent in Newcastle-on-Tyne, at Gateshead, and at North and South Shields. These towns suffer not only from the emanations in their immediate vicinity, but also from those which they convey to each other through the valley of the Tyne, and from the numerous steam-boats…upon the river." (Report of the Commissioners, 1845b: 44)

This severe air pollution, combined with the reduced hours of sunlight between October to April (due to the northern latitude), may have put inhabitants of the industrial North at a heightened risk of developing deficiencies in vitamin D (Pearce and Cheetham, 2010; Macdonald et al., 2011). An investigation by the British Medical Association in 1889 revealed a high prevalence of rickets in coal-mining and industrial districts, and in areas associated with high population density (Owen, 1889). Notably, the North-East demonstrated a very high prevalence of rickets, suggesting a vulnerability to vitamin D deficiency in this region. However, rickets was also reported to be commonplace in London. It is possible that in both London and the North-East child labour, and industry more generally, were similarly contributing factors. However, historical records do not provide sufficient information regarding this and rickets is considered an "invisible disease" in that it will rarely appear as a "cause of death" on bills of mortality. It is therefore the skeletal record that can add clarity to this largely anecdotal reporting regarding this condition in the 19th century.

Historical accounts report that in urban, industrialised centres during this period, not only was infectious disease rife, but diet was also frequently deficient (Gaskell, 1833; Drummond and Wilbraham, 1994; Horrell and Oxley, 2012). It has been suggested that northern diets were nutritionally superior to those in London (Shammas, 1984; Horrell and Oxley, 2012). Those residing in northern counties were more likely to bake their own bread, due to the cheaper price of coal in the North, and potatoes, oatmeal, and milk were more widely available (Shammas,

1984; Snow, 2003). Whereas in the South, less nutritious white bread from bakers was more heavily relied upon, which was commonly adulterated with alum (Snow, 2003). Readily available cheap tea and sugar were more popular, but these lack nutritional quality (Shammas, 1984; Horrell and Oxley, 2012). Therefore, while food variety in London may have been better than other regions due to the importation of goods from overseas, quantity did not necessarily ensure quality, and the poor were likely to have had restricted access to imported food (Sharpe, 2012). The effects of these differences in diets may be responsible for the difference in heights of male military recruits in 1815 with those born in London shorter on average compared to those from urban counties in the North and midlands (Horrell and Oxley, 2012; Floud et al., 1990).

Much has been written from a social and economic historical perspective on the impact of industrialisation on growth in children during the post-medieval period, from a wide range of geographical and social contexts (Floud, 1990; Nicholas and Steckel, 1991; Voth and Leunig, 1996; Oxley, 2003; Humphries and Leunig, 2009; Sharpe, 2012; Kirby, 2013). However, a bias currently exists in bioarchaeological studies of urban health in 18th and 19th centuries England, with most publications focusing on London-based sites (for some examples, see Lewis, 2002a,b; King et al., 2005; Pinhasi et al., 2006; Henderson et al., 2014; Ives and Brickley, 2014, Hassett, 2015). Studies by Mays et al. (Mays et al., 2008; Mays et al., 2009a,b) and Brickley et al. (2007) have indicated that similar trends in urban health also existed within Birmingham at this time. This study adds to the growing corpus of bioarchaeological data on growth and health stress in non-adults from the North and South of England, and aims to examine whether any geographic inequalities in health existed during the 18th-19th century England, or whether industrialisation was universally detrimental.

MATERIALS

The six skeletal collections analysed in this study are summarised in Table 1. Overall, 575 non-adult skeletons were assessed for evidence of pathological indicators of stress (rickets, scurvy, periosteal new bone formation, cribra orbitalia, and dental enamel hypoplasia), and measurements relating to four growth

parameters (tibial diaphyseal length, femoral cortical thickness, vertebral body height, and vertebral transverse diameter) were taken when preservation allowed.

Table 1 – *Summary of sites.*

	Site	Date	Status	No. Individuals	No. Non-adults
North	Coach Lane, N. Shields	1711-1857	Low/ Middle	236	82
	Coronation Street, S. Shields	1816-1855	Low	204	90
South (London)	Chelsea Old Church	1712-1842	High	198	33
	St Benet Sherehog	<1853	Middle	230	64
	Bow Baptist	1816-1856	Middle	416	202
	Cross Bones	1800-1853	Low	148	104

Figure 1 – *Map of England showing location of sites within the north-east, and London.*

Two skeletal collections were identified from the North of England (Fig. 1). Coach Lane was a former Society of Friends burial ground (c.1711-1857 AD) located in North Shields, a town east of Newcastle-upon-Tyne. North Shields in the early 19th century was a rapidly expanding shipping, fishing, and coal-mining community (Gould and Chappel, 2000; Pre-construct Archaeology, 2012). Due to simple Quaker burial practices, the establishment of social status within this group is problematic (PCA-North, 2011; Pre-construct Archaeology Ltd, 2012). While there were plenty of prosperous Quakers within the region it is likely that these individuals represent a mix of low and middle status (Watson, 1864; Boyce, 1889; Grybowska, 2011).

The skeletal collection of St Hilda's churchyard (known as Coronation Street hereafter) is from South Shields (c. AD 1816–1855), located south of the River Tyne. Local industries were similar to that seen in North Shields, centered on shipyards and collieries (Raynor et al., 2011). The individuals of Coronation Street are regarded as being representative of a working-class population.

Four skeletal collections were identified from London (Fig.1). Chelsea Old Church (c. AD 1712-1842) represents those of a higher status who resided in the suburbs of London. By the mid-18th century Chelsea still retained a relatively rural status (Cowie et al., 2008). However, as London continued to grow it did become more urbanised by the mid-19th century (Cowie et al., 2008).

The St Benet Sherehog (c. AD <1853) sample represents relatively prosperous individuals of the middling classes. St Benet Sherehog church was located within the inner city of London (see Fig.1), and this parish has been described as affluent and small (Miles et al., 2008). This sample includes individuals from the 17th-19th century; therefore some individuals may belong to a slightly earlier period than the other samples. The Bow Baptist skeletal collection (c. AD 1816-1856), also of middle class status, resided within the small village of Bow on the outskirts of the city at the beginning of 1816 (Henderson et al., 2013). However, in the second half of the 19th century this community were eventually incorporated into the rapidly expanding city (Henderson et al., 2013).

Lastly, the Cross Bones burial ground (c. AD 1800-1853) is renowned for the pauper status of those interred there. This unconsecrated burial ground was located in the parish of St Saviour's, Southwark (Fig.1), and was reserved for the

very poorest of society, including those who received pauper burials (Brickley and Miles, 1999).

METHODS

Assessment of dental age

Dental age was assessed from dental development using standards for calcification of both the deciduous and permanent dentition (Moorrees et al., 1963a,b; Smith, 1991). These standards were selected for comparability with previous growth studies (Pinhasi et al., 2006). Calcification stages were determined by examination of the dentition radiographically, or macroscopically when loose teeth were present. These stages were used to assign a dental age to each individual, based on the mid-point of the age category that they fell into. For example, those between 0.5-1.49 years of age were classed as 1 year of age (Mays et al., 2008).

Measurement of diaphyseal length

Growth profiles were constructed by plotting measurements of tibial diaphyseal length for each individual against dental age. The tibia was selected for this measurement as it undergoes rapid growth during development, so is thought to be more sensitive to growth disruption (Holliday and Ruff, 2001; Bogin et al., 2002; Pomeroy et al., 2012). The maximum diaphyseal length was measured for the left tibia using a standard osteometric board to 0.01mm (Buikstra and Ubelaker, 1994). When the left tibia was absent it was substituted with the right (Ives and Brickley, 2004). Long bones that had undergone post-mortem damage were omitted from the study. For the London-based skeletal collections, tibial diaphyseal length had previously been recorded according to the above standards for measurement; therefore these data were taken from the WORD database (WORD database, 2012a; WORD database, 2012b; WORD database, 2012c). The Museum of London Archaeology (MoLA) provided data for the Bow Baptist collection. Tibial diaphyseal lengths were compared to existing modern comparative data for individuals 0-18 years of age from the study by Maresh (1955).

Measurement of cortical thickness (CT)

To measure CT, the left femur was selected for radiographic analysis (substituted with the right side when necessary) following Mays et al. (2009a). Antero-posterior radiographs were taken at between 65-80kVp and 4-6mAs from the London based samples using a Kubtec Xtend 100HF x-ray source and Kubtec 3600 CR reader (by Professor Gerald Conlogue, personal communication, 20th April 2015), from the Coronation Street sample using a NOMAD Pro handheld x-ray system (with the assistance of Dr Diana Swales), and from the Coach Lane sample using a Portable GE Medical MPX X-ray unit and Kodak point of care CR System. Measurements of the total bone width (T) and the medullary width (M) were taken from the mid-shaft of the tibia (Mays et al., 2009a). Cortical thickness was determined as T-M, and each measurement was plotted against the dental age to form a CT growth profile for the six samples. Femoral CT measurements were compared to existing modern comparative data for individuals 0-18 years of age from the study by Virtama and Helëla (1969).

Vertebral measurements

The mid-thoracic region of the vertebral column demonstrates the least inherent variation for transverse diameter (see Newman and Gowland, 2015). Therefore measurements of vertebral body height were taken using sliding calipers from the midline of the centra of T6-8 at the point of maximum height (see Newman and Gowland, 2015 for methodology). Measurements were taken to the nearest 0.01mm. Measurements of body height were also taken from adults aged 18-35 years from each sample to provide comparative data. Data was pooled for the sexes, as there is currently no reliable method of determining sex in non-adults (Saunders, 2008).

To assess vertebral neural canal size (VNC), measurements of transverse (TR) diameter of the neural canal of T6-8 were taken using sliding calipers (to the nearest 0.01mm). These measurements represented the furthest distance between the medial surfaces of the left and right pedicles (Watts, 2011; 2013a,b) and could only be taken when the neural arches had fused at the spinous process, though fusion of the neural arch to the vertebral body was not essential.

Averages of the vertebral measurements from T6-8 were calculated for each individual and then plotted against dental age to form growth profiles. While there is unfortunately no modern comparative data set for vertebral body height, a study by Hinck *et al* (1966) provides average transverse diameter measurements for 353 children up to 18 years of age (data averaged into ages 4, 7, 9, 12, 14, and 16), and also averages for measurements from 121 adults (aged 18 years and above).

Recording of pathological indicators of stress

All non-adult individuals were assessed for the presence/absence of five pathological conditions. This assessment was either carried out by the author, or taken from the WORD database (WORD database, 2012a; WORD database, 2012b; WORD database, 2012c). Pathological data pertaining to the Bow Baptist collection was provided by Museum of London Archaeology (MoLA). Each individual was assessed according to the criteria outlined below for skeletal indicators of rickets, scurvy, periosteal new bone formation, cribra orbitalia, and dental enamel hypoplasia.

Rickets was recorded as present when an individual demonstrated several of the following diagnostic criteria: bowing of the long bones, flaring/swelling of the metaphyses, cupping deformities of the metaphyses, coxa vara, and flaring of the sternal rib ends (Mays et al., 2006; Pinhasi et al., 2006; Brickley and Ives, 2008). Other indicators included porosity of the growth plate, new bone formation, porosity of the cranial bones, and thickening of the diaphyses (Ortner and Mays, 1998; Mays et al., 2006; Pinhasi et al., 2006; Brickley and Ives, 2008). The category "rickets" includes all cases indicative of rickets. Cases of both active and healed rickets were considered together in this study, as a previous study by Mays et al. (2009b) suggested that the any effects of rickets on growth between the two forms was not statistically significant.

Scurvy was recorded as present when an individual demonstrated a multitude of skeletal changes such as porosity/new bone formation on the sphenoid, maxillae, mandible, orbits, and on the infra- and supra-spinous regions of the scapulae (Ortner and Erickson, 1997; Brickley and Ives, 2006; Brickley and Ives, 2008; Armelagos et al., 2014; Stark, 2014). Other indicators included new bone formation on the long bones, porosity/new bone formation on the bones of the

cranial vault, and flaring/swelling of the rib ends (Ortner and Erickson, 1997; Brickley and Ives, 2006; Armelagos et al., 2014). The category "scurvy" includes all cases indicative of scurvy.

Scurvy and rickets have many skeletal changes in common, and also frequently co-occur, which can lead to issues regarding their identification (Ortner and Mays, 1998; Stark, 2014). To account for any potential misidentification of these two conditions, individuals with rickets and scurvy were also considered together under a separate category "metabolic disease", including those individuals classed as having "possible rickets" and "possible scurvy".

Periosteal new bone formation refers to areas of bone formation or porosity on the skeleton that cannot be attributed to a specific cause, but may indicate inflammatory processes associated with infection, trauma, or metabolic disease (Ribot and Roberts, 1996; Weston, 2008; Klaus, 2014). It was recorded as present when evidence of periosteal reaction on the cranial bones and long bones was identified (excluding endocranial lesions). Studies have shown that it is beneficial to distinguish between those with active (woven) and healed (lamellar) lesions (DeWitte, 2014), however, this was beyond the scope of this present study. Cribra orbitalia, referring to the marrow hypertrophy seen in the orbits, was recorded using the five stage scoring system of Stuart-Macadam (1991). Lastly, dental enamel hypoplasia (DEH) was recorded as present when an individual demonstrated pit or furrow defects on one or more of the anterior teeth, or on at least one molar.

The overall sample sizes for pathological analysis, diaphyseal length, cortical thickness, and vertebral growth are presented in Table 2.

Statistical analysis

Scatterplots were constructed for tibial diaphyseal length, femoral CT, vertebral body height and TR diameter to determine the homogeneity of the regression slopes (Field, 2013). Once it had been confirmed that all assumptions had been met, the data were statistically assessed via analysis of covariance (ANCOVA) to detect any potential differences in growth between the six samples. ANCOVA allows for the detection of differences between the regression slopes of two datasets, while acknowledging the influence of dental age as a covariate (Pinhasi et al., 2006; Field, 2013). This method was only applied to individuals

between 0-12 years of age to avoid the complications of the sex-differentiated pubertal growth spurt (Shapland and Lewis, 2013; Lewis et al, 2015).

Table 2 – *Sample sizes for pathological and metric analysis (in brackets=adult sample size). BH=vertebral body height; TR= transverse diameter.*

Site	Pathology	Tibial length	Femoral CT	BH (T6-8)	TR (T6-8)
Coach Lane	82	22	20	23 (11)	5
Coronation Street	90	19	31	20 (14)	10
Chelsea Old Church	33	10	10	10 (9)	10
St Benet Sherehog	64	9	13	16 (11)	8
Bow Baptist	202	70	49	42 (27)	46
Cross Bones	104	36	37	38 (5)	22
Total	575	166	160	149 (77)	101

RESULTS

Age-at-death

Figure 2 shows the age-at-death distribution for each sample. This represents the percentage of non-adults that fall into the age categories "foetal and perinatal" (comprising those less than 36 weeks *in* utero, and those aged approximately 36 weeks *in utero* to 4 weeks post-partum), "1-11 months", "1-5 years", "6-11 years", and "12-17 years" of age. The peak age-at-death for the Coach Lane, Chelsea Old Church, St Benet Sherehog, and Bow Baptist samples was 1-5 years of age (Fig.2). There is also a peak in this age category in the Cross Bones sample (30%), and a high percentage of non-adults between 1-11 months and 1-5 years in the Coronation Street sample (at 21% for both groups). However, for both Coronation Street and Cross Bones, the foetal and perinatal age group predominates, making up nearly half of the non-adult samples (at 43% and 48% respectively). It is an inherent feature of cemetery populations that adolescents are poorly represented, as these individuals have already survived the most hazardous stages of the growth period (Lewis, 2007). This pattern is evident in all of the samples with the exceptions of Coach Lane and St Benet Sherehog. These groups show an unusually high representation of individuals aged between 12-17 years.

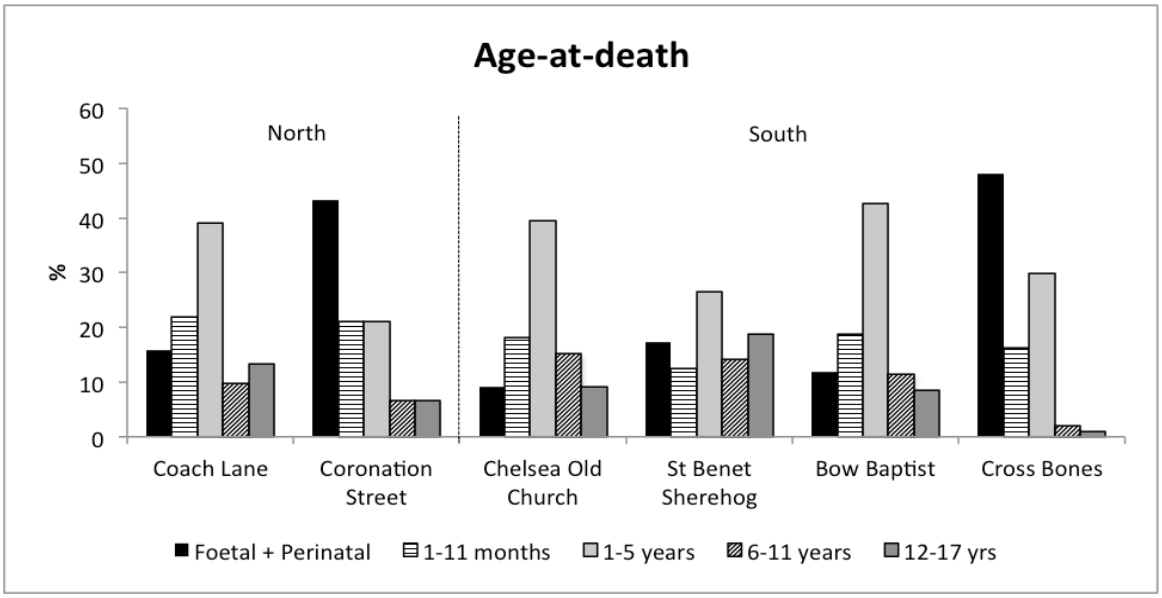

Figure 2 – *Age-at-death distribution of the non-adult sample. Those that could not be assessed for dental age assigned an age category based on indicators of skeletal maturation (diaphyseal length and epipihyseal fusion) (Scheuer and Black, 2000).*

Diaphyseal length

Tibial diaphyseal lengths were plotted against dental age to form a scattergraph, with each data point representing one individual (Fig.3a). The black line shows the averaged data for tibial diaphyseal lengths collected from a healthy, modern comparative data-set (Maresh, 1955). All sites show similar growth patterns to that seen in the modern data for the first two years of life. However, Cross Bones demonstrates some of the lowest tibial diaphyseal length values at birth. Unfortunately, the Cross Bones data set for tibial diaphyseal length does not extend beyond 5 years of age due to preservation. From approximately 2-5 years of age all of the archaeological groups fall below the modern line. St Benet Sherehog and Coach Lane, in general, display some of the highest values throughout the remainder of the growth period. Whereas Chelsea Old Church and Coronation Street exhibit some of the lowest values for tibial diaphyseal length up until 10 years of age. Coronation Street demonstrates significantly lower values for tibial diaphyseal length when compared to the Bow Baptist and Coach Lane data (Table 3). Beyond 10 years, Chelsea Old Church and Coronation Street appear to "catch-up", however this may be due to small sample size. From between 11-13 years of age there is a marked deviation of the archaeological data away from the modern data, so that by 17 years of age, none of the Coach Lane, Coronation Street, Chelsea Old Church, St Benet Sherehog, and Bow Baptist samples have reached the

'normal' values for tibial diaphyseal length. At 16 years of age the Coach Lane, St Benet Sherehog and Bow Baptist samples have still only reached approximately 86%, 84%, and 70% of the modern values respectively. At 15 years of age the Coronation Street group have only reached 81% of the modern values. All archaeological data were significantly lower than the modern data-set (Table 3).

Cortical thickness

Femoral measurements for CT were plotted against dental age (Fig.3b). Modern comparative data exists for measurements of CT, taken from a healthy Finnish population (Virtama and Helelä, 1969). The majority of individuals from all of the archaeological populations fall below this line, revealing statistically significant deficiencies when compared to the modern data (Table 3). This deviation away from the modern data occurs between approximately 1-3 years. Individuals from Cross Bones show a mix of higher and lower values for CT at birth and in infancy. However between 2-5 years of age these data remain particularly low, and are significantly deficient in comparison to Coach Lane (Table 3). Individuals from the Coronation Street, Coach Lane, Bow Baptist, and Chelsea Old Church samples demonstrate some notable deficiencies, particularly at four years of age, and between 9-12 years of age. All of these samples attain higher values once again by 13 years of age. St Benet Sherehog and the Bow Baptists reveal deficiencies throughout the growth period. However, by approximately 15 years of age they appear to have attained similar values to that seen in the modern population, reaching approximately 100% and 94% of the modern data values. Coach Lane and Coronation Street al.so demonstrate deficiencies in CT throughout the growth period, and only attain 88% and 77% of the modern growth values, respectively, by 16 years of age.

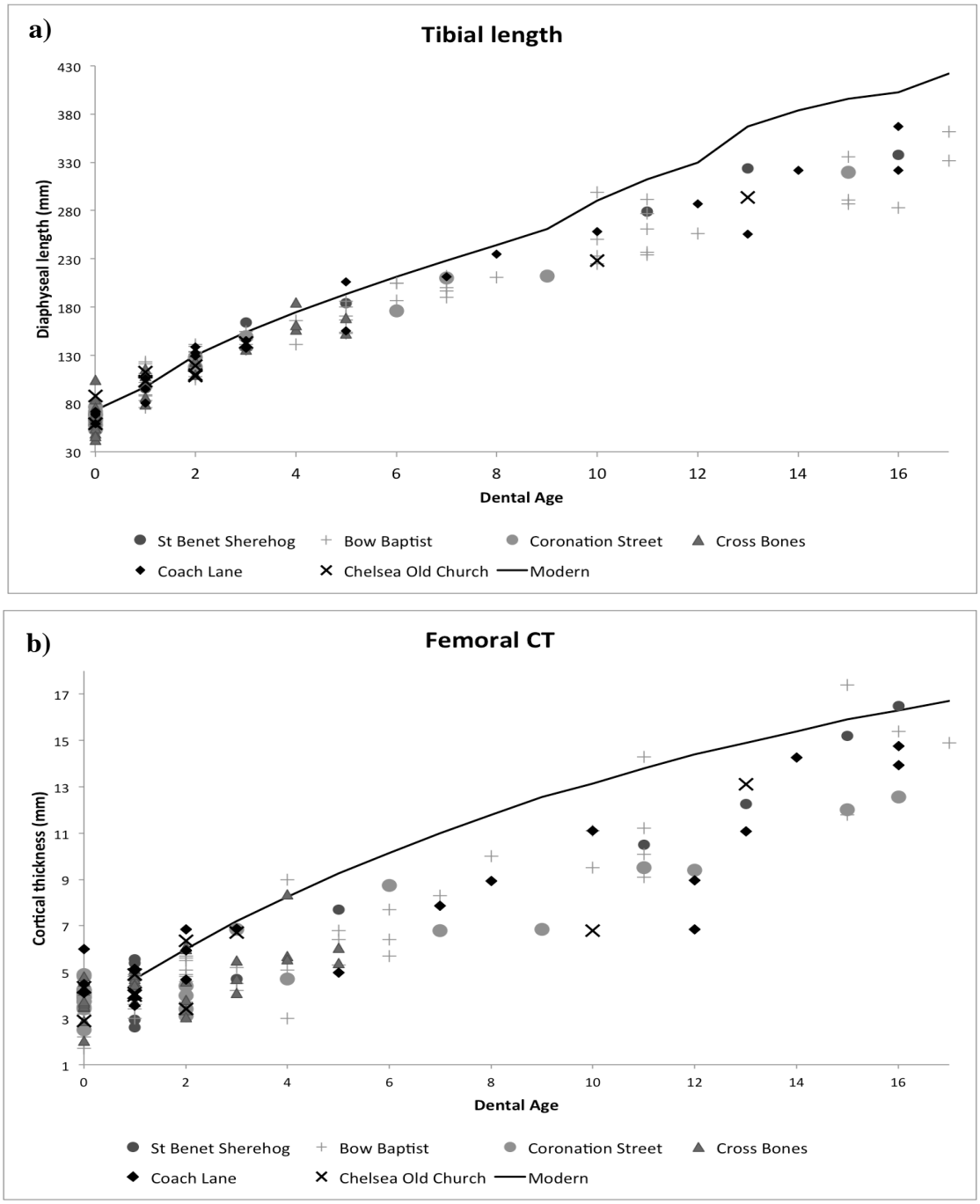

Figure 3 – *Growth profiles for long bone skeletal parameters; a) Tibial length – comparative modern data represented by solid black line, taken from Maresh, 1955; b) Femoral CT – comparative modern data represented by solid black line, taken from Virtama and Helelä, 1969)*

Vertebral body height

Vertebral body height for each individual, based on averaged values for T6-8, was plotted against dental age to form a vertebral growth profile (Fig.4a). Figure 4a also displays data for the average body heights of adult vertebrae from each site, as modern comparative data for vertebral body height is unfortunately not available.

Between 1-4 years of age the growth values for all populations appear to be very similar. At birth, the individuals from the Cross Bones population have the lowest values for body height, and continue to show some of the most deficient values until approximately three years of age, demonstrating significantly deficient values in comparison to the Bow Baptist and Coronation Street samples (Table 3). Coronation Street also demonstrate some of the lower range growth values until 12 years of age, being significantly lower than the Bow Baptist, St Benet Sherehog, and Coach Lane samples (Table 3). Beyond 12 years of age they appear to catch up in growth, reaching 94% of the average adult measurement by 16 years of age. Values for body height at birth are comparatively high for Chelsea Old Church, however the data then begin to level off between 1-3 years of age, and continue to demonstrate some of the lowest growth values until 10 years of age. By 14 years this group appear to have caught up in growth, and have reached approximately 91% of the average adult measurements for this population. Coach Lane and St Benet Sherehog consistently demonstrate some of the highest growth values, and by 15 years of age St Benet Sherehog have reached approximately 97% of the average adult measurement, and by 16 years of age the Coach Lane sample have achieved 90%. Interestingly, the Bow Baptist population appear to lag in growth beyond 12 years of age, and are far from reaching their adult potential for vertebral body height by 17 years of age (at approximately 84% of the adult average).

TR diameter

Measurements of TR diameter for each individual were plotted as a scatter graph against dental age for T6-8 (Fig.4b). Modern data is available for comparison with the archaeological populations. The study by Hinck et al. (1966) provides the average TR diameters of children up to 18 years of age (data averaged into ages 4, 7, 9, 12, 14, and 16 years), and also averages for adult measurements.

The archaeological data sets are in general severely deficient in comparison to the modern data, and this difference is statistically significant (Table 3). The Coronation Street and St Benet Sherehog samples demonstrate consistently higher growth values in comparison to the other archaeological samples, and have achieved 89% and 91% of the modern adult average measurement, respectively, by 16 years of age. The Coach Lane sample has particularly low TR diameters at 10 years of age, only reaching 80% of the average TR diameter for modern children of 9-10 years of age. However, the Coach Lane non-adults have some of the highest measurements for TR diameter in adolescence, reaching 93% of modern adult measurements by 16 years of age. Chelsea Old Church and Cross Bones have the lowest values for TR diameter in the first two years, averaging approximately 72% and 73% of the modern adult values respectively. Chelsea Old Church continues to demonstrate deficient values for the remainder of the growth period, still only achieving approximately 86% of the modern adult values by 13 years of age, and demonstrates significantly lower values than the Bow Baptist and Coronation Street samples (Table 3). All other archaeological inter-site comparisons do not reveal any statistically significant differences (Table 3).

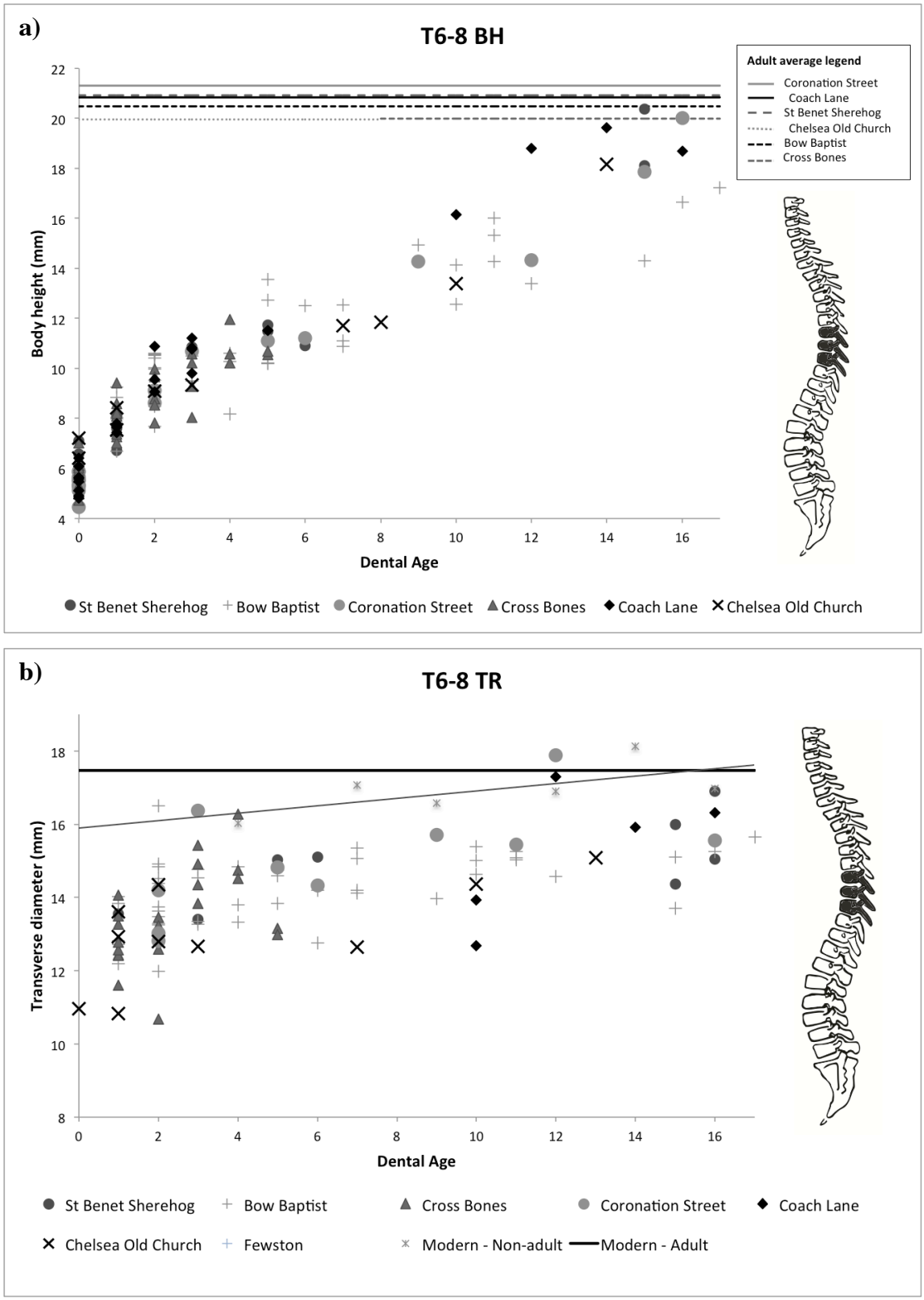

Figure 4 – *Growth profiles for vertebral dimensions; a) Body height measurements for T6-8, plotted with average adult measurements for each site; b) Transverse diameter for T6-8, comparative modern adult data (solid black line) and modern non-adult data (solid grey line) taken from Hinck et al. (1966).*

Table 3 – *ANCOVA results for measurements of tibial diaphyseal length, femoral CT, body height (BH) and transverse diameter (TR) from the four archaeological samples, and compared to modern data. p=<0.05, significant values in bold.*

		Tibial length		Femoral CT		BH		TR	
		F	***p***	**F**	***p***	**F**	***p***	**F**	***p***
Chelsea Old Church vs	St Benet Sherehog	0.106	0.750	0.334	0.571	0.018	0.894	4.444	0.061
	Bow Baptist	0.415	0.522	0.007	0.934	3.414	0.071	10.420	**0.002**
	Cross Bones	0.111	0.741	0.021	0.885	0.159	0.692	3.236	0.083
	Coach Lane	0.580	0.454	1.720	0.203	1.349	0.256	0.000	0.992
	Coronation Street	2.245	0.147	0.003	0.960	2.401	0.134	6.235	**0.025**
St Benet Sherehog vs	Bow Baptist	0.000	0.999	0.527	0.471	4.858	**0.032**	0.455	0.504
	Cross Bones	0.010	0.919	0.470	0.497	0.000	0.985	0.132	0.720
	Coach Lane	0.033	0.857	0.200	0.659	0.125	0.726	2.518	0.188
	Coronation Street	2.043	0.167	0.707	0.406	5.785	**0.023**	0.012	0.915
Bow Baptist vs	Cross Bones	3.234	0.075	0.338	0.563	7.768	**0.007**	1.045	0.311
	Coach Lane	0.296	0.588	0.317	0.576	0.466	0.498	0.408	0.527
	Coronation Street	6.564	**0.012**	0.006	0.940	16.189	**0.000**	3.690	0.061
Cross Bones vs	Coach Lane	0.637	0.428	4.129	**0.047**	0.093	0.762	3.851	0.062
	Coronation Street	1.602	0.211	0.008	0.928	7.327	**0.009**	0.467	0.500
Coach Lane vs	Coronation Street	5.412	**0.026**	2.996	0.091	7.914	**0.008**	2.941	0.120
Modern vs	Archaeological	4.606	**0.000**	10.145	**0.000**	-	-	4.351	**0.001**

Pathological indicators of stress

The crude prevalence rate (CPR) of pathological indicators of stress was calculated as a percentage of individuals within the non-adult samples demonstrating signs of each type of pathology (Fig.5). St Benet Sherehog and the Bow Baptist sample had similar rates of rickets, at 13% and 15% respectively. The rates for Cross Bones and Chelsea Old Church were lower, at 8% and 9%. Coach Lane had the highest rate of rickets at 21%, whereas Coronation Street had no diagnostic cases of this condition. The overall rate of "metabolic disease", while high in the Chelsea Old Church, St Benet Sherehog, and Bow Baptist samples, is much higher in Coach Lane and Cross Bones, at approximately 62% and 44% of the non-adult sample respectively. For Cross Bones this peak is due to the extremely high rate of scurvy, affecting 26% of the non-adult population. While in Coach Lane this high percentage is due to a large number of non-adults falling into the categories "possible scurvy" (CPR 21%) and "possible rickets" (CPR 16%).

Conversely, overall prevalence of metabolic disease is comparatively low for the Coronation Street sample. The CPR rate of scurvy is also minimal in the remaining samples, particularly in the St Benet Sherehog and Bow Baptist samples, and there were no diagnostic cases within Coronation Street. The CPR rate of non-specific infection (NSI) is much higher within Cross Bones (at 28%). The CPR rate for DEH is high amongst the six groups, at a similar rate between Chelsea Old Church, Bow Baptist, Cross Bones, and Coronation Street. However, it is more prevalent in St Benet Sherehog and Coach Lane. Cross Bones has the highest rate of cribra orbitalia (64%) out of all the samples, followed by the Bow Baptist group at 28%. Coronation Street and St Benet Sherehog have the lowest CPR for cribra orbitalia (at 10% and 11%). Overall, while prevalence of pathology is high in all of the archaeological groups, on balance it is the Coach Lane and Cross Bones populations that show the highest rate of skeletal pathologies.

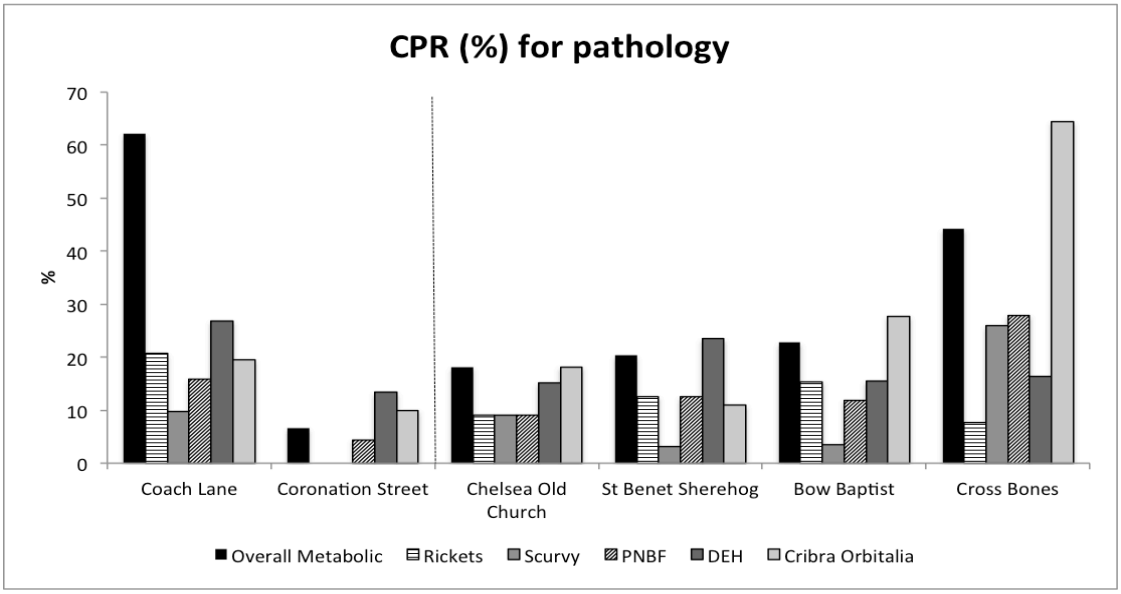

Figure 5 – *Crude prevalence rate (CPR) of pathology seen within the non-adult sample of each site. PNBF = periosteal new bone formation.*

DISCUSSION

"God help 'em! North an' South have each getten their own troubles." Nicholas Higgins – North and South, Elizabeth Gaskell (1855:118)

Historical documentation states that infancy was the most perilous period in the life course, with approximately 41% and 39% of all deaths being in those under five years of age in London and Northumberland, respectively, in 1850 (Registrar-

General, 1854). This is also reflected in the osteological data, with peak age-at-death generally occurring between 1-5 years of age. However, the peak age-at-death occurred much earlier within the Cross Bones and Coronation Street samples, in the foetal and perinatal category. These samples represent populations of lower status; and the early age-at-death is suggestive of poor maternal health and detrimental postnatal exogenous factors associated with being born into poverty (Lewis and Gowland, 2007; Gowland 2015). However, it also must be acknowledged that the burial ground of Cross Bones was unconsecrated, as was a portion of the excavation area within Coronation Street (Brickley and Miles, 1999; Raynor et al., 2011). This "peak" may therefore be more representative of a skew in the data due to the use of these sites for the burial of un-baptised, or still-born infants. That all of the archaeological samples were subject to high risks of morbidity in infancy is also supported by the growth data. All samples demonstrated significant deficiencies in TR diameter when compared to modern values. As this skeletal parameter completes the majority of its growth in the first two years of life (Jinkins, 2000; Scheuer and Black, 2000), this deficiency is indicative of episodes of growth disturbance in infancy. These data are indicative of a universal high risk of high infant/child mortality due to detrimental urban conditions seen within northern and southern industrial centres at this time.

For diaphyseal length and cortical thickness, archaeological values fall away from the modern comparative data sets in general between 1-3 years of age. Evidence of stunting in this age range, alongside timings of peak prevalence of stress indicators, have been used in past studies to discuss onset of weaning and deficiencies in weaning diet (Lewis, 2002a,b). As can be seen in Table 4, similar to that seen in the London populations (with the exception of Cross Bones), peak timings of those classed as having "metabolic disease" within the Coach Lane and Coronation Street samples occurred between 1-5 years of age, corroborating the results from the growth data. For Coach Lane and Coronation Street there is also a peak in prevalence between 1-11 months (however this should be interpreted with caution for Coronation Street as such a low number of individuals were classed as having "metabolic disease"). This may be indicative of a variation in infant feeding practices for these populations.

Table 4 – *Breakdown of metabolic disease by age category. Metabolic disease referring to number of individuals within the skeletal sample demonstrating any sign of rickets, possible rickets, possible scurvy, and scurvy. Percentage in brackets.*

Site	No. Non-adults	Foetal + Perinatal	1-11 mths	1-5 years	6-11 years	12-17 years	Unknown	Overall
Coach Lane	82	7 (8.5)	**11 (13.4)**	**21 (25.6)**	3 (3.7)	9 (11)	0 (0)	51 (62.2)
Coronation Street	90	0 (0)	**2 (2.2)**	**2 (2.2)**	1 (1.1)	0 (0)	0 (0)	5 (5.6)
Chelsea Old Church	33	0 (0)	0 (0)	**3 (9.1)**	1 (3)	2 (6.1)	0 (0)	6 (18.2)
St Benet Sherehog	64	2 (3.1)	1 (1.6)	**6 (9.4)**	1 (1.6)	0 (0)	3 (4.7)	13 (20.3)
Bow Baptist	202	4 (2)	18 (8.9)	**21 (10.4)**	2 (1)	1 (0.05)	0 (0)	46 (22.8)
Cross Bones	104	**24 (23.1)**	**11 (10.6)**	**11 (10.6)**	0 (0)	0 (0)	0 (0)	46 (44.2)

The late 18th and early 19th centuries saw a shift in the popularity of breastfeeding practices, a trend that spanned the social strata (see Newman and Gowland, accepted). Henderson et al. (2014) used incremental isotopic analysis to investigate nutritional status and weaning age within non-adults from St Saviour's Almshouse burial ground in Southwark, London. It was found that, in general, weaning occurred before or by six months of age, and a minimal number of infants were not breastfed at all (Henderson et al., 2014). Additionally, a study by Nitsch et al. (2011) found that a range of infant feeding strategies occurred within the skeletal population of Christ Church, Spitalfields, with some infants receiving little to no breastmilk, whilst others were breastfed until approximately 1.5 years of age. Infants should not develop scurvy prior to six months of age, due to the provision of maternal vitamin C stores during pregnancy, and through the breastmilk after birth (Brickley and Ives, 2008; Buckley et al., 2014). Therefore, evidence of scurvy in infants can be indicative of early cessation of breastfeeding. Alternatively, if the mother is herself vitamin C deficient, it will in turn be passed on to the infant (Cheadle, 1889; Brickley and Ives, 2008). When rates for scurvy and "possible scurvy" are combined for each sample, Cross Bones and Coach Lane have the highest prevalence, at 37% and 31% respectively. This is compared to the relatively low prevalence within the Chelsea Old Church, St Benet Sherehog, Bow Baptist,

and Coronation Street samples (at 9%, 5%, 10%, and 2%, respectively). Thus the peak prevalence of scurvy in the "foetal and perinatal" age category in the Cross Bones sample is of great interest. Diagnostic criteria for scurvy within non-adults has continued to develop since the original analysis of this site in 1999 (Brickley and Miles, 1999; Brickley and Ives, 2008; Armelagos *et al.,* 2014; Stark, 2014; Klaus, 2014a). Additionally, due to the potential misdiagnosis of skeletal changes in individuals less than six months of age, there is a possibility that scurvy may have been over-recorded within the perinatal sample for Cross Bones. However, the London-based non-adult samples were re-categorised into "scurvy", "possible scurvy", and "metabolic disease" in accordance with more recent publications (Brickley and Ives, 2008; Armelagos *et al.,* 2014; Stark, 2014), based on descriptions of skeletal changes from the WORD database. This ensured comparability with the pathological data from the northern samples.

In addition, within the Coach Lane sample, out of all 25 cases of scurvy and possible scurvy within this sample, 28% were perinatal, 32% were 1-11 months, and 32% were aged 1-5 years. These peaks in cases of scurvy and "possible scurvy" may therefore indicate a variety of breastfeeding and weaning practices within these northern and southern populations, as well as being suggestive of poor maternal health. Both northern and southern urban centres became increasingly reliant on the developing industries for employment, which often necessitated an early return of mothers to work following child-birth. This not only led to poor maternal health status, but also left infants exposed to risks of malnutrition from an early cessation of breastfeeding (Newman and Gowland, accepted).

Cribra orbitalia is a condition that is often closely associated with scurvy, as iron deficiency may result from haemorrhaging of weakened blood vessels, and vitamin C aids iron absorption (Baker et al., 2010; Agarwal et al., 2015; Ferrari et al., 2015). It has also been suggested that the substitution of breastmilk with cow's milk at a very early age can result in irritation of the immature digestive tract, leading to iron-deficiency anaemia (Stuart Macadam and Dettwyler, 1995; Lewis, 2002a). This co-morbidity may explain the extremely high rates of both scurvy and cribra orbitalia within the Cross Bones sample (Fig.5). However, the remaining five samples also demonstrated a high rate of cribra orbitalia (ranging from 10-28% CPR). It has been argued that iron-deficiency anaemia is not a sufficient explanation

for the skeletal changes seen in cribra orbitalia. Walker et al. (2009) argue that acquired megaloblastic anaemia is a more likely candidate, resulting from vitamin B12 deficiencies and nutrient loss from gastrointestinal infections, promoted by unhygienic living conditions. While this theory has itself been contested (Oxenham and Cavill, 2010), it is generally accepted that cribra orbitalia is broadly related to unhygienic environments and dietary deficiencies in past populations. Therefore its high prevalence within all of these samples reflects the deleterious environmental conditions associated with life in northern and southern urban centres of this time. Similarly, all of the skeletal samples demonstrated high rates of DEH. These defects are also related to episodes of infection and deficiencies in nutrition during infancy and childhood (Goodman and Rose, 1990; Hillson, 2008). This evidence corroborates the deficiencies seen in the transverse diameters of all six archaeological populations, and is suggestive of the high risks that accompanied infancy during this time, regardless of class or geographic location.

It was hypothesised that the northern-based skeletal collections would demonstrate higher rates of rickets due to the reduced hours of sunlight during the winter months in northern latitudes (Holick, 2004; Pearce and Cheetham, 2010). The Coach Lane skeletal sample does demonstrate a high rate of metabolic disease throughout the growth period (Table 4), particularly bowing deformities in older children and adolescents. A high rate of residual rickets was also recorded amongst the adults of this skeletal collection (Tschinkel, 2013). Overall there was a combined 37% CPR of rickets and "possible rickets" in the Coach Lane non-adult sample. This is much higher than that of the London samples (Bow Baptists = 17%, Cross Bones = 13%, St Benet Sherehog = 14%, Chelsea Old Church = 12%). As in many of the common childhood diseases of the 18th and 19th centuries, there was a degree of seasonality with rickets, with increases in its prevalence in the spring (Hardy, 1992). Infants were said to be "at their most vulnerable to rickets when the dietary imbalances of the weaning process were compounded by the sunlight deprivation of the winter months" (Hardy, 1992: 398). During winter, there is a reduction in not only day length, but also the quantity of UVB photons that reach the earth's surface (Holick, 2004). This is most notable in regions classed as "high latitude", above 37^{o} North, from October to March (Holick, 2004; Macdonald et al., 2011; NICE, 2014). While this region encompasses the majority of Europe, risk of

vitamin D deficiency continues to increase with increasing northern latitude. Recent surveys have revealed that one fifth of adults, and 8-24% of children, in the UK today suffer from deficiencies in vitamin D status (NICE, 2014). Postmenopausal women from Scotland (at latitude 57° North) have been shown to be worse affected when compared to those from the South of England (at 51° North) (Macdonald et al., 2011). The groups most at risk of developing vitamin D deficiencies today (and likewise in the 18th/19th centuries) are children under the age of five years, pregnant and breastfeeding women, individuals over 65 years of age, and those spending long hours indoors (NICE, 2014). Vitamin D status at birth is reliant on maternal health in pregnancy (NICE, 2014); therefore such deficiencies can be "inherited". Approximately 20% of cases of rickets and "possible rickets" combined occurred in those under one year of age in the Coach Lane sample, indicating that maternal vitamin D status within this population was also compromised.

As sunlight is responsible for 80-90% of vitamin D levels in the body, it must be assumed that there is a dependence on stores accumulated during the summer months (Macdonald et al., 2011). However, in regions such as the North-East, where air pollution was at such extreme levels in the 18th and 19th centuries, it is likely that such seasonal deficiencies could not as easily be abated and may have led to deficiencies that were more chronic in nature. Considering the influence that vitamin D is thought to have on immune status, it is likely that such deficiencies left children highly susceptible to future health insults, and it has been shown that those living in lower latitudes, or in those with increased vitamin D intake, have a decreased risk of many chronic diseases (Holick, 2004). A heightened prevalence of pathology certainly exists within the Coach Lane sample, which may be indicative of the reduction in immune status conferred by the elevated rates of metabolic disease seen within this group. However, this potential association with heightened risk of vitamin D deficiency in northern regions is confounded by the palaeopathological data for Coronation Street. Coronation Street demonstrates notably low rates of all pathological conditions assessed in this study. While this evidence may be used to infer that this population were perhaps exposed to fewer environmental hazards than the other samples in this study, the growth data contradicts this assertion. Coronation Street demonstrates some of the lowest growth values for diaphyseal length, vertebral body height, and femoral CT.

According to Wood et al. (1992), skeletal samples demonstrating no sign of pathology are not necessarily "healthier". Skeletal samples represent non-survivors, and it is possible that the non-adults of Coronation Street suffered from acute afflictions, and simply did not survive long enough for skeletal responses to occur (Wood et al., 1992; Vercellotti et al., 2014). In complete absence of vitamin C, an individual may die before skeletal indicators of scurvy can manifest, whether from the deficiency itself, or from susceptibilities in the immune system to other infectious agents (Armelagos et al., 2014; Bourbou, 2014; Krenz-Niedbała, 2015). In addition, infectious disease is classed as an acute stressor, which is more likely to impact on the survival of an individual, rather than their capacity to maintain growth under stress (Vercellotti et al., 2014). Infectious disease was rife in urban environments of the 18th and 19th centuries, with measles, diphtheria, smallpox, whooping cough, and scarlet fever being common risks to the survival of the very young (Levene, 2012). The industrialised towns of North Shields and South Shields exposed their inhabitants to the same polluted environs as experienced by those in London. It has been stated that-

"Among the many places in the England of 1800 where Poverty was pursuing her road in aught but tranquillity, probably none displayed the poorer life of the time within narrower limits or in more varied phase than the twin seaport towns of North and South Shields" (Haswell, 1895: 16).

Those of the lower classes in North and South Shields lived within housing that suffered from a lack of effective drainage and sewerage, and were said to be "struggling too often under the accumulated influences of poverty, disease, and vice…" (The Report of the Commissioners, 1845b: 18). Such an environment would be expected to propagate the types of infectious disease that would be highly detrimental to child health and survival. This may be reflected in the high infant mortality seen in the Coronation Street collection, and the deficient growth values seen within all growth parameters. It has also been suggested that while child labour was common in this region, the children of Coronation Street were more likely to have been employed in industries that involved outside work, such as ship-building, which may have buffered them from vitamin D deficiencies (Raynor et al., 2011). Therefore the Coach Lane non-adults undoubtedly suffered a higher risk of

contracting rickets when compared not only to the four London-based samples, but also Coronation Street. This is likely due to a combination of factors, including the restricted access to sunlight in the winter months, and social practices such as indoor employment. There is evidence that children within the Coach Lane community were employed in factories, with one non-adult aged 12-14 years suffering from "phossy jaw", a necrotising condition occurring from exposure to phosphorus, common in matchmaking (Roberts et al., 2014). Thus it is possible that even within the northern region of Tyneside, variances in local industries had a substantial impact on the health of a community.

Other evidence for child labour practices within these sites, tentatively, comes from the Bow Baptist sample. Figure 4a shows that for vertebral body height, the non-adults of the Bow Baptist group appear to lag in growth in adolescence, and still only achieve 84% of the adult average by 17 years of age. A more in-depth study of the vertebral growth within this site identified that this lag occurs between 9-16 years of age (Newman and Gowland, 2015). Entrance of children into the labour market from 12 years of age was identified by Cardoso and Garcia (2009) as a potential cause for growth delay seen in adolescence within skeletal samples from late medieval to early 20th century Portugal. During this period, such an increase in energetic demands required for manual labour, combined with paucities in nutritional intake and exposure to infectious disease environments, may lead to an inability to meet the energetic demands of the pubertal growth spurt (Cardoso and Garcia, 2009; Duren et al., 2013). The effect that manual labour had on the growing child of the 18th and 19th centuries is well documented. A growth study from the 1830s investigating the heights of children between 8-14 years of age from manufacturing towns revealed significant growth delays that persisted through later development (Sharpe, 2012). From 1833 only children of age nine and above could be employed within cotton mills, and from 1847 children under nine years of age could no longer be apprenticed (Pike, 1966; Pinchbeck and Hewitt, 1973; Burnett, 1984). Therefore, this growth delay seen in the vertebral body height of the Bow Baptist sample may be connected to increased energetic demands during adolescence, and this may in turn be connected to child labour practices. This sample has the second highest rate of rickets and "possible rickets" in this study (17%). This indicates that children were being kept indoors (whether due to illness,

protection, or employment) and/or experienced deficiencies in diet. In contrast, the Coach Lane sample, despite having evidence of factory work within the skeletal collection, shows some of the highest values for vertebral body height between 10-16 years of age. It is possible that this population had access to a superior diet, and as stated previously, London diets were notoriously poor. When children began work to help support the family economy, their diet may have improved (Horrell and Oxley, 2012). Allocation of food resources was weighted towards "wage-earners", who required adequate nutrition to continue to work and support the family (Burnett, 1984; Horrell and Oxley, 2012). While this model meant that the younger children would be left to subsist on a severely deficient diet, older working children may have benefitted from improvements to nutrition later in the growth period, if the food resources available were sufficient to enable this. This may also explain why such a high prevalence of pathology exists within this collection, as better nutrition would increase the likelihood that an individual would survive health insults long enough for skeletal manifestations to occur (Wood et al., 1992). By 17 years of age, none of the Coach Lane, Bow Baptist, St Benet Sherehog, and Coronation Street samples reached the modern day values for tibial diaphyseal length, and it was noted that this deviation away from the modern data began from approximately 11-13 years of age, which is concurrent with the norm for age of entrance into the labour market for this time (Pike, 1966; Pinchbeck and Hewitt, 1973). There is also a disproportionately high representation of those aged 12-17 years of age in the St Benet Sherehog and Coach Lane samples. Such deficiencies may also be indicative of the influence of child labour practices within all these sites on adolescent health. However, deficiencies seen in growth are also likely to have their origins in the well-documented paucities in nutrition and environment experienced within the family home, so these data cannot be definitively attributed to child labour practices (Kirby, 2013).

Overall there were no prominent differences in growth between northern and London based skeletal samples, despite regional differences in diet and industry. Very few of the skeletal parameters revealed statistically significant differences between the archaeological samples, yet showed significantly deficient values when compared to modern data. This suggests that the industrialised centres of the both the North and the South promoted adverse environmental conditions, which proved

to be highly detrimental to child health at this time. Therefore, any variances observed between these samples are more likely to be associated with more nuanced local differences in the social gradient, infant feeding practices, and child labour practices rather than the influence of the "North-South divide" at this time. However, further study is required from a variety of geographic locations to gain a clearer picture of the influence of geography, and related industries, on child health during the 18th-19th centuries.

Although the results of this study have provided convincing evidence for disruption of growth within the samples due to environmental influences and social factors, the limitations of the methods used within this study must be addressed. Growth studies in archaeological populations are, unavoidably, cross-sectional rather than longitudinal. Therefore the possibility that the non-adults of past populations, had they survived, might have demonstrated a different pattern of growth cannot be discounted (Wood et al., 1992). It must also be acknowledged that the individuals within this study are "non-survivors", so the growth patterns and prevalence of pathology demonstrated by them may not necessarily be representative of the experiences of the general population that survived to adulthood (Wood et al., 1992).

In addition, the small sample sizes for the growth parameters in several of the sites within this study are an inherent issue within bioarchaeological study. However, this does not mean that such samples should be excluded from bioarchaeological research, as they can still reveal valuable information regarding health in the past. With the use of appropriate statistical techniques, and multiple modes of analyses, patterns in health stress can still be determined with reliability.

CONCLUSION

While there was no definitive difference in growth between northern and southern populations, the extremely high rate of rickets (and high rates of pathology in general) exhibited by the Coach Lane sample may indicate that certain northern populations faced more severe health insults due to vitamin D deficiency and subsequent reduction in immune resistance. It may be a combination of social factors (such as the keeping of children indoors, or child labour practices) alongside northern latitude that influenced the high rate of metabolic disease seen in the

Coach Lane collection. There is a rising concern that rickets is returning amongst low-income families, generating a high risk for immune suppression and future poor health (Pearce and Cheetham, 2010; Bivins, 2014). The links between vitamin D status, latitude, and chronic disease are well established, and in light of reduced life expectancies and generally poor health outcomes in lower income areas within Scotland and the North-East (Whitehead, 2014; Health and Sport Committee, 2015), it is plausible that vitamin D deficiencies play a substantial role in the pervasive health inequalities experienced by these regions.

This study reveals the benefit of using multiple skeletal indicators to strengthen evidence of health stress in the past. The implementation of growth parameters that target different areas of the developing body in children can provide additional resolution to the determination of timings of episodes of stress. As such, comprehensive growth studies such as this have scope to be further strengthened by the addition of incremental isotopic analysis. To bring together all of these methods would substantially increase our osteobiographical understanding of skeletal assemblages, and enhance our understanding of the impact of health stress on the life course.

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MANUSCRIPT FOUR - Town-made articles of small stature: unravelling growth disruption and pathological indicators of stress in skeletal populations.

To be submitted to The American Journal of Physical Anthropology.

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ABSTRACT:

Objective: Growth disruption has often been used to identify episodes of poor health in past communities. Such disruption can be triggered by a broad spectrum of health stressors. This study aims to investigate whether multiple indicators of stress can be combined to elucidate root causes of "stress" within two urban-based skeletal samples from the 18th/19th centuries.

Methods: Measurements of growth (tibial diaphyseal length, tibial cortical thickness, and vertebral dimensions) and palaeopathological data were recorded for 82 non-adults (0-17 yrs) from Coach Lane, North Shields, and 202 non-adults from the Bow Baptist collection, London.

Results: The Coach Lane sample displayed a higher prevalence of stress indicators in general, and for metabolic disease in particular. Deficiencies in growth for this sample were more influenced by high rates of rickets and scurvy, and their consequent co-morbidities. Whereas poorer growth values within the Bow Baptist sample were associated with cribra orbitalia and dental enamel hypoplasia.

Discussion: The industrial cities of the 18th/19th centuries were infamously detrimental to child health, with frequent occurrence of poor health and dietary deficiency. This is reflected in the patterns of growth and pathology exhibited by the Bow Baptist sample. However, the strong influence of metabolic disease within the Coach Lane sample is indicative of social influences (i.e. child labour practices, insufficient weaning diets, and/or keeping children indoors). The use of multiple indicators of stress within bioarchaeological analysis can begin to tease apart patterns in health stress, and enable a deeper understanding of child health in the past.

INTRODUCTION

Evidence of growth disruption in past populations has often been used to enable discussion in regard to changing conditions between geographical location, time period, and social groups (Ribot and Roberts, 1996; Mays et al., 2008, Mays et al. 2009a,b; Lewis, 2002a,b; Pinhasi et al., 2006; Cardoso and Garcia, 2009). Children require sufficient energetic resources for not only the basic demands of day-to-day activity, but also for basal metabolism, repair, and the growth processes (Møller and Swaddle, 1997; Bogin, 1999; Bogin et al., 2007). When placed under conditions of physical and/or psychological stress, fairly predictable physiological pathways react to ensure "survival" of an individual (McEwen, 1998; Sapolsky, 2004). The stress response is designed to respond to acute stressors, triggering the sympathetic nervous system to release adrenaline, noradrenaline, and cortisol to bring about the "fight or flight" response, placing preference on essential bodily processes for survival (Sapolsky, 2004). However, under conditions of chronic stress, this response can become injurious (McEwen, 1998). Under these conditions, resources are diverted to essential bodily processes, at the expense of growth (McEwen, 1998; Bogin, 1999; Sapolsky, 2004; Bogin et al., 2007; Stulp and Barrett, 2014). While this response means that evidence of growth disruption is by proxy an effective indicator of unfavourable environments and/or poor health in children, there are many discrete triggers that may elicit this response. Children are extremely sensitive to stressors, such as recurrent infection, malnutrition, climatic changes, and low socioeconomic status (Bogin, 1999; Lewis, 2007; Ruff et al., 2013; Stulp and Barrett, 2014). Thus, growth can be impacted by a myriad of factors (both intrinsic and extrinsic to the individual), and when growth profiles demonstrate signs of disruption the causative factors are, by necessity, non-specific in nature.

The epidemic urban environment of the 18th and 19th centuries is well documented, and previous studies have revealed evidence of growth disruption within non-adult samples dating to this period (Lewis, 2002a,b; Mays et al., 2008; Mays et al., 2009a,b; Pinhasi et al., 2006). Life in the city came with significant health risks, ranging from the noxious conditions of the insanitary streets, the virulent spread of disease, limited social mobility, and chronic malnourishment (Booth, 1889; Chadwick, 1842; Engels, 1950; Horrell and Oxley, 2012; Sharpe,

2012). In this context, it becomes difficult to tease apart why one urban population may show similarities or contrasts in growth when compared to another.

Attempts have already been made to identify causative factors in the stunted growth seen in skeletal samples from the 18th/19th centuries. Rickets was a major contributor to the high morbidity and mortality seen amongst children in this period (Hardy, 1992; Gibbs, 1994). The heavily polluted atmosphere from the increasing number of factories, alongside child labour practices that limited exposure to sunlight during the long working hours, meant that deficiencies in vitamin D were rife, and became notoriously associated with the urban centres of this time (Mays et al., 2008,2009b). Mays et al. (2009b) noted deficiencies in bone length in non-adults between the ages of 2-6 years exhibiting skeletal changes associated with rickets. However, Pinhasi et al. (2006) found no evidence for differences in growth between individuals with or without skeletal indicators of rickets. It was suggested that catch-up growth in long bone length may have obscured any significant effects of vitamin D deficiency on growth (Pinhasi et al., 2006). Therefore, the identification of causative factors of growth disruption within skeletal collections of the 18th-19th centuries still requires further study, and the implementation of multiple indicators of stress to combat the veiling effects of catch-up growth.

This paper aims to combine palaeopathological data with a variety of growth parameters (tibial diaphyseal length, tibial cortical thickness, vertebral body height, and vertebral transverse diameter) to identify the key disease processes/stressors that resulted in the growth stunting seen within two skeletal collections dating to the 18th and 19th centuries. Such patterns may reveal variances in the root causes of growth disruption within each site, enabling a more directed discussion as to why each group suffered growth deficiencies in the context of 18th/19th century urban centres.

MATERIALS & METHODS

The two skeletal collections analysed in this study are summarised in Table 1, and provide a combined sample of 284 non-adult skeletons (0-17 years). Measurements relating to tibial diaphyseal length, tibial cortical thickness, and vertebral dimensions were taken when preservation allowed. All individuals were

also assessed for the presence of four skeletal indicators of stress (metabolic disease, dental enamel hypoplasia, cribra orbitalia, and Harris' lines).

Table 1 – *Summary of sites.*

Site	Date	Status	No. Individuals	No. Non-adults
Coach Lane, N. Shields	1711-1857	Low/Middle	236	82
Bow Baptist, London	1816-1856	Middle	416	202

Coach Lane was a former Society of Friends burial ground (c.1711-1857 AD) located in North Shields, to the east of Newcastle-upon-Tyne. North Shields in the early 19th century was a populous shipping, fishing, and coal-mining community (Gould and Chappel, 2000; Pre-construct Archaeology, 2012). The simplicity of the Quaker lifestyle makes the establishment of social status problematic within this sample (PCA-North, 2011; Pre-construct Archaeology Ltd, 2012). However, it is possible that this site contains both low and middle status individuals.

The Bow Baptist skeletal collection (c. AD 1816-1856) resided within the small village of Bow, located on the outskirts of London at the beginning of 1816 (Henderson et al., 2013). In the second half of the 19th century this middle class community were being absorbed into the rapidly expanding city (Henderson et al., 2013).

METHODS

Assessment of dental age

Dental age was assessed from dental development using standards for calcification (Moorrees et al., 1963a,b; Smith, 1991) and eruption (Van Beek, 1983) of both the deciduous and permanent dentition. Calcification stages were determined by examination of the dentition radiographically, or macroscopically when loose teeth were present. These stages were used to assign a dental age to each individual, based on the mid-point of the age category that they fell into. For example, those between 0.5-1.49 years of age were classed as 1 year of age (Mays et al., 2008).

Measurement of diaphyseal length

Growth profiles were constructed by plotting measurements of tibial diaphyseal length for each individual against their dental age. The tibia was selected for this measurement as it undergoes rapid growth during development, so is thought to be more sensitive to growth disruption (Holliday and Ruff, 2001; Bogin et al., 2002; Pomeroy et al., 2012). The maximum diaphyseal length was measured for the left tibia using a standard osteometric board to 0.01mm (Buikstra and Ubelaker, 1994). When the left tibia was absent it was substituted with the right side (Ives and Brickley, 2004). Long bones that had undergone post-mortem damage were omitted from the study. Tibial diaphyseal lengths from the Bow Baptist sample had previously been recorded in accordance with the above standards, this data was provided by the Museum of London Archaeology (MoLA). Tibial diaphyseal lengths were compared to existing modern comparative data for individuals 0-18 years of age from the study by Maresh (1955).

Measurement of cortical thickness (CT)

To measure CT, the left tibia was again selected for radiographic analysis (substituted with the right side when necessary). Antero-posterior radiographs were taken at between 65-80kVp and 4-6mAs from the London based samples using a Kubtec Xtend 100HF x-ray source and Kubtec 3600 CR reader (by Professor Gerald Conlogue, personal communication, 20th April 2015), and from the Coach Lane sample using a Portable GE Medical MPX X-ray unit and Kodak point of care CR System. Measurements of the total bone width (T) and the medullary width (M) were taken from the mid-shaft of the tibia (Mays et al., 2009b). Cortical thickness was determined as T-M, and each measurement was plotted against the dental age to form a CT growth profile for each sample. Tibial CT measurements were compared to existing modern comparative data for individuals 0-18 years of age from the study by Virtama and Helëla (1969).

Vertebral measurements

In a previous study, the cervical and mid-thoracic regions of the vertebral column demonstrated the least inherent variation for transverse (TR) diameter (for methodology see Newman and Gowland, 2015). Therefore, the C5-6 region was

selected for analysis of vertebral dimensions due to larger sample sizes in the Coach Lane sample. Measurements of vertebral body height were taken using sliding calipers (to the nearest 0.01mm) from the midline of the centra at the point of maximum height (Newman and Gowland, 2015). Measurements of body height were also taken from adults aged 18-35 years from each sample to provide comparative data. Adult data was pooled for the sexes, as there is currently no reliable method of determining sex in non-adults (Saunders, 2008).

Measurements of TR diameter of the neural canal were taken using sliding calipers (to the nearest 0.01mm). These measurements represented the furthest distance between the medial surfaces of the left and right pedicles (Watts, 2011; 2013a,b) and could only be taken when the neural arches had fused at the spinous process, though fusion of the neural arch to the vertebral body was not essential. Transverse diameter was compared to modern data from the study by Hinck et al. (1966).

Averages of the vertebral measurements from C5-6 were calculated for each individual and then plotted against dental age to form growth profiles.

Recording of pathological indicators of stress

All non-adult individuals were assessed for the presence/absence of metabolic disease (rickets and/or scurvy), dental enamel hypoplasia (DEH), cribra orbitalia, and Harris' lines (recorded in those selected for CT measurement). This assessment was carried out by the author for Coach Lane. Pathological data from the Bow Baptist collection was provided by the Museum of London Archaeology (MoLA). Each individual was assessed according to the criteria outlined below.

Rickets was recorded as present when an individual demonstrated several of the following diagnostic criteria: bowing of the long bones, flaring/swelling of the metaphyses, cupping deformities of the metaphyses, coxa vara, and flaring of the sternal rib ends (Mays et al., 2006; Pinhasi et al., 2006). Other indicators included porosity of the growth plate, new bone formation, porosity of the cranial bones, and thickening of the diaphyses (Ortner and Mays, 1998; Mays et al., 2006; Pinhasi et al., 2006; Brickley and Ives, 2008). The category "rickets" includes individuals with diagnostic skeletal indicators, and those classed as having "possible rickets". Cases

of both active and healed rickets were considered together, as a previous study by Mays et al. (2009b) found no significant differences in growth between the two.

Scurvy was recorded as present when an individual demonstrated a multitude of skeletal changes such as porosity/new bone formation on the sphenoid, maxillae, mandible, orbits, and on the infra- and supra-spinous regions of the scapulae (Ortner and Erickson, 1997; Brickley and Ives, 2006; Brickley and Ives, 2008; Armelagos et al., 2014; Stark, 2014). Other indicators included new bone formation on the long bones, porosity/new bone formation on the bones of the cranial vault, and flaring/swelling of the rib ends (Ortner and Erickson, 1997; Brickley and Ives, 2006; Armelagos et al., 2014). The category "scurvy" includes individuals demonstrating diagnostic indicators of this condition, and those with "possible scurvy".

To account for any potential misidentification (Ortner and Mays, 1998; Stark, 2014), individuals with rickets and scurvy were also considered together under the separate category "metabolic disease".

Cribra orbitalia, referring to the marrow hypertrophy seen in the orbits, was recorded using the five stage scoring system of Stuart-Macadam (1991). Dental enamel hypoplasia (DEH) was recorded as present when an individual demonstrated pit or furrow defects on one or more of the teeth. Lastly, whilst recording measurements of CT, each individual was also assessed for the presence/absence of Harris lines on the tibiae. Lines were scored as present if they crossed at least half of the diameter of the bone (Ribot and Roberts, 1996; McEwan et al., 2005).

The overall sample sizes for pathological analysis, diaphyseal length, cortical thickness, and vertebral growth can be found in Table 2.

Table 2 – *Sample sizes for pathological and metric analysis (adult sample size).*

Site	Pathology	Tibial length	Tibial CT	BH (C5-6)	TR (C5-6)
Coach Lane	82	22	26	31	12
Bow Baptist	202	70	36	49	42
Total	284	92	62	80	54

Statistical analysis

Inter-site comparisons between the growth data (tibial diaphyseal length, tibial CT, vertebral body height, and TR diameter) of both archaeological sites, and against modern data, were statistically assessed using analysis of covariance (ANCOVA). ANCOVA allows for the detection of differences between the regression slopes of two datasets, while acknowledging the influence of dental age as a covariate (Pinhasi et al., 2006). This method was only applied to individuals between 0-12 years of age to avoid the complications of the sex-differentiated pubertal growth spurt (Lewis et al., 2015). Overall prevalence of pathology was compared between the two sites via a Chi-squared test. All tests used a significance level of 0.001.

RESULTS

Inter-site comparison

Tibial length, tibial CT, vertebral body height, and TR diameter were plotted against dental age for both the Coach Lane and Bow Baptist samples (Figure 1). Each data point represents one individual, and black lines represent comparative modern data for each growth parameter (Maresh, 1955; Hinck et al., 1966; Virtama and Helëla, 1969). Due to the lack of modern data for vertebral body height, average adult measurements for each sample have been plotted for this measurement (Figure 1c).

For tibial diaphyseal length (Fig.1a) both archaeological samples appear closely associated with the modern data throughout infancy and early childhood. However, they begin to fall below the modern data line from approximately three years of age, this deviation becoming particularly apparent after 8-10 years of age. By 16 years of age the Coach Lane and Bow Baptist samples have only reached 86% and 70% of the Maresh data-set, respectively (Fig.1a). There were no significant differences in tibial diaphyseal length for the Coach Lane and Bow Baptist samples, however the archaeological sites were significantly lower than the modern data-set (Table 3).

Figure 1b demonstrates that for tibial CT both archaeological samples exhibit severe deficiencies throughout the growth period, with the majority of individuals from both sites falling below the modern line. Tibial CT values for the

two archaeological sites were significantly lower than the modern data-set (Table 3). While some individuals from both sites appear to have either reached or exceeded modern values by 15-17 years of age, there are still some individuals demonstrating notable deficiencies in tibial CT at this stage. There is no significant difference in tibial CT between the Coach Lane and Bow Baptist samples (Table 3)

For vertebral body height, again there are no significant differences between the two sites (Table 3), however the Bow Baptist sample have slightly lower values for body height between 15-17 years of age (Fig.1c). At 17 years of age the Bow Baptist sample has only reached 81% of the adult average measurement, suggestive of an extension of growth into early adulthood (Fig.1c). This is also true for the Coach Lane sample, which at 15 years of age is marginally higher at 84% of the adult average measurement (Fig.1c).

Lastly, as can be seen in Figure 1d, Coach Lane and the Bow Baptist sample demonstrate severe deficiencies in TR diameter when compared to modern adult and non-adult data, this difference is statistically significant (Table 3). The Bow Baptist sample has generally lower values for TR diameter than Coach Lane, showing notably severe deficiencies in the first three years of life (Fig.1d). This difference is not statistically significant (Table 3). This may be reflective of the small sample size for Coach Lane for this growth parameter.

Table 3 – *ANCOVA results for measurements of tibial diaphyseal length, tibial CT, vertebral body height (BH) and transverse diameter (TR) from the two archaeological samples, and compared to modern data. p=<0.001, significant values in bold.*

ANCOVA	**Tibial length**		**Tibial CT**		**BH (C5-6)**		**TR (C5-6)**	
	F	*p*	F	*p*	F	*p*	F	*p*
Coach Lane vs Bow Baptist	0.296	0.588	0.144	0.706	1.633	0.206	4.952	0.032
Archaeological vs Modern	8.598	**0.000**	8.836	**0.000**	-	-	20.039	**0.000**

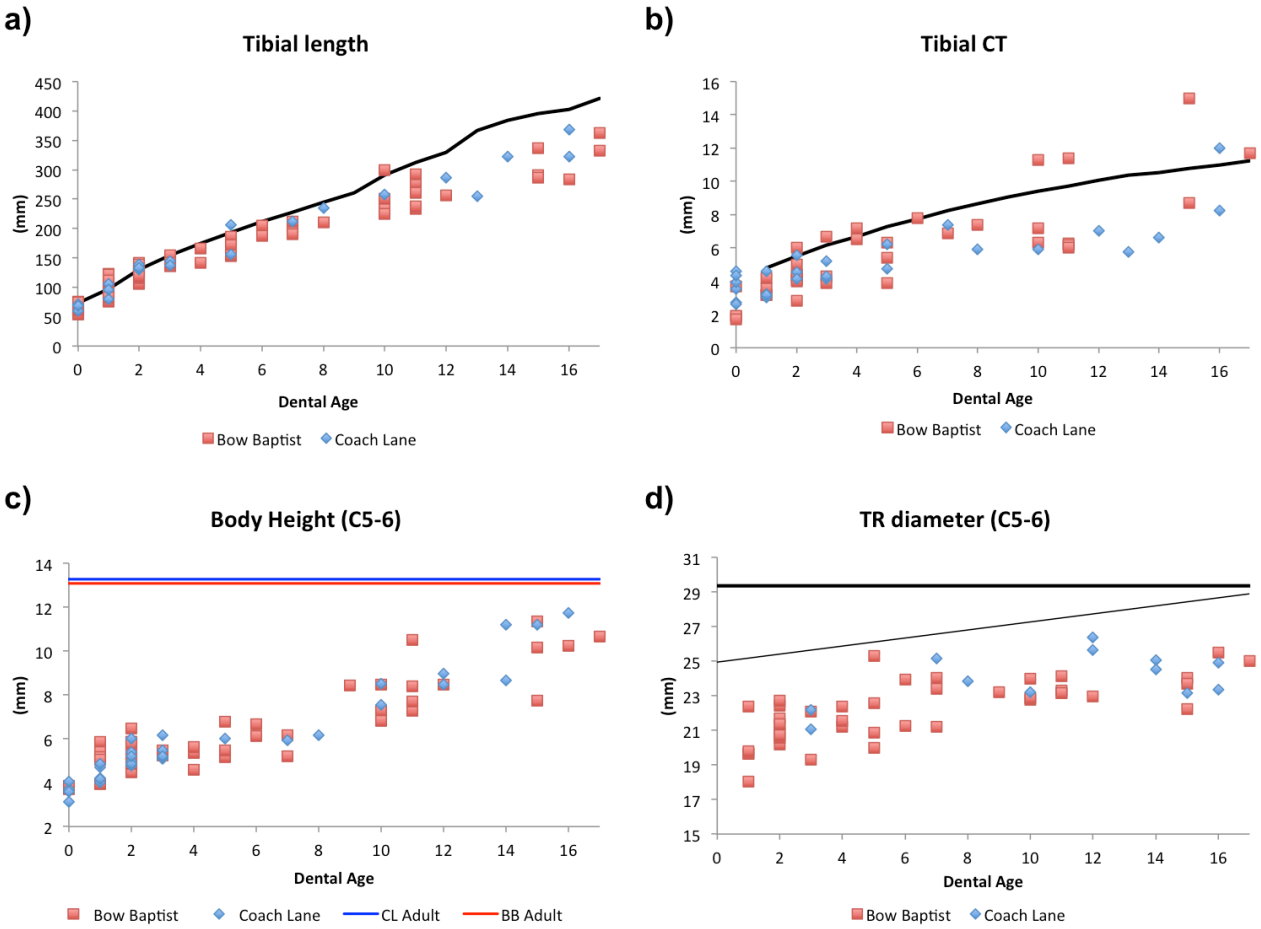

Figure 1 – *Growth profiles for inter-site comparisons; a) Tibial length - comparative modern data represented by solid black line, taken from Maresh, 1955; b) Femoral CT – comparative modern data represented by solid black line, taken from Virtama and Helelä, 1969); c) Body height measurements for C5-6, plotted with average adult measurements for each site; d) Transverse diameter for C5-6, comparative modern adult data (solid black line) and modern non-adult data (solid grey line) taken from Hinck et al. (1966).*

Prevalence of pathological indicators of stress

Of the entire non-adult sample for both sites (Coach Lane = 82 and Bow Baptist = 202), 73% and 52% of non-adults from Coach Lane and the Bow Baptist groups, respectively, demonstrated one or more indicators of stress. A breakdown of the prevalence of pathology by age category within the overall non-adult sample can be seen in Table 4. Metabolic disease peaked for both sites between 1-11 months and 1-5 years, however its prevalence was significantly higher in Coach Lane (62% compared to 23%) (Table 4). Prevalence of cribra orbitalia was more equally weighted between the two sites (at 31% in Coach Lane and 39% in Bow Baptist), and also peaked between 1-5 years. Prevalence of both DEH and Harris' lines were more prevalent in Coach Lane (at 27% and 58% respectively, compared to 15% and 29%), and peaked between 1-5 years of age.

Table 4 – *Breakdown of pathological indicators by age category. Percentage of non-adult population in brackets. HL presented as a percentage of non-adults radiographed. CO presented as percentages of non-adults with orbits/dentition present for analysis.*

	Site	Foetal + Perinatal	1-11 mths	1-5 years	6-11 years	12-17 years	Overall	Chi^2 output
Metabolic	Coach Lane	7 (8.5)	**11 (13.4)**	**21 (25.6)**	3 (3.7)	9 (11)	51 (62.2)	**18.263**
	Bow Baptist	4 (2)	**18 (8.9)**	**21 (10.4)**	2 (1)	1 (0.05)	46 (22.8)	**p<0.001**
Rickets	Coach Lane	0 (0)	5 (6.1)	**15 (18.3)**	3 (3.7)	7 (8.5)	30 (36.6)	7.342
	Bow Baptist	1 (0.5)	**13 (6.4)**	**18 (8.9)**	1 (0.5)	1 (0.5)	34 (16.8)	0.01<p> 0.005
Scurvy	Coach Lane	**7 (8.5)**	**8 (9.8)**	**8 (9.8)**	0 (0)	2 (2.4)	25 (30.5)	9.878
	Bow Baptist	3 (1.5)	10 (5)	**7 (3.5)**	1 (0.5)	0 (0)	21 (10.4)	0.002 <p> 0.001
Cribra Orbitalia	Coach Lane	0 (0)	0 (0)	**9 (17.3)**	3 (5.7)	4 (7.7)	16 (30.8)	1.008
	Bow Baptist	1 (0.7)	10 (7)	**30 (21)**	9 (6.3)	6 (4.2)	56 (39.2)	0.975<p>0.20
DEH	Coach Lane	0 (0)	2 (2.4)	**7 (8.5)**	4 (4.9)	**9 (11)**	22 (26.8)	3.141
	Bow Baptist	0 (0)	0 (0)	8 (4)	**13 (6.4)**	**10 (5)**	31 (15.3)	0.10<p>0.05
Harris' Lines	Coach Lane	0 (0)	0 (0)	**9 (34.6)**	3 (11.5)	3 (11.5)	15 (57.7)	9.656
	Bow Baptist	0 (0)	0 (0)	**7 (13.5)**	4 (7.7)	4 (7.7)	15 (28.8)	0.002 <p> 0.001

Growth and pathology

In order to facilitate a growth comparison between those who demonstrated evidence of pathology versus those that did not (stressed vs. unstressed individuals), each individual within the Coach Lane and Bow Baptist samples was scored based on the number of stress indicators that they exhibited. Therefore each individual fell into one of the following categories – one indicator of stress, two indicators of stress, three indicators of stress, four indicators of stress, or no indicators of stress. Each growth parameter was again plotted for Coach Lane and the Bow Baptist samples (separately), to enable comparison of the above pathological categories within each site (see Figure 2).

As can be seen in Figure 2a and 2e, all individuals demonstrate similar values for tibial diaphyseal length regardless of number of stress indicators exhibited for the Coach Lane and Bow Baptist. However, for tibial CT those with two or more stress indicators tended to have lower growth values than those with

only one in the Coach Lane sample, particularly after eight years of age (Figure 2b). It must be acknowledged that only one individual demonstrated 'no indicators of stress' in this sample. For the Bow Baptist sample, the results were more mixed, with individuals demonstrating two stress indicators in general having lower CT values beyond 8 years of age, some individuals with multiple stress indicators exceeding modern values, and some with "none" appearing to be severely deficient (Fig.2f). Vertebral body height did not show any strong association with number of indicators of stress (Fig.2c and 2g). However, some individuals with two indicators of stress in Bow Baptist group, and three indicators of stress in Coach Lane, did demonstrate greater deficiencies in body height (Fig.2c and 2g). Lastly, for TR diameter, while it is difficult to place too great an emphasis on the results seen in the Coach Lane sample for TR diameter due to the small sample size, those with two or three indicators of stress did demonstrate lower growth values than the one individual with only one indicator of stress (Fig2.d). For the Bow Baptist group, those with two and three indicators of stress demonstrated some of the lower growth values beyond five years of age. However in the first five years of life the results were more mixed, and even those with 'no indicators' demonstrated severe deficiencies in TR diameter (Fig.2h).

It is of interest that for the four growth parameters, the majority of individuals beyond approximately 6-8 years of age all had at least one stress indicator. Although this made comparison with the growth of "unstressed" individuals problematic, it in itself is also suggestive of the cumulative impact of stress in the later growth period.

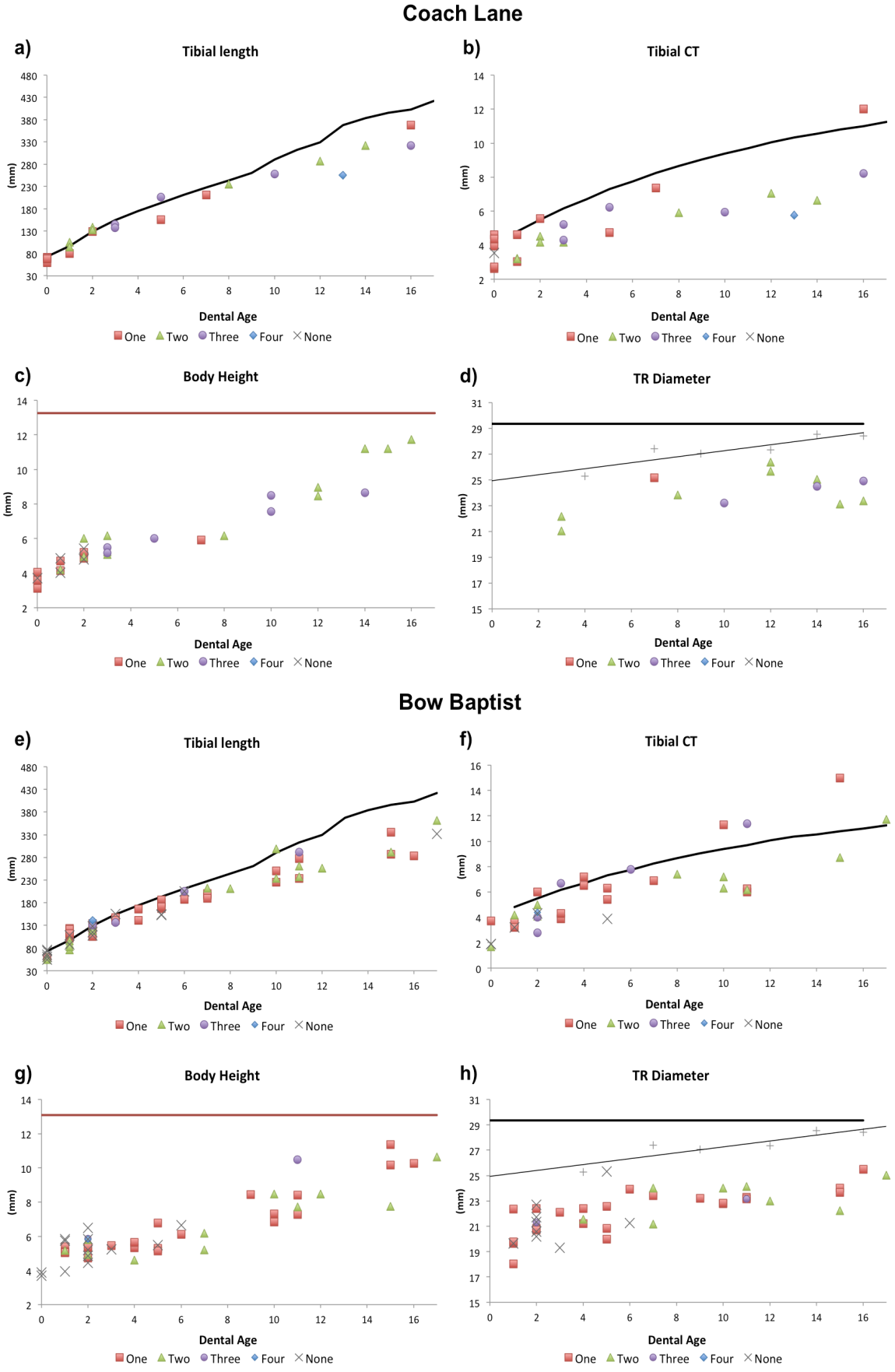

Figure 2 – *Growth profiles for the Coach Lane and Bow Baptist sites for "stressed" and "unstressed" individuals. Solid lines represent comparative data outlined in Figure 1.*

Co-morbidity

In the above analyses, it was established that the presence of multiple indicators of stress did have an impact on the growth patterns described for the Coach Lane and Bow Baptist populations for tibial CT and TR diameter. Therefore, the frequency of co-morbidity between the four stress indicators was determined to identify which conditions most commonly co-occurred within each site.

Overall, 60 non-adults within the Coach Lane sample, and 105 non-adults within the Bow Baptist sample, demonstrated one or more indicator of stress. Within these "pathological samples" 50% and 67% had just one indicator, 33% and 27% had two indicators, 15% and 6% had three indicators, and 2% and 1% had all four indicators (Coach Lane and Bow Baptist respectively).

Figure 3 was constructed to demonstrate the frequency of each type of stress indicator, and the frequency in which they occurred together (their co-morbidities), as a percentage of the "pathological sample" of each site. "Metabolic disease" was by far the most prevalent condition within Coach Lane, followed by DEH, cribra orbitalia, and then Harris' lines. The most commonly occurring co-morbidities were between metabolic disease and DEH (15%), and metabolic disease and cribra orbitalia (8%). When individuals from this site demonstrated three indicators of stress, it was most likely to be a combination of cribra orbitalia, metabolic disease, and DEH (at 8%) (Fig.3). For the Bow Baptist sample, while the most prevalent condition was indeed metabolic disease, followed by cribra orbitalia, and then DEH, these conditions were more equally weighted in terms of their influence on the health of the non-adult sample (Fig.3). The most common co-morbidities for the Bow Baptist sample were between cribra orbitalia and metabolic disease (10%) and cribra orbitalia and DEH (10%) (Fig.3). The most common occurrence of three stress indicators was again a combination of metabolic disease, cribra orbitalia, and DEH (3%) (Fig.3).

Thus, metabolic disease, cribra orbitalia, and DEH have been identified as having a strong impact on the health of the non-adults within this study, and will be assessed alongside the growth parameters to see if any of these individual conditions played a particularly prominent role in influencing growth disruption. Tibial diaphyseal length was omitted from further study, as there was no clear relationship between the presence of stress indicators and growth disruption.

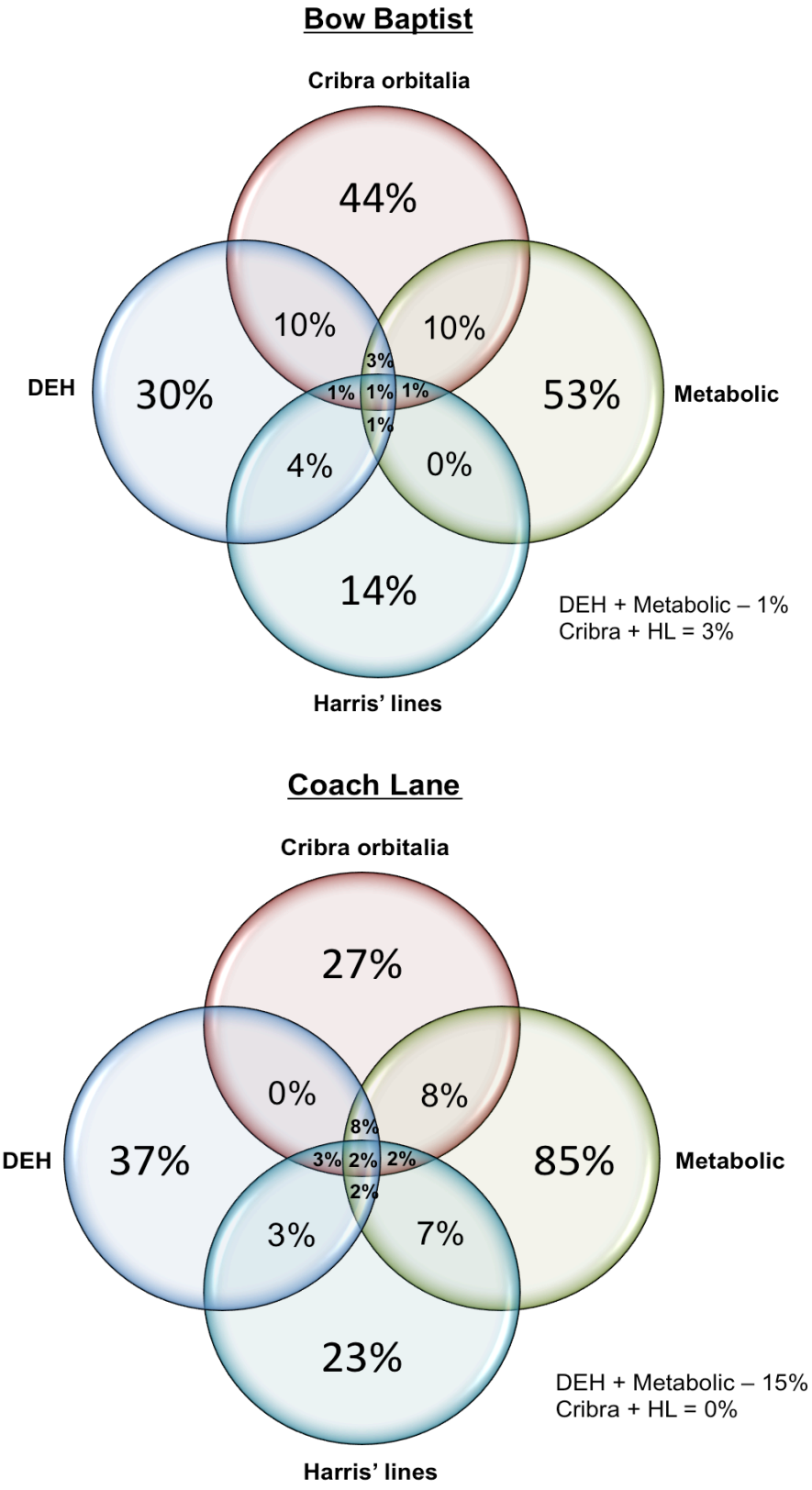

Figure 3 – *Co-morbidity of stress indicators within the Coach Lane and Bow Baptist sites. Prevalence calculated as a percentage of those within each sample demonstrating one or more indicator of stress.*

"Metabolic disease"

Each growth parameter was re-plotted for both sites, and individuals categorised into one of the following groups – "scurvy", "rickets", "both", or "none". As can be seen in Figure 4 sample sizes for those with metabolic disease in the later growth period are very small, which prevents any conclusive inferences regarding the effect of metabolic disease on growth being made. However, there are some interesting observations.

For tibial CT some of the lowest values in the first two years of life for both sites belong to those with "scurvy", "rickets", or "both" (Fig.4a and 4b). While for the Bow Baptist sample the three individuals at 6, 8, and 11 years of age with metabolic disease do not appear to differ in CT values from those without (Fig.4b), in the Coach Lane sample two individuals aged 13 and 16 years with rickets have extremely deficient CT values when compared to the modern data (Fig.4a). However, another individual with rickets aged 16 exceeds modern values (Fig.4a).

There is no obvious relationship between vertebral body height and metabolic disease (Fig. 4c and 4d), with the exception of one individual with rickets in the Coach Lane sample with a particularly low growth value at 14 years of age. However, for TR diameter in the Coach Lane sample the lowest values tended to be associated with those with "rickets", "scurvy", or "both" (Fig.4e). This pattern is not evident in the TR diameters for the Bow Baptist sample (Fig.4f). However, it must be acknowledged that there is a poor representation of individuals with metabolic disease within the growth sample for the Bow Baptist collection.

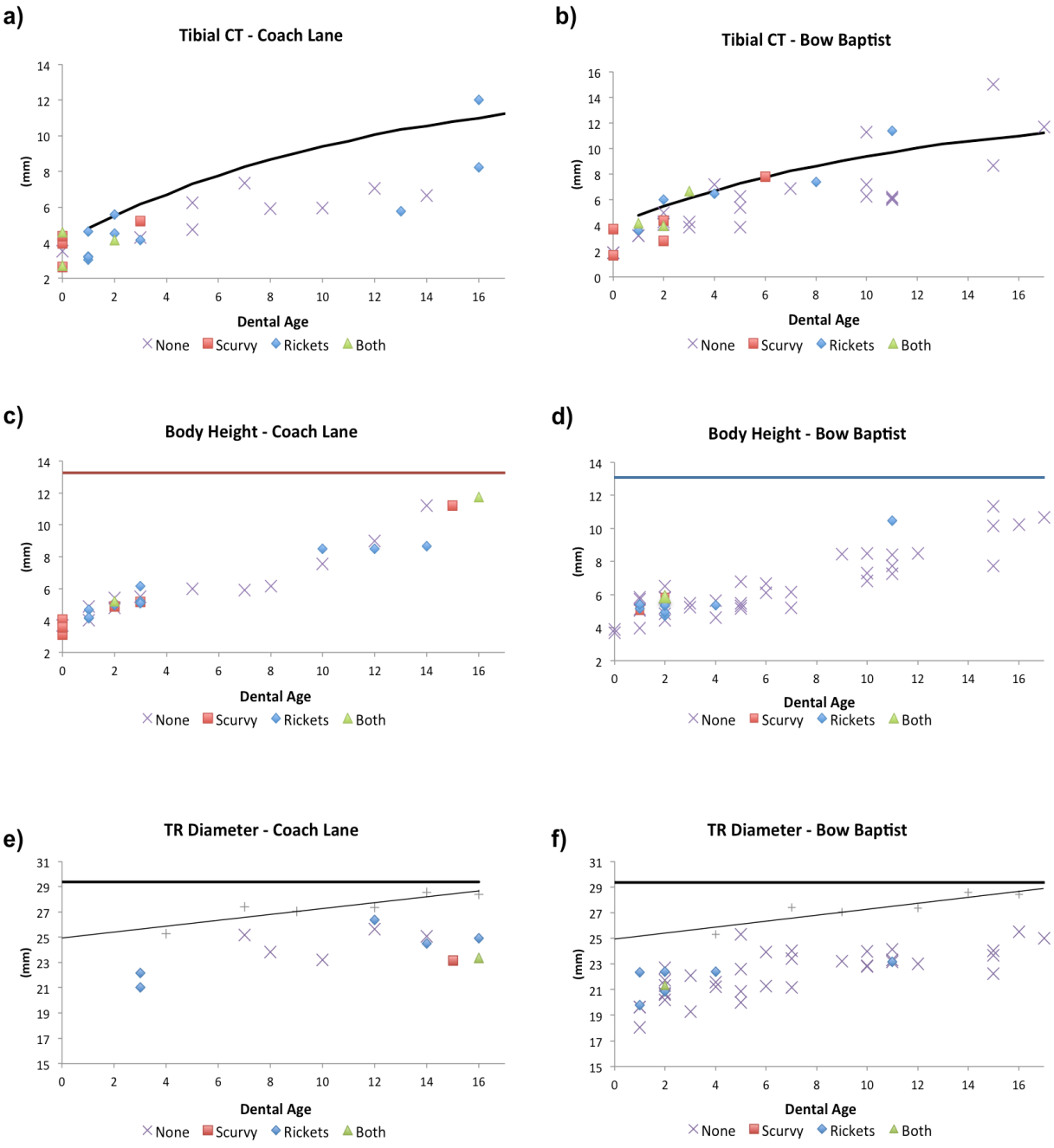

Figure 4 – *Association between metabolic disease and growth within each site. Individuals grouped into the categories "none", "scurvy", "rickets", and "both". Solid lines represent comparative data.*

DEH

The above process was repeated for DEH, with individuals being placed into the categories – "DEH" or "none", based on presence or absence of this stress indicator. As can be seen in Table 4, DEH is most prevalent between 1-5 years (and beyond), and was in general absent in the earlier growth period. This is also reflected in Figure 5, with a clear divide occurring at around 3-5 years of age between those without DEH in infancy and those with DEH for the remainder of the growth period. This is tied into the timings of crown development and dental eruption in non-adults, so is expected, however it is of interest that individuals with DEH tended to exhibit some of the lowest growth values between 8-17 years of age for tibial CT and TR diameter in both the Coach Lane and Bow Baptist samples (Fig.4a, 4b, 4e, and 4f).

Cribra orbitalia

Growth data for individuals with cribra orbitalia (stage 1 and above) were compared to those without cribra orbitalia (stage 0). For both sites there were no notable differences between those with and without this condition for tibial CT and vertebral body height (Fig.4a,b,c,d), however some of the lowest CT values in Coach Lane between 8-13 years of age belonged to individuals with cribra orbitalia (Fig.4a). For the Bow Baptist sample, some of the lower range measurements for vertebral body height between 4-7 years of age were also in those with cribra orbitalia (Fig.6d). However, for TR diameter those with cribra orbitalia did demonstrate some of the lowest values throughout the growth period when compared to those with no skeletal evidence for this stress indicator (Fig.6e,f). This is particularly evident in the first five years of life for the Bow Baptist sample, and at ages three, eight, and 10 for Coach Lane (Fig.4e,f).

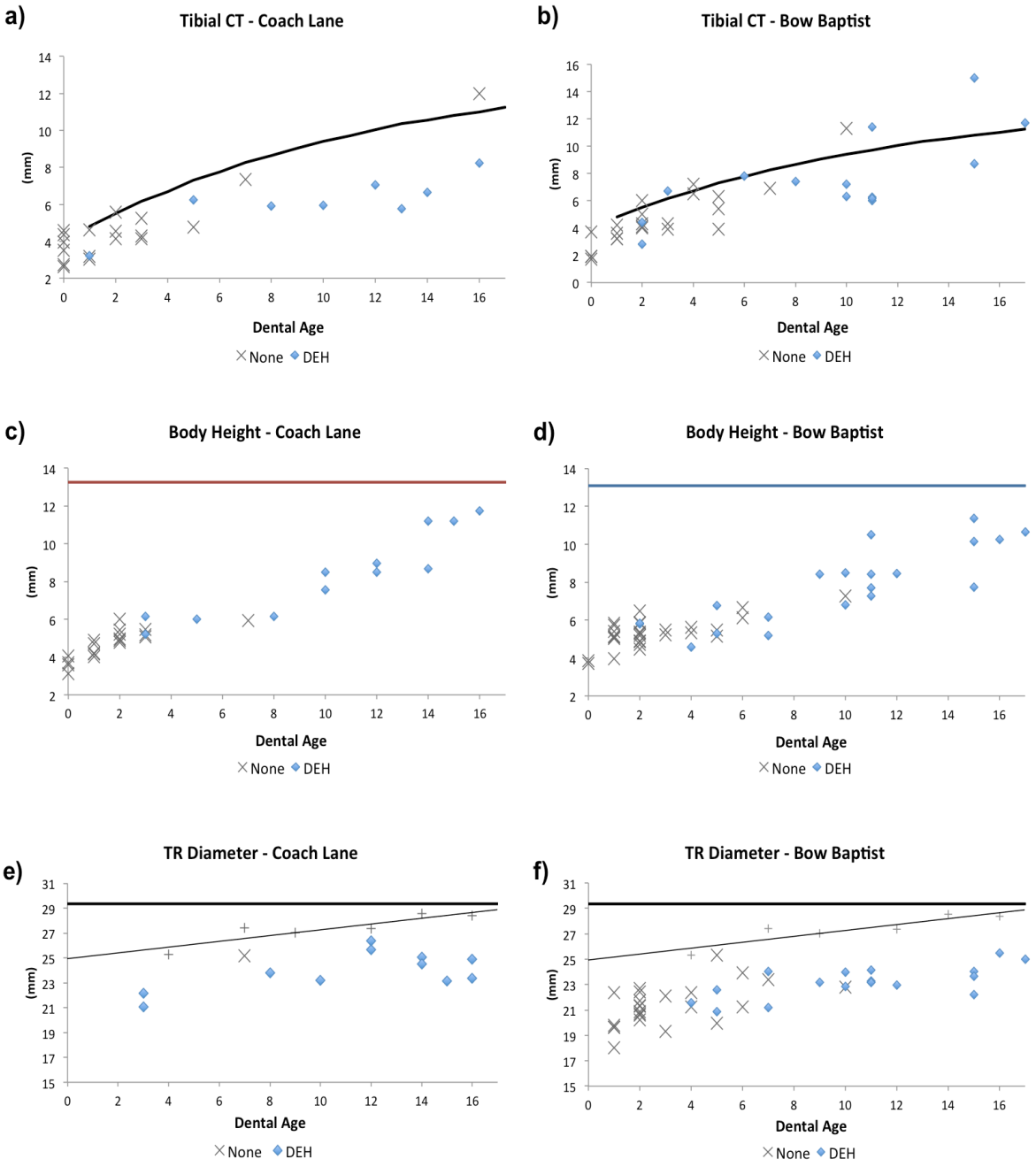

Figure 5 – *Association between DEH and growth within each site. Individuals grouped into the categories "none", and "DEH". Solid lines represent comparative data.*

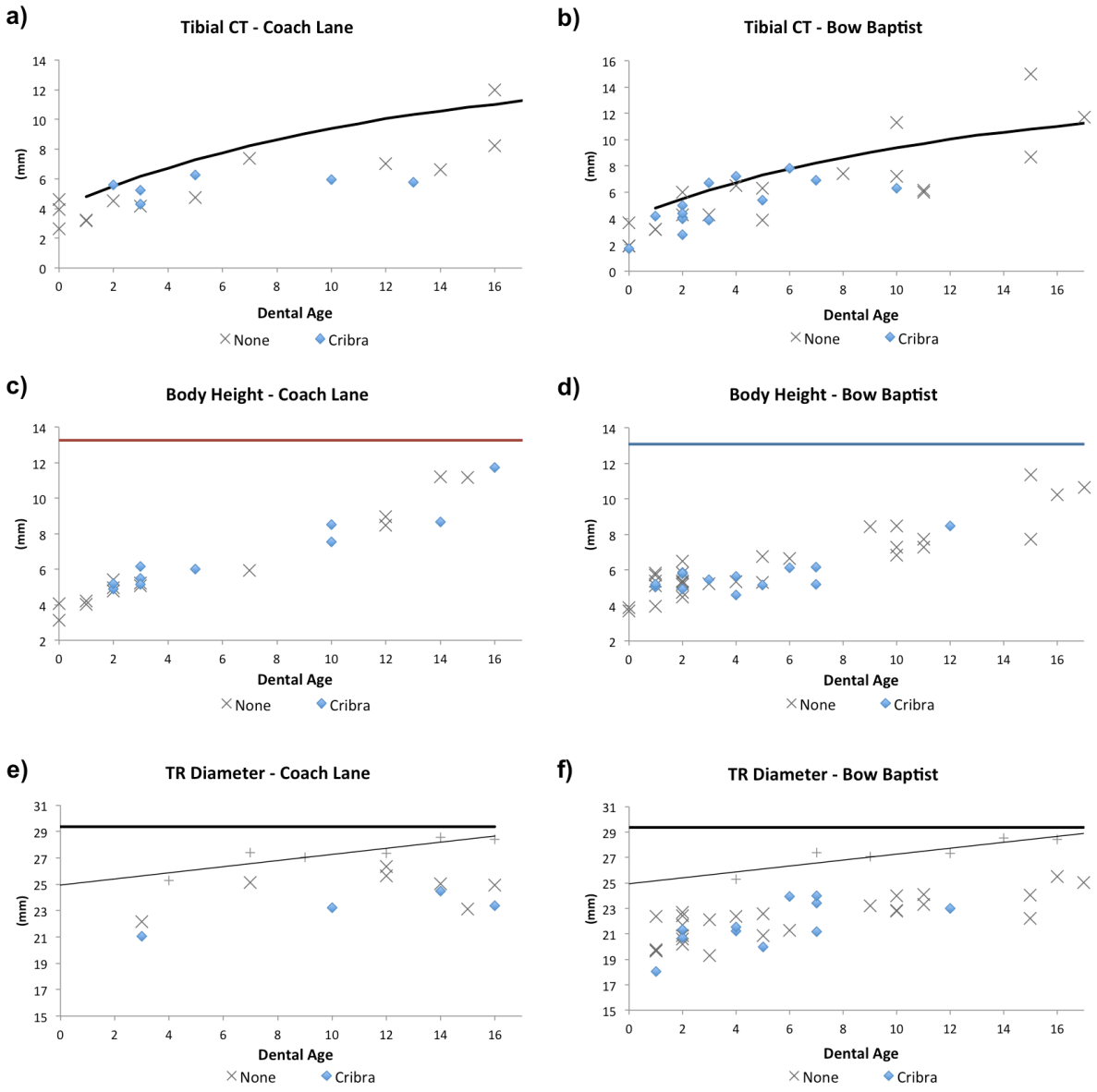

Figure 6 – *Association between cribra orbitalia and growth within each site. Individuals grouped into the categories "none"and "cribra orbitalia". Solid lines represent comparative data.*

DISCUSSION

"He is a town-made article of small stature and weazen features" – Description of Young Smallweed ("Small"), aged approximately 15 years – Bleak House, Charles Dickens (1853: 193)

The inter-site comparisons reveal that there are no statistically significant differences in growth between the two archaeological sites in this study (Table 3). However, both sites revealed significant differences in tibial diaphyseal length, femoral CT, and TR diameter when compared to the modern data (Table 3). Therefore, considering the similarities in growth between the two sites, it is of interest to determine whether the disruptions evident when compared to modern data differ in origin (using pathological data), and can therefore be traced to differences in historical context.

Urban centres of the 18th and 19th centuries came with a multitude of health risks, especially to the very young. Poor sanitation, unprecedented levels of air pollution, and insufficient sewerage and waste disposal all combined to breed an infectious environment in which the spread of disease was rife (Engels, 1950; Hardy, 1993). In addition, social influences such as fashionable, but ultimately detrimental, child-care practices in infancy, the long hours spent indoors due to child labour practices, and insufficient access to adequate nutrition meant that children were highly susceptible to metabolic diseases such as rickets and scurvy (Hardy, 1992; Gibbs, 1994; Kirby, 2013). While it cannot be said with certainty that the non-adults within this study participated in child labour practices, it was likely that children of the lower to middle classes would have been involved in some form of occupational activity (whether within the home, within a family business, or within an apprenticeship) that would have limited time spent out of doors (Earle, 1989). In addition, it has been suggested by Kirby (2013) that many of the severe deficiencies in health in child labourers, particularly rickets, were not exclusively associated with occupation for long hours in factories, but more likely began at home. Therefore, the high prevalence of metabolic disease within these two samples (at 62% and 23% within Coach Lane and the Bow Baptists) is of no surprise. These two sites also demonstrated evidence of anaemia (indicative of an insanitary living environment, high pathogen load, and/or dietary deficiencies), and DEH (often associated with episodes of poor health and malnutrition) (Stuart-Macadam, 1991;

King et al., 2005; Ogden et al., 2007; Hillson, 2008; Walker et al., 2009; Oxenham and Cavill, 2010). The summary pathology data, alongside the often severe deficiencies seen in the growth data when compared to modern "healthy" data-sets, indicates that the non-adults of both sites were exposed to the well-documented detrimental conditions of industrial centres at this time.

The primary aim of this study was to determine whether pathological and growth data can be combined to form a more directed interpretation of the growth disruption seen in past populations. It is clear that the Coach Lane sample experienced a higher prevalence of stress indicators in general than the Bow Baptist sample (Table 4), and it was "metabolic disease" that had the greatest influence on health status for this population (Fig.3). Additionally, individuals with scurvy, rickets, or both within the Coach Lane sample in general showed deficient growth values for tibial CT and TR diameter. For the Bow Baptist sample, the only stress indicator that exceeded Coach Lane in prevalence was cribra orbitalia (at 39% and 31% respectively). The prevalence rates of cribra orbitalia, DEH, and "metabolic disease" were more equally weighted within this site. The most common co-morbidities within the Bow Baptist sample were associated with cribra orbitalia, and lower growth values for tibial CT and TR diameter within this site tended to be more associated with DEH and/or cribra orbitalia.

Therefore, within the Coach Lane sample, the overall health and growth of non-adults was primarily affected by "metabolic disease", and its co-morbidities with cribra orbitalia and DEH. However, within the Bow Baptist sample the results were more indiscriminate, with equal weighting between the three categories of stress indicator, and with more emphasis on cribra orbitalia and its co-morbidities. It is possible that the health patterns seen in the Bow Baptist population reflect the exposure of growing children to the hazards of life in urban centres of the 18th and 19th centuries. Diet was notoriously poor in London, and such dietary deficiencies, when combined with the highly pathogenic environment, would have inevitably led to conditions such as anaemia, and episodes of infection and malnutrition (Shammas, 1984; Snow, 2003; Horrell and Oxley, 2012). Whereas for Coach Lane, while this population likely experienced the same exposure to the deficiencies of urban living, perhaps social factors (such as child-care practices, or occupational activities, which kept children indoors) lead to the higher prevalence of metabolic

disease amongst this sample (Burnett, 1984; Kirby, 2013). As deficiencies in vitamin D are thought to be associated with impairment to immune function (Holick, 2003; Holick, 2004), this may have also left these non-adults highly susceptible to further episodes of poor health.

As well as revealing variances between the two sites in terms of health stress, the results also highlight important differences between the growth parameters implemented in this study. Tibial diaphyseal length and vertebral body height were less likely to show differences between "stressed" and "unstressed" individuals. Both of these measurements continue to increase throughout the growth period, with diaphyseal length of the tibia in general being complete between approximately 18-21 years of age, and vertebral body height typically completing growth between 18-24 years of age (Bick and Copel, 1950; Bogin, 1999; Scheuer and Black, 2000; Duren et al., 2013). Therefore, it is possible that many of the episodes of growth faced by non-adults throughout development are veiled by catch-up growth in these growth parameters. The ability of the long bones to undergo catch-up growth following times of stress is well documented (Eveleth and Tanner, 1990; Mays et al., 2008), however, it is an interesting observation that this may also occur in the vertebral column. A recent study has already suggested that this may occur, and noted general deficiencies in vertebral growth for this population between 9-16 years of age (Newman and Gowland, 2015). Despite the growth disruption seen in the later stages of the growth period for vertebral body height in the Bow Baptist sample, this group still maintain similar average adult measurements for body height to Coach Lane (Fig.1c).

Measurements for CT and TR diameter consistently proved to be more sensitive to the effects of stress on growth, as seen in the tendency for "stressed" individuals to demonstrate lower growth values. It has been proposed that CT measurements may be more sensitive indicators of stress than measurements of long bone length, suggesting that catch-up growth in the long bones may occur at the expense of cortical thickness (Mays et al., 2009a; Stulp and Barrett, 2014). The recovery of appositional growth following instances of malnutrition and/or infection is slow; therefore deficiencies in this growth parameter are more likely to continue throughout the growth period (Hummert, 1983). In addition, the majority of growth in TR diameter is complete at the time of fusion of the spinous process between

approximately 1-2 years of age, effectively "locking in" evidence of early growth disruption for the remainder of the life course, with little opportunity for growth recovery (Jinkins, 2000; Scheuer and Black, 2000; Watts, 2013a,b).

Figure 5 demonstrates that from approximately six years onwards, the majority of individuals within the growth sample demonstrated DEH, and for tibial CT in particular this stress indicator was also often associated with severely deficient growth values for both sites. As crown development of the permanent dentition begins from as early as six months of age, and completes around eight years of age, DEH within the permanent dentition is indicative of episodes of stress occurring during infancy and early childhood (Goodman and Rose, 1990). Therefore, it is of interest that adolescent individuals with DEH tended to exhibit some of the lowest growth values for tibial CT and TR diameter in both the Coach Lane and Bow Baptist samples. That individuals with DEH still show growth deficiencies later in the growth period suggests that early life stressors, such as malnutrition, poor health, and weaning stress, can continue to influence the growth trajectory for the remainder of the developmental period, or confer a degree of frailty in the individual that leaves them susceptible to future health insults.

Many of the older individuals within the growth samples demonstrating one or multiple indicators of stress also often had severely deficient values for TR diameter. As this growth parameter fuses between 1-2 years of age, no significant increases in size can occur beyond this age, and not all of the indicators of stress present in the older children/adolescents will have necessarily developed prior to fusion. That they still show such deficiencies may also indicate the influence of early life stressors on susceptibility to future health insults. Infancy is the most vulnerable time in the life course, with risks of mortality and morbidity high (Lewis, 2007; Gowland, 2015). Disease load and nutritional stress during infancy was exceedingly high during the 18th and 19th centuries, with 42% of deaths in 1837-1838 being in those under the age of five (Registrar-General, 1839). This may also explain why there is often little stratification in growth values between "stressed" and "unstressed" individuals prior to two years of age for TR diameter, as all those dying at this stage in the life course were likely under extreme stress, leading to premature mortality.

The prevalence of Harris' lines, and its interactions with growth and the other stress indictors, has been largely ignored within this study. The aetiology of these "indicators of stress" is somewhat controversial, with suggestions that these lines of growth arrest are more likely a consequence of normal growth patterns and spurts (Papageogopoulou et al., 2011; Alfonso-Durruty, 2011). Their appearance does not always show a consistent correlation with stressful events. Many studies have failed to detect a clear relationship between the occurrence of these lines in individuals who have suffered severe illness or malnutrition, and they have also been observed in healthy children (Nowak and Piontek, 2002; Alfonso et al., 2005; Papageorgopoulou et al., 2011; Alfonso-Durruty, 2011). However, in a recent study by Geber et al. (2014) Harris' lines were highly prevalent amongst children of the Great Irish Famine (1845-1852), indicating the occurrence of periods of intense catch-up growth amongst those suffering from malnutrition. Therefore, it was concluded that in the famine context, Harris' lines should not be overlooked as an indicator of stress in past populations (Geber, 2014).

Within this study there does appear to be a degree of co-morbidity of Harris' lines with DEH, metabolic disease, and cribra orbitalia (see Figure 3), which may be indicative of a correlation with episodes of stress. However, it is possible that these associations are coincidental, resulting from its common occurrence within growing individuals (Alfonso-Durruty, 2011). We can rarely gain a true observation of the prevalence of Harris' lines within a whole sample, as time constraints and accessibility of radiographic equipment necessitate sub-samples to be made, which may skew data. In addition, while Figure 3 may indicate that there was a higher prevalence of Harris' lines within the Coach Lane sample, it must be noted that there was also a relatively high association of this indicator with metabolic disease. "Scurvy lines", and "white lines of Frankel" are characteristic radiographic indicators of vitamin C deficiency (Brickley and Ives, 2008). It is possible that when growth recommences following reintroduction of vitamin C into the diet, these lines will remodel and take on the appearance of Harris' lines (Stark, 2014). In such instances, Harris' lines may therefore be indicative of episodes of stress. It is clear that more research is required on the aetiology of this indicator, and its association with conditions such as scurvy, to evaluate its continued use as an indicator of stress in bioarchaeology.

While this study has revealed some interesting patterns in terms of health stress and growth disruption, some limitations must be considered. As can be seen in Table 3, all of the individuals within this study, whether deemed to be "stressed" or "unstressed", exhibited significant deficiencies in growth parameters when compared to modern data. All of the individuals within this study were "non-survivors"; therefore even those without any overt manifestation of stress indicators were under some form of health stress that led to their premature death (Wood et al., 1992). It has also been suggested that active vs. inactive indicators of stress is of great importance when considering health patterns within skeletal assemblages, as those where remodelling has occurred survived the episode of stress, whereas those with active lesions succumbed to it (Pinhasi et al., 2013). Pinhasi et al. (2013) found that non-adults with healed cribra orbitalia or periosteal new bone formation demonstrated larger long bone dimensions when compared to those with active lesions. Therefore it is necessary to further develop the techniques used within this study in reference to the prevalence of inactive and active lesions, and their impact on growth.

Sample sizes within this study were also low, and were further reduced by the splitting of growth data into pathological categories. As a consequence, statistical analysis for many of the data comparisons within this study was not possible; therefore any differences observed cannot be validated in terms of significance. While nuances in growth patterns in relation to pathology were indeed evident, further study on larger assemblages is necessary to fully evaluate the benefits of combining growth and pathology data in this way.

CONCLUSION

It is impossible to gain a full picture of the experience of childhood health through growth parameters alone, but the combination of multiple indicators of stress, and the assessment of co-morbidity, may reveal which factors were of greatest influence within a skeletal sample. Results of this study revealed greater pressures placed on the non-adults of Coach Lane due to a heightened prevalence of metabolic disease within the sample. This may have resulted from social practices of the time (related to child care and/or child labour) that propagated deficiencies in vitamin D and vitamin C, and led to susceptibilities to further health insults from the

poor environmental conditions of industrial centres. Whereas in the Bow Baptist sample, the stronger influence of cribra orbitalia and DEH is more suggestive of the generalised influence of dietary deficiencies and the insanitary urban conditions of London on child health in the 18th and 19th centuries. Therefore, a break down of growth data using indicators of stress in this way can begin to lift the veil of inhibitory factors such as catch-up growth, and the non-specific nature of indicators of stress, on the assessment of child development in the past.

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CHAPTER SIX

Discussion

6.1 Introduction

The previous four manuscripts have each addressed questions relating to the impact of the urban environment, social status, and geographic location on child health, as well as exploring new avenues for the detection of growth disruption and episodes of stress in non-adults within bioarchaeology. This chapter will now synthesise all of the results and conclusions from the four manuscripts in order to facilitate a wider discussion of the impact of industrialisation on child health, as well as the enduring impact of social factors on health during the 18th and 19th centuries. It will also evaluate the techniques currently in use in bioarchaeology, and the feasibility of their application in constructing this type of osteobiographical data.

6.2 Child health and the urban environment

One of the key research questions of this study was whether there were observable differences in the skeletal indicators of poor health in non-adults between urban populations from the North and South of England in the 18th-19th century. Fatality from disease occurred up to four times more frequently in towns at this time (Engels, 1950), and infant mortality was at unprecedented levels (Pinchbeck and Hewitt, 1969; Hardy, 1992). The industrial environment of the 18th and 19th centuries was characterised by the ubiquitous spread of disease, and commonplace instances of malnutrition and undernutrition – '…annually more and more human beings become engulfed in the vortex of mighty London, and disease and death dog the footsteps of the "madding crowd"' (Bowers, 1902:11). As can be seen in Manuscript Three, such detrimental conditions were not confined to London. Northern urban centres such as Newcastle, North Shields, and South Shields were significant centres of industrial development via manufactories, shipping industries, and the coal trade (Butler, 2012; Pre-construct Archaeology, 2012; Raynor *et al*., 2011). Considering the health risks that accompanied life in the cities during this time, it was no wonder that child health suffered so drastically in both the North and the South. All six archaeological sites were significantly

deficient in tibial diaphyseal length, femoral CT, and TR diameter when compared to modern data (see Table 3: Manuscript Three). This reflects the health risks that accompanied life in the city for all children. However, no significant differences were detected between any of the growth parameters in terms of northern and southern comparisons, but instead appear to be related to differences in social status (Table 3: Manuscript Three). Age-at-death rates for each site also reflected the high risks of morbidity and mortality during the vulnerable stage of infancy at this time. Around 42% of total deaths in London between 1837-1838 were those of five years of age and under (Registrar-General, 1839). Accordingly, peak age-at-death for the Coach Lane, Chelsea Old Church, Bow Baptist, and St Benet Sherehog samples occurred between 1-5 years of age (see Figure 2: Manuscript Three). While age-at-death was also high between 1-5 years for Cross Bones and Coronation Street, the peak age-at-death occurred earlier, in the "Foetal + Perinatal" age category. This was suggestive of elevated infant mortality risks amongst lower status groups.

Growth values for tibial diaphyseal length and femoral CT indicated a general deviation away from modern comparative data between 1-5 years of age, and deficiencies in TR diameter also reflected health stress in infancy. These growth deficits, combined with timings of peak prevalence in metabolic disease, may be suggestive of weaning stress from early cessation of breastfeeding, and/or insufficient weaning diets (Lewis, 2002a). As described in Manuscript Two, breastfeeding practices could be highly variable in the 18th/19th centuries, and a falling popularity in breastfeeding often meant substitution with inadequate artificial infant feeds (Buchan, 1778; Wickes, 1953; Fildes, 1995; Crawford, 2010). For Chelsea Old Church, the Bow Baptist sample, and St Benet Sherehog, this peak in prevalence of metabolic disease occurred between 1-5 years, for Coach Lane and Coronation Street between 1 month-5 years, and for Cross Bones from the perinatal period right through to 5 years of age (see Table 4: Manuscript Three). This suggests that the process of weaning may have occurred earlier in Coach Lane, Coronation Street, and Cross Bones. However, it must also be considered that deficiencies in vitamin C and D can be passed on from the mother, therefore may represent inherited deficiencies.

Overall rates of pathology were generally high for most sites (see Figure 5: Manuscript Three). With the exception of Coronation Street, all sites demonstrated

high rates of rickets, which is reflective of the types of child-care practices implemented throughout the social strata that limited exposure to sunlight (see Manuscript Two). A high prevalence of DEH was also notable amongst the sites, particularly for Coach Lane and St Benet Sherehog. This dental defect is associated with episodes of infection and malnutrition occurring during crown development of the dentition, from the second trimester up until approximately eight years of age (Goodman and Rose, 1990). Therefore this too is indicative of exposure to significant health risks during infancy and childhood within urban centres. Pathology in general was much more prevalent within Coach Lane and Cross Bones, which supports the preceding evidence (relating to age-at-death and patterns in growth deficits) that it was factors relating to social status or social practices that had the greatest influence on child health at this time, rather than geographic location, as the industrial environment itself was universally poor.

This raises an important question. When studying archaeological populations from such similar environmental backgrounds as these six groups, how do we begin to identify nuances in health stress when prevalence of pathology was so high overall? All of these sites demonstrated stunted growth in comparison to modern data, and all non-adults in this study are by nature "non-survivors". Therefore within these six sites endogenous and exogenous influences were acting to increase risk of morbidity and mortality (Lewis and Gowland, 2007). Manuscript Four used a combination of growth and pathology data to identify the central causative factors in the high morbidity and mortality demonstrated by the Coach Lane and Bow Baptist groups. By looking at co-morbidity it was possible to pin-point metabolic disease, and therefore subsequent deficits in immune response, as the strongest influence within the Coach Lane non-adult sample. This may have been related to social factors, such as child-care and/or child labour practices, combined with the northern latitude of this site. Whereas for the Bow Baptist group, cribra orbitalia and DEH had a greater influence on child health, indicating a more general response to poor environmental conditions within urban centres. It is therefore of interest to assess co-morbidity patterns within the remaining four sites. Following the methodology set out in Manuscript Four, percentages of individuals with DEH, cribra orbitalia, metabolic disease, and Harris' lines (and co-morbidities)

within the "pathological samples" were calculated. As can be seen in Figure 6.1a, the most prevalent stress indicator for Coronation Street was DEH, with notable co-morbidities with Harris' lines and metabolic disease. However, the extremely low prevalence of pathology within this sample means that these high percentages are somewhat misleading. While it is of interest that DEH (indicative of the presence of early life stressors) was most prevalent, all other associations cannot be assessed with reliability. For Chelsea Old Church (see Fig.6.1b), similar to that seen within the Bow Baptist sample, there seems to be a more equal distribution of prevalence of the four stress indicators. The most frequent co-morbidity was between DEH and cribra orbitalia (at 17%), which is suggestive of poor environmental conditions, deficiencies in diet, and episodes of poor health, alongside contributory factors for metabolic disease (Stuart-Macadam, 1991; King *et al*., 2005; Walker *et al*., 2009;

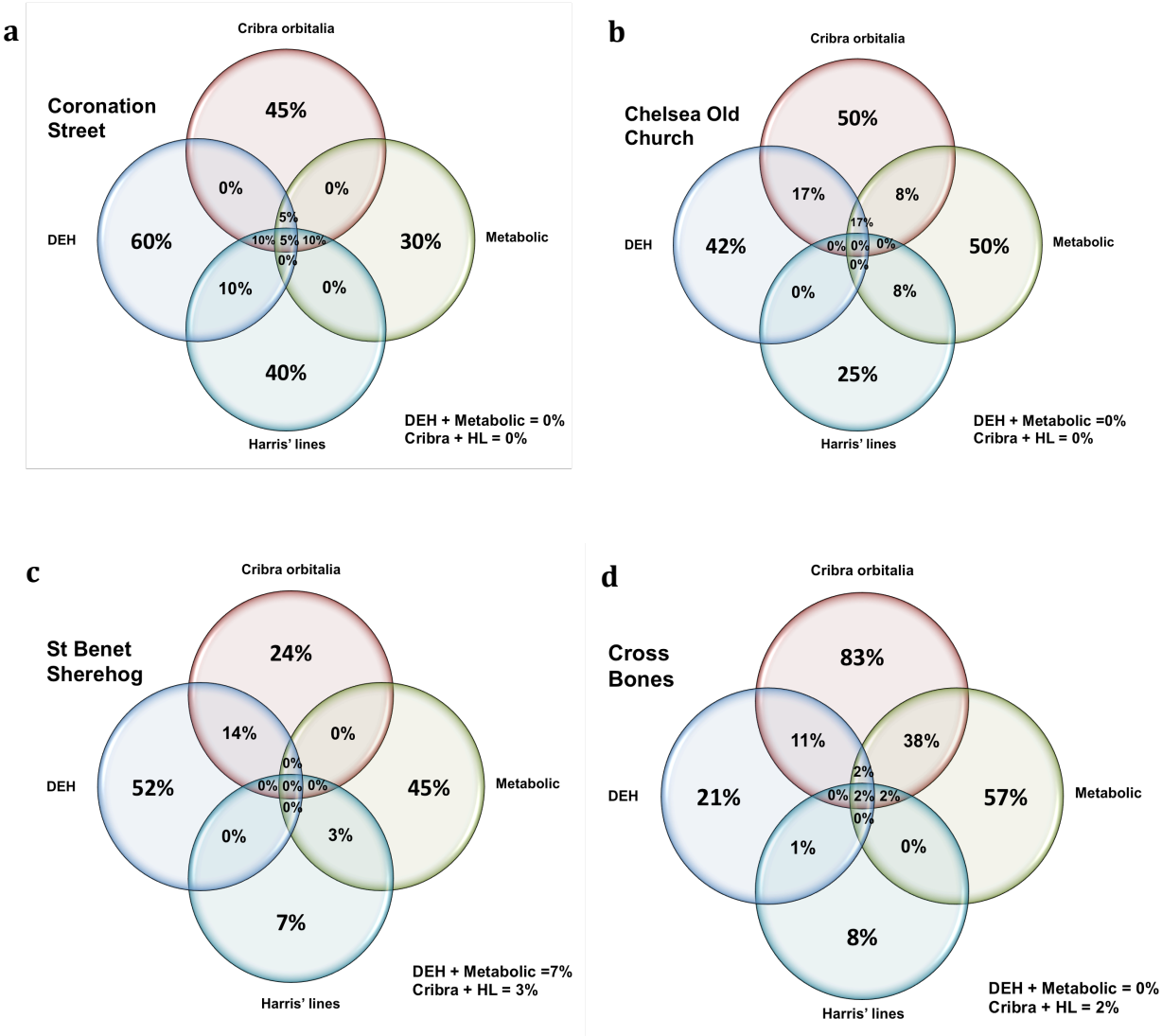

Figure 6.1 – *CPR (%) of co-morbidity of the four stress indicators, calculated as a percentage of non-adults demonstrating one or more indicators of stress within each sample.*

Oxenham and Cavill, 2010). These patterns in health stress may therefore be reflective of the influence of child-care practices implemented within wealthier populations, which frequently resulted in vitamin D deficiencies and inadequate infant diets (Miles *et al*., 2008b; Giuffra *et al*., 2013). A similar pattern is also evident in St Benet Sherehog (Fig.6.1c). However, for Cross Bones the distribution of pathological prevalence slightly shifts (Fig.6.1d). Metabolic disease is again high, however it is cribra orbitalia that is most prevalent (at 83%). The most frequent co-morbidity occurred between cribra orbitalia and metabolic disease, which may be related to the documented association of anaemia and scurvy, related to the haemorrhaging that results from vitamin C deficiency, or a reduced capacity for iron absorption (Weinstein *et al*., 2001; Wapler *et al*., 2004; Baker *et al*., 2010; Agarwal *et al*., 2015; Ferrari *et al*., 2015). Due to the high number of perinates within this sample, it is likely that this pattern is therefore suggestive of poor maternal health, and inherited deficiencies in vitamin C.

The main findings for each site will now be summarised to aid further discussion in relation to child health and the urban environment of the 18th and 19th centuries.

While all sites showed significant deficiencies in TR diameter when compared to modern data, the non-adults of Chelsea Old Church demonstrated significantly lower TR diameters (for vertebral grouping T6-8) when compared to Coronation Street and the Bow Baptists (see Table 3: Manuscript Three). This is suggestive of particularly high risks of exposure to stress during the first two years of life within this sample. As stated in Manuscript Two, such deficiencies in growth as demonstrated by the high status site of Chelsea Old Church are most likely related to fashionable child-care practices and infant feeding strategies in operation during this period. The peak in prevalence of metabolic disease between 1-5 years also supports this. The high prevalence rates of rickets (9%) and scurvy (9%) demonstrated by this sample are indicative of the insufficiencies in weaning diet, early cessation of breastfeeding, and limited exposure to sunlight brought about by such practices as keeping children indoors, and use of paps and panadas (Fildes, 1995; Miles *et al*., 2008b; Crawford, 2010; Giuffra *et al*., 2013). Analysis of co-morbidity of stress indicators within the non-adults of this collection revealed a relatively equal distribution of prevalence, slightly more weighted towards DEH

and cribra orbitalia. The high rate of DEH provides further support for the presence of early life stressors. A study by Millard *et al*. (2014) also revealed that this comparatively wealthier population were exposed to high risks of lead poisoning in childhood, most likely originating from use of vessels coated in lead glazes. Therefore, overall, the growth and pathology data collected for Chelsea Old Church reveals high risks for wealthy children in infancy. However, non-adults from Chelsea Old Church do appear to "catch-up" in growth for tibial diaphyseal length beyond 10 years of age (see Fig.4: Manuscript Two). This suggests that insults to health experienced during infancy may be overcome by social buffering later in childhood. However, due to the cross-sectional nature of cemetery populations, and the small sample size of this site, this should be applied with caution. Higher status children did also experience poor health in infancy, but due to advantages in family income affording a better quality of care, they were more likely to recover with little effect to future well-being (Hardy, 1992).

The non-adult sample of St Benet Sherehog in general demonstrated some of the highest growth values for tibial diaphyseal length, femoral CT, vertebral body height, and TR diameter. However, very few of the growth parameters revealed significant differences between St Benet Sherehog and the remaining archaeological populations, with the exception of this sample having significantly higher values for vertebral body height (T6-8) between 0-12 years of age than Coronation Street, but significantly lower values than the Bow Baptist sample. The non-adults of St Benet Sherehog also demonstrated peaks in metabolic disease prevalence between 1-5 years of age, indicative of weaning stress, however this sample had a comparatively low prevalence rate of scurvy (3%). It is important to note, that it is possible that some of the individuals within this collection date to the 17th century, due to the lengthy cemetery usage of this site. Therefore, that this sample should show comparatively lower rates of pathology, and higher growth values, may be due to differing conditions in the city centre of London prior to the deterioration of the urban environment in the 18th and 19th centuries. However, for tibial diaphyseal length, non-adults of this sample were still not reaching modern values by 16 years of age, at 84% of the average measurement for the Maresh sample. There was also a relatively high rate of DEH (23%), indicative of early life stress, and this group was still significantly deficient in tibial diaphyseal length, cortical thickness, and TR

diameter between 0-12 years of age when compared to modern data. Therefore, despite the potential advantages of this middling status group, growth and pathology data still indicate the influence of health pressures that accompanied urban life, even prior to the 18th century (Porter, 1994).

For the Bow Baptist sample, similar to that seen in the St Benet Sherehog group, results are suggestive of exposure to the detrimental urban environment but also a degree of social buffering due to their middling status. Bow Baptist non-adults between 0-12 years of age had significantly higher tibial diaphyseal lengths than Coronation Street, and significantly larger vertebral body heights (T6-8) than St Benet Sherehog, Cross Bones, and Coronation Street. Therefore, the Bow Baptist sample also showed some of the best growth values. Analysis of co-morbidity revealed that cribra orbitalia and DEH had the greatest influence on growth disruption within this sample, which alongside a peak in metabolic disease between 1-5 years of age, may be indicative of early life stress related to weaning practices. However, the low rate of scurvy (3%) within this sample means that the very high rate of cribra orbitalia amongst the non-adults (28%) may be more related to high pathogen load associated with insanitary environmental conditions, alongside nutritional deficiencies (Walker *et al.,* 2009). The village of Bow became increasingly industrialised during the 18th and 19th centuries, so it is possible that the increasing levels of pollution and declining sanitary conditions had an inevitable impact on child health, despite social buffering. The London diet was also notably poor during this time compared to other regions within the country (Horrell and Oxley, 2012). On analysis of vertebral body height in Manuscript One, a lag in growth was identified between 9-16 years of age, this is also evident in Figure 3 of Manuscript Two, and Figure 4 of Manuscript Three. It was suggested in Manuscript Three that this growth delay in adolescence is likely due to an increase in energetic demands alongside nutritional deficiencies (Duren *et al*., 2013). During this period of the growth process a much higher intake of protein, calcium, vitamin D, and calories is required to support bone growth and development during the adolescent growth spurt (Duren *et al*., 2013). Axial growth is more sensitive to growth disruption in adolescence (Bass *et al*., 1999; Riggs *et al*., 1999; Bradney et al., 2000; Seeman *et al*., 2000), and as growth delay during this section of the growth period has previously been used to identify entrance to the labour market

(Cardoso and Garcia, 2009), it was suggested that this growth disturbance may reflect employment within apprenticeships. It is unlikely that middle class children would not be expected to work and contribute to the family budget, however the types of work they would be expected to employ may have been "lighter" than that experienced by children of the poor, and they were perhaps apprenticed within the family business (Earle, 1989). However, due to lack of supporting documentary evidence, this is purely speculation.

For the Cross Bones sample, growth and pathology data were suggestive of the risks that accompanied being born into poverty, and this will be discussed in more detail in section 6.4. Growth deficits in tibial diaphyseal length, femoral CT, and TR diameter were not only evident in the significant deficiencies compared to modern data, but also in the significantly lower femoral CT values when compared to Coach Lane, and significantly reduced vertebral body height (T6-8) when compared to the Bow Baptist sample. While it has been seen that a peak in metabolic disease between 1-5 years of age was the norm for the middling and wealthier classes at this time, rates of metabolic disease within Cross Bones were high from the "foetal and perinatal" period right through to five years of age. This was primarily due to the extremely high rate of scurvy amongst the perinates, and was indicative of maternal health deficiencies. The unconsecreated burial ground of Cross Bones has been associated with the burial of "single woman" (prostitutes), and certainly in the 19th century was reserved for the very poor, including pauper burials (Brickley and Miles, 1999). Therefore, the health deficits seen within the Cross Bones sample are reflective of poor maternal health and the exogenous influences of an unsanitary environment associated with living in poverty.

Identification of social status of the Coach Lane cemetery sample has been problematic, due to paucities in biographical information for this site, and the lack of grave goods associated with Quaker burial practices (further discussed in section 4.3.1). On examination of the results from the non-adult sample of this cemetery site, it does demonstrate characteristics of both low status and middling status groups. Peak age-at-death occurred between 1-5 years of age, comparable to Chelsea Old Church, St Benet Sherehog, and the Bow Baptists, and growth values tended to be higher than some of the other archaeological samples. For example, for femoral CT, Coach Lane demonstrated significantly higher values when compared

to Cross Bones, and for vertebral body height (T6-8) this sample was significantly higher than Coronation Street. Coach Lane also shows some of the highest values for vertebral body height in adolescence, particularly when compared to the Bow Baptist sample (see Fig. 4a: Manuscript Three, and Fig. 1c: Manuscript Four). For TR diameter (C5-6), Coach Lane also had significantly larger values than the Bow Baptist sample. However, in contrast to the comparatively superior growth values seen in this sample, overall rates of pathology were some of the highest seen within the six archaeological sites, second only to the Cross Bones sample. There were extremely high rates of metabolic disease within this site (at 21% for rickets, 10% for scurvy, and 62% for "metabolic disease"), and a large proportion of these occurred from a very early age. The peak in metabolic disease prevalence occurred from one month to five years of age, more comparable with the low status groups of Cross Bones and Coronation Street. This high rate of scurvy and rickets in the foetal and perinatal periods, and between 1-11 months suggests that maternal health within this community was also compromised. On analysis of co-morbidity, metabolic disease had by far the greatest influence on health status and growth, which may have a connection to the northern latitude of North Shields, but is also likely related to child labour practices that necessitated long hours spent indoors. It is therefore possible that this population is perhaps more representative of a lower status group, however there must have been some buffering (whether due to superior northern diets, intra-community care associated with Quaker poor relief strategies, or a combination of many factors) that meant these children were more likely to survive long enough for skeletal indicators of pathology to manifest (DeWitte and Stojanowski, 2015). Lower status families were often attracted to dissenter lifestyles, due to the oppressive nature of the church and parish poor laws on those living in poverty (Cherryson *et al.,* 2012; Henderson *et al.,* 2013). This is particularly true for Methodism, but it is also possible that this may have occurred within Quaker communities (Mathias, 2001). Within Quaker communities there was an obligation to help members experiencing difficulties, and it had been boasted, "…no Quaker received poor relief" (Mathias, 2001: 144). Like that seen in the Chelsea Old Church sample, a study by Ostrander (2013) revealed significant exposure to lead in childhood from the industrial environment of North Shields. It has been reported that iron deficiency anaemia increases intestinal lead absorption

(Baker *et al*., 2010). While Ostrander (2013) and Millard *et al*. (2014) found no association between lead exposure and cribra orbitalia in their samples, Ostrander (2013) did find a correlation between lead exposure and metabolic disease in Coach Lane. This is suggestive of a complex interplay of severe industrial environmental conditions, nutritional deficiency in infancy, and social practices that impacted on child health, and consequently led to the high levels of skeletal pathology seen in this sample.

Lastly, for Coronation Street the dichotomy that exists between the growth and pathology data was unexpected. While the growth data revealed significant growth deficits for all skeletal parameters in reference to both modern comparative data and the other archaeological samples (being significantly lower than the Bow Baptist sample for tibial diaphyseal length, and significantly smaller than the St Benet Sherehog, Bow Baptist, Cross Bones, and Coach Lane samples for vertebral body height – T6-8), this site demonstrated surprisingly low rates of pathology. While prevalence of pathology is regularly used within palaeopathological study to indicate overall population health (Larsen, 1997; DeWitte and Stojanowski, 2015), it is important to note that the absence of overt markers of pathology is not necessarily suggestive of absence of health stress (Wood *et al*., 1992). While the above sections have demonstrated varying degrees of environmental and social influences on child health within archaeological sites using elevated prevalence rates of select indicators of stress, the notably low prevalence rates of pathology seen within Coronation Street is also of significance. As already stated in Manuscript Three, acute stressors, such as the myriad of infectious disease known to have affected children during the 18th and 19th centuries, are more likely to have directly affected survival prospects rather than impacting on growth or allowing time for manifestation of pathological markers (McDade, 2003; McDade *et al*., 2008; Vercelotti *et al*., 2014). To look at Figure 5 in Manuscript Three, it would be easy to assume that the non-adults within Coronation Street were less "stressed" than the comparative archaeological populations. However, as described above, the growth parameters revealed significant deficiencies, as well as a lag in growth of vertebral body height between 5-9 years of age (see Manuscript One). In addition, the peak in age-at-death in the "Foetal + Perinatal" age category is not expressive of a "healthy" population, and high rates of DEH indicates presence of early life

stressors such as malnutrition and infection (King *et al*., 2005; Hillson, 2008). It has been stated that –

> *"Infant mortality is the most sensitive index we possess of social welfare and of sanitary administration, especially under urban conditions." Sir Arthur Newsholme, quoted in Titmuss (1943: 12)*

However, it is important to reiterate that a large proportion of the foetal and perinatal individuals within this sample came from the western side of the lower burial horizon of the excavation site (Raynor *et al*., 2011). This section spreads beyond the original cemetery boundaries, so it has been suggested that these individuals may represent un-baptised infants, buried in unconsecrated ground. While this may skew the data for Coronation Street somewhat, it does make this sample more comparable in terms of infant health to the Cross Bones sample. Therefore, using multiple methodologies to access evidence of stress within this study has helped identify deficiencies in infant health that would have previously been overlooked.

In summary, the above differences between sites detected in mortality, growth, and pathology patterns stem from differences in social status (and associated social practices), rather than geographical differences between the North and South. In short, this is due to the fact that there were no substantial differences in northern and southern industrial centres during this time, due to pervasive deficiencies in environmental conditions associated with large towns and cities. However, Manuscript Three did indicate that the high rate of rickets demonstrated within the Coach Lane non-adult sample might be related to the northern latitude of North Shields. As metabolic disease was identified as being a primary influence on the growth of affected individuals in Manuscript Four, this may indicate that the higher risk of vitamin D deficiency within this community led to overall health deficits, due to the association between vitamin D and immune resistance. In this sense, the northern latitude of North Shields may have generated this difference in susceptibility to vitamin D deficiency when compared to the South. The British Medical Association identified "…Newcastle, Gateshead, and practically all of Tyneside…" as having a particularly high prevalence of rickets (Owen, 1889: 114). This may have had significant implications for future health risks for the non-adults

of Coach Lane due to suppressed immunity, and this population did demonstrate some of the highest prevalence of pathology. Recent studies have revealed a high prevalence of vitamin D deficiencies in the UK today, and lower levels of UVB exposure have been identified in the North compared to the South (Macdonald *et al.,* 2011; NICE, 2014). This is therefore suggestive of a high risk for health inequalities in the North of England today, due to the impact of vitamin D deficiency on immunocompetence, and its association with an increased risk of many chronic diseases (Holick, 2004).

6.3 Impact of child health on longevity

While it has been shown that urban life came with significant health hazards for the very young, none of the four manuscripts have directly addressed the resultant effects of such early life stress on adult health. The long term health consequences of early life adversity has become the focus of intense research interest over the last two decades, known as the Developmental Origins of Health and Disease (DOHaD) hypothesis (see Chapter Two: section 2.5). As a consequence, the well-being of infants and young children is now known to be central to overall population health (Gowland, 2015). In line with the DOHaD hypothesis, unhealthy children would be expected to eventually become unhealthy adults. To recap, early life stressors are associated with an increased susceptibility to significant health issues such as cardiovascular disease, diabetes, stomach cancer, hypertension, and respiratory disease (Barker and Osmond, 1986; Barker, 1991,1992,1994). With copious evidence that the 18th-19th century industrial environment was poor, and diet frequently compromised in the young, it is of interest to establish what effect this had on those who survived childhood.

It is clear that the non-adults within these six archaeological sites were exposed to such early life stressors, thus it is now necessary to assess the state of health amongst the adults of each group within this study. This will enable a discussion of the consequences of poor childhood health on future adult health in the 18th/19th centuries. Unfortunately, it is not currently within the scope of palaeopathological research to access information such as cardiovascular health and evidence of diabetes, and evidence of cancer in the past is rare and limited to those affecting the bone (Steckel, 2005). Recent research has begun to identify evidence

of cardiovascular disease in the past through identification of calcified blood vessels (potentially indicative of atherosclerosis) associated with human remains (Binder and Roberts, 2014), and studies of dental calculus (Adler *et al*., 2013). Therefore future studies may be able to incorporate such evidence into discussions of the DOHaD hypothesis. Evidence of reduced longevity within adult samples can also used as a marker of suboptimal health within skeletal collections, and when combined with data reflecting early life stress, may be used to assess population health in line with the DOHaD hypothesis within bioarchaeology. Evidence of growth disruption has been the primary mode of accessing child health within this study. Periods of growth stunting have both short and long term consequences for health. For example, a longitudinal study of boys from Helsinki showed that those who were taller had a longer life-span; however if the boys achieved a tall height after a period of stunting, followed by rapid catch-up growth, they experienced a shorter life-span (Barker *et al*., 2011). Therefore, growth patterns, along with the interplay of early life stressors with health status, can also impact on longevity. As described in Chapter Two: section 2.5, as an individual grows and develops, a multitude of biological trade-offs may have to be made when resources are limited, thus tipping the balance between the processes necessary for growth, maintenance, and survival (Worthman and Kuzara, 2005). In the same way that growth stunting stems from the diversion of resources toward survival, the elevated energetic requirements of catch-up growth may too come at the expense of essential maintenance processes, with implications for future health (Bogin, 1999; Cardoso and Garcia, 2009). Thus, the analysis of non-specific indicators of stress (namely age-at-death distributions, stature comparisons, and prevalence of pathology) amongst the adult sample of each group may permit discussion regarding longevity, overall health status, and child health histories.

Age-at-death distributions were plotted for the age categories 18-25, 26-35, 36-45, and >45 for each site. All adult mortality and pathology data for the Bow Baptist population is only representative of the PAY05 portion of the skeletal collection, due to availability of adult data. Classic "attritional mortality" profiles expected within cemetery populations are characterised by a low proportion of adolescent individuals within the cemetery population, followed by a gradual increase in number of young adults through to older adults (Gowland and

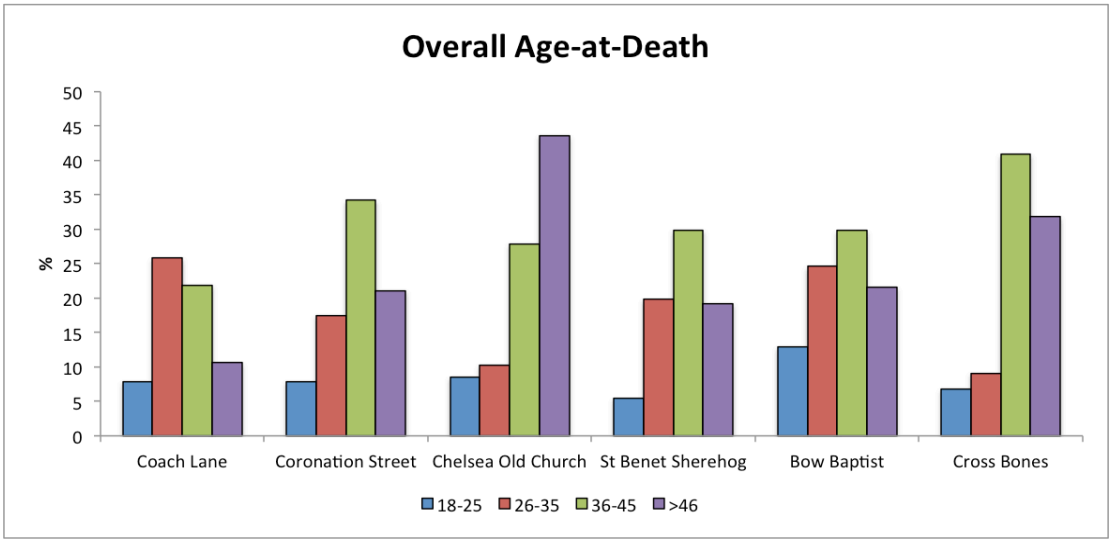

Figure 6.2 – *Age-at-death distribution for the adult sample of each site.*

Chamberlain, 2005). This follows the assumption that with increasing age comes increasing risk of death. However, the underestimation of age in older adults is an issue inherent in osteological study (Gowland, 2007). While ageing of non-adults can be done to a greater degree of reliability due to the reliance on fairly predictable developmental stages, ageing of adults relies on a uniform progression of degeneration (Gowland, 2007). The methodologies used to assess adult age (primarily the auricular surface and pubic symphysis of the os coxae) come with a large potential for error from not only inter-observer differences in recording, but also individual variability in the ageing process, and differential preservation of the skeletal elements required for analysis (Gowland, 2007). This pattern is evident in Figure 6.2. With the exception of Chelsea Old Church and Coach Lane, all archaeological sites demonstrate a peak age-at-death between 36-45 years of age. Whereas the peak age-at-death for Chelsea Old Church occurred in the older adult age category of ">46 years", and for Coach Lane between 26-35 years of age. It is of note that there is still a high proportion of older adults within the Cross Bones sample, despite it not being the peak category for age-at-death. This under-representation of older adults in the Coach Lane, Coronation Street, St Benet Sherehog, and Bow Baptist samples may reflect biases in adult ageing methodology, but could also be suggestive of the often reduced life expectancies in the pre-antibiotic era of the 18th and 19th centuries (Chadwick, 1842).

The reported annual deaths from the Registrar General for the year 1850 (Registrar-General, 1854) have been calculated for each age group as a percentage of the total number of deaths for England and Wales, London, and the Northern Counties (see Fig. 6.3). Due to the data available, the 18-25 years age category implemented in the osteological analysis must be compared to the 15-25 years age category in the historical data. From these data it is possible to ascertain that the the proportion of adult age-at-death within London increased gradually until peaking between 45-54 years of age, and continued at a steady proportion before beginning to drop from 65-74 years onwards (Fig.6.3). For the Northern counties, and England and Wales as a whole, there was a slightly higher proportion of death between 15-24 years when compared to London, but comparatively lower values from 25-64 years of age. For these two groups the peak age-at-death for adults was between 65-74 years of age, after which numbers dropped. Therefore, the historical data reveals that the majority of adult deaths in the 19th century occurred in those aged 45 years and above, and a slightly higher risk of death existed in London throughout adulthood. Therefore, the mortality profiles based on the osteological data are either skewed by biases in ageing methodologies, or these archaeological populations suffered from heightened risks of reduced longevity. In this case, the peak in age-at-death within the ">46 years" category in the Chelsea Old Church group could be indicative of their wealthier position within society, which may have buffered them

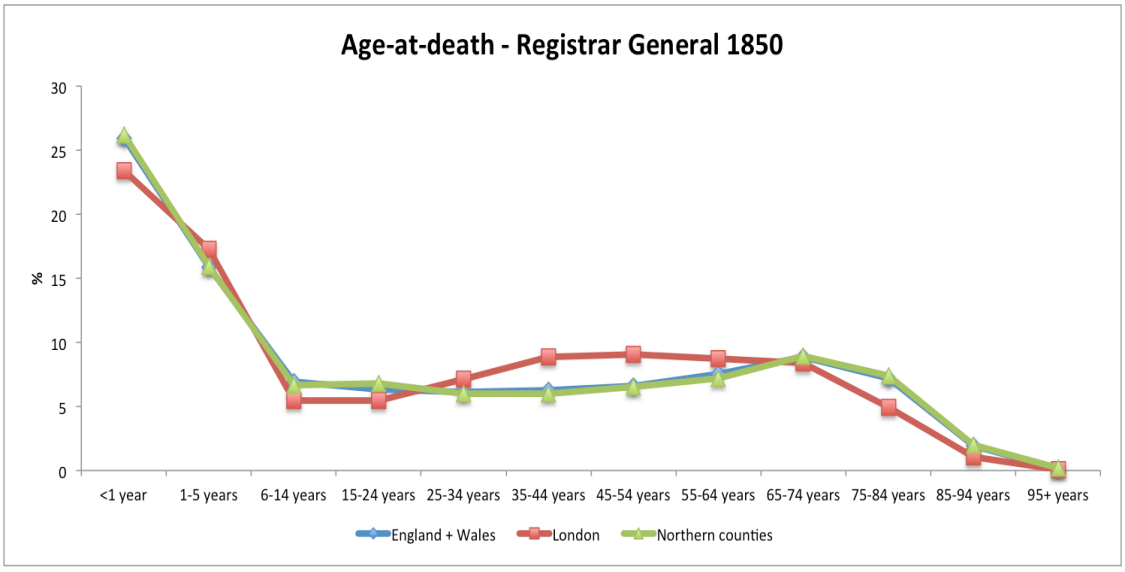

Figure 6.3 – *Percentage of individuals dying within each age category from the overall number of deaths within England and Wales, London, and the northern counties in the year 1850. Data taken from the Registrar-General (1854).*

from some of the more severe health insults of the urban environment. This adheres to the results of the non-adult growth data, as adolescents within Chelsea Old Church were more likely to show higher growth values despite evidence of stressors in infancy. As can be seen in Figure 6.2, all six sites have a low representation of 18-25 year olds, which is expected within an attritional mortality profile, and is also reflected in the historical data in Figure 6.3. However, that the Coach Lane population should show such a high proportion of 26-35 year olds in comparison to the rest of the population, the remaining archaeological samples, and expected levels inferred from historical data, is of interest. This may represent a heightened risk of reduced longevity within this group, reflective of the very poor health of the infants at this site.

Another method through which overall population health can be assessed is through the analysis of stature. As the growth of an individual is determined by factors such as nutritional status, exposure to infectious agents, and socio-economic status, as well as being genetically determined, final adult stature is considered a sensitive indicator of both childhood health and environmental conditions (Johnson and Nicholas, 1997; Nicholas and Oxley, 1993; Cameron and Bogin, 2012). As previously discussed in Chapter Two, variation in stature between groups has been used by a multitude of past studies to discuss the impact of disease on populations from the 18th and 19th centuries (Floud, 1990; Nicholas and Steckel, 1991; Voth and Leunig, 1996; Oxley, 2003; Humphries and Leunig, 2009; Sharpe, 2012; Kirby, 2013). The mean stature (calculated using Trotter, 1970 from femoral length) for males and females from each site has been plotted in Figure 6.4, and reveals that stature was in general very similar, regardless of social status and geographic location. Females were consistently smaller in height than males, and this difference is most marked in the Coach Lane and Bow Baptist populations. These differences are most likely due to sexual dimorphism in height (Wells, 2007; Stulp and Barrett, 2014), but the greater variation seen between females could be suggestive of female disadvantage. A study of Irish and English born convicts from 1817-1840 revealed that Irish women had a height advantage over urban English women (Nicholas and Steckel, 1991). It was suggested that this may be due to the "breadwinner" system in England, whereas all members within Irish families would go out to work (Nicholas and Steckel, 1991). Chelsea Old Church demonstrates the highest female

stature, supporting research by Hughes-Morey (2015), whereas Coach Lane demonstrates the lowest. As the non-adult sample of Coach Lane showed evidence of poor maternal health through the high prevalence of deficiency diseases in infancy, this shorter stature may too indicate the enduring influences of heritable deficiency alongside social factors that led to female health disadvantage at this time. This will be discussed further in section 6.4.

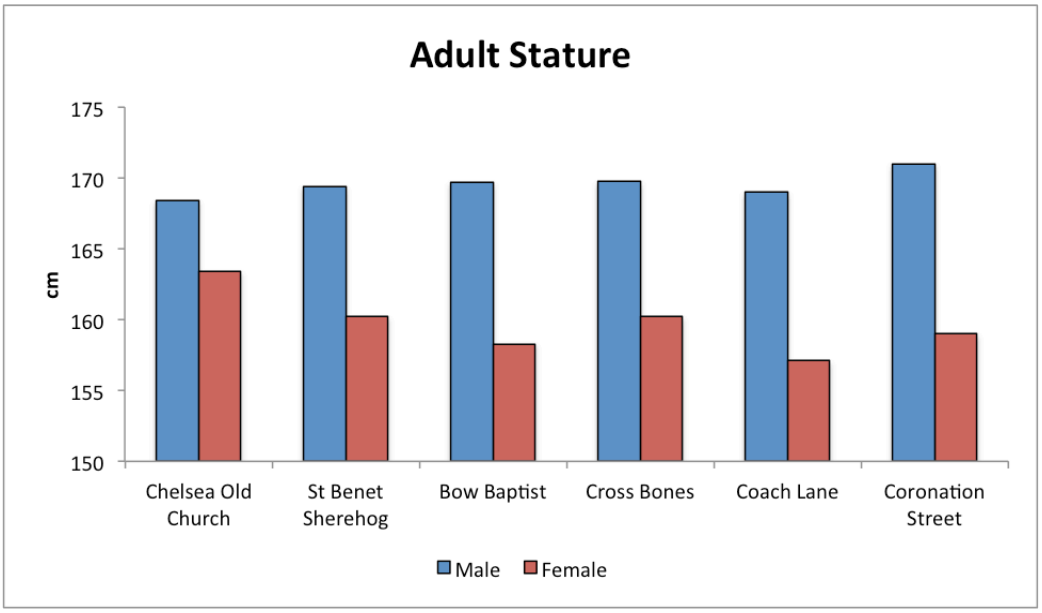

Figure 6.4 – *Average adult stature for males and females of each site. Calculated using Trotter (1970) using femoral length.*

The lack of substantial differences between the sites in terms of stature is not surprising, considering the fact that very few differences in diaphyseal length were detected between the non-adult samples of each site. As stature is representative of health status throughout the growth period (i.e. infancy, childhood, and adolescence), episodes of stress would have to be chronic in nature for there to be a permanent influence on future adult height, and catch-up growth frequently veils such episodes of infection and malnutrition (Pinhasi *et al.*, 2006; Mays *et al.*, 2009a,b). When assessing the stature of adults within a population, it is also important to consider the fact that these individuals survived the vulnerable periods of infancy, childhood, and adolescence, therefore may either have not experienced the substantial health stressors exhibited by the non-adults of each site, or were simply more robust (Wood *et al.*, 1992).

To determine whether the adults of each site did in fact experience stress in infancy and childhood, and whether these early life stressors impacted on future adult health and survival prospects, crude prevalence rates for residual rickets, periosteal new bone formation, DEH, and cribra orbitalia, were calculated for the adult population of each cemetery site. For the Bow Baptist collection, pathology data is again only based on PAY05 due to availability of data.

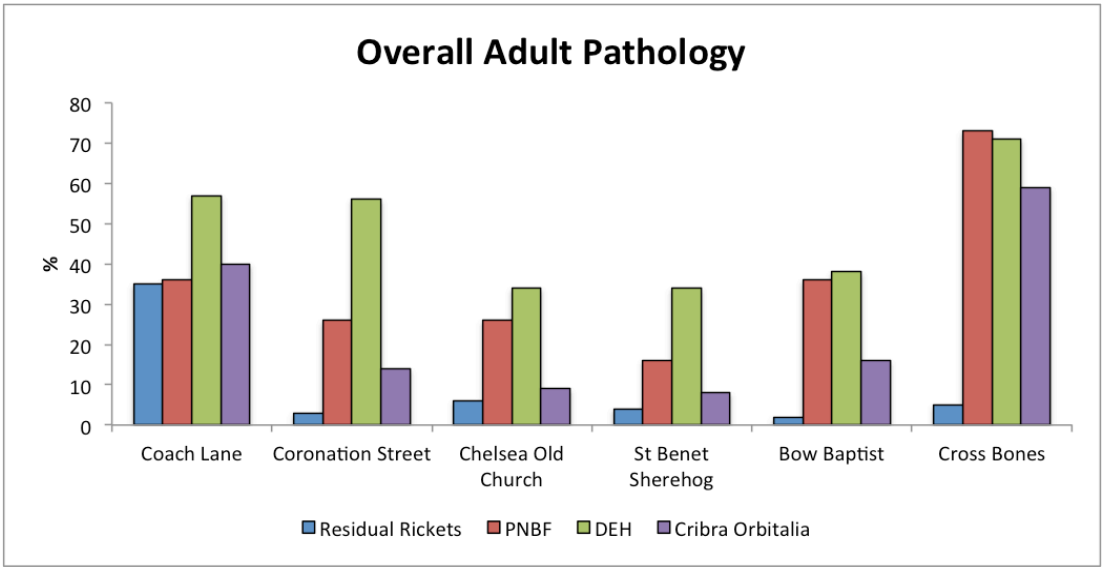

Figure 6.5 – *CPR (%) of pathology within adult samples for each site. PNBF = Periosteal new bone formation. Calculated as a percentage of total number of adults in each population.*

As can be seen in Figure 6.5, the overall adult pathology data reveals that Coach Lane and Cross Bones in general have the highest rates of pathology amongst the six archaeological sites. The highest rates for all sites tend to be for DEH (ranging from 34% to 71%). Coach Lane and Cross Bones have a particularly high prevalence of cribra orbitalia (at 40% and 59%). This condition, like DEH, is associated with health insults experienced during childhood while centres for red blood cell production are still located above the orbits (Walker *et al.,* 2009). There is also a relatively high rate of periosteal new bone formation amongst the sites, particularly for Cross Bones (at 73%). Periosteal new bone formation has been used by DeWitte (2010, 2014; DeWitte *et al.,* 2015) as an indicator of "frailty" when used alongside evidence of earlier age-at-death in adults. This follows the rationale that a correlation between the two indicates a heightened susceptibility to health risks (i.e. infection), and therefore an increased risk of premature death. The high rate of residual rickets (35%) is also notable within the Coach Lane sample, and is

unsurprising considering the extremely high rate of rickets within the non-adult population of this site. Therefore many individuals within the adult samples demonstrate evidence of having survived to maturity despite exposure to early life stressors (such as malnutrition and/or infection).

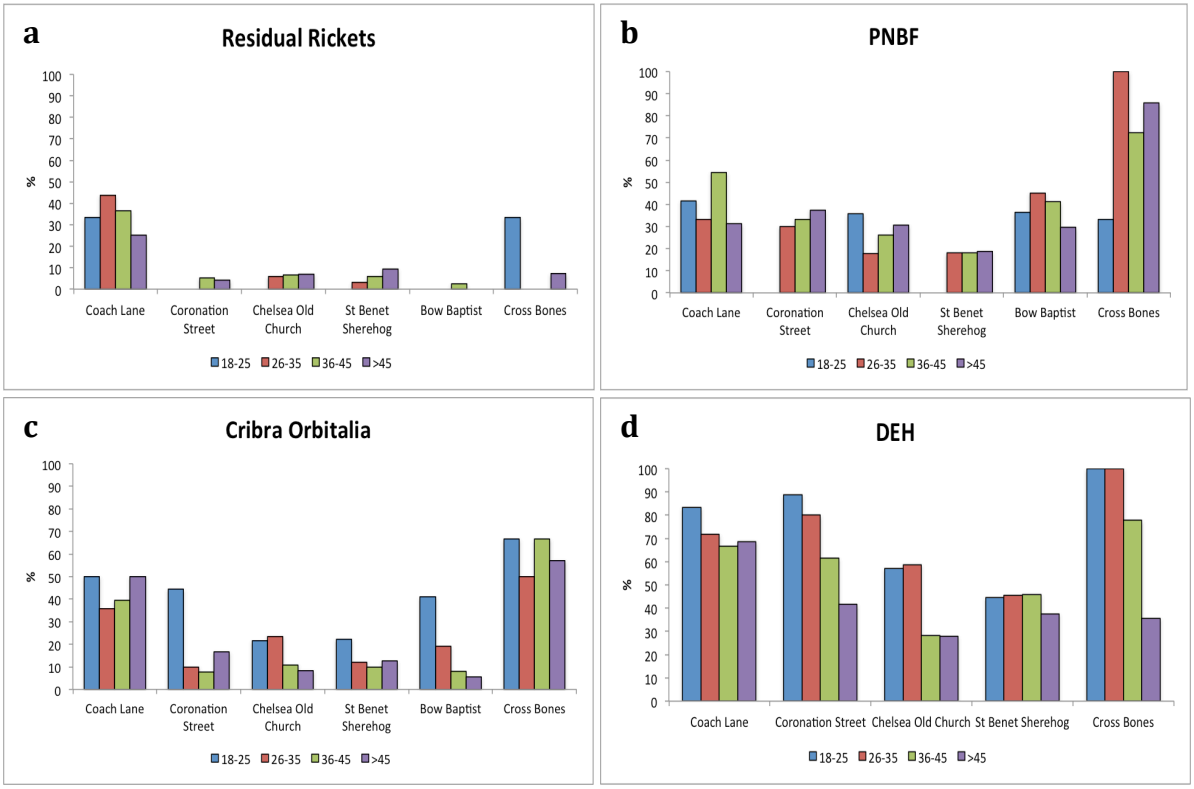

Figure 6.6 – *CPR (%) of pathology for each site for the age categories; 18-25 years, 26-35 years, 36-45 years, 46+ years. PNBF = Periosteal new bone formation. Calculated as a percentage of total number of adults within each age category.*

To determine whether the presence of these non-specific indicators of stress correlated with a reduction in longevity, prevalence rates were also assessed within each of the adult age categories. For residual rickets, there are an extremely high proportion of individuals affected (at 44%) within the 26-35 year age category of the Coach Lane sample (see Figure 6.6a). There is also a comparatively high proportion of individuals affected by residual rickets within Coach Lane for those aged 18-25 years, 36-45 years, and >45 years. A previous study has already revealed a high prevalence of residual bowing and "knock-kneed" individuals within Coach Lane (Tschinkel, 2013). That this condition should show an association with earlier mortality in the 26-35 years age category may be suggestive of the impact of vitamin D deficiency in childhood on future immunocompetency. There was also a very high proportion of individuals with residual rickets within the

18-25 years age category of Cross Bones, however it must be noted that this category is only representative of three individuals. There was no other discernible correlation between residual rickets and age within the remaining sites. Likewise, there was no evident connection between age-at-death and presence of periosteal new bone formation (see Fig.6.6b), with the exception of the Coach Lane sample where those between 18-25 and 36-45 years of age were more likely to demonstrate evidence of periosteal reaction. While a higher proportion of 18-25 year olds within the Chelsea Old Church sample showed evidence of periosteal reaction, there were no individuals affected for this age group within Coronation Street and St Benet Sherehog. Again, prevalence rates seen at Cross Bones are likely to be skewed by small sample size for those between 18-25 and 26-35 years of age. For cribra orbitalia, those in Coach Lane exhibiting this condition of childhood (and therefore those who experienced episodes of stress during development) were more likely to be aged between 18-25 and >45 years, and prevalence rates were generally high (see Fig. 6.6c). Coronation Street, Chelsea Old Church, St Benet Sherehog, and the Bow Baptist sample all showed a higher proportion of individuals affected by cribra orbitalia in the earlier age categories. Lastly, a large proportion of individuals demonstrated enamel defects indicative of DEH throughout the sites and the four age categories. The highest proportions of individuals affected tended to occur between 18-25 and 26-35 years of age (Fig.6.6d). Unfortunately, sufficient DEH data to assess prevalence within the discrete age groups was not available for analysis with the Bow Baptist sample. While prevalence of periosteal new bone formation, cribra orbitalia, and DEH have shown some degree of association with an earlier age at death, it must be noted that older adults are scarcer within archaeological samples, due to the biases associated with under-ageing mentioned previously (Gowland, 2007). This therefore may skew the data.

Another method that is being increasingly used within bioarchaeological study to access evidence of health stress in past populations, and the impact of early life stress on longevity, is the analysis of vertebral neural canal size. As discussed in Chapter Two, stenosis of the vertebral neural canal may be associated with disrupted neural and immune development, and therefore could lead to a reduction in longevity (Clark *et al.,* 1986). Adult TR diameters were compared through an ANOVA test to identify any statistically significant differences in average TR

Table 6.1 – *Sample sizes for ANOVA inter-site comparison of adult TR diameters, pooled for the sexes.*

		T10	T11	T12	L1	L2	L3	L4
Coach Lane	Mean	16.75	17.94	20.59	21.98	21.74	21.54	22.06
	N	11	13	13	16	18	20	17
	S.E.	0.33	0.40	0.45	0.41	0.37	0.33	0.39
Coronation Street	Mean	17.20	19.27	20.75	22.86	22.80	23.48	23.59
	N	11	10	9	12	13	12	11
	S.E.	0.61	0.84	0.80	0.72	0.63	0.90	1.16
Chelsea Old Church	Mean	17.31	18.13	19.87	20.79	20.89	20.57	20.71
	N	9	9	9	9	12	12	12
	S.E.	0.52	0.75	0.66	0.66	0.54	0.56	0.46
St Benet Sherehog	Mean	17.41	18.40	21.12	22.43	22.60	22.90	22.72
	N	7	7	8	11	10	11	8
	S.E.	0.51	0.41	0.50	0.44	0.61	0.61	0.70
Bow Baptist	Mean	17.25	18.16	20.30	21.76	21.74	21.64	21.49
	N	29	30	30	29	32	33	32
	S.E.	0.28	0.28	0.29	0.24	0.25	0.26	0.33
Cross Bones	Mean	15.61	17.22	18.02	19.90	20.88	20.88	20.80
	N	5	4	4	4	5	5	2
	S.E.	0.42	0.35	0.90	0.94	0.42	0.35	0.54

Table 6.2 - *ANOVA results for inter-site comparison of adult TR diameters. Tukey post-hoc analysis performed to identify which groups were significantly different from one another. p=<0.05, significant values in bold. CL= Coach Lane, CS= Coronation Street, COC= Chelsea Old Church, SBS= St Benet Sherehog, BB= Bow Baptist.*

ANOVA results		**T10**		**T11**		**T12**		**L1**		**L2**		**L3**		**L4**	
		F	***p***	**F**	***p***	**F**	***p***	**F**	***p***	**F**	***p***	**F**	***p***	**F**	***p***
Inter-site comparison		1.238	0.301	1.080	0.379	1.996	**0.090**	2.879	**0.020**	2.349	**0.048**	4.113	**0.002**	2.694	**0.027**
Tukey post-hoc analysis		**T10**		**T11**		**T12**		**L1**		**L2**		**L3**		**L4**	
		S.E.	***p***	**S.E.**	***p***	**S.E.**	***p***	**S.E.**	***p***	**S.E.**	***p***	**S.E.**	***p***	**S.E.**	***p***
CL vs	**CS**	0.647	0.981	0.733	0.465	0.760	1.000	0.651	0.753	0.615	0.518	0.682	0.060	0.831	0.439
	COC	0.681	0.963	0.755	1.000	0.760	0.933	0.710	0.547	0.629	0.760	0.682	0.713	0.809	0.562
	SBS	0.733	0.943	0.816	0.993	0.788	0.984	0.667	0.985	0.666	0.789	0.701	0.382	0.920	0.978
	BB	0.537	0.936	0.578	0.999	0.582	0.997	0.531	0.998	0.497	1.000	0.529	1.000	0.644	0.952
	CB	0.818	0.733	0.996	0.978	1.002	0.122	0.952	0.258	0.854	0.917	0.933	0.981	1.60	0.969
CS vs	**COC**	0.681	1.000	0.800	0.713	0.826	0.892	0.751	0.075	0.676	0.064	0.762	**0.003**	0.896	**0.023**
	SBS	0.733	1.000	0.858	0.913	0.852	0.998	0.711	0.990	0.710	1.000	0.779	0.976	0.997	0.951
	BB	0.537	1.000	0.636	0.511	0.666	0.985	0.585	0.424	0.555	0.411	0.629	**0.050**	0.750	0.068
	CB	0.818	0.385	1.030	0.359	1.053	0.113	0.984	**0.040**	0.889	0.270	0.993	0.105	1.650	0.538
COC vs	**SBS**	0.764	1.000	0.878	1.000	0.852	0.687	0.766	0.278	0.723	0.183	0.779	**0.040**	0.980	0.324
	BB	0.579	1.000	0.662	1.000	0.666	0.986	0.650	0.662	0.572	0.670	0.629	0.531	0.727	0.890
	CB	0.846	0.351	1.047	0.952	1.053	0.502	1.024	0.954	0.899	1.000	0.993	1.000	1.639	1.000
SBS vs	**BB**	0.639	1.000	0.731	0.999	0.697	0.851	0.603	0.881	0.612	0.732	0.650	0.387	0.848	0.698
	CB	0.888	0.337	1.092	0.886	1.073	**0.057**	0.995	0.127	0.925	0.439	1.01	0.346	1.697	0.865
BB vs	**CB**	0.734	0.238	0.927	0.910	0.933	0.155	0.909	0.326	0.812	0.896	0.896	0.957	1.564	0.998

Table 6.3 – *Sample sizes for ANOVA age comparison (18-25 years vs, 26-35 years) of adult TR diameters, pooled for the sexes.*

			T10	T11	T12	L1	L2	L3	L4
Coach Lane	*18-25*	Mean	16.98	18.7	20.46	22.09	22.02	21.89	22.82
		N	3	2	4	4	5	4	3
		S.E.	0.37	0.04	0.66	0.07	0.35	0.71	0.38
	26-35	Mean	16.66	17.80	20.64	21.94	21.63	21.45	21.89
		N	8	11	9	12	13	16	14
		S.E.	0.44	0.46	0.60	0.55	0.50	0.37	0.46
Coronation Street	*18-25*	Mean	15.89	17.22	20.80	23.28	24.81	26.73	28.15
		N	2	1	1	3	2	2	2
		S.E.	0.93	-	-	2.70	3.84	5.51	5.62
	26-35	Mean	17.49	19.49	20.74	22.72	22.43	22.82	22.58
		N	9	9	8	9	11	10	9
		S.E.	0.70	0.90	0.91	0.57	0.46	0.49	0.68
Chelsea Old Church	*18-25*	Mean	16.52	17.47	19.74	20.05	20.38	20.04	20.53
		N	5	6	6	6	8	6	7
		S.E.	0.63	0.93	0.99	0.80	0.70	0.83	0.51
	26-35	Mean	18.29	19.45	20.13	22.25	21.91	21.10	20.97
		N	4	3	3	3	4	6	5
		S.E.	0.61	1.05	0.48	0.67	0.64	0.76	0.89
St Benet Sherehog	*18-25*	Mean	18.58	19.40	22.02	22.36	22.52	22.69	23.39
		N	2	2	3	4	3	4	3
		S.E.	1.23	0.70	0.47	0.46	1.20	0.55	0.59
	26-35	Mean	16.95	18.00	20.58	22.46	22.63	23.02	22.32
		N	5	5	5	7	7	7	5
		S.E.	0.47	0.41	0.67	0.67	0.76	0.93	1.07
Bow Baptist	*18-25*	Mean	16.74	17.75	19.97	21.81	21.77	21.51	21.11
		N	11	11	13	10	11	12	13
		S.E.	0.36	0.37	0.33	0.40	0.43	0.37	0.35
	26-35	Mean	17.56	18.39	20.56	21.74	21.73	21.71	21.75
		N	18	19	17	19	21	21	19
		S.E.	0.39	0.38	0.44	0.31	0.31	0.35	0.50
Cross Bones	*18-25*	Mean	15.16	16.71	18.85	20.45	20.65	20.81	20.26
		N	2	2	2	2	2	2	1
		S.E.	0.19	0.08	0.33	0.36	0.11	0.25	-
	26-35	Mean	15.92	17.73	17.19	19.36	21.04	20.92	21.33
		N	3	2	2	2	3	3	1
		S.E.	0.68	0.46	1.85	2.13	0.74	0.61	-

diameter between the sites (see Table 6.1 for sample sizes), and between the age groups 18-25 years and 26-35 years within each site (see Table 6.3 for sample sizes). The results of the inter-site comparisons revealed that adult TR diameters for Cross Bones were significantly smaller than Coronation Street at L1 (see Table 6.2). The TR diameters for Chelsea Old Church were significantly smaller than Coronation Street at L3 and L4, and St Benet Sherehog at L3 (Table 6.2). The Bow Baptist TR diameters were also significantly reduced when compared to Coronation

Street at L3 (Table 6.2). Therefore the adults of Chelsea Old Church and Cross Bones demonstrated a tendency towards having smaller TR diameters in comparison to the remaining archaeological samples. This corroborates the non-adult data for these sites, which also demonstrated substantial deficiencies in TR diameter.

Table 6.4 - *ANOVA results age comparisons of adult TR diameters. p=<0.05, significant values in bold.*

	T10		T11		T12		L1		L2		L3		L4	
	F	***p***	**F**	***p***	**F**	***p***	**F**	***p***	**F**	***p***	**F**	***p***	**F**	***p***
Coach Lane	0.172	0.688	0.644	0.439	0.035	0.855	0.023	0.883	0.216	0.649	0.284	0.601	0.805	0.384
Coronation Street	1.014	0.340	0.634	0.449	0.000	0.984	0.105	0.752	1.980	0.187	3.085	0.110	4.752	0.057
Chelsea Old Church	3.918	0.088	1.691	0.235	0.068	0.802	3.082	0.123	1.945	0.193	0.890	0.368	0.214	0.654
St Benet Sherehog	2.568	0.170	3.251	0.131	2.275	0.182	0.010	0.921	0.005	0.943	0.060	0.812	0.520	0.498
Bow Baptist	2.071	0.162	1.255	0.272	1.025	0.320	0.015	0.904	0.005	0.944	0.138	0.713	0.916	0.346
Cross Bones	0.739	0.453	4.893	0.157	0.780	0.470	0.252	0.665	0.165	0.712	0.019	0.898	-	-

As previously discussed in Manuscript Two, these deficiencies are most likely related to child-care practices amongst the wealthy for Chelsea Old Church, and a combination of maternal deficiency and detrimental exogenous influences for the low status group of Cross Bones. It is also of interest that Coronation Street demonstrated significantly larger TR diameters than Chelsea Old Church, Cross Bones, and the Bow Baptist sample. This may suggest exposure to a lesser degree of "stress" in early life in those who survived to adulthood, or alternatively the influence of strong selection pressures that meant that those who suffered poor health in infancy were less likely to survive to adulthood. In terms of the impact of early life stress on longevity, there were no significant differences between those aged between 18-25 years and 26-35 years for any of the sites (Table 6.4). Therefore, despite the substantial impact on morbidity in infancy associated with the urban environment of the 18th and 19th centuries, this did not appear to influence age-at-death in any of these samples for this skeletal parameter. However, a study by Watts (2015) that focused on adult vertebral dimensions (including the adult samples from Chelsea Old Church, St Benet Sherehog, and Cross Bones) did

identify an association between reduced TR diameter and an increased risk of adult mortality. Watts (2015) included the age categories 36-45 years, and 46+ years in the analysis, which may explain why no differences were detected within this thesis.

To summarise, the analysis of the adult samples in relation to the impact of early life stress on longevity has been somewhat inconclusive. While there was a clear under-representation of "older adults" (those aged >45 years) in the Coach Lane, Coronation Street, St Benet Sherehog, and Bow Baptist samples, it remains unclear as to whether this is due to differential risk of early mortality, or due to inherent issues within osteological methodology. However, that the Chelsea Old Church sample shows such a preponderance of "older adults", with a distinctly different mortality pattern in comparison to the other archaeological samples, suggests that this osteological bias may not have skewed the data significantly. In Whitechapel in 1842 it was reported that the expected lifespan for labourers was 22, whereas for "Gentlemen" it was 45 years (Chadwick, 1842). Therefore it would be expected that wealthier populations such as Chelsea Old Church would have such a high proportion of "older adults". This would signify that the peak age-at-death of between 36-45 years in the Coronation Street, St Benet Sherehog, and Bow Baptist sample, and between 26-35 years in the Coach Lane sample, may represent a higher risk of death at comparatively younger ages within these skeletal samples. Adult data often paralleled the results seen in the non-adult data, with a highest overall rate in pathology amongst the adults of Coach Lane and Cross Bones, and significantly reduced adult TR diameters in Chelsea Old Church and Cross Bones. This, alongside the high rates of DEH seen within all of the six samples, suggests that the adult proportion of each cemetery sample survived to maturity despite exposure to episodes of poor health in infancy. This condition appeared to particularly affect those between 18-25 years and 26-35 years, which is suggestive of some impact of early life stress on longevity in these sites. Miszkiewicz (2015) identified status related differentials in DEH prevalence and resultant life-span in a cemetery sample from Canterbury, UK. It was found that a correlation existed between a high rate of DEH and a reduced life-span within the lower status group of this cemetery population (Miszkiewicz, 2015). In addition, there did appear to be a potential correlation between cribra orbitalia and reduced age-at-death, which has also been demonstrated by Steckel (2005). Interestingly this pattern was also true

for periosteal new bone formation within Coach Lane and Chelsea Old Church, which in light of the research by DeWitte (2010, 2014; DeWitte *et al*., 2015) may be suggestive of the influence of frailty on survival prospects within these populations. However, despite this potential evidence of stress in early life impacting on future survival prospects, and by proxy adult health, there were no significant differences in TR diameter between those died between 18-25 years of age and 26-35 years of age. As adults within a cemetery population represent those who survived childhood they are by nature more robust than those who succumbed to poor health prior to maturity, with the weakest members of society succumbing to stressors in infancy and childhood (Armelagos *et al*., 2009; DeWitte *et al*., 2015). Due to historical populations being placed under heavier loads of stress due to the lack of antibiotics when infectious disease was rife, and food resources often being affected by seasonal shortages or chronic periods of famine, selection pressures were likely extremely high (Steckel, 2005). It is likely that such high selection pressures may have instead led to a high infant mortality, with many affected by early life stress being more likely to die before maturity due to inherent frailty. In this context, it may be more difficult to assess principles of the DOHaD hypothesis within 18th-19th century skeletal collections. DeWitte *et al*. (2015) found little effect of status on adult mortality within skeletal collections from the 18th-19th centuries, however she identified significant selective mortality amongst children of lower status. However, Watts (2015) did identify an association between deficiencies in TR diameter and a reduction in longevity within three of the samples used within this study (see above). It was concluded that growth disruption occurring during late childhood and early adolescence affected longevity within these samples, thus highlighting the impact of growth disruption on future adult health (Watts, 2015).

Alternatively, it is possible that the lack of difference seen in this study is due to the fact that, as seen in the non-adult results for TR diameter, the majority of infants at this time were exposed to substantial health risks, regardless of social status or geographical location. In this standpoint, it is expected that all adults should show deficiencies in TR diameter. To put this in perspective, the mean adult TR diameter for a modern, healthy, population is 25mm at L1 (Hinck *et al*., 1966). When this is compared to the mean TR diameters displayed in Table 6.1 and Table 6.3, the adult TR diameters for all of the six sites, and within both age groups

assessed, fall substantially below that of modern data. It may be the case that the lack of significant differences within this growth parameter, and the differences seen in mortality risk and prevalence of pathology, reflects more on the generally unhealthy society of urban centres of the 18th and 19th centuries. Poor health in childhood, particularly infancy, was ubiquitous during this period, but life chances beyond this would be substantially influenced by the situation of an individual within the social strata. This is particularly evident within the non-adult and adult data for Chelsea Old Church. It would therefore be of interest to assess differences in TR diameter between those aged 18-25 years and 26-35 years compared to the age categories 36-45 years and >46 years, as this has proven to be more successful at identifying adult mortality risks (Watts, 2015).

While it has proven difficult to determine whether stress in childhood had any major impact on future adult health, there is one factor within this analysis that is of particular interest. Prevalence of residual rickets was in general low, with the exception of Coach Lane. It has already been noted that rickets was rife amongst the non-adults within this site, but it is also of interest that there is a peak in prevalence of residual rickets between 26-35 years of age. This correlates with the peak age-at-death for this site. It is therefore possible that vitamin D deficiency in childhood was a significant influence on future adult health within this community. This could be connected with the reduction in immune resistance associated with deficiencies in vitamin D (Holick, 2003; Uriu-Adams *et al*., 2013).

Assessing the impact of poor health in infancy, in line with the DOHaD hypothesis is problematic, and it also cannot be overlooked that the reduction in longevity seen could also be related to an accumulation of health insults throughout the life-course, and not purely dictated by early experiences (Blackwell *et al*., 2001). This is particularly pertinent when considering social status, as despite the poor start in infancy evident in the Chelsea Old Church sample, social buffering appears to have enabled improvements to survival prospects.

6.4 Health, wealth, and the poverty trap

"What changes, too, this addition of power is introducing into the Social System; how wealth has more and more increased, and at the same time gathered itself more and more into masses, strangely altering the old relations, and increasing the distance between the rich and the poor…" – Carlyle (1852: 188)

Manuscript Two concluded that children throughout the social strata experienced significant health risks, though these differed in origin. As expected, health was worst in the Cross Bones sample; however, a surprising amount of pathology was evident amongst the children of the middling and upper class samples of Chelsea Old Church, St Benet Sherehog, and the Bow Baptists. This was also reflected in the data for the northern-based sites, seen in the deficiencies in growth and high rate of perinatal deaths demonstrated by the low status Coronation Street, and the high rates of pathology demonstrated by the low/middle status Coach Lane. Within London, the rate of death in children between 0-5 years of age in the area of Bethnal Green (located roughly halfway between the St Benet Sherehog and Bow Baptist populations) was 1 in 5 amongst the gentry and professional families, and 1 in 2 amongst labouring families (Chadwick, 1842). Between 5-10 years of age this rate equaled 1 in 20 and 1 in 15 respectively (Chadwick, 1842). Therefore, mortality rates for children were high in all classes, and this is further evidenced by the deficient growth seen in all four archaeological populations when compared to modern data.

6.4.1 Children of the wealthy

"Wealth, the slave of fashion, and poverty, the victim of necessity, are, in some degree, subject to the same retributive afflictions. The offspring of the one early become phthisieal, nervous, dyspeptic, and laterally curved in their spines; while a corresponding portion of the other become the melancholy objects of rickets, mesenteric disease, scrofula, anteriorly bent spines, and twisted limbs." – Black (1833: 143-144)

Indirect social determinants of health, such as child-care practices, were identified as having a significant impact on child health. The types of child-care strategies implemented by a family will be strongly influenced by their social status. The 18th century English physician Cadogan spoke out strongly against the upper class care of their children, and held the belief that the simple child-care provided

by the lower classes was far more beneficial to health than the indulgences of the wealthy (Levene, 2006).

> *"The Mother who has only a few rags to cover her child loosely, and little more than her own breast to feed it, sees it healthy and strong…while the puny insect, the heir and hope of a rich family, lies languishing under a load of finery that overpowers his limbs, abhorring and rejecting the dainties he is crammed with, till he dies a victim to the mistaken care and tenderness of his fond Mother."*
> *Cadogan (1748: 8-9)*

It was also stated by Beeton "Some women of fashion, moving constantly in society, deny that they have time to give to their little ones" (1861: 1896). Consequently, artificial feeding or the employment of wet nurses were popular alternatives to maternal breastfeeding, despite the espousal of "simple" infant care (Cadogan, 1748; Buchan, 1778, 1804; Wickes, 1953; Fildes, 1995). In addition, women of "fashion" were often considered to be physically unable to breastfeed, as their constitutions were considered too delicate and prone to nervous disorders (Buchan, 1778). The immature immune system of the newborn infant relies on the transmission of maternal antibodies via breast milk for protection from environmental pathogens (Cunningham, 1995; Katzenberg *et al*., 1996). This passive immunity is vital to the health and survival of the infant, and supports the development of the infant's own immune system (Cunningham, 1995; Katzenberg *et al*., 1996). To put this in context, breastfed children in developing countries today have at least a six times greater chance of survival in the early months than non-breastfed children (UNICEF, 2015). Formula-fed children in both developed and developing countries have been found to suffer from more frequent and severe bouts of acute illnesses, ranging from intestinal disorders, gastrointestinal infections, diarrhoea, Sudden Infant Death Syndrome, respiratory disease, and chronic health problems (Stuart-Macadam and Dettwyler, 1995; Cunningham, 1995; Haggerty and Rutstein, 1999; Ip *et al*., 2007; Horta and Victoria, 2013).

Practices such as swaddling led Rousseau to lament "Of our own accord we cripple them to prevent their laming themselves" (1889: 16). However, a study of children from Mongolia found no association between swaddling from the first month after birth, and the onset of rickets (Urnaa *et al*., 2006). During the 18th-19th centuries keeping children indoors was also common practice; therefore it is likely a

combination of child-care practices that restricted sunlight that led to a high prevalence of vitamin D deficiencies amongst the wealthy.

"Many children of the well-to-do were, no doubt, ridiculously pampered and coddled. Not only were they shut up in stuffy rooms, and clad in masses of tight clothing; but they were often kept indoors for days together." (Bayne-Powell, 1939: 3).

Medicines such as "infant soothers" were also widely used throughout the social strata to treat a variety of illnesses (Pinchbeck and Hewitt, 1973; Finch, 1999; Crawford, 2010; Sharpe, 2012). Mrs Beeton in her 1861 *Book of Household Management* frequently refers to the use of a few drops of laudanum for illnesses such as thrush, whooping cough, and diarrhoea (Beeton, 1861). Preparations such as this were highly detrimental to the health of children, and often had fatal consequences (Buchan, 1804; Finch, 1999; Sharpe, 2012). It was also not uncommon for opiates such as "syrup of poppies" to be used to induce docility amongst children (Buchan, 1778; Beeton, 1861; Sharpe, 2012). For example, Godfrey's Cordial (containing ginger, wine, water, treacle, oil of sassafras, and opium), was given to infants from as young as a few days old and often with fatal consequences (Buchan, 1804; Sharpe, 2012):

"The result of this terrible practice is that a great number of infants perish, either suddenly from an overdose, or, as more commonly happens, slowly, painfully and insidiously. Those who escape with life become pale and sickly children, often half idiotic, and always with a ruined constitution." (Pinchbeck and Hewitt, 1973: 406)

The child-care practices and infant feeding strategies of the 18th and 19th meant that children of the upper classes were likely to experience deficiencies in exposure to sunlight and paucities in nutrition, especially in infancy. The results of Manuscript Two and Manuscript Three revealed that rickets was a pervasive condition throughout the sites (with the exception of Coronation Street), regardless of class. Therefore, rickets was not just a disease of the poor. In a time of high morbidity from the polluted urban environment, susceptibility in immune status through lack of passive immunity from maternal breast milk, malnutrition from a poor weaning diet, and immunosuppression resulting from vitamin D deficiency,

would have brought significant risks to survival. However, despite these disadvantages experienced in infancy, it is possible that wealthier children who survived this perilous stage of the life course were more likely to recover with little effect to future health due to social buffering (Hardy, 1992). Despite exhibiting some of the lowest values for TR diameter, tibial diaphyseal length, and femoral CT in infancy, beyond 10 years of age the non-adults of Chelsea Old Church demonstrate higher growth values. This was also reflected in the mortality profile for the adult data for Chelsea Old Church, where adults were more likely to live to older age categories, and research by Hughes-Morey (2015). These results are suggestive of the social gradient in health that was likely in operation at this time.

Likewise, the middling status St Benet Sherehog and Bow Baptist groups were more likely to show higher growth values for tibial diaphyseal length, femoral CT, and vertebral body height, and both demonstrated very low prevalence rates of scurvy. It is possible that children of middle status families experienced the "best of both worlds". From the 18th century family life was becoming more of a private affair, in which children became the centre of a "nuclear family" (Cunningham, 2005). This movement began within the middle classes, and by the mid-19th century ideologies of childhood as being a vital period for determining future adult character had taken hold (Cunningham, 2005). Middle class mothers, released from necessity to work to support the family economy, may therefore have been more likely to rear their children in adherence to the simple practices disseminated by Buchan and Cadogan (Booth, 1902).

"This class is not of necessity pecuniarily better off than the artisan class below it, but the wholesome theory that the man should be the breadwinner of the household pervades it. It is keenly sensitive to social distinctions, is fairly educated, and its anxiety to keep up appearances demands an increasing expenditure on dress, furniture, and food, and perhaps even service, which is more than commensurate with its income. The daughters as they grow up make themselves useful at home either in housework or by assisting in the shop, and are to a certain extent distinguished from the class below them by the fact that they need not go out to work." Booth's description of the middle class family (1902:296)

Away from the deleterious conditions of poverty faced by lower class families, the superior nutrition afforded to middle class mothers alongside a preference for maternal breastfeeding may have buffered their infants somewhat

from the high morbidity and mortality of the urban environment. Analysis of the Christ Church, Spitalfields collection has also suggested that this "middling" status group may have been somewhat buffered from health stress (Lewis, 2002a,b). However, Lewis (2002b) commented that even they were not safe from the ill effects of the industrial environment, noting evidence of indicators of stress in infancy, and the influence of insufficient infant feeding practices. Indeed, Nitsch *et al*. 2011) identified a range of breastfeeding practices in operation within the "middling" status Christ Church, Spitalfields collection. They found that some infants were not breastfed at all, while others were breastfed up until 1.5 years of age (Nitsch *et al*., 2011).

6.4.2 Poverty and maternal health

In this time of rapid change in both society and economy, there were those that profited, but many more still who were left in poverty. Given the well-documented phenomenon of the social gradient of health, and what is known about the environment of industrial centres during this period, it seems reasonable to hypothesise that the children of the lower classes would exhibit an overall greater prevalence of skeletal indicators of poor health and evidence of growth stunting than the middling and upper classes. Children of the lower classes were exposed to higher risks of recurrent poor health and premature death (Fildes, 1995). This is also reflected in the in the high rates of pathology amongst the Cross Bones children, heightened perinatal age-at-death, and the stunting observed in all growth parameters. This heightened perinatal age-at-death was also seen within the low status Coronation Street group, although due to potential problems in cemetery sampling, this should be approached with caution for both sites.

The results of Manuscript Two and Manuscript Three indicate that many infants within the Cross Bones, Coach Lane, and Coronation Street collections experienced insults to health *in utero,* which by nature reflects on maternal health status. For example, there was a high rate of deciduous enamel hypoplasia seen in Cross Bones, high rates of perinatal "metabolic disease" in the Cross Bones and Coach Lane collections, and poor growth values in infancy for Cross Bones, Coach Lane, and Coronation Street. While the attribution of a lower social status can only be tentatively applied to the Coach Lane collection, the evidence for child labour

practices within this group (Roberts *et al*., 2014), alongside evidence for inherited conditions of deficiency (see Manuscript Three), means that the data for this site can be discussed in relation to working class groups and maternal health. As previously discussed in section 6.2, dissenter religions often attracted large working-class followings (Cherryson *et al*., 2012).

In addition to the severe social inequalities in operation during the 18th and 19th centuries, women also faced gender inequalities that may have impacted on health (Rendall, 1990; Perkin, 1993). Women often sought employment to supplement family income, though working conditions and wages for women were generally poor. Furthermore, the requirement to be away from the home for long hours had a significant impact on the health of the children as well as the mother (Engels, 1950; Rendall, 1990; Perkin, 1993). It was not until 1891 that factory legislation began to recognise the significant health risks that accompanied pregnancy in the work place (Perkin, 1993). Expectant mothers in employment would continue to work up to eighteen hours a day until birth, resulting in a high risk of miscarriage (Engels, 1950; Perkin, 1993). Buchan stated that such women had:

> *"…no alternative but to perish with famine, or run the risk of miscarrying by continued exertions at the washing-tub, or at some other toilsome work, for sixteen or eighteen hours, according to the caprice of the sordid views of her unfeeling employer" (Buchan, 1804: 54).*

The lack of reliable contraceptives at this time, combined with the loss of contraceptive benefits gained from breastfeeding due to declining weaning ages (necessitated by poor health or the need to work), meant that birth intervals were usually short (Fildes, 1995; Lane, 2001). Such closely spaced births not only depleted maternal reserves, and added an extra strain on family resource, but also greatly reduced chances of survival of newborns (Stuart-Macadam and Dettwyler, 1995). Risk of death increases by 77% when birth intervals fall below 12 months (Stuart-Macadam and Dettwyler, 1995). Paucities in nutrition due to food allocation practices (Horrell and Oxley, 1999; Horrell and Oxley, 2012), maternal depletion from repeated pregnancies, and/or detrimental working conditions, meant that maternal health suffered greatly in the 18th and 19th centuries (Gowland and Newman, in press). A study by Horrell *et al*. (2009) revealed that women admitted to a London House of Correction from 1858-1878 often gained weight despite the

hard labour requirements, indicating that the harsh conditions inside were in fact preferable to the conditions they experienced outside. Maternal health is a significant determinant in prenatal development and survival, therefore the impact of heightened energetic demands and inadequate nutrition experienced by pregnant working mothers likely had dire consequences for infant health and survival (Wadsworth and Butterworth, 2006; Gowland and Newman, in press).

The loss of a father in the household, through death or absconding, meant that families were at high risk of being pushed further into extreme poverty, which in turn would impact on future employment opportunities of the children (Horrell *et al.,* 2001). Without the influence of the male "breadwinner" on family income, women and children would be required to seek employment in poorly paid and low skilled labour (Horrell *et al.,* 2001). Not only did this limit access to the type of education and training opportunities required to seek higher paid employment prospects in the future, they were required to perform this labour on insufficient diets and often under conditions of significant occupational hazard (Burnett, 1984; Nicholas and Oxley, 1993; Horrell *et al.,* 2001). Inability to pursue skilled employment, combined with deficiencies in health and nutrition, meant that the children of the poor were unequipped to move up the social ladder and out of the poverty trap, and it could take generations for families to achieve this transition. Significantly, this intergenerational poverty is not limited to family economics. The inheritance of maternal health deficits by future generations has already been discussed (see Chapter Two, Manuscript Two, and section 6.3), however low socioeconomic status in childhood has also been shown to impact cognitive development (Chavez *et al.,* 2000; Noble *et al.*, 2015). Therefore early life experiences, combined with maternal health, will determine not only physical growth and future health, but also long-term outcomes such as educational attainment and future economic status (Wilkinson and Pickett, 2010; Aizer and Currie, 2014; Elgar *et al.*, 2015).

With maternal health being such a significant determinant of survival prospects, and future adult health, it is of interest to determine whether the adult data for each site reflects any health inequalities in females compared to males. For calculation of CPR for each pathological indicator, those classed as "female?" and "male?" were included in the female and male categories respectively. The sites that

did not demonstrate a near equal proportion of males and females were St Benet Sherehog (Males = 48.5%, Females = 27.5%), Cross Bones (Males = 27.3%, Females = 61.4%), Bow Baptist (Males = 40.9%, Females = 53%), and Coach Lane (Males = 47.7%, Females = 39.7%). This will be considered when analysing the results, as it could potentially skew the data.

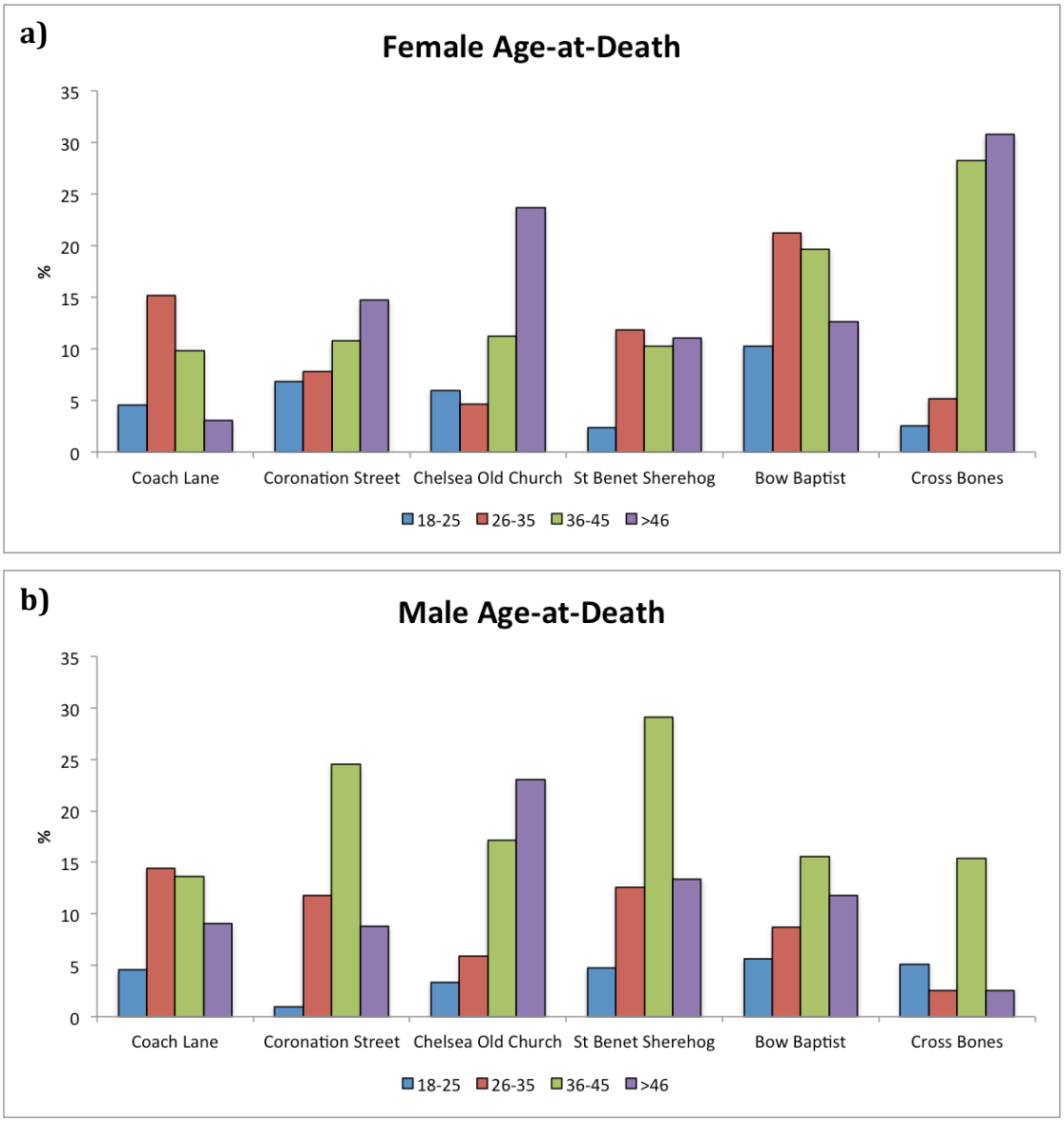

Figure 6.7 – *Age-at-death distributions for a) Females (including those classed as F?), and b) Males (including those classed as M?). Calculated as a percentage of total males and females for each site.*

Figure 6.7 shows the age-at-death distributions for males and females within each site. For Coach Lane, despite an under-representation of females compared to males overall, there is a peak in females dying between the ages of 26-35 years of

age (Fig.6.7a). For Coronation Street there is a larger proportion of females aged 18-25 years, but a larger proportion of males dying between 26-35 years and 36-45 years, and a greater number of females surviving to ">46 years" (Figure 6.7). Chelsea Old Church has a larger proportion of females aged 18-25 years than males, but a higher rate of males between 36-45 years. The peak in males between 36-45 years of age in St Benet Sherehog may reflect the preponderance of males over females in this site (Fig.6.7a). There are, overall, more females than males in the Bow Baptist site, so the elevated number of females compared to males between 26-35 years cannot be attributed with reliability to a true discrepancy between males and females in this younger age category. However, it is interesting that this is the peak age-at-death for females within this site. Lastly, there are an extremely high number of females within Cross Bones compared to males, rendering it difficult to assess mortality patterns. There is a larger proportion of males in the 18-25 age category compared to females, despite their overall underrepresentation within the adult sample (Fig.6.7b). However, this may be a result of a skew in the data due to this collection being a sub-sample of the cemetery, as well as the very small sample size of adults within the Cross Bones collection. A Kolmogorov-Smirnov test was implemented to determine if any statistically significant differences existed between male and female age-at-death distributions for each site. For Cross Bones there was a significant difference between the age distributions ($0.025<p>0.01$), however see above regarding the potential for skewed data within this sample. For the remaining sites there were no significant differences ($p= >0.05$).

To summarise, Coronation Street and Chelsea Old Church show heightened rates of mortality for females in the 18-25 years age categories when compared to males, and females within the Coach Lane, St Benet Sherehog, and Bow Baptist samples have a peak age-at-death between 26-35 years. This may reflect heightened risks of early death for women within these sites, either due to maternal disadvantage (more likely in the lower status groups), or due to the risks that accompanied childbirth. Women of middling to upper classes also frequently underwent repeated pregnancies, with short birth intervals, which likely took a large toll on their health (Lane, 2001; Crawford, 2010). In addition, in this pre-antibiotic era, the potential for complications during pregnancy and childbirth were extremely high. Conditions such as toxaemia, haemorrhage, and puerperal fever were of

significant risk to the survival of the mother, and approximately 1 in 200 births led to the death of the mother during this time (Perkin, 1993: 65). Within the lower classes, 1 in 5 deaths of women between the ages 24-34 years were related to pregnancy or childbirth (Crawford, 2010). The age-at-death distribution for Coronation Street, despite showing a higher rate of young females compared to young males, resembled a normal attritional mortality profile, with a much larger proportion of women aged ">46 years" (Fig.6.7a). This high proportion of "older" females also existed within Chelsea Old Church and St Benet Sherehog, which suggests that female disadvantage was not so much a contributory factor in survival prospects within these populations. Therefore in these cases, risks of pregnancy and childbirth more likely dominated as the primary determinants of female longevity, alongside the ubiquitous risks of disease from the urban environment. For the Coronation Street, Chelsea Old Church, St Benet Sherehog, Bow Baptist, and Cross Bones collections, there was a much higher risk of mortality for males between 36-45 years of age (Fig.6.7b). It is commonly reported that women in general live longer than men, and are more resistant to disease and the influences of environmental stress on health (DeWitte, 2010). However, the number of females surviving to ">46 years" of age within the Coach Lane adult sample was notably low (Fig.6.7a). There were peaks in age-at-death for both males and females between 26-35 years and 36-45 years within this sample, which may be suggestive of a generally lower life expectancy in this northern industrial centre.

General comparisons of prevalence of pathology within males and females within each site reveal that there is a much greater prevalence of residual rickets within males from Coach Lane (Fig.6.8a), which may suggest that young boys were more likely to be employed in apprenticeships or factory work that kept them indoors, while girls were perhaps more likely to be kept within the domestic sphere. Male/female differentials in prevalence of periosteal new bone formation are negligible (Fig.6.8b), with the exception of a much higher rate amongst females in Cross Bones. However, this may be skewed by the generally higher rate of females within this collection. This may also be reflected in the prevalence rates for DEH and cribra orbitalia (Fig.6.8c,d). However, males within the Coach Lane, Coronation Street, Chelsea Old Church, and St Benet Sherehog samples show a consistently higher prevalence of DEH when compared to females from each site

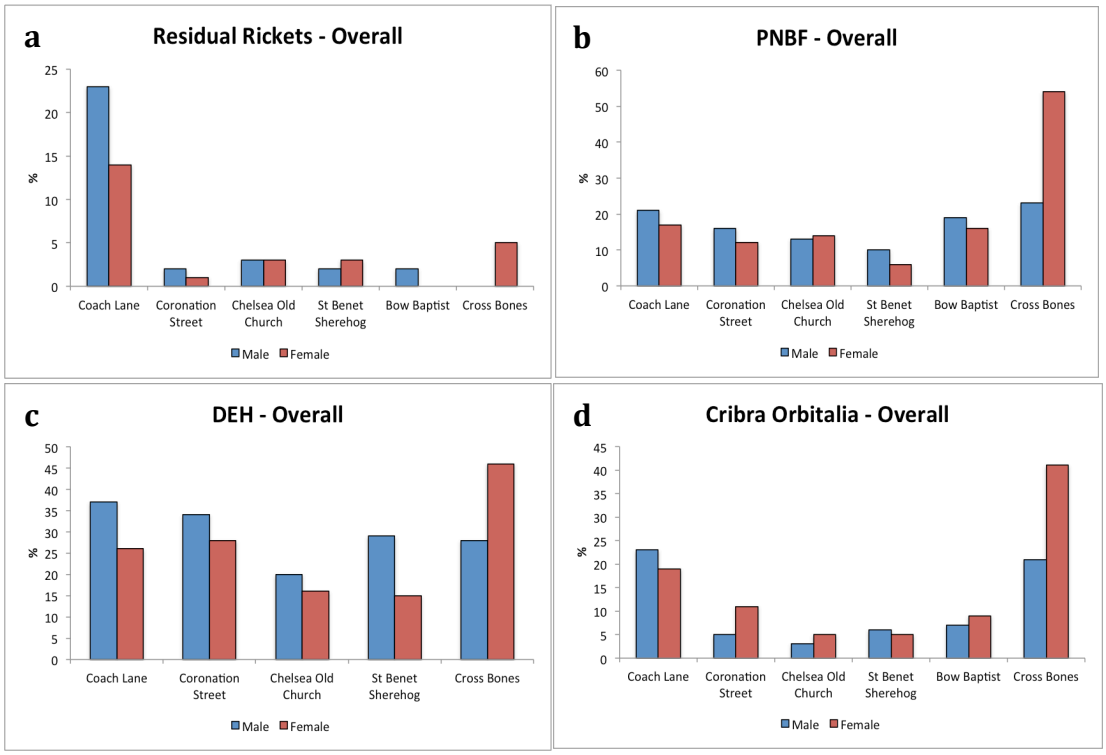

Figure 6.8 – *CPR (%) of pathology for males and females from each site. PNBF = Periosteal new bone formation. Calculated as a percentage of total males and females for each site.*

(Fig.6.8c). Guatelli-Steinberg and Lukacs (1999) found that sex biases in DEH prevalence are generally skewed towards males. They suggested that this may beweakly related to greater vulnerability of males to environmental stressors, and/or female buffering (Guatelli-Steinberg and Lukacs, 1999). Females are known to have stronger immune systems than males, with males being more susceptible to infections (Møller *et al*., 1998; Scmid-Hempel, 2003; Bourman *et al*., 2005; Furman *et al*., 2014). These differentials in immune response are influenced by the sex hormones, with testosterone being immunosuppressive, and oestrogen stimulating antibody production (Bourman *et al*., 2005; Furman *et al*., 2014). However, it is important to note that reproductive events, such as pregnancy and lactation, are energetically costly, therefore during the reproductive years females may undergo periods of immunosuppression as reproductive-immune trade-offs in resource allocation occur (Abrams and Miller, 2011; Pazos *et al*., 2012). There are also many studies that reveal a higher prevalence of stress indicators within females, and in the study by King *et al*. (2005) females from 18th-19th century London demonstrated a higher prevalence of DEH. Only in the Coronation Street adult sample is there a reliably higher rate of cribra orbitalia amongst females when compared to males

(Fig.6.8d).

When these prevalence rates of pathology are broken down by age for both sexes some interesting patterns emerge. Within Coach Lane, there was a generally high proportion of individuals affected by residual rickets for both males and females of all age groups (with the exception of older females) (Fig.6.9). Older females and younger males were also more likely to demonstrate evidence of this condition in the Coronation Street, Chelsea Old Church, and St Benet Sherehog samples (Fig.6.9a).

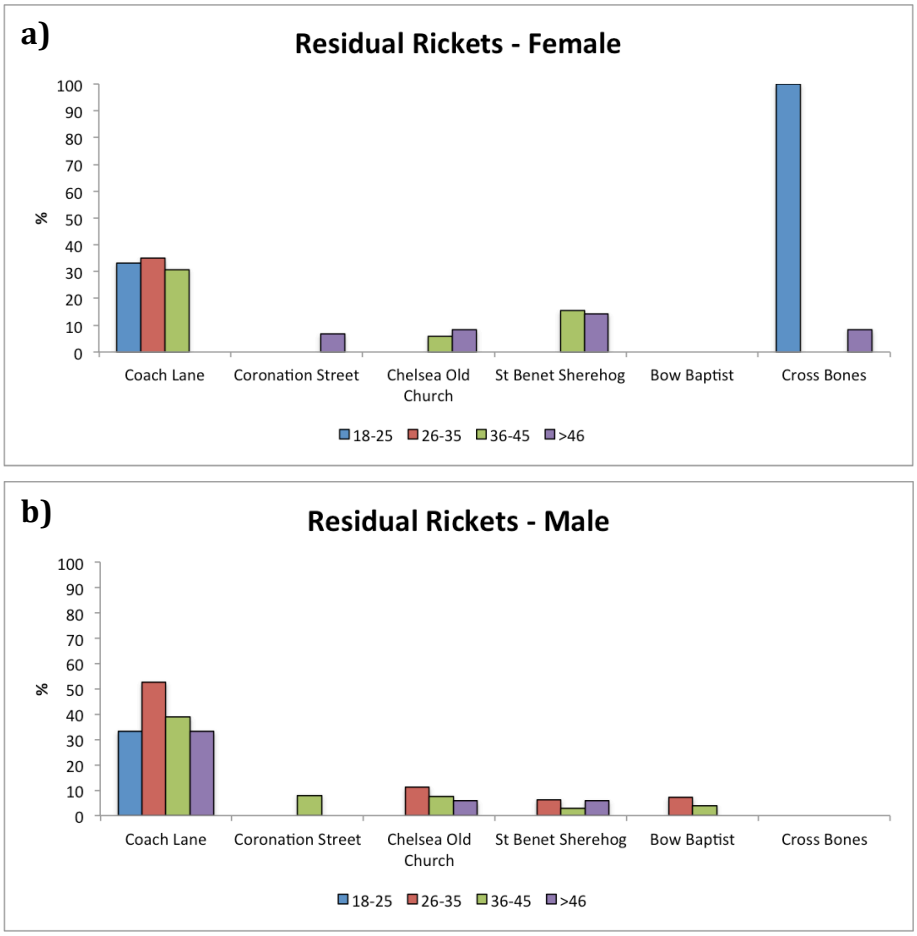

Figure 6.9 – *CPR (%) of residual rickets within each age category for a) Females (including F?), and b) Males (including M?). Calculated as a percentage of total females or males within each age category respectively.*

In general, younger males (particularly aged between 18-25 years of age) and older females were more likely to demonstrate evidence of periosteal new bone formation in Coach Lane (Fig.6.10). A greater proportion of young males were also affected in the Bow Baptist sample. In line with the research by DeWitte (2010), this may too be indicative of innate frailty and susceptibility to infection in male

individuals, as well as the propensity for female buffering. However, a large proportion of young females also demonstrated evidence of periosteal reaction in Chelsea Old Church.

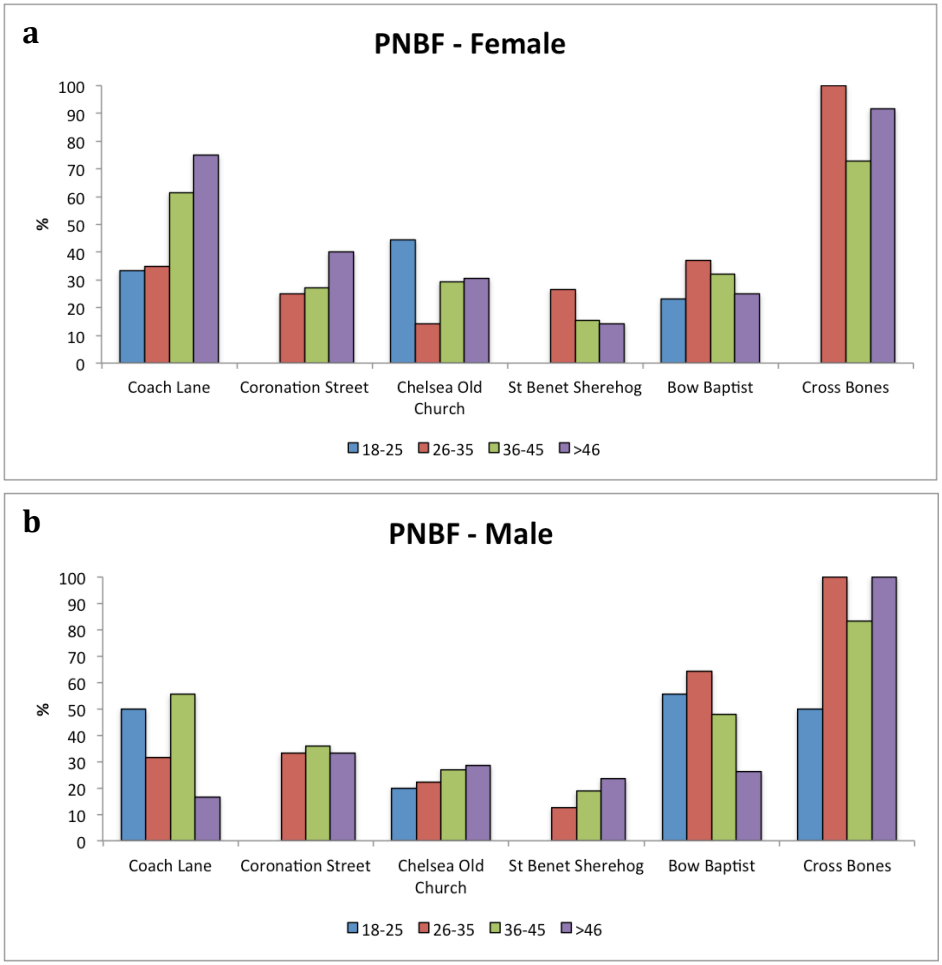

Figure 6.10 - *CPR (%) of non-specific infection within each age category for a) Females (including F?), and b) Males (including M?). PNBF = Periosteal new bone formation. Calculated as a percentage of total females or males within each age category respectively.*

A very high proportion of females aged between 18-25 years and 26-35 years had DEH when compared to later age groups within the Coach Lane, Coronation Street, Chelsea Old Church, St Benet Sherehog, and Cross Bones (Fig.6.11a). This is also seen for male individuals within Chelsea Old Church and tentatively Coronation Street (Fig.6.11b). This may indicate a susceptibility to early age at death in those who experienced poor health in infancy. Lastly, an extremely high proportion of females aged 18-25 years appear to have been affected by cribra orbitalia (and therefore stress in childhood) within all the sites (Fig.6.12a). Therefore, analysis of pathology by age and sex reveals varying susceptibilities to

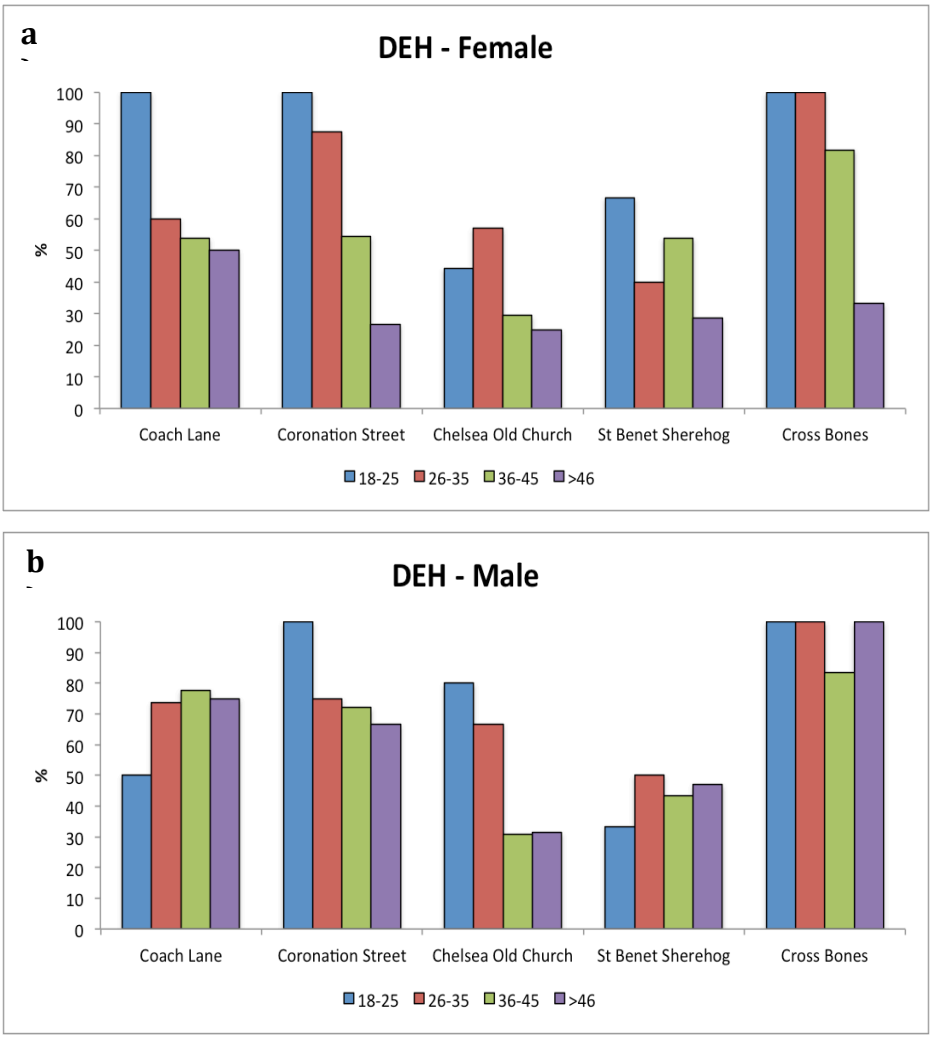

Figure 6.11 - *CPR (%) of DEH within each age category for a) Females (including F?), and b) Males (including M?). Calculated as a percentage of total females or males within each age category respectively.*

early age-at-death related to early life stressors. Some of these associations were indicative of potential female disadvantage, as seen in the association of DEH and cribra orbitalia with a reduction in longevity within the sites. As Cross Bones may not be entirely representative of a true adult mortality and pathology profile (due to issues with excavation – see section 4.3.6), the data for this site have been largely discounted within the analysis. A study by Holland (2013) found that males were more likely to succumb to stressors in childhood, thus males survivors are more likely to demonstrate lower rates of stress indicators than female survivors. Females are therefore more likely to overcome periods of stress, so that the resultant indicators (such as DEH and cribra orbitalia) may be more likely to persist into the adult mortality sample (Holland, 2013). Thus it is important to consider that sex related differentials in frailty may skew data such as this.

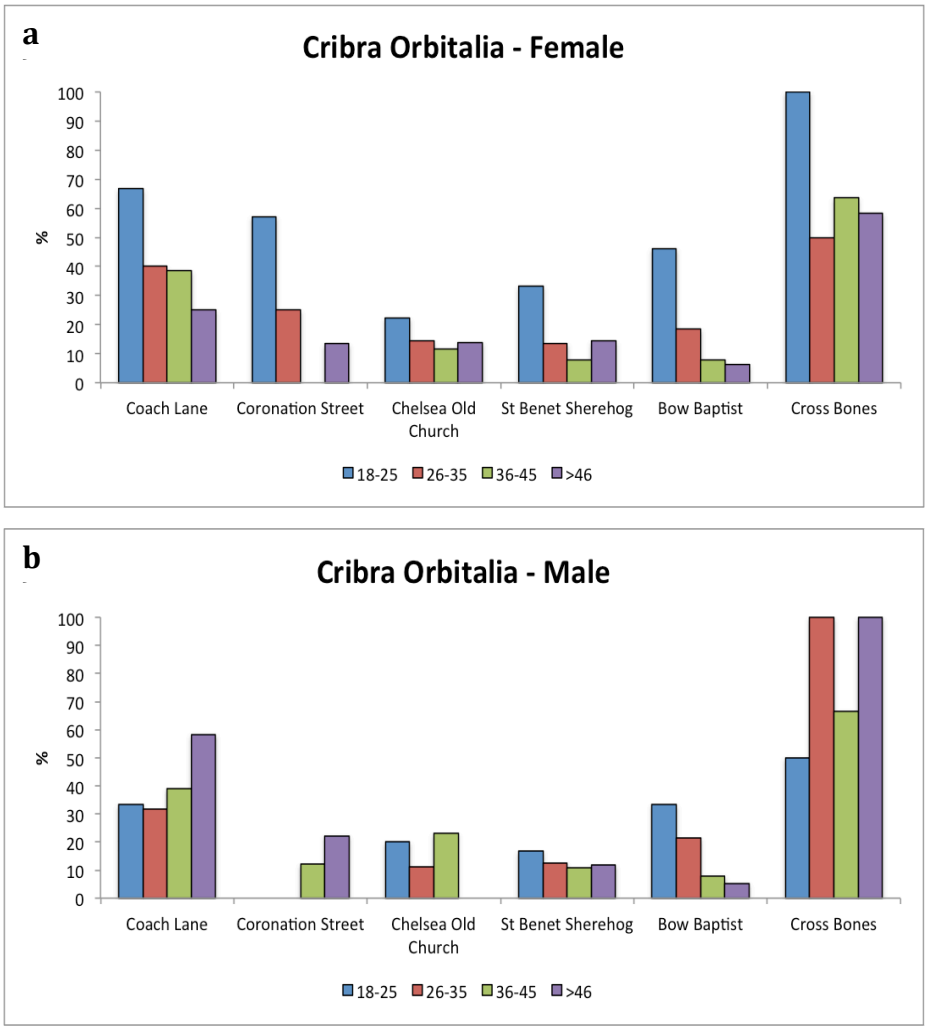

Figure 6.12 - *CPR (%) of cribra orbitalia within each age category for a) Females (including F?), and b) Males (including M?). Calculated as a percentage of total females or males within each age category respectively.*

Finally a statistical comparison of the TR diameters of adult males and females from each site was undertaken through an ANOVA test. The sample sizes for each site can be found in Table 6.5. As can be seen in Table 6.5, the only significant differences between male and female TR diameters occurred at L1 and L2 for the St Benet Sherehog sample, and L1, L2, L3, and L4 for the Bow Baptist sample. In all of these cases, female TR diameters were significantly smaller than male TR diameters. There were no significant differences within the remaining four sites (Table 6.6). As this growth parameter is representative of the first few years of life, it is unlikely that any disparities should occur between males and females, and sexual dimorphism in TR diameter has been found to be limited (Clark *et al.*, 1986; Watts, 2011). In theory, this could be indicative of preferential treatment of male

Table 6.5 – *Sample sizes for ANOVA sex comparison Males vs Females (including M? and F? individuals).*

			T10	T11	T12	L1	L2	L3	L4
Coach Lane	*Male*	Mean	16.33	17.51	20.45	21.83	21.82	21.63	21.90
		N	7	9	7	9	11	12	11
		S.E.	0.39	0.51	0.76	0.67	0.57	0.48	0.55
	Female	Mean	17.48	18.9	20.74	22.18	21.60	21.40	22.34
		N	4	4	6	7	7	8	6
		S.E.	0.42	0.20	0.47	0.39	0.36	0.40	0.52
Coronation Street	*Male*	Mean	17.71	19.64	20.75	22.56	22.63	23.30	23.14
		N	7	6	9	8	8	6	6
		S.E.	0.90	1.31	-	0.63	0.60	0.70	0.89
	Female	Mean	16.32	18.7	-	23.47	23.07	23.66	24.14
		N	4	4	-	4	5	6	5
		S.E.	0.48	0.88	-	1.91	1.45	1.76	2.45
Chelsea Old Church	*Male*	Mean	17.38	17.18	19.63	20.87	21.32	20.97	21.05
		N	3	3	4	4	5	6	6
		S.E.	0.18	1.29	1.10	1.46	1.26	1.06	0.86
	Female	Mean	17.27	18.60	20.06	20.72	20.52	20.02	20.37
		N	6	6	5	5	6	5	6
		S.E.	0.80	0.94	0.90	0.54	0.39	0.52	0.35
St Benet Sherehog	*Male*	Mean	17.33	18.76	21.99	23.33	23.53	23.74	23.30
		N	4	3	4	6	7	7	4
		S.E.	0.52	0.41	0.38	0.46	0.54	0.66	1.15
	Female	Mean	17.53	18.13	20.25	21.34	20.43	21.43	22.15
		N	3	4	4	5	3	4	4
		S.E.	1.14	0.67	0.73	0.47	0.32	0.87	0.85
Bow Baptist	*Male*	Mean	17.65	18.44	20.60	22.97	22.85	22.52	22.63
		N	8	9	10	9	8	11	12
		S.E.	0.48	0.30	0.28	0.26	0.16	0.47	0.62
	Female	Mean	17.10	18.04	20.16	21.22	21.38	21.20	20.81
		N	21	21	20	20	24	22	20
		S.E.	0.34	0.37	0.41	0.24	0.29	0.27	0.28
Cross Bones	*Male*	Mean	15.95	16.95	19.11	20.79	20.68	20.70	-
		N	2	2	2	2	2	2	-
		S.E.	0.98	0.32	0.07	0.70	0.14	0.36	-
	Female	Mean	15.39	17.48	16.93	19.02	20.32	20.42	20.80
		N	3	2	2	2	2	2	2
		S.E.	0.45	0.70	1.59	1.79	0.44	0.14	-

Table 6.6 - *ANOVA results for sex comparisons of adult TR diameters. p=<0.05, significant values in bold.*

	T10		T11		T12		L1		L2		L3		L4	
	F	***p***	**F**	***p***	**F**	***p***	**F**	***p***	**F**	***p***	**F**	***p***	**F**	***p***
Coach Lane	3.495	0.094	3.020	0.110	0.097	0.761	0.175	0.682	0.084	0.776	0.120	0.733	0.271	0.610
Coronation Street	1.194	0.303	0.278	0.612	-	-	0.331	0.578	0.107	0.750	0.036	0.853	0.172	0.688
Chelsea Old Church	0.009	0.926	0.780	0.406	0.094	0.768	0.010	0.923	0.434	0.526	0.560	0.473	0.529	0.484
St Benet Sherehog	0.032	0.865	0.527	0.500	4.464	0.079	9.035	**0.015**	12.529	**0.008**	4.498	0.063	0.644	0.453
Bow Baptist	0.757	0.392	0.441	0.512	0.510	0.481	18.569	**0.000**	8.239	**0.007**	6.873	**0.013**	9.161	**0.005**
Cross Bones	0.364	0.589	0.474	0.562	1.876	0.304	0.857	0.452	0.608	0.517	0.525	0.544	-	-

infants within the middle status groups. However, due to the stronger immune response in females, alongside social buffering, females within middle status households may have been more likely to survive episodes of stress experienced during infancy (Bourman *et al.,* 2005; Furman *et al.,* 2014).

In this case, those with deficiencies in TR diameter would be more likely to survive to adulthood. Whereas, due to inherent frailty in males, and the adverse conditions experienced by those in the lower classes, within these groups only those robust enough to overcome early stressors (or those who did not experience growth disruption in infancy) survived to adulthood. For these groups it is therefore more likely that survivors would have larger TR diameters.

While overall there was no persuasive evidence of maternal disadvantage within the adult data, apart from mortality profiles suggestive of the risks that accompanied pregnancy and childbirth, there still remains compelling evidence of poor maternal health within the non-adult data. This is evident in the presence of growth restriction and high prevalence of deficiency diseases seen in infancy in the Cross Bones and Coach Lane non-adult samples. Vitamin D deficiency has been shown to be a particularly strong influence on population health within the Coach Lane collection, and as demonstrated in section 6.3, the female sample from Coach Lane had the lowest stature of all the sites. Women of the industrial cities of the North and London were generally shorter in a study of female prisoners from 1817-1876 (Johnson and Nicholas, 1997). It was felt that this reflected on the inequalities in living standards for women in industrialising areas (Johnson and Nicholas, 1997).

Approximately 37% of non-adults in the Coach Lane sample demonstrated skeletal changes associated with rickets or possible rickets, and 6% were aged between 0-1 years of age, which may be indicative of early cessation of breastfeeding. However, if mothers were indeed breastfeeding but were themselves vitamin D deficient, this deficiency would in turn be transferred to the infant (Shin *et al.,* 2010; Uriu-Adams *et al.,* 2013; NICE, 2014). The high rate of rickets within the non-adult sample, the high rate of residual rickets within the adult sample compared to the other sites, the overall high proportion of females aged 18-36 and males of all age categories affected by residual rickets within the sample, and evidence for potential osteomalacia within four adults within this sample (Tschinkel, 2013), indicates that this population were exposed to high risks of developing deficiencies in vitamin D. This may have had implications for future morbidity and mortality. A study of adolescent girls and elderly women living in northern Europe (Denmark, Finland, Ireland, and Poland) found that during winter approximately one-third of the adolescent girls demonstrated deficiencies in vitamin D and two-thirds of the elderly women in the sample (Anderson *et al.,* 2005). Suboptimal maternal vitamin D status has been associated with a heightened risks of preeclampsia, gestational diabetes, pre-term delivery, obstructed labour, miscarriage, and low birth weight babies (Shin *et al.,* 2010; Uriu-Adams *et al.,* 2013). Maternal vitamin D deficiency has also been connected with lower tibial total bone cross-sectional area in infants, with potential long term consequences for future growth trajectory (Uriu-Adams *et al.,* 2013). Disruption to increases in bone mass (as seen in the analysis of cortical thickness in this study) may influence the peak bone mass attainable at maturity, and this in turn may increase risk of osteoporosis and fracture in later life (Anderson *et al.,* 2005; Urnaa *et al.,* 2006; Pettifor and Prentice, 2011; Duren *et al.,* 2013). In addition, deficiencies in vitamin D can inhibit adaptive immunity, thus increasing risk of contraction of infectious disease (Uriu-Adams *et al.,* 2013). It has also been associated with chronic diseases, such as cardiovascular disease, diabetes, arthritis, multiple sclerosis, and cancer (Huotari and Herzig, 2008; Uriu-Adams *et al.,* 2013). Thus, there are potentially long-term implications for both bone health, and overall well-being, in populations exposed to a heightened risk of vitamin D deficiency, particularly when such deficiencies occur from such an early age.

This data reinforces the importance of the analysis of growth and pathology data within non-adult samples, as they not only reflect child health at this time, but also the health of the mother. It is well documented that women experienced significant health inequalities in the 18th and 19th centuries, particularly those of the lower classes. However, from adult data alone, the magnitude of the impact that this disadvantage could have had on future generations cannot be fully appreciated.

6.4.3 Modern-day perspectives

"Rich people would doubtless continue to be; they would only be less rich by contrast with the common lot of humanity. Their social functions would remain what they are now, and they would fill their place more usefully and profitably, and above all more happily, under a state of things which would secure the final divorce of poverty from labour." Charles Booth on equality (1889: 171).

The success of the higher and middle classes depended on the employment of the working classes to produce capital, and in turn the working classes depended on the wages earned to support their families (Engels, 1950). However, despite this symbiotic relationship, it was only the lower classes that lived within conditions of extreme poverty (Kay, 1832; Gaskell, 1833; Engels, 1950). Many members of the upper and middle classes in the 18th and 19th centuries viewed the deterioration of the urban environment and high levels of morbidity and mortality amongst the lower classes to be the fault of the poor, arising from their idleness and low morals (Hudson, 1992). It was Chadwick's (1842) belief that improvements to sanitation, drainage, and clean water supplies would improve health and would be of benefit to all of society. However, such improvement was expensive, and as in many unequal societies today, there existed an aversion by the upper classes to the plight of the lower classes, with the belief that many existed in this position due to deficiencies in their moral character (Booth, 1889; Engels, 1950; Wilkinson and Pickett, 2010; Kirby, 2013). However, the root causes of the majority of cases of poverty were due to issues of employment and family circumstance rather than idleness, drink, or a "lack of thriftiness" (Booth, 1889). This inequality and class aversion meant that issues of sanitation did not readily improve; disease flourished and spread, and ultimately risked the health of all children, regardless of class. In this way, the social system of the 18th and 19th centuries, by which the few gained at the expense

of the many, ensured that vast numbers of families were being trapped in poverty, propagating severe health inequalities that were inherited by future generations. The cyclic interaction of poor health on human capital attainment (such as productivity and educational outcome), and availability of resources on recovery rates following poor health, can generate "poverty traps" that are difficult to overcome (Ngonghala *et al*., 2014). It is clear that the "poverty traps" that were set in the 18th and 19th centuries have persisted due, in part, to the dangerous cycle of malnutrition, poor health, and intergenerational poverty.

There has been a rising income inequality within developed countries in the past four decades, and in 2010 the UK represented one of the most unequal societies in the world, alongside the USA, Singapore, and Portugal (Wilkinson and Pickett, 2010; Aizer and Currie, 2014; Elgar *et al*., 2015; Pickett and Wilkinson, 2015). As already stated, social inequality can influence birth-weight, life expectancy, future health prospects, and cognitive and social development. In a recent government study, children from lower income families performed less well in cognitive tests than children from higher income families at five years of age (McKnight, 2015). Children from higher income families were also more likely to have higher income jobs later in life, regardless of whether they scored highly on the cognitive skill assessments (McKnight, 2015). This study emphasised that upward mobility in lower status families was not just limited by fewer opportunities to accumulate transferrable skills necessary for the labour market, but also by a "glass floor" effect that protected low attaining children from wealthier families from downward mobility, thus reducing employment opportunities for those lower on the income scale (McKnight, 2015). Class inequalities and marginalisation of the poorest groups also leads to enhanced psychosocial stress, with increased chronic disease risk (Marmot and Wilkinson, 2006).

According to Booth, in 1891 St. Saviour's Southwark (parish of Cross Bones burial ground) sat on a "wretched throne" of poverty (1891: 395). Over 100 years after Booth first produced his poverty maps of London, many areas still remain zones of marked social and health inequality (Dorling *et al*., 2000). The "…fundamental relation between spatial patterns of social deprivation and spatial patterns of mortality is so robust that a century of change in inner London has failed to disrupt it" (Dorling *et al*., 2000: 1). In the 1980s a series of UK Government

reports highlighted the continuing existence of marked social and health inequalities within the UK (Townsend *et al*., 1992). *The Black Report* revealed that the risk of death at birth and during the first month of life in families of unskilled workers was double that of professional families (Townsend *et al*., 1992: 43). Marked class differences in the occurrence of cancer, heart disease, and respiratory disease also existed amongst adults, which has an intriguing link to the DOHaD hypothesis, as early life stressors are believed to heighten risks of these chronic diseases (Townsend *et al*., 1992; Barker and Osmond, 1986; Barker, 1992,1994). *The Health Divide* exposed the inadequate response by the government to the findings of *The Black Report* (Townsend *et al*., 1992). The importance of giving children the best start to life was highlighted as a key means of tackling health inequalities (Townsend *et al*., 1992). These findings were reiterated by the more recent Marmot Review (Department of Health, 2010), which argued powerfully that 'health is a matter of social justice'. However there has been a general resistance to tackle health inequalities in any substantive and sustained way. In arguments strikingly reminiscent of Victorian sentiments, factors such as risky behavior (e.g., smoking, drinking) and idleness amongst the poor are still deemed responsible for health inequalities today, rather than social injustice (Townsend *et al*., 1992). A recent calculation indicates that 2.3 million children in the UK were classed as living in relative poverty in 2013-14 (Shale *et al*., 2015) and this is forecast to rise over the next few years. The life-long consequences of child poverty for health suggests that a lack of early-life intervention will be costly in terms of future population well-being and economic resources.

6.5 Accessing child health in bioarchaeology: evaluating new and existing methodologies

The study of growth has progressed substantially in recent years, with a greater appreciation of the necessity to view growth as not just a discrete experience of childhood, but as a contributory determinant of future adult health and a vital component of the life course. Traditionally bioarchaeological analysis has sought to access growth, and by proxy health, in the past through evidence of disruption to diaphyseal length. Through this methodology research has revealed a wealth of information such as the impact of urbanisation and industrialisation on population

health, shifts in subsistence to agriculture, influences of social status on health, and interactions with disease processes (Hummert, 1983; Van Gerven *et al*., 1985; Ribot and Roberts, 1996; Lewis, 2002a,b; Pinhasi *et al*., 2006; Mays *et al*., 2008, Mays *et al*., 2009a,b; Cardoso and Garcia, 2009; Ruff *et al*., 2013; Pinhasi *et al*., 2013; Robbins Schug and Goldman, 2014). However, it is now prudent to seek additional avenues to access evidence of growth disruption, to fully encompass the variety of ways in which such disruption can manifest within the skeletal remains of individuals and populations (Ruff *et al*., 2013).

Novel methodologies that have sought to gain additional insight into the osteobiographical histories of past populations are continuing to increase within the bioarchaeological literature, such as the re-introduction of measurement of VNC diameter (Watts, 2011, 2013a,b, 2015), measurement of appositional growth disruption through cortical thickness (Mays *et al*., 2009a), measurement of compact bone geometry (Robbins Schug and Goldman, 2014), and incremental isotopic analysis of dentine (Beaumont *et al*., 2013b; Beaumont *et al*., 2015; Henderson *et al*., 2014). The bioarchaeological tool-kit for the analysis of non-adult skeletal remains has grown considerably, and it is now time to seek to implement such methodologies into wider research whenever possible -

Taking advantage of the full range of possible skeletal growth analyses is essential both for reconstructing past health and behavior, and for interpreting variation in juvenile morphology in the archaeological and human fossil record. (Ruff et al., 2013: 34)

To attempt to gain an appreciation of the diversity of the childhood experience amongst urban children of the 18th and 19th centuries, this study has used a combination of existing, underused, and novel methodologies to assess growth. As will be described below, the combination of measurements of diaphyseal length, cortical thickness, and vertebral growth have proven to be effective indicators of child health in the past, revealing intriguing links to the interaction of social status, environment, and health. However, it is essential to assess the efficacy of applying each of these techniques reliably to skeletal populations. This can be achieved through the analysis of intra-observer error. As can be seen in Table 6.6, all four growth parameters implemented in this study have a coefficient of reliability of

around 0.99, this means that there is an extremely low probability that any variances observed within the data are due to human error (Goto and Mascie-Taylor, 2007).

It is the combination of these methods that has allowed such a comprehensive analysis of growth within this study. The use of multiple indicators of stress has long been supported within bioarchaeological study (Goodman *et al*., 1984), and this is becoming increasingly attainable within the study of growth.

Table 6.7 – *Intra-observer error scores for each measurement, where R= coefficient of reliability (0= not reliable, 1=completely reliable).*

	Tibial length	**Femoral CT**	**TR diameter**	**Vertebral body height**
R	0.99998	0.99743	0.9999	0.99984

The Bow Baptist population, despite demonstrating relatively higher growth values in childhood for diaphyseal length and cortical thickness, appear to have experienced deficiencies in adolescence, and this was only evident through measurement of vertebral body height. While Coach Lane demonstrates some of the highest values for diaphyseal length out of the samples in this study, this group also show notable deficiencies in femoral CT. Therefore, Coach Lane appears to have maintained diaphyseal length at the expense of cortical thickness. This corroborates the findings by Mays *et al*. (2009b), that measurements of cortical thickness are more sensitive to episodes of disruption than diaphyseal length. Coronation Street, despite demonstrating a low prevalence of pathology shows significant deficiencies in growth, revealing episodes of stress with no overt manifestation on the skeleton. Thus, non-adults of all sites suffered in infancy, as corroborated by the deficient growth values seen for TR diameter, but how children fared beyond this period of the life course was site dependent, and highly socially influenced. Therefore, this combination of growth parameters revealed differential timings of growth disruption throughout the sites throughout infancy, childhood, and adolescence.

While infancy is a particularly vulnerable phase, being a vital period for future health and survival prospects, adolescent health is often overlooked. Recent studies have sought to access crucial developmental stages within the growth period, such as when puberty begins, and ends (Shapland and Lewis, 2013; Lewis *et*

al., 2015). Lewis *et al*. (2015) have identified delays in both onset of menarche in females, and delays in completion of maturation in males, within a sample of adolescents from urban medieval sites in London. Such delays were potentially related to physical exertion, nutritional deficiencies, and/or episodes of infection (Lewis *et al*., 2015). These developing techniques therefore have scope to significantly further our knowledge regarding adolescent health in the past. In a socio-cultural context, during the 18th and 19th centuries late childhood and adolescence took a variety of forms depending on social status. Within the lower classes, from roughly nine years of age children would have been expected to work to help support the family economy (Pike, 1966; Pinchbeck and Hewitt, 1973; Humphries, 2010), thus exposing them to new occupational and environmental hazards (Kirby, 2013). There are references to both instances of premature and delayed puberty amongst factory children (Gaskell, 1833; Engels, 1950). However, entrance in the labour market may have brought benefits, such as increased agency, and contributions to the family budget would bring with it greater bargaining rights for food allocation (Horrell and Oxley, 2012). In contrast, within this stage of the life course children of the middle to higher classes may have experienced a more protected, and more limited sphere. Thus, it would be of interest to assess pubertal stages of maturation within skeletal collections dating to the 18th-19th centuries of differing social status. Therefore there is still much to learn regarding the impact of such varieties in life on the development of older children within urban regions of the 18th and 19th centuries.

A wider application of vertebral body height measurements to the analysis of skeletal collections is also now encouraged. It would also be of benefit to test this technique on populations of individuals of known age and medical background. This would provide valuable comparative data from healthy individuals, and would enable a deeper understanding of the level of growth disruption experienced within the vertebral column under conditions of stress. Manuscript One revealed potential evidence that the vertebral column can undergo catch-up growth into early adulthood, up until it completes growth at approximately 18-24 years of age (Bick and Copel, 1950; Scheuer and Black, 2000). Further study is therefore needed to ascertain what effect this may have on the estimation of stature, and the analysis of body proportions.

Manuscript Four also demonstrated the benefits of manipulating pathology data in new ways to enhance our understanding of the interactions of disease and growth disruption in the past. This study has not only considered prevalence of pathology within the six sites, but also the impact of co-morbidity on health and growth. There are many common co-morbidities, such as scurvy and rickets, and scurvy and cribra orbitalia (Weinstein *et al.,* 2001; Wapler *et al.,* 2004; Walker *et al.,* 2009; Stark, 2014; Agarwal *et al.,* 2015; Ferrari *et al.,* 2015), and such interactions clearly had a greater impact on growth in the Coach Lane and Bow Baptist collections. Urban centres of the 18th and 19th centuries are associated with high pathogen loads, and nutritional stress, therefore such co-morbidities would be expected to occur more frequently in such an insanitary environment. Through this type of analysis not only presence of pathology, but also intensity and disease load can start to be considered within past populations. Whether due to environmental hazard, social practice, or a combination of the two factors, Coach Lane, Cross Bones, and Chelsea Old Church demonstrated some of the highest health risks out of all the sites in this study.

6.6 Limitations

The analysis of growth and pathology within skeletal collections is not without limitation, and this must be considered in reference to the results of this study. The production of growth profiles provided the primary means of assessment of child health within this thesis, however such analysis comes with the caveat that the data output is representative of a cross-sectional sample, rather than a longitudinal study of growth within a population (Saunders, 2008; Gowland, 2006; Klaus, 2014b; DeWitte and Stojanowski, 2015). While this allows for an assessment of which samples demonstrated the most deficient growth values when compared to dental age, it cannot be ascertained how these individuals would have continued to grow had they survived (Wood *et al.,* 1992; Saunders, 2008). This is particularly important in discussions of catch-up growth. While the "improved" growth values evident for tibial diaphyseal length and femoral CT within the Chelsea Old Church sample can indicate the presence of catch-up growth following early growth deficits, catch-up growth is a longitudinal process. Therefore it is impossible to differentiate between individuals that did indeed experience catch-up growth, and

those who simply did not experience poor health in infancy.

Cemetery samples by nature often encompass a wide temporal range. As a result, individuals interred at the beginning of the cemetery usage may have experienced vastly different conditions to those buried prior to closure. Periods of food shortages, epidemic disease, and/or fluctuations in environmental conditions can rarely be accounted for (Wood *et al.,* 1992; DeWitte and Stojanowski, 2015). This becomes particularly relevant when considering the fast paced decline in environmental conditions of 18th/19th century urban centres. The adult samples within the six sites also represent those who survived infancy, childhood, and adolescence, therefore either were not exposed to the harsher social and/or environmental stressors that lead to the premature mortality of the non-adult sample, or were simply physiologically more robust (Wood *et al.,* 1992; DeWitte and Stojanowski, 2015). This may explain the relatively inconclusive results regarding the impact of early life stressors on adult longevity (see section 6.3). Additionally, post-medieval cemeteries, particularly within urban centres, are likely highly heterogenous. Migration from rural areas to the growing cities was commonplace at this time, as more of the population sought new employment opportunities provided by the developing industry (DeWitte *et al.,* 2015). This means that it is possible that the older children, and adults, within these samples could have lived elsewhere during infancy and early childhood, therefore may have experienced different health risks.

The "osteological paradox" has already been discussed within this thesis in reference to the differential manifestation of pathological indicators of stress within Coach Lane and Coronation Street (see Manuscript Three). The issue of "survivors" versus "non-survivors" as petitioned by Wood *et al.* (1992) is also relevant to the assessment of growth profiles, and the impact of childhood stress on future adult health. Non-adult samples represent "non-survivors", therefore those who were more vulnerable to premature mortality due to a myriad of factors such as chronic illness and/or socio-cultural circumstance (Wood *et al.,* 1992). Therefore the growth profiles formed for these individuals are representative of the "weakest" members of the population under study, and may not be reflective of the experience of the general population who survived to maturity. Selection pressures, such as poor environmental conditions, act on the weakest members of a population (DeWitte

and Stojanowski, 2015). These non-adult individuals are therefore more likely to demonstrate evidence of growth stunting than their more robust peers. However, a study by Saunders and Hoppa (1993) found that differences in growth between "non-survivors" and those who died at an older age are likely to be small. It has also been suggested in recent studies (DeWitte, 2014, DeWitte *et al.,* 2015) that the analysis of pathological indicators of stress should include differentiation between active and healed lesions. This could allow for the identification of populations that were more susceptible to early mortality in response to stress (i.e. those with a high prevalence of active lesions), and those that were able to buffer stressors more efficiently (i.e. those with a higher prevalence of healed lesions). While this type of analysis was not possible within this thesis, it does provide a future avenue for study for these populations, particularly to determine whether the wealthier populations demonstrated a higher rate of healed lesions when compared to lower status groups.

While the author collected the majority of the data presented within this thesis, pathology data, and tibial lengths for skeletal collections based at the Museum of London were taken from the WORD database, therefore were collected by other researchers during the original analysis of these sites. Precautions were taken during data collection for this study to limit any potential interobserver error that may haven arisen from this, such as the implementation of the same age categorisations and standards for skeletal analysis as used by the Museum of London (Powers, 2012). However, as previously noted in Section 5.7.2 in reference to scurvy, diagnostic criteria for pathology may have changed in the time between the original analysis and the present study. While this is not a significant issue for the majority of the sites as the original skeletal recording occurred at a relatively recent date, the site report for Cross Bones was published in 1998 (Brickley and Miles, 1999). For this reason, detailed descriptions of pathological changes were assessed from the relevant WORD databases in line with more recent publications (see Section 5.7.2). Additionally, reliance on the WORD databases for recording of pathology for the London-based sites meant that differentiation between active and healed lesions could not be made. Therefore this level of analysis was not included within the present study, but would make a valuable contribution to this research in future studies.

The use of modern data (Maresh, 1955, Virtama and Helelá, 1969, and

Hinck *et al*., 1966) as comparative samples for archaeological data should always be approached with caution. These measurements were taken from the radiographs of living subjects, so it is possible that some of the measurements may be amplified due to radiographic enlargement. For the comparisons of cortical thickness, this amplification may not be a significant issue as the measurements from archaeological samples were also taken from radiographs. However, it may be more problematic when comparing the data from Maresh (1955) to data recorded using an osteometric board. In an attempt to compensate for this amplification in the Maresh data set, the mean values for each age category were reduced by 1.5% (as suggested by MacCord, 2009).

Lastly, the level of preservation required to enable metric analysis within the non-adult sample meant that sample sizes were frequently small. This is an issue inherent within bioarchaeology and should not deter the implementation of this type of analysis. The use of multiple indicators of stress and growth parameters can ensure that the maximum number of non-adults within a sample can be assessed, as while one individual may not have sufficient preservation of the long bones, they may still have enough vertebrae present for metric analysis. The small sample sizes within many of the excavated cemeteries of this study also mean that they are only representative of a "snap-shot" of child health in England at this time. Further study is required on other 18th/19th century skeletal collections from differing geographic location and socio-economic status to continue to broaden our knowledge regarding the experience of childhood during this period.

6.7 Summary

To summarise, urban centres of the 18th and 19th centuries had a severe impact on child health, this is well documented in historical research, but is also evident in the analysis of growth disruption and palaeopathological data within this study. While geographical location appeared to have little impact on child health, indicating that northern urban centres were just as detrimental to child health as London, social status reigned as the most significant factor in determining infant health and future developmental potential.

All sites demonstrated evidence of experiencing significant early life stressors, which inevitably affected health status, growth, and survival prospects.

Following the precarious period of infancy, wealth did seem to buffer the more severe effects of the urban environment, as seen in the normal "attritional" mortality profile within the adult sample of Chelsea Old Church. However, the remaining sites did reveal tentative evidence of reduced life-span, with a much smaller proportion of adults surviving to ">46 years", and peaks in age-at-death between 26-35 years and 36-45 years, often associated with DEH and cribra orbitalia (conditions associated with childhood stress). This may be related to the reduction in life expectancy associated with life in the urban centres at this time, or may also indicate the influence of early life stress on adult longevity as suggested by the DOHaD hypothesis.

Assessment of female disadvantage was somewhat inconclusive amongst the adult samples. While women undoubtedly experienced a higher risk of health inequality at this time, particularly amongst the lower classes, it was only the increased risk of death between 18-25 years and 26-35 years as seen in the mortality profiles of some of the sites that was indicative of relative female disadvantage. This, however, may also be related to risks that accompanied pregnancy and childbirth. It was only through the assessment of infant health within the six sites, using growth parameters and non-specific indicators of stress, that maternal health disadvantage was conclusively evident, highlighting the importance of non-adult health analysis for accessing not only child health in the past, but also other stages within the life course.

CHAPTER SEVEN

Conclusion

7.1 Research aims

This study aimed to undertake a comprehensive analysis of childhood health in post-medieval England, thus constructing a broader appreciation of child health across geographical and social contexts within the urban sphere. It also aimed to utilise and evaluate emerging methodologies for the detection of poor health within skeletal remains, to determine whether new approaches such as these can enrich our knowledge of child health in the past. The research questions outlined in the introduction to this thesis will be revisited to determine whether these aims have been met.

7.2 Research questions

1. **Are there observable differences in the skeletal indicators of poor health in non-adults between Northern and Southern populations of seemingly similar environmental background?**

There were no significant differences in growth values (tibial diaphyseal length, femoral CT, vertebral body height, and TR diameter) overall between northern-based and London-based skeletal collections within this study. This suggests that northern urban centres in the $18^{th}/19^{th}$ century experienced the same well-documented declines in sanitation, over-crowding, and spread of disease as that seen in London. Historical documentation also attests to poor conditions in the North. Urban centres of this time had a substantial impact on morbidity and mortality of those less than five years of age, and this is also evident in the skeletal data. However, a regional specific risk of vitamin D deficiency was identified within the Coach Lane sample, whether due to northern latitude, social factors (such as child-care practices and/or child labour practices), or a combination of the two.

2. **Are there observable differences in the skeletal indicators of poor health in non-adults from differing socio-economic backgrounds?**

Socio-economic status was identified as being the primary influence on child health

within this study. Lower status groups did demonstrate some of the most deficient growth values, and high levels of pathology. Severe deficiencies alongside high prevalence rates of metabolic disease in infancy were suggestive of poor maternal health and inherited health deficits related to health inequalities propagated by the social system of the 18th/19th centuries. However, Chelsea Old Church, despite their wealthier status and semi-rural location, also demonstrated some of the most significant growth and health deficits, particularly in infancy. These originated from "fashionable" child-care practices at this time, such as early onset of weaning, the use of artificial infant feeds, and keeping children indoors. St Benet Sherehog and the Bow Baptist sample generally demonstrated higher growth values and comparatively lower rates of pathology, which may indicate that growing up in a middle class family afforded a "best of both worlds" approach to child care.

3. **At what stage of the life course do these skeletal indicators of poor health become apparent, and how might they relate to child-care and child labour practices?**

All six archaeological sites demonstrated deficiencies in growth values in infancy. Most notable were the deficits seen in TR diameter when compared to modern data. As this skeletal feature completes the majority of its growth between 1-2 years of age, these deficits represent growth disruption resulting from episodes of stress in infancy. Peaks in metabolic disease also occurred, in general, between 1-5 years of age, suggestive of onset of weaning and insufficient weaning diets. However, for some sites these peaks were much earlier, demonstrating a variety of weaning times during a time when breastfeeding was falling in popularity. Analysis of vertebral body height revealed deficiencies in growth between 9-16 years of age within the Bow Baptist sample, and 5-9 years of age within Coronation Street. These ages correlate with the normal age for entrance into the labour market (~9 years of age) at this time, so these deficiencies may be related to increased energetic demands associated with child labour practices. There is also putative evidence of child labour practices within the Coach Lane sample, which may be related to the heightened prevalence of rickets and "knock-kneed" individuals seen in this collection. However, conditions that may have led to growth stunting and vitamin D deficiency were not unique to the factory system, and such conditions may have

also been propagated by poor environment in the home.

4. **Do vertebral indicators of stress and measurements of cortical thickness provide a complementary understanding of the living environments and health status of non-adults within a population when combined with more commonly utilised techniques (e.g. long bone length, enamel hypoplasia, periosteal new bone formation, cribra orbitalia, and Harris' lines)?**

The four growth parameters utilised within this study have proven to be both reliable and effective for the assessment of child health and development. The combination of these four methods enabled not only the identification of significant deficiencies in growth when compared to modern data, but also for a commentary on the timings of growth disruption within each sample. The combination of measurement of TR diameter and vertebral body height can reveal differential timings in exposure to stressors within sites, with TR diameter being indicative of health in infancy, and vertebral body height being most sensitive to disruption in adolescence. As growth in vertebral height completes in early adulthood, catch-up growth can also be discussed when compared to average adult measurements from each site. Cortical thickness did demonstrate a propensity to act as a more sensitive indicator of stress than diaphyseal length, as suggested by Mays *et al*. (2009a). This is best displayed by the deficiencies seen in femoral CT values compared to the relatively higher tibial diaphyseal length values within Coach Lane. Therefore, this study revealed that a combination of these four growth parameters provided a deeper understanding of living environment and health status.

5. **Is there an association between evidence of growth disruption in past populations and the presence of indicators of stress and/or metabolic disease?**

Non-specific indicators of stress (DEH, metabolic disease, and cribra orbitalia) also corroborated the growth data, and could be combined to form site-specific explanations for the main influences on growth deficits. Growth disruption within the Bow Baptist site appeared to be more influenced by the presence of cribra orbitalia and DEH, indicative of some of the more general environmental and

dietary pressures associated with urban centres at this time. However, metabolic disease had a greater impact on child health and growth within the Coach Lane sample, potentially indicative of stronger social influences (such as child labour practices). Therefore analysis of the association between indicators of stress (particularly their co-morbidities), and growth disruption is of great value. However, Harris' lines remain of questionable value, while there did appear to be some association with indicators of stress, their origins still remain problematic and further study is required.

7.3 Summary

By identifying novel ways of approaching growth and palaeopathological analysis, this study has contributed to the growing corpus of research related to childhood and child health within bioarchaeology. The 18th/19th centuries were a dynamic time in British history, in which both the landscape and society underwent fundamental changes. While the field of historical research has approached the topic of childhood and child health from a myriad of geographic and social contexts, this has yet to be fully explored within bioarchaeological study. Through the analysis of under-represented groups (those living in northern centres of industry, and those of the middling to higher classes), this study has broadened current knowledge of the impact of industrialisation on child health during this time.

Through a combination of methodologies it has revealed vulnerabilities in the life course (particularly infancy) following which, if they survived, a child could either thrive or continue to experience deprivation, depending on the influence of social status.

As the wealth, power, cities, and population size of England grew, many of its inhabitants were left behind, predominantly within the lower classes. The declining health of the nation particularly affected the very young. These early life stressors had a significant impact on both the immediate health and survival prospects of the young, but also on future adult health. With the present high rate of children living within conditions of poverty in the UK, and further afield, studies in past populations can provide profound and crucial messages regarding the impact of social inequality on generations to come.

7.4 Suggestions for future research

The results of this study have provided many future avenues for research to continue to build on our knowledge of 18th/19th century health, and novel ways of assessing non-adult skeletal development (and disruption) within bioarchaeology –

- In order to further develop the new technique of measuring non-adult vertebral dimensions as indicators of stress, this method requires application to more skeletal collections of differing temporal and geographic contexts. It is also crucial to apply vertebral body height measurements to a non-adult collection of known medical background, so that standards for what are "normal" growth values versus deficient growth values can be established.
- While this study has implemented multiple indicators of stress and growth parameters to form a comprehensive approach to the study of child health, it would be of great interest to integrate these data with incremental isotopic analysis. These additional data would allow for an exploration of exact timings that episodes of stress occurred within the life course, and how these related to breastfeeding practices, or influences such as metabolic disease.
- More northern-based studies from skeletal collections of this time are necessary, along with those from western and South-Western regions of England to counteract the current bias towards London-based studies.
- Continuing to seek out new methods of assessing growth disruption within the developing skeleton would provide additional ways in which timings of episodes of stress can be assessed in greater resolution. In addition, the impact of variances in body proportion as a result of episodes of stress requires further research, as this study has identified the potential for catch-up growth in vertebral body height into early adulthood. Anthropological studies frequently use "sitting-height" as a measure of health in current populations, which highlights the importance of the vertebral column in the growth process and final adult stature.
- Adolescent health is frequently overlooked, and the results from the vertebral body height analysis are suggestive of differential exposure to stress throughout the six sites. Therefore it would be of interest to assess

pubertal timing, as Engels (1950) stated that the onset of puberty was often premature or delayed amongst factory workers.

- The most prominent outcome of this study was the impact of social status, and most importantly social inequality, on health. It is vital to continue to study social inequality in the past, from a diverse range of temporal and geographic contexts, to enable a better understanding of its origins and impact on populations today. In this way we may be able to begin to dig out the "roots of poverty".

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APPENDICES

Appendix 1 – Example non-adult recording form

Non-adult Recording Form						
Site:	**Site Code:**	**ID:**	**Radiographs?**			
Skeletal Age:	**Dental Age:**	**Age Category:**				
Pathology:	**DEH?**	**Cribra L**	**Cribra R**	**Metabolic?**	**HL?**	**Other:**
DENTITION						

DECIDUOUS

MFH										
DEH										
	e	**d**	**c**	**b**	**a**	**a**	**b**	**c**	**d**	**e**
	e	**d**	**c**	**b**	**a**	**a**	**b**	**c**	**d**	**e**
DEH										
MFH										

PERMANENT

MFH															
DEH															
	8	**7**	**6**	**5**	**4**	**3**	**2**	**1**	**1**	**2**	**3**	**4**	**5**	**6**	**7**	**8**
	8	**7**	**6**	**5**	**4**	**3**	**2**	**1**	**1**	**2**	**3**	**4**	**5**	**6**	**7**	**8**
DEH																
MFH																

P= present; E= erupting; X = lost antemortem; / = lost postmortem; NP = not present
U = unerupted.

Notes:	
DENTAL AGE:	**Age Category:**

Additional Measurements

CORTICAL THICKNESS

Bone	T	M	CT	CI		HL?
Humerus L						
Humerus R						
Femur L						
Femur R						
Tibia L						
Tibia R						
Radius L						
Radius R						
Ulna L						
Ulna R						

VERTEBRAL MEASUREMENTS

	Present?	VNC		VBH		
Bone		AP	TR	AVBH	PVBH	MVBH
C1						
C2						
C3						
C4						
C5						
C6						
C7						
T1						
T2						
T3						
T4						
T5						
T6						
T7						
T8						
T9						
T10						
T11						
T12						
L1						
L2						
L3						
L4						
L5						

LENGTH (mm)		R	L	Notes:
	Humerus			
	Radius			
	Ulna			
	Femur			
	Tibia			
	Clavicle			

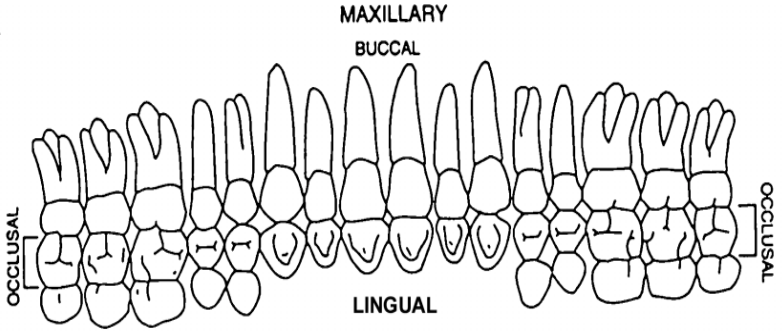
Appendix 2 – Example adult recording form

Adult Recording Form		
Site:	**Site Code:**	**ID:**
Pathology:		
Sex:	**Age Cateory:**	
VERTEBRAL MEASUREMENTS:		

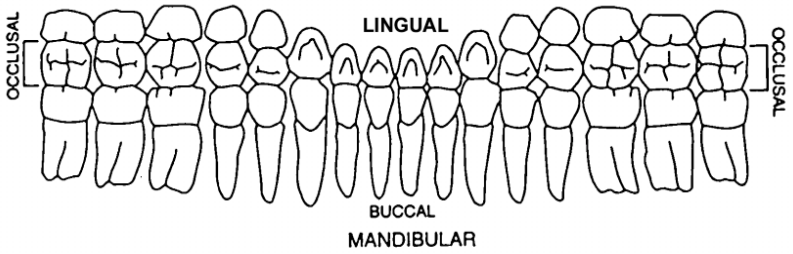
	VNC		VBH	Notes
	AP	**TR**	**MVBH**	
C1				
C2				
C3				
C4				
C5				
C6				
C7				
T1				
T2				
T3				
T4				
T5				
T6				
T7				
T8				
T9				
T10				
T11				
T12				
L1				
L2				
L3				
L4				
L5				

LENGTH (mm)		**L**	**R**	
	Humerus			
	Radius			
	Ulna			
	Femur			
	Tibia			
	Clavicle			

PERMANENT DENTITION



MFH															
DEH															
	8	**7**	**6**	**5**	**4**	**3**	**2**	**1**	**1**	**2**	**3**	**4**	**5**	**6**	**7**	**8**
	8	**7**	**6**	**5**	**4**	**3**	**2**	**1**	**1**	**2**	**3**	**4**	**5**	**6**	**7**	**8**
DEH															
MFH															



P= present; E= erupting; X = lost antemortem; / = lost postmortem;
NP = not present; U = unerupted.

Notes:

Appendix 3 – Sample sizes for adult metric data for each site

Table A3.1 – *Sample sizes for adult metric data. *Adults with measureable vertebral body height (BH) and TR (transverse diameter)*

Site	Overall no. Adults	Adults aged 18-25	Adults ages 26-35	Adults in metric sample	BH*	TR*
Coach Lane	151	12	39	29	26	29
Coronation Street	114	9	20	23	22	23
Chelsea Old Church	165	14	17	19	19	19
St Benet Sherehog	167	9	33	23	20	23
Bow Baptist	171	22	42	41	39	41
Cross Bones	44	3	4	7	7	7
Total	812	69	155	142	133	142

Appendix 4 – Breakdown of growth parameter sample sizes by age for each site

Table A4.1 – *Tibial diaphyseal length by age*

Tibial Length	Site					
Age	**Coach Lane**	**Coronation Street**	**Chelsea Old Church**	**St Benet Sherehog**	**Bow Baptist**	**Cross Bones**
0	4	12	2	3	8	20
1	3	-	2	1	11	5
2	3	2	3	-	14	4
3	2	1	1	1	5	2
4	-	-	-	-	2	3
5	2	-	-	1	6	2
6	-	1	-	-	3	-
7	1	1	-	-	4	-
8	1	-	-	-	1	-
9	-	1	-	-	-	-
10	1	-	1	-	4	-
11	-	-	-	1	5	-
12	1	-	-	-	1	-
13	1	-	1	1	-	-
14	1	-	-	-	-	-
15	-	1	-	-	3	-
16	2	-	-	1	1	-
17	-	-	-	-	2	-
Total	22	19	10	9	70	36

Table A4.2 – *Tibial CT by age*

Tibial CT	Site					
Age	**Coach Lane**	**Coronation Street**	**Chelsea Old Church**	**St Benet Sherehog**	**Bow Baptist**	**Cross Bones**
0	6	13	2	3	4	15
1	4	-	2	2	4	8
2	3	3	2	-	7	5
3	3	1	1	1	3	2
4	-	-	-	-	2	3
5	2	-	-	1	3	2
6	-	1	-	-	1	-
7	1	1	-	-	1	-
8	1	-	-	-	1	-
9	-	1	-	-	-	-
10	1	-	1	-	3	-
11	-	1	-	1	4	-
12	1	1	-	-	-	-
13	1	-	1	1	-	-
14	1	-	-	-	-	-
15	-	1	-	1	2	-
16	2	-	-	1	-	-
17	-	-	-	-	1	-
Total	26	23	9	11	36	35

Table A4.3 – *Femoral CT by age*

Femoral CT	Site					
Age	**Coach Lane**	**Coronation Street**	**Chelsea Old Church**	**St Benet Sherehog**	**Bow Baptist**	**Cross Bones**
0	3	18	2	2	4	15
1	3	-	3	5	10	8
2	3	4	2	-	10	6
3	1	1	1	1	3	3
4	-	1	-	-	3	3
5	1	-	-	1	3	2
6	-	1	-	-	3	-
7	1	1	-	-	1	-
8	1	-	-	-	1	-
9	-	1	-	-	-	-
10	1	-	1	-	2	-
11	-	1	-	1	4	-
12	2	1	-	-	-	-
13	1	-	1	1	-	-
14	1	-	-	-	-	-
15	-	1	-	1	2	-
16	2	1	-	1	1	-
17	-	0	-	-	1	-
Total	20	31	10	13	48	37

Table A4.4 – *Body height (BH) C5-6 by age*

BH C5-6	Site					
Age	**Coach Lane**	**Coronation Street**	**Chelsea Old Church**	**St Benet Sherehog**	**Bow Baptist**	**Cross Bones**
0	4	9	2	4	2	15
1	5	-	2	5	8	10
2	6	5	-	1	12	4
3	5	1	-	1	2	3
4	-	-	-	-	3	3
5	1	1	-	2	4	2
6	-	1	-	1	2	-
7	1	-	1	-	2	-
8	1	-	-	-	-	-
9	-	1	-	-	1	-
10	2	-	1	-	3	-
11	-	1	-	1	4	-
12	2	-	-	-	1	-
13	-	-	-	1	-	-
14	2	-	1	-	-	-
15	1	-	-	1	3	-
16	1	1	-	1	1	-
17	-	-	-	-	1	-
Total	31	20	7	18	49	37

Table A4.5 – *Body height (BH) T6-8 by age*

BH T6-8	Site					
Age	**Coach Lane**	**Coronation Street**	**Chelsea Old Church**	**St Benet Sherehog**	**Bow Baptist**	**Cross Bones**
0	8	11	2	5	-	14
1	3	-	2	4	7	10
2	3	2	1	1	12	5
3	4	1	1	1	2	4
4	-	-	-	-	3	3
5	1	1	-	2	4	2
6	-	1	-	1	1	-
7	-	-	1	-	3	-
8	-	-	1	-	-	-
9	-	1	-	-	1	-
10	1	-	1	-	2	-
11	-	-	-	-	3	-
12	1	1	-	-	1	-
13	-	-	-	-	-	-
14	1	-	1	-	-	-
15	-	1	-	2	1	-
16	1	1	-	-	1	-
17	-	-	-	-	1	-
Total	23	20	10	16	42	38

Table A4.6 – *Body height (BH) L2-4 by age*

BH L2-4	Site					
Age	**Coach Lane**	**Coronation Street**	**Chelsea Old Church**	**St Benet Sherehog**	**Bow Baptist**	**Cross Bones**
0	6	9	1	3	-	16
1	1	-	2	4	6	9
2	4	2	1	1	11	4
3	4	1	1	1	4	1
4	-	-	-	-	2	3
5	1	1	-	1	3	2
6	-	1	-	-	-	-
7	1	-	1	-	4	-
8	-	-	1	-	-	-
9	-	1	-	-	1	-
10	1	-	1	-	2	-
11	-	1	-	-	3	1
12	1	1	-	-	1	-
13	-	-	1	-	-	-
14	-	-	1	1	-	-
15	-	1	-	2	1	-
16	-	1	-	1	1	-
17	-	-	-	-	1	-
Total	19	19	10	14	40	36

Table A4.7 – *Transverse diameter (TR) C5-6 by age*

TR C5-6	Site					
Age	**Coach Lane**	**Coronation Street**	**Chelsea Old Church**	**St Benet Sherehog**	**Bow Baptist**	**Cross Bones**
0	-	-	-	-	-	-
1	-	-	1	3	5	3
2	-	4	1	-	9	3
3	2	1	-	2	2	2
4	-	-	-	-	3	3
5	-	1	-	1	4	2
6	-	1	-	1	2	1
7	1	-	1	-	3	-
8	1	-	-	-	-	-
9	-	1	-	1	1	-
10	1	-	1	-	3	-
11	-	1	-	1	4	-
12	2	-	-	-	1	-
13	-	-	-	1	-	-
14	2	-	1	-	-	-
15	1	-	-	2	3	-
16	2	1	-	1	1	-
17	-	-	-	-	1	-
Total	12	10	5	13	42	14

Table A4.8 – *Transverse diameter (TR) T6-8 by age*

TR T6-8	Site					
Age	**Coach Lane**	**Coronation Street**	**Chelsea Old Church**	**St Benet Sherehog**	**Bow Baptist**	**Cross Bones**
0	-	-	1	-	-	-
1	-	-	3	1	5	8
2	-	3	2	-	12	5
3	-	1	1	1	4	4
4	-	-	-	-	3	3
5	-	1	-	1	3	2
6	-	1	-	1	2	-
7	-	-	1	-	4	-
8	-	-	-	-	-	-
9	-	1	-	-	1	-
10	2	-	1	-	3	-
11	-	1	-	-	4	-
12	1	1	-	-	1	-
13	-	-	1	-	-	-
14	1	-	-	-	-	-
15	-	-	-	2	2	-
16	1	1	-	2	1	-
17	-	-	-	-	1	-
Total	5	10	10	8	46	22

Table A4.9 – *Transverse diameter (TR) L2-4 by age*

TR L2-4	Site					
Age	**Coach Lane**	**Coronation Street**	**Chelsea Old Church**	**St Benet Sherehog**	**Bow Baptist**	**Cross Bones**
0	-	1	-	-	-	1
1	1	-	1	1	4	4
2	1	2	1	1	10	4
3	2	-	1	1	4	1
4	-	-	-	-	3	3
5	-	1	-	1	2	1
6	-	1	-	-	2	1
7	1	-	1	-	4	-
8	-	-	1	-	-	-
9	-	1	-	-	1	-
10	2	-	1	-	3	-
11	-	1	-	1	4	1
12	2	1	-	-	1	-
13	-	-	1	-	-	-
14	2	-	1	-	-	-
15	-	1	-	2	2	-
16	1	1	-	1	2	-
17	-	-	-	-	1	-
Total	12	10	8	8	43	16

Appendix 5 – Summary statistics for adult pathology data – Overall

All	*N*	Residual Rickets	%	PNBF	%	Cribra Orbitalia	%	DEH	%
Coach Lane	151	53	35.1	54	35.8	60	39.7	86	57
Coronation Street	114	3	2.6	30	26.3	16	14	64	56.1
Chelsea Old Church	165	10	6.1	42	25.5	15	9.1	56	33.9
St Benet Sherehog	167	7	4.2	26	15.6	14	8.4	57	34.1
Bow Baptist	171	4	2.3	62	36.3	27	15.8	65	37.9
Cross Bones	44	2	4.5	32	72.7	26	59.1	31	70.5

Table A5.1 – *Overall pathology CPR (%) for whole adult sample. PNBF= periosteal new bone formation; DEH= dental enamel hypoplasia. Cribra orbitalia category includes individuals classed as stage 1 and above.*

18-25 years	*N*	Residual Rickets	%	PNBF	%	Cribra Orbitalia	%	DEH	%
Coach Lane	12	4	2.6	5	3.3	6	4	19	6.6
Coronation Street	9	0	0	0	0	4	3.5	8	7
Chelsea Old Church	14	0	0	5	3	3	1.8	8	4.8
St Benet Sherehog	9	0	0	0	0	2	1.2	4	2.4
Bow Baptist	22	0	0	8	4.7	9	5.3	-	-
Cross Bones	3	1	2.3	1	2.3	2	4.5	3	6.8

Table A5.2 – *Overall pathology CPR (%) for adult sample 18-25 years. CPR (%) calculated as a percentage of the overall number of adults in each site, see Table A5.1.*

26-35 years	*N*	Residual Rickets	%	PNBF	%	Cribra Orbitalia	%	DEH	%
Coach Lane	39	17	11.3	12	8.6	14	9.3	28	18.5
Coronation Street	20	0	0	6	5.3	2	1.7	16	14
Chelsea Old Church	17	1	0.6	3	1.8	4	2.4	10	6.1
St Benet Sherehog	33	1	0.6	6	3.6	4	2.4	15	9
Bow Baptist	42	1	0.6	19	11.1	8	4.7	-	-
Cross Bones	4	0	0	4	9.1	2	4.5	4	9.1

Table A5.3 – *Overall pathology CPR (%) for adult sample 26-35 years. CPR (%) calculated as a percentage of the overall number of adults in each site, see Table A5.1.*

36-45 years	*N*	Residual Rickets	%	PNBF	%	Cribra Orbitalia	%	DEH	%
Coach Lane	33	12	7.9	18	11.9	13	8.6	22	14.6
Coronation Street	39	2	1.6	13	11.4	3	2.6	24	21.1
Chelsea Old Church	46	3	1.8	12	7.3	5	3	13	7.9
St Benet Sherehog	50	3	1.8	9	5.4	5	3	23	13.8
Bow Baptist	51	1	0.6	21	12.3	4	2.3	-	-
Cross Bones	18	0	0	13	29.5	12	27.3	14	31.8

Table A5.4 – *Overall pathology CPR (%) for adult sample 36-45 years. CPR (%) calculated as a percentage of the overall number of adults in each site, see Table A5.1.*

46+ years	*N*	Residual Rickets	%	PNBF	%	Cribra Orbitalia	%	DEH	%
Coach Lane	16	4	2.6	5	3.3	8	5.3	11	7.3
Coronation Street	24	1	0.9	9	7.9	4	3.5	10	8.8
Chelsea Old Church	72	5	3	22	13.3	6	3.6	20	12.1
St Benet Sherehog	32	3	1.8	6	3.6	4	2.4	12	7.2
Bow Baptist	37	0	0	11	6.4	2	1.2	-	-
Cross Bones	14	1	2.3	12	27.3	8	18.2	5	11.4

Table A5.5 – *Overall pathology CPR (%) for adult sample 46+ years. CPR (%) calculated as a percentage of the overall number of adults in each site, see Table A5.1.*

Appendix 6 – Summary statistics for adult pathology data – Females

All	*N*	Residual Rickets	%	PNBF	%	Cribra Orbitalia	%	DEH	%
Coach Lane	60	19	14.4	23	17.4	25	18.9	34	25.8
Coronation Street	52	1	1	12	11.8	11	10.8	28	27.5
Chelsea Old Church	74	5	3.3	21	13.8	8	5.3	25	16.4
St Benet Sherehog	46	4	3.1	8	6.3	6	4.7	19	15
Bow Baptist	91	0	0	25	15.5	14	8.7	-	-
Cross Bones	27	2	5.1	21	53.8	16	41	18	46.2

Table A6.1 – *Overall pathology CPR (%) for whole female sample. Includes those classified as F (female) and F? (possible female). CPR (%) calculated as percentage of total number of M, M?, F, and F? individuals within each site. PNBF= periosteal new bone formation; DEH= dental enamel hypoplasia. Cribra orbitalia category includes individuals classed as stage 1 and above.*

18-25 years	*N*	Residual Rickets	%	PNBF	%	Cribra Orbitalia	%	DEH	%
Coach Lane	6	2	1.5	2	1.5	4	3	6	4.5
Coronation Street	7	0	0	0	0	4	3.9	7	6.9
Chelsea Old Church	9	0	0	4	2.6	2	1.3	4	2.6
St Benet Sherehog	3	0	0	0	0	1	0.8	2	1.6
Bow Baptist	13	0	0	3	1.9	6	3.7	-	-
Cross Bones	1	1	2.6	0	0	1	2.6	1	2.6

Table A6.2 – *Pathology CPR (%) for female sample 18-25 years. CPR (%) calculated as a percentage of the total number of M, M?, F, and F? individuals within each site.*

26-35 years	*N*	Residual Rickets	%	PNBF	%	Cribra Orbitalia	%	DEH	%
Coach Lane	20	7	5.3	7	5.3	8	6.1	12	9.1
Coronation Street	8	0	0	2	2	2	2	7	6.9
Chelsea Old Church	7	0	0	1	0.7	1	0.7	4	2.6
St Benet Sherehog	15	0	0	4	3.1	2	1.6	6	4.7
Bow Baptist	27	0	0	10	6.2	5	3.1	-	-
Cross Bones	2	0	0	2	5.1	1	2.6	2	5.1

Table A6.3 – *Pathology CPR (%) for female sample 26-35 years. CPR (%) calculated as a percentage of the total number of M, M?, F, and F? individuals within each site.*

36-45 years	*N*	Residual Rickets	%	PNBF	%	Cribra Orbitalia	%	DEH	%
Coach Lane	13	4	3	8	6.1	5	3.8	7	5.3
Coronation Street	11	0	0	3	2.9	0	0	6	5.9
Chelsea Old Church	17	1	0.7	5	3.3	2	1.3	5	3.3
St Benet Sherehog	13	2	1.6	2	1.6	1	0.8	7	5.5
Bow Baptist	25	0	0	8	5	2	1.2	-	-
Cross Bones	11	0	0	8	20.5	7	17.9	9	23.1

Table A6.4 – *Pathology CPR (%) for female sample 36-45 years. CPR (%) calculated as a percentage of the total number of M, M?, F, and F? individuals within each site.*

46+ years	*N*	Residual Rickets	%	PNBF	%	Cribra Orbitalia	%	DEH	%
Coach Lane	4	0	0	3	2.3	1	0.8	2	1.5
Coronation Street	15	1	1	6	5.9	2	2	4	3.9
Chelsea Old Church	36	3	2	11	7.2	5	3.3	9	5.9
St Benet Sherehog	14	2	1.6	2	1.6	2	1.6	4	3.1
Bow Baptist	16	0	0	4	2.5	1	0.6	-	-
Cross Bones	12	1	2.6	11	28.2	7	17.9	4	10.3

Table A6.5 – *Pathology CPR (%) for female sample 46+ years. CPR (%) calculated as a percentage of the total number of M, M?, F, and F? individuals within each site.*

Appendix 7 – Summary statistics for adult pathology data – Males

All	*N*	Residual Rickets	%	PNBF	%	Cribra Orbitalia	%	DEH	%
Coach Lane	72	30	22.7	28	21.2	30	22.7	49	37.1
Coronation Street	50	2	2	16	15.7	5	4.9	35	34.3
Chelsea Old Church	78	5	3.3	20	13.2	5	3.3	30	19.7
St Benet Sherehog	81	3	2.4	13	10.2	8	6.3	37	29.1
Bow Baptist	70	3	1.9	31	19.3	11	6.8	-	-
Cross Bones	12	0	0	9	23.1	8	20.5	11	28.2

Table A7.1 – *Overall pathology CPR (%) for whole male sample. Includes those classified as M (male) and M? (possible male). PNBF= periosteal new bone formation; DEH= dental enamel hypoplasia. Cribra orbitalia category includes individuals classed as stage 1 and above.*

18-25 years	*N*	Residual Rickets	%	PNBF	%	Cribra Orbitalia	%	DEH	%
Coach Lane	6	2	1.5	3	2.3	2	1.5	3	2.3
Coronation Street	1	0	0	0	0	0	0	1	1
Chelsea Old Church	5	0	0	1	0.7	1	0.7	4	2.6
St Benet Sherehog	6	0	0	0	0	1	0.8	2	1.6
Bow Baptist	9	0	0	5	3.1	3	1.9	-	-
Cross Bones	2	0	0	1	2.6	1	2.6	2	5.1

Table A7.2 – *Pathology CPR (%) for male sample 18-25 years. CPR (%) calculated as a percentage of the total number of M, M?, F, and F? individuals within each site.*

26-35 years	*N*	Residual Rickets	%	PNBF	%	Cribra Orbitalia	%	DEH	%
Coach Lane	19	10	7.6	6	4.5	6	4.5	14	10.6
Coronation Street	12	0	0	4	3.9	-	-	9	8.8
Chelsea Old Church	9	1	0.7	2	1.3	1	0.7	6	3.9
St Benet Sherehog	16	1	0.8	2	1.6	2	1.6	8	6.3
Bow Baptist	14	1	0.6	9	5.6	3	1.9	-	-
Cross Bones	1	0	0	1	2.6	1	2.6	1	2.6

Table A7.3 – *Pathology CPR (%) for male sample 26-35 years. CPR (%) calculated as a percentage of the total number of M, M?, F, and F? individuals within each site.*

36-45 years	*N*	Residual Rickets	%	PNBF	%	Cribra Orbitalia	%	DEH	%
Coach Lane	18	7	5.3	10	7.6	7	5.3	14	10.6
Coronation Street	25	2	2	9	8.8	3	2.9	18	17.6
Chelsea Old Church	26	2	1.3	7	4.6	6	3.9	8	5.3
St Benet Sherehog	37	1	0.8	7	5.5	4	3.1	16	12.6
Bow Baptist	25	1	0.6	12	7.5	2	1.2	-	-
Cross Bones	6	0	0	5	12.8	4	10.3	5	12.8

Table A7.4 – *Pathology CPR (%) for male sample 36-45 years. CPR (%) calculated as a percentage of the total number of M, M?, F, and F? individuals within each site.*

46+ years	*N*	Residual Rickets	%	PNBF	%	Cribra Orbitalia	%	DEH	%
Coach Lane	12	4	3	2	1.5	7	5.3	9	6.8
Coronation Street	9	0	0	3	2.9	2	2	6	5.9
Chelsea Old Church	35	2	1.3	10	6.6	0	0	11	7.2
St Benet Sherehog	17	1	0.8	4	3.1	2	1.6	8	6.3
Bow Baptist	19	0	0	5	3.1	1	0.6	-	-
Cross Bones	1	0	0	1	2.6	1	2.6	1	2.6

Table A7.5 – *Pathology CPR (%) for male sample 46+ years. CPR (%) calculated as a percentage of the total number of M, M?, F, and F? individuals within each site.*

Please see CD provided for Appendices 8-10.

CD - Appendix 8 – Summary table for non-adult metric analysis for each site

CD - Appendix 9 – Summary table for pathological data for each site

CD - Appendix 10 – Summary table for adult metric analysis for each site