Spring 2019

The Intersections of Health and Wealth: Socioeconomic Status, Frailty, and Mortality in Industrial England

Samantha Lee Yaussy

THE INTERSECTIONS OF HEALTH AND WEALTH: SOCIOECONOMIC STATUS, FRAILTY, AND MORTALITY IN INDUSTRIAL ENGLAND

by

Samantha Lee Yaussy

Bachelor of Arts
Wake Forest University, 2013

Master of Arts
University of South Carolina, 2015

Submitted in Partial Fulfillment of the Requirements

For the Degree of Doctor of Philosophy in

Anthropology

College of Arts and Sciences

University of South Carolina

2019

Accepted by:

Sharon N. DeWitte, Major Professor

Carlina M. de la Cova, Committee Member

Katrina M. Walsemann, Committee Member

Andrew A. White, Committee Member

Cheryl L. Addy, Vice Provost and Dean of the Graduate School

DEDICATION

To Côa,

who did more than any human ever could.

ACKNOWLEDGEMENTS

I would like to express my deepest gratitude to my dissertation advisor, Sharon DeWitte, for her patience, direction, humor, and invaluable feedback throughout my graduate career. All of my successes to this point are due in no small part to your guidance and understanding. I would also like to thank my committee: Dr. Carlina de la Cova, Dr. Katrina Walsemann, and Dr. Andy White. Your encouragement and support have helped me persevere through the exhaustion of a doctoral dissertation, and your thoughtful questions, comments, and suggestions have immeasurably improved my project.

I am grateful for the assistance of several individuals who provided access to the skeletal collections used in this study: Jelena Bekvalac at the Museum of London, Don Walker at Museum of London Archaeology, Dr. Rebecca Gowland at Durham University, and Dr. Jo Buckberry at the University of Bradford. Also, my sincere thanks to David Westbury in the Department of Mechanical Engineering at the University of South Carolina, who trusted me to not irreparably damage the College of Engineering and Computing's sole Microscribe Digitizer. I am still not confident you knew I took it overseas, so belated thanks for that too. I am also grateful to Dr. Katherine Weisensee and Dr. Christopher Klingenberg, who provided helpful advice and training in geometric morphometrics. Collectively, they are probably the reason I did not break the College of Engineering and Computing's sole Microscribe Digitizer.

Thank you to the entities that funded my research and made this project possible: the National Science Foundation (Doctoral Dissertation Improvement Grant, BCS-1649757), the Office of the Vice President for Research at the University of South Carolina (SPARC Graduate Research Grant, Russell J. and Dorothy S. Bilinski Dissertation Fellowship), and the Department of Anthropology (travel grants and Ann Kingsolver Research & Professional Skills Award).

To Preston, Mattie, Ken, Joanna, and the many other vital members of the Department of Anthropology, thank you for your letters of reference, your written and spoken encouragement, and your confidence in me as a scholar. To my family and friends—Mom, Elise, Joshua, Tyler, Ben, Megan, other Joshua, Dr. Caroline Neely, grandparents, godparents, and so many more—thank you for your tireless support, and for pretending to listen while I waxed poetic about the importance of intersectionality. Lastly, this dissertation would not have been completed without the help of Indy, who put my heart back together when it had broken into a million pieces. Thank you.

ABSTRACT

Socioeconomic status (SES) is considered one of the most powerful predictors of mortality today. However, studies of health in living populations and bioarchaeological studies of health in the past often oversimplify the connection between SES and mortality and overlook heterogeneity in frailty within a population and the potential for multiple types of marginalization to be layered within a single individual. This dissertation project uses skeletal samples to examine the interactions of SES, demographic characteristics (e.g., age and sex), exposure to physiological stressors, and mortality in the context of industrialization in 18th- and 19th-century England. Skeletal data from four industrial-era cemeteries (St. Bride's Fleet Street, Coach Lane, St. Peter's Wolverhampton, and New Bunhill Fields) were analyzed with paleodemographic approaches (hazard modeling, hierarchical log-linear analysis, and analysis of variance) to (1) determine how morbidity and mortality patterns in industrial England differed between SES groups; (2) investigate how physiological stressors throughout life interacted with socially meaningful categories such as age and sex to produce layered marginalizations that influenced frailty and mortality in industrial England; and (3) evaluate the potential of stressors underrepresented in bioarchaeology to enhance our understanding of marginalization, intersectionality, and mortality in the past. Generally, the results of the hierarchical log-linear analyses and the analyses of variance suggest that marginalized identities (i.e., low SES or female sex) were associated with earlier ages at death. Furthermore, several skeletal indicators of early life stress (cribra orbitalia, tooth size, and the anteroposterior

diameter of the lumbar vertebral neural canal) were associated with low SES and earlier age at death, providing additional bioarchaeological support for the Developmental Origins of Health and Disease hypothesis. The results of the hazard analyses, the analyses of the transverse diameter of the lumbar vertebral neural canal, and the fluctuating asymmetry analyses underscore the importance of considering the issues associated with the Osteological Paradox (e.g., selective mortality) when interpreting patterns of frailty and mortality observed in skeletal samples. This project provides a novel model for exploring intersectionality and the effects of social identity in bioarchaeological studies.

TABLE OF CONTENTS

LIST OF TABLES

LIST OF FIGURES

LIST OF SYMBOLS

a	age in the Siler and Gompertz-Makeham models of mortality
α_1	the risk of death associated with immaturity in the Siler model of mortality
β_1	the rate at which the risk of death associated with immaturity declines with age in the Siler model of mortality
α_2	the age-independent component in the Siler model of mortality
α_3	the risk of death at birth associated with senescence in the Siler model of mortality
β_3	the rate at which risk of death at birth associated with senescence increases with age in the Siler model of mortality
α_1	the age-independent component of the Gompertz-Makeham model of mortality
α_2	the senescent component of the Gompertz-Makeham model of mortality
β	the rate at which risk of death associated with senescence increases with age in the Gompertz-Makeham model of mortality

LIST OF ABBREVIATIONS

ANOVA(s).....Analysis of Variance (pl. Analyses of Variance)

AP ..... Anteroposterior

CO..... Cribra Orbitalia

FA ..... Fluctuating Asymmetry

LEH.....Linear Enamel Hypoplasia

PNB.....Periosteal New Bone

SES..... Socioeconomic Status

TR ..... Transverse

CHAPTER 1

INTRODUCTION

Socioeconomic status (SES) has long been known to mediate exposure to hazards and disease, as well as access to those resources necessary for growth, development, tissue maintenance, and immune response (Floud, Wachter, & Gregory, 1990; Larsen, 1995; Martorell & Habicht, 1986; Nicholas & Steckel, 1991; Robb, Bigazzi, Lazzarini, Scarsini, & Sonego, 2001; Schell, 1997; Steckel, 2009; Stinson, 2000). Consequently, SES is considered one of the most powerful predictors of mortality (Saunders & Hoppa, 1993). However, the connection between SES and mortality is often oversimplified and overlooks heterogeneity in frailty within a population (as defined by Vaupel et al., 1979) and the potential for multiple types of marginalization to be layered within a single individual (Hancock, 2007). As a result, many investigations into SES and mortality in living populations are biased at best, and, in more extreme cases, could misinform health policy and treatment protocols about the risks faced by individuals in a variety of contexts.

Like modern public health studies (Bowleg, 2012; Hankivsky et al., 2010), studies of past population health should also be aware of intersectionality, and recognize that multiple inequalities are mutually constituted and cannot be understood by simplistic theoretical approaches and statistical methodologies that treat previous stress, sex, age, and SES as distinct subjects of inquiry. Further, we should avoid uncritically applying modern patterns of SES and health onto the past. This study addresses the limitations of

previous studies of SES in the past by incorporating statistical approaches that account for heterogeneity in frailty and interpreting the results in light of current theoretical perspectives espoused by public health researchers investigating the connection between SES and health in living populations. This project provides a model for exploring SES in past societies and broadens our understanding of the variable effects of SES across time by focusing on a context that differs from the modern, industrialized populations to which these methods have been applied. This study uses skeletal samples to examine the interactions of SES, demographic characteristics (such as age and sex), exposure to physiological stressors, and mortality in the context of industrialization in 18th- and 19th-century England.

1.1 Research objectives and hypotheses

This project uses paleodemographic approaches to achieve the following objectives:

Objective 1: Determine how morbidity and mortality patterns in industrial England differed between SES groups.

Objective 2: Investigate how physiological stressors throughout life interacted with socially meaningful categories such as age and sex to produce layered marginalizations that influenced frailty and mortality in industrial England.

Objective 3: Evaluate the potential of stressors underrepresented in biological anthropology literature to enhance our understanding of marginalization, intersectionality, and mortality in the past.

This dissertation focuses on the human skeletal remains interred in the crypts at St. Bride's Church on Fleet Street in London, the overflow cemetery of St. Peter's Collegiate Church in Wolverhampton, the Quaker cemetery (COL10) on Coach Lane in North Shields, and the New Bunhill Fields burial ground in London, which are curated at St. Bride's Church, the University of Bradford, Durham University, and Museum of London Archaeology (MoLA), respectively. Hazard analysis is used to compare age and sex patterns of mortality within and between different SES groups to evaluate differences in mortality during the industrialization process. Additionally, hierarchical log-linear analysis and 2- and 3-way ANOVA are used to analyze the interactions among social and biological factors (i.e., age, sex, and SES) and skeletal indicators of stress (craniofacial fluctuating asymmetry, periosteal new bone formation, linear enamel hypoplasia, tooth size, and vertebral neural canal size) to enhance our understanding of intersectionality and the developmental origins of health and disease in the past. This study uses these analytical approaches to test the following hypotheses:

> **Hypothesis 1:** Given the effects of differential resource access and hazard exposure associated with SES observed in living populations, risks of mortality and stress marker frequencies will be higher in the lower-SES group ("working" and "middle" classes combined) than in the higher-SES group ("upper" class).
>
> **Hypothesis 2:** Given that social and biological differences within SES groups can intersect to meaningfully influence the effect of socioeconomic marginalization, risk of mortality will be higher for children and adult females than for adult males in each status group.

Hypothesis 3: Given the potential for early life stress events to influence later adult mortality, stress markers reflecting active or early life stress events (i.e., linear enamel hypoplasia, fluctuating asymmetry, tooth size, vertebral neural canal size, and active periosteal new bone formation) will be more frequent in lower-SES group and those markers reflecting the accumulation of nonlethal stressors over a relatively long life (i.e., healed periosteal new bone formation) will be associated with the higher-SES group. Alternatively, these stress markers will be more strongly associated with earlier age-at-death in the lower-SES group, reflecting greater buffering against stress associated with higher SES.

1.2 Studies of SES and Health

1.2.1 Modern Studies of SES and Health

Scholars have long been interested in the connection between socioeconomic status (SES) and health. Early population health research recognized an inverse relationship between socioeconomic variables and risk of disease, but primarily focused on distinguishing stark differences in disease prevalence and mortality between the poorest and wealthiest classes without further investigation (Antonovsky, 1967). Eventually, scholars would identify a gradient in health that extended from the lower to the upper echelons of the status hierarchy (Adler & Ostrove, 1999; Marmot, Shipley, & Rose, 1984), and, by the mid-1980s, studies had also found evidence of graded decreases in infant mortality and chronic disease at higher levels of SES (Adler et al., 1994). Public health researchers also worked to identify specific diseases for which there was a

consistent negative relationship between SES and risk, such as tuberculosis (Cantwell, Mckenna, Mccray, & Onorato, 1998) and cardiovascular disease (Kaplan & Keil, 1993).

However, studies of SES and health faced the question of generalizability: Is the SES gradient consistent across all populations? By the 1990s, it was apparent that the patterns in health and disease observed in nations like the U.S. and Britain were not as pronounced (J. S. Feinstein, 1993), were reversed (Bunker et al., 1992), or disappeared altogether (Marmot, 1992) in other contexts. For example, the negative relationship between SES and coronary heart disease risk does not exist in some non-industrialized contexts (Marmot, 1992), and the health gradient by SES is reduced in relatively more egalitarian countries like Sweden and Norway (J. S. Feinstein, 1993). The connection between SES and health was complicated further when studies verified that the strength of the relationship also fluctuated within populations, particularly by race, age, or sex (Adler & Ostrove, 1999). For example, Williams and colleagues (2010) demonstrate that the SES-health gradient weakens or disappears entirely for some racial or ethnic groups. Examples include: "the Hispanic paradox", in which Hispanic immigrants of low SES exhibit levels of health that are equivalent or superior to whites of higher SES; the "diminishing returns hypothesis", in which Blacks do not exhibit the same improvements in health with increasing SES compared to whites; and a complete reversal of the association between SES and health for black men, in which education was associated with a poor lipid profile (high triglycerides, high LDL cholesterol, low HDL cholesterol) compared to black women and whites (Williams et al., 2010). In another study of mortality patterns in 19th-century Sweden, Edvinsson and Broström (2012) found no social gradient in health among the elderly (aged 60+ years).

Many studies of socioeconomic status and health in the last two decades have attempted to identify and understand the complex social and biological mechanisms that produce the observed patterns of disease and mortality by SES. One key recognition has been that both disadvantage and advantage can be conveyed to individuals and groups at a variety of scales. Chen and Miller (2013) review a substantial amount of evidence for multi-scalar pathways linking socioeconomic status and health, including at the levels of neighborhood (e.g., violence, social capital), family (e.g., parenting practices, family conflict, routines), and individual (e.g., psychological characteristics, health behaviors). Importantly, researchers have recognized that health outcomes can be influenced by the intersection of causative factors from all three levels, and therefore should not be studied in isolation. For example, violence at the neighborhood level is associated with greater risk of cardiovascular disease (Sundquist et al., 2006), and could compound with the negative health outcomes associated with a stressed family environment (Dong et al., 2004) and individual psychological risk factors (S. M. Lynch, 2006). The factors and negative health outcomes involved in this simplistic example—neighborhood violence, strained or overworked parents causing familial stress, and individual factors like depression—have all been associated with low SES (E. Chen & Miller, 2013).

Enumerating the proximate factors linking SES and health is important, because the resulting list emphasizes how the relationship can persist across time and space—even when societies change—and reveals opportunities for intervention. Many of the mechanisms linking SES to disease and mortality are related to resources like knowledge, prestige, power, and social connections, which can be used to enhance health or avoid disease. Examples include: knowing about and asking for beneficial health procedures;

quitting smoking; getting flu shots; eating fruits and vegetables; and exercising regularly (Phelan, Link, Diez-Roux, Kawachi, & Levin, 2004). There are other mechanisms at levels beyond the individual (e.g., the household and neighborhood, as suggested by Chen and Miller (2013). Examples include: low income housing that is located near noise or pollution and to which police, fire, and sanitation services are less attentive; blue-collar jobs are often more dangerous and stressful, yet also carry fewer health benefits than white-collar jobs; high-SES neighborhoods and social networks are less likely to expose an individual to violence and second-hand smoke, and are more likely to share new health-related knowledge or refer an individual to the best physicians or specialists (Phelan et al., 2004). Further, the financial hardships and difficult jobs associated with low-SES families means that parents are less able to implement stable routines and have less patience with children and more stress at home. Low-SES parenting is often more controlling and restrictive, and often involves harsher punishments, which has been related to coronary heart disease and early mortality. Low SES is also associated with negative emotional states and psychological wellbeing, such as hostility, pessimism, depression, and anxiety. In turn, these psychological factors and stressors are associated with coronary heart disease, strokes, and early mortality (E. Chen & Miller, 2013).

Additional studies detail how exposure to different proximate factors can be connected to disease and mortality biologically. Exposure to violence is more common in low-SES neighborhoods and is related to a variety of disease outcomes such as asthma, cardiovascular disease (CVD), and chronic pain. In contrast, family routines (e.g., eating dinner together on a regular basis)—which are more common in high-SES households—coincide with fewer asthma symptoms, adherence to daily medications, lower

inflammation, and lower blood pressure. At the cellular and molecular levels, factors associated with low SES (such as lower levels of family support or high levels of chronic stress) may prime the immune system to react strongly to asthma stimuli (e.g., higher eosinophil counts, which are recruited by cytokine IL-5 to orchestrate a prolonged inflammatory response to allergens in the airway) (E. Chen & Miller, 2013). Other studies have demonstrated that early life psychosocial stress (e.g., mouse pups being separated from their dam) impacts the hypothalamic-pituitary-adrenal (HPA) axis, which negatively impacts health outcomes throughout later life. Compared to controls, mice who had experienced separation stress as pups exhibited greater proinflammatory cytokine secretion and viral replication, but decreased virus-induced glucocorticoid secretion, in response to infection with the influenza virus as adults (Avitsur, Hunzeker, & Sheridan, 2006). Miller and colleagues (2009) demonstrate diminished expression of and signaling by the glucocorticoid receptor (GR) is present in humans who experienced low-SES conditions in early life, which has important consequences for health outcomes like CVD and respiratory conditions. Low SES in early life generates a "defensive phenotype" that limits the negative feedback inhibition of cortisol in the HPA axis (i.e., permits relatively higher levels of stress hormones), enables humans to mount stronger adrenocortical and inflammatory responses to challenges in their environment (e.g., a fight-or-flight response). However, after the acute threat has passed, continuous activation of this system produces an allostatic toll on the individual that contributes to diseases like CVD (G. E. Miller et al., 2009).

Importantly, proximate mechanisms and behavior-specific health outcomes can change across time and space, yet the relationship between SES and health persists. The

continued relationship between SES and disease or mortality is a result of the adaptability of knowledge and resources among high-SES individuals. High-SES individuals are more aware of new threats to their health and can consciously utilize their privileged access to health information, resources, and connections to avoid risks or mitigate damages (Phelan et al., 2004). When the negative health effects of smoking were exposed, high-SES individuals were more likely to cease smoking or not start in the first place, leading to an inverse association between SES and smoking (Pierce, Fiore, Novotny, Hatziandreu, & Davis, 1989). The related disease and mortality outcomes, such as coronary heart disease and lung cancer mortality, also shifted. The relationship between SES and coronary heart disease switched from positive to negative (Beaglehole, 1990), and an inverse association between SES and lung cancer mortality developed (W. P. Logan, 1982). High-SES people likely had better access to health research (from physicians or through social connections), better support from social networks, and access to resources devoted to quitting (e.g., enough money to afford nicotine patches or see a lifestyle counselor). Similar patterns are evident in other areas: high-SES individuals were more able to accommodate lifestyle changes associated with low-fat diets, such as eating less fast food and more fruits and vegetables (Popkin, Siega-Riz, & Haines, 1996); and knowledge about how to prevent HIV infection was more quickly disseminated among individuals in the high-SES group, leading to an eventual association between low SES and HIV/AIDS (Fife & Mode, 1992).

The term “fundamental causes” has been coined for social causes (like SES) that affect multiple disease outcomes via multiple risk factors in a multiscalar fashion, ultimately causing inequalities in mortality (Phelan et al., 2004). Phelan and colleagues

argued that previous investigations emphasized isolated mechanisms or proximate causes (e.g., risk behaviors like smoking) to explain the relationship between SES and health, ignoring the ultimate source of the disparity in mediating factors between status groups. In contrast, a fundamental cause perspective stresses how the relationship between SES and mortality or disease is maintained over time, because access to the knowledge or resources that allow individuals to avoid risks or mitigate damage is determined by SES. In other words, as new knowledge amasses about novel diseases, disease prevention, risk management, and disease intervention, this knowledge is differentially accumulated within and utilized by the higher-SES groups, preserving the SES-health pattern. These groups also benefit from access to a variety of resources (e.g., money, knowledge, power, social connections) which can be purposefully and effectively utilized at that status level. Consequently, even when the proximate factors themselves are eliminated or altered (e.g., diet changes, quitting smoking, or decreasing neighborhood violence), the SES-health disparity continues to exist because the overarching cause involves SES-based access to health-determining resources and knowledge. In their study, Phelan and colleagues (2004) hypothesize that highly preventable diseases will show the greatest degree of separation by SES in terms of mortality patterns, while the diseases for which we have the least knowledge (and are thus the least preventable) will show less differentiation between SES groups. The results show that, particularly for the adult age groups, the health outcomes for the least-preventable diseases (e.g., malignant neoplasm of the gallbladder, multiple sclerosis, malignant neoplasm of the brain) showed no significant differences between SES groups, while 14/18 of the highly preventable diseases (e.g., ischemic heart disease, atherosclerosis, hypertensive renal disease) were

significantly associated with SES group (Phelan et al., 2004). As Chen and Miller (2013) argued, factors and mechanisms like neighborhood violence, social capital, parenting approaches, family conflict, routines, psychological characteristics, and health behaviors are a few major mediators and pathways through which SES affects health, rather than isolated causes to be separately diagnosed and treated.

Other recent research has criticized the "one size fits all" approach to the link between SES and health, including the continued emphasis on adult SES without adequate consideration of the relative importance of childhood SES on later health (Braveman et al., 2005). One example of improvement in this area is a study of the impact of SES throughout life on mortality in middle-aged Finnish men, which found that persistently low SES through childhood and adulthood negatively affected both all-cause and cardiovascular disease mortality risk (Lynch et al., 1994). In a similar vein, Currie and Stabile (2002) analyzed longitudinal data from Canadian children to investigate the variation in responses of high- and low-SES children to health shocks. Their results suggest that low-SES children experience a higher rate of health shocks, rather than merely that they respond slower to a given shock compared to high-SES children, and that the observed negative outcomes (e.g., poor health, poor academic performance) accumulate over time and likely negatively impact future SES. These studies, in conjunction with those presented in the preceding paragraphs, suggest that a study of socioeconomic status and health should include several elements: (1) a consideration of the context of the population under study, and how circumstances may differ for different subpopulations or groups within the larger population; (2) a consideration of how macro-level factors and processes intersect with micro-level factors to create multiple risk

factors at multiple scales affecting multiple disease outcomes and mortality patterns; and (3) a consideration of how early life health events (often related to childhood SES) may influence both adult disease and mortality patterns *and* adult SES.

1.2.2 Bioarchaeological Studies of SES and Health in the Past

Bioarchaeological research into the existence of the health-SES gradient in the past has followed a similar trajectory of increasing recognition of complexity and diversification of methodologies. In an early investigation, Haviland (1967) compared the stature of Mayan individuals interred in tombs (high status) with those buried elsewhere, finding that the high status individuals were, on average, taller than their lower status counterparts. Haviland concluded that upper class people were taller because of a nutritional advantage associated with their social position. Later, additional methodologies and skeletal indicators were explored for their potential contributions to the investigations of the associations between status and health. Schoeninger (1979) compared strontium levels in bone to the amounts of burial goods accompanying individuals, finding that lower status individuals had higher mean levels of strontium, suggesting they ate less meat than higher status individuals that were accompanied by more burial goods. Additional research from a variety of geographical locations confirmed that high status individuals were taller, had better nutrition, or were healthier than their lower status counterparts (Angel, 1984; Hatch & Willey, 1974; J. C. Rose, 1985).

Later investigations problematized the relationship between SES and skeletal indicators of health, noting that the association is not always consistent across contexts and social groups. In a study of individuals from Iron Age Italy, Robb and colleagues (2001) found perplexing patterns of association between burial inclusions and health: there was no statistically significant relationship between grave goods and several commonly-used indicators of health for females, whereas for males varying levels of grave goods were significantly associated with some health indicators but not others. Particularly, males with no grave goods were associated with higher frequencies of Schmorl's nodes, periosteal new bone formation, and trauma, while men buried with weapons had lower frequencies of Schmorl's nodes and periosteal new bone formation, and men buried with pottery (but not weapons) were, health-wise, somewhere in-between (Robb et al., 2001). In their study of Classic Maya settlements in Mexico, Cucina and Tiesler (2003) found that antemortem tooth loss frequencies were higher among the elite, which may be related to behavior and diet differences between the higher and lower class. However, the frequencies of dental caries varied across status and sex groups, being less common in elite males than they were in elite females or low-status individuals of either sex. Similarly, the experiences of racial categories altered the effects of SES on health in African- and Euro-American males of low SES in a study by de la Cova (2011). Although some disease patterns exhibited no difference between race groups, 19th-century African-Americans had significantly higher rates of treponematosis and tuberculosis compared to Euro-Americans of the same status (de la Cova, 2011). Finally, in a study of Middle Iron Age Britain, significant differences in health were not apparent between high- and low-status samples for health indicators like cribra orbitalia, periosteal

new bone formation, and dental caries and abscesses, but indicators like upper limb degenerative joint disease and antemortem tooth loss significantly varied by age and/or sex between status groups (Peck, 2013). As in modern population health studies, the studies of SES and health presented here demonstrate the importance of context in evaluating the connection between SES and health in the bioarchaeological record. Despite similar methodologies, the relationship between skeletal indicators of health and SES appears to have varied with time, location, and samples examined.

1.3 Industrialization in England

1.3.1 Social and Economic Restructuring in Industrializing England

Industrialization, particularly in England, involved changes in the relationships between men, women, and children, as well as a restructuring of the class system and status-based ideologies. Before discussing health and SES, it is important to address the economic and social changes that took place in society, to demonstrate how industrialization itself influenced patterns of marginalization that affect health and disease outcomes among gender, age, and SES groups.

Prior to industrialization, much of England's population lived in rural settings and relied on agriculture, and households (the nuclear and extended family) produced and consumed in local markets (Gazeley & Horrell, 2013; May, 1987). Much of this system was dismantled with the progression of industrialization and urbanization. One change that may have allowed England to rapidly become the global center of industrialization was a pronounced increase in population size. England's population was increasing at a

moderate rate between the 1740s and 1790s, but dramatically increased in the early 19th century, with records suggesting a change from 6.5 million people in 1751 to 17.9 million people in 1851 (G. Clark, 2007; May, 1987; Stevenson, 1993). Although it may be somewhat related to decreased mortality due to reduced infectious disease virulence or improved climate patterns influencing crop yield (and thus the improved nutrition and immunological resistance of the English population), the early 18th-century increase in population is most closely tied to fertility increases prompted by earlier ages at marriage and decreased time periods between births (Stevenson, 1993; Boberg-Fazlic et al., 2011; for a more thorough discussion of mortality patterns during the 2nd epidemiological transition, see DeWitte, 2014c; Gage, 2005). Regardless, the pressure of a growing population led to employment restructuring in terms of how men, women, and children were hired and treated in the workplace, further complicated by the increased mechanization of production. Though evidence is lacking for women and children—particularly the working poor—during the 1750-1850 period (Gage, 2005), a picture of the transitioning society can be generated by examining the changing roles of women and children at the time.

Prior to the industrial period, agriculture largely employed live-in farm workers (young, unmarried individuals of both sexes). Bythell (1993) argues that this is because relatively high mortality, late marriage, and small families meant that labor was unreliable and an 18th-century farmer's best bet was to employ young, unmarried workers. However, a constant, live-in labor force was not necessary for arable farming, so when cereal production became economically more attractive than animal husbandry, so did seasonal, wage-supported male workers (conveniently supplied by the rise in

population that increased the number of available poor, landless, adult male workers willing to work for low wages). This transition was particularly pronounced in in the south and east of England; however, some live-in workers (of both sexes) continued to be hired in regions where animal husbandry predominated, as livestock require daily attention (Bythell, 1993).

A similar shift to male-centered employment was evident in factory work. Although women were traditionally employed in the textile and clothing-making industries, labor typically devoted to women (e.g., spinning) gradually disappeared into the factories between 1750 and 1850. In the tasks that remained outside of the factory (i.e., that a woman could complete at home, such as stitching, seaming, and finishing) the labor was for extremely low pay. Even when they obtained positions within factories, women were primarily given low-wage, unskilled positions, while better-paying jobs as clerks, overseers, and managers were given almost exclusively to men. By 1851, only about a fifth of the adult female population (i.e., females over the age of 15, who are defined as "adults" by the census of 1851) was employed (Checkland, 1964). For domestic service—the area that employed the most women by far—the question becomes whether the increase was led by supply or demand: there was an increase in young, unmarried females who struggled to find other sources of employment, yet there was also an increase in servant-employing middle-class households because of the successful [male] employers in the industrial, commercial, and professional sectors. Although some other sources of employment existed for women in the service sector (e.g., governesses, midwives, shopkeepers, innkeepers, schoolmistresses), it remained that women were

largely absent from the building trades, mining, iron- and machine-works, craft trades, or clerks and secretaries (Bythell, 1993).

Like women, children were gradually removed from the workforce as the process of industrialization wore on. Though child labor was not a new concept, the exploitation of children in factory jobs eventually led to child labor laws in Britain. The number of hours children could work in factories was progressively reduced by government mandates, such as the Factory Act of 1833. Children 9-14 years old could not work more than 8 hours per day in most textile mills, and individuals under the age of 18 could not work more than 12 hours. The Act of 1844 further reduced the hours of children 9-16 years old to 6.5 working hours (May, 1987). In addition, Lord Ashley's Mines Act of 1842 banned women and boys under the age of 10 from mine work entirely (May, 1987; Stearns, 2007). Children were instead funneled into compulsory education, causing them to become a household expense rather than a resource, leading both fertility and mean family size to decrease again by the 1870s (Stearns, 2007; Stevenson, 1993).

Interestingly, the political acts expelling women and children from the mines and factories appear to have been chiefly symbolic. For example, by the 1840s, women had already been pushed out of the mining sector by coal owners and adult male miners. Owners preferred a disciplined workforce, and the removal of women and children undermined the internally-connected workforce of family teams that had previously dominated. Male miners, on the other hand, were threatened by the possibility of unemployment, and sought to ensure the exclusion of low-wage-accepting women and children (Bythell, 1993).

Importantly, these demographic changes and workplace transitions were reinforced by changing ideologies at the cultural level. In the years following 1750, the increase in the marriage rate, decrease in the age of marriage, and increases in fertility and mean family size meant that a larger portion of a married woman's life was relegated to the unpaid job of managing the home, and opportunities for full-time or regular employment diminished. Unmarried women had the greatest chance of gaining employment, but—because of their position as young spinsters—they were perceived to have no need of wages as high as a man's, who, presumably, needed to earn enough to support an entire family (Bythell, 1993). The economic importance of women dropped precipitously—middle-class women were not expected to hold jobs at all and working-class women were only to contribute to the household income as factory workers or domestic servants until marriage in young adulthood (Stearns, 2007). Because of the assumption that young women would exit the workforce when they married, skills were also considered unnecessary for young female workers. This perception extended to ideas that women were mentally and physically incapable of certain types of work, further emphasizing a gendered division of labor (Bythell, 1993).

Even for men, however, the structure of employment and cultural notions of class changed dramatically during the industrializing period. Decentralized supervision gave way to formalized supervision in the form of foremen overseeing employees unfamiliar with being "bossed". Semiskilled work opportunities increased and replaced many skilled positions, leading to a limited sense of achievement and loss of control on the part of the workers (Stearns, 2007; Stevenson, 1993). Finally, the aristocracy and peasantry were replaced by a tense relationship between middle-class employers and the working class

that they employed. This class conflict existed on both material and ideological grounds, with the Industrial Revolution seeing a rise in working class riots, protests, Marxism, Socialism, labor unions, and even cultural protest in the form of art (Stearns, 2007).

1.3.2 Industrialization and Health: Historical and Bioarchaeological Research

Although neo-liberal arguments from the 1980s suggested that industrialization fits a pattern of pronounced economic growth ultimately generating health improvements, historical evidence from England suggests that industrialization exerted health impacts that were primarily negative and were especially intense among marginalized workers (e.g., women, children, migrants). It appears that the health improvements typically associated with industrialization are only realized when industrialization-fueled economic growth *materially* improves population health (e.g., enhancement of urban preventative health infrastructures including improved sewage disposal and clean water access). Thus, though England's industrialization was in full swing by 1850, it was not until the democratically-forced implementation of infrastructure improvements in the 1870s that the nation saw a decrease in negative health consequences and urban mortality (Szreter, 2004).

Historians are limited in the types of data that they can employ to explore industrialization's impact on health. In addition to wage data from particular classes (e.g., G. Clark, 2001, 2005, 2007; C. H. Feinstein, 1998; Hunt, 1986) and birth and death records (e.g., G. Clark & Cummins, 2015), historians have used documented height data to access the health and standard of living of individuals in industrializing England (e.g.,

Nicholas and Steckel, 1991; Nicholas and Oxley, 1993; Cinnirella, 2008). Feinstein (1998) and Clark (2001, 2005) use available evidence on wages to investigate standards of living among manual laborers in the building and agricultural sectors. Feinstein (1998) combines wage patterns with estimated costs of living, finding only a marginal increase in living standards (10-15%) between the 1780s and 1850s. Clark's (2001, 2005) data show a similar pattern, with no substantial improvement in wages or standard of living until after 1820. However, despite their arguments to the contrary, wage data (complete with "corrections" for costs of living and urban health hazards) do not adequately represent the health of the industrializing English population.

Other historians have examined height and weight over the industrial period as proxies for changing health, but these data have their own biases. Nicholas and Steckel (1991) and Nicholas and Oxley (1993) examine the heights of male and female laborers being transported to the penal colony of New South Wales between 1817 and 1840. Men in rural settings appeared to fare slightly better than those in urban settings, particularly those areas in and around London (Nicholas & Steckel, 1991). The heights of urban women, in contrast, exceeded that of their rural counterparts, indicating that rural women were experiencing a more rapid nutritional decline relative to men. Importantly, Nicholas and Oxley (1993) connect the health patterns in their data to the social and economic changes taking place during the industrial revolution (detailed in above sections): they suggest the decline in health of rural women may be related to the declining availability of agricultural employment for women, and thus the declining importance of the woman as a wage-earner in the rural household. Limited employment in agriculture, manufacturing, and formal apprenticeship led to increased gender inequality within the

home, impacting food allocation. Rural areas in particular were limited in their employment opportunities, leading to increased gender inequality within the home, and forcing women and children to accept a smaller portion of the household's resources in order to ensure their continued survival (Nicholas & Oxley, 1993). Similarly, Cinnirella (2008) examined the heights of British Army recruits, finding that stature declined over the industrializing period, with pronounced declines in the last half of the 18th century and in the mid-19th century. Declines were especially pronounced among rural-to-urban migrants, who may have been forced to migrate because of declining agricultural employment opportunities for women and children (Cinnirella, 2008). Scholars examining documented weight changes in British prisons argue that observed weight fluctuations are suggestive of the resource change that occurs when moving from the household diet to the prison diet: adult males lost weight because the prison diet was less than what they received as the primary bread-winners in the household, boys and old men maintained the same weight, and adult women gained weight because the meager prison diets were more substantial than the meals they received in the household (Meredith & Oxley, 2015). Importantly—and like patterns found in other studies (Cinnirella, 2008; Hunt, 1986; Nicholas & Oxley, 1993; Nicholas & Steckel, 1991)—the extent of nutritional disparities between subpopulations was often region-dependent; for example, Meredith and Oxley (2015) demonstrated better health outcomes (i.e., optimal heights and weights) for women and children (*and* men) in regional economies in which women and children earned more.

Although informative, the historical data presented above are biased and require various assumptions and corrections to be considered accurate. The studies of wages are

focused within one particular class—usually agricultural laborers—offering little information on the experiences of other English men and women, such as the middle-class. Furthermore, as Gazeley and Horrell (2013) point out, data on wages or earnings do not predict nutritional inadequacy or health as well as other, even less-widely available historical data, such as household size. Finally, although height and weight data have been used extensively as proxies for health across multiple disciplines, the available historical data on body size is often biased towards the working class. In fact, *all* of the height and weight studies presented here (Cinnirella, 2008; Meredith & Oxley, 2015; Nicholas & Oxley, 1993; Nicholas & Steckel, 1991) use data from prison logs. The historical evidence about convicts is informative, but limited in that it may not accurately represent the working class or the British population as a whole. Finally, although data on industrialization-era mortality is available from other historical sources (e.g., the London Bills of Mortality begin recording causes of death in the 17th century), the descriptions in such sources are often inaccurate and do not provide an adequate description of the health of the living population from which the deceased come (DeWitte, 2014c).

Because historical data lack detail, generalizability, and a solid portion of the population (i.e., women are often relegated to positions of "shopkeeper's wife", while children are reduced to mere birth and death records), other sources of evidence can be used to get a better picture of overall population health, as well as the exposures and hazards experienced by different subpopulations. Bioarchaeological studies offer an opportunity to explore some of these lost voices in greater detail, broadening our understanding of past lives and the origins of modern health patterns.

Although not covered exhaustively here, bioarchaeological studies of SES and health in industrializing England have investigated the health and mortality patterns of individuals typically omitted from the historical literature: women, children, and the poor. Multiple studies have investigated the health of non-adults (children) in industrial-era England (e.g., DeWitte, Hughes-Morey, Bekvalac, & Karsten, 2016; M. E. Lewis, 2002; M. E. Lewis & Gowland, 2007; Mays, Brickley, & Ives, 2008; Mays, Ives, & Brickley, 2009; Newman & Gowland, 2017; Pinhasi, Shaw, White, & Ogden, 2006), generally suggesting that industrialization—not just urbanization—was detrimental to the health of non-adults. Lewis (2002) demonstrated children in industrial environments were smaller and had higher frequencies of stress and metabolic disease indicators than their rural counterparts. These results would be corroborated by a later study, which found that neonatal deaths in urban environments were primarily driven by exogenous factors like disease, nutrition, and accidents, compared to rural environments where genetics and maternal influence played a greater role (M. E. Lewis & Gowland, 2007). In a study of non-adults of differing SES, Newman and Gowland (2017) found that a middle-class site exhibited better long bone growth values and vertebral neural canal sizes (i.e., two skeletal measures considered indicative of developmental stability or stress), while the high-SES and low-SES sites exhibited the lowest growth values. The authors argue that status-driven cultural norms and child-care practices (i.e., breastfeeding substitutes, swaddling, sun exposure limitation) left high-SES non-adults susceptible to compromised immunocompetence, as well as iron and vitamin D deficiencies. In the same study, indicators of early life stress among low-SES non-adults are the result of poor maternal and childhood environmental conditions, suggesting a connection with studies finding

negative health outcomes among adults in London cemeteries (i.e., the developmental origins of health and disease hypothesis) (Newman & Gowland, 2017). Other bioarchaeological studies underscore the importance of childhood SES conditions for later health and mortality risk, demonstrating that children experienced the bulk of the negative consequences of industrialization (e.g., enamel hypoplasia and metabolic diseases) and selective mortality primarily targeted low-SES children while their high-SES counterparts experienced a buffering effect (DeWitte, 2014c; DeWitte et al., 2016).

Bioarchaeological studies have also examined the patterns of skeletal indicators of stress and mortality among adults in differing SES groups. A study of two industrial-era cemeteries from London found no significant differences in the rates of caries or tooth loss in individuals separated by sex or social status (Mant & Roberts, 2015). Using the same London cemeteries, Hughes-Morey (2016) found that low-status females had lower mean age at death than high status males, high status females had a greater mean tibia length than low-status females, and low-status males were less likely to have short tibiae than low-status females or high-status males. According to Hughes-Morey, the results suggest that low-status females experienced the greatest amount of stress throughout life, making them relatively more frail and less likely to survive to adulthood when compared to any other subgroup. In contrast, high-status males who experienced childhood insults that affected achieved stature were still able to survive to adulthood because they were less likely to be exposed to severe and repeated disease and had preferential access to resources (Hughes-Morey, 2016). Hughes-Morey's (2016) results are similar to those of DeWitte and colleagues (2016), which suggested that selective mortality among non-adults influenced later population frailty among surviving adults. In that study, the

removal of high frailty children of low SES led to relatively low-frailty adults in the low-SES group, while buffered high-SES children would survive childhood and the emergent high-SES adults would be of more heterogeneous frailty than the low-SES adults (DeWitte et al., 2016).

The bioarchaeological studies of SES and health in industrializing England highlight the importance of context and methodology in understanding patterns of frailty and mortality in the past. As the Mant and Roberts (2015) and Hughes-Morey (2016) examples show, studies using the same collections but different skeletal indicators of health can generate wholly different results that elucidate different aspects of health in the past. Therefore, bioarchaeology has the opportunity to provide a multi-faceted glimpse into the lives of individuals in the past. Incorporating a variety of data sources (e.g., alternative skeletal indicators of stress) and interpreting the results in light of current theoretical approaches (e.g., the explicit use of the developmental origins of health and disease hypothesis by (Newman & Gowland, 2017) allows bioarchaeology to examine entirely new and previously inaccessible aspects of past patterns of disease and mortality, rather than merely commenting on the biases of the historical data.

1.4 Intersectional Approaches

1.4.1 Origins

Intersectionality theory (also described as a concept, framework, or approach; see Davis, 2008) argues that outcomes tied to intersecting marginalizations (such as race, sex, and class) at a local level are intertwined with structural inequalities in society (such as

racism, sexism, and classism) (Bowleg, 2012). Though the theoretical form used in this study stems primarily from recent quantitative health research (e.g., Stirratt et al., 2008; Veenstra, 2011; Warner and Brown, 2011; Hinze et al., 2012; Sen and Iyer, 2012; Seng et al., 2012; Longman Marcellin et al., 2013), the term originated in Black feminist scholarship decades earlier and emanates from a much older social movement.

Kimberlé Crenshaw (1989, 1991), a Black feminist legal scholar, is credited with coining the term "intersectionality" to describe the perspective that multiple marginalized identities and processes meaningfully intersect to influence individuals' lives. In her 1989 publication, Crenshaw specifically uses the Black woman as a starting point, particularly to highlight how dominant conceptions of discrimination lead us to consider only the experiences of the most privileged discriminated identities, effectively erasing the multiply marginalized members of society (e.g., sexual discrimination legal cases focus on the privileged women in race and class categories; legal cases involving race discrimination focus on the sex- and class-privileged Blacks). In the same vein, because feminist scholarship and antiracist policy discussions focus on a limited and biased set of experiences, multiply marginalized individuals (e.g., Black women) are erased and excluded from these discourses (Crenshaw, 1989).

Although many scholars trace the term to Crenshaw, the underlying perspective that socially salient categories like race and gender meaningfully intersect has been recognized for decades (Collins, 2015). One prominent example includes hooks's (1981) book "Ain't I a Woman: Black Women and Feminism", a title which alludes to the 1851 speech by freed slave Sojourner Truth in which Truth challenged the idea that her gender and race were mutually exclusive. In her book, hooks addresses the perpetual silence of

black female scholars and black women more generally, arguing that black women were conditioned to silently accept their oppression by racist feminists (white women) and sexist black activists (black men). She discusses how black male sexism was largely ignored by the budding feminist movement that attacked [white] patriarchal power and argues that white feminists subtly exposed their racist tendencies by only acknowledging the experiences and oppressions of white women (hooks, 1981). Therefore, in historical perspective, intersectionality stems from an implicitly white, middle-class feminist movement that denied the access of individuals multiply-marginalized by the patriarchy.

1.4.2 Intersectionality in Studies of Modern Health

The historical background provided by Black feminists like hooks (and Truth) has encouraged the growth of intersectionality as a theoretical perspective in recent decades. Intersectionality approaches acknowledge that multiple marginalizations (e.g., race and sex for Black women) are mutually constituted and reciprocally constructed, and cannot be fully investigated or understood by methodologies that treat them as distinct categories of analysis (Collins, 2015). Researchers incorporating intersectionality, therefore, must be aware that intersections among advantaged and disadvantaged axes—which vary within the larger structural context—can be more central to the nature of experiences or health outcomes than any of the axes of inequality considered separately (Veenstra, 2011). In studies of health, several themes can be identified: (1) social identities are multiple and mutually constituted, rather than independent subjects of inquiry; (2) historically oppressed groups and multiply-marginalized peoples are an important starting

point for analysis, but perhaps are not sufficient alone; and (3) micro-level characteristics and identities interact with macro-level structural factors and processes, thereby producing, magnifying, or mitigating the observed outcomes (Bauer, 2014; Bowleg, 2012).

Studies of health that have touched upon each theme abound. The central tenet of multiple, mutually constitutive identities presents a challenge to quantitative researchers (i.e., the interaction among more than two variables is difficult to interpret). However, scholars have approached the issue using a variety of analytical methods and with a great degree of success (e.g., Stirratt et al., 2008; Hinze et al., 2012; Seng et al., 2012). The second theme—multiply-marginalized identities as a starting, but not concluding, point—has received an increased amount of attention in the last decade, following Hancock's (2007) argument that *all* intersections should be of interest to researchers. In the last several years, scholars have acknowledged that in systems oppressing multiply-disadvantaged individuals, there are also identities and processes that permit multiply-advantaged individuals and individuals that experience combinations of advantage and disadvantage. Sen and Iyer's (2012) study of health access in Indian households examines these "middle groups" (i.e., non-poor women and poor men), finding that "middle groups" may exhibit similar health outcomes, but the advantages that they leverage and the mechanisms involved vary (e.g., poor men leverage their gender and non-poor women leverage their class). Interestingly, the authors find that the two types of advantages differ in their efficacy: gender appears to be the more powerful lever for gaining access to, continuing, and receiving quality healthcare (Sen & Iyer, 2012). Finally, a variety of health studies have approached the third theme, which investigates

the interplay between micro-level factors (such as sex and class) and macro-level factors (such as sexism and classism). Hankivsky and colleagues (2010) provide several examples of Canadian researchers who have successfully investigated this aspect of intersectionality in their work, including studies of how aging gay and lesbian adults experience marginalization and discrimination in multiple ways (Brotman, Ryan, & Cormier, 2003), and how macro-level structures like colonization and racism impact Aboriginal womens' risks of violence and HIV (Varcoe & Dick, 2008).

1.4.3 Intersectional as Biocultural: Using Intersectionality in Bioarchaeology

Intersectionality approaches explore the intersections of various categories of social and biological difference (e.g., age, sex, class), yet also acknowledge the interplay and negotiation between these individual, micro-level characteristics and larger, macro-level processes and systems of power and oppression (e.g., ageism, sexism, classism) (Springer, Hankivsky, & Bates, 2012). Because of this emphasis on both personal and societal intersections, it could be argued that intersectionality's use within bioarchaeology is inherently biocultural. Biocultural studies, which highlight the interconnectedness of macro- and micro-level contexts, require bioarchaeologists to look beyond the physical, biological sources of data in front of them to consider a skeletal collection's embeddedness in cultural and historical contexts (Alan H. Goodman & Leatherman, 1998). Biocultural approaches recognize a "dynamic interaction between humans and their larger social, cultural, and physical environments" (M. K. Zuckerman, Turner, & Armelagos, 2012, p. 39), meaning that biological processes and outcomes are

inseparable from their cultural context (Dufour, 2006; Alan H. Goodman & Leatherman, 1998; M. K. Zuckerman & Armelagos, 2011). Consequently, this dissertation project considers the biocultural approaches forged from political-economic anthropology and biological anthropology to be complementary to the intersectionality approaches from social theory and public health. Though stemming from different disciplinary backgrounds, the two theoretical orientations advocate for many of the same investigations and interpretations within the context of bioarchaeology (e.g., the biological/health consequences of sex and class differences in resource access within the larger sociocultural setting).

Considering the compatibility of the two perspectives, it is somewhat surprising that biocultural archaeologists have not yet embraced intersectionality as a theoretical framework. In an article published in the *Annual Review of Anthropology*, Tom Boellstorff (2007) discusses "Queer studies in the house of anthropology", including a section on intersectionality. However, Boellstorff addresses intersectionality primarily as an assumed theoretical approach rather than an arena for further study of the mutually-constituted identities available to individuals beyond "lesbian" or "gay". Boellstorff intentionally excludes archaeology from his article, and instead references a forthcoming piece by Barbara Voss. However, neither Voss's (2008) article nor an earlier article that specifically addresses "black feminist-inspired" approaches to archaeology (Franklin, 2001) reference this now prominent social theory and the potential it holds for archaeology and biological anthropology.

Of the authors who integrate intersectionality into their archaeological or bioarchaeological study, few explicitly discuss the tenets of the perspective or generate

methods that could be replicated by future scholars interested in applying such an approach. For example, Kjellström (2014) considers violence in the archaeological record as a form of discrimination, underscoring patterns of violence and types of trauma between adult females—who exhibited few signs of violence-related trauma—and adult males—who received traumatic lesions on the cranium that were structurally-reified "marks of honor" (Kjellström, 2014, p. 245). However, Kjellström's work does not fully access the effects of intersecting identity categories beyond sex (e.g., cribra orbitalia and linear enamel hypoplasia are simultaneously used as indicators of status and childhood health). A study by Fahlander (2012) focuses primarily on delineating the methodological advancements necessary to address elements of intersectionality beyond age, sex, and status identities. For example, Fahlander (2012) discusses how to access features like gender through facial construction, body shape (e.g., fat distribution), and other bodily characteristics that "hint" at dispositions and preferences (e.g., hormonal effects on bones—such as relative index and ring finger lengths—which he argues hint at personality traits or dispositions) (Fahlander, 2012). Like Fahlander, an article by Conkey (2005) focuses less on delineating the goals of the perspective or the ways in which intersectionality theory might be applied to archaeological materials. Rather, Conkey (2005) argues that intersectional and indigenous archaeologies offer an opportunity for reflexivity in archaeology, allowing us to "recover the multiplicity of voices that are normally suppressed in archaeological discourse" (Conkey, 2005 quoting Joyce, 1998).

The most comprehensive study to concretely integrate bioarchaeological data with an intersectional perspective—as well as a multi-level investigation typical of biocultural approaches (Dressler, 1995)—comes from Torres-Rouff and Knudson (2017), who use a

multiscalar approach ("integrative multiscalar model") to explore identities held at a variety of levels, including the individual, community, and region. Using data from their research in northern Chile, Torres-Rouff and Knudson examine immutable and mutable aspects of identity: immutable aspects are accessed via cranial metric and nonmetric traits (to explore the biological relatedness of populations—biological distance—across space and time), as well as strontium and oxygen isotopic analyses (to examine geographic origins and mobility at the individual level); mutable aspects are accessed via data on sex (to access gender), physiological age (to access social age), cranial modification (to access community identities), and mortuary context (to access individual and group social identities) (Torres-Rouff & Knudson, 2017). Because Torres-Rouff and Knudson leverage multiple lines of evidence to examine the individual- and population-level changes over time, they examine how social positions are negotiated within larger structures of power and oppression.

The article by Torres-Rouff and Knudson (2017) provides one example of how an intersectionality approach can be applied in bioarchaeological studies to answer larger anthropological questions. An intersectional bioarchaeology can demonstrate how micro-level identities intersect with macro-level power structures and processes in a variety of contexts, adding temporal depth to examinations of how power relations are produced and maintained. Bioarchaeology is uniquely situated to contribute an entirely nuanced perspective on the generation and maintenance of social identities within systems of oppression and privilege across cultures. Further, intersectionality can drastically change how archaeologists and biological anthropologists approach patterns of health in the past, given that the perspective's focus requires an investigation of more than one

characteristic of interest producing the observed patterns of disease and mortality (e.g., *only* sex and health outcomes or *only* SES and health outcomes). In this way, bioarchaeologists are equipped to produce literature that extends beyond the modern, industrialized contexts researched by our colleagues in other social sciences, yet also complements that scholarship and encourages interdisciplinary (and inter-anthropological-subfield) discussion of the origins of intersections and their associated health outcomes across space and time. Lastly, to echo Conkey (2005), an intersectional bioarchaeology offers an opportunity to recover multiply-marginalized voices that have been largely omitted from the history books. As Fahlander (2012) mentions, an intersectional archaeology encourages scholars to search for new ways to examine identity negotiation in the past via the archaeological record (e.g., Torres-Rouff and Knudson's [2017] emphasis on sources of data on the "mutable" and "immutable" aspects of individual and group identity).

This dissertation project provides an additional example of how a biocultural bioarchaeology can be used to explore the intersections of social identities that are accessible via the archaeological record. Using age and sex estimations gleaned from the skeletal material and archaeological evidence of SES, this study quantitatively examines how these individual, micro-level characteristics interact to produce patterns of frailty and mortality in different subpopulations of society. Importantly, this study also uses historical information to contextualize the paleodemographic analyses and consider the macro-level processes taking hold during the industrialization of England. Particularly, the forms of sexism and classism recognizable today emerged during this period, allowing this study to provide temporal depth to the structures of oppression being

studied by intersectionality theorists working with living populations. By considering intersections at a formative time in England's history, this study examines how sex, age, and SES may have acted independently or in conjunction to influence frailty and survivorship, and it investigates how SES, sex, and age may have impacted individuals and subpopulations constrained by increasingly restrictive social and political structures, which produced and reproduced the marginalizations and health outcomes seen in England today.

1.4.4 Limitations to Intersectionality in Bioarchaeology

The bioarchaeological application of intersectionality theory is limited by the evidence available in the archaeological and historical records. Like biocultural studies, studies of the past that seek to employ an intersectional framework can be limited by the social and cultural information available in the historical literature. Since biocultural and intersectional studies explicitly address the influences of macro-level structures and processes, fully applying those theoretical perspectives can be challenging when historical data are unavailable or incredibly biased (see Dufour, 2006) and (M. K. Zuckerman et al., 2012) for a discussion of limitations in biocultural studies). However, as the Torres-Rouff and Knudson (2017) example shows, it is possible to use skeletal and archaeological evidence to examine community- and population-level influences on individual health outcomes in some cases.

Currently, intersectionality research in bioarchaeology is also limited by the characteristics available for study. For example, most studies are limited to age, sex,

status, and ancestry groups, as these are the characteristics most bioarchaeologists would agree are directly accessible from the skeletal data or archaeological context. Arguments can be made for using ancestry to comment on socially-based racial categories or using biological sex to assess gendered disparities in health outcomes (see, for example, Torres-Rouff and Knudson's [2017] use of sex to access gender), but many archaeologists are hesitant to make such a leap, as it could expose archaeological data to modern-day categorizations and biases. However, advances in the field provide some opportunity for optimism. Migrant status, for example, may be exposed by strontium and oxygen isotope values (e.g., Evans et al., 2006), while improvements in age estimation methods provide an opportunity to examine age-related intersections and health outcomes among the elderly (e.g., Jepser L. Boldsen, Milner, Konigsberg, & Wood, 2002).

1.5 The Developmental Origins of Health and Disease (DOHaD) Hypothesis

1.5.1 Origins

The foundations of the Developmental Origins of Health and Disease (DOHaD) hypothesis lie in studies of the patterns of mortality produced by coronary heart disease (CHD) in Britain. The original concept for CHD was that unhealthy lifestyles associated with rising prosperity combined with genetic predisposition to produce observed patterns of mortality from the disease; however, geographical studies of Britain proved to be at odds with this model, as rates of heart disease were lowest in affluent areas and dramatically higher in poorer areas (Barker, 2007). In a study of CHD mortality in England and Wales, Barker and Osmond (1986) found that the regions with high rates of

CHD mortality between 1968 and 1978 coincided with high neonatal mortality rates in the past. Further, lifestyle factors such as cigarette smoking, psychosocial stress, and dietary fat consumption showed entirely different geographic patterning. Therefore, Barker and Osmond reasoned that the poor social conditions causing infant deaths in the past were not also giving rise to risky behaviors. Instead, the "Barker hypothesis" posited that poor living conditions influenced the metabolic, hormonal, and nutritional environment within which the fetus developed, causing changes in the phenotype of the infant (e.g., low birthweight) that enabled survival in the short-term but put the individual at greater risk of disease (e.g., CHD) and mortality in later life (Barker & Osmond, 1986). Barker's argument for human developmental plasticity (i.e., a single genotype can give rise to a range of different physiological states due to different developmental environments) impacting mortality patterns was later replicated in other studies (Barker et al., 1993; Barker, Osmond, Winter, Margetts, & Simmonds, 1989).

Barker's early work emphasized the "thrifty phenotype" and "developmental plasticity" hypotheses, which led to the finding that adverse environmental conditions during different critical phases of fetal development corresponded to different consequences in adult life (e.g., the Dutch Hunger Winter data demonstrated that late-gestation adversity was connected to insulin resistance and impaired glucose tolerance, while periconceptional adversity was related to high serum cholesterol and coronary heart disease) (Wadhwa, Buss, Entringer, & Swanson, 2009). Developmental plasticity would later morph into the "predictive adaptive response" (PAR) model, which posits that critical windows exist (periconception to postnatal) during which the mechanisms of developmental plasticity may operate, fine-tuning the developing organism's physiology

to the predicted adult environment. The fetal environment (conveyed by the mother) acts as a cue to the fetus and programs infant body composition. In brief, PARs use maternal cues to predict the postnatal environment and prepare the fetal physiology to survive in that environment. However, should the predicted environment and the actual environment mismatch, the risk of certain diseases (e.g., CHD and atherosclerosis) increases as the individual matures. For example, if the maternal environment suggests resource scarcity, physiological alterations like insulin resistance may be considered adaptive; however, if the actual postnatal environment permits adequate or over-nutrition, the risks associated with glucose intolerance (e.g., Type 2 diabetes) are increased. Thus, adaptive responses occurring during development can ultimately produce negative consequences in adult life (Gluckman, Cutfield, Hofman, & Hanson, 2005; Gluckman, Hanson, Cooper, & Thornburg, 2008; Hanson & Gluckman, 2008).

Expansion of the DOHaD model has also branched beyond nutrition into maternal stress, which may influence later adult health even if the infant does not present with adverse phenotypes like low birthweight (Entringer, Buss, & Wadhwa, 2010; Wadhwa et al., 2009). The study of epigenetics (particularly in animal models) has been especially fruitful in understanding the variety of mechanisms and consequences of maternal diet and stress on offspring (e.g., obesity, neural and endocrine responses to stress, and common adult disorders like depression, anxiety, diabetes, and heart disease). A variety of epigenetic changes (e.g., changes in the methylation of cytidine-guanosine nucleotides in the promoter regions of specific genes, changes to chromatin structure through histone acetylation or methylation, and post-transcriptional modification via microRNA) have been identified, which lengthens the developmental window for predictive adaptive

responses beyond birth, likely from conception to early childhood (Gluckman et al., 2008).

Recent research foci include the impact of early-life stressors on the response of the hypothalamic-pituitary-adrenal axis to later life stress and infection (e.g., Avitsur et al., 2006; G. E. Miller et al., 2009; Prentice & Moore, 2005), which likely stems from modifications to the epigenome (Entringer et al., 2010). In addition to the mechanisms underlying the patterning identified in DOHaD studies, many researchers emphasize the causes of the fetal or childhood stress. Socioeconomic status (SES) has been a key topic of interest, particularly in that it is rarely restricted to a singular event or critical period, but often also exerts an impact on life trajectories (socially and biologically), causing disadvantages to accumulate across the life course (Gagnon & Bohnert, 2012; Hertzman, 1999; Willson, Shuey, & Elder, 2007). A variety of health studies of have demonstrated associations between childhood SES and overall cardiovascular disease mortality in adults, as well as other causes of adult mortality, such as stroke, coronary heart disease, and accidental or violent death (Galobardes, Lynch, & Davey Smith, 2004; Galobardes, Smith, & Lynch, 2006; Kittleson et al., 2006). As suggested above, associations between childhood SES and adult mortality may be related to early-life modifications to mRNA (e.g., GR and TLR4 regions), which then promote exaggerated inflammatory reactions in response to infection, and, eventually, produce diseases associated with chronic inflammation (e.g., cardiovascular disease) (G. Miller & Chen, 2007; G. E. Miller et al., 2008). An association has even been found between early life SES and susceptibility to infectious disease in adulthood; results from Cohen and colleagues (2004) indicated that risk of contracting a cold (rhinovirus) decreased with an increase in the number of years

that parents owned the home that the child lived in, and a decrease in the number of years of home ownership was associated with greater infection and greater illness expression.

1.5.2 DOHaD in Bioarchaeology

Within the last decade, scholars have applied the DOHaD perspective in bioarchaeological studies, using skeletal evidence to tackle new questions and previously unexplored research directions, such as the link between early life stress and later life morbidity and mortality (Gowland, 2015). Skeletal indicators of developmental disruption such as limb length, enamel hypoplasia, and vertebral size have been found to correlate with younger ages at death in adulthood (e.g., Amoroso, Garcia, & Cardoso, 2014; Armelagos, Goodman, Harper, & Blakey, 2009; Jesper L. Boldsen, 2007; Newman & Gowland, 2015, 2017; Temple, 2014; R. Watts, 2011, 2013a, 2015). In the case of vertebral dimensions, the transverse and anteroposterior dimensions of the lumbar portion of the neural canal reach their final size at different stages of childhood and adolescence (and the vertebral bodies even later), suggesting that these features can be used to examine the effects of stress events occurring at different phases of the developmental period (Newman and Gowland, 2015; Watts, 2015). Similarly, the relationship between enamel hypoplasia and earlier adult mortality suggests that the developmental time frame of the analyzed tooth or teeth could provide information about stress events experienced during that portion of childhood (Armelagos et al., 2009; Jesper L. Boldsen, 2007; A. H. Goodman & Armelagos, 1985a; Temple, 2014). In conjunction, these skeletal indicators

could provide information about the entire subadult period, and could be analyzed to examine mortality risk, as well as longevity and survivorship.

In addition to the examples provided by modern population health studies, bioarchaeological studies have also demonstrated ways to integrate a focus on SES into a DOHaD framework. First and foremost, successful bioarchaeological studies employing a DOHaD perspective to evaluate the effects of early life SES must select skeletal indicators of stress produced during childhood yet maintained through development (e.g., stature or long bone length, enamel hypoplasia on select permanent or deciduous teeth, vertebral neural canal dimensions). Second, studies must statistically evaluate the association between developmental disturbance and later mortality. Examples of such studies are rare but are becoming more numerous, confirming that this is an active area of research in bioarchaeology. In a study of linear enamel hypoplasia presence in a skeletal collection from Lisbon, Portugal, Amoroso and colleagues (2014) found that SES exerted an early yet cumulative effect on health, decreasing adult longevity. Another study by Hughes-Morey (2016) indicated differences in tibia length and mortality risk by SES and sex in a collection from industrial-era London. Hughes-Morey (2016) argues that SES and sex groups had different early life experiences, which impacted observed patterns of adult mortality risk (e.g., the short tibiae of low-SES females compared to low-SES males may be explained by males experiencing less stress during growth and development than females). Finally, Newman and Gowland (2017) use evidence from multiple London cemeteries to investigate whether children of middle- or high-SES groups were buffered by privilege from the negative aspects of urban life. Interestingly, although low SES was associated with several indicators of perinatal and childhood stress, high SES was also

associated with low long bone growth values (longitudinal and appositional) and small vertebral neural canal size values. In a brilliant demonstration of the importance of cultural context, Newman and Gowland (2017) explain that the poor health outcomes associated with high SES children may be the result of status-driven childcare practices (e.g., breastfeeding substitutes, swaddling, sun exposure limitation).

As with intersectionality research, bioarchaeology has a unique perspective to offer studies of developmental health. Particularly, bioarchaeological studies using a biocultural approach can answer the call of population health scholars like Arline Geronimus (2013), who argue that findings from DOHaD research should be considered only within a much larger social and cultural framework. Though animal models are useful, but their benefit is also their detriment: the short lives of rodents prevent us from seeing the decades-long exposures that some human populations contend with. Geronimus (2013) correctly asserts that, to better incorporate the cultural context and stressors throughout the life course, a variety of disciplines and researchers must collaborate. Studies like the ones presented above add temporal depth to the study of early-life health events and cumulative disadvantage throughout the life course. Bioarchaeological studies can answer questions about the range of contributing factors (and outcomes) exhibited across cultures and time.

1.5.3 Limitations to DOHaD in Bioarchaeology

Bioarchaeological engagement with the DOHaD hypothesis is primarily limited by the nature of the available evidence (i.e., osteological data). First, skeletal data limits

the indicators and markers of early life insults that are available to bioarchaeologists. As noted by Klaus (2014), exciting new areas of research in studies of modern populations, like epigenetics, are largely closed to bioarchaeologists for the present. Additionally, one of the only bioarchaeological indicators of prenatal disturbances—enamel hypoplasia on deciduous teeth—is only available for study if the individual dies as a child, meaning that we cannot currently look at the effects of prenatal stress events on adult mortality patterns (Armelagos et al., 2009).

Second, skeletal data also limits the observable health outcomes of interest to DOHaD researchers. For example, a bioarchaeological study could not replicate Barker and Osmond's (1986) original study, because dry bone offers no access to data on birthweight or coronary heart disease mortality. Finally, bioarchaeological data lack the specificity of most DOHaD data sets. Because indicators like enamel hypoplasia, vertebral neural canal size, and long bone length are nonspecific indicators of developmental disturbance, bioarchaeological studies are unable to examine the effects of specific physiological disruptions like malnutrition or disease upon adult health outcomes. However, despite these limitations, bioarchaeologists can access an abundance of data on nonspecific developmental disturbances and their influences on all-cause mortality patterns, which is interesting and worth exploring.

1.6 Skeletal Indicators of Stress

1.6.1 Fluctuating Asymmetry

1.6.1.1 Background and Definitions

Morphological asymmetry in bilateral features has been explored extensively by biologists interested in how and why asymmetries develop in a variety of organisms (Klingenberg, 2015; A. P. Møller, 1997). The interest in asymmetry stems from the assumption that corresponding structures on the left and right side of a bilaterally symmetrical organism are under the control of the same genome and should experience the same environmental conditions, and thus the right and left sides *should* both produce identical copies of the "target" phenotype specified by the genome. However, because of subtle variations in processes occurring at the molecular level, subtle differences are generated across developing tissues and organs (Klingenberg, 2015). Thus, researchers investigating asymmetry are often interested in the sources or effects of developmental noise and developmental stability. "Developmental noise" refers to the effects of environmental perturbations (stress events) upon the ideal phenotypic development of an individual, while "developmental stability" refers to the organism's the ability to correct for deviations from the developmental ideal (A R Palmer, 1994; A R Palmer & Strobeck, 1992). The premise of studies examining the relationship between stress and developmental noise/stability is that stress in particular individuals or groups leads to a diminished capacity to produce the target phenotype (Klingenberg, 2015). Thus, although asymmetry—the deviation from symmetry in an organism's bilateral features—may not

be of great interest on its own, it serves as a proxy measurement of the stable development of the organism's phenotype under given environmental conditions.

Human and non-human animal studies of asymmetry have suggested—in some cases, quite strongly—that a negative relationship exists between developmental instability and components of Darwinian fitness: symmetrical individuals grow faster, exhibit higher fecundity, and survive longer than asymmetrical individuals. Importantly, these studies indicate that the components of fitness related to developmental instability are not limited to those influenced exclusively by either sexual or natural selection, and the effect can be direct or indirect. For example, asymmetrical individuals may be at greater risk of predation or parasitic infection than more symmetrical individuals, lowering the former's competitiveness and indirectly reducing measures of fitness (see Møller, 1997 for a review).

In practice, asymmetry can be broadly divided into three types: directional asymmetry, antisymmetry, and fluctuating asymmetry. The three are most easily characterized by the right-minus-left (R – L) frequency distributions that they generate at the sample or population levels (**Figure 1.1**). Directional asymmetry exhibits a normal frequency distribution with a mean above or below zero, signifying that the bilateral feature of interest consistently differs in size on one side of the body among sampled individuals. For example, the tusk of a male narwhal (which is actually a grossly enlarged canine tooth) is *always* found on the left side of the individual. In contrast, features that exhibit a large degree of antisymmetry will produce frequency distributions with a mean of zero but that are non-normal in some way (e.g., platykurtic or bimodal). For example,

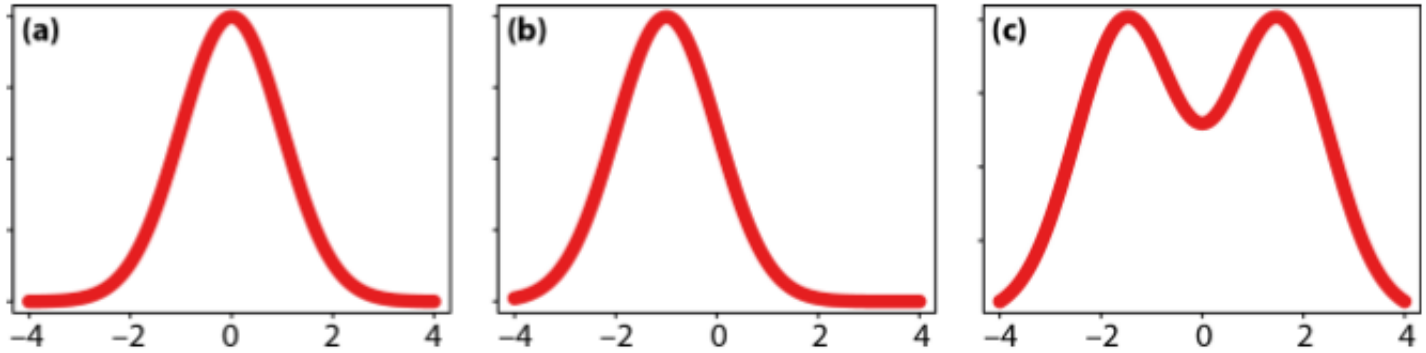

Figure 1.1 The frequency distributions produced by (a) fluctuating asymmetry, (b) directional asymmetry, and (c) antisymmetry (from Klingenberg, 2015).

the oversized major claw of the male fiddler crab is equally likely to be found on the right or left side of the body of a given individual. Lastly, fluctuating asymmetry (FA) is traditionally defined by a R – L frequency distribution that is normally distributed about a mean of zero (Palmer and Strobeck, 1992; 2003; cf. Klingenberg, 2015).

Since studies of asymmetry are typically interested in the influence of developmental noise on between-sides variation, FA is of greater interest than directional asymmetry or antisymmetry (Klingenberg & McIntyre, 1998). True developmental noise involves the cumulative effect of subtle, random between-sides variation produced by environmental perturbations (stress) and thus should produce normal distributions (although there may be empirical evidence to contradict this assertion, for example Babbitt et al., 2006). If one assumes that FA produces normal distributions with a mean of zero, a trait that exhibits directional asymmetry (i.e., bias towards one side of the body among individuals in the sample, usually indicating a strong genetic basis for the trait) or non-normality (i.e., bimodality indicating antisymmetry, as well as skew or leptokurtosis) is not considered suitable for analyses of developmental noise (A R Palmer, 1994).

1.6.1.2 Collecting Data and Preparing for Statistical Analysis

In addition to excluding traits that exhibit directional asymmetry or non-normality, studies of FA must consider several other important factors before interpreting the results of their analysis. First, the bilateral trait or traits selected for measurement should not experience wear or remodeling (A R Palmer & Strobeck, 2003). Second, these traits should be easily and precisely found and replicated within and among individuals.

Many morphometric studies use the terminology of Bookstein (1997) and collect only "type 1" or "type 2" landmarks. Type 1 landmarks are defined by the intersection of boundaries or the juxtaposition of tissues (e.g., the intersections of suture lines on the human skull), while type 2 landmarks are points of maximum curvature on relatively smooth surfaces or contours (Bookstein, 1997). In both cases, landmarks must be unambiguously defined prior to data collection to minimize measurement error and ensure that the same point is collected from all specimens. Finally, once data has been collected, it should be evaluated for antisymmetry prior to any formal statistical testing. As described above, antisymmetry would be suggested by bimodality in the distribution of signed asymmetry values (or, for multivariate data, a principal component analysis of the vectors containing the individual left-right asymmetries would exhibit abnormal clustering of data points).

Once data has been properly collected and checked for antisymmetry, a generalized Procrustes superimposition (or fit) can be used to extract information about asymmetry in shape from the landmark coordinates collected for each individual. A Procrustes fit removes variation in size, position, and orientation, standardizing them so that the only remaining component of variation is shape. Beginning with the raw data (the digitized landmarks for each specimen), all landmark configurations are scaled to a centroid size of 1.0. The centroids of all configurations are then shifted (translated), so that they fall into the same position (the origin, or coordinates of 0,0). Finally, the sum of squared distances between each specimen's corresponding landmarks is minimized, rotating each landmark configuration into the same position. After the distance between corresponding landmarks has been minimized with the Procrustes superimposition, the

remaining difference is purely shape difference (Klingenberg, 2015; Klingenberg & McIntyre, 1998). In a generalized Procrustes analysis (which standardizes more than two individuals), all landmark configurations are fit to the first, or target, landmark configuration. An average configuration is then generated, and the superimposition process repeated. A new average configuration is created, and the superimposition process is repeated yet again (and so on), until the newest average configuration does not differ from the previous one (Klingenberg, 2015). Following the Procrustes superimposition, a conventional analysis of variance (two-factor ANOVA, or MANOVA in the multivariate case) can be used to evaluate shape asymmetry (Klingenberg & McIntyre, 1998; A R Palmer, 1994; A R Palmer & Strobeck, 1986).

Because FA results from random events during development (A R Palmer, 1994; A R Palmer & Strobeck, 1992), some studies have generated a measure of the magnitude of FA for each individual to evaluate developmental imprecision by proxy (e.g., Anders Pape Møller & Swaddle, 1997; Polak, 2003). One such measure (or individual "score" of asymmetry) is the Procrustes distance between the original and reflected copies of the landmark configuration (in structures with object symmetry, such as the human skull) (Klingenberg, 2015). Note that this is a measure of the overall magnitude of shape difference per individual, rather than an actual measurement of "distance" per se. In addition to animal studies not mentioned here, Procrustes distance has been used in recent morphometrics studies of the human skull (Pound et al., 2014; Weisensee, 2013).

1.6.1.3 Fluctuating Asymmetry in Humans and the DOHaD Hypothesis

Studies have generally indicated that environmentally-produced FA is most pronounced when the population exists in a marginal environment and the stressor (e.g., temperature extremes, pollutants or toxins, etc.) is experienced prenatally (Parsons, 1990). In human studies, this has led to an interest in socioeconomic status (SES) differences in FA, or changes in FA levels within a single population over time. Most human studies have focused on the skull, though examples of studies of asymmetry in the postcranial skeleton exist (e.g., Albert & Greene, 1999; Barış Özener, 2010b). This discussion focuses on studies of the human skull, as the skull is less subject to directional asymmetry issues generated by biomechanic factors such as hand dominance.

The last decade has produced several anthropological studies of FA in the human skull. DeLeon (2007) evaluated craniofacial FA as a measure of environmental stress in two cemeteries from medieval Kulubnarti, Sudanese Nubia. Her results indicated that the early Christian period, which is believed to have been the more stressful time period, exhibited greater overall FA in the craniofacial skeleton compared to the late Christian period. Another study of skulls from medieval and 20th-century Poland has revealed higher values of FA in the modern individuals, which the authors argue is related to the environmental pollutants associated with a modern environment (e.g., toxins, tobacco smoke, etc.) (Gawlikowska et al., 2007). A study of individuals from prehistoric Japan by Hoover and Matsumura (2008) produced mixed results, suggesting that FA decreased somewhat (not significantly) between the more stable Middle Jomon and Okhotsk periods compared to the Late/Final Jomon period. The results of this study appear to contradict previous studies of FA *and* previous archaeological findings about the

nutritional and developmental stability of the examined historical periods; however, these results may be related to the study's limited number of FA measurements (3 distances), small sample size (n = 49), and large number of sampled archaeological contexts (13 sites).

In an interesting study of the differences in facial FA between the sexes under different socioeconomic conditions, Özener (2010a) found that FA was greater in the lower SES group, and males had greater FA than females (but only in the lower SES group). Özener notes that literature on sex-based FA differences is mixed, and does not necessarily support the assertion that females have a higher buffering capacity (and therefore greater developmental stability and decreased FA) compared to males. That statement aside, the lack of a significant difference between the sexes in the higher SES group suggests that males and females experience similar developmental stability when environmental stresses are low, but poor living conditions (higher environmental stress) may differentially affect developmental stability in males (Barış Özener, 2010a). Another study of SES and facial FA in adults from ten Latin American cities appears to conflict with other studies supporting a significant association between FA and SES, as it found no significant correlation between facial FA and measures of SES (Quinto-Sánchez et al., 2017). However, the interpretations of the study may be dependent upon the significance level selected for analysis ($p \leq 0.01$), which altered their conclusions despite correlation values between facial FA and four principle component scores for indicators of wealth that would typically be considered statistically significant ($p = 0.051, 0.100, 0.028, 0.036$). Regardless, the same study also demonstrated associations between FA and age (increased FA at older ages) and FA and sex (increased FA in females) (Quinto-Sánchez

et al., 2017). As in the study by Özener (2010a), Quinto-Sánchez and colleagues (2017) acknowledge that the finding that FA was more pronounced in females differs from much of the published literature, which generally shows higher FA in males.

Finally, some studies have explicitly addressed the potential for FA to serve as a skeletal indicator of early life stress, which can be used to evaluate bioarchaeological support for the Developmental Origins of Health and Disease (DOHaD) hypothesis. In a study of British children, Pound and colleagues (2014) found no evidence of an association between facial FA and measures of childhood health. However, the authors suggest that their results may have to do with their sample: modern medicine potentially limits exposure to pathogens and nutritional stress, and could therefore have affected the expression of FA due to developmental stress. Pound and colleagues (2014) also admit that previous demographic analyses on their data set (the Avon Longitudinal Study of Parents and Children, ALSPAC) have demonstrated that the data have an overrepresentation of affluent children, perhaps limiting the applicability of their findings to other populations. I would also add that an overrepresentation of wealthy children may limit the observed pattern between FA and childhood health, given that lower SES is expected to correlate with negative health outcomes and more pronounced facial FA. In contrast to the study by Pound and colleagues (2014), studies by Watts (2013a) and Weisensee (2013) found associations between early life developmental stability (evinced by FA values) and later life morbidity and mortality. In a study of childhood health indicators and adult mortality, Watts (2013a) found that cribra orbitalia presence and FA values corresponded with earlier mortality in females living in northern England prior to 1700 A.D. Age at death in males, however, was associated with cribra orbitalia presence,

small vertebral neural canals, and short femoral lengths. Likewise, age at death in males and females in a group from after 1700 A.D. was only associated with small vertebral neural canals. Watts (2013a) suggests that environmental and economic changes in the post-1700 A.D. period protected that population from the early mortality associated with developmental insults (e.g., higher FA values) seen in the pre-1700 A.D. period. In the study by Weisensee (2013), individuals from 19th- and 20th-century Portugal exhibited greater degrees of FA if they were dying of degenerative diseases, compared to those individuals dying of infectious diseases. In contrast to Watts (2013a), Weisensee (2013) found a non-significant trend between FA and sex (i.e., males had higher FA values than females), which is broadly consistent with similar trends in the FA literature and possibly related to enhanced male sensitivity (or female buffering). The inconsistent relationship between sex and FA in the anthropological and biological literature calls for a closer look at the contexts of the studied samples, as some unexamined cultural and environmental factors may be influencing observed patterns of FA by sex between studies and over time.

The articles by Watts (2013a) and Weisensee (2013) highlight the potential utility of FA as an indicator of developmental instability in bioarchaeological studies of the DOHaD hypothesis. Studies that want to explore support for the DOHaD hypothesis in the bioarchaeological record need to find and use as many skeletal indicators of childhood health as possible. Watts (2013a), for example, used cribra orbitalia presence, femur length, vertebral neural canal size, and FA. Indicators like vertebral neural canal size, enamel hypoplasia, and FA are particularly important because they are largely permanent and are not remodeled in later life. As a result, a combination of skeletal

indicators can be used as gauges of health during specific portions of infancy or childhood (e.g., linear enamel hypoplasia and vertebral neural canal size), or during the developmental period generally (e.g., cribra orbitalia and craniofacial FA), and can be evaluated for associations with age at death or mortality risk.

1.6.2 Linear Enamel Hypoplasia

Linear enamel hypoplasia (LEH) are horizontal grooves or bands that develop on teeth in response to a variety of developmental disruptions (A. H. Goodman & Rose, 1990). The macroscopically identifiable defects result when ameloblast cells cease functioning, causing a deficit in enamel volume on that portion of the tooth (Hillson, 1996). Although biological anthropologists recognize that microscopically detecting hypoplastic defects is a more reliable method, the time requirements and analytical tools necessary to perform microscopic analyses make it impractical in the field (Hassett, 2014). Instead, this study (and most others) macroscopically detect LEH on the surface of the tooth.

According to Goodman and Armelagos (1985a, 1985b), the frequency of hypoplastic defects is not solely determined by the timing of tooth crown development. In other words, teeth forming at the same time are not equally likely to display LEH. A variety of possible explanations for this phenomenon exist, such as ameloblast concentration at a given time during development, ameloblast secretion rate at a given time, enamel prism length, or enamel prism direction, but evidence from Goodman and Armelagos (1985a) suggests that certain teeth are preferable for the study of developmental disruption, as they are more likely to exhibit macroscopic defects like

LEH. Particularly, the maxillary central incisors are most sensitive during early childhood (it develops from birth to 4.5 years), while the mandibular canine is most sensitive during the overall childhood period (it develops from 0.5 to 6.5 years).

In addition, Goodman and Armelagos (1985a) suggest the polar teeth (i.e., the more anterior teeth in each tooth class) are more likely to display LEH and other enamel defects than the non-polar (posterior) teeth, because they are under greater genetic control and are limited in their responses to developmental disturbance; that is, the developmentally stable teeth (e.g., the central incisors) are constrained in their responses to developmental disruption, while the less developmentally stable teeth (e.g., the lateral incisors) are able to respond to environmental perturbations in a greater number of ways (see discussion of Tooth Size, below).

Importantly, a review of the literature on humans and non-human primates shows that enamel formation, composition, and development is not seen to differ by sex. Instead, it is more likely that post-natal cultural biases (e.g., preferential treatment of males) influence LEH expression compared to biological differences between the sexes (Guatelli-Steinberg & Lukacs, 1999). Because LEH is susceptible to cultural biases, it is also a useful indicator to include in an intersectional framework, as it may expose important intersections between sex and other social and biological categories. Indeed, previous archaeological work suggests that this indicator intersects meaningfully with socioeconomic status (SES). For example, in Peck's (2013) study, LEH was more common among females in a lower status group (non-elites) than in higher status males or females.

In addition to LEH's utility as an indicator of meaningful age, sex, and SES intersections, this study's use of the DOHaD hypothesis also necessitates that LEH be included among a variety of skeletal indicators to examine health in the past. Particularly, a study using multiple indicators should ensure that indicators are informative about as much of the life course as possible, so that pertinent health events are not "missed", falling between the gaps of each skeletal indicator's timespan. Therefore, in the interest of efficiency during data collection, the mandibular canine was selected for study, because it covers a sizeable portion of childhood development (0.5 to 6.5 years, according to Goodman and Armelagos, 1985a) and is sensitive to hypoplastic defects compared to most other teeth. In this study, LEH serves as an "early childhood" skeletal indicator, providing age-specific information on physiological insults experienced between birth and 6 years of age. This use of LEH as an indicator of early life (compared to indicators of adolescence and later life) health insults has already been successfully employed in previous bioarchaeological studies (e.g., Watts, 2015).

1.6.3 Periosteal New Bone Formation

Periosteal new bone formation (PNBF) is a proliferation of bone in response to trauma or strain to the overlying periosteum (Ortner, 2003). Like LEH, bioarchaeologists often characterize PNBF as a nonspecific indicator of physiological insult (Larsen, 1997a). Injury, infection, nutritional imbalance, neoplastic disease, congenital disease, and other "stresses" have all been connected to periosteal lesion presence (E. M. Chen, Masih, Chow, Matcuk, & Patel, 2012; Ortner, 2003; Paine & Brenton, 2006). This study

is unconcerned with the etiology of the lesions but joins previous studies in using them as a general, nonspecific source of information about health insults in the past.

Numerous bioarchaeological studies have suggested that PNBF may be more frequently associated with older individuals (DeWitte, 2014b; Grauer, 1993; Jerome C. Rose & Hartnady, 1991; Yaussy, DeWitte, & Redfern, 2016). The positive association between age and PNBF may relate to diminished diseased resistance with increased age (Jerome C. Rose & Hartnady, 1991) or the accumulation of nonlethal insults over time (Grauer, 1993). Previous research has also demonstrated that PNBF presence can vary by sex, for biological or cultural reasons (Yaussy et al., 2016). Skeletal indicators that may demonstrate patterns of variation with age or sex are of interest to population health researchers employing an intersectional perspective (i.e., in studies that examine how biological, social, and cultural characteristics determine patterns of health and wellbeing for different domains within the larger population).

In addition to exploring how PNBF presence varies among subpopulations, part of this research project examines whether PNBF activity (active vs. healed lesions) is indicative of heterogeneity in frailty within past populations. Previous research by DeWitte (2014a) suggests that healed PNBF lesions are associated with higher survivorship (lower frailty), while active lesions and no lesions at all are associated with relatively lower survivorship (higher frailty). Therefore, to capture heterogeneity in frailty (the Osteological Paradox) and examine the impacts of intersecting biological and social distinctions on health (intersectionality), this study analyzes data on both the presence/absence and activity level of PNBF.

1.6.4 Cribra Orbitalia

Individuals with cribra orbitalia (CO) exhibit porous lesions on the orbital roofs resulting from marrow expansion in the diploe of the skull (Ortner, 2003). Although most bioarchaeologists associate CO with iron deficiency anemia (i.e., iron deficiencies instigate systemic responses to hemoglobin levels by expanding red blood cell-producing marrow), the etiology of CO has been subject to debate. Walker and colleages (2009) suggested that iron deficiency is inconsistent with the marrow expansion and outer table resorption associated with the lesions, as it impedes hemoglobin synthesis and decreases red blood cell production. Instead, the authors argued that CO is best explained by scurvy, rickets, hemangiomas, and traumatic injuries causing subperiosteal hematomas.

Scholars swiftly responded to Walker et al. (2009), arguing that their dismissal of the iron deficiency anemia hypothesis was premature. Oxenham and Cavill (2010, p. 200) agree that there are potentially multiple etiologies for CO, but explain that a lack of available iron incurs a "vastly increased level of ineffective erythropoiesis" resulting in "erythroid hyperplasia of the bone marrow". In addition to agreeing with Oxenham and Cavill (2010), McIlvaine (2013) argues that dismissal of the iron-deficiency anemia hypothesis has unintended consequences in terms of the effect its dismissal would have on previous publications. McIlvaine's (2013) argument is that vitamin B_{12} deficiency and iron deficiency likely could (and do) co-occur, and—if Walker and colleagues (2009) are correct—would lead to unacknowledged hidden heterogeneity in the archaeological record; that is, inhibition of marrow hypertrophy caused by iron deficiency (as described by Walker et al., 2009) could mask the co-occurrence of vitamin B_{12} deficiency, and individuals would not show CO and, despite being incredibly frail, would mimic

"healthy" individuals without deficiencies (because both groups would lack lesions). Importantly, the McIlvaine (2013) argues that CO, regardless of etiology, is still an indicator of nutritional inadequacies or disease in a population and is useful as a bioarchaeological indicator of frailty in a population.

McIlvaine's (2013) comments are particularly significant for this study, because it intentionally seeks out hidden heterogeneity without attempting to differentially diagnose the etiology of particular lesions. As McIlvaine (2013) noted, CO is an indicator of frailty in the past that is available to bioarchaeologists studying human skeletal remains. Even if we are uncertain about CO's etiologies, previous research has demonstrated that CO presence is associated with elevated risks of mortality (e.g., DeWitte, 2014a), making it a useful inclusion in studies of past population health. Further, this study directly responds to the concerns of authors like McIlvaine (2013) by investigating the activity level of CO lesions, potentially providing meaningful information about heterogeneity and differential frailty in the past (the Osteological Paradox). In addition, since CO is typically associated with childhood anemia, it can serve as a useful indicator of how childhood health insults influence later health and mortality patterns (DOHaD).

1.6.5 Tooth Size

Guagliardo (1982) presented one of the earliest arguments for tooth crown size as an nonspecific indicator of chronic developmental disruption. Based on evidence that lower birth size is associated with smaller teeth (Garn, Osborne, & McCabe, 1979), Guagliardo (1982) reasoned that early chronic stress influences later life frailty, causing earlier mortality in stressed individuals compared to their less- or non-stressed

counterparts. Guagliardo's (1982) results confirmed that individuals dying as juveniles (the "more stressed" group) had smaller teeth than individuals surviving to adulthood (the "less stressed" group).

Stojanowski and colleagues (2007) have since supported Guagliardo's (1982) findings in a study of tooth size and age-at-death. Their results show that individuals who were most stressed (evinced by their smaller teeth) also died at a younger age (evinced by their death as non-adults). Stojanowski et al. (2007) provide supporting evidence that nongenetic, environmental stresses (e.g., poor diet or illness) influence tooth size, particularly in the nonpolar teeth (i.e., the posterior teeth in each tooth class). These results are consistent with the arguments presented by Goodman and Armelagos (1985a), who suggested that the developmentally stable polar teeth will exhibit enamel defects rather than size variations, while the non-polar teeth, which are genetically less constrained, are able to respond to environmental perturbations by slowing development and decreasing size.

Like the study by Stojanowski et al. (2007), this study uses tooth size as a record of childhood health. The permanent non-polar premolars and molars form during childhood, with the crown of the second premolar forming between ages 2-7 years and the crown of the second molar forming between ages 3-8 years (W. H. Logan & Kronfeld, 1933; C. F. Moorrees, Fanning, & Hunt, 1963). Like LEH, tooth size serves as an indelible record of physiological insults occurring during a very specific period of development, and thus can be informative about how developmental disturbances influence adult health and mortality (DOHaD).

1.6.6 Vertebral Neural Canal Size

Although the connection between diminished neural canal size and premature mortality was recognized over thirty years ago (G. A. Clark et al., 1986), few bioarchaeological studies have utilized vertebral neural canal (VNC) size as an indicator of developmental insult. Clark and colleagues (1986) were the first biological anthropologists to suggest that stressors occurring during the prenatal and early postnatal periods of growth and development impacted the adult lifespan. They argued that small VNC size reflects reduced genetic growth potential due to environmental stress during development, and VNC stunting is accompanied by impaired neural and immune function that may be connected to poor health in adulthood and reduced longevity. The results of the study supported the authors' hypotheses: individuals who died at younger ages (i.e., 15-25 years of age) had significantly smaller transverse and anteroposterior VNC diameters in the lumbar region compared to individuals who died at later ages (G. A. Clark et al., 1986).

In the decades since the study by Clark and colleagues (1986), the work of Rebecca Watts (2011, 2013a, 2013b, 2015) has been invaluable in the advancement of vertebral neural canal research in bioarchaeology. Particularly, her study investigating the exact ages at which the midsagittal (i.e., the anteroposterior) and interpedicular (i.e., transverse) diameters of the neural canal reached their final adult sizes in a historical population has been foundational to studies seeking to use the two neural canal diameters as indicators of different developmental periods in childhood and adolescence. Watts determined that, in individuals from the East Smithfield Black Death cemetery in London (AD 1348 – 1350), the midsagittal diameter reached adult size by 3-5 years of age,

whereas the interpedicular diameters continued to increase in size until 15-17 years of age (R. Watts, 2013b). Another study by Watts (2011) confirmed the relationship between reduced VNC size and earlier age at death, and, perhaps more importantly, demonstrated the utility of using multiple skeletal elements to show which periods of development are most closely linked with adult mortality. The results of that study suggested that stress during early childhood (evinced by reduced VNC size) had a more profound impact on adult health than later stress episodes (evinced by reduced stature), at least among males (Watts, 2011). A third study by Watts (R. Watts, 2013a) found that cribra orbitalia, small transverse VNC diameters, and short femoral length in males and cribra orbitalia and craniofacial fluctuating asymmetry in females were associated with a younger age-at-death in individuals from Lincolnshire, England (AD 1150-1855). Most recently, Watts (2015) used hazards analysis to evaluate the effect of reduced VNC size on adult age-at-death, finding that growth disruptions during infancy and early childhood (evinced by small anteroposterior VNC diameters) were not associated with reduced longevity, but growth disruptions during late childhood and early adolescence (evinced by small transverse VNC diameters) was associated with a significantly increased risk of mortality in adulthood. Importantly, the results indicated that the association between small transverse VNC diameters and age-at-death was influenced by sex and socioeconomic status, suggesting that sex and status mediated exposure to hazards and resistance to stressors in later medieval and post-medieval London (Watts, 2015). In the same issue of the American Journal of Physical Anthropology, Newman and Gowland (2015) presented their findings regarding the potential for vertebral body height and transverse VNC diameter to be used as skeletal indicators of stress in non-adult

individuals. The authors conclude that the two measurements provide complementary information about the timing and ramifications of early life stress events and can be used to construct growth profiles akin to those produced using long bone length (Newman and Gowland, 2015).

Like the study by Watts (2015), this dissertation project examines the influence of sex and socioeconomic status on VNC size. Particularly, the anteroposterior and transverse diameters are used as separate skeletal indicators that reflect two different developmental periods—that of infancy and early childhood (evinced by the anteroposterior diameter) and later childhood and adolescence (evinced by the transverse diameter). However, this study also incorporates an intersectional perspective into the analyses (i.e, 2- and 3-way ANOVA) and interpretations, and thus investigates how the two variables interact with each other and macro-level systems of oppression to produce patterns of frailty and mortality in skeletal remains from industrializing England.

1.7 The Osteological Paradox

The term "osteological paradox" was originally conceived by Wood and colleagues (1992) to describe the problems inherent in using skeletal evidence to make conclusions about the health of past populations. In their publication ("The Osteological Paradox: Problems of Inferring Prehistoric Health from Skeletal Samples"), the authors address three of the biggest problems confronting paleodemography and paleopathology today: demographic non-stationarity, selective mortality, and hidden heterogeneity in risks. Stationary populations are closed to migration, have constant age-specific fertility and mortality, have a zero growth rate, and have an equilibrium age distribution, whereas

nonstationary populations are growing or declining because birth and death rates are not equal. Importantly, in moderately nonstationary populations (such as those typical of bioarchaeological studies), fertility impacts the age-at-death distribution more than changes in mortality, and mean age at death is the inverse of the birth rate rather than an approximation of the expectation of life (Sattenspiel & Harpending, 1983). The primary focus of the article by Wood and colleagues (1992), however, is on selective mortality (i.e., all skeletal collections are made up of individuals at the highest risk of death at a given age, instead of individuals that survived that age) and hidden heterogeneity (i.e., individuals vary in their frailty—their susceptibility to disease and death—which stems from a variety of sources and makes it nearly impossible to interpret population-level age-specific mortality rates in terms of individual risk of death) (J. Wood et al., 1992).

Heterogeneity is problematic because it means that an aggregate measure of the population does not approximate the real risk of death for any subpopulation until that subpopulation makes up a large portion of the total population; therefore, population-level measures mortality patterns (e.g., life tables) are utterly useless for determining individual or subgroup risks of death (Wood et al., 1992). However, Wood and colleagues (1992) were not the first scholars to identify the problem of heterogeneity in individual frailty. Vaupel and colleagues (Vaupel et al., 1979; Vaupel & Yashin, 1985) identified the issues posed by standard life table methods more than a decade earlier, noting that traditional life tables required correcting to reflect actual mortality patterns. Vaupel and colleagues (1979) note that the gradual removal of relatively frail individuals from a cohort due to selective mortality causes a progressive decrease in the cohort's average frailty, and, at the very least, modifications to life table methods should include

adjustments for age-invariant heterogeneity in frailty. Without alterations, standard life table methods have likely produced biased estimates (e.g., underestimation of individual aging rates, progress in mortality reduction, and differences in mortality between populations; overestimation of current life expectancy and the gains in life expectancy that can be expected by avoiding certain causes of death). Such biases can lead to confusing results and erroneous conclusions when comparing the mortality rates of multiple groups. The "gradual removal of relatively frail individuals" mentioned above is particularly salient when the primary difference between the groups is advantage or resource access. A disadvantaged group may appear to do better than an advantaged group in terms of frailty and mortality at later ages; however, this phenomenon is actually produced because a greater number of relatively frail individuals in the disadvantaged group have been removed by mortality, causing a decrease in the average frailty of the disadvantaged cohort as a whole, causing it to approach or drop below the average frailty of the advantaged cohort at later ages (Vaupel et al., 1979). Vaupel and Yashin (1985) would later reiterate this point on "heterogeneity's ruses", stressing that an overall population's hazard rate (or risk of death) was dependent on the changing proportions of the subpopulations included in the aggregate, and no population-level mortality pattern would accurately approximate the patterns of the subpopulations that made up the larger population.

Likewise, Wood and colleagues (1992) argued that selective mortality is problematic because individuals with bony lesions may have been at greater risk of death, meaning that the frequency of active bony lesions in a skeletal sample likely overestimates the actual prevalence in the living population (because those with active

lesions were frail and thus "selected" for death). Further, eliminating one cause of death—typically considered an "improvement" in health—causes an increase in the proportional mortality from the remaining causes of death (e.g., decreases in infectious disease mortality increases the rate of cancer mortality) (Wood et al., 1992).

Lesion presence as an indicator of poor health was an especially problematic assumption in previous studies of population health using skeletal data. These early studies posited that an increase in lesion frequencies in a skeletal sample indicated an elevation in the population prevalence of the conditions or stressors that create the lesions of interest (e.g., the adoption of agriculture was associated with high frequencies of skeletal lesions associated with undernutrition and infectious disease). However, the issues of selective mortality and hidden heterogeneity combine to influence lesion presence. Wood et al. (1992) describe a fictitious population composed of three subpopulations that vary in their frailty and exposure to a lesion-causing condition (or stressor): the first subpopulation never experiences the condition, and never develops lesions; the second subpopulation experiences the condition, survives long enough to produce skeletal lesions, and ultimately lives to be killed by some other factor later on; the third subpopulation experiences the condition and dies so quickly that they are unable to develop lesions. When examining the skeletal data, however, there appear to be only two subpopulations—those with lesions (the seemingly "unhealthy" group) and those without (the seemingly "healthy" group). This example illustrates the problem with using skeletal lesions as straightforward indicators of "health" (however it is defined). Some individuals may not have encountered the lesion-causing condition or stressor. Some individuals may have experienced the stressor and been so frail that they died almost

immediately. Both groups would be included in the "healthy" category because they lack lesions, when, in reality, the subpopulation that died quickly was actually at greater risk of death (frailer) than either of the other two subpopulations (i.e., the one that avoided the stressor and the one that survived the stressor). At the same time, the group with lesions may actually represent relatively less frail individuals with an enhanced ability to survive episodes of illness or stress (Wood et al., 1992).

Subsequent replies and progress updates (M. N. Cohen, 1994; DeWitte & Stojanowski, 2015; Wright & Yoder, 2003) further clarified the issues faced by bioarchaeological studies, but offered hope in the form of methodological advancements. Wright and Yoder (2003) review progress in several areas, identifying recent improvements in sex estimation (e.g., supero-inferior femoral neck diameter, hand/foot bones, and DNA-based methods), age assessment (e.g., dental histology, sternal rib end morphology, and transition analysis), hazard modeling (as an alternative to life tables; see Gage, 1988), ancient DNA and isotope analyses, and Bayesian or maximum likelihood approaches. Continued growth was described by DeWitte and Stojanowski (2015), who recognized several themes in bioarchaeological studies that have directly addressed the issues of the Osteological Paradox. In the decades following the original publication, studies have echoed Wood et al.'s (1992) suggestion to focus on simple/egalitarian societies or short-term use cemeteries that limit demographic nonstationarity, and encourage consideration of intrasite/interindividual patterns of variation. Scholars have also begun considering individuals with a young age-at-death to be non-survivors who succumbed to death sooner than survivors that lived to older ages. Additionally, growth in related fields has contributed to improvements and a diverse array of applications

within bioarchaeology (e.g., genetic determinants of disease, epigenetics, and DOHaD). However, the integration of the Osteological Paradox into bioarchaeological methodologies and interpretations is far from ubiquitous and must continue to spread among the field's scholars and researchers (DeWitte & Stojanowski, 2015).

Following DeWitte and Stojanowski (2015), this study addresses some of the issues presented by Wood and colleagues (1992) by selecting a diverse range of skeletal indicators of stress and utilizing them to their fullest. Recent discussions and developments in paleopathology have made progress in determining or clarifying the etiologies or progression of common indicators of frailty, such as cribra orbitalia, porotic hyperostosis, and periosteal new bone formation (e.g., the discussion between Walker et al., 2009 and authors like Oxenham and Cavill, 2010 and McIlvaine, 2013). Although many of the skeletal indicators used in bioarchaeological studies lack the specificity available in modern studies of health (i.e., many indicators are "nonspecific" and cannot be differentially diagnosed or traced to a particular disease or stressor), bioarchaeologists have expanded the number of indicators that can be used to examine past health, as well as the information that can be gleaned from each indicator. For example, a study by DeWitte (2014a) suggests that the activity of periosteal lesions (i.e., active, mixed, or healed) may be indicative of varying levels of frailty. As a result, what was previously considered a single indicator (periosteal new bone formation, and perhaps lesions like cribra orbitalia and porotic hyperostosis as well) has been divided into multiple indicators that can be informative about different levels of frailty. By approaching skeletal indicators as multifaceted indicators of frailty, bioarchaeological studies have the opportunity to capture more heterogeneity in frailty than previously possible and generate

new and varied hypotheses about how selective mortality operated in the past. In addition, studies that utilize a diverse range of indicators can examine different aspects of past health, as different indicators may be more or less sensitive to the stresses experienced by individuals in different contexts. For example, Yaussy and colleagues (2016) determined that linear enamel hypoplasia (instead of cribra orbitalia) was the more sensitive indicator of frailty in the context of famine in medieval London, but recognized that cribra orbitalia could still be informative in other contexts and studies.

Lastly, as mentioned above, studies that employ a variety of indicators can also examine health and stress at different ages, which allows bioarchaeologists to examine stress and frailty throughout the life course. Assessing age-structured data is one way that bioarchaeological researchers have approached the issues of the Osteological Paradox. An age-structured approach is comparative, as it considers non-adult individuals to be "non-survivors" (i.e., individuals who succumbed to a stressor at a young age, particularly physiologically risky periods like weaning), while older individuals are "survivors" (i.e., individuals who survived the same stressor or period). Survivors are expected to be less frail than non-survivors, allowing bioarchaeologists to directly evaluate how lesion frequencies differ between frail individuals (non-survivors) and less-frail individuals (survivors). Focusing on non-adults is particularly useful for bioarchaeologists interested in simultaneously exploring the DOHaD hypothesis with skeletal data, and studies have already begun investigating how developmental disturbances influence later life frailty and survival. Stojanowski and colleagues (2007), for example, have demonstrated that small crown size was more common in non-adults (non-survivors) than in adults (survivors), suggesting that physiological disturbances in

early life—evinced by small teeth—impacted later survivorship. In this dissertation project, multiple skeletal indicators that are relevant to specific non-adult age ranges or to the non-adult period generally are examined to expose patterns in frailty associated with the non-adult period. In other words, by adding a variety of age-based skeletal indicators of health to new studies, bioarchaeologists can expose patterns of heterogeneity in frailty that were previously "hidden" and expose how developmental stress impacts the action of selective mortality in adulthood.

1.8 Dissertation Structure

This dissertation is separated into four complementary chapters that each address one or more of the research objectives outlined above. In Chapter 2, I introduce intersectionality as a theoretical approach and provide an example of how three frequently-used skeletal indicators of stress (cribra orbitalia, periosteal new bone formation, and linear enamel hypoplasia) can be analyzed with hierarchical log-linear analysis to examine patterns of frailty and mortality across various age, sex, and socioeconomic axes. In Chapter 3, I use tooth size and vertebral neural canal dimensions to examine the complex associations among adult age at death, sex, status, and early life stress in the context of industrial-era England. Particularly, I emphasize how tooth size and VNC size can be used as bioarchaeological evidence for the DOHaD hypothesis. In Chapter 4, I use craniofacial fluctuating asymmetry as an additional example of a nonspecific skeletal indicator of early life stress that can be utilized by bioarchaeological studies incorporating intersectionality and the DOHaD hypothesis. Chapter 5 is devoted to an examination of sex and SES patterns of mortality, and the ways in which hazard

analysis can be employed to examine intersectionality in the context of industrializing England. Lastly, in Chapter 6, I summarize the findings of the dissertation and discuss contributions and potential future directions.

CHAPTER 2

THE INTERSECTIONS OF INDUSTRIALIZATION: VARIATION IN SKELETAL INDICATORS OF FRAILTY BY AGE, SEX, AND SOCIOECONOMIC STATUS IN 18TH- AND 19TH-CENTURY ENGLAND[1]

[1] Yaussy, SL. Submitted to the *American Journal of Physical Anthropology*, 02/28/2019.

2.1 Introduction

2.1.1 Intersectionality and Health

Intersectionality theory argues that outcomes tied to intersecting marginalizations (such as race, sex, and class) at an individual level are intertwined with structural inequalities in society (such as racism, sexism, and classism) (Bowleg, 2012). Originally articulated by Black feminist scholars (Collins, 2015; Crenshaw, 1989, 1991), intersectionality approaches acknowledge that multiple marginalizations (e.g., race and sex for Black women) are mutually constituted and reciprocally constructed—neither identity exists in a vacuum and each identity is influenced by the existence of the other identity—and cannot be fully investigated or understood by methodologies that treat them as distinct categories of analysis (Collins, 2015). Researchers incorporating intersectionality, therefore, must be aware that intersections among advantaged and disadvantaged axes—which vary within the larger structural context—can be more central to the nature of experiences or health outcomes than any of the axes of inequality considered separately (Veenstra, 2011). Although intersectionality's central tenet of multiple, mutually constitutive identities presents a challenge to quantitative researchers (i.e., the interaction among more than two variables is difficult to interpret), researchers investigating intersectionality in living populations have successfully approached the issue using a variety of analytical methods (e.g., Hinze, Lin, & Andersson, 2012; Seng, Lopez, Sperlich, Hamama, & Reed Meldrum, 2012; Stirratt, Meyer, Ouellette, & Gara, 2008).

Compared to researchers working with living populations (e.g., Hinze et al., 2012; Longman Marcellin, Bauer, & Scheim, 2013; Sen & Iyer, 2012; Seng et al., 2012; Stirratt et al., 2008; Veenstra, 2011; Warner & Brown, 2011), bioarchaeologists have largely

avoided intersectionality theory when examining patterns of health in the past. Of the authors who incorporate intersectionality into their studies, few explicitly discuss the tenets of the perspective or generate methods that could be replicated by scholars interested in applying such an approach. The most comprehensive study to integrate bioarchaeological data with an intersectional perspective comes from Torres-Rouff and Knudson (2017), who use data from northern Chile to explore mutable and immutable aspects of identities held at the individual, community, and regional levels. Because the authors leverage multiple lines of evidence (e.g., cranial metric and nonmetric traits, isotope analyses, mortuary context) to examine individual- and population-level changes over time, they examine how social positions are negotiated within larger structures of power and oppression. The article by Torres-Rouff and Knudson (2017) provides one example of how an intersectionality approach can be applied in bioarchaeological studies to answer larger anthropological questions. For example, an intersectional bioarchaeology can demonstrate how micro-level identities intersect with macro-level power structures in a variety of contexts and can add temporal depth to intersectional research being conducted in other disciplines. Further, intersectionality can drastically change how bioarchaeologists approach patterns of health in the past, given that the perspective involves an investigation of how multiple characteristics of interest produce observed patterns of physiological stress and mortality. In this article, the intersections of age, sex, socioeconomic status (SES), and previous health (evinced by nonspecific skeletal indicators of frailty) are analyzed to examine how these factors intersected to influence patterns of frailty and mortality in industrializing England.

2.1.2 Marginalization of Women in Industrial England

Industrialization in England (*c.* 1760-1840) involved changes in the positions of men and women within society, the larger economy, and household economies, as well as a restructuring of the class system and status-based ideologies. Therefore, an intersectional bioarchaeological study of health in industrial-era England must address the economic and social changes that took place in society, to demonstrate how industrialization itself influenced patterns of marginalization that affect health outcomes among gender, age, and SES groups. Though evidence is sparse for women during the industrial period (Gage, 2005), a picture of the transitioning society can be generated by examining what is known about the changing roles of women at the time.

Prior to industrialization, much of England's population lived in rural settings and households produced and consumed in local markets (Gazeley & Horrell, 2013; May, 1987). However, between 1751 and 1851, England experienced a substantial increase in population size, particularly in urban areas (G. Clark, 2007; May, 1987). This dramatic population growth provided the labor force that fueled industrialization, but it was also accompanied by a shift to male-centered employment. For example, although women were traditionally employed in the textile and clothing-making industries, many of the tasks typically relegated to women (e.g., spinning) gradually disappeared into factories as industrialization progressed. Even when they obtained positions within factories or worked from home (e.g., by completing seaming and finishing tasks), women were primarily given low-wage, unskilled positions, while better-paying jobs as clerks, overseers, and managers were given almost exclusively to men. Though domestic service employed large numbers of young, unmarried women and some other sources of

employment existed (e.g., governesses, midwives, shopkeepers, schoolmistresses), it remained that women were largely absent from the building and craft trades, mining, machine-works, and professional positions (Bythell, 1993).

Importantly, these workplace transitions were reinforced by changing ideologies at the cultural level. In the years following 1750, the economic importance of women dropped precipitously—middle-class women were not expected to hold jobs at all and working-class women were only to contribute to the household income as factory workers or domestic servants until marriage (Stearns, 2007). Because of the assumption that women would exit the workforce when they married, skills were considered unnecessary for young female workers and their wages were often substantially lower than those of men, who, presumably, needed to earn enough to support an entire family (Bythell, 1993). In addition to wage inequality, women faced discrimination at work as well. For instance, in the agricultural and mining sectors, both employers and male laborers openly discriminated against female laborers (Bythell, 1993; Nicholas & Oxley, 1993). Eventually, the idea developed that women were mentally and physically incapable of certain types of work, further reinforcing a gendered division of labor in industrial England (Bythell, 1993).

2.1.3 Industrialization and Health

Studies of standards of living among manual laborers, based on wage data, reveal only a marginal increases in living standards—and, by extension, health—between the 1780s and 1850s (C. H. Feinstein, 1998), with no substantial improvement in wages or standard of living until at least 1820 (G. Clark, 2001, 2005). Analyses of height and

weight data suggest a more complex pattern, indicating that the effects of industrialization varied by age, sex, or location (e.g., Meredith and Oxley, 2015). However, despite the usefulness of historical data, scholars have long recognized the biases of documentary sources. For example, wage data—even when corrected for costs of living and urban health hazards—do not adequately represent the health of the industrializing English population. Further, studies of historical data are often biased towards potentially unrepresentative segments of the working class (e.g., agricultural laborers, convicts, military recruits), offering little information on the experiences of other English men and women, such as the middle class. Finally, although data on industrialization-era mortality are available from other historical sources (e.g., the London Bills of Mortality begin recording causes of death in the 17th century), the descriptions in such sources are often inaccurate and do not provide an adequate description of the health of the living population from which the deceased come (DeWitte, 2014c).

Bioarchaeological studies have improved our understanding of SES and health in industrializing England by including individuals typically omitted from the historical literature. A study of two industrial-era cemeteries from London found that low-status females had lower mean age at death than high status males, high status females had a greater mean tibia length than low-status females, and low-status males were less likely to have short tibiae than low-status females or high-status males (Hughes-Morey, 2016). According to Hughes-Morey, the results suggest that low-status females experienced the greatest amount of stress throughout life, making them relatively more frail and less likely to survive to adulthood when compared to any other subgroup. In contrast, high-

status males who experienced childhood insults that affected achieved stature were still able to survive to adulthood because they were less likely to be exposed to severe and repeated disease and had preferential access to resources (Hughes-Morey, 2016). Hughes-Morey's (2016) results are similar to those of DeWitte, Hughes-Morey, Bekvalac, and Karsten (2016), which suggested that selective mortality among non-adults influenced later population frailty among surviving adults. In that study, the removal of high frailty children of low SES led to relatively low-frailty adults in the low-SES group, while buffered high-SES children would survive childhood and the emergent high-SES adults would be of more heterogeneous frailty than the low-SES adults (DeWitte et al., 2016).

This study uses three skeletal indicators of stress (cribra orbitalia, periosteal new bone formation, and linear enamel hypoplasia) and hierarchical log-linear analysis to examine previously unexposed patterns of frailty and mortality across various age, sex, and socioeconomic axes. Particularly, this study tests the hypothesis that multiply-marginalized individuals (i.e., low-SES females) and individuals exhibiting a mix of marginalized and privileged identities (i.e., low-SES males, high-SES females) will exhibit higher frequencies of skeletal indicators of stress compared to multiply-privileged individuals (i.e., high-SES males). This hypothesis is based on one of the main tenets of intersectionality theory, which suggests that identity categories are not independent, but rather intersect to collectively influence health outcomes within larger structural contexts. Additionally, it is expected that earlier ages at death will be associated with individuals exhibiting at least one marginalized identity (i.e., low SES or female sex).

2.2 Materials and Methods

2.2.1 Skeletal Samples

The skeletal samples used in this study (n = 537)—St. Bride's Fleet Street, Coach Lane, St. Peter's Wolverhampton, and New Bunhill Fields—are dated to the 18th and 19th centuries, which corresponds to the rise of industrialization in England. Samples were selected to represent two of the SES groups existing at the time: the working class (lower-status individuals) and higher-status individuals who had more prestigious occupations, such as doctors or clerks. As such, the skeletal samples used in this study are ideal for investigating status-based differences in frailty and mortality developing during the industrial period, as well as the intersections between biologically- and socially-generated identities that likely played a major role in influencing those mortality patterns.

The higher status skeletal collection used in this study comes from the crypt burials associated with St. Bride's church in central London (also known as St. Bride's Fleet Street). The sample analyzed here is confined to adult individuals known to be interred between 1740 and 1852, and information from the parish register, associated coffin plates, and historical accounts all suggest that the crypt assemblage is composed of higher status individuals (Gustav Milne, 1997). St. Bride's Fleet Street differs from many bioarchaeological skeletal collections in that it has known ages at death for many of the individuals. However, to avoid comparing known ages with estimated ages, age at death was estimated for all individuals in the St. Bride's Fleet Street collection using transition analysis, as described below.

The Coach Lane skeletal sample comes from a Quaker burial ground in North Shields, England, which was known to have been in use from 1711 until its official

closure in 1857. The cemetery was associated with the Society of Friends Meeting House, established in 1698 (Proctor, Gaimster, & Langthorne, 2014). Although the occupations recorded in the Society of Friends monthly meeting minutes suggest that some of the Quakers buried in the cemetery were middle-class individuals (Tancock & Lee, 2014), the social status of the people interred there is not known for certain (Roberts, Caffell, Filipek-Ogden, Gowland, & Jakob, 2016). However, as noted by Roberts and colleagues (2016) in their earlier analysis of the collection, the Quakers were devoted to dressing plainly and adopting simple lifestyles that separated them from what they believed to be modern corruption (Dandelion, 2008), leading this study to conservatively consider the Coach Lane assemblage to be composed of working- or middle-class individuals. As such, the individuals from Coach Lane are included in the lower status group in the analyses presented here.

The overflow cemetery of St. Peter's Collegiate Church in Wolverhampton, England was opened in 1819 and, according to the parish record, likely received its last burial in 1853. Like Coach Lane, the individuals of the Wolverhampton collection are understood to be working- and middle-class individuals and were most commonly buried in earth-cut graves. Although records indicate brick-lined graves existed in the overflow cemetery and would suggest slightly more disposable income among some of the populace, no evidence of brick-lined graves were found upon excavation. The wealthiest individuals in the parish would have afforded burials elsewhere, in family vaults on more attractive parts of the grounds or within the church itself (Adams, 2007). As a result, adults from the St. Peter's Wolverhampton collection are included in the lower status group in this study.

New Bunhill Fields burial ground includes working class individuals from 19th-century London, England. Like St. Peter's Wolverhampton, New Bunhill Fields was used for a relatively brief period, as it was thought to have opened in 1821 and was closed in 1853. Although no burial registers for New Bunhill Fields survive, historical records for the area show that the burial ground catered largely to the local Nonconformist population. Available occupational information suggests that the area's inhabitants were primarily working-class individuals, similar to those found in the Coach Lane and St. Peter's Wolverhampton collections. Moreover, the fees for New Bunhill Fields were relatively low, making it one of the cheapest burial grounds in the area and therefore affordable for even the poorest working-class individuals (Miles & Connell, 2012). Consequently, the individuals from New Bunhill Fields burial ground are included in the lower status group analyzed in this study.

2.2.2 Age Estimation

In this study, adult age at death was estimated for all individuals using transition analysis. Transition analysis uses maximum likelihood estimation to produce point estimates of age for even the oldest individuals in a collection, thus avoiding two of the primary limitations of traditional age estimation methods: age mimicry of the reference sample and broad terminal age categories. For this study, cranial suture closure and skeletal age indicators on the pubic symphysis and iliac auricular surface were used to generate age estimates, as described by Boldsen and colleagues (2002). For the analyses described below, all adults (aged 18+ years) were assigned to the following age at death categories: 18.0 – 24.9, 25.0 – 29.9, 30.0 – 44.9, 45.0 – 64.9, 65.0 + years. The age at

death categories used in this study are irregular in breadth to maximize the number of categories used while still maintaining sufficient sample sizes for each analysis. The age-at-death distributions for each site are presented in **Tables 2.1 – 2.3**, which are separated by skeletal indicator of stress because each indicator is analyzed separately to maximize sample sizes, as described below.

2.2.3 Sex Estimation

This study was limited to only those individuals for whom sex could be estimated using the sexually dimorphic features of the skull and pelvis (Buikstra & Ubelaker, 1994a). When possible, features of the pelvis were given more weight in determining sex compared to features of the skull. For analysis, cases of "probable female" and "probable male" were collapsed into the "female" (0) and "male" (1) categories, respectively. Individuals for which sex was indeterminable or inestimable were not included in the analysis.

2.2.4 Skeletal Indicators of Stress

To assess the intersecting effects of age, sex, status, and physiological stress on mortality during the industrial period, trends in the presence of the following skeletal stress markers were examined: cribra orbitalia, periosteal new bone (periosteal lesions), and linear enamel hypoplasia. Previous studies have shown these lesions to be associated with disease or malnutrition and with elevated risks of mortality (DeWitte & Wood, 2008; Yaussy et al., 2016). The sample sizes and lesion frequencies for each skeletal indicator of stress are presented in **Tables 2.4 – 2.6**, subdivided by cemetery and sex.

Table 2.1 Age-at-death distributions in the periosteal new bone sample, subdivided by site and sex. % = percentages of male or female individuals from each site within each age category.

Periosteal New Bone								
Age	**St. Bride's Fleet Street**		**Coach Lane**		**St. Peter's Wolverhampton**		**New Bunhill Fields**	
	Female	**Male**	**Female**	**Male**	**Female**	**Male**	**Female**	**Male**
18.0 - 24.9	8 (9.6%)	8 (8.2%)	2 (4.2%)	12 (18.2%)	2 (13.3%)	1 (6.3%)	12 (23.1%)	8 (13.8%)
25.0 - 29.9	7 (8.4%)	5 (5.1%)	5 (10.4%)	4 (6.1%)	4 (26.7%)	1 (6.3%)	11 (21.2%)	8 (13.8%)
30.0 - 44.9	15 (18.1%)	19 (19.4%)	16 (33.3%)	14 (21.2%)	5 (33.3%)	8 (50.0%)	19 (36.5%)	26 (44.8%)
45.0 - 64.9	27 (32.5%)	17 (17.3%)	9 (18.8%)	3 (4.5%)	1 (6.7%)	2 (12.5%)	5 (9.6%)	5 (8.6%)
65.0 +	26 (31.3%)	49 (50.0%)	16 (33.3%)	33 (50.0%)	3 (20.0%)	4 (25.0%)	5 (9.6%)	11 (19.0%)
Total	83 (100%)	98 (100%)	48 (100%)	66 (100%)	15 (100%)	16 (100%)	52 (100%)	58 (100%)

Table 2.2 Age-at-death distributions in the linear enamel hypoplasia sample, subdivided by site and sex. % = percentages of male or female individuals from each site within each age category.

Linear Enamel Hypoplasia								
Age	**St. Bride's Fleet Street**		**Coach Lane**		**St. Peter's Wolverhampton**		**New Bunhill Fields**	
	Female	**Male**	**Female**	**Male**	**Female**	**Male**	**Female**	**Male**
18.0 - 24.9	7 (13.0%)	7 (10.8%)	1 (3.7%)	10 (23.3%)	3 (20.0%)	2 (10.5%)	11 (32.4%)	7 (19.4%)
25.0 - 29.9	6 (11.1%)	7 (10.8%)	4 (14.8%)	4 (9.3%)	5 (33.3%)	4 (21.1%)	8 (23.5%)	8 (22.2%)
30.0 - 44.9	13 (24.1%)	17 (26.2%)	10 (37.0%)	10 (23.3%)	4 (26.7%)	10 (52.6%)	13 (38.2%)	17 (47.2%)
45.0 - 64.9	15 (27.8%)	10 (15.4%)	5 (18.5%)	2 (4.7%)	1 (6.7%)	0 (0.0%)	1 (2.9%)	1 (2.8%)
65.0 +	13 (24.1%)	24 (36.9%)	7 (25.9%)	17 (39.5%)	2 (13.3%)	3 (15.8%)	1 (2.9%)	3 (8.3%)
Total	54 (100%)	65 (100%)	27 (100%)	43 (100%)	15 (100%)	19 (100%)	34 (100%)	36 (100%)

Table 2.3 Age-at-death distributions in the cribra orbitalia sample, subdivided by site and sex. % = percentages of male or female individuals from each site within each age category.

Cribra Orbitalia								
Age	**St. Bride's Fleet Street**		**Coach Lane**		**St. Peter's Wolverhampton**		**New Bunhill Fields**	
	Female	**Male**	**Female**	**Male**	**Female**	**Male**	**Female**	**Male**
18.0 - 24.9	7 (8.1%)	8 (8.2%)	2 (4.0%)	10 (16.9%)	4 (16.7%)	3 (11.5%)	16 (29.1%)	8 (13.6%)
25.0 - 29.9	9 (10.5%)	7 (7.1%)	5 (10.0%)	5 (8.5%)	6 (25.0%)	4 (15.4%)	11 (20.0%)	10 (16.9%)
30.0 - 44.9	15 (17.4%)	20 (20.4%)	12 (24.0%)	9 (15.3%)	7 (29.2%)	13 (50.0%)	18 (32.7%)	27 (45.8%)
45.0 - 64.9	29 (33.7%)	19 (19.4%)	10 (20.0%)	3 (5.1%)	2 (8.3%)	1 (3.8%)	4 (7.3%)	5 (8.5%)
65.0 +	26 (30.2%)	44 (44.9%)	21 (42.0%)	32 (54.2%)	5 (20.8%)	5 (19.2%)	6 (10.9%)	9 (15.3%)
Total	86 (100%)	98 (100%)	50 (100%)	59 (100%)	24 (100%)	26 (100%)	55 (100%)	59 (100%)

Table 2.4 The sample sizes and lesion frequencies for the periosteal new bone analyses, subdivided by site and sex. % = percentages of individuals within each sex category and site with and without periosteal lesions.

Periosteal New Bone					
Skeletal Sample	**Females**		**Males**		**Total**
	Present	**Absent**	**Present**	**Absent**	
St. Bride's Fleet Street	34 (41.0%)	49 (59.0%)	38 (38.8%)	60 (61.2%)	181
Coach Lane	15 (31.3%)	33 (68.7%)	21 (31.8%)	45 (68.2%)	114
St. Peter's Wolverhampton	5 (33.3%)	10 (66.7%)	7 (43.8%)	9 (56.2%)	31
New Bunhill Fields	27 (51.9%)	25 (48.1%)	34 (58.6%)	24 (41.4%)	110
Total	81 (40.9%)	117 (59.1%)	100 (42.0%)	138 (58.0%)	436

Table 2.5 The sample sizes and hypoplasia frequencies for the linear enamel hypoplasia analyses, subdivided by site and sex. % = percentages of individuals within each sex category and site with and without linear enamel hypoplasia.

Linear Enamel Hypoplasia					
Skeletal Sample	**Females**		**Males**		**Total**
	Present	**Absent**	**Present**	**Absent**	
St. Bride's Fleet Street	35 (64.8%)	19 (35.2%)	45 (69.2%)	20 (30.8%)	119
Coach Lane	20 (74.1%)	7 (25.9%)	27 (62.8%)	16 (37.2%)	70
St. Peter's Wolverhampton	5 (33.3%)	10 (66.7%)	14 (73.7%)	5 (26.3%)	34
New Bunhill Fields	22 (64.7%)	12 (35.3%)	20 (55.6%)	16 (44.4%)	70
Total	82 (63.1%)	48 (36.9%)	106 (65.0%)	57 (35.0%)	293

Table 2.6 The sample sizes and lesion frequencies for the cribra orbitalia analyses, subdivided by site and sex. % = percentages of individuals within each sex category and site with and without cribra orbitalia lesions.

Cribra Orbitalia					
Skeletal Sample	**Females**		**Males**		**Total**
	Present (%)	**Absent (%)**	**Present (%)**	**Absent (%)**	
St. Bride's Fleet Street	3 (3.5%)	83 (96.5%)	18 (18.4%)	80 (81.6%)	184
Coach Lane	14 (28.0%)	36 (72.0%)	22 (37.3%)	37 (62.7%)	109
St. Peter's Wolverhampton	4 (16.7%)	20 (83.3%)	2 (7.7%)	24 (92.3%)	50
New Bunhill Fields	10 (18.2%)	45 (81.8%)	7 (11.9%)	52 (88.1%)	114
Total	31 (14.4%)	184 (85.6%)	49 (20.2%)	193 (79.8%)	457

Cribra orbitalia consists of porotic lesions on the roofs of the orbits caused by bone marrow hypertrophy (Ortner, 2003; Stuart-Macadam, 1991; Walker, Bathurst, Richman, Gjerdrum, & Andrushko, 2009). Although these lesions are often attributed to iron-deficiency anemia, this etiology is debated (Walker et al., 2009; however, cf. Oxenham & Cavill, 2010 and McIlvaine, 2013), and studies have indicated a variety of sources for the lesions, including localized inflammation, osteoporosis, and rickets (Wapler, Crubézy, & Schultz, 2004). In this study, no attempt was made to determine the source of the lesions. Instead, cribra orbitalia is considered a nonspecific indicator of physiological stress during childhood. Cribra orbitalia was scored using the grading system of Buikstra and Ubelaker (1994a), and activity scores were collapsed into a binary score of "absent" (0) or "present" (1) for analysis.

Linear enamel hypoplasias (LEH) are grooves or pits on the tooth surface caused by the disruption of the enamel formation process (A. H. Goodman, Armelagos, & Rose, 1980; Larsen, 1997b). Often related to bouts of malnutrition or infection, LEH on the permanent teeth are an enduring mark of physiological stress that occurred during childhood, as the tooth was formed (Ortner, 2003). Here, LEH were identified macroscopically on the mandibular canines. These teeth were selected for analysis because they are both sensitive to physiological stress and develop over a relatively long period of time (A. H. Goodman et al., 1980). LEH were scored as "present" (1) if one or more defects were visible to the naked eye under good lighting and "absent" (0) if no defects were visible.

Periosteal new bone formation, which is generated in response to inflammation or trauma to the periosteum (Weston, 2008), differs from the other two skeletal indicators

examined in this study in that it is not restricted to the childhood period. In this study, as with cribra orbitalia and LEH, periosteal lesions were not used to differentially diagnose individuals with a specific disease, but were used as a general indicator of exposure to physiological stressors that may have influenced frailty. Data were only collected from individuals possessing at least two-thirds of the diaphysis of either tibia. The tibiae were selected for analysis because they have relatively little overlying tissue separating them from the external environment, are particularly susceptible to trauma and infection, and have elevated osteogenic potential compared to other skeletal elements (Klaus, 2014; Roberts & Manchester, 2005). In addition, the tibia is relatively robust and, as a result, is often well-preserved in skeletal samples (Galloway, Willey, & Snyder, 1997; C. M. Stojanowski, Seidemann, & Doran, 2002; Waldron, 1997).

2.2.5 Hierarchical Log-Linear Analysis

Hierarchical log-linear analysis—a nonparametric form of analysis that accommodates both categorical and binary variables—was used to investigate the associations among adult age, sex, status, and the skeletal stress markers. Hierarchical analyses are particularly useful in studies of intersectionality (e.g., Stirratt et al., 2008), because they examine multiple intersections of social and biological variables. Particularly, groups in the middle of the social spectrum, who exhibit different combinations of social advantage and disadvantage (e.g., high SES women and low SES men), may experience different health outcomes from individuals who are multiply-advantaged or multiply-disadvantaged (Sen & Iyer, 2012). In addition to identifying multi-way interactions between the variables, this approach uses backwards elimination

to remove nonsignificant interactions among variables, and thus reveals whether significant associations exist between SES and each skeletal stress marker, independent of age or sex (DeWitte & Bekvalac, 2011; J. A. Green, 1988). In all cases in which a statistically significant relationship existed, Chi square tests and histograms of the data were used to determine the nature of the relationship between the variables. In this study, p-values of less than 0.10 were considered statistically significant and reported below. However, in agreement with researchers from a variety of fields (H. W. Cohen, 2011; S. N. Goodman, 1999; Lang, Rothman, & Cann, 1998; Rothman, 1998; Trafimow & Marks, 2015) statistical significance is presented with caution.

To ensure all associations between status groups were the result of socioeconomic inequality rather than geographic differences between the four sites, additional hierarchical log-linear analyses were conducted on a reduced sample that included only the high and low status sites from London (St. Bride's Fleet Street and New Bunhill Fields, respectively). Because the sample sizes for each reduced sample analysis were markedly smaller than the full samples that included all four sites, the results should be viewed with caution. Particularly, for the analysis of LEH presence, age, sex, and status, two analyzed cells contained fewer than five individuals (i.e., for the low status group, the age categories 45.0 – 64.9 years and ages 65.0+ years contained only 2 and 4 individuals, respectively), meaning that any statistically significant age-based associations may not be valid for that analysis.

2.3 Results

Table 2.7 shows the significant two- and three-way interactions found for each skeletal indicator of stress when all sites were examined together (i.e., the full sample). In two cases, three-way interactions existed among the variables analyzed: among periosteal lesions, sex, and age, and among cribra orbitalia, SES, and sex. Chi-square tests revealed the direction of the relationships indicated by the hierarchical log-linear analyses: middle-aged males (30.0 – 44.9 years) exhibited a higher frequency of periosteal lesions compared to females of all ages or males of younger or older ages (**Figures 2.1** and **2.2**), and high-SES females had lower frequencies of cribra orbitalia relative to other groups (**Figures 2.3** and **2.4**). The three-way associations remained consistent when analyses were limited to the London sites (reduced sample). Additionally, cribra orbitalia was more common in males, young adults (18.0 – 29.9 years), and individuals of low SES. No associations were observed between the presence of linear enamel hypoplasia (LEH) and age, sex, or SES in the full sample. Within the samples constructed for each skeletal indicator, relationships were also found between SES and age and between sex and age, indicating that low-SES individuals and females were more common in the young and middle age groups, regardless of the skeletal indicator being examined.

Table 2.8 shows the significant results of the hierarchical log-linear analyses in which only the London sites were included (i.e., the reduced samples). Many of the associations observed in the full samples were also observed in this reduced samples, but several differences are notable. In the reduced sample for LEH, a significant four-way interaction between LEH presence, age, sex, and SES was detected. The Chi-square analysis for low-SES males revealed a higher frequency of LEH in the middle age at

Table 2.7 The significant associations among each skeletal indicator of stress, age, sex, and SES in the full sample (all four sites). Non-significant relationships not shown. PNB = periosteal new bone, LEH = linear enamel hypoplasia, CO = cribra orbitalia.

Skeletal Indicator Sample	Association	Chi-Square	Sig.	Relationship (from Chi Square)
Periosteal New Bone (n = 436)	PNB*Sex*Age	11.410	.022	PNB more common in middle-aged males (30.0-44.9 years)
	SES*Age	36.006	.000	Low SES more common in young and middle ages (18.0-44.9 years)
	Sex*Age	18.432	.001	Females more common in young and middle ages (25.0-29.9, 45.0-64.9 years)
Linear Enamel Hypoplasia (n = 293)	SES*Age	26.194	.000	Low SES more common in young and middle ages (18.0-44.9 years)
	Sex*Age	9.499	.050	Females more common in young and middle ages (18.0-29.9, 45.0-64.9 years)
Cribra Orbitalia (n = 457)	CO*SES*Sex	9.591	.002	CO less common among high-SES females
	CO*SES	7.282	.007	CO more common in low SES
	CO*Sex	3.853	.050	CO more common in males
	CO*Age	18.340	.001	CO more common in young ages (18.0-29.9 years)
	SES*Age	38.754	.000	Low SES more common in young and middle ages (18.0-44.9 years)
	Sex*Age	13.304	.010	Females more common in young ages (18.0-29.9 years)

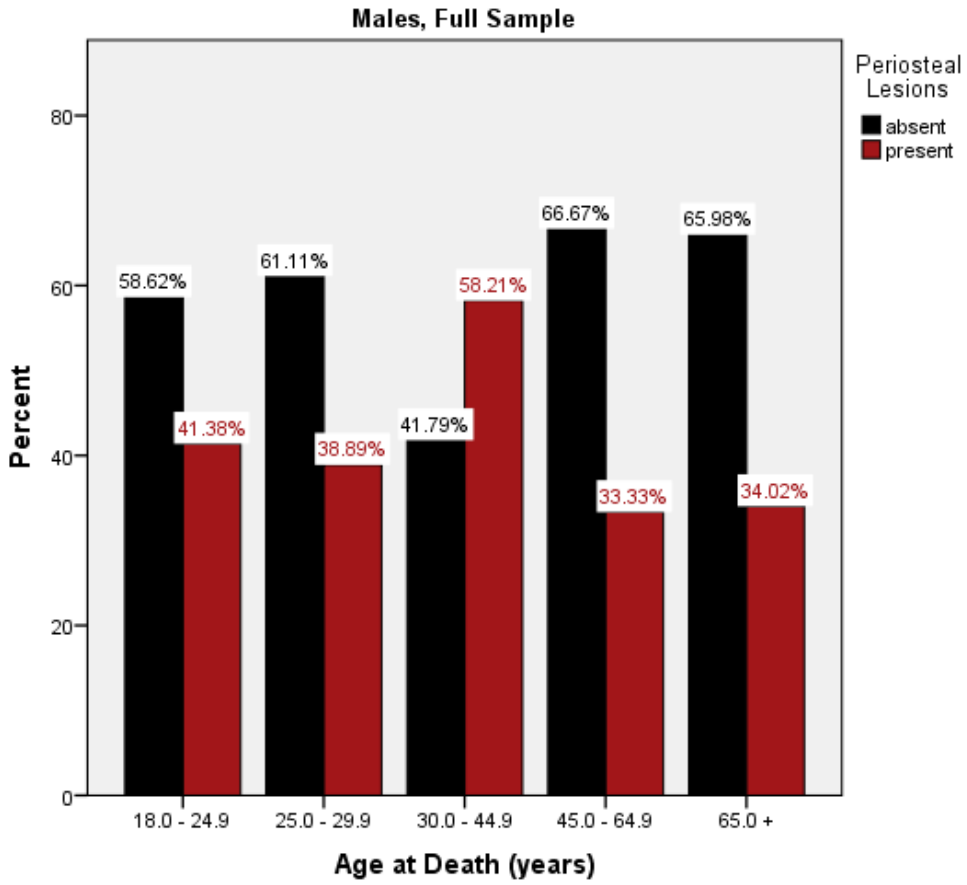

Figure 2.1 Frequencies of periosteal lesions by age in males from all four sites (full sample). Chi-square for periosteal lesions × age in males: χ^2 = 10.669, p = 0.031. Results were consistent in the reduced sample.

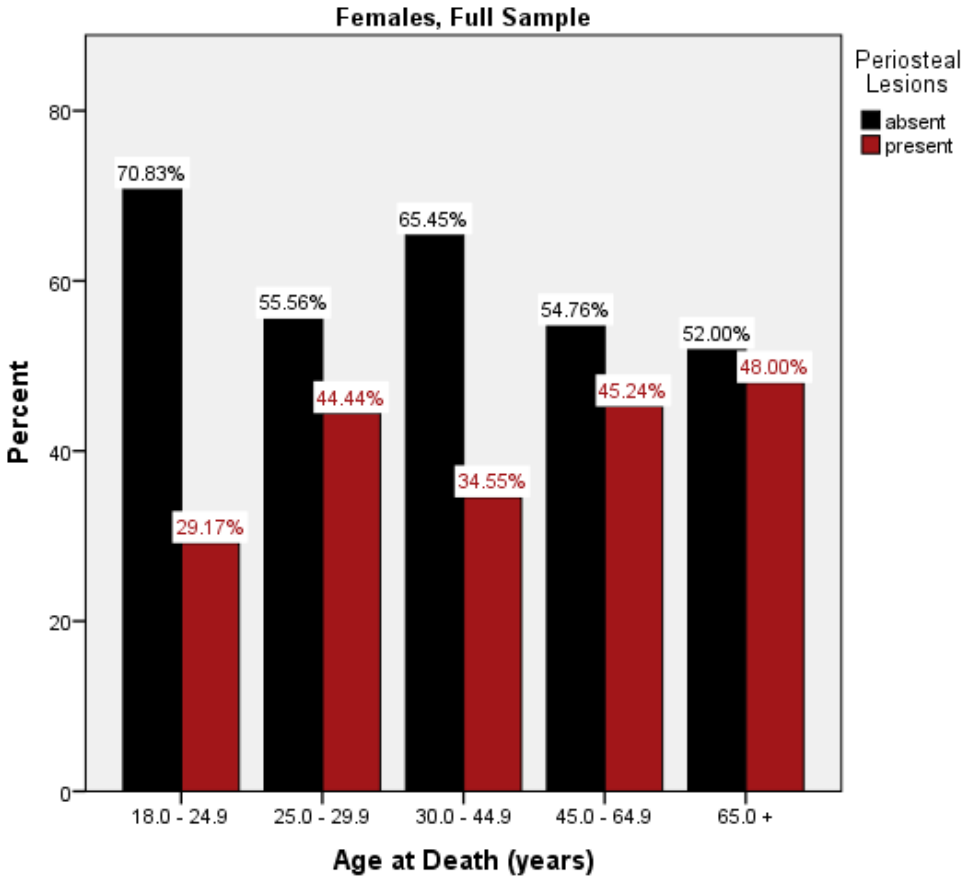

Figure 2.2 Frequencies of periosteal lesions by age in females from all four sites (full sample). Chi-square for periosteal lesions × age in females: χ^2 = 3.796, p = 0.434. Results were consistent in the reduced sample.

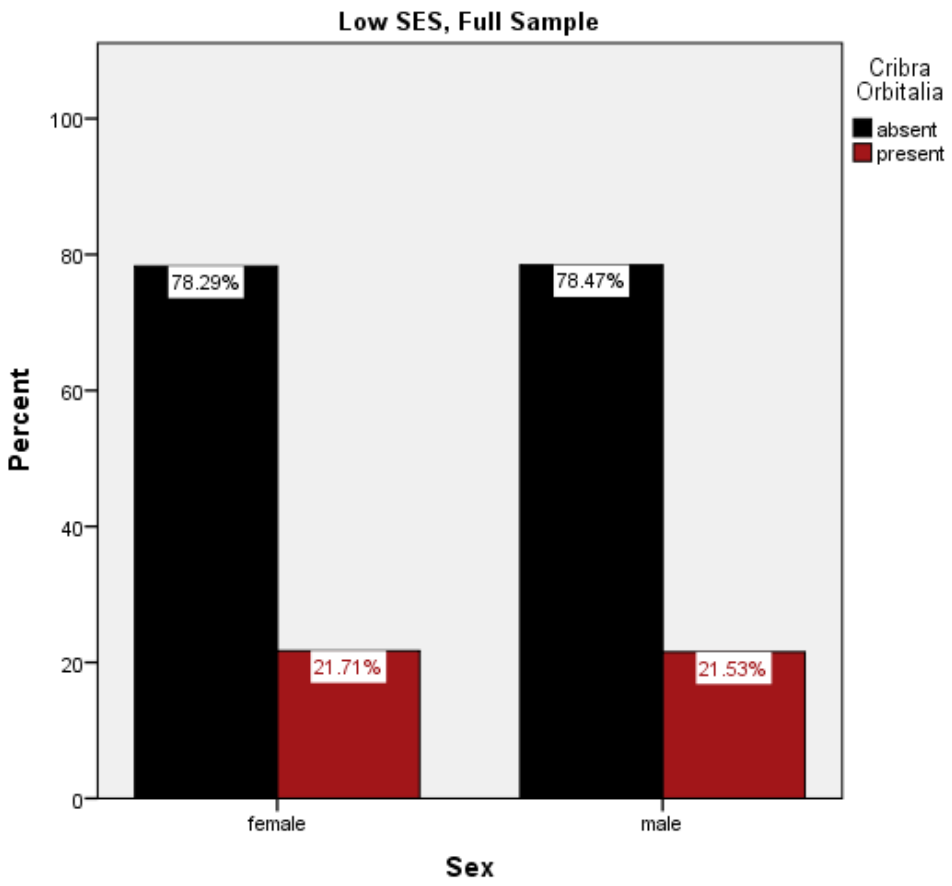

Figure 2.3 Frequencies of cribra orbitalia by sex in the low-SES group from all four sites (full sample). Chi-square for cribra orbitalia × sex in the low-SES group: $\chi^2 = 0.001$, $p = 0.972$. Results were consistent in the reduced sample.

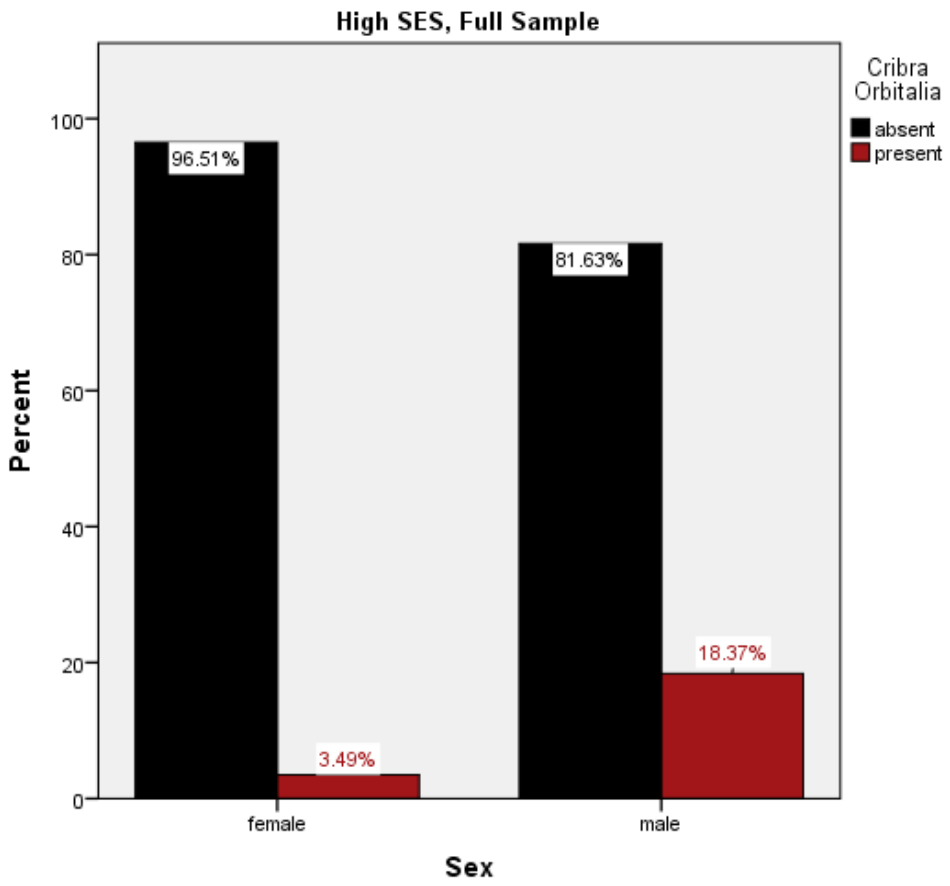

Figure 2.4 Frequencies of cribra orbitalia by sex in the high-SES group from all four sites (full sample). Chi-square for cribra orbitalia × sex in the high-SES group: $\chi^2 = 10.030$, $p = 0.002$. Results were consistent in the reduced sample.

Table 2.8 The significant associations among each skeletal indicator of stress, age, sex, and SES in the reduced sample (London sites). Non-significant relationships not shown. PNB = periosteal new bone, LEH = linear enamel hypoplasia, CO = cribra orbitalia.

Skeletal Indicator Sample	Association	Chi-Square	Sig.	Relationship (from Chi Square)
Periosteal New Bone (n = 291)	PNB*SES*Age	8.306	.081	PNB more common in middle and oldest ages (25.0-44.9, 65.0+ years) in low SES
	PNB*Sex*Age	11.158	.025	PNB more common in middle-aged males (30.0-44.9 years)
	PNB*SES	4.800	.028	PNB more common in low SES
	SES*Age	48.731	.000	Low SES more common in young and middle ages (18.0-44.9 years)
	Sex*Age	12.504	.014	Females common in young and middle ages (18.0-29.9, 45.0-64.9 years)
Linear Enamel Hypoplasia (n = 189)	LEH*SES*Sex*Age	7.997	.092	LEH more common in middle-aged low-SES males (25.0-64.9 years)
	SES*Age	41.135	.000	Low SES more common in young and middle ages (18.0-44.9 years)
Cribra Orbitalia (n = 298)	CO*SES*Sex	8.996	.003	CO least common among high-SES females
	CO*Sex	4.898	.027	CO more common in males
	CO*Age	11.177	.025	CO more common in young ages (18.0-29.9 years)
	SES*Age	53.842	.000	Low SES more common in young and middle ages (18-44.9 years)
	Sex*Age	12.026	.017	Females more common in young and middle ages (18.0-29.9, 45.0-64.9 years)

death categories (25.0 – 64.9 years), compared to the youngest and oldest age categories (**Figure 2.5**). Comparatively, Chi-square tests revealed that high-SES females, high-SES males, and low-SES females had relatively high frequencies of LEH regardless of age at death (**Figures 2.6 – 2.8**). Other associations differed between the full (all sites) and reduced (London sites only) samples, including a three-way association between periosteal lesions, SES, and age in the reduced sample that was not indicated in the full sample. In the high-SES group, periosteal lesion frequency was not associated with age (**Figure 2.9**). In contrast, the low-SES group exhibited a dramatic spike in periosteal lesion frequency in the middle-aged and oldest age categories (25.0 – 44.9 years and 65.0+ years) (**Figure 2.10**). Additionally, there was an association between periosteal lesions and SES in the reduced sample that was not indicated in the full sample. When limited to the London sites, periosteal lesions were more common in low-SES individuals. In the LEH sample, the sex and age association found in the full sample was not found in the reduced sample. Lastly, the significant association between cribra orbitalia presence and SES in the full sample was not found in the reduced sample.

2.4 Discussion

Intersectionality theory argues that the axes of multiple identities (i.e., such as those based on age, sex, or SES) would intersect to collectively influence health outcomes, rather than each identity exerting a distinct influence. The findings of this study do not support the hypothesis constructed based on intersectionality theory, given that none of the analyzed samples indicated two- or three-way associations among the skeletal indicators of physiological stress and the SES and sex categories of multiply-

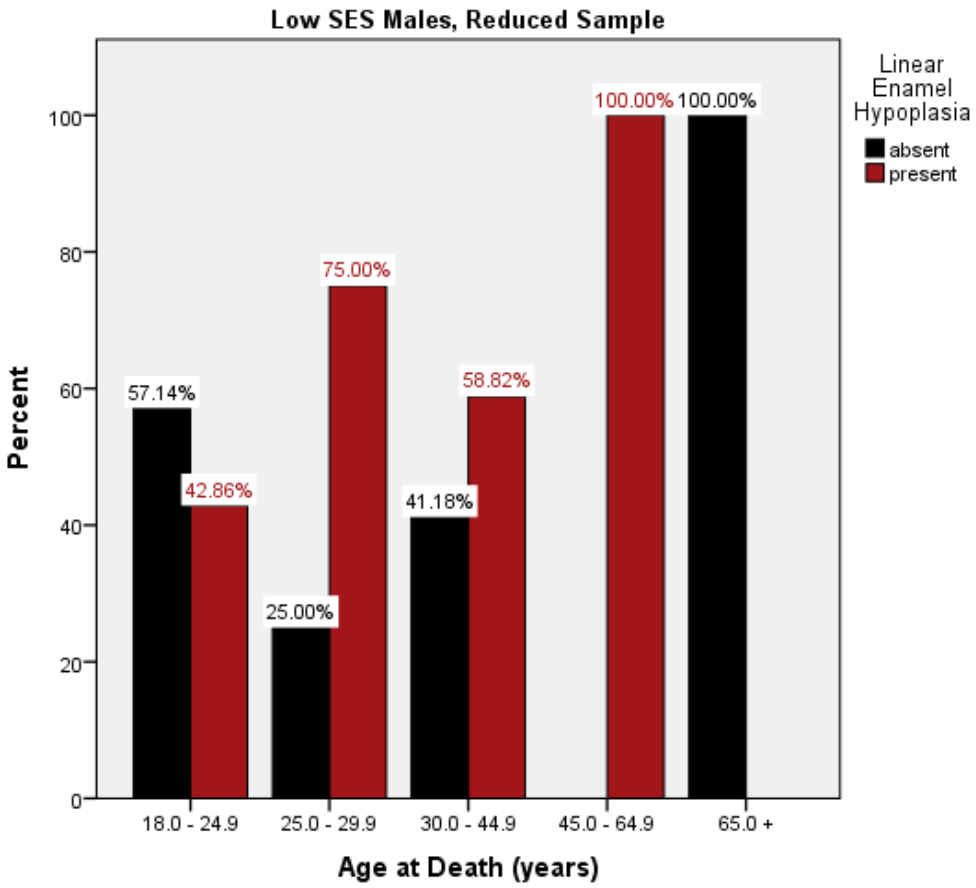

Figure 2.5 Frequencies of linear enamel hypoplasia by age in low-SES males from the London sites only (reduced sample). Chi-square for linear enamel hypoplasia × age in low-SES males: $\chi^2 = 6.306$, $p = 0.177$.

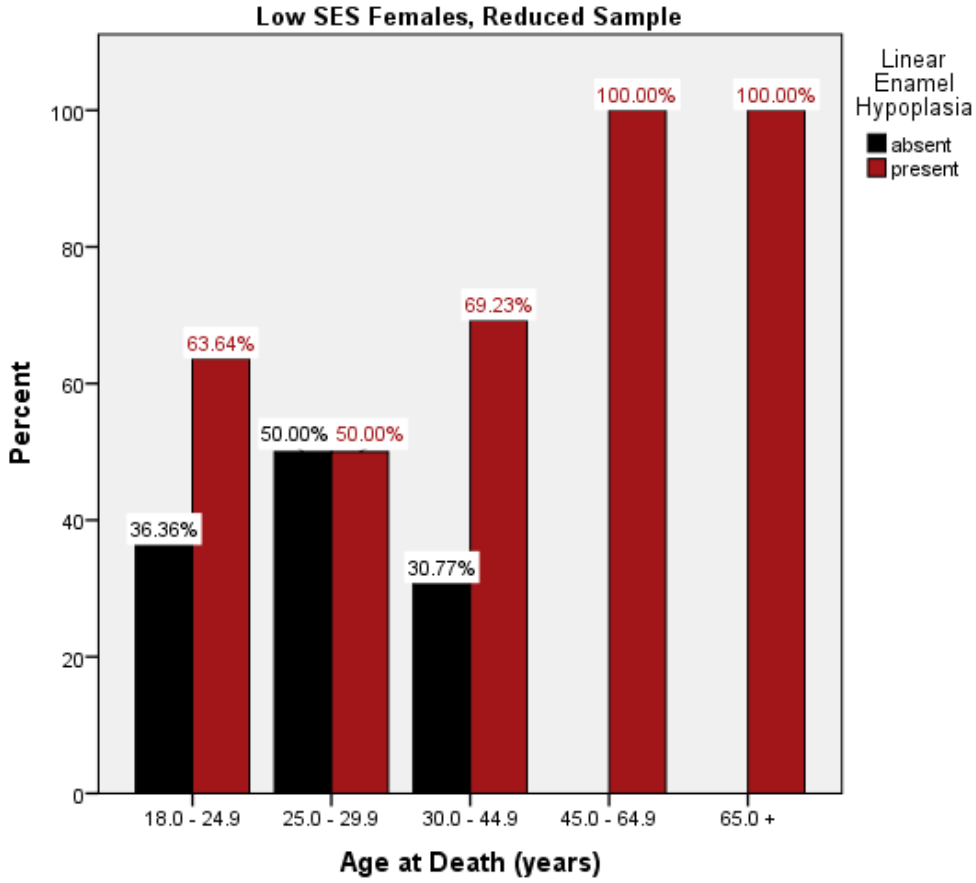

Figure 2.6 Frequencies of linear enamel hypoplasia by age in low-SES females from the London sites only (reduced sample). Chi-square for linear enamel hypoplasia × age in low-SES females: $\chi^2 = 1.971$, $p = 0.741$.

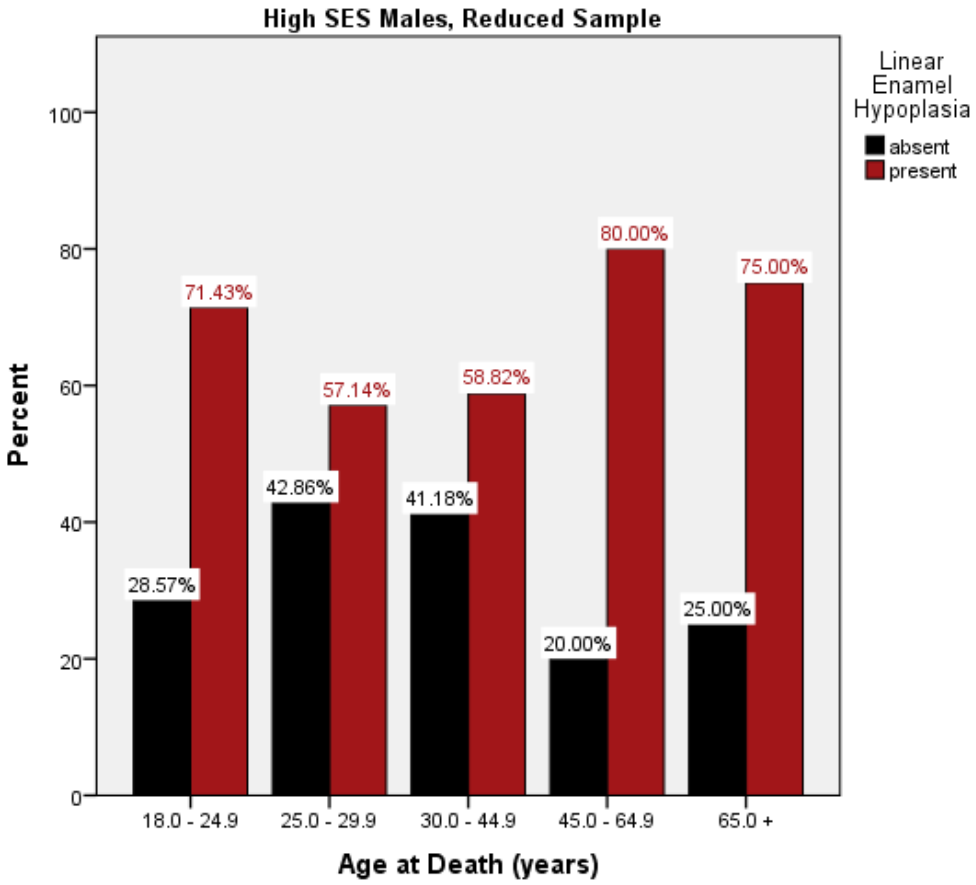

Figure 2.7 Frequencies of linear enamel hypoplasia by age in high-SES males from the London sites only (reduced sample). Chi-square for linear enamel hypoplasia × age in high-SES males: $\chi^2 = 2.280$, $p = 0.684$.

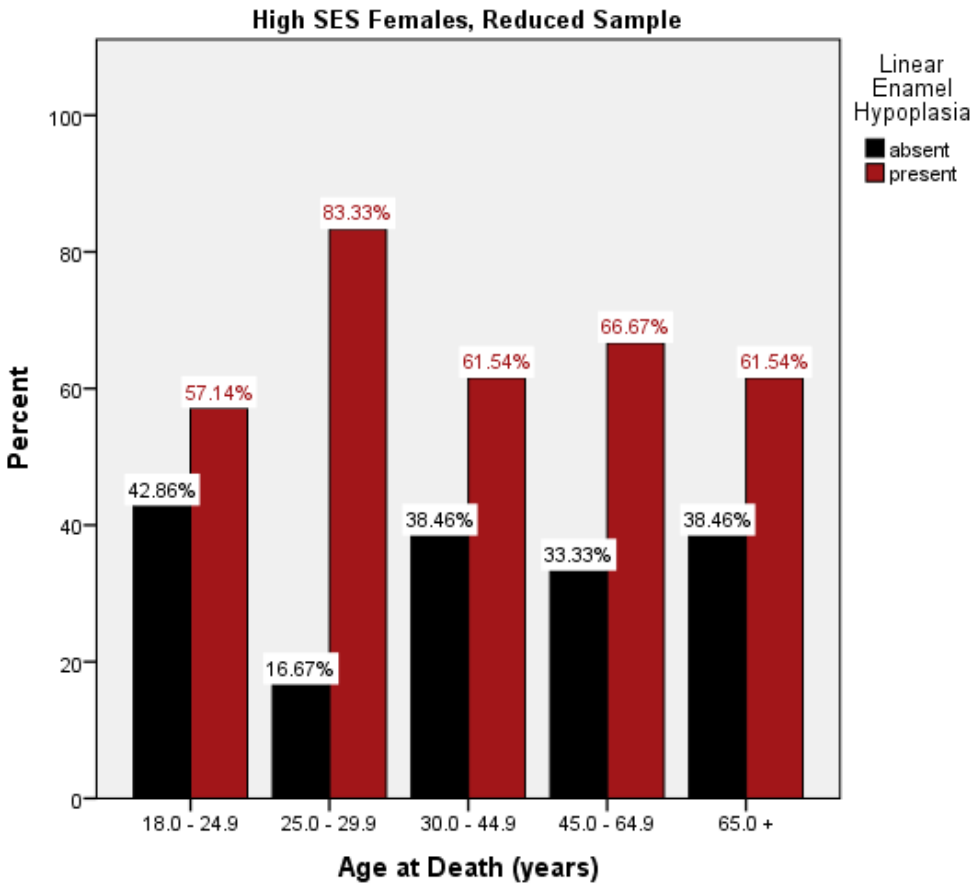

Figure 2.8 Frequencies of linear enamel hypoplasia by age in high-SES females from the London sites only (reduced sample). Chi-square for linear enamel hypoplasia × age in high-SES females: $\chi^2 = 1.228$, $p = 0.873$.

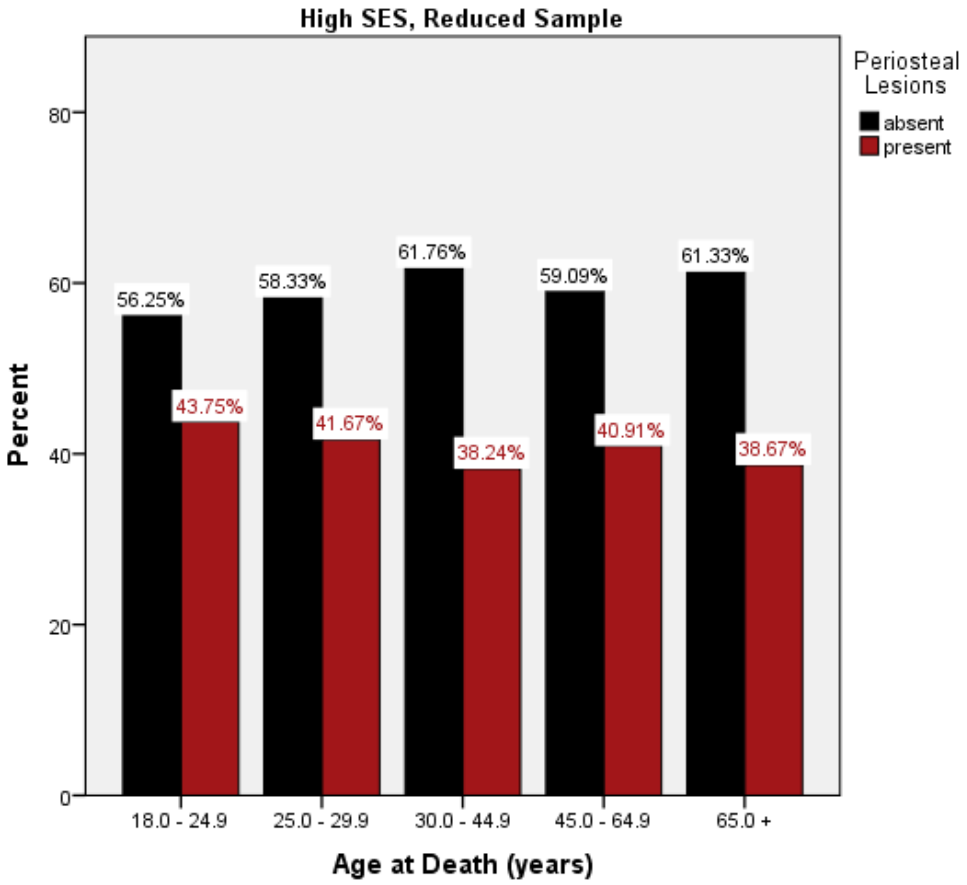

Figure 2.9 Frequencies of periosteal lesions by age in the high-SES group from the London sites only (reduced sample). Chi-square for periosteal lesions × age in the high-SES group: $\chi^2 = 0.219$, $p = 0.994$.

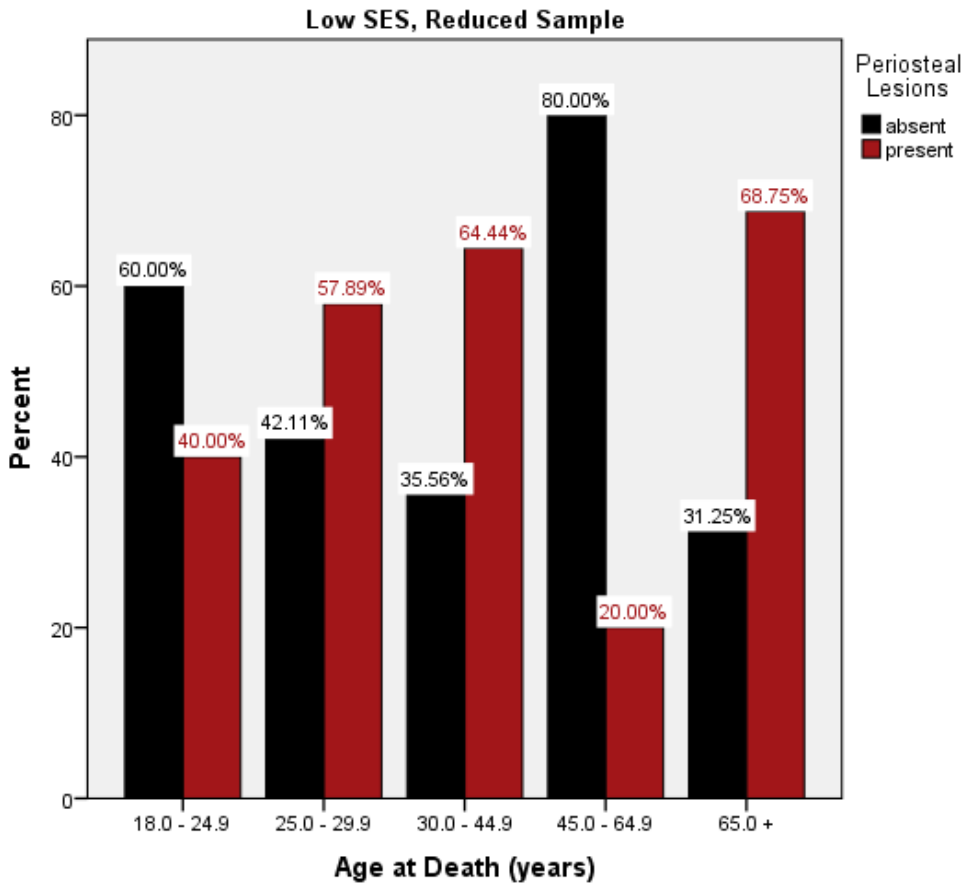

Figure 2.10 Frequencies of periosteal lesions by age in the low-SES group from the London sites only (reduced sample). Chi-square for periosteal lesions × age in the low-SES group: $\chi^2 = 9.685$, $p = 0.046$.

marginalized individuals (i.e., low-SES females). However, the results did support the hypothesis that earlier age at death would be associated with marginalized identities (i.e., low SES and female sex). Historical data are used to situate the findings in their cultural context to illustrate the interaction between individual-level health outcomes and the structural forces that were changing during the process of industrialization in England.

2.4.1 Periosteal New Bone Formation

A three-way association existed between periosteal lesions, sex, and age, and the association was consistent between the full (all sites) and reduced (London sites only) samples. Particularly, periosteal lesions were significantly more common in middle-aged males (30.0 – 44.9 years) than in younger or older males. In contrast, periosteal lesions were generally more common in older females than in the youngest females (although Chi-square tests indicate the association is not significant), which may indicate an accumulation of nonfatal physiological stressors over a relatively long life and therefore lower frailty relative to younger peers who died before developing lesions, as suggested elsewhere (DeWitte, 2014b; Grauer, 1993; Yaussy et al., 2016). The patterns of lesion frequency in males and females were similar between the full and reduced samples, suggesting that the three-way association between age, sex, and periosteal lesions was not due to the combination of low-SES samples from different regions of England.

Males exhibited a substantial increase in the frequency of periosteal lesion presence in the middle age category (30.0 – 44.9 years) followed by a decrease in lesion frequency in the older two age categories (45.0 – 64.9 years and 65.0+ years). This result suggests that the presence of periosteal lesions on the tibia—caused by infections or

trauma—increased the risk of death for middle-aged men. Therefore, the drastic late-age decrease in periosteal lesion frequency among males may indicate selective mortality, as discussed by Wood, Milner, Harpending, and Weiss (1992): males with periosteal lesions perished by 45 years of age, whereas males without lesions survived to die at older ages. The differing patterns of periosteal lesion frequency by sex may result from biological or cultural differences between males and females that influenced exposure to pathogens and physiological stressors that generate periosteal lesions, such as repeated minor trauma or infection (Weston, 2008). In general, male sex steroid hormones (androgens) lower several aspects of immunity, whereas female sex steroid hormones (estrogens) enhance immunity (Klein, 2000b, 2000a). As a result, males are biologically more susceptible to a variety of parasitic, fungal, bacterial, and viral infections compared to females (e.g., Ferrer et al., 1998; Sabra L. Klein et al., 2004; Webster et al., 1997; Zuk & McKean, 1996). Behavioral factors could also influence the frequency of periosteal lesions in males, given that males engage in "risk-taking" behaviors—such as aggression—more often than females (Klein, 2000b), which could impact frequencies of traumatic lesions between the sexes. Finally, cultural factors likely played a role in the middle-aged increase in periosteal lesion frequency among males in this study. Compared to females of the same age, young and middle-aged males in industrial Britain were more often employed in occupations like agriculture, metal manufacture, building, transport, and mining (Rule, 2014), which likely exposed them to an abundance of workplace hazards capable of causing trauma or infection. Moreover, an injured male in a hazardous setting would be at risk of further injury, infection, or death, possibly explaining the

pattern of males with periosteal lesions dying at middle-age and males without lesions surviving to older ages.

Two additional associations were found for the reduced (London sites only) sample that were not found in the full (all sites) sample: a three-way association among periosteal lesions, SES, and age at death, and a two-way association between periosteal lesions and SES. Regarding the two-way association, a Chi-square test indicated that periosteal lesions were more common in low-SES individuals compared to high-SES individuals, independent of age or sex. However, Chi-square tests of the three-way association among periosteal lesions, SES, and age at death indicated that only the low-SES group exhibited a relationship between periosteal lesions and age at death (i.e., periosteal lesions were more frequent in low-SES individuals aged 25.0 – 44.9 years and 65.0+ years), whereas periosteal lesions were largely unassociated with age in high-SES individuals. The results suggest that, regardless of sex, low-SES individuals in London may have been at greater risk of trauma and infection capable of producing periosteal lesions compared to high-SES individuals from London or low-SES individuals from other urban settings in England (i.e., the three-way association between periosteal lesions, SES, and age at death was not observed in the low-SES sample that included individuals from Wolverhampton and North Shields). Analyses of documentary data from the industrial period have suggested that working-class men and women in London and the surrounding areas were shorter and weighed less than urban laborers in northern England and Scotland (Meredith & Oxley, 2015; Nicholas & Steckel, 1991). In agreement with those studies, the findings of this study suggest that the source of the disparity may be the disamenities associated with London compared to other urban areas. Although the water,

sanitation, and living conditions were all considered inadequate in Wolverhampton (Adams, 2007) and North Shields (Tancock & Lee, 2014), it may be that the conditions in London were more severe.

2.4.2 Linear Enamel Hypoplasia

When all four sites were examined together, the presence of LEH was not significantly associated with age, sex, or SES. However, when the sample was reduced to only the London sites, a significant four-way association existed between LEH presence, SES, sex, and age. Chi-square tests revealed no significant relationships between age and high-SES males, low-SES males, high-SES females, or low-SES females, but the largest Chi-square value belonged to low-SES males, who more frequently exhibited high frequencies of LEH in the middle age categories and comparatively low frequencies in the youngest and oldest age categories. All other tested groups—high-SES males, high-SES females, and low-SES females—exhibited LEH more often than not for every age category. Although a four-way association between SES, sex, age, and early life health (evinced by LEH) would be of interest to intersectional researchers, it is more likely that this result is a consequence of the small sample sizes involved in the reduced (London sites only) hierarchical log-linear analyses.

2.4.3 Cribra Orbitalia

A three-way association was found among the presence of cribra orbitalia, SES, and sex in both the full (all sites) and reduced (London sites only) samples. High-SES females had significantly lower frequencies of cribra orbitalia compared to any other

group, which suggests that high-SES females experienced a buffering effect that substantially impacted the development of cribrotic lesions. Interestingly, this relationship existed in addition to the significant associations that were found between cribra orbitalia and sex in the full and reduced samples and between cribra orbitalia and SES in the full sample. That is, because hierarchical log-linear analysis determines the associations between and among variables *independent of other variables*, the three-way association among cribra orbitalia, high SES, and female sex is independent of the associations between cribra orbitalia and SES or sex. Thus, this result suggests—in accordance with intersectionality theory—that the unique domain or position produced by the intersection of high SES and female sex exerted an effect on cribra orbitalia presence beyond the effect of SES or sex separately.

As mentioned above, this study considers the presence of cribra orbitalia lesions to be reflective of physiological insults (e.g., dietary deficiency, diarrheal disease, parasitic infection) that occurred during childhood and that may have impacted frailty and risk of mortality for marginalized individuals in adulthood. Historical scholars have argued that dietary deficiencies and heavy disease loads caused substantial stunting among the urban poor in industrial England (Floud et al., 1990; Nicholas & Oxley, 1993; Nicholas & Steckel, 1991; Sharpe, 2012). In large working-class families, food was preferentially allocated to the adult male in the family who earned a larger portion of the household's income. As a result, working-class children in England were chronically malnourished and shorter than their peers from regions where children earned higher wages and thus had better nutrition (Meredith & Oxley, 2015; Sharpe, 2012). As these working-class children aged—presumably after developing cribrotic lesions—their

earlier life experiences may have impacted their overall frailty, causing cribra orbitalia to be associated with younger ages at death in the full (all sites) and reduced (London sites only) samples. In addition, cribra orbitalia was associated with male sex (in the full and reduced samples), and low SES (in the full sample only). The association between cribra orbitalia and sex may suggest that male non-adults were particularly nutritionally deprived or exposed to more pathogens and parasites capable of generating anemia, making them more likely to develop cribrotic lesions. Lastly, though nutritional deprivation and urban disamenities likely account for the association between cribra orbitalia and the low-SES group, it is worth noting that the association only existed in the full (all sites) sample. An inconsistency between the full and reduced samples indicates a discrepancy between the low-SES skeletal sample from London and the samples from Wolverhampton and North Shields. Particularly, individuals in the samples from farther north were more likely to have cribra lesions than their London counterparts. This result may have to do with more severe nutritional deficiencies or increased exposure to pathogens among individuals in the low-SES sites from Wolverhampton and North Shields, compared to individuals in the low-SES site from London.

2.4.4 Additional Associations

2.4.4.1 Socioeconomic Status and Age at Death

Within each of the lesion-specific samples, a two-way association was found between SES and age at death. The associations were consistent between the full (all sites) and reduced (London sites only) samples, indicating that the relationships were not the result of geographic differences in risk. For each sample, individuals of low SES were

more common in the young and middle age at death categories (18.0 – 44.9 years), whereas individuals of high SES were more common in the older age at death categories (45.0 – 65.0+ years). The results of this study closely match the historical data from cities throughout England, indicating that individuals in higher socioeconomic strata (e.g., professionals and members of the aristocracy) more frequently lived beyond age 45, whereas individuals in the lower strata (e.g., laborers and servants) often died at younger ages (Bailey & Day, 1863; Morris, 1964; Titmuss, 1943).

For decades, studies have demonstrated relationships between SES and life expectancy, SES and survival (e.g., following cancer diagnosis), and SES and mortality in a variety of contexts (e.g., Adelstein, 1980; Antonovsky, 1967; Carnon et al., 1994; Cavigelli & Chaudhry, 2012; Kogevinas & Porta, 1997; Marmot, Shipley, & Rose, 1984). Research on living populations has repeatedly demonstrated that SES is correlated with a variety of environmental hazards and pollutants. Modern individuals of low SES are more likely to live near toxic waste dumps (Bullard & Wright, 1993), lack access to clean drinking water (Calderon et al., 1993), and live in substandard housing (G. W. Evans, 2004) compared to their higher-SES peers. Historical evidence suggests that low-SES individuals in industrializing England experienced similar threats to their health, including inadequate or contaminated food, degraded and congested living conditions, pollution, and infectious disease (G. Clark, Huberman, & Lindert, 1995; C. H. Feinstein, 1998; Komlos, 1998; Nicholas & Steckel, 1991; Stevenson, 1993). In sum, low-SES individuals in industrializing England likely faced overcrowding, undernutrition, and disease, putting them at greater risk of earlier mortality compared to their high-SES peers

who could afford access to better food and healthcare, and lived in relatively less crowded, less polluted, and less hazardous conditions.

2.4.4.2 Sex and Age at Death

A two-way association was also found between sex and age at death in nearly all of the analyzed samples, with the exception of the reduced LEH sample (i.e., individuals from the two London sites who possessed one or both mandibular canines). The high degree of consistency between the full (all sites) and reduced (London sites only) samples suggests that the association is not an artifact of geographic differences. Females were generally more common in the younger and middle-aged age at death categories (i.e., 18.0 – 29.9 and 45.0 – 64.9 years), whereas males were typically more common in the oldest age at death category (i.e., 65.0+ years).

The results of this study suggest that, independent of SES, women died at younger ages than men, who more frequently lived to older ages. These findings are consistent with the available historical evidence, which describes dramatic changes in gender dynamics during the industrial period. Multiple scholars have suggested the decline in health of women may be related to the declining availability of employment for women, and thus the declining importance of the woman as a wage-earner in the household. Limited employment in agriculture, manufacturing, and formal apprenticeship led to increased gender inequality within the home and forced women to accept a smaller portion of the household's resources in order to ensure their continued survival (Bythell, 1993; Nicholas & Oxley, 1993). As mentioned above, working class women were often malnourished, because nutrient-rich foods like meat, vegetables, and beer were given to

the husband, who was the primary source of the family's income (Meredith & Oxley, 2015; Nicholas & Oxley, 1993; Oren, 1973). Malnutrition is known to increase susceptibility to infection by compromising the innate and adaptive immune responses, which, in turn, increases risk of morbidity and mortality (Jackson & Calder, 2004; Scrimshaw, Taylor, & Gordon, 1968; Wintergerst, Maggini, & Hornig, 2007). As employment opportunities dwindled and food allocation shifted towards the adult males in the household, females likely became malnourished and thus more vulnerable to mortality resulting from infection.

Women in industrializing England were also at risk of maternal mortality, or death during childbirth. Earlier ages at marriage and decreased birth intervals from 1750 to 1850 led to a population boom that fueled the industrial labor force (Boberg-Fazlic et al., 2011; Stevenson, 1993), but may have also increased the likelihood of maternal mortality. For the city of London, the rates of maternal mortality in 1760 and 1781 were estimated to be 16.7 and 15 maternal deaths per 1,000 births, respectively (Buer, 2013). Interestingly, evidence suggests that women of higher SES were at greater risk of maternal mortality in the early 19th century, which is likely related to the training of the person performing the delivery. At the time, the deliveries of upper-class women were attended by physicians, who were more likely to unnecessarily interfere in the birthing process, inviting infection or hemorrhage. In contrast, the deliveries of lower-class women were primarily attended by midwives, who were much less likely to interfere in childbirth (Loudon, 1986b, 2000). Therefore, the combined effects of earlier marriages, decreased birth intervals, and dangerous birthing conditions may have influenced the association between sex and age at death found by this study, increasing the number of

adult females dying in the childbearing years (i.e., the younger and middle-aged age at death categories) relative to males, who died at older ages.

2.5 Conclusion

The goal of this study was to integrate intersectionality theory into a bioarchaeological study of health and mortality in industrializing England. The results confirmed that skeletal indicators of frailty were associated with social and biological factors separately and in conjunction with each other. Particularly, a three-way association existed among periosteal lesions, sex, and age, indicating that middle-aged males were more likely to exhibit periosteal lesions compared to individuals in other age and sex categories. Likewise, a three-way association existed among cribra orbitalia, SES, and sex, indicating that high-SES females were less likely to exhibit cribra orbitalia compared to high-SES males, low-SES females, and low-SES males. In each case, individual-level marginalizations and negative health outcomes were connected to larger structural inequalities developing at the time: the classist inequality of living conditions in urban areas and the newly-amplified devaluation of women in the economic and sociocultural spheres.

CHAPTER 3

TOOTH SIZE AND VERTEBRAL NEURAL CANAL SIZE AS BIOARCHAEOLOGICAL EVIDENCE OF THE DEVELOPMENTAL ORIGINS OF HEALTH AND DISEASE HYPOTHESIS[2]

[2] Yaussy, SL. To be submitted to the *American Journal of Physical Anthropology*.

3.1 Introduction

The Developmental Origins of Health and Disease (DOHaD) hypothesis posits that developmental disruptions in the prenatal period, infancy, and childhood can negatively influence later life health outcomes. Epidemiological investigations of the connection between early life stress and later life disease were pioneered by Barker and colleagues, who suggested that poor nutrition during pregnancy could alter the development of the fetus, leading to low birthweight and increased risk of cardiovascular disease in adulthood (Barker & Osmond, 1986; Barker et al., 1989). In the decades since, scholars have refined Barker's hypothesis to include epigenetic mechanisms (i.e., DNA methylation, post-translational histone modification, and non-coding RNA) that connect early life stress events to adult morbidity (Rosenfeld, 2015; Wadhwa et al., 2009; Waterland & Michels, 2007).

Although bioarchaeology cannot directly observe many of the early life stressors, physiological responses to stress, or adult disease outcomes on which the DOHaD hypothesis is predicated, alterations in skeletal or dental development provide an enduring record of adaptive reaction norms that ensured short-term survival but exacted long-term costs in terms of optimal development (i.e., the concepts of adaptive plasticity and physiological constraint). That is, certain skeletal indicators of stress can provide evidence of instances when resources were diverted to ensure the short-term survival of an individual during a stress event (e.g., boosted immune activity in response to infection), and, as a result, depleted the resources available for competing processes (e.g., growth and maintenance) (Temple, 2018). Thus, bioarchaeologists have contributed to investigations of the DOHaD hypothesis, utilizing a variety of skeletal indicators of stress

to assess the effects of developmental disturbance in the past on later life frailty, morbidity, and mortality (Agarwal, 2016; Gowland, 2015; Temple, 2018). Armelagos and colleagues (2009) introduced the potential for linear enamel hypoplasia and other enamel defects to be used as skeletal evidence in support of the DOHaD hypothesis, citing several prior bioarchaeological studies linking dental defects to earlier mortality (Cook & Buikstra, 1979; Duray, 1996; Alan H. Goodman & Armelagos, 1988; Jerome C. Rose, Armelagos, & Lallo, 1978; White, 1978). Temple (2014) elaborated on the connection between enamel defects and risk of death, connecting age-at-first-defect formation with age at death and numbers of linear enamel hypoplasia among Late/Final Jomon period foragers from Japan. Particularly, individuals who formed defects in infancy and early childhood were at greater risk of forming additional defects during later developmental stages and dying at younger ages, suggesting that individuals' investment in survival during early life weakened their abilities to respond to future stress events and exacerbated their mortality schedules (Temple, 2014). Weisensee (2013) demonstrated a connection between craniofacial fluctuating asymmetry—a measure of developmental instability—and death from degenerative diseases in individuals from an identified skeletal collection. Stojanowski and colleagues (2007) found that non-adults exhibited smaller tooth crown sizes compared to adults, indicating that early life stressors capable of preventing complete crown development could also increase risk of early death (i.e., individuals with small teeth did not survive to reach adulthood). Steckel (2005) found that risk of death between ages 15 and 30 increased by 4.6% for individuals with short femora. Watts (2015) found that growth disruptions in late childhood and early

adolescence evinced by vertebral neural canal sizes were associated with increased risk of mortality in adulthood among individuals from later medieval and post-medieval London.

However, some bioarchaeological studies have challenged relatively simplistic views of the relationship between developmental disruption and premature mortality. A study of 19th- and early 20th-century individuals from Lisbon found that the association between linear enamel hypoplasia and earlier age at death became nonsignificant when adult socioeconomic status (SES) was considered. The authors suggest that adversity accumulated for individuals of low SES, compounding to produce increased morbidity and premature mortality (Amoroso et al., 2014). Similarly, Hughes-Morey (2016) found a significant negative association between short tibia length and risk of mortality in high-SES females, but not in high-SES males, low-SES males, or low-SES females. Hughes-Morey argues that her results are explained by SES-influenced differences in subadult mortality patterns, in which low-status females individuals (i.e., the multiply-marginalized group in her study, though she does not use that phrasing) experienced the greatest amount of stress in early life, making them relatively more frail and less likely to survive to adulthood when compared to other population subgroups. In contrast, high-status males who experienced childhood insults that affected adult stature were still able to survive to adulthood because they had preferential access to resources and were less likely to be exposed to severe and repeated disease (Hughes-Morey, 2016). In sum, bioarchaeological research has indicated that cultural factors may significantly affect the relationship between early life stress and later health outcomes, amplifying the effects of adversity in some populations while minimizing the impact of stress in others. In this study, tooth size and vertebral neural canal dimensions are used to examine the complex

associations among adult age at death, sex, and status in the context of industrial-era England.

3.2 Materials and Methods

3.2.1 Skeletal Samples

The skeletal samples used in this study (see **Tables 3.1** and **3.2**) are dated to the 18th and 19th centuries, when England was undergoing industrialization. Three of the four skeletal collections—Coach Lane, St. Peter's Wolverhampton, and New Bunhill Fields—are comprised of individuals of lower socioeconomic status (SES). The Coach Lane collection (A.D. 1711 – 1857) comes from a burial ground associated with the Society of Friends Meeting House in North Shields, England (Proctor et al., 2014). Though the SES of the area's inhabitants is not known for certain, the Meeting House was a Quaker establishment, which suggests that the individuals in the collection were devoted to simple lifestyles devoid of modern corruption (Dandelion, 2008). Consequently, this study and others (Roberts et al., 2016) conservatively consider the individuals in the Coach Lane collection to be of relatively low SES (i.e., the working- or lower middle-class). The St. Peter's Wolverhampton collection (A.D. 1819 – 1853) comes from the overflow cemetery associated with the St. Peter's Collegiate Church in Wolverhampton, England. The earth-cut graves from the cemetery suggest that the St. Peter's Wolverhampton collection is composed of working-class individuals, compared to wealthier parishioners who could afford family vaults or burial on the church grounds (Adams, 2007). Lastly, the New Bunhill Fields burial ground (A.D. 1821 – 1853) was an affordable funerary option for even London's poorest inhabitants (Miles & Connell,

Table 3.1 The sample sizes for each tooth measurement, separated by site. The sample size for all individuals (10+ years) is provided first, followed by the sample size of only adults (18+ years) for whom sex could be estimated, in parentheses.
MD = maximum mesiodistal length, BL = maximum buccolingual length.

Tooth (Measurement)	Site				Total
	St. Bride's Fleet Street	Coach Lane	St. Peter's Wolverhampton	New Bunhill Fields	
right maxillary premolar (MD)	42 (41)	48 (45)	21 (17)	52 (49)	163 (152)
right maxillary premolar (BL)	42 (41)	53 (50)	21 (17)	54 (51)	170 (159)
left maxillary premolar (MD)	50 (48)	54 (49)	21 (17)	60 (55)	185 (169)
left maxillary premolar (BL)	50 (48)	58 (53)	20 (16)	60 (55)	188 (172)
right maxillary molar (MD)	52 (50)	45 (40)	19 (14)	57 (49)	173 (153)
right maxillary molar (BL)	51 (49)	46 (41)	20 (15)	55 (47)	172 (152)
left maxillary molar (MD)	48 (45)	50 (41)	22 (17)	57 (49)	177 (152)
left maxillary molar (BL)	49 (46)	49 (40)	22 (17)	57 (48)	177 (151)
right mandibular premolar (MD)	80 (77)	66 (57)	30 (27)	67 (60)	243 (221)
right mandibular premolar (BL)	84 (81)	61 (52)	30 (27)	69 (62)	244 (222)
left mandibular premolar (MD)	67 (66)	70 (61)	28 (25)	68 (61)	233 (213)
left mandibular premolar (BL)	68 (67)	63 (55)	28 (25)	68 (61)	227 (208)
right mandibular molar (MD)	67 (64)	55 (44)	23 (19)	56 (47)	201 (174)
right mandibular molar (BL)	66 (63)	52 (42)	23 (19)	57 (48)	198 (172)
left mandibular molar (MD)	58 (56)	48 (40)	22 (18)	58 (49)	186 (163)
left mandibular molar (BL)	62 (60)	49 (41)	25 (21)	56 (47)	192 (169)

Table 3.2 The sample sizes for each vertebral neural canal measurement, separated by site. The sample size for all individuals (10+ years for AP diameter, 15+ years for TR diameter) is provided first, followed by the sample size of only adults (18+ years) for whom sex could be estimated, in parentheses. MD = maximum mesiodistal length, BL = maximum buccolingual length.

Vertebra (Measurement)	Site				Total
	St. Bride's Fleet Street	Coach Lane	St. Peter's Wolverhampton	New Bunhill Fields	
L1 (TR)	163 (161)	66 (57)	29 (26)	96 (89)	354 (333)
L1 (AP)	157 (155)	61 (52)	27 (24)	96 (89)	341 (320)
L2 (TR)	167 (165)	74 (65)	31 (28)	93 (86)	365 (344)
L2 (AP)	159 (157)	66 (67)	28 (25)	93 (86)	346 (335)
L3 (TR)	165 (162)	74 (62)	29 (27)	97 (90)	365 (341)
L3 (AP)	155 (152)	65 (54)	27 (25)	95 (88)	342 (319)
L4 (TR)	160 (158)	67 (58)	30 (25)	94 (87)	351 (328)
L4 (AP)	144 (142)	59 (50)	28 (23)	92 (86)	323 (301)
L5 (TR)	147 (144)	49 (43)	23 (19)	90 (83)	309 (289)
L5 (AP)	138 (135)	40 (35)	21 (17)	82 (76)	281 (263)

2012). As a result, New Bunhill Fields is believed to be composed of working-class individuals, comparable to the Coach Lane and St. Peter's Wolverhampton collections. To ensure adequate sample sizes for multi-way statistical analyses, these three skeletal samples were combined to form the low-SES group analyzed in this study.

The high-SES group analyzed in this study comes from the St. Bride's Fleet Street collection (A.D. 1740 – 1852), which is composed of skeletonized individuals from the crypts of St. Bride's Church in London, England. In addition to the relative expense of crypt burials compared to earth-cut or brick-lined burials, the parish register, associated coffin plates, and historical accounts all suggest that the crypt interments were the final resting place of relatively wealthy individuals, such as merchants (G Milne, 1997). All of the analyses in this study (described below) compare the higher-status burials from St. Bride's Fleet Street with the lower-status burials from Coach Lane, St. Peter's Wolverhampton, and New Bunhill Fields.

3.2.2 Age and Sex Estimation

Non-adult age at death was estimated based on dental development, diaphyseal length, and epiphyseal fusion (Buikstra & Ubelaker, 1994b). When both dental and skeletal estimates are available, dental age is used, as this has the strongest correlation with chronological age (A. B. Lewis & Garn, 1960). Given that this study focuses on the effects of developmental disturbances on vertebral neural canal and tooth crown sizes via analysis of the lumbar vertebrae and second premolars and molars, respectively, only those non-adult individuals old enough to have completely formed tooth crowns and lumbar neural canals were included in the analyses, as described below.

Age at death was estimated for all adult individuals (18+ years of age) using transition analysis (Jepser L. Boldsen et al., 2002). This method uses the ADBOU (Anthropological Database, Odense University) age estimation software to avoid the primary bias associated with traditional methods—imposing the age distribution of a known-age reference sample on the sample of interest. Further, transition analysis provides point estimates of age and probability distributions for age estimates, rather than broad age intervals, as most age estimation methods do. The point estimates of age provided by transition analysis are particularly helpful in studies investigating age-based patterns of mortality, as it identifies variation in even the oldest individuals, rather than consolidating advanced-age adults in a single, broad age category. Consequently, this study examines late adult age patterns in mortality that are typically unavailable. The age at death categories included in this study are: 10.0-10.9, 11.0-11.9, 12.0-12.9, 13.0-13.9, 14.0-14.9, 15.0-15.9, 16.0-16.9, 17.0-17.9, 18.0-24.9, 25.0-29.9, 30.0-34.9, 35.0-39.9, 40.0-44.9, 45.0-49.9, 50.0-54.9, 55.0-59.9, 60.0-64.9, 65.0-69.9, 70.0-74.9, 75.0-79.9, and 80+ years.

Sex was estimated for all adult individuals (18+ years of age) using sexually dimorphic features of the pelvis and cranium, following Buikstra and Ubelaker (1994b).

3.2.3 Tooth Size

The maximum mesiodistal and buccolingual crown dimensions were collected for the maxillary and mandibular second premolars and second molars, following the methods outlined by Moorrees and colleagues (1957) and Hillson (1996). Measurements were made with a Neiko digital caliber and recorded to the nearest 0.1 mm. The second

premolars and molars were chosen for measurement and analysis based on the field theory of dental development, which suggests that polar teeth (i.e., the first tooth in each tooth class, such as the first premolar or first molar) are under the greatest genetic control and are therefore limited in their responses (e.g., hypoplasia) to environmental perturbations during development (Butler, 1939; Dahlberg, 1945). In contrast, research may suggest that the nonpolar teeth (i.e., the more distal members of each tooth class, such as the second premolar or second molar) can reduce in size in response to developmental stress (Garn et al., 1979; Guagliardo, 1982; Larsen, 1983; Christopher M. Stojanowski et al., 2007).

Tooth formation literature has indicated that permanent non-polar premolars and molars form during childhood, with the crown of the second premolar forming between ages 2-7 years and the crown of the second molar forming between ages 3-8 years (W. H. Logan & Kronfeld, 1933; C. F. Moorrees et al., 1963). Therefore, to ensure that only individuals with fully developed tooth crowns were being compared, only individuals over 10 years of age were included in the analyses (see **Table 3.1**). Visibly worn teeth, broken teeth, and teeth with large pathologies (e.g., caries) were not measured and were not included in the study. However, to further ensure that attrition was not affecting tooth size, tooth measurements were tested for correlations between estimated age at death and tooth size. In this test, a negative Pearson correlation coefficient would be expected if attrition was a causative agent (i.e., crown size decreases with increasing age). None of the tooth measurements were found to be negatively correlated with estimated age at death.

To examine the associations among tooth size, age at death, and SES, a 2-way analysis of variance (ANOVA) was conducted for each tooth measurement, including all individuals. Likewise, to examine the associations among tooth size, age at death, SES, and sex, a 3-way ANOVA was conducted for each tooth measurement, including all adults (18+ years) for whom sex could be estimated. Prior to the 2- and 3-way ANOVAs for each tooth measurement, boxplots, histograms, and Q-Q plots were used to identify extreme outliers in the data set. As a result, 22 individual teeth measurements were removed from the analyses, and **Table 3.1** reflects the final sample sizes used in the 2- and 3-way ANOVAs. Kolmogorov-Smirnov tests were used to ensure that none of the teeth measurements significantly deviated from a normal distribution, and Levene's test was used to determine the homogeneity of variances for each tooth measurement. For the 2-way ANOVA of tooth size, Levene's test indicated unequal variances for six of the sixteen tooth measurements. Natural log and reciprocal (inverse) transformations of the data did not improve the homogeneity of the variances, so those six measurements were removed from the 2-way analyses. For the 3-way ANOVA of tooth size, Levene's test indicated unequal variances for eight of the sixteen tooth measurements. Natural log and reciprocal (inverse) transformations of the data did not improve the homogeneity of the variances, so those eight measurements were removed from the 3-way analyses. All 16 tooth measurements are listed in the results tables, and the tables specify which measurements were removed due to unequal variances

3.2.4 Vertebral Neural Canal Size

The maximum anteroposterior (AP) and transverse (TR) neural canal diameters were collected for all available lumbar vertebrae (L1–L5) for all individuals, following the methods outlined by Watts (2011). Measurements were made with a Neiko digital caliber and recorded to the nearest 0.1 mm. Each diameter of each vertebra was measured twice, and the average length was recorded to minimize measurement error. Although the lumbar neural canals complete the majority of their growth in utero, there is some continued expansion of the vertebral canal throughout infancy and childhood (Hinck, Clark, & Hopkins, 1966). A recent study by Watts (2013b) indicated that the anteroposterior (AP) diameter reaches adult size around ages 3-5 years and the transverse (TR) diameter reaches adult size around age 15(R. Watts, 2013)(R. Watts, 2013). Therefore, to ensure only individuals with fully developed neural canals were being compared, only individuals over 10 years of age were included in analyses of the AP diameter and only individuals over 15 years of age were included in analyses of the TR diameter (see **Table 3.2**). Broken vertebrae and vertebrae with obvious pathologies or trauma were not measured and were not included in the study. However, to further ensure that age-related pathology was not affecting VNC size, each measurement was tested for correlations between estimated age at death and VNC size. In this test, a negative Pearson correlation coefficient would be expected if age-related pathology was a causative agent (i.e., VNC size decreases with increasing age). None of the VNC measurements were found to be negatively correlated with estimated age at death.

To examine the associations among vertebral neural canal (VNC) size, age at death, and SES, a 2-way analysis of variance (ANOVA) was conducted for each VNC

measurement, including all individuals. Likewise, to examine the associations among VNC size, age at death, SES, and sex, a 3-way ANOVA was conducted for each VNC measurement, including all adults (18+ years) for whom sex could be estimated. Prior to the 2- and 3-way ANOVAs for each VNC measurement, boxplots, histograms, and Q-Q plots were used to identify extreme outliers in the data set. As a result, 20 individual VNC measurements were removed from the analyses, and **Table 3.2** reflects the final sample sizes used in the 2- and 3-way ANOVAs. Kolmogorov-Smirnov tests were used to ensure that none of the VNC measurements significantly deviated from a normal distribution, and Levene's test was used to determine the homogeneity of variances for each VNC measurement. Initial tests revealed that large portions of the data failed the Normality and equal variances tests. Natural log and reciprocal transformations were applied to address issues of non-Normality and unequal variances, and the reciprocal transformation most improved the Normality and homogeneity of variances. However, following transformation, one of the ten measurements (the anteroposterior diameter of L1) continued to fail Normality tests and Levene's test for unequal variances, so that measurement was removed from the 2-way analyses. In addition, four of the ten measurements, all in the anteroposterior dimension, were consistently non-Normal or exhibited unequal variances and were removed from the 3-way analyses. All 10 VNC measurements are listed in the results tables, and the tables specify which measurements were removed due to unequal variances.

3.3 Results

Table 3.3 shows the statistically significant results for the 2-way ANOVAs examining the effects of age and SES on tooth size for all individuals over the age of 10 years. Of the ten teeth measurements that were analyzed, five measurements exhibited significant associations between tooth size and SES, or tooth size and SES*Age. An examination of the estimated mean tooth size for each group (high SES vs. low SES, male vs. female, etc.) indicated that, in all teeth exhibiting a significant relationship between tooth size and SES, small teeth were associated with the low-SES group. Similarly, in all teeth exhibiting a significant relationship between tooth size and SES*Age, small teeth were associated with the younger individuals in the low-SES group (i.e., most age categories under 50 years old). However, the confidence intervals for the estimated means overlapped across age and SES categories.

Table 3.4 shows the statistically significant results for the 3-way ANOVAs examining the effects of age, SES, and sex on tooth size for adults (≥18 years of age) in the sample. Of the eight teeth measurements that were analyzed, six measurements exhibited significant associations between tooth size and SES, sex, SES*sex, or SES*age. An examination of the estimated mean tooth size for each group indicated that, in the four cases exhibiting a significant relationship between tooth size and sex, small teeth were associated with females. In the single case exhibiting a significant relationship between tooth size and SES, small teeth were associated with low SES. In the two cases exhibiting a significant relationship between tooth size and SES*sex, the largest tooth sizes were associated with the high-SES males, whereas the group with the smallest teeth differed (low-SES males or the high-SES females). In the two cases exhibiting a significant

Table 3.3 All significant effects and interactions for each tooth measurement and the relationship between tooth size and SES, age, or age*SES in a 2-way ANOVA. Dashes indicate teeth that were removed from further analyses due to unequal variances. MD = maximum mesiodistal length, BL = maximum buccolingual length.

Tooth (Measurement)	Significant Effects and Interactions	*p*	Relationship
right maxillary premolar (MD)	None		
right maxillary premolar (BL)	–	–	–
left maxillary premolar (MD)	SES SES*Age	0.011 0.052	Smallest teeth in low SES Smallest teeth in young low SES (10.0-24.9 years old)
left maxillary premolar (BL)	SES*Age	0.018	Smallest teeth in young low SES (10.0-17.9 years old)
right maxillary molar (MD)	–	–	–
right maxillary molar (BL)	SES	0.081	Smallest teeth in low SES
left maxillary molar (MD)	–	–	–
left maxillary molar (BL)	None		
right mandibular premolar (MD)	None		
right mandibular premolar (BL)	None		
left mandibular premolar (MD)	SES	0.020	Smallest teeth in low SES
left mandibular premolar (BL)	None		
right mandibular molar (MD)	SES	0.032	Smallest teeth in low SES
right mandibular molar (BL)	–	–	–
left mandibular molar (MD)	–	–	–
left mandibular molar (BL)	–	–	–

Table 3.4 All significant effects and interactions for each tooth measurement and the relationship between tooth size and SES, age, sex, age*SES, age*sex, sex*SES, or age*SES*sex in a 3-way ANOVA. Dashes indicate teeth that were removed from further analyses due to unequal variances. MD = maximum mesiodistal length, BL = maximum buccolingual length.

Tooth (Measurement)	Significant Effects and Interactions	*p*	Relationship
right maxillary premolar (MD)	None		
right maxillary premolar (BL)	–	–	–
left maxillary premolar (MD)	SES*Sex	0.044	Smallest teeth in low-SES males
left maxillary premolar (BL)	Sex SES*Sex SES*Age	0.043 0.040 0.030	Smallest teeth in females Smallest teeth in high-SES females Smallest teeth in young or old low SES (18.0-29.9, 70.0-79.9 years old)
right maxillary molar (MD)	–	–	–
right maxillary molar (BL)	Sex	0.067	Smallest teeth in females
left maxillary molar (MD)	–	–	–
left maxillary molar (BL)	None		
right mandibular premolar (MD)	SES	0.059	Smallest teeth in low SES
right mandibular premolar (BL)	Sex	0.065	Smallest teeth in females
left mandibular premolar (MD)	–	–	–
left mandibular premolar (BL)	Sex SES*Age	0.003 0.068	Smallest teeth in females Smallest teeth in young low SES (18.0-39.9 years old)
right mandibular molar (MD)	–	–	–
right mandibular molar (BL)	–	–	–
left mandibular molar (MD)	–	–	–
left mandibular molar (BL)	–	–	–

relationship between tooth size and SES*age, small teeth were associated with young low-SES individuals (< 25 years old). However, in nearly all cases, the confidence intervals for the estimated means overlapped to some degree.

Table 3.5 shows the statistically significant results for the 2-way ANOVAs examining the effects of age and SES on VNC size for all individuals over the age of 10 years (for AP diameter) or 15 years (for TR diameter), using the reciprocal-transformed VNC data. For clarity, the relationships presented here are in the original, untransformed units. Of the 9 VNC measurements that were analyzed, seven measurements exhibited significant associations between VNC size and SES, age, or SES*Age. An examination of the estimated means for each group indicated that, in the samples exhibiting a significant relationship between VNC size and SES, small AP diameters were associated with the low-SES group, whereas small TR diameters were associated with the high-SES group. Similarly, in all samples exhibiting a relationship between VNC size and age, larger VNC diameters were found in individuals over approximately 60 years of age, whereas the smallest VNC diameters were associated with the middle-aged (50-59.9 years) groups. In the single case exhibiting an SES*age interaction, VNC sizes were smallest among young (10.0-15.9 years) and middle-aged (50.0-64.9 years) individuals in the low-SES group. However, the confidence intervals for the estimated means overlapped across age and SES categories.

Table 3.6 shows the statistically significant results for the 3-way ANOVAs examining the effects of age, SES, and sex on VNC size for adults (≥18 years of age) in the sample, using the reciprocal-transformed VNC data. For clarity, the relationships presented here are in the original, untransformed units. Of the six VNC measurements

Table 3.5 All significant effects and interactions for each VNC measurement and the relationship between VNC size and SES, age, or age*SES in a 2-way ANOVA. Dashes indicate vertebrae that were removed from further analyses due to unequal variances. TR = maximum transverse diameter, AP = maximum anteroposterior diameter. For clarity, relationships are presented in the original, untransformed units (i.e., "small" refers to a small diameter in millimeters, rather than the inverse of millimeters).

Vertebra (Measurement)	Significant Effects and Interactions	*p*	Relationship
L1 (TR)	SES Age	0.049 0.000	Smallest in high SES Smallest in ages 50-54.9; increases after 55 years old
L1 (AP)	–	–	–
L2 (TR)	Age	0.003	Smallest in ages 50-54.9; increases after 55 years old
L2 (AP)	None		
L3 (TR)	SES Age	0.081 0.000	Smallest in high SES Smallest in 50-54.9; increases after 55 years old
L3 (AP)	None		
L4 (TR)	Age	0.006	Smallest in ages 50-54.9; increases after 55 years old
L4 (AP)	SES Age SES*Age	0.069 0.003 0.063	Smallest in low SES Smallest in ages 10-13.9, 17-17.9, 50-64.9 Smallest in young & middle-aged low SES (10.0-15.9, 50.0-64.9 years old)
L5 (TR)	Age	0.055	Smallest in ages 50-59.9; increases after 60 years old
L5 (AP)	SES	0.098	Smallest for low SES

Table 3.6 All significant effects and interactions for each VNC measurement and the relationship between VNC size and SES, age, and sex in a 3-way ANOVA. Dashes indicate vertebrae that were removed from further analyses due to unequal variances. TR = maximum transverse diameter, AP = maximum anteroposterior diameter. For clarity, relationships are presented in the original, untransformed units (i.e., "small" refers to a small diameter in millimeters, rather than the inverse of millimeters).

Vertebra (Measurement)	Significant Effects and Interactions	*p*	Relationship
L1 (TR)	SES Age Sex	0.002 0.004 0.000	Smaller in high SES Small in ages 18-39.9, smallest in ages 50-59.9; increases after 60 years old Smaller in females
L1 (AP)	–	–	–
L2 (TR)	SES Age Sex	0.045 0.030 0.000	Smaller in high SES Small in ages 18-39.9, smallest in ages 50-59.9; increases after 60 years old Smaller in females
L2 (AP)	–	–	–
L3 (TR)	SES Age Sex	0.009 0.003 0.002	Smaller in high SES Small in ages 18-39.9, smallest in 50-64.9; increases after 65 years old Smaller in females
L3 (AP)	–	–	–
L4 (TR)	SES Age Sex	0.013 0.028 0.001	Smaller in high SES Small in ages 18-39.9, smallest in ages 50-59.9; increases after 60 years old Smaller in females
L4 (AP)	None		
L5 (TR)	Sex	0.062	Smaller in females
L5 (AP)	–	–	–

that were analyzed, five measurements exhibited significant associations between VNC size and SES, age, or sex. No significant interactions (e.g., SES*sex) were found. An examination of the estimated means for each group indicated that, in the samples exhibiting a significant relationship between VNC size and SES, small VNC diameters were associated with the high-SES group. In all samples exhibiting a relationship between VNC size and age, larger VNC diameters were associated with individuals over 60 or 65 years of age, whereas smaller VNC diameters were common in the younger (18.0-39.9) and middle-aged (50-59.9 years) groups. In all samples exhibiting a relationship between VNC size and sex, VNC sizes were smallest among females. However, the confidence intervals for the estimated means overlapped in most of the analyses, except for the confidence intervals for the estimated means for males and females in the L1, L2, and L3 samples.

3.4 Discussion

The goal of this study was to examine the associations between multiple skeletal indicators of developmental disruption and age at death, yet also determine how culturally-salient factors such as sex (as a proxy for gender) and burial location (as a proxy for SES) influenced the relationship between early life adversity and later life health (evinced by skeletal indicators of stress and age at death) in industrializing England. Collectively, the results indicate that skeletal indicators of developmental disruption were concentrated among individuals of low SES, and the age at death of individuals who experienced early life adversity was consistently younger than individuals who did not exhibit skeletal indicators of developmental stress. Consequently,

the results of this study generally agree with the results of studies examining the evidence for the DOHaD hypothesis in living populations: developmental disruption can negatively affect later life morbidity and mortality, and the relationship is particularly strong in individuals of low socioeconomic status (Galobardes et al., 2004, 2006; Kelly-Irving et al., 2013). However, the results of this study also complicate this seemingly straightforward association, especially when the developmental timing of each skeletal indicator is considered.

3.4.1 Stress during the Prenatal, Infant, and Early Childhood Periods

As mentioned above, crown formation of the second premolars and molars occurs between ages 2-8 (W. H. Logan & Kronfeld, 1933; C. F. Moorrees et al., 1963). Similarly, the anteroposterior (AP) diameter of the neural canal reaches adult size by age 5 (Watts, 2013). Together, the tooth crown measurements (buccolingual and mesiodistal lengths) and neural canal AP diameter measurement are representative of the prenatal, infant, and early childhood periods. The results of this study were consistent for these skeletal indicators: smaller teeth were associated with low-SES individuals, females, and people who died before the age of 50; smaller AP diameters were associated with low-SES individuals and people who died before the age of 65. Thus, adversity in the prenatal, infant, and early childhood periods had an impact on age at death in industrial England, and the effects of early life adversity were apparently stronger for females, in general, and people of low SES.

The 18th and 19th centuries in England were rife with opportunities for stress in the prenatal, infant, and early childhood developmental periods. The rise of industry

produced a host of new health hazards, including exposure to pollution and toxins, urban crowding, unsanitary conditions, and heightened risk of disease (Storey, 1992). Rickets—a skeletal disorder caused by a lack of vitamin D, calcium, or phosphate—was commonplace in industrial cities and mining centers, in part because coal smoke and air pollution from factories drastically reduced sunlight exposure, which is necessary for vitamin D synthesis (Kirby, 2013). Sharpe (2012) notes that the risks associated with industrialization were particularly concentrated in the lower class and—in agreement with the tooth size and AP neural canal diameter data presented in this study—low-SES children were more likely to exhibit stunting associated with nutritional deprivation and illnesses like tuberculosis, diarrhea, and rheumatic fever. Recovery from lung disease, which was particularly evident in the unsanitary and overcrowded living areas of the 18th- and 19th-century poor, required access to resources that were only available to the wealthy, such as "extra-nutritious food, warmth, nursing, and to be able to stay away from work" (Sharpe, 2012, p. 1479). Wealthy mothers, for example, were able to stay home to nurse their infants, or could afford a wet nurse (V Fildes, 1988, 1995). An isotopic study of feeding practices among wealthy individuals from Christ Church Spitalfields (18th- and 19th-century London) has suggested that some infants were breastfed for 1.5 years or more (Nitsch, Humphrey, & Hedges, 2011). In contrast, working-class women were frequently expected to return to work shortly after giving birth (Engels, 2005; Perkin, 1995), and, as a result, often accelerated the weaning process and ceased breastfeeding at earlier ages (V Fildes, 1995). Popular breastmilk substitutes were prepared from nutritionally deficient ingredients, such as rice, flour, or bread mixed with milk or water (Valerie Fildes, 1986). Additionally, breastmilk substitutes made from

cow's milk may have irritated the digestive tracts of infants or served as a source of bacterial infections, such as tuberculosis and scarlet fever (Atkins, 1992; M. E. Lewis, 2002). The results of the tooth size analyses also indicate that females were more likely to exhibit developmental stress-related crown size reduction. One possibility is preferential parental investment in male offspring (Voland, Dunbar, Engel, & Stephan, 1997), which may have forced female infants and children to devote energetic resources towards ensuring survival, rather than optimal tooth growth. In contrast, this study found that high-SES males had larger teeth compared to every other subgroup. This distinction indicates that even at very young ages, upper-class boys possessed two advantageous social identities (i.e., male gender and high status), which may have conferred nutritional or immunological benefits to young boys and buffered them from the developmental insults associated with reduced tooth crown size.

The associations between small teeth and small AP neural canal diameters and earlier age at death—particularly among low-SES individuals, who were exposed to the unsanitary and overcrowded living conditions of industrializing England—suggest that physiological stress in early life affected morbidity and risk of mortality in adulthood. These findings are consistent with the clinical data from modern populations, which indicates that low SES in childhood is associated with heightened immune responsiveness (Azad et al., 2012) and decreased resistance to upper respiratory infections (S. Cohen et al., 2004) in adulthood. The mechanism connecting early life stress and negative adult health outcomes may be the chronic stress-related dysregulation of glucocorticoid receptor signaling by the hormonal products of the hypothalamic-pituitary-adrenal axis (e.g., cortisol). Prolonged elevated cortisol secretion, such as that

exhibited by individuals who experienced low SES in childhood (Li, Power, Kelly, Kirschbaum, & Hertzman, 2007), may produce a downregulation of the expression or function of glucocorticoid receptors, which influence inflammatory and anti-inflammatory processes throughout the body (G. E. Miller, Cohen, & Ritchey, 2002). For example, glucocorticoid receptor signaling inhibits proinflammatory transcription factors like NF-κB, which facilitate the production of the inflammatory cytokine interleukin-6 (J. I. Webster, Tonelli, & Sternberg, 2002). As a result, the downregulation of glucocorticoid receptor signaling reduces the ability of the immune system to respond to anti-inflammatory signals and enables exaggerated cytokine-mediated inflammatory processes that contribute to the pathogenesis of inflammation-related respiratory, infectious, and cardiovascular diseases in adulthood (E. Chen & Miller, 2013; G. Miller & Chen, 2007; G. E. Miller et al., 2009, 2008).

3.4.2 Stress during Later Childhood and Adolescence

As mentioned above, the transverse (TR) diameter of the vertebral neural canal completes much of its growth in utero, but continues growing—and is thus susceptible to physiological constraint—until 15 years of age (Hinck et al., 1966; R. Watts, 2013b). Consequently, the TR diameter registers developmental disruptions occurring during the prenatal, infant, and childhood periods—similar to tooth crown size and the AP diameter of the neural canal—but also provides evidence of stresses occurring during adolescence. In this study, small TR diameters were associated with females, younger age at death (< 60 years of age), and individuals of high SES. The associations between small TR diameters and females and younger age at death were largely consistent with the tooth

size and AP diameter results, and likely reflect the same processes. That is, physiological disruptions throughout the developmental period resulted in smaller neural canal dimensions and earlier ages at death, and these developmental disruptions were particularly concentrated in female individuals.

The transverse (TR) neural canal diameter analyses suggest an association between later developmental disruptions and individuals of high SES. Although the tooth size and AP diameter results suggested that high-SES children were buffered from the detrimental effects of urban life, the TR diameter results suggest some health disruptions were concentrated among the upper-class. Other studies of skeletal material from industrial England have also demonstrated an unexpectedly poor growth values and high frequencies of metabolic disease among high-SES children (Hughes-Morey, 2016; M. E. Lewis, 2002; Newman & Gowland, 2017), suggesting that social privilege did not fully buffer high status individuals from developmental disruptions. In fact, some childcare practices, such as keeping upper-class children indoors to protect them from "moral or physical contamination" (Burnett & Burnett, 2013a, p. 48), may have had a detrimental effect on the development of high-SES children. Preventing children from venturing outdoors may have predisposed them to vitamin D deficiencies, which has been linked to a variety of medical conditions, infectious diseases, and neurological disorders (Nimitphong & Holick, 2011). Interestingly, although these later childhood and adolescent disruptions were recorded in the small TR diameters of high-SES children, the associations between small TR diameters and high SES and between small TR diameters and earlier age at death do not interact as they do in the tooth size and AP diameter analyses (e.g., small teeth are consistently associated with low-SES individuals who died

by age 50). Therefore, though wealth did not completely buffer high-SES children from stress during childhood and adolescence, it may have allowed them to survive to older ages than their low-SES counterparts.

3.5 Conclusion

This study examined the associations among skeletal indicators of developmental disruption (tooth crown size and vertebral neural canal dimensions) and age at death, sex, and socioeconomic status (SES) in skeletal samples from industrializing England. Broadly, the results suggested that developmental disruption was associated with low-SES individuals and earlier age at death (i.e., before approximately 60 years of age). These findings agree with the results of studies examining the evidence for the DOHaD hypothesis in living populations, which indicate that early life stress negatively impacts health in later life and the effect is often mediated by SES. However, the results of this study also suggest the relationship is complex and influenced by cultural context. Small teeth and anteroposterior neural canal diameters were related to the marginalization of females and low-SES individuals during the prenatal, infant, and early childhood periods and were associated with younger ages at death among individuals of low SES. In contrast, small transverse neural canal diameters were associated with high-SES individuals and reflect stress events occurring in later childhood and adolescence that primarily affected upper-class individuals. Importantly, there was no interaction among small transverse diameters, high SES, and age at death, suggesting that the developmental disruptions impacting high-SES individuals did not necessarily imply risk of earlier death in adulthood. In contrast, the multi-way associations among developmental stress

(evinced by small teeth and AP diameters), low SES, and age at death suggested that stress in early life was related to premature mortality in adulthood for low-status individuals.

CHAPTER 4

USING CRANIOFACIAL FLUCTUATING ASYMMETRY TO EXAMINE THE INTERSECTIONS OF SEX, AGE, SOCIOECONOMIC STATUS, AND EARLY LIFE EXPERIENCES IN INDUSTRIAL ENGLAND[3]

[3] Yaussy, SL. To be submitted to the *American Journal of Physical Anthropology*.

4.1 Introduction

The developmental origins theory (or, as it is now known, the Developmental Origins of Health and Disease (DOHaD) hypothesis) was originally formulated by epidemiologist David Barker (Barker, 2007), who recognized that, in Britain, geographic patterns of mortality due to coronary heart disease did not coincide with regional differences in dietary fat consumption or lifestyle factors like cigarette smoking, but with regions that exhibited high rates of neonatal mortality in the preceding decades. In that study and others, Barker reasoned that poor living conditions may influence the metabolic, hormonal, and nutritional environment within which a fetus develops, causing changes in the phenotype of the infant, such as low birthweight (Barker et al., 1993, 1989; Barker & Osmond, 1986). Barker's hypothesis (or the fetal origins hypothesis) posited that the fetal environment acts as a cue to alter the physiology of the developing fetus to match the predicted postnatal environment. "Mismatch" can occur, however, when the post-birth environment does not match predicted environmental conditions. In sum, adaptive responses during development ensure the survival of the fetus (e.g., low birthweight if maternal cues implied resource scarcity) but could ultimately produce negative consequences (e.g., insulin resistance, impaired glucose tolerance, high serum cholesterol, coronary heart disease) in adult life (Gluckman, Cutfield, et al., 2005; Gluckman et al., 2008; Hanson & Gluckman, 2008). Recent research has expanded the scope of Barker's work beyond maternal nutrition and fetal adaptive responses, and current foci include the epigenetic effects of maternal stress (e.g., Wadhwa et al., 2009; Entringer et al., 2010; Rodney and Mulligan, 2014; Mulligan, 2016) and the impact of early-life stressors on the response of the hypothalamic-pituitary-adrenal axis to later life

stress and infection (Avitsur et al., 2006; G. E. Miller et al., 2009; Prentice & Moore, 2005).

Within the last decade, scholars have applied the DOHaD perspective in bioarchaeological studies, using skeletal evidence to tackle new questions and previously unexplored research directions, such as the link between early life stress and later life morbidity and mortality (Gowland, 2015; Temple, 2018). Skeletal indicators of developmental disruption such as limb length, enamel hypoplasia, and vertebral size have been found to correlate with younger ages at death in adulthood (e.g., Amoroso et al., 2014; Armelagos et al., 2009; Jesper L. Boldsen, 2007; Newman & Gowland, 2015; Temple, 2014; R. Watts, 2011, 2013a, 2015). Importantly, bioarchaeological studies add temporal depth to the study of early-life health events and can answer questions about how developmental disturbances have impacted frailty and mortality across cultures throughout history.

One theoretical perspective that has received relatively little recognition in bioarchaeology is intersectionality, which emphasizes the potential for multiple marginalizations to intersect in a single individual. Researchers examining health in living populations have adopted the perspective and demonstrated that social identities, such as those based on race, sex, class, age, or sexual orientation, cannot be sufficiently studied using methodologies that treat them as distinct categories of analysis (Collins, 2015). In intersectional studies (e.g., Stirratt et al., 2008; Warner and Brown, 2011; Hinze et al., 2012; Sen and Iyer, 2012; Seng et al., 2012; Longman Marcellin et al., 2013), the intersections of various social and biological axes are foregrounded, because they

considered to be more central to the nature of life experiences of health outcomes than any of the axes considered separately (Veenstra, 2011).

The purpose of this study is to provide an example of how intersectionality and the DOHaD hypothesis could be incorporated into bioarchaeological studies of health and mortality in the past. In accordance with the DOHaD hypothesis, this study analyzes data on fluctuating asymmetry (FA), a measure of developmental instability, to investigate how early life experiences impacted age at death in populations from Industrial England. At the same time, data on sex and socioeconomic status (SES) are included to determine how social axes of inequality impacted FA and if the effects of those variables are distinct or interact to some degree. These results are situated within their larger cultural context, given that a goal of intersectionality is to acknowledge the interplay between individual-level characteristics and the structural processes that contribute to their expression (Bauer, 2014; Bowleg, 2012).

4.1.1 Fluctuating Asymmetry

Morphological asymmetry in bilateral features has been explored extensively by biologists interested in how and why asymmetries develop in a variety of organisms (Klingenberg, 2015; A. P. Møller, 1997). Interest in asymmetry stems from the assumption that corresponding structures on the left and right side of a bilaterally symmetrical organism are under the control of the same genome and should experience the same environmental conditions, and thus the right and left sides should both produce identical copies of the "target" phenotype specified by the genome. However, because of subtle, random variations in processes occurring at the molecular level, subtle differences

are generated across developing tissues and organs (Klingenberg, 2015). Thus, researchers investigating fluctuating asymmetry (FA) are often interested in the sources or effects of 'developmental noise', or those environmental perturbations or stressors that could disrupt the ideal phenotypic development of an individual or feature (A R Palmer, 1994; A R Palmer & Strobeck, 1992). Studies have generally indicated that environmentally-produced FA is most pronounced when the population exists in a marginal environment and the stressor (e.g., temperature extremes, pollutants or toxins, etc.) is experienced prenatally (Parsons, 1990). In human studies, this has led to an interest in SES differences in FA (i.e., as a morphological indicator of developmental instability or as evidence in support of the DOHaD hypothesis) or changes in FA levels within a single population over time (Bigoni, Krajíček, Sládek, Velemínský, & Velemínská, 2013; DeLeon, 2007; Livshits et al., 1988; Bariş Özener & Fink, 2010; Barış Özener, 2010b; Barış Özener & Ertuğrul, 2011; Weisensee & Spradley, 2018).

Although examples of anthropological studies of asymmetry in the postcranial skeleton exist (e.g., Albert & Greene, 1999; Barış Özener, 2010b), most human studies have focused on the skull, as it is less subject to directional asymmetry (i.e., asymmetry in one or more bilateral features that favors either the right or left side) generated by genetics or biomechanic factors such as hand dominance. DeLeon (2007) evaluated craniofacial FA as a measure of environmental stress in two cemeteries from medieval Kulubnarti, Sudanese Nubia. Her results indicated that the early Christian period, which is believed to have been the more stressful time period, exhibited greater overall FA in the craniofacial skeleton compared to the late Christian period. Another study of skulls from medieval and 20th-century Poland has revealed higher values of FA in the modern

individuals, which the authors argue is related to the environmental pollutants associated with a modern environment (e.g., toxins, tobacco smoke, etc.) (Gawlikowska et al., 2007). A study of individuals from prehistoric Japan by Hoover and Matsumura (2008) produced mixed results, suggesting that FA decreased somewhat (not significantly) between the more stable Middle Jomon and Okhotsk periods compared to the Late/Final Jomon period. The results of this study appear to contradict previous studies of FA and previous archaeological findings about the nutritional and developmental stability of the examined historical periods; however, these results may be related to the study's limited number of FA measurements (3 craniometric distances), small sample size (n = 49), and large number of sampled archaeological contexts (13 sites).

Another study of SES and facial FA in adults from ten Latin American cities appears to conflict with other studies supporting a significant association between FA and SES, as it found no significant correlation between facial FA and measures of SES (Quinto-Sánchez et al., 2017). However, the interpretations of the study may be dependent upon the significance level selected for analysis ($p \leq 0.01$), which altered their conclusions despite correlation values between facial FA and four principle component scores for indicators of wealth that would by many scholars' standards be considered statistically significant ($p = 0.051, 0.100, 0.028, 0.036$). Regardless, the same study also demonstrated associations between FA and age (increased FA at older ages) and FA and sex (increased FA in females) (Quinto-Sánchez et al., 2017).

Some studies have explicitly addressed the potential for FA to serve as a skeletal indicator of early life stress, which can be used to evaluate bioarchaeological support for the DOHaD hypothesis. In a study of British children, Pound and colleagues (2014)

found no evidence of an association between facial FA and measures of childhood health. However, the authors suggest that their results may have to do with their sample: modern medicine potentially limits exposure to pathogens and nutritional stress and could therefore have affected the expression of FA due to developmental stress. Pound and colleagues (2014) also acknowledge that previous demographic analyses on their data set (the Avon Longitudinal Study of Parents and Children, ALSPAC) have demonstrated that the data have an overrepresentation of affluent children, perhaps limiting the applicability of their findings to other populations. Particularly, an overrepresentation of wealthy children may limit the observed pattern between FA and childhood health, given that lower SES is expected to correlate with negative health outcomes and more pronounced facial FA. In contrast to the study by Pound and colleagues (2014), studies by Watts (2013a) and Weisensee (2013) found associations between early life developmental stability (evinced by FA values) and later life morbidity and mortality. In a study of childhood health indicators and adult mortality, Watts (2013a) found that cribra orbitalia presence and FA values corresponded with earlier mortality in females living in northern England prior to 1700 A.D. Age at death in males, however, was associated with cribra orbitalia presence, small vertebral neural canals, and short femoral lengths. Likewise, age at death in males and females in a group from after 1700 A.D. was only associated with small vertebral neural canals. Watts (2013a) suggests that environmental and economic changes in the post-1700 A.D. period protected that population from the early mortality associated with developmental insults (e.g., higher FA values) seen in the pre-1700 A.D. period. In the study by Weisensee (2013), individuals from 19th- and 20th-century Portugal exhibited greater degrees of FA if they were dying of degenerative diseases,

compared to those individuals dying of infectious diseases. The conflicting findings among studies of FA in different contexts might indicate that there is regional, temporal, or cultural variation with respect to the risk of developing asymmetric features, particularly within SES groups. This study examines the associations among craniofacial FA, SES, sex, and age at death in adults from industrial-era cemeteries in England. Particularly, this study tests the hypothesis that individuals exhibiting multiple marginalizations (i.e., low-SES females) will exhibit higher levels of FA compared to other analyzed groups (i.e., low-SES males, high-SES females, high-SES males), and that higher levels of FA (as a skeletal indicator of developmental instability) will be associated with earlier ages at death in marginalized individuals.

4.2 Materials and Methods

4.2.1 Skeletal Samples

The osteological data for this study come from four cemeteries in England that were in use during the early period of industrialization from the mid-18th to the mid-19th centuries: St. Bride's Church Fleet Street (n = 101), New Bunhill Fields (n = 50), St. Peter's Church Wolverhampton (n = 10), and Coach Lane (n = 7). St. Bride's Fleet Street is a collection of crypt burials from London dated to A.D. 1740-1852. Information gleaned from historical records associated with St. Bride's Church (e.g., the parish register) and the archaeological context (e.g., coffin plates) suggest that the crypt is associated with individuals of relatively high status, such as clerks, merchants, and gentlemen (Gustav Milne, 1997). Consequently, St. Bride's Fleet Street is considered high SES compared to the other three collections analyzed in this study.

To access differences in frailty and mortality associated with socioeconomic position, this study compares the high-status individuals interred in St. Bride's Church to individuals from three cemeteries that catered largely to the working class of industrial England. New Bunhill Fields burial ground is located in London and dates to A.D. 1821-1853. No burial register is available, but historical documents suggest that the burial ground served a largely Nonconformist population and was cheap enough to be considered affordable for working-class (low-SES) individuals (Miles & Connell, 2012). St. Peter's Collegiate Church in Wolverhampton, England opened an overflow cemetery in 1819 and received the last burials in 1853. The overflow cemetery primarily catered to working-class individuals, given that most of the individuals buried there were interred in earth-cut graves. Brick-lined graves—which indicate the disposable income available to wealthier parishioners—were not found during excavation of the overflow cemetery, suggesting the burial ground is composed of individuals of lower SES (Adams, 2007). The third low-SES skeletal sample used in this study is Coach Lane, a skeletal collection from North Shields, England. Coach Lane was associated with the Quaker Society of Friends Meeting House and was in use from 1711 to 1857 (Proctor et al., 2014). Like New Bunhill Fields and St. Peter's Wolverhampton, the Coach Lane skeletal collection is believed to be primarily composed of working-class (low SES) individuals. Although historical documents suggest that some of the Quakers in the region were in the middle class (Tancock & Lee, 2014), the simple lifestyles adopted by Quakers (Dandelion, 2008) and the uncertainty surrounding their higher social status (Roberts et al., 2016) leads this study to conservatively consider the Coach Lane assemblage to be composed of low- or

working-class individuals (i.e., of lower SES than the St. Bride's Fleet Street skeletal sample).

4.2.2 Age Estimation

Transition analysis (Jepser L. Boldsen et al., 2002) was used to estimate age at death for all adults (18+ years) in the sample. Unlike conventional age estimation methods, transition analysis avoids "age mimicry", in which the age estimates produced are biased by the composition of the of the reference sample. In addition, transition analysis is appropriate for small sample sizes and provides age at death estimates for even the oldest individuals in a sample, allowing for the evaluation of patterns of mortality at older ages than is typically possible. The age-at-death distributions for each site are provided in **Table 4.1**.

4.2.3 Sex Estimation

The sexually dimorphic features of the pelvis and skull were used to estimate sex for all individuals in this study, following Buikstra and Ubelaker (1994a). Only those individuals for which age at death and sex could be estimated were included in this study; that is, non-adults (<18 years of age) and individuals for which sex was indeterminable or inestimable were excluded from the analysis. Any individual determined to be a "probable female" or "probable male" was analyzed as part of the "female" (0) or "male" (1) categories, respectively.

Table 4.1 Age-at-death distributions of male and female individuals from each site. Total sample size = 168. % = percentages of female or male individuals within each age category for each site.

Age	St. Bride's Fleet Street		Coach Lane		St. Peter's Wolverhampton		New Bunhill Fields	
	Female	Male	Female	Male	Female	Male	Female	Male
18.0 - 24.9	4 (8.3%)	2 (3.8%)	0 (0.0%)	0 (0.0%)	1 (25.0%)	1 (16.7%)	6 (27.3%)	4 (14.3%)
25.0 - 29.9	4 (8.3%)	4 (7.5%)	0 (0.0%)	0 (0.0%)	2 (50.0%)	1 (16.7%)	5 (22.7%)	4 (14.3%)
30.0 - 34.9	3 (6.3%)	7 (13.2%)	0 (0.0%)	1 (25.0%)	1 (25.0%)	2 (33.3%)	2 (9.1%)	6 (21.4%)
35.0 - 39.9	3 (6.3%)	3 (5.7%)	0 (0.0%)	1 (25.0%)	0 (0.0%)	2 (33.3%)	4 (18.2%)	4 (14.3%)
40.0 - 44.9	5 (10.4%)	6 (11.3%)	0 (0.0%)	0 (0.0%)	0 (0.0%)	0 (0.0%)	2 (9.1%)	3 (10.7%)
45.0 - 49.9	5 (10.4%)	1 (1.9%)	1 (33.3%)	1 (25.0%)	0 (0.0%)	0 (0.0%)	0 (0.0%)	2 (7.1%)
50.0 - 54.9	4 (8.3%)	1 (1.9%)	0 (0.0%)	0 (0.0%)	0 (0.0%)	0 (0.0%)	1 (4.5%)	0 (0.0%)
55.0 - 59.9	4 (8.3%)	4 (7.5%)	0 (0.0%)	0 (0.0%)	0 (0.0%)	0 (0.0%)	0 (0.0%)	0 (0.0%)
60.0 - 64.9	4 (8.3%)	4 (7.5%)	0 (0.0%)	0 (0.0%)	0 (0.0%)	0 (0.0%)	0 (0.0%)	0 (0.0%)
65.0 - 69.9	8 (16.7%)	6 (11.3%)	0 (0.0%)	0 (0.0%)	0 (0.0%)	0 (0.0%)	1 (4.5%)	2 (7.1%)
70.0 - 74.9	2 (4.2%)	12 (22.6%)	1 (33.3%)	1 (25.0%)	0 (0.0%)	0 (0.0%)	1 (4.5%)	2 (7.1%)
75.0 - 79.9	2 (4.2%)	3 (5.7%)	0 (0.0%)	0 (0.0%)	0 (0.0%)	0 (0.0%)	0 (0.0%)	1 (3.6%)
80+	0 (0.0%)	0 (0.0%)	1 (33.3%)	0 (0.0%)	0 (0.0%)	0 (0.0%)	0 (0.0%)	0 (0.0%)
Total	48 (100%)	53 (100%)	3 (100%)	4 (100%)	4 (100%)	6 (100%)	22 (100%)	28 (100%)

4.2.4 Fluctuating Asymmetry Data Collection

Three-dimensional landmark coordinate data were collected for this study using a Microscribe G2X Digitizer. To minimize measurement error and ensure the same reference point was collected from all individuals, most of the landmarks selected for this study are considered "type 1" landmarks, which are defined by the intersection of boundaries or the juxtaposition of tissues, such as the intersections of suture lines on the human skull (Bookstein, 1997). Exceptions to this rule included easily replicable measurements (e.g., ectoconchion) or visually apparent points of maximum curvature (e.g., opisthion). All bilateral and midsagittal landmarks were unambiguously defined prior to data collection (**Table 4.2** and **Figure 4.1**) following the definitions provided by Howells (1973), Buikstra and Ubelaker (1994a), and Fleischman et al. (2018).

A generalized Procrustes superimposition was used to extract information about asymmetry in shape from the landmark coordinates collected for each individual. A Procrustes fit removes variation in size, position, and orientation, standardizing those aspects for all individuals in the sample so the only remaining component of variation is shape. That is, after the distance between corresponding landmarks has been minimized with the Procrustes superimposition, the remaining difference is purely shape difference (Klingenberg and McIntyre, 1998; Klingenberg, 2015). To assess measurement error in the sample, a subsample of crania (n = 19) were digitized at least 4 times apiece, with each repetition on a separate day. A Procrustes analysis of variance (i.e., a conventional two-factor ANOVA) was then used to evaluate shape asymmetry and measurement error. Levene's test for equality of variances was used to test for heterogeneity of variances

Table 4.2 Names and definitions of the landmarks used in this study. Abbreviations listed coincide with labels in **Figure 4.1**.

Landmark	Definition
alare (alar)	Most lateral point of the nasal aperture
asterion (ast)	Intersection of the lambdoid, parietomastoid, and occipitomastoid sutures
basion (bas)	Midpoint of the anterior margin of the foramen magnum
bregma (brg)	Intersection of the coronal and sagittal sutures
dacryon (dac)	Intersection of the frontolacrimal and lacrimomaxillary sutures
ectoconchion (ect)	Most lateral point of the orbital lateral wall
fob maximum breadth point (fob)	Most lateral point on either side of the foramen magnum
glabella (glb)	Most anterior point of the frontal bone in the midsagittal plane
hormion (hor)	Median point of attachment of vomer and sphenoid bones
jugale (jug)	Deepest points of curvature between the frontal and temporal processes of the malars
krotaphion (kro)	Most posterior point of the sphenoparietal suture
lambda (lam)	Intersection of the sagittal and lambdoidal sutures
most inferior nasal border (nlhi)	Most inferior point of the nasal aperture
nasale inferius (nasi)	Most inferior point of the nasomaxillary suture
nasale superius (nass)	Most superior point of the nasomaxillary suture
nasion (nas)	Intersection of nasofrontal suture and midsagittal plane
opisthion (ops)	Midpoint of the posterior margin of the foramen magnum
porion (por)	Most superior point on the margin of the external auditory meatus
prosthion, Howells (pro)	Most anterior point in the midline on the alveolar border between the central incisors
radiometer point (radpt)	Point in the center of the external auditory meatus
sphenion (sph)	Most anterior point of the parietal where the parietal and sphenoid meet the coronal suture
sphenofrontale (jnz)	Intersection of the frontal, sphenoid, and zygomatic
stephanion (stp)	Intersection of the coronal suture and the inferior temporal line
subspinale (ssp)	Deepest point seen in the profile below the anterior nasal spine
zygomaxilare (zygom)	Intersection of the zygomaxillary suture and the attachment of the masseter muscle
zygoorbitale (zygoo)	Intersection of the orbital margin and the zygomaxillary suture
zygotemporale inferior (zyti)	Point at the inferior zygotemporal suture on the zygomatic process
zygotemporale superior (zyts)	Point at the superior zygotemporal suture on the zygomatic process

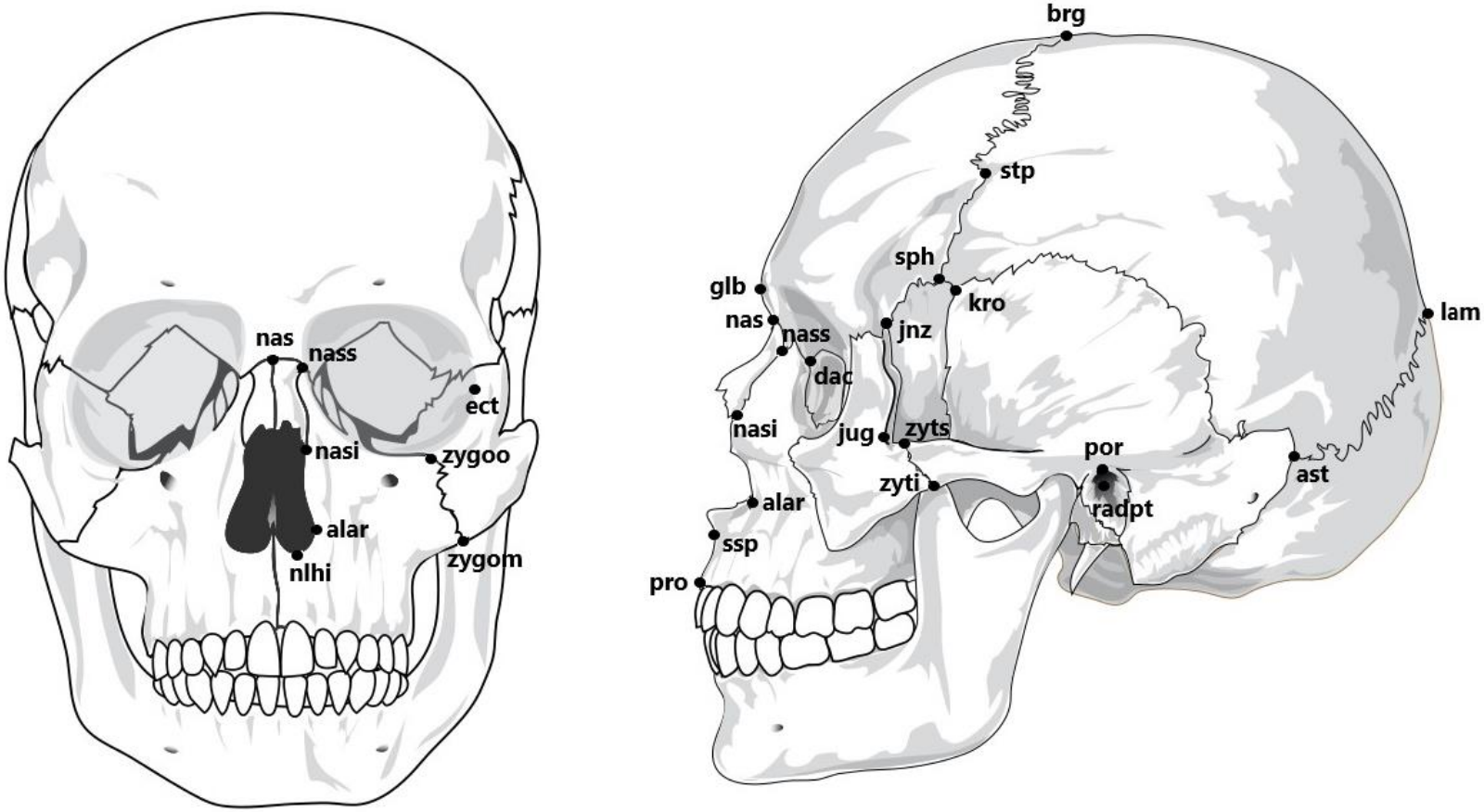

Figure 4.1 Anterior and lateral view of the landmarks used in the study. Not pictured: bas, fob, hor, ops.

among age at death categories, between males and females, and between the high- and low- SES groups. The Procrustes superimposition and Procrustes ANOVA were performed using the MorphoJ software package produced by Klingenberg (2011).

Because FA results from random events during development, scholars have found it useful to generate a measure of the magnitude of FA for each individual to evaluate developmental imprecision by proxy (e.g., Møller and Swaddle, 1997; Polak, 2003). One such measure is the Procrustes distance between the original and reflected copies of the landmark configuration (Klingenberg, 2015). As in previous morphometric studies of the human skull (Pound et al., 2014; Weisensee, 2013), this study used this method to generate a single, multivariate estimate of the overall magnitude of shape difference per individual: the Mahalanobis distance or FA score. Mahalanobis FA scores were calculated for all individuals in MorphoJ (Klingenberg, 2011). The sample kurtosis value was examined to determine if the frequency distribution of the Mahalanobis FA scores exhibited antisymmetry (i.e., a bimodal or platykurtic distribution).

Initial tests of the data indicated nonnormality and unequal variances in the age at death, sex, and SES groups. The data were first transformed using the natural log of the Mahalanobis FA scores, but this transformation did not improve the heterogeneity of variances indicated by Levene's test. Consequently, the data were transformed using the reciprocal of the Mahalanobis FA scores, and this transformed dataset exhibited normal distributions and equal variances in all analyzed groups. The associations among the reciprocally-transformed Mahalanobis FA scores, adult age at death, sex, and SES were assessed using three-way ANOVA, which allows for an assessment of age, sex, and SES as separate effects, as well as the interactions among the variables and their association

with FA value. Here, p-values equal to or less than 0.10 are considered to be significant. However, given the relatively small sample size analyzed by this study (n = 168) and the issues concerning *p*-values that have been raised by numerous scholars (H. W. Cohen, 2011; S. N. Goodman, 1999; Lang et al., 1998; Rothman, 1998; Trafimow & Marks, 2015), the *p*-values included in this study should be taken with caution.

4.3 Results

The results from the Procrustes ANOVA to determine shape asymmetry and measurement error are presented in **Table 4.3**. The test indicates that the variance in FA was greater than the variance due to measurement error ($F = 5.70$). Levene's test was used to evaluate the homogeneity of variances in all analyzed groups, and none of the data returned significant results (p-value for sex = 0.943, for age = 0.428, and for SES = 0.440). The kurtosis value for the transformed data was -0.017 (s.e. = 0.373), which is well under the significance threshold for kurtotic distributions. These results suggest that antisymmetry does not substantially affect the sample.

The results of the 3-way ANOVA of the association among the reciprocal-transformed Mahalanobis FA score, age, sex, and SES are presented in **Table 4.4**. The results indicate that the effect of SES is significant ($p < 0.001$), the effect of Sex is significant ($p = 0.056$), and the interaction among age, sex, and SES is significant ($p = 0.077$). Evaluation of the estimated means indicates that reciprocal-transformed Mahalanobis FA scores were higher among low-SES individuals in general, females in general, and young low-SES females (18.0 – 34.9 years) and mid-age low-SES males (35.0 – 44.9 years). In untransformed terms (i.e., in terms of the original units, rather than

Table 4.3 The results of the Procrustes ANOVA to determine shape asymmetry (Individual × Side) and Measurement Error using the untransformed data.

Source	df	SS	MS	*F*
Individual	8232	0.7205426	0.0000875272	4.30[a]
Side	43	0.00675886	0.0001571827	7.73[a]
Individual × Side	7224	0.14687645	0.0000203317	5.70[a]
Measurement Error	5612	0.02002389	0.0000035680	

[a] = $p < 0.0001$

Table 4.4 The results of the 3-way ANOVA to determine the association among Mahalanobis FA score (reciprocal-transformed data), age at death, sex, and SES.

Mahalanobis FA Score (reciprocal-transformed)	**SS**	**F**	***p*-value**
Sex	0.005	3.732	0.056
Age	0.008	0.494	0.915
SES	0.036	28.317	<0.001
Sex * Age	0.015	1.097	0.369
Sex * SES	0.00007242	0.057	0.812
Age * SES	0.01	0.902	0.526
Sex * Age * SES	0.017	1.885	0.077

in reciprocally-transformed units), asymmetry was most pronounced among males, high-SES individuals, and young high-SES males (18.0 – 24.9 years).

4.4 Discussion

The results of the 3-way ANOVA of the reciprocally-transformed FA scores suggest that SES and sex were independently, significantly associated with FA score. However, the results did not support the hypothesis that FA score would be associated with marginalized SES and sex categories (i.e., low SES and female sex); instead, when converted to standard, untransformed FA score units, high-SES individuals and males were more likely to exhibit higher FA scores. In addition, an interaction between SES, sex, and age at death was significantly associated with reciprocally-transformed FA score and, when converted to untransformed FA score units, indicates that young high-SES males (18.0 –24.9 years) were more likely to have higher FA scores compared to individuals in other age, sex, or SES groups.

Studies of the developmental origins of health in living populations have emphasized the relationship between early life SES and adult morbidity and mortality. SES is particularly interesting in that it is rarely restricted to a singular event or critical period, but often also exerts an impact on life trajectories both socially and biologically, causing disadvantages to accumulate across the life course (Gagnon & Bohnert, 2012; Hertzman, 1999; Willson et al., 2007). Studies of living populations have demonstrated associations between childhood SES and overall cardiovascular disease mortality in adults, as well as other causes of adult mortality, such as stroke, coronary heart disease, and accidental or violent death (Galobardes et al., 2004, 2006; Kittleson et al., 2006).

Research has suggested that associations between childhood SES and adult mortality may be related to early-life modifications to mRNA (e.g., GR and TLR4 regions), which then promote exaggerated inflammatory reactions in response to infection, and, eventually, produce diseases associated with chronic inflammation, like cardiovascular disease (G. Miller & Chen, 2007; G. E. Miller et al., 2008). An association has even been found between early life SES and susceptibility to infectious disease in adulthood; results from Cohen and colleagues (2004) indicated that risk of contracting a cold (rhinovirus) decreased with an increase in the number of years that parents owned the home that the child lived in, and a decrease in the number of years of home ownership was associated with greater infection and greater illness expression.

As indicated above, bioarchaeological investigations of the DOHaD hypothesis have yielded similar results, demonstrating that skeletal indicators of early life physiological disruption (e.g., diminished long bone length or vertebral size, enamel hypoplasia) often correlate with younger ages at death in adulthood. Bioarchaeological studies of DOHaD in the context of industrializing England have suggested that SES and sex groups had different early life experiences, which impacted patterns of later life morbidity and mortality. In her study of two industrial-era skeletal collections from London (Lower Saint Bride's, a low-status skeletal collection, and Chelsea Old Church, a high-status skeletal collection), Hughes-Morey (2016) found differences in tibia length by sex in the low-SES skeletal collection. Hughes-Morey argues that the short tibiae of low-SES females compared to low-SES males may be explained by males experiencing less stress during growth and development than females. In another study, Newman and Gowland (2017) use evidence from multiple London cemeteries to investigate whether

children of middle or high-SES groups were buffered by privilege from the negative aspects of urban life. Interestingly, although low SES was associated with several indicators of perinatal and childhood stress, high SES was also associated with low long bone growth values (longitudinal and appositional) and small vertebral neural canal size values (Newman & Gowland, 2017).

As in previous bioarchaeological studies of the connection between early life SES and later life morbidity and mortality in Industrial England, the results of this study suggest that the relationship was not straightforward and may have been impacted by the cultural contexts to which individuals in different sex and SES groups may have been exposed. Given that human craniofacial growth is completed throughout childhood and adolescence (i.e., with different areas of the skull reaching maturity in shape and size between ages 7.7 and 15.7 years of age) (Bastir, Rosas, & O'Higgins, 2006), the presence of craniofacial asymmetry reflects developmental noise occurring during the non-adult period generally and it may be useful to compare FA with the findings of studies of other nonspecific skeletal indicators of developmental disruption. In a study of non-adults from industrial England, Newman and Gowland (2015) found that vertebral body height growth was disrupted to a greater degree and overall crude prevalence rates for rickets, non-specific infection, and cribra orbitalia were higher in their more prosperous skeletal sample from Bow Baptist (eastern outskirts of London) compared to their working-class sample from South Shields, England. In that study and their later publication (Newman and Gowland, 2015, 2017), the authors suggest environmental and cultural practices that may have influenced the unexpected concentration of poor health outcomes among high-SES children. Specifically, breastfeeding practices may have played a substantial role in

the health outcomes of high-SES children. In the mid- to late-18th century, it became fashionable for the wealthy to feed infants by hand or employ a dry-nurse in the home, rather than employ wet nurses (V Fildes, 1988, 1995). Popular substitute foods included pap, which consisted of bread soaked in water or cow's milk (Radbill, 1981), and panada, which consisted of cereals cooked in broth (Wickes, 1953). In addition to unclean artificial feeding devices, the cow's milk used in many breastmilk substitutes made them vulnerable to diarrheal infections and was often contaminated with bacteria that contributed to the spread of diseases like tuberculosis and scarlet fever (Atkins, 1992). Records from the 19th century revealed that hand-fed infants had higher mortality rates than breastfed infants—more than one third of dry-fed infants died in the first year of life (V Fildes, 1995; Knodel & Kintner, 1977; Weinberg, 1993). If infants born into high-SES families survived the first year of life, they were at risk of a variety of illnesses due to their underdeveloped immunocompetence (Cunningham, 1995; Oddy, 2001). As a result, infant feeding practices during industrialization in England may have negatively impacted the development of high-SES males, causing them to have higher degrees of FA and impacting their lifelong frailty such that they were more likely to die at younger adult ages than their peers who survived childhood with less asymmetrical features.

The plight of upper-class males does not necessarily overshadow the difficulties experienced by the English working class. Bioarchaeological studies of non-adults frequently mention the high infant and child mortality of the poor during the industrial period. Newman and Gowland (2017) note that the low-status skeletal collection used in their study (Cross Bones cemetery) was largely composed of perinates, and parish registers associated with the cemetery indicate a large majority of the non-adult burials

for the year 1845 were infants less than a year old (Brickley & Miles, 1999). Whereas high-SES families could afford to hand-feed or breastfeed their infants or hire wet- or dry-nurses, working-class families were often limited in the food and time they could offer young family members. Food, especially nutritious meat or vegetables, was often given to the father, since he was the primary source of income for the household (Horrell & Oxley, 2012; Meredith & Oxley, 2015; Nicholas & Oxley, 1993). As a result, young children in low-SES families were often malnourished, which is known to have negative effects on immune competence (Scrimshaw, 2003; Scrimshaw et al., 1968). Therefore, in addition to the overcrowding, unsanitary living conditions, and pollutants of the 18th- and 19th-century urban environment, individuals of low SES were vulnerable to a variety of infectious diseases that could cause non-adult mortality. Thus, the findings of this study—that FA is associated with high-SES individuals and high-SES males, in particular—may be the result of selective mortality. If low-SES individuals who experienced developmental disruptions (and thus had higher FA scores) were relatively more frail than their high-SES peers, they may have died prior to reaching adulthood, causing this study to find no association between FA and mortality in the working-class sample. The findings of this study are similar the results of other bioarchaeological studies of frailty and mortality in industrial-era England (DeWitte et al., 2016; Hughes-Morey, 2016), and suggests that non-adult mortality among the poor may have removed highly frail low-status children, resulting in surviving low-status adults who were less frail than their high-status counterparts.

In addition to the cultural differences that could have impacted the relationship between sex and FA, some research has suggested a biological component to the differing

sensitivity of the sexes to environmental perturbation during development. In an early review, Stinson (1985) notes that some investigations suggest males are less buffered than females against the environment during growth but acknowledges that many studies—particularly those of the postnatal period—are inconclusive about the sensitivity of males to stressors compared to females. However, recent studies have demonstrated that males may be more susceptible than females to a variety of environmental stressors, including viral, bacterial, and parasitic infections (Klein, 2000b, 2004; Klein & Huber, 2010). If males are more sensitive to and less buffered against developmental instability, it would be expected that they would exhibit greater amounts of FA, as seen in this study. However, the literature on sex differences in FA is mixed and does not necessarily support the assertion that females have a higher buffering capacity—and thus greater developmental stability and decreased FA—compared to males. Some studies have reported greater degrees of asymmetry in females (Bigoni et al., 2013; Ercan et al., 2008; Quinto-Sánchez et al., 2017), some studies have reported no difference between males and females (Ferrario, Sforza, Miani Jr, & Serrao, 1995; Hallgrímsson, 1999), and other papers agree with the findings of this study and suggest that facial asymmetry is more pronounced in males (Claes et al., 2012; Farkas & Cheung, 1981; Barış Özener, 2010a; Weisensee, 2013). The inconsistent relationship between sex and FA in the anthropological and biological literature calls for a closer look at the contexts of the studied samples, as some unexamined cultural and environmental factors may be influencing observed patterns of FA by sex between studies and over time.

4.5 Conclusion

This study examined the differences in craniofacial fluctuating asymmetry among age, sex, and socioeconomic status groups in populations from industrializing England. FA scores were significantly associated with SES and sex, suggesting that males and individuals of high SES were more likely to develop asymmetrical crania. In addition, a moderately significant interaction among age at death, sex, and SES suggests that young high-SES males in industrializing England were particularly likely to have more asymmetrical craniofacial features compared to individuals in other age, sex, or SES categories. The results of this study suggest that the unique domains generated by the intersections of sex and status categories profoundly influenced patterns of craniofacial FA and age at death in industrial-era England.

CHAPTER 5

THE INTERSECTIONAL EFFECTS OF SEX AND SOCIOECONOMIC STATUS ON RISK OF MORTALITY IN INDUSTRIALIZING ENGLAND[4]

[4] Yaussy, SL. To be submitted to the *American Journal of Physical Anthropology*.

5.1 Introduction

Though research with living populations has identified socioeconomic status (SES) as one of the most powerful predictors of mortality in the world today (Stringhini et al., 2017, 2014, 2010), historians have maintained a heated debate about whether a negative income-mortality relationship became more pronounced or existed at all during the period of industrialization in Britain (*c.* 1760-1840) or elsewhere, such as in Sweden, France, Canada, and the United States (Antonovsky, 1967; Bengtsson & Dribe, 2011; Bengtsson & van Poppel, 2011; Haines & Ferrie, 2011; Marmot, 2005). Contemporaneous assessments lend support to the presence of an income-mortality gradient in 19th-century Britain, with one observer claiming that "the greatest mortality of all occurs in that locality where filth and dissipation are conjoined…[and] these influences have their origin in the inferior standard of comfort and cleanliness attendant upon poverty" (Chadwick, 1842, p. 284). Later historians used wage data from working-class men to argue that standards of living increased substantially during the industrial era (Lindert & Williamson, 1983), but they were met with pessimism that increases in real wages and standard of living were not nearly as pronounced as initially believed (G. Clark, 2001, 2005; C. H. Feinstein, 1998; Schwarz, 1985). Studies of the recorded heights of men in the 18th- and 19th-century British military have been equally contentious. Early analyses suggested that height—a proxy for adequate nutrition and health—increased in England during the industrial period (Floud et al., 1990), whereas later analyses of the same data suggested that stature declined (Komlos & Küchenhoff, 2012). Studies of infant mortality records provide further evidence supporting the pessimistic view, demonstrating that there was no substantial improvement in the standard of living of the

working class until the mid-19th century (Huck, 1994, 1995). Likewise, analyses of life expectancy at birth suggested that between 1781 and 1826 expectation of life improved from about 35 years to 40 years, but this marginal improvement is tempered by a comparison with the figure from two centuries prior, which indicates life expectancy in the period from 1566-1621 was 38.6 years (Wrigley & Schofield, 1989).

Largely absent from these discussions has been a recognition of the heterogeneity that existed within classes in Industrial England and how the standards of living and health outcomes of other subpopulations may have differed from that of the working-class adult male. Part of this omission may be related to a dearth of comparable historical records and reliable demographic data for the 1780-1850 period (Szreter & Mooney, 1998). Historical documentation regarding the income and real wages of women (especially prior to 1841), for example, are inadequate or entirely non-existent (Nicholas & Oxley, 1993). In recent years, however, bioarchaeologists and anthropometric historians have worked to rectify the implicit homogenization of patterns of health and mortality in the industrial era, and have produced studies of the heights, weights, and health of women and children in the laboring classes of 18th- and 19th-century England. Studies comparing the heights of boys recorded by the Royal Military Academy at Sandhurst and the Marine Society revealed a dramatic height gap between the upper- and lower-class individuals, which was particularly pronounced when the lower-class boys were orphans or came from female-headed households (Horrell, Humphries, & Voth, 1998; Komlos, 2007). A bioarchaeological study of long bone length and bone cortical thickness in children from a 19th-century churchyard in Birmingham found no relationship between long bone length and socioeconomic status, but found decreased

cortical bone thickness in lower-status children compared to higher-status children of the same age, likely because of insufficient nutrition among lower-status children (Mays et al., 2009). Another bioarchaeological study demonstrated that low-status children in industrial London exhibited elevated mortality and reduced survival compared to their high-status counterparts, and the authors argue that the lack of a similar discrepancy between high- and low-status adults may be the result of status-based selective mortality in childhood (i.e., no significant difference in risk of mortality or survival was found between high- and low-status adults because frail low-status individuals die in childhood and the surviving low-status adults are comparatively less frail) (DeWitte et al., 2016).

Studies examining the health of adult women from the working class have been equally fruitful. Nicholas and Oxley (1993) examined the heights and literacy of convict women transported to Australia in the years 1826-1840 and found that average height decreased and illiteracy increased. The authors suggest that their findings are indicative of declining living standards among working-class women during the process of industrialization (Nicholas & Oxley, 1993). Similarly, Horrell and colleagues (2009) studied the available height, weight, and body mass data for working-class individuals imprisoned in a Victorian-era London House of Correction. Their results indicate that working-class children of both sexes experienced early life deprivation in industrializing London, but the body mass of working-class women declined with age while the body mass of working-class men was largely maintained (Horrell et al., 2009). These results would be echoed in a study of working-class prisoners in the Surrey House of Correction outside of London, which found that the body mass of men was largely stable throughout

adulthood, whereas the body mass of women steadily declined with age (Meredith & Oxley, 2015).

However, the studies mentioned above continue to overlook subgroups of the larger British population, such as high-status women, who may have exhibited a unique combination of advantageous and disadvantageous social identities—high status and female gender—which could have affected their life experiences and health outcomes in ways that differed from the high-status male, low-status female, or low-status male. In accordance with intersectionality theory—which posits that there are interactions between various categories of social identity (e.g., class and gender) and macro-level systems of oppression (e.g., classism and sexism) (Collins & Bilge, 2016; Crenshaw, 1989, 1991)—this study examines the relationship between socioeconomic status (SES) and mortality in skeletal samples from 18th- and 19th-century England, with a particular focus on the intersection between SES and sex categories and their embeddedness in oppressive structural systems that were operating at the time.

5.2 Materials and Methods

5.2.1 Skeletal Samples

All of the burial grounds used in this study accepted individuals for interment during the 18th and 19th centuries, when England was undergoing industrialization. The burial grounds themselves are described in greater detail elsewhere (Milne and Betts, 1997; Adams and Colls, 2007; Alan Williams Archaeology, 2008; Miles and Connell, 2012; Tancock, 2014; Roberts et al., 2016; Yaussy, in prep) and will not be repeated here. The skeletal samples from these cemeteries (see **Table 5.1**) were selected because

Table 5.1 Sample sizes used in the analyses, divided by site. Non-adults were not included in the analyses intended to assess patterns of mortality related to sex.

Site	Adult Females	Adult Males	Non-adults	Total
St. Bride's Fleet Street	97	111	13	221
Coach Lane	60	76	76	212
St. Peter's Wolverhampton	34	35	53	122
New Bunhill Fields	60	64	406	530
Total	251	286	548	1085

(1) they each conform to a relatively narrow chronological window centered on the industrial period in England and (2) there is historical and archaeological evidence available for each site to assert the SES of the individuals interred there. The St. Bride's Fleet Street skeletal collection consists of crypt burials (analyzed individuals interred between 1740 and 1852; London, England) that, along with evidence from the parish register and historical accounts, indicate the skeletal sample is composed of individuals of relatively high SES (Milne and Betts, 1997). In contrast, the individuals sampled from the Coach Lane (1711–1857; North Shields, England), St. Peter's Collegiate Church (1819–early 1850s; Wolverhampton, England), and New Bunhill Fields (1821-1853; London, England) burial grounds were all buried in earth-cut graves—the less expensive burial option, compared to crypt burial—and likely belonged to the working or lower-middle classes (Adams and Colls, 2007; Miles and Connell, 2012; Roberts et al., 2016). Therefore, in this study, St. Bride's Fleet Street is considered the high-SES group, whereas Coach Lane, St. Peter's Wolverhampton, and New Bunhill Fields are collectively considered the low-SES group.

5.2.2 Age Estimation

Non-adult age at death was estimated using a combination of dental development, diaphyseal length, and epiphyseal fusion (Buikstra and Ubelaker, 1994). When both dental and skeletal estimates were available, dental age estimates were given more weight as they have the strongest correlation with chronological age (Lewis and Garn, 1960). Adult age at death was estimated using transition analysis, as described by Boldsen and colleagues (2002). Transition analysis provides a point estimate of age for each

individual, eliminating the broad terminal age category often produced by conventional methods. Additionally, transition analysis avoids mimicking the age at death distribution of the known-age reference sample, a common problem with traditional methods, and works well with the fragmented remains typical of bioarchaeological skeletal samples.

5.2.3 Sex Estimation

Sex was estimated for adult individuals (aged 18+ years) who retained the necessary sexually dimorphic features of the skull and pelvis (Buikstra and Ubelaker, 1994). In this study, individuals scored as "probable females" or "probable males" were consolidated into binary categories of "female" (0) and "male" (1) for analysis. Non-adults were not included in the analyses intended to assess sex-based patterns of mortality.

5.2.4 Siler Model

To determine the relationship between SES and risk of death in Industrial England, SES (i.e., low SES = 0, high SES = 1) was modeled as a covariate affecting the Siler model of mortality:

$$h(a) = \alpha_1 e^{-\beta_1 a} + \alpha_2 + \alpha_3 e^{\beta_3 a}$$

where the first component, $\alpha_1 e^{-\beta_1 a}$, is associated with the juvenile period when risk of death decreases exponentially (i.e., a is age, α_1 is the risk of death associated with immaturity, and β_1 is the rate at which that risk declines with age); the second component, α_2, is associated with the constant risk of death that is unrelated to age (i.e., the age-independent component); and the third component, $\alpha_3 e^{\beta_3 a}$, is associated with the

senescent period when risk of death increases exponentially (i.e., *a* is age, α_3 is the risk of death at birth associated with senescence, and β_3 is the rate at which that risk increases with age) (Gage, 1988). Both non-adult and adult individuals were included in the analyses.

To ensure any effects of the covariate were a product of status differentials rather than geographic location, the Siler model was applied within a "combined sample" composed of individuals from all four sites (n = 1085; high SES = St. Bride's Fleet Street; low SES = St. Peter's Wolverhampton, Coach Lane, and New Bunhill Fields), as well as within a "London sample" composed of only those individuals from the London sites (n = 751; high SES = St. Bride's Fleet Street; low SES = New Bunhill Fields). Given that previous research has suggested the age-independent component of the Siler model (α_2) often cannot be estimated from paleodemographic data (Gage, 1988; Herrmann & Konigsberg, 2002), two versions of each model were estimated: a full model including all of the parameters and the covariate parameter, and a reduced model in which the age-independent component was set to 0.

A likelihood ratio test (LRT) was used to compare the fits of the models in which the value of the SES covariate was and was not estimated (i.e., the SES covariate was set to 0, making it an additional "reduced" model, as above). Thus, the LRT tests the null hypothesis that SES had no effect on risk of mortality (i.e., high SES was not associated with increased nor decreased risk of death). The LRT was computed using the following formula: $LRT = -2[\ln(L_{reduced}) - \ln(L_{full})]$, where LRT approximates a χ^2 distribution with df = 1. A *p*-value of less than 0.05 was considered statistically significant.

5.2.5 Gompertz Model

To determine the relationship between sex and risk of mortality (i.e., whether men and women faced similar risks of dying) in Industrial England, sex (i.e., females = 0, males = 1) was modeled as a covariate affecting the Gompertz-Makeham model of adult mortality:

$$h(a) = \alpha_1 + \alpha_2 e^{\beta a}$$

where a is age, α_1 is the risk of death that is constant with respect to age (i.e., the age-independent component), α_2 is the risk of death associated with senescence at the moment of birth, and β is the rate at which this risk increases with age (Gage, 1988). Only adults (aged 18+ years) for whom sex could be estimated were included in the Gompertz-Makeham hazard model analyses. Parameters were estimated using maximum likelihood analyses with the program *mle* (Holman, 2005). A positive estimate for the parameter representing the effect of the covariate would suggest males were at an increased risk of death compared to females, whereas a negative estimate for the parameter representing the effect of the covariate would suggest males were at a decreased risk of death compared to females.

To ensure any effects of the covariate were caused by sex-based differences in risk rather than geographic location, the Gompertz-Makeham model was applied to each skeletal sample separately (St. Bride's Fleet Street, n = 208; Coach Lane, n = 136; St. Peter's Wolverhampton, n = 69; New Bunhill Fields, n = 124), as well as to a combined sample composed of individuals from the three low-SES sites (St. Peter's Wolverhampton, Coach Lane, and New Bunhill Fields, n = 329). For the reasons detailed above for the Siler model, two versions of each model were estimated: a full model

including all three Gompertz-Makeham parameters and the covariate parameter, and a reduced model in which the age-independent component was set to 0 (which eliminates the effect of the age-independent component and reduces the model to a Gompertz model). Finally, as in the Siler hazard analyses described above, a likelihood ratio test (LRT) was used to compare the fits of the Gompertz models in which the value of the sex covariate was and was not estimated (i.e., the sex covariate was set to 0 in the reduced version). In this case, the LRT tests the null hypothesis that sex had no effect on risk of mortality (i.e., male sex was not associated with increased nor decreased risk of death). As in the Siler analyses, a *p*-value of less than 0.05 was considered statistically significant.

5.3 Results

In every analysis, at least one full model (i.e., the five-parameter Siler model and the Gompertz-Makeham model) did not yield good estimates of the age-independent component (i.e., α_1 in the Gompertz-Makeham model and α_2 in the Siler model). Consequently, the results for the reduced models (i.e., the models in which the age-independent component is set to 0) are presented below.

Table 5.2 shows the estimated values of the parameter representing the effect of SES on the risk of mortality, the 95% confidence intervals for the estimates, and the associated likelihood ratio tests for the combined (all four sites) and London (London sites only) samples. In both cases, the negative value of the parameter indicates that people of higher SES were at reduced risk of death compared to people in lower-SES groups. The results of the likelihood ratio tests indicate that, in both the combined and the

Table 5.2 Maximum likelihood estimate of the effect of the socioeconomic status (SES) covariate on risk of mortality for the combined and London samples, the associated 95% confidence interval (CI), and results of the likelihood ratio test (LRT).

Sample	Covariate effect (95% CI)	LRT	*p*
Combined sample (all sites)	-0.8439 (-1.065, -0.6380)	140.544	< 0.0001
London sample (London sites only)	-1.4487 (-1.670, -1.243)	305.494	< 0.0001

London samples, including the SES covariate improved the fit of the Siler model to the data.

Table 5.3 shows the estimated value of the parameter representing the effect of sex on the risk of mortality, the 95% confidence interval for the estimate, and the associated likelihood ratio test. The negative value of the parameter for the St. Bride's (higher-SES) sample indicates that males of higher SES were at reduced risk of death compared to females of higher SES. The result of the likelihood ratio test indicates that including the sex covariate improved the fit of the Gompertz model to the data. Although the estimated value of the parameter representing the effect of sex is also negative in the lower-SES samples (Coach Lane, New Bunhill Fields, St. Peter's Wolverhampton, and the combined low-SES sample), the associated confidence intervals span zero and the likelihood ratio tests indicate that including the sex covariate does not improve the fit of the Gompertz model in those instances.

5.4 Discussion

The Siler hazard analysis results indicate that SES significantly influenced risk of death, and that high-SES individuals were at reduced risk of death compared to low-SES individuals. The results of this study differ from that of DeWitte and colleagues (2016), who examined the effect of SES on risk of mortality in industrial London and found that high-status non-adults were at reduced risk of death compared to low-status non-adults but found no difference in risk of death between high- and low-status adults. One possible difference between the two studies is the skeletal samples analyzed: this study compared the high-status individuals from St. Bride's Fleet Street (London) with the low-

Table 5.3 Maximum likelihood estimates of the effect of the sex covariate on risk of mortality, the associated 95% confidence intervals (CI), and results of the likelihood ratio tests (LRT).

Sample	Covariate effect (95% CI)	LRT	*p*
St. Bride's Fleet Street (higher SES)	-0.3023 (-0.619, -0.0157)	4.6184	0.0316
Coach Lane	-0.1326 (-0.5196, 0.2103)	0.5836	0.4449
New Bunhill Fields	-0.2642 (-0.6884, 0.1075)	2.144	0.1431
St. Peter's Wolverhampton	-0.1558 (-0.744, 0.336)	0.4184	0.5177
Combined lower SES	-0.1788 (-0.4285, 0.0515)	2.602	0.1067

status individuals from New Bunhill Fields (London), Coach Lane (North Shields), and St. Peter's Collegiate Church (Wolverhampton), whereas DeWitte et al. (2016) compared the high-status individuals from St. Bride's Fleet Street (London) and Chelsea Old Church (from an affluent suburb of London) with the low-status individuals from St. Bride's Lower Churchyard (London). Another possible difference between the two studies that could have affected the results were the age estimates used. As mentioned above, this study used transition analysis to estimate adult age, thereby avoiding some of the issues associated with traditional age estimation methods. DeWitte et al.'s (2016) use of conventional age estimates may have obfuscated patterns of risk among adults in their sample. Therefore, this study provides support for a "pessimistic" view of living standards among the working class in 18th- and 19th-century England and suggests that the SES-mortality relationship observed in modern populations (Stringhini et al., 2017, 2014, 2010) also existed throughout Britain's industrialization process.

Phelan and colleagues (2004; 2010) argue that the SES-mortality association persists throughout time because SES allows select individuals to benefit from access to a variety of health-determining resources (e.g., money to afford better housing and a nutritious diet, knowledge about risk management or disease prevention, etc.) that affect multiple disease outcomes via multiple risk factors in a multiscalar fashion. For example, the connection between cardiovascular disease mortality and low SES in living populations is related to the combined effects of multiple risk factors at multiple scales (E. Chen & Miller, 2013), including violence at the neighborhood level (Sundquist et al., 2006), abusive or stressful family environments (Dong et al., 2004), and individual psychological risk factors like depression (J. W. Lynch, Kaplan, Cohen, Tuomilehto, &

Salonen, 1996). In a similar fashion, urban disamenities in industrializing England would have posed numerous health risks to low-status children and adults that were mitigated or avoided altogether by high-status individuals. One of the main problems associated with industrialization was the overcrowding generated by increased urbanization. By the mid-19th century, over half of the population of England lived in cities, which caused housing congestion and the propagation of infectious diseases and unsanitary living conditions in poorer areas (Schofield, 1994; Steckel & Floud, 2008; Williamson, 1994). Disease was common in the crowded living spaces inhabited by the working class, and cities like London regularly experienced epidemics of tuberculosis, influenza, measles, typhoid, scarlet fever, smallpox, and cholera (Woods & Woodward, 1984). Additionally, food access in industrial cities was largely limited to what could be bought in stores, and, when it could be afforded, bread and other food items purchased by the working class were of low quality, calorically deficient, and, in many cases, adulterated (Burnett & Burnett, 2013b; Shammas, 1984). Given that the relationship between malnutrition and infection is well documented in living populations (Katona & Katona-Apte, 2008; Scrimshaw et al., 1968), inadequate food access likely made the urban poor even more vulnerable to the diseases that already existed in their communities. Moreover, although the importance of sanitation and adequate sewage disposal was well understood and the technology for providing clean water to city dwellers existed, there was little improvement in the health-preserving aspects of urban infrastructures until the late 1840s, when doctors and poor law reformers called attention to the sanitation and hygiene issues and their connection to increased mortality among the lower-class (Hill, 1985; Morgan, 2002; Szreter, 2004). In contrast, many of the wealthy lived in far less crowded

conditions or could afford properties outside of urban centers, which offered a great degree of protection from the cramped, unsanitary, and disease-ridden environments relegated to the poor (Woods & Woodward, 1984).

The results of this study also suggest that the relationship between SES and mortality in industrializing England is not straightforward and the risks and benefits associated with different levels of SES were not evenly distributed in the population. Particularly, the Gompertz hazard analyses suggested that sex significantly influenced risk of death in the high-SES group, with high-SES males at reduced risk of death relative to high-SES females. Few studies have been conducted comparing the experiences and health outcomes of high-status women and high-status men in the industrial era, and thus inequalities between these two groups has been largely overlooked. Though upper-class women exhibited one privileged identity (i.e., high status) and may have benefitted from the classist structures of industrial England, they were still subject to other forms of structural oppression, such as sexism. Like their sisters in the working class, high-SES women could not vote, had limited access to an education, and did not have property rights—their properties were transferred to their husbands when they married (Hill, 1985). In addition, the social status of high-SES females may have worked against them: wealthy women in 18th- and 19th-century Britain were often at greater risk of mortality associated with childbirth compared to women in the working class. During the industrial period, wealthy women could afford to give birth in a hospital or at home and were often attended by a general practitioner, whereas working-class women could not afford to be seen by a physician and were most often attended by a midwife (Loudon, 1986b, 1986a). Although midwives in England were not required to undergo formal training until 1902,

historical data suggests that deliveries performed by midwives were safer than those performed by physicians, who were more likely to unnecessarily intervene in the birthing process using forceps that had not been properly sanitized, thus increasing the risk of puerperal sepsis, the main cause of maternal mortality at the time (De Brouwere, De Brouwere, Tonglet, & Van Lerberghe, 1998; Loudon, 1986a, 1986b, 2000). Consequently, the intersection of gender and class identities in industrializing England may have increased the risk of death during childbirth for women of high SES.

Additionally, the results of this study are similar to those found by Hughes-Morey (2012) in her study of high-status males and females from industrial-era London, in which high-status females exhibited higher hazard ratios (i.e., greater risk of death) associated with cribra orbitalia and porotic hyperostosis, two skeletal indicators that are often attributed to iron deficiency anemia in childhood (Patricia Stuart-Macadam, 1991; Patricia Stuart-Macadam & Kent, 1992; Patty Stuart-Macadam, 1985). In her study, Hughes-Morey (2012) suggests that upper-class women may have been at increased risk of multi-cause anemia produced by the combined effects of excessive alcohol consumption and lead poisoning. In modern populations, megaloblastic anemia is common in individuals who drink alcohol regularly, and is usually the result of folate deficiency (R. Green & Mitra, 2017; Savage & Lindenbaum, 1986). In 18th- and 19th-century England, high status women could afford to regularly drink port and other fortified wines imported from Spain and Portugal, which is now known to have had lead concentrations in excess of 1900 mg/L (Lessler, 1988; Nriagu, 1983). Lead poisoning contributes to anemia by decreasing heme synthesis, which decreases the lifespan and number of red blood cells, as well as the amount of hemoglobin per cell. The blood-lead

concentration typically associated with anemia (≥80 µg per 100 mL) is also the concentration at which clinical sources report permanent brain damage, convulsions, coma, and death, if left untreated (Chisolm, 1971). Consequently, repeated lead poisoning, combined with the effects of heavy alcohol consumption and the iron demands of pregnancy (Kalaivani, 2009), could have caused lethal cases of anemia and increased the risks of mortality in high-SES women relative to high-SES men or low-SES women.

Interestingly, the pattern of reduced male risk was also consistently observed in the low-SES groups, but the associated confidence intervals spanned zero and the LRTs were not statistically significant. This result suggests that low-SES females may have been at greater risk compared to low-SES males, but the difference was not as substantial as the difference in risk between high-SES females and males. It could be that, in low-SES settings, sex did not have as profound of a negative effect for women or protective effect for men. Given the historical evidence suggesting that the physical and mental effort associated with unpaid domestic labor—particularly if it was paired with the heavy burden of wage labor—was associated with stunting and excess mortality among industrial-era females (Harris, 2008; Horrell & Oxley, 2016; Humphries, 1991), it is unlikely that women did not experience negative health outcomes associated with their sex. Rather, the protective effects of being male may have been minimized in the working-class environment, preventing as significant of a difference in risk of mortality from developing between low-SES females and males as was observed in high-SES females and males.

5.5 Conclusion

The results of this study suggest that socioeconomic status (SES) affected risk of death in 18th- and 19th-century England. Particularly, individuals in the high-SES sample (the crypt assemblage associated with St. Bride's Fleet Street) were at reduced risk of death compared to individuals in the low-SES samples (the New Bunhill Fields, St. Peter's Wolverhampton, and Coach Lane skeletal assemblages). Increased risk of mortality among lower-class individuals is likely due to the overcrowded and unsanitary living conditions of cities in industrializing England, whereas upper-class individuals likely benefitted from better nutrition and hygiene, reduced risk of disease, and less-crowded living conditions. However, the benefits of high SES were unequally distributed among adults in the high-SES sample. High-SES males were at reduced risk of death compared to high-SES females, suggesting that men primarily benefitted from the protective aspects of high SES.

CHAPTER 6

CONCLUSION

6.1 Socioeconomic Status

One of the three objectives of this dissertation project was to determine how morbidity and mortality patterns in industrial England differed between socioeconomic status (SES) groups. Particularly, it was hypothesized that risks of mortality and stress marker frequencies would be higher in the lower-SES group than in the higher-SES group. Indeed, most of the samples that were analyzed indicated a relationship between low SES and earlier age at death. All the samples for the hierarchical log-linear analyses (i.e., the samples for periosteal lesions, cribra orbitalia, and linear enamel hypoplasia) exhibited associations between low SES and earlier ages at death, and the hazard analyses to determine the relationship between SES and risk of death indicated that low-SES individuals were at increased risk of mortality compared to high-SES individuals. Additionally, the presence of cribra orbitalia, small tooth crown sizes, and reduced anteroposterior (AP) neural canal diameters were associated with low SES, indicating that low-SES individuals were more likely to exhibit skeletal indicators of early childhood adversity compared to their high-status peers.

The results also suggest, however, that the relationship is more complex than high frequencies of skeletal indicators of stress being associated with low SES. In fact, in some cases, skeletal indicators of stress were associated with the privileged status group: small transverse (TR) neural canal diameters were found in the high-SES group,

particularly when the sample was limited to adults (18+ years of age); and increased craniofacial fluctuating asymmetry (FA) was associated with high-SES adults. Two interpretations—which are not necessarily mutually exclusive—fit the TR neural canal and FA data: (1) given that both FA and TR neural canal diameters develop throughout the prenatal, infant, childhood, and adolescent periods, it could be that high-SES individuals were more likely to experience adversity or physiological stress in the later non-adult period compared to low-SES individuals who, as summarized above, were more likely to exhibit skeletal indicators related to early childhood stress; (2) given that both the FA and TR samples were limited to individuals over the age of 15, it is possible that these data are the result of selective mortality, as described by Wood and colleagues (1992). That is, the second interpretation here suggests that—in agreement with other studies of industrial England (DeWitte et al., 2016; Hughes-Morey, 2016)—the high-SES individuals who exhibited increased FA and reduced TR neural canal diameters may have been relatively *less* frail than their low-SES counterparts who died before the age of 15 and thus were not included in the samples analyzed by this study. Therefore, the two interpretations provided above are not necessarily mutually exclusive, because the individuals surviving to exhibit certain skeletal indicators of stress in late adolescence and adulthood may be the more robust members of the low-SES group and the majority of the high-SES group, which is comparably more heterogenous in frailty. In these two cases, it was not that privilege allowed high-SES individuals to avoid adversity or physiological stress during adolescence, but that privilege and resource access may have enabled those individuals to survive to adulthood.

6.2 Intersectionality

The second objective of this dissertation project was to investigate how physiological stressors throughout life interacted with socially meaningful categories such as age and sex to produce layered marginalizations that influenced frailty and mortality in industrial England. In other words, a large part of this dissertation is devoted to developing an intersectional bioarchaeology and providing other scholars with a model of how intersectionality can be incorporated into bioarchaeological studies. Given the focus of the dissertation project on the effects of SES on patterns of health and mortality in industrializing England, it was hypothesized that risk of mortality would be higher for children and adult females than for adult males in each status group.

Although the hierarchical log-linear analyses consistently suggested that females died at younger ages than males, there was no evidence that multiply-marginalized adults (i.e., low-SES females) exhibited health outcomes that were drastically different than those exhibited by any other group. Instead, the two three-way interactions observed in both the full (all four sites) and reduced (London sites) samples (i.e., periosteal lesions significantly more common among men ages 30.0 – 44.9 years, and cribra orbitalia lesions significantly less common among high-SES females) were associated with "middle groups" (i.e., individuals exhibiting combinations of marginalized and privileged identities, such as low-SES males or high-SES females), as described by Sen and Iyer (2012). In accordance with intersectionality theory, it is the intersection—the mutually-constitutive action of *both* identities—that produced the health disparities observed. For example, in the 18th- and 19th-centuries, working-class women and children were gradually expelled from the industrial workforce, upper-class women were expected to

stay home and uphold the budding Victorian ideals of a domestic housewife, and upper-class men were employed in relatively safe occupations befitting their status (e.g., clerk or physician). In contrast, working-class men were almost exclusively employed in difficult and dangerous working conditions that put them at greater risk of injury and infection (i.e., the two primary causes of periosteal new bone formation), which, in turn, may have put them at increased risk of death in the middle ages.

Generally, the analyses of tooth size and vertebral neural canal size supported the hypothesis that women and children were at greater risk of exhibiting skeletal indicators of stress or earlier age at death compared to adult men. In the 2-way ANOVAs of tooth size (i.e., the only analyses to include a substantial number of non-adults, aside from the AP neural canal diameters, which were largely inconclusive or excluded from analysis), small tooth crowns were associated with non-adults in the low-SES group. This finding has additional implications for the developmental origins of health and disease hypothesis, discussed below; however, it also implies that children born into working-class families occupied a particularly marginalized position, and, as a result, were more likely to experience physiological stress in early childhood. Likewise, smaller teeth and reduced TR neural canal diameters were consistently associated with females in the 3-way ANOVAs of adult individuals, suggesting that females were more likely to have experienced early life adversity compared to males. The positions of women and children were likely influenced by intersecting cultural ideologies and structural processes operating in industrializing England, specifically the idea that, especially among the working class, resources should be preferentially allocated to those individuals who were able to contribute the most to the family's income, namely adult males.

The hazard analyses may also indicate an intersectional relationship, this time highlighting the multiply-privileged individuals in the study (i.e., high-SES males). In those analyses, high SES was associated with reduced risk of death (among both non-adult and adult individuals included in the Siler model), but only high-SES males exhibited a significantly reduced risk of death when SES groups were analyzed separately (among adult individuals included in the Gompertz model). Consequently, the results of the hazard analyses suggest that high-SES males benefitted from the privileges associated with male sex and high status, whereas other groups may have experienced hazards associated with their disadvantaged identities. Risks experienced by the men, women, and children of the working class were likely related to urban disamenities common in the industrial period, many of which were not improved or fixed until the 1840s (Hill, 1985; Morgan, 2002; Szreter, 2004). Likewise, high-SES females faced dangers specifically associated with being a wealthy housewife, such as drinking expensive wine saturated with lethal concentrations of lead or giving birth attended by an impatient and inexperienced male physician. The privileges and resources associated with high status and male sex may also explain the seemingly contradictory results of the FA analyses, which indicated that increased FA was associated with high-SES males. As mentioned above, this finding may be a product of selective mortality. Specifically, marginalized individuals who exhibited increased FA may have been relatively more frail and thus less likely to survive to adulthood compared to high-SES males, who had preferential access to resources and thus were able to survive to adulthood despite experiencing early life stress capable of producing craniofacial asymmetry. Therefore, the results of this dissertation project suggest that the intersections of status and gender identities, in

conjunction with classism and sexism at the structural level, affected patterns of health in industrial England. Specifically, the findings of this project indicate that high-SES males experienced reduced risk of death (during and likely prior to adulthood), enabling them to survive physiological stress in early life while their disadvantaged peers perished at younger ages.

6.3 The Developmental Origins of Health and Disease Hypothesis

The third and final objective of this dissertation project was to evaluate the potential of stressors underrepresented in biological anthropology literature to enhance our understanding of marginalization, intersectionality, and mortality in the past. Although this objective initially emphasized the need to increase the number of informative stress markers used by bioarchaeologists to assess health in the past, it eventually morphed into a study of how these diverse skeletal indicators of stress might produce novel understandings about how early life stress events influenced later life mortality in the past (i.e., the Developmental Origins of Health and Disease, or DOHaD, hypothesis). Particularly, it was hypothesized that stress markers reflecting active or early life stress events (i.e., linear enamel hypoplasia, fluctuating asymmetry, tooth size, vertebral neural canal size, and active periosteal new bone formation) will be more frequent in lower-SES group and those markers reflecting the accumulation of nonlethal stressors over a relatively long life (i.e., healed periosteal new bone formation) will be associated with the higher-SES group. Alternatively, these stress markers will be more strongly associated with earlier age-at-death in the lower-SES group, reflecting greater buffering against stress associated with higher SES. It is worth noting that portions of this

hypothesis were analyzed but have not been presented as a part of this dissertation (e.g., data on active vs. healed periosteal new bone formation), because of concerns regarding sample sizes and dissertation length. However, these omitted analyses provide fruitful areas for future research, as discussed below.

In the hierarchical analyses, cribra orbitalia was associated with the low-SES group (in the full sample containing all four sites) and younger age at death (in both the full sample and the reduced sample containing only the London sites), but linear enamel hypoplasia was not associated with SES or age at death. In the 2- and 3-way ANOVAs with tooth size as the dependent variable, small teeth were associated with low SES and earlier age at death in the low-SES group. In the 2- and 3-way ANOVAs with vertebral neural canal size as the dependent variable, small AP diameters were associated with low SES and earlier age at death in the low-SES group, but small TR diameters were associated with the high-SES group. Lastly, in the 3-way ANOVA with craniofacial FA as the dependent variable, increased FA was associated with the high-SES group and with earlier ages at death in the high-SES group. Generally, the results of this study support the hypothesis as constructed: half of the analyzed samples indicated that skeletal indicators of early life stress were associated with the low-SES group, and those same skeletal indicators were associated with earlier age at death, especially among low-SES individuals. These results suggest that individuals who experienced early life stress severe enough to cause cribrotic lesions or interfere with tooth crown development or AP neural canal diameter were more likely to die at younger ages in later life. Although bioarchaeologists cannot currently examine the specific early life physiological effects or later life disease outcomes associated with developmental stress, the results of this study,

in general, provide bioarchaeological support for the DOHaD hypothesis. Comparatively, the presence of linear enamel hypoplasia may not be particularly informative about early life stress in the context of industrializing England. Finally, as discussed above, the TR neural canal diameter and FA results may be indicative of adversity experienced primarily by the upper class during adolescence, or, more importantly, may highlight why bioarchaeologists should consider the implications of the Osteological Paradox (J. W. Wood et al., 1992), including the effects of selective mortality on patterns of pathology in skeletal samples.

6.4 Contributions

This dissertation project contributes new, bioarchaeological evidence to the study of industrialization in England, including findings about how various factors, such as sex and socioeconomic status, intersected to influence patterns of health among individuals living in the cities of London, Wolverhampton, and North Shields. Although historical records provide some information about people living in 18th- and 19th-century England, these records are often biased, inaccurate, and do not provide adequate descriptions of the health of the English population. Data on wages or earnings do not predict nutritional inadequacy or health as well as other, less-widely available historical data, such as household size (Gazeley & Horrell, 2013). Records of height and weight are often biased towards unrepresentative portions of the working class, such as convicts, army recruits, or agricultural laborers (e.g., Cinnirella, 2008; Meredith & Oxley, 2015; Nicholas & Oxley, 1993; Nicholas & Steckel, 1991). Even the British Census, first conducted in 1801, is not considered “reliable” or suitable for analysis until at least 1851 (Checkland,

1964; Richards, 1974). Large gaps and omissions are evident in historical sources, which brings their representativeness, accuracy, and usefulness into question. For example, many women are only identified as "occupied" or "unoccupied" in terms of employment in the 1851 census (Richards, 1974). Bioarchaeological studies, therefore, provide an opportunity to learn about the lives and experiences of these omitted individuals via analysis of their skeletal remains.

This study, in particular, challenges the notion that patterns of health (i.e., patterns of mortality and skeletal indicators of stress) were uniform within age, class, or sex categories. Studies of health in living populations have demonstrated that outcomes vary due to the effects of intersecting social identities and structural processes (Bauer, 2014; Bowleg, 2012) and early life experiences (Gluckman, Hanson, & Pinal, 2005), but relatively few bioarchaeological studies incorporate intersectionality theory or the DOHaD hypothesis into their analytical designs or interpretations. This project, in contrast, uses a suitable analytical technique (i.e., hierarchical log-linear analysis) to expose associations among multiple biological and social variables to investigate how intersectionality produced patterns of frailty and mortality in industrial-era England. Additionally, this research emphasizes the importance of studying "middle groups" who exhibit combinations of advantaged and disadvantaged identities and may not present with the same health outcomes as multiply-marginalized or multiply-privileged individuals. Indeed, the results of the periosteal lesion and cribra orbitalia analyses (Chapter 2) indicated that low-SES men and high-SES women exhibited drastically different health outcomes compared to other analyzed groups. Moreover, as addressed in Chapter 3, this project contributes to the growing body of bioarchaeological evidence in

support of the developmental origins of health and disease hypothesis (see, for example, Gowland, 2015; Temple, 2018), adding tooth size to the toolkit of skeletal indicators of stress that bioarchaeologists could use to examine the effects of early life adversity in the past. In sum, this study further develops the bioarchaeological literature in support of the DOHaD hypothesis, and theoretically contributes to the development of the discipline by presenting a unique model for scholars interested in studying intersectionality in the past.

6.5 Future Directions

As mentioned above, some of the analyses conducted for this study were excluded due to space restrictions and sample size concerns. Particularly, data were collected and analyzed regarding cribra orbitalia activity (i.e., absent, active, mixed, healed), periosteal lesion activity (i.e., absent, active, mixed, healed), number of linear enamel hypoplasias per individual, and earliest age at hypoplasia formation. However, due to the statistical requirements of hierarchical analysis (i.e., >80% of cells should contain 5 or more individuals), it is difficult to conduct crosstabulation analyses with small sample sizes and large numbers of variable categories (e.g., the four activity categories listed above compared to the two categories required for the presence vs. absence analyses). Thus, these analyses were excluded from the final version of the dissertation presented here but would be a potentially fruitful area for future research if sample sizes could be increased. Specifically, the sample from New Bunhill Fields (n = 535) that was analyzed by this study was only a fraction of the individuals who were excavated from the burial ground, so additional data on low-SES individuals from London are available yet not incorporated into this study.

The methods used in this study to address the ways in which intersectionality impacted patterns of frailty and mortality in industrializing England could be applied to other contexts, such as other industrializing contexts or other periods of transition more generally. Bioarchaeological and paleodemographic methods could also be used to examine the effects of intersectionality in 20th- and 21st-century skeletal collections in regions that are understudied and underrepresented in the intersectional literature (e.g., the Khon Kaen University skeletal collection, which could be used to examine patterns of health and mortality in 20th-century southeast Asia). Additional bioarchaeological studies of intersectionality could generate new information about the production and maintenance of social inequalities and health disparities throughout time and across cultures, thus forging connections between bioarchaeological studies of intersectionality in the past and research on living populations currently being conducted by other disciplines.

REFERENCES

Adams, J. L. (2007). “Out of Darkness, Cometh Light”: Life and Death in Nineteenth-century Wolverhampton: Excavation of the Overflow Burial Ground of St Peter’s Collegiate Church, Wolverhampton, 2001-2002. Archaeopress.

Adelstein, A. M. (1980). Life-style in occupational cancer. Journal of Toxicology and Environmental Health, Part A Current Issues, 6(5–6), 953–962.

Adler, N. E., Boyce, T., Chesney, M. A., Cohen, S., Folkman, S., Kahn, R. L., & Syme, S. L. (1994). Socioeconomic status and health: the challenge of the gradient. American Psychologist, 49(1), 15.

Adler, N. E., & Ostrove, J. M. (1999). Socioeconomic status and health: what we know and what we don’t. Annals of the New York Academy of Sciences, 896, 3–15.

Agarwal, S. C. (2016). Bone morphologies and histories: Life course approaches in bioarchaeology: BONE MORPHOLOGIES AND HISTORIES. American Journal of Physical Anthropology, 159, 130–149. https://doi.org/10.1002/ajpa.22905

Albert, A. M., & Greene, D. L. (1999). Bilateral asymmetry in skeletal growth and maturation as an indicator of environmental stress. American Journal of Physical Anthropology, 110(3), 341–349. https://doi.org/10.1002/(SICI)1096-8644(199911)110:3<341::AID-AJPA6>3.0.CO;2-8

Amoroso, A., Garcia, S. J., & Cardoso, H. F. V. (2014). Age at death and linear enamel hypoplasias: Testing the effects of childhood stress and adult socioeconomic circumstances in premature mortality. American Journal of Human Biology, 26(4), 461–468. https://doi.org/10.1002/ajhb.22547

Angel, J. L. (1984). Health as a crucial factor in the changes from hunting to developed farming in the eastern Mediterranean. Paleopathology at the Origins of Agriculture, 51–74.

Antonovsky, A. (1967). Social class, life expectancy and overall mortality. The Milbank Memorial Fund Quarterly, 45(2), 31–73.

Armelagos, G. J., Goodman, A. H., Harper, K. N., & Blakey, M. L. (2009). Enamel hypoplasia and early mortality: Bioarcheological support for the Barker hypothesis. Evolutionary Anthropology: Issues, News, and Reviews, 18(6), 261–271. https://doi.org/10.1002/evan.20239

Atkins, P. J. (1992). White Poison? The Social Consequences of Milk Consumption, 1850–1930. Social History of Medicine, 5(2), 207–227. https://doi.org/10.1093/shm/5.2.207

Avitsur, R., Hunzeker, J., & Sheridan, J. F. (2006). Role of early stress in the individual differences in host response to viral infection. Brain, Behavior, and Immunity, 20(4), 339–348.

Azad, M. B., Lissitsyn, Y., Miller, G. E., Becker, A. B., HayGlass, K. T., & Kozyrskyj, A. L. (2012). Influence of socioeconomic status trajectories on innate immune responsiveness in children. PloS One, 7(6), e38669.

Babbitt, G. A., Kiltie, R., & Bolker, B. (2006). Are Fluctuating Asymmetry Studies Adequately Sampled? Implications of a New Model for Size Distribution. The American Naturalist, 167(2), 230–245. https://doi.org/10.1086/498621

Bailey, A. H., & Day, A. (1863). On the rate of mortality prevailing amongst the families of the peerage during the nineteenth century. Journal of the Statistical Society of London, 26(1), 49–71.

Barker, D. J. (2007). The origins of the developmental origins theory. Journal of Internal Medicine, 261(5), 412–417.

Barker, D. J., Godfrey, K. M., Gluckman, P. D., Harding, J. E., Owens, J. A., & Robinson, J. S. (1993). Fetal nutrition and cardiovascular disease in adult life. The Lancet, 341(8850), 938–941.

Barker, D. J., & Osmond, C. (1986). Infant mortality, childhood nutrition, and ischaemic heart disease in England and Wales. The Lancet, 327(8489), 1077–1081.

Barker, D. J., Osmond, C., Winter, P. D., Margetts, B., & Simmonds, S. J. (1989). Weight in infancy and death from ischaemic heart disease. The Lancet, 334(8663), 577–580.

Bastir, M., Rosas, A., & O'Higgins, P. (2006). Craniofacial levels and the morphological maturation of the human skull. Journal of Anatomy, 209(5), 637–654.

Bauer, G. R. (2014). Incorporating intersectionality theory into population health research methodology: challenges and the potential to advance health equity. Social Science & Medicine (1982), 110, 10–17. https://doi.org/10.1016/j.socscimed.2014.03.022

Beaglehole, R. (1990). International trends in coronary heart disease mortality morbidity and risk factors. Epidemiologic Reviews, 12, 1–15.

Bengtsson, T., & Dribe, M. (2011). The late emergence of socioeconomic mortality differentials: A micro-level study of adult mortality in southern Sweden 1815–1968. Explorations in Economic History, 48(3), 389–400. https://doi.org/10.1016/j.eeh.2011.05.005

Bengtsson, T., & van Poppel, F. (2011). Socioeconomic inequalities in death from past to present: An introduction. Explorations in Economic History, 48(3), 343–356. https://doi.org/10.1016/j.eeh.2011.05.004

Bigoni, L., Krajíček, V., Sládek, V., Velemínský, P., & Velemínská, J. (2013). Skull shape asymmetry and the socioeconomic structure of an early medieval central european society. American Journal of Physical Anthropology, 150(3), 349–364. https://doi.org/10.1002/ajpa.22210

Boberg-Fazlic, N., Sharp, P., & Weisdorf, J. (2011). Survival of the richest? Social status, fertility and social mobility in England 1541-1824. European Review of Economic History, 15(3), 365–392.

Boellstorff, T. (2007). Queer studies in the house of anthropology. Annu. Rev. Anthropol., 36, 17–35.

Boldsen, Jepser L., Milner, G. R., Konigsberg, L. W., & Wood, J. W. (2002). Transition analysis: A new method for estimating age from skeletons. In R. D. Hoppa & J. W. Vaupel (Eds.), Paleodemography: Age distributions from skeletal samples (pp. 73–106). Cambridge: Cambridge University Press.

Boldsen, Jesper L. (2007). Early childhood stress and adult age mortality—A study of dental enamel hypoplasia in the medieval Danish village of Tirup. American Journal of Physical Anthropology, 132(1), 59–66. https://doi.org/10.1002/ajpa.20467

Bookstein, F. L. (1997). Morphometric Tools for Landmark Data: Geometry and Biology. Cambridge University Press.

Bowleg, L. (2012). The problem with the phrase women and minorities: intersectionality-an important theoretical framework for public health. American Journal of Public Health, 102(7), 1267–1273. https://doi.org/10.2105/AJPH.2012.300750

Braveman, P. A., Cubbin, C., Egerter, S., Chideya, S., Marchi, K. S., Metzler, M., & Posner, S. (2005). Socioeconomic status in health research: one size does not fit all. Jama, 294(22), 2879–2888.

Brickley, M., & Miles, A. (1999). The Cross Bones Burial Ground, Redcross Way, Southwark, London: Archaeological Excavations (1991-1998) for the London Underground Limited Jubilee Line Extension Project. MoLAS Monograph 3. London: Museum of London.

Brotman, S., Ryan, B., & Cormier, R. (2003). The health and social service needs of gay and lesbian elders and their families in Canada. The Gerontologist, 43(2), 192–202.

Buer, M. C. (2013). Health, wealth and population in the early days of the industrial revolution. Routledge.

Buikstra, J. E., & Ubelaker, D. H. (Eds.). (1994b). Standards for data collection from human skeletal remains: Proceedings of a seminar at the Field Museum of Natural History (Arkansas Archaeology Research Series 44). Fayetteville, AR: Arkansas Archeological Survey Press.

Bullard, R. D., & Wright, B. H. (1993). Environmental justice for all: community perspectives on health and research. Toxicology and Industrial Health, 9(5), 821–841.

Bunker, C. H., Ukoli, F. A., Nwankwo, M. U., Omene, J. A., Currier, G. W., Holifield-Kennedy, L., … Kuller, L. H. (1992). Factors associated with hypertension in Nigerian civil servants. Preventive Medicine, 21(6), 710–722.

Burnett, P. J., & Burnett, J. (2013a). Destiny Obscure: Autobiographies of Childhood, Education and Family from the 1820s to the 1920s. Routledge.

Burnett, P. J., & Burnett, J. (2013b). Plenty and Want: a social history of food in England from 1815 to the present day. Routledge.

Butler, P. M. (1939). Studies of the Mammalian Dentition.–Differentiation of the Post-canine Dentition. In Proceedings of the Zoological Society of London (Vol. 109, pp. 1–36). Wiley Online Library.

Bythell, D. (1993). Women in the Workforce. The Industrial Revolution and British Society, 31–53.

Calderon, R. L., Johnson Jr, C. C., Craun, G. F., Dufour, A. P., Karlin, R. J., Sinks, T., & Valentine, J. L. (1993). Health risks from contaminated water: Do class and race matter? Toxicology and Industrial Health, 9(5), 879–900.

Cantwell, M. F., Mckenna, M. T., Mccray, E., & Onorato, I. M. (1998). Tuberculosis and race/ethnicity in the United States: impact of socioeconomic status. American Journal of Respiratory and Critical Care Medicine, 157(4), 1016–1020.

Carnon, A. G., Ssemwogerere, A., Lamont, D. W., Hole, D. J., Mallon, E. A., George, W. D., & Gillis, G. R. (1994). Relation between socioeconomic deprivation and pathological prognostic factors in women with breast cancer. BMJ: British Medical Journal, 309(6961), 1054.

Cavigelli, S. A., & Chaudhry, H. S. (2012). Social status, glucocorticoids, immune function, and health: can animal studies help us understand human socioeconomic-status-related health disparities? Hormones and Behavior, 62(3), 295–313. https://doi.org/10.1016/j.yhbeh.2012.07.006

Chadwick, E. (1842). Report on the sanitary condition of the labouring population og Great: Britain: supplementary report on the results of special inquiry into the practice of interment in towns (Vol. 1). HM Stationery Office.

Checkland, S. G. (1964). The rise of industrial society in England, 1815-1885. Addison-Wesley Longman Ltd.

Chen, E. M., Masih, S., Chow, K., Matcuk, G., & Patel, D. (2012). Periosteal Reaction: Review of Various Patterns Associated With Specific Pathology. Contemporary Diagnostic Radiology, 35(17), 6. https://doi.org/10.1097/01.CDR.0000418465.87546.2d

Chen, E., & Miller, G. E. (2013). Socioeconomic Status and Health: Mediating and Moderating Factors. Annual Review of Clinical Psychology, 9, 723–749. https://doi.org/10.1146/annurev-clinpsy-050212-185634

Chisolm, J. J. (1971). Lead poisoning. Scientific American, 224(2), 15–23.

Cinnirella, F. (2008). Optimists or pessimists? A reconsideration of nutritional status in Britain, 1740–1865. European Review of Economic History, 12(3), 325–354.

Claes, P., Walters, M., Shriver, M. D., Puts, D., Gibson, G., Clement, J., … Suetens, P. (2012). Sexual dimorphism in multiple aspects of 3D facial symmetry and asymmetry defined by spatially dense geometric morphometrics. Journal of Anatomy, 221(2), 97–114.

Clark, G. (2001). Farm wages and living standards in the industrial revolution: England, 1670–1869. The Economic History Review, 54(3), 477–505.

Clark, G. (2005). The Condition of the Working Class in England, 1209-2004. Journal of Political Economy, 113(6), 1307–1340. https://doi.org/10.1086/498123

Clark, G. (2007). The long march of history: Farm wages, population, and economic growth, England 1209–1869 1. The Economic History Review, 60(1), 97–135.

Clark, G. A., Hall, N. R., Armelagos, G. J., Borkan, G. A., Panjabi, M. M., & Wetzel, F. T. (1986). Poor growth prior to early childhood: Decreased health and life-span in the adult. American Journal of Physical Anthropology, 70(2), 145–160.

Clark, G., & Cummins, N. (2015). Malthus to modernity: wealth, status, and fertility in England, 1500–1879. Journal of Population Economics, 28(1), 3–29.

Clark, G., Huberman, M., & Lindert, P. H. (1995). A British Food Puzzle, 1770-1850. The Economic History Review, 48(2), 215–237. https://doi.org/10.2307/2598401

Cohen, H. W. (2011). P Values: Use and Misuse in Medical Literature. American Journal of Hypertension, 24(1), 18–23.

Cohen, M. N. (1994). The osteological paradox reconsidered. Current Anthropology, 35(5), 629–631.

Cohen, S., Doyle, W. J., Turner, R. B., Alper, C. M., & Skoner, D. P. (2004). Childhood socioeconomic status and host resistance to infectious illness in adulthood. Psychosomatic Medicine, 66(4), 553–558.

Collins, P. H. (2015). Intersectionality's Definitional Dilemmas. Annual Review of Sociology, 41(1), 1–20. https://doi.org/10.1146/annurev-soc-073014-112142

Collins, P. H., & Bilge, S. (2016). Intersectionality. John Wiley & Sons.

Conkey, M. W. (2005). Dwelling at the margins, action at the intersection? Feminist and indigenous archaeologies, 2005. Archaeologies, 1(1), 9–59.

Cook, D. C., & Buikstra, J. E. (1979). Health and differential survival in prehistoric populations: prenatal dental defects. American Journal of Physical Anthropology, 51(4), 649–664. https://doi.org/10.1002/ajpa.1330510415

Crenshaw, K. (1989). Demarginalizing the Intersection of Race and Sex: A Black Feminist Critique of Antidiscrimination Doctrine, Feminist Theory and Antiracist Politics. University of Chicago Legal Forum, 1(1, Article 8), 139–167.

Crenshaw, K. (1991). Mapping the Margins: Intersectionality, Identity Politics, and Violence Against Women of Color. Stanford Law Review, 43(6), 1241–1299.

Cucina, A., & Tiesler, V. (2003). Dental caries and antemortem tooth loss in the Northern Peten area, Mexico: a biocultural perspective on social status differences among the Classic Maya. American Journal of Physical Anthropology: The Official Publication of the American Association of Physical Anthropologists, 122(1), 1–10.

Cunningham, A. (1995). Breastfeeding: Adaptive behavior for child health and longevity. In P Stuart-Macadam & K. Dettwyker (Eds.), Breastfeeding: biocultural perspectives (pp. 243–264). Hawthorne, NY: Aldine De Gruyter.

Currie, J., & Stabile, M. (2002). Socioeconomic Status and Health: Why Is the Relationship Stronger for Older Children? NBER Working Paper.

Dahlberg, A. A. (1945). The changing of dentition of man. JADA, 32, 676–690.

Dandelion, P. (2008). The Quakers: A very short introduction (Vol. 177). Oxford University Press.

De Brouwere, V., De Brouwere, V., Tonglet, R., & Van Lerberghe, W. (1998). Strategies for reducing maternal mortality in developing countries: what can we learn from the history of the industrialized West? Tropical Medicine & International Health, 3(10), 771–782.

de la Cova, C. (2011). Race, health, and disease in 19th-century-born males. American Journal of Physical Anthropology, 144(4), 526–537.

DeLeon, V. B. (2007). Fluctuating asymmetry and stress in a medieval Nubian population. American Journal of Physical Anthropology, 132(4), 520–534. https://doi.org/10.1002/ajpa.20549

DeWitte, S. N. (2014a). Differential survival among individuals with active and healed periosteal new bone formation. International Journal of Paleopathology, 7, 38–44. https://doi.org/10.1016/j.ijpp.2014.06.001

DeWitte, S. N. (2014b). Health in post-black death London (1350–1538): Age patterns of periosteal new bone formation in a post-epidemic population. American Journal of Physical Anthropology, 155(2), 260–267. https://doi.org/10.1002/ajpa.22510

DeWitte, S. N. (2014c). Modeling the second epidemiological transition in London: Patterns of mortality and frailty during industrialization. In M. Zuckerman (Ed.), Moving the Middle to the Foreground: Revisiting the Second Epidemiological Transition. (pp. 35–54). Hoboken: Wiley-Blackwell.

DeWitte, S. N., & Bekvalac, J. (2011). The association between periodontal disease and periosteal lesions in the St. Mary Graces cemetery, London, England A.D. 1350-1538. American Journal of Physical Anthropology, 146(4), 609–618. https://doi.org/10.1002/ajpa.21622

DeWitte, S. N., Hughes-Morey, G., Bekvalac, J., & Karsten, J. (2016). Wealth, health and frailty in industrial-era London. Annals of Human Biology, 43(3), 241–254. https://doi.org/10.3109/03014460.2015.1020873

DeWitte, S. N., & Stojanowski, C. M. (2015). The Osteological Paradox 20 Years Later: Past Perspectives, Future Directions. Journal of Archaeological Research, 23(4), 397–450. https://doi.org/10.1007/s10814-015-9084-1

DeWitte, S. N., & Wood, J. W. (2008). Selectivity of Black Death mortality with respect to preexisting health. Proceedings of the National Academy of Sciences of the United States of America, 105(5), 1436–1441. https://doi.org/10.1073/pnas.0705460105

Dong, M., Giles, W. H., Felitti, V. J., Dube, S. R., Williams, J. E., Chapman, D. P., & Anda, R. F. (2004). Insights into causal pathways for ischemic heart disease: adverse childhood experiences study. Circulation, 110(13), 1761–1766.

Dressler, W. W. (1995). Modeling biocultural interactions: examples from studies of stress and cardiovascular disease. American Journal of Physical Anthropology, 38(S21), 27–56.

Dufour, D. L. (2006). Biocultural approaches in human biology. American Journal of Human Biology, 18(1), 1–9.

Duray, S. M. (1996). Dental indicators of stress and reduced age at death in prehistoric Native Americans. American Journal of Physical Anthropology, 99(2), 275–286. https://doi.org/10.1002/(SICI)1096-8644(199602)99:2<275::AID-AJPA5>3.0.CO;2-Y

Edvinsson, S., & Broström, G. (2012). Old age, health, and social inequality: Exploring the social patterns of mortality in 19th century northern Sweden. Demographic Research, 26, 633–660.

Engels, F. (2005). The condition of the working class in England. In The Sociology and Politics of Health (pp. 22–27). Routledge.

Entringer, S., Buss, C., & Wadhwa, P. D. (2010). Prenatal stress and developmental programming of human health and disease risk: concepts and integration of empirical findings. Current Opinion in Endocrinology, Diabetes, and Obesity, 17(6), 507.

Ercan, I., Ozdemir, S. T., Etoz, A., Sigirli, D., Tubbs, R. S., Loukas, M., & Guney, I. (2008). Facial asymmetry in young healthy subjects evaluated by statistical shape analysis. Journal of Anatomy, 213(6), 663–669.

Evans, G. W. (2004). The environment of childhood poverty. American Psychologist, 59(2), 77.

Evans, J., Stoodley, N., & Chenery, C. (2006). A strontium and oxygen isotope assessment of a possible fourth century immigrant population in a Hampshire cemetery, southern England. Journal of Archaeological Science, 33(2), 265–272.

Fahlander, F. (2012). Facing gender. Corporeality, materiality, intersectionality and resurrection (pp. 137–152). Stockholm University. Retrieved from http://www.diva-portal.org/smash/record.jsf?pid=diva2:572151 files/3748/record.html

Farkas, L. G., & Cheung, G. (1981). Facial asymmetry in healthy North American Caucasians: an anthropometrical study. The Angle Orthodontist, 51(1), 70–77.

Feinstein, C. H. (1998). Pessimism perpetuated: real wages and the standard of living in Britain during and after the industrial revolution. The Journal of Economic History, 58(3), 625–658.

Feinstein, J. S. (1993). The relationship between socioeconomic status and health: a review of the literature. The Milbank Quarterly, 279–322.

Ferrario, V. F., Sforza, C., Miani Jr, A., & Serrao, G. (1995). A three-dimensional evaluation of human facial asymmetry. Journal of Anatomy, 186(Pt 1), 103.

Ferrer, J. F., Jonsson, C. B., Esteban, E., Galligan, D., Basombrio, M. A., Peralta-Ramos, M., … Segovia, A. (1998). High prevalence of hantavirus infection in Indian communities of the Paraguayan and Argentinean Gran Chaco. The American Journal of Tropical Medicine and Hygiene, 59(3), 438–444.

Fife, D., & Mode, C. (1992). AIDS incidence and income. Journal of Acquired Immune Deficiency Syndromes, 5(11), 1105–1110.

Fildes, V. (1988). The English wet-nurse and her role in infant care 1538-1800. Medical History, 32(2), 142–173.

Fildes, V. (1995). The culture and biology of breastfeeding: an historical review of Western Europe. In P Stuart-Macadam & K. Dettwyker (Eds.), Breastfeeding: biocultural perspectives (pp. 101–126). Hawthorne, NY: Aldine De Gruyter.

Fildes, Valerie. (1986). Breasts, bottles and babies-a history of infant feeding. Edinburgh University Press.

Floud, R., Wachter, K. W., & Gregory, A. (1990). Height, health and history: nutritional status in the United Kingdom, 1750-1980. Cambridge: Cambridge University Press.

Franklin, M. (2001). A Black feminist-inspired archaeology? Journal of Social Archaeology, 1(1), 108–125.

Gage, T. B. (1988). Mathematical hazard models of mortality: an alternative to model life tables. American Journal of Physical Anthropology, 76(4), 429–441.

Gage, T. B. (2005). Are modern environments really bad for us?: revisiting the demographic and epidemiologic transitions. Am J Phys Anthropol, Suppl 41, 96–117.

Gagnon, A., & Bohnert, N. (2012). Early life socioeconomic conditions in rural areas and old-age mortality in twentieth-century Quebec. Social Science & Medicine, 75(8), 1497–1504.

Galloway, A., Willey, P., & Snyder, L. (1997). Human bone mineral densities and survival of bone elements: a contemporary sample. In W. D. Haglund & M. H. Sorg (Eds.), Forensic taphonomy: the postmortem fate of human remains (pp. 295–317). Boca Raton, FL: CRC Press, LLC.

Galobardes, B., Lynch, J. W., & Davey Smith, G. (2004). Childhood socioeconomic circumstances and cause-specific mortality in adulthood: systematic review and interpretation. Epidemiologic Reviews, 26(1), 7–21.

Galobardes, B., Smith, G. D., & Lynch, J. W. (2006). Systematic review of the influence of childhood socioeconomic circumstances on risk for cardiovascular disease in adulthood. Annals of Epidemiology, 16(2), 91–104.

Garn, S. M., Osborne, R. H., & McCabe, K. D. (1979). The effect of prenatal factors on crown dimensions. American Journal of Physical Anthropology, 51(4), 665–677. https://doi.org/10.1002/ajpa.1330510416

Gawlikowska, A., Szczurowski, J., Czerwiński, F., Miklaszewska, D., Adamiec, E., & Dzięciołowska, E. (2007). The fluctuating asymmetry of mediaeval and modern human skulls. HOMO, 58(2), 159–172. https://doi.org/10.1016/j.jchb.2006.10.001

Gazeley, I., & Horrell, S. (2013). Nutrition in the E nglish agricultural labourer's household over the course of the long nineteenth century. The Economic History Review, 66(3), 757–784.

Geronimus, A. T. (2013). Deep integration: Letting the epigenome out of the bottle without losing sight of the structural origins of population health. American Journal of Public Health, 103(S1), S56–S63.

Gluckman, P. D., Cutfield, W., Hofman, P., & Hanson, M. A. (2005). The fetal, neonatal, and infant environments—the long-term consequences for disease risk. Early Human Development, 81(1), 51–59.

Gluckman, P. D., Hanson, M. A., Cooper, C., & Thornburg, K. L. (2008). Effect of in utero and early-life conditions on adult health and disease. New England Journal of Medicine, 359(1), 61–73.

Gluckman, P. D., Hanson, M. A., & Pinal, C. (2005). The developmental origins of adult disease. Maternal & Child Nutrition, 1(3), 130–141.

Goodman, A. H., & Armelagos, G. J. (1985a). Factors affecting the distribution of enamel hypoplasias within the human permanent dentition. Am J Phys Anthropol, 68(4), 479–493.

Goodman, A. H., & Armelagos, G. J. (1985b). The chronological distribution of enamel hypoplasia in human permanent incisor and canine teeth. Arch Oral Biol, 30(6), 503–507.

Goodman, A. H., Armelagos, G. J., & Rose, J. C. (1980). Enamel hypoplasias as indicators of stress in three prehistoric populations from Illinois. Hum Biol, 52(3), 515–528.

Goodman, A. H., & Rose, J. C. (1990). Assessment of systemic physiological perturbations from dental enamel hypoplasias and associated histological structures. Yearbook of Physical Anthropology, 33, 59–110.

Goodman, Alan H., & Armelagos, G. J. (1988). Childhood Stress and Decreased Longevity in a Prehistoric Population. American Anthropologist, 90(4), 936–944. https://doi.org/10.1525/aa.1988.90.4.02a00120

Goodman, Alan H., & Leatherman, T. L. (1998). Building a New Biocultural Synthesis: Political-Economic Perspectives on Human Biology. Ann Arbor: University of Michigan Press.

Goodman, S. N. (1999). Toward Evidence-Based Medical Statistics. 1: The P Value Fallacy. Annals of Internal Medicine, 130, 995–1004.

Gowland, R. L. (2015). Entangled lives: Implications of the developmental origins of health and disease hypothesis for bioarchaeology and the life course. American Journal of Physical Anthropology, n/a-n/a. https://doi.org/10.1002/ajpa.22820

Grauer, A. L. (1993). Patterns of anemia and infection from medieval York, England. American Journal of Physical Anthropology, 91(2), 203–213. https://doi.org/10.1002/ajpa.1330910206

Green, J. A. (1988). Loglinear Analysis of Cross-Classified Ordinal Data: Applications in Developmental Research. Child Development, 59(1), 1–25.

Green, R., & Mitra, A. D. (2017). Megaloblastic anemias: nutritional and other causes. Medical Clinics, 101(2), 297–317.

Guagliardo, M. F. (1982). Tooth crown size differences between age groups: a possible new indicator of stress in skeletal samples. American Journal of Physical Anthropology, 58(4), 383–389. https://doi.org/10.1002/ajpa.1330580405

Guatelli-Steinberg, D., & Lukacs, J. R. (1999). Interpreting sex differences in enamel hypoplasia in human and non-human primates: Developmental, environmental, and cultural considerations. American Journal of Physical Anthropology, 110(S29), 73–126.

Haines, M. R., & Ferrie, J. P. (2011). Socioeconomic inequalities in death from past to present: A postscript. Explorations in Economic History, 48(3), 441–443. https://doi.org/10.1016/j.eeh.2011.06.003

Hallgrímsson, B. (1999). Ontogenetic patterning of skeletal fluctuating asymmetry in rhesus macaques and humans: evolutionary and developmental implications. International Journal of Primatology, 20(1), 121–151.

Hancock, A.-M. (2007). When Multiplication Doesn't Equal Quick Addition: Examining Intersectionality as a Research Paradigm. Perspectives on Politics, 5(1), 63–79. https://doi.org/10.1017/S1537592707070065

Hankivsky, O., Reid, C., Cormier, R., Varcoe, C., Clark, N., Benoit, C., & Brotman, S. (2010). Exploring the promises of intersectionality for advancing women's health research. International Journal for Equity in Health, 9, 5. https://doi.org/10.1186/1475-9276-9-5

Hanson, M. A., & Gluckman, P. D. (2008). Developmental origins of health and disease: new insights. Basic & Clinical Pharmacology & Toxicology, 102(2), 90–93.

Harris, B. (2008). Gender, health, and welfare in England and Wales since industrialisation. In Research in Economic History (pp. 157–204). Emerald Group Publishing Limited.

Hassett, B. R. (2014). Missing defects? A comparison of microscopic and macroscopic approaches to identifying linear enamel hypoplasia. American Journal of Physical Anthropology, 153(3), 463–472. https://doi.org/10.1002/ajpa.22445

Hatch, J., & Willey, P. (1974). Stature and status in Dallas society. Tennessee Archaeology, 30, 107–131.

Haviland, W. A. (1967). Stature at Tikal, Guatemala: Implications for Ancient Maya Demography and Social Organization. American Antiquity, 32(3), 316–325.

Herrmann, N. P., & Konigsberg, L. W. (2002). A re-examination of the age-at-death distribution of Indian Knoll. In R. D. Hoppa & J. W. Vaupel (Eds.), Paleodemography : age distribution from skeletal samples. (pp. 243–257). Cambridge: Cambridge University Press.

Hertzman, C. (1999). The biological embedding of early experience and its effects on health in adulthood. Annals of the New York Academy of Sciences, 896(1), 85–95.

Hill, C. (1985). British Economic and Social History 1700-1982. London: Edward Arnold Publishers.

Hillson, S. (1996). Dental anthropology. Cambridge: Cambridge University Press.

Hinck, V. C., Clark, W. M., & Hopkins, C. E. (1966). NORMAL INTERPEDICULATE DISTANCES (MINIMUM AND MAXIMUM) IN CHILDREN AND ADULTS. American Journal of Roentgenology, 97(1), 141–153. https://doi.org/10.2214/ajr.97.1.141

Hinze, S. W., Lin, J., & Andersson, T. E. (2012). Can we capture the intersections? Older Black women, education, and health. Women's Health Issues: Official Publication of the Jacobs Institute of Women's Health, 22(1), e91-98. https://doi.org/10.1016/j.whi.2011.08.002

Holman, D. J. (2005). mle: A programming language for building likelihood models. Retrieved from http://faculty.washington.edu/djholman/mle/

hooks, bell. (1981). Ain't I a Woman Black Women and Feminism. Boston, MA: South End Press.

Hoover, K. C., & Matsumura, H. (2008). Temporal variation and interaction between nutritional and developmental instability in prehistoric Japanese populations. American Journal of Physical Anthropology, 137(4), 469–478. https://doi.org/10.1002/ajpa.20892

Horrell, S., Humphries, J., & Voth, H.-J. (1998). Stature and relative deprivation: fatherless children in early industrial Britain. Continuity and Change, 13(1), 73–115.

Horrell, S., Meredith, D., & Oxley, D. (2009). Measuring misery: Body mass, ageing and gender inequality in Victorian London. Explorations in Economic History, 46(1), 93–119.

Horrell, S., & Oxley, D. (2012). Bringing home the bacon? Regional nutrition, stature, and gender in the industrial revolution [1]: REGIONAL NUTRITION, STATURE, AND GENDER. The Economic History Review, 65(4), 1354–1379. https://doi.org/10.1111/j.1468-0289.2011.00642.x

Horrell, S., & Oxley, D. (2016). Gender bias in nineteenth-century England: Evidence from factory children. Economics & Human Biology, 22, 47–64.

Howells, W. (1973). Cranial Variation in Man: A Study by Multivariate Analysis of Patterns of Difference among Recent Human Populations. Peabody Museum of Archaeology and Ethnology, Harvard Univ. Retrieved from https://ci.nii.ac.jp/naid/10006682479/#cit

Huck, P. (1994). Infant mortality in nine industrial parishes in Northern England, 1813–1836. Population Studies, 48(3), 513–526.

Huck, P. (1995). Infant Mortality and Living Standards of English Workers During the Industrial Revolution. The Journal of Economic History, 55, 528–550.

Hughes-Morey, G. (2012). Body Size and Mortality in Medieval England. PhD Dissertation. Department of Anthropology. (PhD). University at Albany, SUNY.

Hughes-Morey, G. (2016). Interpreting adult stature in industrial London. American Journal of Physical Anthropology, 159(1), 126–134. https://doi.org/10.1002/ajpa.22840

Humphries, J. (1991). "Bread and a pennyworth of treacle": excess female mortality in England in the 1840s. Cambridge Journal of Economics, 15(4), 451–473.

Hunt, E. H. (1986). Industrialization and regional inequality: wages in Britain, 1760–1914. The Journal of Economic History, 46(4), 935–966.

Jackson, A., & Calder, P. (2004). Severe undernutrition and immunity. In M. Gershwin, P. Neste, & C. Keen (Eds.), Handbook of Nutrition and Immunity (pp. 71–92). Totowa, NJ: Humana Press.

Kalaivani, K. (2009). Prevalence & consequences of anaemia in pregnancy. Indian J Med Res, 130(5), 627–33.

Kaplan, G. A., & Keil, J. E. (1993). Socioeconomic factors and cardiovascular disease: a review of the literature. Circulation, 88(4), 1973–1998.

Katona, P., & Katona-Apte, J. (2008). The interaction between nutrition and infection. Clinical Infectious Diseases: An Official Publication of the Infectious Diseases Society of America, 46(10), 1582–1588. https://doi.org/10.1086/587658

Kelly-Irving, M., Lepage, B., Dedieu, D., Bartley, M., Blane, D., Grosclaude, P., … Delpierre, C. (2013). Adverse childhood experiences and premature all-cause mortality. European Journal of Epidemiology, 28(9), 721–734.

Kirby, P. (2013). Child Workers and Industrial Health in Britain, 1780-1850. Boydell & Brewer Ltd.

Kittleson, M. M., Meoni, L. A., Wang, N.-Y., Chu, A. Y., Ford, D. E., & Klag, M. J. (2006). Association of childhood socioeconomic status with subsequent coronary heart disease in physicians. Archives of Internal Medicine, 166(21), 2356–2361.

Kjellström, A. (2014). Interpreting Violence: A bioarchaeological perspective of violence from medieval central Sweden. In C. Knüsel & M. Smith (Eds.), The Routledge Handbook of the Bioarchaeology of Human Conflict (pp. 237–250). Abingdon, Oxon, UK: Routledge.

Klaus, H. D. (2014). Frontiers in the bioarchaeology of stress and disease: Cross-disciplinary perspectives from pathophysiology, human biology, and epidemiology. American Journal of Physical Anthropology, 155(2), 294–308. https://doi.org/10.1002/ajpa.22574

Klein, S. L. (2000a). Hormones and mating system affect sex and species differences in immune function among vertebrates. Behavioural Processes, 51(1), 149–166. https://doi.org/10.1016/S0376-6357(00)00125-X

Klein, S. L. (2000b). The effects of hormones on sex differences in infection: from genes to behavior. Neurosci Biobehav Rev, 24(6), 627–638.

Klein, S. L. (2004). Hormonal and immunological mechanisms mediating sex differences in parasite infection. Parasite Immunology, 26(6–7), 247–264.

Klein, S. L., Cernetich, A., Hilmer, S., Hoffman, E. P., Scott, A. L., & Glass, G. E. (2004). Differential expression of immunoregulatory genes in male and female Norway rats following infection with Seoul virus. Journal of Medical Virology, 74(1), 180–190.

Klein, S. L., & Huber, S. (2010). Sex differences in susceptibility to viral infection. In S. L. Klein & C. Roberts (Eds.), Sex Hormones and Immunity to Infection (pp. 93–122). Heidelberg: Springer.

Klingenberg, C. P. (2011). MorphoJ: an integrated software package for geometric morphometrics. Molecular Ecology Resources, 11(2), 353–357.

Klingenberg, C. P. (2015). Analyzing Fluctuating Asymmetry with Geometric Morphometrics: Concepts, Methods, and Applications. Symmetry, 7(2), 843–934. https://doi.org/10.3390/sym7020843

Klingenberg, C. P., & McIntyre, G. S. (1998). Geometric Morphometrics of Developmental Instability: Analyzing Patterns of Fluctuating Asymmetry with Procrustes Methods. Evolution, 52(5), 1363–1375. https://doi.org/10.1111/j.1558-5646.1998.tb02018.x

Knodel, J., & Kintner, H. (1977). The Impact of Breast Feeding Patterns on the Biometric Analysis of Infant Mortality. Demography, 14(4), 391–409. https://doi.org/10.2307/2060586

Kogevinas, M., & Porta, M. (1997). Socioeconomic differences in cancer survival: a review of the evidence. IARC Scientific Publications, 138(138), 177–206.

Komlos, J. (1998). Shrinking in a Growing Economy? The Mystery of Physical Stature during the Industrial Revolution. The Journal of Economic History, 58(3), 779–802.

Komlos, J. (2007). On English Pygmies and giants: the physical stature of English youth in the late 18th and early 19th centuries. Research in Economic History, 25, 149–168. https://doi.org/10.1016/s0363-3268(07)25003-7

Komlos, J., & Küchenhoff, H. (2012). The diminution of the physical stature of the English male population in the eighteenth century. Cliometrica, Journal of Historical Economics and Econometric History, 6(1), 45–62.

Lang, J. M., Rothman, K. J., & Cann, C. I. (1998). That Confounded P-Value. Epidemiology, 9, 7–8.

Larsen, C. S. (1983). Deciduous tooth size and subsistence change in prehistoric Georgia coast populations. Current Anthropology, 24(2), 225–226.

Larsen, C. S. (1995). Biological Changes in Human Populations with Agriculture. Annual Review of Anthropology, 24, 185–213.

Larsen, C. S. (1997a). Bioarchaeology: interpereting behavior from the human skeleton. New York, NY: Cambridge University Press.

Larsen, C. S. (1997b). Stress and deprivation during the years of growth and development and adulthood. In C. S. Larsen (Ed.), Bioarchaeology: Interpreting Behavior from the Human Skeleton (pp. 6–61). Cambridge: Cambridge University Press.

Lessler, M. A. (1988). Lead and lead poisoning from antiquity to modern times. Ohio Journal of Science, 88, 78–84.

Lewis, A. B., & Garn, S. M. (1960). The relationship between tooth formation and other maturational factors. The Angle Orthodontist, 30(2), 70–77.

Lewis, M. E. (2002). Impact of industrialization: Comparative study of child health in four sites from medieval and postmedieval England (A.D. 850-1859). American Journal of Physical Anthropology, 119(3), 211–223. https://doi.org/10.1002/ajpa.10126

Lewis, M. E., & Gowland, R. (2007). Brief and precarious lives: Infant mortality in contrasting sites from medieval and post-medieval England (AD 850-1859). American Journal of Physical Anthropology, 134(1), 117–129. https://doi.org/10.1002/ajpa.20643

Li, L., Power, C., Kelly, S., Kirschbaum, C., & Hertzman, C. (2007). Life-time socio-economic position and cortisol patterns in mid-life. Psychoneuroendocrinology, 32(7), 824–833.

Lindert, P. H., & Williamson, J. G. (1983). English workers' living standards during the industrial revolution: a new look. The Economic History Review, 36(1), 1–25.

Livshits, G., Davidi, L., Kobyliansky, E., Ben-Amitai, D., Levi, Y., Merlob, P., … Reynolds, J. F. (1988). Decreased developmental stability as assessed by fluctuating asymmetry of morphometric traits in preterm infants. American Journal of Medical Genetics, 29(4), 793–805. https://doi.org/10.1002/ajmg.1320290409

Logan, W. H., & Kronfeld, R. (1933). Development of the human jaws and surrounding structures from birth to the age of fifteen years. Journal of the American Dental Association, 20(3), 379–428.

Logan, W. P. (1982). Cancer mortality by occupation and social class 1851-1971.

Longman Marcellin, R., Bauer, G. R., & Scheim, A. I. (2013). Intersecting impacts of transphobia and racism on HIV risk among trans persons of colour in Ontario, Canada. Ethnicity and Inequalities in Health and Social Care, 6(4), 97–107. https://doi.org/10.1108/EIHSC-09-2013-0017

Longman Marcellin, R., R. Bauer, G., & I. Scheim, A. (2013). Intersecting impacts of transphobia and racism on HIV risk among trans persons of colour in Ontario, Canada. Ethnicity and Inequalities in Health and Social Care, 6(4), 97–107.

Loudon, I. (1986a). Deaths in childbed from the eighteenth century to 1935. Medical History, 30(1), 1–41.

Loudon, I. (1986b). Obstetric care, social class, and maternal mortality. British Medical Journal (Clinical Research Ed.), 293(6547), 606.

Loudon, I. (2000). Maternal mortality in the past and its relevance to developing countries today–. The American Journal of Clinical Nutrition, 72(1), 241S–246S.

Lynch, J. W., Kaplan, G. A., Cohen, R. D., Tuomilehto, J., & Salonen, J. T. (1996). Do cardiovascular risk factors explain the relation between socioeconomic status, risk of all-cause mortality, cardiovascular mortality, and acute myocardial infarction? American Journal of Epidemiology, 144(10), 934–942.

Lynch, S. M. (2006). Explaining life course and cohort variation in the relationship between education and health: the role of income. J Health Soc Behav, 47(4), 324–338.

Mant, M., & Roberts, C. (2015). Diet and Dental Caries in Post-Medieval London. International Journal of Historical Archaeology, 19(1), 188–207. https://doi.org/10.1007/s10761-014-0286-x

Marmot, M. (1992). Coronary heart disease: rise and fall of a modern epidemic. In Coronary Heart Disease Epidemiology (pp. 3–19). Oxford University Press, New York.

Marmot, M. (2005). The Status Syndrome: How Social Standing Affects Our Health and Longevity. London: Bloomsbury Publishing. Retrieved from http://www.amazon.com/The-Status-Syndrome-Standing-Longevity/dp/0805078541

Marmot, M., Shipley, M. J., & Rose, G. (1984). Inequalities in death—specific explanations of a general pattern? The Lancet, 323(8384), 1003–1006.

Martorell, R., & Habicht, J.-P. (1986). Growth in early childhood in developing countries. In F. Falkner & J. Tanner (Eds.), Human Growth. Volume 3. Methodology. Ecological, Genetic, and Nutritional Effects on Growth. Second Edition (pp. 241–262). New York: Plenum Press.

May, T. (1987). An economic and social history of Britain, 1760-1970. Longman.

Mays, S., Brickley, M., & Ives, R. (2008). Growth in an English population from the Industrial Revolution. American Journal of Physical Anthropology, 136(1), 85–92. https://doi.org/10.1002/ajpa.20780

Mays, S., Ives, R., & Brickley, M. (2009). The effects of socioeconomic status on endochondral and appositional bone growth, and acquisition of cortical bone in children from 19th century Birmingham, England. American Journal of Physical Anthropology, 140, 410–416.

McIlvaine, B. K. (2013). Implications of Reappraising the Iron-Deficiency Anemia Hypothesis. International Journal of Osteoarchaeology, 25(6), 997–1000.

Meredith, D., & Oxley, D. (2015). Blood and bone: body mass, gender and health inequality in nineteenth-century British families. The History of the Family, 20(2), 204–230. https://doi.org/10.1080/1081602X.2015.1036902

Miles, A., & Connell, B. (2012). New Bunhill Fields Burial Ground, Southwark: Excavations at Globe Academy, 2008. Museum of London Archaeology.

Miller, G., & Chen, E. (2007). Unfavorable Socioeconomic Conditions in Early Life Presage Expression of Proinflammatory Phenotype in Adolescence. Psychosomatic Medicine, 69(5), 402. https://doi.org/10.1097/PSY.0b013e318068fcf9

Miller, G. E., Chen, E., Fok, A. K., Walker, H., Lim, A., Nicholls, E. F., … Kobor, M. S. (2009). Low early-life social class leaves a biological residue manifested by decreased glucocorticoid and increased proinflammatory signaling. Proceedings of the National Academy of Sciences of the United States of America, 106(34), 14716–14721. https://doi.org/10.1073/pnas.0902971106

Miller, G. E., Chen, E., Sze, J., Marin, T., Arevalo, J. M., Doll, R., … Cole, S. W. (2008). A functional genomic fingerprint of chronic stress in humans: blunted glucocorticoid and increased NF-κB signaling. Biological Psychiatry, 64(4), 266–272.

Miller, G. E., Cohen, S., & Ritchey, A. K. (2002). Chronic psychological stress and the regulation of pro-inflammatory cytokines: A glucocorticoid-resistance model. Health Psychology, 21(6), 531–541. https://doi.org/10.1037/0278-6133.21.6.531

Milne, G. (1997). St. Bride's Church, London: Archaeological research 1952-60 and 1992-5. London: English Heritage.

Milne, Gustav. (1997). St. Bride's Church, London: Archaeological Research 1952-60 and 1992-5 (Vol. 11). English Heritage London.

Møller, A. P. (1997). Developmental stability and fitness: a review. The American Naturalist, 149(5), 916–932. https://doi.org/10.1086/286030

Møller, Anders Pape, & Swaddle, J. P. (1997). Asymmetry, Developmental Stability and Evolution. Oxford University Press, UK.

Moorrees, C. F. A., Thomsen, S. Ø., Jensen, E., & Yen, P. K.-J. (1957). Mesiodistal Crown Diameters of the Deciduous and Permanent Teeth in Individuals. Journal of Dental Research, 36(1), 39–47. https://doi.org/10.1177/00220345570360011501

Moorrees, C. F., Fanning, E. A., & Hunt, E. E., Jr. (1963). Age Variation of Formation Stages for Ten Permanent Teeth. J Dent Res, 42, 1490–1502.

Morgan, N. (2002). Infant mortality, flies and horses in later-nineteenth-century towns: a case study of Preston. Continuity and Change, 17(1), 97–132.

Morris, N. (1964). Uses of Epidemiology (2nd Revised edition edition). Edinburgh: Harcourt Brace/Churchill Livingstone.

Mulligan, C. (2016). Early Environments, Stress, and the Epigenetics of Human Health. Annual Review of Anthropology, 45(1), 233–249. https://doi.org/10.1146/annurev-anthro-102215-095954

Newman, S. L., & Gowland, R. L. (2015). The use of non-adult vertebral dimensions as indicators of growth disruption and non-specific health stress in skeletal populations. American Journal of Physical Anthropology, 158(1), 155–164.

Newman, S. L., & Gowland, R. L. (2017). Dedicated Followers of Fashion? Bioarchaeological Perspectives on Socio-Economic Status, Inequality, and Health in Urban Children from the Industrial Revolution (18th–19th C), England. International Journal of Osteoarchaeology, 27(2), 217–229. https://doi.org/10.1002/oa.2531

Nicholas, S., & Oxley, D. (1993). The living standards of women during the industrial revolution, 1795-18201. The Economic History Review, 46(4), 723–749. https://doi.org/10.1111/j.1468-0289.1993.tb01359.x

Nicholas, S., & Steckel, R. H. (1991). Heights and living standards of English workers during the early years of industrialization,1770-1815. The Journal of Economic History, 51, 937–957.

Nimitphong, H., & Holick, M. F. (2011). Vitamin D, neurocognitive functioning and immunocompetence. Current Opinion in Clinical Nutrition & Metabolic Care, 14(1), 7. https://doi.org/10.1097/MCO.0b013e3283414c38

Nitsch, E. K., Humphrey, L. T., & Hedges, R. E. M. (2011). Using stable isotope analysis to examine the effect of economic change on breastfeeding practices in Spitalfields, London, UK. American Journal of Physical Anthropology, 146(4), 619–628. https://doi.org/10.1002/ajpa.21623

Nriagu, J. O. (1983). Lead and lead poisoning in antiquity. New York: John Wiley.

Oddy, W. H. (2001). Breastfeeding protects against illness and infection in infants and children: a review of the evidence. Breastfeeding Review, 9(2), 11.

Oren, L. (1973). The welfare of women in laboring families: England, 1860-1950. Feminist Studies, 1(3/4), 107–125.

Ortner, D. J. (2003). Identification of pathological conditions in human skeletal remains (2nd ed.). San Diego, CA: Academic Press.

Oxenham, M. F., & Cavill, I. (2010). Porotic hyperostosis and cribra orbitalia: the erythropoietic response to iron-deficiency anaemia. Anthropological Science, 118(3), 199–200. https://doi.org/10.1537/ase.100302

Özener, Bariş, & Fink, B. (2010). Facial symmetry in young girls and boys from a slum and a control area of Ankara, Turkey. Evolution and Human Behavior, 31(6), 436–441.

Özener, Barış. (2010a). Brief communication: Facial fluctuating asymmetry as a marker of sex differences of the response to phenotypic stresses. American Journal of Physical Anthropology, 143(2), 321–324. https://doi.org/10.1002/ajpa.21357

Özener, Barış. (2010b). Fluctuating and directional asymmetry in young human males: Effect of heavy working condition and socioeconomic status. American Journal of Physical Anthropology, 143(1), 112–120. https://doi.org/10.1002/ajpa.21300

Özener, Barış, & Ertuğrul, B. (2011). Relationship between shortness of final body height and fluctuating asymmetry in Turkish young males. Annals of Human Biology, 38(1), 34–38.

Paine, R. R., & Brenton, B. P. (2006). The paleopathology of pellagra: investigating the impact of prehistoric and historical dietary transitions to maize. Journal of Anthropological Sciences, 84(2006), 125–135.

Palmer, A R. (1994). Fluctuating asymmetry analyses: a primer. In T. Markow (Ed.), Developmental Instability: Its Origins and Evolutionary Implications. Contemporary Issues in Genetics and Evolution (Vol. 2, pp. 335–364). Dordrecht, The Netherlands: Springer. Retrieved from https://link-springer-com.pallas2.tcl.sc.edu/chapter/10.1007/978-94-011-0830-0_26

Palmer, A R, & Strobeck, C. (1986). Fluctuating Asymmetry: Measurement, Analysis, Patterns. Annual Review of Ecology and Systematics, 17(1), 391–421. https://doi.org/10.1146/annurev.es.17.110186.002135

Palmer, A R, & Strobeck, C. (2003). Fluctuating asymmetry analyses revisited. In M. Polack (Ed.), Developmental Instability (DI): Causes and Consequences (pp. 279–319). Oxford: Oxford University Press.

Palmer, A R, & Strobeck, C. (1992). Fluctuating asymmetry as a measure of developmental stability: implications of non-normal distributions and power of statistical tests. Acta Zoologica Fennica, 191, 57–72.

Parsons, P. A. (1990). Fluctuating Asymmetry: An Epigenetic Measure of Stress. Biological Reviews, 65(2), 131–145. https://doi.org/10.1111/j.1469-185X.1990.tb01186.x

Peck, J. J. (2013). Status, health, and lifestyle in Middle Iron Age Britain: A bioarcheological study of elites and non-elites from East Yorkshire, Northern England. International Journal of Paleopathology, 3(2), 83–94.

Perkin, J. (1995). Victorian women. NYU Press.

Phelan, J. C., Link, B. G., Diez-Roux, A., Kawachi, I., & Levin, B. (2004). "Fundamental causes" of social inequalities in mortality: a test of the theory. Journal of Health and Social Behavior, 45(3), 265–285.

Phelan, J. C., Link, B. G., & Tehranifar, P. (2010). Social conditions as fundamental causes of health inequalities: theory, evidence, and policy implications. Journal of Health and Social Behavior, 51 Suppl, S28-40. https://doi.org/10.1177/0022146510383498

Pierce, J. P., Fiore, M. C., Novotny, T. E., Hatziandreu, E. J., & Davis, R. M. (1989). Trends in cigarette smoking in the United States: educational differences are increasing. Jama, 261(1), 56–60.

Pinhasi, R., Shaw, P., White, B., & Ogden, A. R. (2006). Morbidity, rickets and long-bone growth in post-medieval Britain--a cross-population analysis. Annals of Human Biology, 33(3), 372–389. https://doi.org/10.1080/03014460600707503

Polak, M. (2003). Developmental Instability: Causes and Consequences. Oxford, New York: Oxford University Press.

Popkin, B. M., Siega-Riz, A. M., & Haines, P. S. (1996). A comparison of dietary trends among racial and socioeconomic groups in the United States. New England Journal of Medicine, 335(10), 716–720.

Pound, N., Lawson, D. W., Toma, A. M., Richmond, S., Zhurov, A. I., & Penton-Voak, I. S. (2014). Facial fluctuating asymmetry is not associated with childhood ill-health in a large British cohort study. Proceedings of the Royal Society B: Biological Sciences, 281(1792), 20141639. https://doi.org/10.1098/rspb.2014.1639

Prentice, A. M., & Moore, S. E. (2005). Early programming of adult diseases in resource poor countries. Archives of Disease in Childhood, 90(4), 429–432.

Proctor, J., Gaimster, M., & Langthorne, J. (2014). A quaker burial ground in North Tyneside: excavations at Coach Lane, North Shields. London: Preconstruct Archaeology Monograph Series.

Quinto-Sánchez, M., Cintas, C., Cerqueira, C. C. S. de, Ramallo, V., Acuña-Alonzo, V., Adhikari, K., … González-José, R. (2017). Socioeconomic Status Is Not Related with Facial Fluctuating Asymmetry: Evidence from Latin-American Populations. PLOS ONE, 12(1), e0169287. https://doi.org/10.1371/journal.pone.0169287

Radbill, S. X. (1981). Infant feeding through the ages. Clinical Pediatrics, 20(10), 613–621. https://doi.org/10.1177/000992288102001001

Richards, E. (1974). Women in the British economy since about 1700: An interpretation. History, 59(197), 337–357.

Robb, J., Bigazzi, R., Lazzarini, L., Scarsini, C., & Sonego, F. (2001). Social "status" and biological "status": a comparison of grave goods and skeletal indicators from Pontecagnano. American Journal of Physical Anthropology, 115(3), 213–222.

Roberts, C. A., Caffell, A., Filipek-Ogden, K. L., Gowland, R., & Jakob, T. (2016). 'Til Poison Phosphorous Brought them Death': A potentially occupationally-related disease in a post-medieval skeleton from north-east England. International Journal of Paleopathology, 13, 39–48.

Roberts, C. A., & Manchester, K. (2005). The archaeology of disease. Ithaca, NY: Cornell University Press.

Rodney, N. C., & Mulligan, C. J. (2014). A biocultural study of the effects of maternal stress on mother and newborn health in the Democratic Republic of Congo. American Journal of Physical Anthropology, 155, 200–209. https://doi.org/10.1002/ajpa.22568

Rose, J. C. (Ed.). (1985). Gone to a Better Land. Fayetteville, Arkansas: Arkansas Archeological Survey.

Rose, Jerome C., Armelagos, G. J., & Lallo, J. W. (1978). Histological enamel indicator of childhood stress in prehistoric skeletal samples. American Journal of Physical Anthropology, 49(4), 511–516.

Rose, Jerome C., & Hartnady, P. (1991). Interpretation of infectious skeletal lesions from a historic Afro-American cemetery. In D. J. Ortner & A. C. Aufderheide (Eds.), Human paleopathology: current syntheses and future options (pp. 119–127). Washington: Smithsonian Institution Press.

Rosenfeld, C. S. (2015). The epigenome and developmental origins of health and disease. Academic Press.

Rothman, K. J. (1998). Writing for Epidemiology. Epidemiology, 9, 333–337.

Rule, J. (2014). The labouring classes in early industrial England, 1750-1850. Routledge.

Sattenspiel L, & Harpending H. (1983). Stable populations and skeletal age. American Antiquity, 48(3), 489–498.

Saunders, S. R., & Hoppa, R. D. (1993). Growth deficit in survivors and non-survivors: Biological mortality bias in subadult skeletal samples. American Journal of Physical Anthropology, 36(S17), 127–151. https://doi.org/10.1002/ajpa.1330360608

Savage, D., & Lindenbaum, J. (1986). Anemia in alcoholics. Medicine, 65(5), 322–338.

Schell, L. (1997). Culture as a stressor: A revised model of biocultural interaction. American Journal of Physical Anthropology, 102, 67–77.

Schoeninger, M. J. (1979). Diet and status at Chalcatzingo: some empirical and technical aspects of strontium analysis. American Journal of Physical Anthropology, 51(3), 295–310. https://doi.org/10.1002/ajpa.1330510302

Schofield, R. (1994). British population change, 1700-1871. In R Floud & D. McCloskey (Eds.), The Economic History of Britain Since 1700 Volume 1: 1700-1860 (pp. 60–95). Cambridge: Cambridge University Press.

Schwarz, L. D. (1985). The standard of living in the long run: London, 1700–1860. The Economic History Review, 38(1), 24–36.

Scrimshaw, N. S. (2003). Historical concepts of interactions, synergism and antagonism between nutrition and infection. The Journal of Nutrition, 133(1), 316S-321S.

Scrimshaw, N. S., Taylor, C. E., & Gordon, J. E. (1968). Interactions of nutrition and infection. American Journal of the Medical Sciences, 237(3), 367–403.

Sen, G., & Iyer, A. (2012). Who gains, who loses and how: Leveraging gender and class intersections to secure health entitlements. Social Science & Medicine, 74(11), 1802–1811. https://doi.org/10.1016/j.socscimed.2011.05.035

Seng, J. S., Lopez, W. D., Sperlich, M., Hamama, L., & Reed Meldrum, C. D. (2012). Marginalized identities, discrimination burden, and mental health: empirical exploration of an interpersonal-level approach to modeling intersectionality. Social Science & Medicine (1982), 75(12), 2437–2445. https://doi.org/10.1016/j.socscimed.2012.09.023

Shammas, C. (1984). The eighteenth-century English diet and economic change. Explorations in Economic History, 21(3), 254.

Sharpe, P. (2012). Explaining the short stature of the poor: chronic childhood disease and growth in nineteenth-century England. Economic History Review, 65(4), 1475–1494. https://doi.org/10.1111/j.1468-0289.2011.00629.x

Springer, K. W., Hankivsky, O., & Bates, L. M. (2012). Gender and health: relational, intersectional, and biosocial approaches. Social Science & Medicine (1982), 74(11), 1661–1666. https://doi.org/10.1016/j.socscimed.2012.03.001

Stearns, P. N. (2007). The social impact of the industrial revolution. In The Industrial Revolution in World History (pp. 69–90). Boulder, CO: Westview Press.

Steckel, R. H. (2005). Young adult mortality following severe physiological stress in childhood: skeletal evidence. Economics and Human Biology, 3(2), 314–328. https://doi.org/10.1016/j.ehb.2005.05.006

Steckel, R. H., & Floud, R. (2008). Health and welfare during industrialization. University of Chicago Press.

Steckel, R. H. (2009). Heights and human welfare: Recent developments and new directions. Explorations in Economic History, 46, 1–23.

Stevenson, J. (1993). Social aspects of the Industrial Revolution. In P. O'Brien & R. Quinault (Eds.), The Industrial Revolution and British Society (pp. 229–253). Cambridge: Cambridge University Press.

Stinson, S. (1985). Sex differences in environmental sensitivity during growth and development. American Journal of Physical Anthropology, 28(S6), 123–147.

Stinson, S. (2000). Growth variation: biological and cultural factors. In S. Stinson, B. Bogin, R. Huss-Ashmore, & D. H. O'Rourke (Eds.), Human Biology: an evolutionary and biocultural perspective (pp. 434–438). New York: Wiley-Liss.

Stirratt, M. J., Meyer, I. H., Ouellette, S. C., & Gara, M. A. (2008). Measuring identity multiplicity and intersectionality: Hierarchical classes analysis (HICLAS) of sexual, racial, and gender identities. Self and Identity, 7(1), 89–111.

Stojanowski, C. M., Seidemann, R. M., & Doran, G. H. (2002). Differential skeletal preservation at Windover Pond: causes and consequences. Am J Phys Anthropol, 119(1), 15–26.

Stojanowski, Christopher M., Larsen, C. S., Tung, T. A., & McEwan, B. G. (2007). Biological structure and health implications from tooth size at Mission San Luis de Apalachee. American Journal of Physical Anthropology, 132(2), 207–222. https://doi.org/10.1002/ajpa.20489

Storey, R. (1992). Preindustrial urbanlifestyle and health. In R. Huss-Ashmore, J. Schall, & M. Hediger (Eds.), Health and Lifestyle Change (pp. 33–42). Philadelphia: University Museum of Archaeology and Anthropology.

Stringhini, S., Carmeli, C., Jokela, M., Avendaño, M., Muennig, P., Guida, F., … Bochud, M. (2017). Socioeconomic status and the 25 × 25 risk factors as determinants of premature mortality: a multicohort study and meta-analysis of 1· 7 million men and women. The Lancet, 389(10075), 1229–1237.

Stringhini, S., Rousson, V., Viswanathan, B., Gedeon, J., Paccaud, F., & Bovet, P. (2014). Association of socioeconomic status with overall and cause specific mortality in the Republic of Seychelles: results from a cohort study in the African region. PloS One, 9(7), e102858.

Stringhini, S., Sabia, S., Shipley, M., Brunner, E., Nabi, H., Kivimaki, M., & Singh-Manoux, A. (2010). Association of socioeconomic position with health behaviors and mortality. Jama, 303(12), 1159–1166.

Stuart-Macadam, Patricia. (1991). Porotic hyperostosis: Changing interpretations. In D. J. Ortner & A. C. Aufderheide (Eds.), Human paleopathology: current syntheses and future options. (pp. 36–39). Washington, DC: Smithsonian Institution Press.

Stuart-Macadam, Patricia, & Kent, S. (1992). Diet, demography, and disease : changing perspectives on anemia. New York: Aldine de Gruyter.

Stuart-Macadam, Patty. (1985). Porotic Hyperostosis: Representative of a Childhood Condition. American Journal of Physical Anthropology, 66, 391–398.

Sundquist, K., Theobald, H., Yang, M., Li, X., Johansson, S.-E., & Sundquist, J. (2006). Neighborhood violent crime and unemployment increase the risk of coronary heart disease: a multilevel study in an urban setting. Social Science & Medicine, 62(8), 2061–2071.

Szreter, S. (2004). Industrialization and health. British Medical Bulletin, 69(1), 75–86. https://doi.org/10.1093/bmb/ldh005

Szreter, S., & Mooney, G. (1998). Urbanization, mortality, and the standard of living debate: new estimates of the expectation of life at birth in nineteenth-century British cities. The Economic History Review, 51(1), 84–112.

Tancock, D., & Lee, K. (2014). Congenital Defects in 18th and 19th Century Populations from Rural and Urban Northeast England (PhD Thesis). Durham University.

Temple, D. H. (2014). Plasticity and constraint in response to early-life stressors among late/final jomon period foragers from Japan: Evidence for life history trade-offs from incremental microstructures of enamel. American Journal of Physical Anthropology, 155(4), 537–545. https://doi.org/10.1002/ajpa.22606

Temple, D. H. (2018). Bioarchaeological evidence for adaptive plasticity and constraint: Exploring life-history trade-offs in the human past. Evolutionary Anthropology: Issues, News, and Reviews, 28(1), 34–46. https://doi.org/10.1002/evan.21754

Titmuss, R. M. (1943). Birth, Poverty and Wealth. A Study of Infant Mortality. Birth, Poverty and Wealth. A Study of Infant Mortality.

Torres-Rouff, C., & Knudson, K. J. (2017). Integrating Identities: An Innovative Bioarchaeological and Biogeochemical Approach to Analyzing the Multiplicity of Identities in the Mortuary Record. Current Anthropology, 58(3), 381–409. https://doi.org/10.1086/692026

Trafimow, D., & Marks, M. (2015). Editorial. Basic and Applied Social Psychology, 37(1), 1–2. https://doi.org/10.1080/01973533.2015.1012991

Varcoe, C., & Dick, S. (2008). The intersecting risks of violence and HIV for rural Aboriginal women in a neo-colonial Canadian context. International Journal of Indigenous Health, 4(1), 42–52.

Vaupel, J. W., Manton, K. G., & Stallard, E. (1979). The impact of heterogeneity in individual frailty on the dynamics of mortality. Demography, 16, 439–454.

Vaupel, J. W., & Yashin, A. I. (1985). Heterogeneity's ruses: some surprising effects of selection on population dynamics. American Statistician, 39, 176–185.

Veenstra, G. (2011). Race, gender, class, and sexual orientation: intersecting axes of inequality and self-rated health in Canada. International Journal for Equity in Health, 10(1), 3.

Voland, E., Dunbar, R. I. M., Engel, C., & Stephan, P. (1997). Population Increase and Sex-Biased Parental Investment in Humans: Evidence from 18th-and 19th-Century Germany. Current Anthropology, 38(1), 129–135.

Voss, B. L. (2008). Sexuality studies in archaeology. Annual Review of Anthropology, 37, 317–336.

Wadhwa, P. D., Buss, C., Entringer, S., & Swanson, J. M. (2009). Developmental Origins of Health and Disease: Brief History of the Approach and Current Focus on Epigenetic Mechanisms. Seminars in Reproductive Medicine, 27(5), 358–368. https://doi.org/10.1055/s-0029-1237424

Waldron, T. (1997). The relative survival of the human skeleton: implications for paleopathology. In A. Boddington, A. N. Garland, & R. C. Janaway (Eds.), Death, decay and reconstruction. (pp. 55–64). Manchester: Manchester University Press.

Walker, P. L., Bathurst, R. R., Richman, R., Gjerdrum, T., & Andrushko, V. A. (2009). The causes of porotic hyperostosis and cribra orbitalia: A reappraisal of the iron-deficiency-anemia hypothesis. American Journal of Physical Anthropology, 139(2), 109–125.

Wapler, U., Crubézy, E., & Schultz, M. (2004). Is cribra orbitalia synonymous with anemia? Analysis and interpretation of cranial pathology in Sudan. American Journal of Physical Anthropology, 123(4), 333–339.

Warner, D. F., & Brown, T. H. (2011). Understanding how race/ethnicity and gender define age-trajectories of disability: An intersectionality approach. Social Science & Medicine, 72(8), 1236–1248.

Waterland, R. A., & Michels, K. B. (2007). Epigenetic epidemiology of the developmental origins hypothesis. Annual Review of Nutrition, 27, 363–388. https://doi.org/10.1146/annurev.nutr.27.061406.093705

Watts, R. (2011). Non-specific indicators of stress and their association with age at death in Medieval York: Using stature and vertebral neural canal size to examine the effects of stress occurring during different periods of development. International Journal of Osteoarchaeology, 21(5), 568–576. https://doi.org/10.1002/oa.1158

Watts, R. (2013a). Childhood development and adult longevity in an archaeological population from Barton-upon-Humber, Lincolnshire, England. International Journal of Paleopathology, 3(2), 95–104.

Watts, R. (2013b). Lumbar vertebral canal size in adults and children: Observations from a skeletal sample from London, England. HOMO, 64(2), 120–128. https://doi.org/10.1016/j.jchb.2013.01.002

Watts, R. (2015). The long-term impact of developmental stress. Evidence from later medieval and post-medieval London (AD1117–1853). American Journal of Physical Anthropology, 158(4), 569–580. https://doi.org/10.1002/ajpa.22810

Webster, J. I., Tonelli, L., & Sternberg, E. M. (2002). Neuroendocrine regulation of immunity. Annual Review of Immunology, 20(1), 125–163.

Webster, M., Libranda-Ramirez, B. D. L., Aligui, G. D., Olveda, R. M., Ouma, J. H., Kariuki, H. C., … Butterworth, A. E. (1997). The influence of sex and age on antibody isotype responses to Schistosoma mansoni and Schistosoma japonicum in human populations in Kenya and the Philippines. Parasitology, 114(4), 383–393.

Weinberg, F. (1993). Infant feeding through the ages. Canadian Family Physician, 39, 2016–2020.

Weisensee, K. E. (2013). Assessing the relationship between fluctuating asymmetry and cause of death in skeletal remains: a test of the developmental origins of health and disease hypothesis. American Journal of Human Biology, 25(3), 411–417. https://doi.org/10.1002/ajhb.22390

Weisensee, K. E., & Spradley, M. K. (2018). Craniofacial asymmetry as a marker of socioeconomic status among undocumented Mexican immigrants in the United States. Economics & Human Biology, 29, 122–127. https://doi.org/10.1016/j.ehb.2018.02.007

Weston, D. A. (2008). Investigating the specificity of periosteal reactions in pathology museum specimens. American Journal of Physical Anthropology, 137(1), 48–59. https://doi.org/10.1002/ajpa.20839

White, T. D. (1978). Early hominid enamel hypoplasia. American Journal of Physical Anthropology, 49(1), 79–83.

Wickes, I. G. (1953). A history of infant feeding. II. Seventeenth and eighteenth centuries. Archives of Disease in Childhood, 28(139), 232–240; contd.

Williams, D. R., Mohammed, S. A., Leavell, J., & Collins, C. (2010). Race, socioeconomic status, and health: complexities, ongoing challenges, and research opportunities. Annals of the New York Academy of Sciences, 1186(1), 69–101.

Williamson, J. (1994). Coping with city growth. In R Floud & D. McCloskey (Eds.), The Economic History of Britain since 1700 (pp. 332–356). Cambridge: Cambridge University Press.

Willson, A. E., Shuey, K. M., & Elder, G. H. (2007). Cumulative advantage processes as mechanisms of inequality in life course health. American Journal of Sociology, 112(6), 1886–1924.

Wintergerst, E. S., Maggini, S., & Hornig, D. H. (2007). Contribution of selected vitamins and trace elements to immune function. Annals of Nutrition and Metabolism, 51(4), 301–323.

Wood, J., Milner, G., Harpending, H., & Weiss, K. (1992). The osteological paradox: problems of inferring prehistoric health from skeletal samples. Current Anthropology, 33(4), 343–370.

Wood, J. W., Milner, G. R., Harpending, H. C., & Weiss, K. M. (1992). The Osteological Paradox: Problems of Inferring Prehistoric Health from Skeletal Samples. Current Anthropology, 33(4), 343–370.

Woods, R., & Woodward, J. (1984). Urban Disease and Mortality in Nineteenth-Century England. London: Batsford Academic and Educational.

Wright, L. E., & Yoder, C. J. (2003). Recent progress in bioarchaeology: Approaches to the osteological paradox. Journal of Archaeological Research, 11, 43–70.

Wrigley, E. A., & Schofield, R. S. (1989). The population history of England 1541-1871 (Vol. 46). Cambridge University Press.

Yaussy, S. L., DeWitte, S. N., & Redfern, R. C. (2016). Frailty and famine: Patterns of mortality and physiological stress among victims of famine in medieval London. American Journal of Physical Anthropology, 160(2), 272–283.

Zuckerman, M. K., & Armelagos, G. J. (2011). The origins of biocultural dimensions in bioarchaeology. Social Bioarchaeology, 13–43.

Zuckerman, M. K., Turner, B. L., & Armelagos, G. J. (2012). Evolutionary Thought in Paleopathology and the Rise of the Biocultural Approach. In A Companion to Paleopathology (pp. 34–57). John Wiley & Sons, Ltd. https://doi.org/10.1002/9781444345940.ch3

Zuk, M., & McKean, K. A. (1996). Sex differences in parasite infections: patterns and processes. International Journal for Parasitology, 26(10), 1009–1024.

APPENDIX A

HAZARD ANALYSIS RESULTS

A.1 Siler Model, All Sites, All Individuals, SES Covariate

1085 lines read from file Age_SESgroup.dat
1085 Observations kept and 0 observations dropped.

NAME	age	SESgroup
MEAN	25.3535484	0.20368664
VAR	733.498541	0.16234802
STDEV	27.0831782	0.40292434
MIN	0.00000000	0.00000000
MAX	81.3000000	1.00000000

Age_SESgroup.mle
Program file: Age_SESgroup.mle
Input data file name: Age_SESgroup.dat
2 variables read.

Model 1 Run 1 : Age_SESgroup.mle

METHOD = SIMPLEX MAXITER = 5000 MAXEVALS = 100000
Convergence at EPSILON = 0.0000010000
LogLikelihood: -4118.921 AIC: 8245.8412 Del(LL): 0.0000008703
Iterations: 163 Function evals: 258 Time: 00:00:01
Converged normally

Results with estimated standard errors. (35 evals)
Solution with 4 free parameters

Name	Form	Estimate	Std Error	t	against
a1	LOGLIN	0.268598786839	0.018299266491	14.6781176705	0.0
b1	LOGLIN	0.463934693492	0.031860853670	14.5612763016	0.0
a3	LOGLIN	0.006267343729	0.000930241107	6.73733259249	0.0
b3	LOGLIN	0.036170152325	0.003983936615	9.07899794069	0.0

Results with estimated standard errors. (40 evals)
Solution with 4 free parameters

Name Form	Estimate	Std Error	t	against
a1 LOGLIN	0.268598786839	0.018168938920	14.7834052406	0.0
b1 LOGLIN	0.463934693492	0.030380141427	15.2709853117	0.0
a3 LOGLIN	0.006267343729	0.000715917558	8.75428135584	0.0
b3 LOGLIN	0.036170152325	0.002269675561	15.9362654919	0.0

Likelihood CI Results: (129 evals)
Solution with 4 free parameters

Name Form	Estimate	Lower CI	Upper CI
a1 LOGLIN	0.268598786839	0.229596413604	0.311468275832
b1 LOGLIN	0.463934693492	0.403048001117	0.535723494376
a3 LOGLIN	0.006267343729	0.005475255941	0.007131381885
b3 LOGLIN	0.036170152325	0.033448928366	0.038753894959

Model 1 Run 2 : Age_SESgroup.mle

METHOD = SIMPLEX MAXITER = 5000 MAXEVALS = 100000
Convergence at EPSILON = 0.0000010000
LogLikelihood: -4048.649 AIC: 8107.2976 Del(LL): 0.0000009575
Iterations: 276 Function evals: 440 Time: 00:00:01
Converged normally

Results with estimated standard errors. (41 evals)
Solution with 5 free parameters

Name Form	Estimate	Std Error	t	against
a1 LOGLIN	0.307434170232	0.021254508871	14.4644212716	0.0
b1 LOGLIN	0.450633386037	0.032106635483	14.0355219181	0.0
a3 LOGLIN	0.008168746767	0.001286732550	6.34844184807	0.0
b3 LOGLIN	0.036877193153	0.003581114634	10.2976857540	0.0
b_SESgroup	-0.84389237751	0.148589998934	-5.6793349725	0.0

Results with estimated standard errors. (60 evals)
Solution with 5 free parameters

Name Form	Estimate	Std Error	t	against
a1 LOGLIN	0.307434170232	0.020934400400	14.6855971202	0.0
b1 LOGLIN	0.450633386037	0.030398757673	14.8240724470	0.0
a3 LOGLIN	0.008168746767	0.000933876886	8.74713453904	0.0
b3 LOGLIN	0.036877193153	0.002240720284	16.4577405794	0.0
b_SESgroup	-0.84389237751	0.076381471694	-11.048391171	0.0

Likelihood CI Results: (166 evals)
Solution with 5 free parameters

Name Form	Estimate	Lower CI	Upper CI
a1 LOGLIN	0.307434170232	0.262519823311	0.356814220828
b1 LOGLIN	0.450633386037	0.389499290014	0.523013711549

a3 LOGLIN	0.008168746767	0.007136791245	0.009294058173
b3 LOGLIN	0.036877193153	0.034160489961	0.039455644843
b_SESgroup	-0.84389237751	-1.06496617849	-0.63797661834

Model 1 Run 3 : Age_SESgroup.mle

METHOD = SIMPLEX MAXITER = 5000 MAXEVALS = 100000
Convergence at EPSILON = 0.0000010000
LogLikelihood: -4031.404 AIC: 8072.8071 Del(LL): 0.0000009810
Iterations: 855 Function evals: 1354 Time: 00:00:03
Converged normally

Results with estimated standard errors. (54 evals)
Solution with 5 free parameters

Name	Form	Estimate	Std Error	t	against
a1	LOGLIN	0.293662593972	0.023542314884	12.4738198183	0.0
b1	LOGLIN	0.538763637621	0.041233622384	13.0661243536	0.0
a2	LOGLIN	0.015716783797	0.001426949186	11.0142561153	0.0
a3	LOGLIN	2.19585E-0011	6.41801E-0011	0.34213860834	0.0
b3	LOGLIN	0.310277372843	0.001082759587	286.561649098	0.0

Results with estimated standard errors. (60 evals)
Solution with 5 free parameters

Name	Form	Estimate	Std Error	t	against
a1	LOGLIN	0.293662593972	0.022113528610	13.2797709106	0.0
b1	LOGLIN	0.538763637621	0.037374593156	14.4152375215	0.0
a2	LOGLIN	0.015716783797	0.000797646121	19.7039556522	0.0
a3	LOGLIN	2.19585E-0011	0.000000001387	0.01583694131	0.0
b3	LOGLIN	0.310277372843	0.001193949840	259.874713635	0.0

Likelihood CI Results: (160 evals)
Solution with 5 free parameters

Name	Form	Estimate	Lower CI	Upper CI
a1	LOGLIN	0.293662593972	0.224258031276	0.324902982514
b1	LOGLIN	0.538763637621	0.481544665757	0.670351945387
a2	LOGLIN	0.015716783797	0.014717588471	0.023122258925
a3	LOGLIN	2.19585E-0011	1.65198E-0011	2.83384E-0011
b3	LOGLIN	0.310277372843	0.306399706157	0.313681922584

Model 1 Run 4 : Age_SESgroup.mle

METHOD = SIMPLEX MAXITER = 5000 MAXEVALS = 100000
Convergence at EPSILON = 0.0000010000
LogLikelihood: -4053.842 AIC: 8119.6830 Del(LL): 0.0000009666

Iterations: 607 Function evals: 833 Time: 00:00:02
Converged normally

Results with estimated standard errors. (59 evals)
Solution with 6 free parameters

Name	Form	Estimate	Std Error	t	against
a1	LOGLIN	0.275429611607	0.018749369383	14.6900733555	0.0
b1	LOGLIN	0.364180312321	0.032665349321	11.1488265053	0.0
a2	LOGLIN	3.79823E-0010	0.009828734968	0.00000003864	0.0
a3	LOGLIN	0.007549900063	0.006035814687	1.25085020902	0.0
b3	LOGLIN	0.038160833136	0.011962427721	3.19005757247	0.0
b_SESgroup		-0.84923654893	0.171289307338	-4.9579075433	0.0

Results with estimated standard errors. (84 evals)
Solution with 6 free parameters

Name	Form	Estimate	Std Error	t	against
a1	LOGLIN	0.275429611607	0.018510519214	14.8796264670	0.0
b1	LOGLIN	0.364180312321	0.020807402734	17.5024397315	0.0
a2	LOGLIN	3.79823E-0010	-oo	+oo	0.0
a3	LOGLIN	0.007549900063	-oo	+oo	0.0
b3	LOGLIN	0.038160833136	-oo	+oo	0.0
b_SESgroup		-0.84923654893	0.075474556514	-11.251958119	0.0

Likelihood CI Results: (181 evals)
Solution with 6 free parameters

Name	Form	Estimate	Lower CI	Upper CI
a1	LOGLIN	0.275429611607	0.225702977938	0.308369833476
b1	LOGLIN	0.364180312321	0.340100879687	0.505285614781
a2	LOGLIN	3.79823E-0010	1.08420E-0019	0.004111999837
a3	LOGLIN	0.007549900063	0.006599433917	0.008618463621
b3	LOGLIN	0.038160833136	0.035570351193	0.040863866473
b_SESgroup		-0.84923654893	-1.08153805843	-0.65398544096

A.2 Siler Model, London Sites Only, All Individuals, SES Covariate

751 lines read from file Age_SESgroup_LondonOnly.dat
751 Observations kept and 0 observations dropped.

NAME	age	SESgroup
MEAN	22.3513981	0.29427430
VAR	679.706661	0.20795384
STDEV	26.0711845	0.45601956
MIN	0.00000000	0.00000000
MAX	79.9000000	1.00000000

Age_SESgroup_LondonOnly.mle
Program file: Age_SESgroup_LondonOnly.mle
Input data file name: Age_SESgroup_LondonOnly.dat
2 variables read.

Model 1 Run 1 : Age_SESgroup_LondonOnly.mle

METHOD = SIMPLEX MAXITER = 5000 MAXEVALS = 100000
Convergence at EPSILON = 0.0000010000
LogLikelihood: -2713.181 AIC: 5434.3620 Del(LL): 0.0000006706
Iterations: 176 Function evals: 281 Time: 00:00:00
Converged normally

Results with estimated standard errors. (33 evals)
Solution with 4 free parameters

Name	Form	Estimate	Std Error	t	against
a1	LOGLIN	0.301210433565	0.023060736088	13.0616140099	0.0
b1	LOGLIN	0.420192929272	0.032387593962	12.9738853020	0.0
a3	LOGLIN	0.005689288574	0.001099614475	5.17389385452	0.0
b3	LOGLIN	0.039054533969	0.004874795308	8.01152284318	0.0

Results with estimated standard errors. (40 evals)
Solution with 4 free parameters

Name	Form	Estimate	Std Error	t	against
a1	LOGLIN	0.301210433565	0.022087686288	13.6370296840	0.0
b1	LOGLIN	0.420192929272	0.029663637831	14.1652528146	0.0
a3	LOGLIN	0.005689288574	0.000858110009	6.63002238896	0.0
b3	LOGLIN	0.039054533969	0.003009203997	12.9783603918	0.0

Likelihood CI Results: (123 evals)
Solution with 4 free parameters

Name	Form	Estimate	Lower CI	Upper CI

a1 LOGLIN	0.301210433565	0.253232390197	0.354510541807
b1 LOGLIN	0.420192929272	0.359600098725	0.492462026341
a3 LOGLIN	0.005689288574	0.004772612516	0.006714428364
b3 LOGLIN	0.039054533969	0.035506266662	0.042373606084

Model 1 Run 2 : Age_SESgroup_LondonOnly.mle

METHOD = SIMPLEX MAXITER = 5000 MAXEVALS = 100000
Convergence at EPSILON = 0.0000010000
LogLikelihood: -2560.434 AIC: 5130.8688 Del(LL): 0.0000009230
Iterations: 313 Function evals: 513 Time: 00:00:02
Converged normally

Results with estimated standard errors. (38 evals)
Solution with 5 free parameters

Name Form	Estimate	Std Error	t	against
a1 LOGLIN	0.398028242888	0.030963932188	12.8545767530	0.0
b1 LOGLIN	0.371530380707	0.032065790526	11.5865030803	0.0
a3 LOGLIN	0.010000115009	0.002103332267	4.75441525236	0.0
b3 LOGLIN	0.046629182410	0.004072218730	11.4505593871	0.0
b_SESgroup	-1.44870183452	0.134753807058	-10.750730285	0.0

Results with estimated standard errors. (60 evals)
Solution with 5 free parameters

Name Form	Estimate	Std Error	t	against
a1 LOGLIN	0.398028242888	0.029090787159	13.6822781972	0.0
b1 LOGLIN	0.371530380707	0.028850589001	12.8777398858	0.0
a3 LOGLIN	0.010000115009	0.001509517415	6.62470992883	0.0
b3 LOGLIN	0.046629182410	0.003015436952	15.4634910795	0.0
b_SESgroup	-1.44870183452	0.086936781136	-16.663854074	0.0

Likelihood CI Results: (155 evals)
Solution with 5 free parameters

Name Form	Estimate	Lower CI	Upper CI
a1 LOGLIN	0.398028242888	0.334134119459	0.469100700974
b1 LOGLIN	0.371530380707	0.311978965488	0.443104114422
a3 LOGLIN	0.010000115009	0.008387726267	0.011803757005
b3 LOGLIN	0.046629182410	0.043088404120	0.049941100357
b_SESgroup	-1.44870183452	-1.66973811772	-1.24274855932

Model 1 Run 3 : Age_SESgroup_LondonOnly.mle

METHOD = SIMPLEX MAXITER = 5000 MAXEVALS = 100000
Convergence at EPSILON = 0.0000010000

LogLikelihood: -2761.317 AIC: 5532.6346 Del(LL): 0.0000008404
Iterations: 340 Function evals: 436 Time: 00:00:01
Converged normally

Results with estimated standard errors. (53 evals)
Solution with 5 free parameters

Name	Form	Estimate	Std Error	t	against
a1	LOGLIN	0.152401378028	0.011846834026	12.8643127513	0.0
b1	LOGLIN	0.202042739374	0.029452460811	6.85996123286	0.0
a2	LOGLIN	2.89067E-0010	0.013580348301	0.00000002129	0.0
a3	LOGLIN	0.004767849445	0.006771104076	0.70414653080	0.0
b3	LOGLIN	0.041704823294	0.021199936407	1.96721454690	0.0

Results with estimated standard errors. (60 evals)
Solution with 5 free parameters

Name	Form	Estimate	Std Error	t	against
a1	LOGLIN	0.152401378028	0.013257175265	11.4957654993	0.0
b1	LOGLIN	0.202042739374	0.017712747292	11.4066291380	0.0
a2	LOGLIN	2.89067E-0010	0.012878099560	0.00000002245	0.0
a3	LOGLIN	0.004767849445	0.006563432338	0.72642623538	0.0
b3	LOGLIN	0.041704823294	0.018883364024	2.20854839426	0.0

Likelihood CI Results: (137 evals)
Solution with 5 free parameters

Name	Form	Estimate	Lower CI	Upper CI
a1	LOGLIN	0.152401378028	0.141015853602	0.219543788444
b1	LOGLIN	0.202042739374	0.188548577941	0.362977414226
a2	LOGLIN	2.89067E-0010	1.08420E-0019	0.004203015662
a3	LOGLIN	0.004767849445	0.003986053716	0.005689070370
b3	LOGLIN	0.041704823294	0.038383932010	0.045311319981

Model 1 Run 4 : Age_SESgroup_LondonOnly.mle

METHOD = SIMPLEX MAXITER = 5000 MAXEVALS = 100000
Convergence at EPSILON = 0.0000010000
LogLikelihood: -2599.398 AIC: 5210.7963 Del(LL): 0.0000007650
Iterations: 712 Function evals: 943 Time: 00:00:02
Converged normally

Results with estimated standard errors. (60 evals)
Solution with 6 free parameters

Name	Form	Estimate	Std Error	t	against
a1	LOGLIN	0.255620177867	0.022417008086	11.4029569375	0.0
b1	LOGLIN	0.177136921313	0.032875611894	5.38809503780	0.0
a2	LOGLIN	0.000000001123	0.027721233350	0.00000004049	0.0

a3 LOGLIN	0.010876077458	0.012626068506	0.86139857809	0.0
b3 LOGLIN	0.044796820415	0.016060007855	2.78933988189	0.0
b_SESgroup	-1.54015782322	0.166754809114	-9.2360624044	0.0

Results with estimated standard errors. (84 evals)
Solution with 6 free parameters

Name Form	Estimate	Std Error	t	against
a1 LOGLIN	0.255620177867	0.021669121174	11.7965179951	0.0
b1 LOGLIN	0.177136921313	0.015671010880	11.3034776549	0.0
a2 LOGLIN	0.000000001123	0.012695689896	0.00000008842	0.0
a3 LOGLIN	0.010876077458	0.005933370278	1.83303534889	0.0
b3 LOGLIN	0.044796820415	0.007967858405	5.62219082466	0.0
b_SESgroup	-1.54015782322	0.086271767723	-17.852396721	0.0

Likelihood CI Results: (175 evals)
Solution with 6 free parameters

Name Form	Estimate	Lower CI	Upper CI
a1 LOGLIN	0.255620177867	0.209395852759	0.293926602165
b1 LOGLIN	0.177136921313	0.168101544204	0.427706908131
a2 LOGLIN	0.000000001123	1.08420E-0019	0.004912480683
a3 LOGLIN	0.010876077458	0.008844560992	0.012673901184
b3 LOGLIN	0.044796820415	0.041808377223	0.048922482801
b_SESgroup	-1.54015782322	-1.74556730360	-1.31732961818

A.3 Gompertz Model, St. Bride's Fleet Street Only, Adult Individuals, Sex Covariate

208 lines read from file SB79_sex.dat
208 Observations kept and 0 observations dropped.

NAME	age	sex
MEAN	53.3610577	0.53365385
VAR	368.630602	0.25006968
STDEV	19.1997553	0.50006967
MIN	18.0000000	0.00000000
MAX	79.9000000	1.00000000

SB79_sex.mle
Program file: SB79_sex.mle
Input data file name: SB79_sex.dat
2 variables read.

Model 1 Run 1 : SB79_sex.mle

METHOD = SIMPLEX MAXITER = 5000 MAXEVALS = 100000
Convergence at EPSILON = 0.0000010000
LogLikelihood: -890.7140 AIC: 1785.4280 Del(LL): 0.0000003863
Iterations: 48 Function evals: 80 Time: 00:00:00
Converged normally

Results with estimated standard errors. (21 evals)
Solution with 2 free parameters

Name	Form	Estimate	Std Error	t	against
a2	LOGLIN	0.001968936496	0.000516018185	3.81563393144	0.0
b	LOGLIN	0.055245205044	0.005865806982	9.41817642637	0.0

Results with estimated standard errors. (12 evals)
Solution with 2 free parameters

Name	Form	Estimate	Std Error	t	against
a2	LOGLIN	0.001968936496	0.000475407022	4.14158059041	0.0
b	LOGLIN	0.055245205044	0.004334390958	12.7457826450	0.0

Likelihood CI Results: (61 evals)
Solution with 2 free parameters

Name	Form	Estimate	Lower CI	Upper CI
a2	LOGLIN	0.001968936496	0.001567358093	0.002434051007
b	LOGLIN	0.055245205044	0.051134708077	0.059037695859

Model 1 Run 2 : SB79_sex.mle

METHOD = SIMPLEX MAXITER = 5000 MAXEVALS = 100000
Convergence at EPSILON = 0.0000010000
LogLikelihood: -888.4048 AIC: 1782.8096 Del(LL): 0.0000004983
Iterations: 88 Function evals: 148 Time: 00:00:00
Converged normally

Results with estimated standard errors. (25 evals)
Solution with 3 free parameters

Name	Form	Estimate	Std Error	t	against
a2	LOGLIN	0.002210483019	0.000622237052	3.55247732520	0.0
b	LOGLIN	0.056308364140	0.005821232392	9.67292840219	0.0
b_sex		-0.30232405929	0.159997968860	-1.8895493577	0.0

Results with estimated standard errors. (24 evals)
Solution with 3 free parameters

Name	Form	Estimate	Std Error	t	against
a2	LOGLIN	0.002210483019	0.000545897097	4.04926685408	0.0
b	LOGLIN	0.056308364140	0.004380074412	12.8555724944	0.0
b_sex		-0.30232405929	0.139919970549	-2.1606927025	0.0

Likelihood CI Results: (95 evals)
Solution with 3 free parameters

Name	Form	Estimate	Lower CI	Upper CI
a2	LOGLIN	0.002210483019	0.001759620727	0.002732627985
b	LOGLIN	0.056308364140	0.052196845936	0.060102331426
b_sex		-0.30232405929	-0.61899007785	-0.01574326360

Model 1 Run 3 : SB79_sex.mle

METHOD = SIMPLEX MAXITER = 5000 MAXEVALS = 100000
Convergence at EPSILON = 0.0000010000
LogLikelihood: -867.0680 AIC: 1740.1359 Del(LL): 0.0000009649
Iterations: 386 Function evals: 644 Time: 00:00:00
Converged normally

Results with estimated standard errors. (34 evals)
Solution with 3 free parameters

Name	Form	Estimate	Std Error	t	against
a1	LOGLIN	0.013608058384	0.002077899589	6.54894897654	0.0
a2	LOGLIN	0.000000057801	0.000000010010	5.77460799580	0.0
b	LOGLIN	0.204506899570	0.002992448222	68.3409985450	0.0

Results with estimated standard errors. (24 evals)
Solution with 3 free parameters

Name	Form	Estimate	Std Error	t	against
a1	LOGLIN	0.013608058384	0.001372838835	9.91234953020	0.0
a2	LOGLIN	0.000000057801	0.000000002889	20.0065655825	0.0
b	LOGLIN	0.204506899570	0.001674926440	122.099033563	0.0

Likelihood CI Results: (87 evals)
Solution with 3 free parameters

Name	Form	Estimate	Lower CI	Upper CI
a1	LOGLIN	0.013608058384	0.009726885625	0.018329804328
a2	LOGLIN	0.000000057801	0.000000038830	0.000000081295
b	LOGLIN	0.204506899570	0.198999944893	0.209252565719

Model 1 Run 4 : SB79_sex.mle

METHOD = SIMPLEX MAXITER = 5000 MAXEVALS = 100000
Convergence at EPSILON = 0.0000010000
LogLikelihood: -864.1093 AIC: 1736.2185 Del(LL): 0.0000005904
Iterations: 592 Function evals: 948 Time: 00:00:01
Converged normally

Results with estimated standard errors. (39 evals)
Solution with 4 free parameters

Name	Form	Estimate	Std Error	t	against
a1	LOGLIN	0.016150366572	0.002683196448	6.01907720292	0.0
a2	LOGLIN	0.000000067335	0.000000011435	5.88851105612	0.0
b	LOGLIN	0.205640958863	0.003067676816	67.0347533904	0.0
b_sex		-0.34454068154	0.143327098993	-2.4038767544	0.0

Results with estimated standard errors. (40 evals)
Solution with 4 free parameters

Name	Form	Estimate	Std Error	t	against
a1	LOGLIN	0.016150366572	0.001942903668	8.31248961963	0.0
a2	LOGLIN	0.000000067335	0.000000002901	23.2120658734	0.0
b	LOGLIN	0.205640958863	0.002071885502	99.2530517100	0.0
b_sex		-0.34454068154	0.140750478119	-2.4478828502	0.0

Likelihood CI Results: (121 evals)
Solution with 4 free parameters

Name	Form	Estimate	Lower CI	Upper CI
a1	LOGLIN	0.016150366572	0.011533221158	0.021775509057
a2	LOGLIN	0.000000067335	0.000000045376	0.000000094491
b	LOGLIN	0.205640958863	0.200172511614	0.210359405236
b_sex		-0.34454068154	-0.66128510278	-0.05803830715

A.4 Gompertz Model, Coach Lane Only, Adult Individuals, Sex Covariate

136 lines read from file COL10_sex.dat
136 Observations kept and 0 observations dropped.

NAME	age	sex
MEAN	54.3522059	0.55882353
VAR	439.608588	0.24836601
STDEV	20.9668450	0.49836333
MIN	18.0000000	0.00000000
MAX	81.3000000	1.00000000

COL10_sex.mle
Program file: COL10_sex.mle
Input data file name: COL10_sex.dat
2 variables read.

Model 1 Run 1 : COL10_sex.mle

METHOD = SIMPLEX MAXITER = 5000 MAXEVALS = 100000
Convergence at EPSILON = 0.0000010000
LogLikelihood: -593.1107 AIC: 1190.2214 Del(LL): 0.0000007195
Iterations: 47 Function evals: 83 Time: 00:00:00
Converged normally

Results with estimated standard errors. (21 evals)
Solution with 2 free parameters

Name	Form	Estimate	Std Error	t	against
a2	LOGLIN	0.002394680529	0.000910087619	2.63126371476	0.0
b	LOGLIN	0.049187721174	0.009494677235	5.18055748035	0.0

Results with estimated standard errors. (12 evals)
Solution with 2 free parameters

Name	Form	Estimate	Std Error	t	against
a2	LOGLIN	0.002394680529	0.000697882948	3.43134981935	0.0
b	LOGLIN	0.049187721174	0.005124276246	9.59895970029	0.0

Likelihood CI Results: (59 evals)
Solution with 2 free parameters

Name	Form	Estimate	Lower CI	Upper CI
a2	LOGLIN	0.002394680529	0.001801255862	0.003105642651
b	LOGLIN	0.049187721174	0.044156837626	0.053738324317

Model 1 Run 2 : COL10_sex.mle

METHOD = SIMPLEX MAXITER = 5000 MAXEVALS = 100000
Convergence at EPSILON = 0.0000010000
LogLikelihood: -592.8189 AIC: 1191.6377 Del(LL): 0.0000006378
Iterations: 100 Function evals: 167 Time: 00:00:01
Converged normally

Results with estimated standard errors. (25 evals)
Solution with 3 free parameters

Name	Form	Estimate	Std Error	t	against
a2	LOGLIN	0.002549123762	0.001084365645	2.35079723634	0.0
b	LOGLIN	0.049438766490	0.009623235474	5.13743705252	0.0
b_sex		-0.13264357511	0.209135568716	-0.6342468473	0.0

Results with estimated standard errors. (24 evals)
Solution with 3 free parameters

Name	Form	Estimate	Std Error	t	against
a2	LOGLIN	0.002549123762	0.000769777509	3.31150719853	0.0
b	LOGLIN	0.049438766490	0.005134175893	9.62934802412	0.0
b_sex		-0.13264357511	0.173035062443	-0.7665705045	0.0

Likelihood CI Results: (92 evals)
Solution with 3 free parameters

Name	Form	Estimate	Lower CI	Upper CI
a2	LOGLIN	0.002549123762	0.001917667408	0.003306354090
b	LOGLIN	0.049438766490	0.044409189536	0.053990965385
b_sex		-0.13264357511	-0.51960148522	0.210279401340

Model 1 Run 3 : COL10_sex.mle

METHOD = SIMPLEX MAXITER = 5000 MAXEVALS = 100000
Convergence at EPSILON = 0.0000010000
LogLikelihood: -541.5537 AIC: 1089.1075 Del(LL): 0.0000007366
Iterations: 796 Function evals: 1332 Time: 00:00:00
Converged normally

Results with estimated standard errors. (34 evals)
Solution with 3 free parameters

Name	Form	Estimate	Std Error	t	against
a1	LOGLIN	0.013398036323	0.002083820846	6.42955288095	0.0
a2	LOGLIN	1.45647E-0013	7.90199E-0011	0.00184316404	0.0
b	LOGLIN	0.375355437484	0.001491874659	251.599848045	0.0

Results with estimated standard errors. (24 evals)

Solution with 3 free parameters

Name	Form	Estimate	Std Error	t	against
a1	LOGLIN	0.013398036323	0.001592278457	8.41438019995	0.0
a2	LOGLIN	1.45647E-0013	0.000000002450	0.00005944434	0.0
b	LOGLIN	0.375355437484	0.001880497682	199.604307446	0.0

Likelihood CI Results: (81 evals)
Solution with 3 free parameters

Name	Form	Estimate	Lower CI	Upper CI
a1	LOGLIN	0.013398036323	0.008967455965	0.019054703060
a2	LOGLIN	1.45647E-0013	9.04441E-0014	2.17737E-0013
b	LOGLIN	0.375355437484	0.369013414310	0.380719068935

Model 1 Run 4 : COL10_sex.mle

METHOD = SIMPLEX MAXITER = 5000 MAXEVALS = 100000
Convergence at EPSILON = 0.0000010000
LogLikelihood: -541.1165 AIC: 1090.2329 Del(LL): 0.0000007503
Iterations: 1069 Function evals: 1707 Time: 00:00:01
Converged normally

Results with estimated standard errors. (39 evals)
Solution with 4 free parameters

Name	Form	Estimate	Std Error	t	against
a1	LOGLIN	0.014627615397	0.002643742920	5.53291898668	0.0
a2	LOGLIN	1.51129E-0013	8.05888E-0011	0.00187530989	0.0
b	LOGLIN	0.376229453308	0.001975054258	190.490692515	0.0
b_sex		-0.16256958433	0.160497449261	-1.0129107040	0.0

Results with estimated standard errors. (40 evals)
Solution with 4 free parameters

Name	Form	Estimate	Std Error	t	against
a1	LOGLIN	0.014627615397	0.002183869148	6.69802740229	0.0
a2	LOGLIN	1.51129E-0013	0.000000002458	0.00006148555	0.0
b	LOGLIN	0.376229453308	0.002360030232	159.417217720	0.0
b_sex		-0.16256958433	0.173462336340	-0.9372039358	0.0

Likelihood CI Results: (114 evals)
Solution with 4 free parameters

Name	Form	Estimate	Lower CI	Upper CI
a1	LOGLIN	0.014627615397	0.009790361018	0.020805980636
a2	LOGLIN	1.51129E-0013	9.39114E-0014	2.25880E-0013
b	LOGLIN	0.376229453308	0.369895947676	0.381590319487
b_sex		-0.16256958433	-0.54965822673	0.180222647921

A.5 Gompertz Model, St. Peter's Only, Adult Individuals, Sex Covariate

69 lines read from file HCW01_sex.dat
69 Observations kept and 0 observations dropped.

NAME	age	sex
MEAN	41.2971014	0.50724638
VAR	354.504109	0.25362319
STDEV	18.8282795	0.50361016
MIN	18.2000000	0.00000000
MAX	77.8000000	1.00000000

HCW01_sex.mle
Program file: HCW01_sex.mle
Input data file name: HCW01_sex.dat
2 variables read.

Model 1 Run 1 : HCW01_sex.mle

METHOD = SIMPLEX MAXITER = 5000 MAXEVALS = 100000
Convergence at EPSILON = 0.0000010000
LogLikelihood: -293.5466 AIC: 591.09313 Del(LL): 0.0000009037
Iterations: 37 Function evals: 63 Time: 00:00:00
Converged normally

Results with estimated standard errors. (17 evals)
Solution with 2 free parameters

Name	Form	Estimate	Std Error	t	against
a2	LOGLIN	0.012538062637	0.006916290378	1.81283057127	0.0
b	LOGLIN	0.027231309397	0.013725251754	1.98402986585	0.0

Results with estimated standard errors. (12 evals)
Solution with 2 free parameters

Name	Form	Estimate	Std Error	t	against
a2	LOGLIN	0.012538062637	0.003648558509	3.43644280447	0.0
b	LOGLIN	0.027231309397	0.006415935514	4.24432404851	0.0

Likelihood CI Results: (56 evals)
Solution with 2 free parameters

Name	Form	Estimate	Lower CI	Upper CI
a2	LOGLIN	0.012538062637	0.008342704296	0.017951650469
b	LOGLIN	0.027231309397	0.018111424212	0.035029904067

Model 1 Run 2 : HCW01_sex.mle

METHOD = SIMPLEX MAXITER = 5000 MAXEVALS = 100000
Convergence at EPSILON = 0.0000010000
LogLikelihood: -293.3374 AIC: 592.67487 Del(LL): 0.0000002560
Iterations: 64 Function evals: 111 Time: 00:00:00
Converged normally

Results with estimated standard errors. (21 evals)
Solution with 3 free parameters

Name	Form	Estimate	Std Error	t	against
a2	LOGLIN	0.013515478886	0.007677957487	1.76029613453	0.0
b	LOGLIN	0.027403113760	0.013530448128	2.02529239979	0.0
b_sex		-0.15584719644	0.241083826215	-0.6464440145	0.0

Results with estimated standard errors. (24 evals)
Solution with 3 free parameters

Name	Form	Estimate	Std Error	t	against
a2	LOGLIN	0.013515478886	0.004208905680	3.21116221466	0.0
b	LOGLIN	0.027403113760	0.006420280756	4.26821112688	0.0
b_sex		-0.15584719644	0.241014217855	-0.6466307168	0.0

Likelihood CI Results: (88 evals)
Solution with 3 free parameters

Name	Form	Estimate	Lower CI	Upper CI
a2	LOGLIN	0.013515478886	0.008992367875	0.019349581886
b	LOGLIN	0.027403113760	0.018282651842	0.035200285028
b_sex		-0.15584719644	-0.74411786174	0.336043172926

Model 1 Run 3 : HCW01_sex.mle

METHOD = SIMPLEX MAXITER = 5000 MAXEVALS = 100000
Convergence at EPSILON = 0.0000010000
LogLikelihood: -293.5466 AIC: 593.09314 Del(LL): 0.0000009797
Iterations: 192 Function evals: 262 Time: 00:00:00
Converged normally

Results with estimated standard errors. (26 evals)
Solution with 3 free parameters

Name	Form	Estimate	Std Error	t	against
a1	LOGLIN	0.000000067930	0.086720681003	0.00000078332	0.0
a2	LOGLIN	0.012543643334	0.065414849945	0.19175528713	0.0
b	LOGLIN	0.027231924950	0.068234902139	0.39909084788	0.0

Results with estimated standard errors. (24 evals)

Solution with 3 free parameters

Name	Form	Estimate	Std Error	t	against
a1	LOGLIN	0.000000067930	0.051921142071	0.00000130833	0.0
a2	LOGLIN	0.012543643334	0.035378783053	0.35455270791	0.0
b	LOGLIN	0.027231924950	0.032226002998	0.84502955428	0.0

Likelihood CI Results: (67 evals)
Solution with 3 free parameters

Name	Form	Estimate	Lower CI	Upper CI
a1	LOGLIN	0.000000067930	1.08420E-0019	0.013511592185
a2	LOGLIN	0.012543643334	0.008342466163	0.017951171914
b	LOGLIN	0.027231924950	0.018102193412	0.035021171927

Model 1 Run 4 : HCW01_sex.mle

METHOD = SIMPLEX MAXITER = 5000 MAXEVALS = 100000
Convergence at EPSILON = 0.0000010000
LogLikelihood: -293.3374 AIC: 594.67487 Del(LL): 0.0000007635
Iterations: 171 Function evals: 224 Time: 00:00:00
Converged normally

Results with estimated standard errors. (31 evals)
Solution with 4 free parameters

Name	Form	Estimate	Std Error	t	against
a1	LOGLIN	0.000000008214	0.089871479858	0.00000009139	0.0
a2	LOGLIN	0.013519263832	0.067933054055	0.19900862724	0.0
b	LOGLIN	0.027401026659	0.065261718423	0.41986370143	0.0
b_sex		-0.15611586614	0.250219987013	-0.6239144522	0.0

Results with estimated standard errors. (40 evals)
Solution with 4 free parameters

Name	Form	Estimate	Std Error	t	against
a1	LOGLIN	0.000000008214	0.052162355239	0.00000015747	0.0
a2	LOGLIN	0.013519263832	0.035237626271	0.38365989037	0.0
b	LOGLIN	0.027401026659	0.030001117926	0.91333352066	0.0
b_sex		-0.15611586614	0.242441492672	-0.6439321274	0.0

Likelihood CI Results: (99 evals)
Solution with 4 free parameters

Name	Form	Estimate	Lower CI	Upper CI
a1	LOGLIN	0.000000008214	1.08420E-0019	0.014637486000
a2	LOGLIN	0.013519263832	0.008994365729	0.019353884099
b	LOGLIN	0.027401026659	0.018279728452	0.035197526096
b_sex		-0.15611586614	-0.74431026235	0.335850771842

A.6 Gompertz Model, New Bunhill Fields Only, Adult Individuals, Sex Covariate

124 lines read from file DVL05_sex.dat
124 Observations kept and 0 observations dropped.

NAME	age	sex
MEAN	37.8459677	0.51612903
VAR	271.317301	0.25177026
STDEV	16.4717121	0.50176714
MIN	18.0000000	0.00000000
MAX	78.1000000	1.00000000

DVL05_sex.mle
Program file: DVL05_sex.mle
Input data file name: DVL05_sex.dat
2 variables read.

Model 1 Run 1 : DVL05_sex.mle

METHOD = SIMPLEX MAXITER = 5000 MAXEVALS = 100000
Convergence at EPSILON = 0.0000010000
LogLikelihood: -513.0177 AIC: 1030.0355 Del(LL): 0.0000007672
Iterations: 36 Function evals: 65 Time: 00:00:00
Converged normally

Results with estimated standard errors. (17 evals)
Solution with 2 free parameters

Name	Form	Estimate	Std Error	t	against
a2	LOGLIN	0.014931202385	0.005283801556	2.82584465496	0.0
b	LOGLIN	0.028193851993	0.008504132470	3.31531194872	0.0

Results with estimated standard errors. (12 evals)
Solution with 2 free parameters

Name	Form	Estimate	Std Error	t	against
a2	LOGLIN	0.014931202385	0.003151790902	4.73737086253	0.0
b	LOGLIN	0.028193851993	0.005047865856	5.58530135237	0.0

Likelihood CI Results: (60 evals)
Solution with 2 free parameters

Name	Form	Estimate	Lower CI	Upper CI
a2	LOGLIN	0.014931202385	0.011074694622	0.019594758919
b	LOGLIN	0.028193851993	0.020957463335	0.034614030689

Model 1 Run 2 : DVL05_sex.mle

METHOD = SIMPLEX MAXITER = 5000 MAXEVALS = 100000
Convergence at EPSILON = 0.0000010000
LogLikelihood: -511.9457 AIC: 1029.8914 Del(LL): 0.0000005817
Iterations: 82 Function evals: 135 Time: 00:00:00
Converged normally

Results with estimated standard errors. (21 evals)
Solution with 3 free parameters

Name	Form	Estimate	Std Error	t	against
a2	LOGLIN	0.017089462721	0.006271613678	2.72489084918	0.0
b	LOGLIN	0.028460538276	0.008343852548	3.41095892017	0.0
b_sex		-0.26416638564	0.174027090360	-1.5179612846	0.0

Results with estimated standard errors. (24 evals)
Solution with 3 free parameters

Name	Form	Estimate	Std Error	t	against
a2	LOGLIN	0.017089462721	0.003886906634	4.39667435592	0.0
b	LOGLIN	0.028460538276	0.005034606814	5.65298132022	0.0
b_sex		-0.26416638564	0.179812864940	-1.4691183844	0.0

Likelihood CI Results: (93 evals)
Solution with 3 free parameters

Name	Form	Estimate	Lower CI	Upper CI
a2	LOGLIN	0.017089462721	0.012674081564	0.022424598126
b	LOGLIN	0.028460538276	0.021225171069	0.034873269777
b_sex		-0.26416638564	-0.68839931736	0.107516873275

Model 1 Run 3 : DVL05_sex.mle

METHOD = SIMPLEX MAXITER = 5000 MAXEVALS = 100000
Convergence at EPSILON = 0.0000010000
LogLikelihood: -513.0188 AIC: 1032.0375 Del(LL): 0.0000007866
Iterations: 115 Function evals: 142 Time: 00:00:00
Converged normally

Results with estimated standard errors. (26 evals)
Solution with 3 free parameters

Name	Form	Estimate	Std Error	t	against
a1	LOGLIN	0.000000009490	0.060051616223	0.00000015803	0.0
a2	LOGLIN	0.014944590304	0.044675831092	0.33451174692	0.0
b	LOGLIN	0.028079062187	0.038965470478	0.72061396520	0.0

Results with estimated standard errors. (24 evals)

Solution with 3 free parameters

Name	Form	Estimate	Std Error	t	against
a1	LOGLIN	0.000000009490	0.019880717273	0.00000047736	0.0
a2	LOGLIN	0.014944590304	0.012866464503	1.16151490565	0.0
b	LOGLIN	0.028079062187	0.010155081098	2.76502589364	0.0

Likelihood CI Results: (72 evals)
Solution with 3 free parameters

Name	Form	Estimate	Lower CI	Upper CI
a1	LOGLIN	0.000000009490	1.08420E-0019	0.010743798916
a2	LOGLIN	0.014944590304	0.011122623249	0.019680407035
b	LOGLIN	0.028079062187	0.020936834630	0.034595660116

Model 1 Run 4 : DVL05_sex.mle

METHOD = SIMPLEX MAXITER = 5000 MAXEVALS = 100000
Convergence at EPSILON = 0.0000010000
LogLikelihood: -511.9457 AIC: 1031.8914 Del(LL): 0.0000009215
Iterations: 159 Function evals: 202 Time: 00:00:00
Converged normally

Results with estimated standard errors. (31 evals)
Solution with 4 free parameters

Name	Form	Estimate	Std Error	t	against
a1	LOGLIN	0.000000006583	0.063875765626	0.00000010305	0.0
a2	LOGLIN	0.017087809403	0.047298427531	0.36127647990	0.0
b	LOGLIN	0.028455862254	0.035659390064	0.79799071725	0.0
b_sex		-0.26400698456	0.180929303865	-1.4591720574	0.0

Results with estimated standard errors. (40 evals)
Solution with 4 free parameters

Name	Form	Estimate	Std Error	t	against
a1	LOGLIN	0.000000006583	0.025458281906	0.00000025856	0.0
a2	LOGLIN	0.017087809403	0.016398908977	1.04200891826	0.0
b	LOGLIN	0.028455862254	0.011382571144	2.49995030935	0.0
b_sex		-0.26400698456	0.180849442312	-1.4598164151	0.0

Likelihood CI Results: (104 evals)
Solution with 4 free parameters

Name	Form	Estimate	Lower CI	Upper CI
a1	LOGLIN	0.000000006583	1.08420E-0019	0.012225177600
a2	LOGLIN	0.017087809403	0.012675279195	0.022426719654
b	LOGLIN	0.028455862254	0.021225459022	0.034873544962
b_sex		-0.26400698456	-0.68812286782	0.107793347271

A.7 Gompertz Model, High SES (St. Bride's) Only, Adult Individuals, Sex Covariate

208 lines read from file SEShigh_sex.dat
208 Observations kept and 0 observations dropped.

NAME	age	sex
MEAN	53.3610577	0.53365385
VAR	368.630602	0.25006968
STDEV	19.1997553	0.50006967
MIN	18.0000000	0.00000000
MAX	79.9000000	1.00000000

SEShigh_sex.mle
Program file: SEShigh_sex.mle
Input data file name: SEShigh_sex.dat
2 variables read.

Model 1 Run 1 : SEShigh_sex.mle

METHOD = SIMPLEX MAXITER = 5000 MAXEVALS = 100000
Convergence at EPSILON = 0.0000010000
LogLikelihood: -890.7140 AIC: 1785.4280 Del(LL): 0.0000003863
Iterations: 48 Function evals: 80 Time: 00:00:00
Converged normally

Results with estimated standard errors. (21 evals)
Solution with 2 free parameters

Name	Form	Estimate	Std Error	t	against
a2	LOGLIN	0.001968936496	0.000516018185	3.81563393144	0.0
b	LOGLIN	0.055245205044	0.005865806982	9.41817642637	0.0

Results with estimated standard errors. (12 evals)
Solution with 2 free parameters

Name	Form	Estimate	Std Error	t	against
a2	LOGLIN	0.001968936496	0.000475407022	4.14158059041	0.0
b	LOGLIN	0.055245205044	0.004334390958	12.7457826450	0.0

Likelihood CI Results: (61 evals)
Solution with 2 free parameters

Name	Form	Estimate	Lower CI	Upper CI
a2	LOGLIN	0.001968936496	0.001567358093	0.002434051007
b	LOGLIN	0.055245205044	0.051134708077	0.059037695859

Model 1 Run 2 : SEShigh_sex.mle

METHOD = SIMPLEX MAXITER = 5000 MAXEVALS = 100000
Convergence at EPSILON = 0.0000010000
LogLikelihood: -888.4048 AIC: 1782.8096 Del(LL): 0.0000004983
Iterations: 88 Function evals: 148 Time: 00:00:00
Converged normally

Results with estimated standard errors. (25 evals)
Solution with 3 free parameters

Name	Form	Estimate	Std Error	t	against
a2	LOGLIN	0.002210483019	0.000622237052	3.55247732520	0.0
b	LOGLIN	0.056308364140	0.005821232392	9.67292840219	0.0
b_sex		-0.30232405929	0.159997968860	-1.8895493577	0.0

Results with estimated standard errors. (24 evals)
Solution with 3 free parameters

Name	Form	Estimate	Std Error	t	against
a2	LOGLIN	0.002210483019	0.000545897097	4.04926685408	0.0
b	LOGLIN	0.056308364140	0.004380074412	12.8555724944	0.0
b_sex		-0.30232405929	0.139919970549	-2.1606927025	0.0

Likelihood CI Results: (95 evals)
Solution with 3 free parameters

Name	Form	Estimate	Lower CI	Upper CI
a2	LOGLIN	0.002210483019	0.001759620727	0.002732627985
b	LOGLIN	0.056308364140	0.052196845936	0.060102331426
b_sex		-0.30232405929	-0.61899007785	-0.01574326360

Model 1 Run 3 : SEShigh_sex.mle

METHOD = SIMPLEX MAXITER = 5000 MAXEVALS = 100000
Convergence at EPSILON = 0.0000010000
LogLikelihood: -867.0680 AIC: 1740.1359 Del(LL): 0.0000009649
Iterations: 386 Function evals: 644 Time: 00:00:00
Converged normally

Results with estimated standard errors. (34 evals)
Solution with 3 free parameters

Name	Form	Estimate	Std Error	t	against
a1	LOGLIN	0.013608058384	0.002077899589	6.54894897654	0.0
a2	LOGLIN	0.000000057801	0.000000010010	5.77460799580	0.0
b	LOGLIN	0.204506899570	0.002992448222	68.3409985450	0.0

Results with estimated standard errors. (24 evals)

Solution with 3 free parameters

Name	Form	Estimate	Std Error	t	against
a1	LOGLIN	0.013608058384	0.001372838835	9.91234953020	0.0
a2	LOGLIN	0.000000057801	0.000000002889	20.0065655825	0.0
b	LOGLIN	0.204506899570	0.001674926440	122.099033563	0.0

Likelihood CI Results: (87 evals)
Solution with 3 free parameters

Name	Form	Estimate	Lower CI	Upper CI
a1	LOGLIN	0.013608058384	0.009726885625	0.018329804328
a2	LOGLIN	0.000000057801	0.000000038830	0.000000081295
b	LOGLIN	0.204506899570	0.198999944893	0.209252565719

Model 1 Run 4 : SEShigh_sex.mle

METHOD = SIMPLEX MAXITER = 5000 MAXEVALS = 100000
Convergence at EPSILON = 0.0000010000
LogLikelihood: -864.1093 AIC: 1736.2185 Del(LL): 0.0000005904
Iterations: 592 Function evals: 948 Time: 00:00:01
Converged normally

Results with estimated standard errors. (39 evals)
Solution with 4 free parameters

Name	Form	Estimate	Std Error	t	against
a1	LOGLIN	0.016150366572	0.002683196448	6.01907720292	0.0
a2	LOGLIN	0.000000067335	0.000000011435	5.88851105612	0.0
b	LOGLIN	0.205640958863	0.003067676816	67.0347533904	0.0
b_sex		-0.34454068154	0.143327098993	-2.4038767544	0.0

Results with estimated standard errors. (40 evals)
Solution with 4 free parameters

Name	Form	Estimate	Std Error	t	against
a1	LOGLIN	0.016150366572	0.001942903668	8.31248961963	0.0
a2	LOGLIN	0.000000067335	0.000000002901	23.2120658734	0.0
b	LOGLIN	0.205640958863	0.002071885502	99.2530517100	0.0
b_sex		-0.34454068154	0.140750478119	-2.4478828502	0.0

Likelihood CI Results: (121 evals)
Solution with 4 free parameters

Name	Form	Estimate	Lower CI	Upper CI
a1	LOGLIN	0.016150366572	0.011533221158	0.021775509057
a2	LOGLIN	0.000000067335	0.000000045376	0.000000094491
b	LOGLIN	0.205640958863	0.200172511614	0.210359405236
b_sex		-0.34454068154	-0.66128510278	-0.05803830715

A.8 Gompertz Model, Low SES (Coach Lane, St. Peter's, New Bunhill) Only, Adult Individuals, Sex Covariate

329 lines read from file SESlow_sex.dat
329 Observations kept and 0 observations dropped.

NAME	age	sex
MEAN	45.3930091	0.53191489
VAR	414.518762	0.24974053
STDEV	20.3597338	0.49974046
MIN	18.0000000	0.00000000
MAX	81.3000000	1.00000000

SESlow_sex.mle
Program file: SESlow_sex.mle
Input data file name: SESlow_sex.dat
2 variables read.

Model 1 Run 1 : SESlow_sex.mle

METHOD = SIMPLEX MAXITER = 5000 MAXEVALS = 100000
Convergence at EPSILON = 0.0000010000
LogLikelihood: -1427.656 AIC: 2859.3122 Del(LL): 0.0000007922
Iterations: 40 Function evals: 69 Time: 00:00:00
Converged normally

Results with estimated standard errors. (20 evals)
Solution with 2 free parameters

Name	Form	Estimate	Std Error	t	against
a2	LOGLIN	0.008553031324	0.002033614538	4.20582719215	0.0
b	LOGLIN	0.031327108834	0.006225096341	5.03238939878	0.0

Results with estimated standard errors. (12 evals)
Solution with 2 free parameters

Name	Form	Estimate	Std Error	t	against
a2	LOGLIN	0.008553031324	0.001236228733	6.91864789711	0.0
b	LOGLIN	0.031327108834	0.002943549206	10.6426312734	0.0

Likelihood CI Results: (63 evals)
Solution with 2 free parameters

Name	Form	Estimate	Lower CI	Upper CI
a2	LOGLIN	0.008553031324	0.007144504876	0.010136635633
b	LOGLIN	0.031327108834	0.027642083419	0.034767676199

Model 1 Run 2 : SESlow_sex.mle

METHOD = SIMPLEX MAXITER = 5000 MAXEVALS = 100000
Convergence at EPSILON = 0.0000010000
LogLikelihood: -1426.355 AIC: 2858.7109 Del(LL): 0.0000008357
Iterations: 110 Function evals: 187 Time: 00:00:00
Converged normally

Results with estimated standard errors. (23 evals)
Solution with 3 free parameters

Name	Form	Estimate	Std Error	t	against
a2	LOGLIN	0.009340403286	0.002323551477	4.01988222616	0.0
b	LOGLIN	0.031571579261	0.006141524173	5.14067491546	0.0
b_sex		-0.17883004070	0.115596895828	-1.5470142119	0.0

Results with estimated standard errors. (24 evals)
Solution with 3 free parameters

Name	Form	Estimate	Std Error	t	against
a2	LOGLIN	0.009340403286	0.001434885076	6.50951315844	0.0
b	LOGLIN	0.031571579261	0.002947949697	10.7096736753	0.0
b_sex		-0.17883004070	0.110636327138	-1.6163772364	0.0

Likelihood CI Results: (98 evals)
Solution with 3 free parameters

Name	Form	Estimate	Lower CI	Upper CI
a2	LOGLIN	0.009340403286	0.007801546387	0.011068847081
b	LOGLIN	0.031571579261	0.027884874759	0.035010442257
b_sex		-0.17883004070	-0.42846874006	0.051528201435

Model 1 Run 3 : SESlow_sex.mle

METHOD = SIMPLEX MAXITER = 5000 MAXEVALS = 100000
Convergence at EPSILON = 0.0000010000
LogLikelihood: -1362.488 AIC: 2730.9769 Del(LL): 0.0000004647
Iterations: 641 Function evals: 1076 Time: 00:00:01
Converged normally

Results with estimated standard errors. (34 evals)
Solution with 3 free parameters

Name	Form	Estimate	Std Error	t	against
a1	LOGLIN	0.022657708417	0.002273816189	9.96461742603	0.0
a2	LOGLIN	1.53613E-0013	7.29146E-0011	0.00210675543	0.0
b	LOGLIN	0.375482162206	0.001343594126	279.461003056	0.0

Results with estimated standard errors. (24 evals)
Solution with 3 free parameters

Name	Form	Estimate	Std Error	t	against
a1	LOGLIN	0.022657708417	0.001478539568	15.3243842114	0.0
a2	LOGLIN	1.53613E-0013	0.000000001759	0.00008732733	0.0
b	LOGLIN	0.375482162206	0.001680097464	223.488321544	0.0

Likelihood CI Results: (88 evals)
Solution with 3 free parameters

Name	Form	Estimate	Lower CI	Upper CI
a1	LOGLIN	0.022657708417	0.018300056940	0.027643122145
a2	LOGLIN	1.53613E-0013	1.01070E-0013	2.19463E-0013
b	LOGLIN	0.375482162206	0.369902544289	0.380249485139

Model 1 Run 4 : SESlow_sex.mle

METHOD = SIMPLEX MAXITER = 5000 MAXEVALS = 100000
Convergence at EPSILON = 0.0000010000
LogLikelihood: -1367.608 AIC: 2743.2158 Del(LL): 0.0000008916
Iterations: 804 Function evals: 1326 Time: 00:00:01
Converged normally

Results with estimated standard errors. (43 evals)
Solution with 4 free parameters

Name	Form	Estimate	Std Error	t	against
a1	LOGLIN	0.018345085889	0.002146453037	8.54669800302	0.0
a2	LOGLIN	9.55930E-0013	7.32192E-0011	0.01305572885	0.0
b	LOGLIN	0.349697213992	0.001875413456	186.464063646	0.0
b_sex		0.180579533192	0.120951999194	1.49298510479	0.0

Results with estimated standard errors. (40 evals)
Solution with 4 free parameters

Name	Form	Estimate	Std Error	t	against
a1	LOGLIN	0.018345085889	0.001644674179	11.1542371870	0.0
a2	LOGLIN	9.55930E-0013	0.000000001744	0.00054822282	0.0
b	LOGLIN	0.349697213992	0.001898133321	184.232166479	0.0
b_sex		0.180579533192	0.111915020767	1.61354152422	0.0

Likelihood CI Results: (122 evals)
Solution with 4 free parameters

Name	Form	Estimate	Lower CI	Upper CI
a1	LOGLIN	0.018345085889	0.015978524533	0.025273161639
a2	LOGLIN	9.55930E-0013	6.35130E-0013	1.35675E-0012
b	LOGLIN	0.349697213992	0.344271765713	0.354434908514
b_sex		0.180579533192	-0.16022764882	0.345013322915

APPENDIX B

HIERARCHICAL LOG-LINEAR ANALYSIS RESULTS

B.1 Cribra Orbitalia Presence, Full Sample (All Four Sites), Adult Individuals

Step[a]		Effects	Chi-Square[c]	df	Sig.	Number of Iterations
0	Generating Class[b]	SES*Sex*Cribra_presence*Age_Cat_Collapsed	.000	0	.	
	Deleted Effect 1	SES*Sex*Cribra_presence*Age_Cat_Collapsed	2.701	4	.609	4
1	Generating Class[b]	SES*Sex*Cribra_presence, SES*Sex*Age_Cat_Collapsed, SES*Cribra_presence*Age_Cat_Collapsed, Sex*Cribra_presence*Age_Cat_Collapsed	2.701	4	.609	
	Deleted Effect 1	SES*Sex*Cribra_presence	9.591	1	.002	3
	2	SES*Sex*Age_Cat_Collapsed	.770	4	.942	4
	3	SES*Cribra_presence*Age_Cat_Collapsed	2.606	4	.626	4
	4	Sex*Cribra_presence*Age_Cat_Collapsed	6.805	4	.147	4
2	Generating Class[b]	SES*Sex*Cribra_presence, SES*Cribra_presence*Age_Cat_Collapsed, Sex*Cribra_presence*Age_Cat_Collapsed	3.471	8	.901	
	Deleted Effect 1	SES*Sex*Cribra_presence	9.104	1	.003	3
	2	SES*Cribra_presence*Age_Cat_Collapsed	2.037	4	.729	4
	3	Sex*Cribra_presence*Age_Cat_Collapsed	6.807	4	.146	3
3	Generating Class[b]	SES*Sex*Cribra_presence, Sex*Cribra_presence*Age_Cat_Collapsed, SES*Age_Cat_Collapsed	5.508	12	.939	
	Deleted Effect 1	SES*Sex*Cribra_presence	10.021	1	.002	4
	2	Sex*Cribra_presence*Age_Cat_Collapsed	7.773	4	.100	4
	3	SES*Age_Cat_Collapsed	38.754	4	.000	2
4	Generating Class[b]	SES*Sex*Cribra_presence, SES*Age_Cat_Collapsed, Sex*Age_Cat_Collapsed, Cribra_presence*Age_Cat_Collapsed	13.281	16	.652	
	Deleted Effect 1	SES*Sex*Cribra_presence	8.204	1	.004	4
	2	SES*Age_Cat_Collapsed	36.940	4	.000	4
	3	Sex*Age_Cat_Collapsed	13.304	4	.010	3
	4	Cribra_presence*Age_Cat_Collapsed	18.340	4	.001	3

5	Generating Class[b]	SES*Sex*Cribra_presence, SES*Age_Cat_Collapsed, Sex*Age_Cat_Collapsed, Cribra_presence*Age_Cat_Collapsed	13.281	16	.652

a. At each step, the effect with the largest significance level for the Likelihood Ratio Change is deleted, provided the significance level is larger than .050.
b. Statistics are displayed for the best model at each step after step 0.
c. For 'Deleted Effect', this is the change in the Chi-Square after the effect is deleted from the model.

B.2 Cribra Orbitalia Presence, Reduced Sample (London Sites), Adult Individuals

Step[a]		Effects	Chi-Square[c]	df	Sig.	Number of Iterations
0	Generating Class[b]	SES*Sex*Cribra_presence*Age_Cat_Collapsed	.000	0	.	
	Deleted Effect 1	SES*Sex*Cribra_presence*Age_Cat_Collapsed	6.731	4	.151	4
1	Generating Class[b]	SES*Sex*Cribra_presence, SES*Sex*Age_Cat_Collapsed, SES*Cribra_presence*Age_Cat_Collapsed, Sex*Cribra_presence*Age_Cat_Collapsed	6.731	4	.151	
	Deleted Effect 1	SES*Sex*Cribra_presence	8.996	1	.003	3
	2	SES*Sex*Age_Cat_Collapsed	.919	4	.922	4
	3	SES*Cribra_presence*Age_Cat_Collapsed	4.250	4	.373	4
	4	Sex*Cribra_presence*Age_Cat_Collapsed	2.471	4	.650	4
2	Generating Class[b]	SES*Sex*Cribra_presence, SES*Cribra_presence*Age_Cat_Collapsed, Sex*Cribra_presence*Age_Cat_Collapsed	7.650	8	.468	
	Deleted Effect 1	SES*Sex*Cribra_presence	10.000	1	.002	2
	2	SES*Cribra_presence*Age_Cat_Collapsed	4.948	4	.293	4
	3	Sex*Cribra_presence*Age_Cat_Collapsed	2.549	4	.636	4
3	Generating Class[b]	SES*Sex*Cribra_presence, SES*Cribra_presence*Age_Cat_Collapsed, Sex*Age_Cat_Collapsed	10.199	12	.599	
	Deleted Effect 1	SES*Sex*Cribra_presence	9.469	1	.002	3
	2	SES*Cribra_presence*Age_Cat_Collapsed	4.562	4	.335	5
	3	Sex*Age_Cat_Collapsed	12.026	4	.017	2
4	Generating Class[b]	SES*Sex*Cribra_presence, Sex*Age_Cat_Collapsed, SES*Age_Cat_Collapsed, Cribra_presence*Age_Cat_Collapsed	14.761	16	.542	
	Deleted Effect 1	SES*Sex*Cribra_presence	8.325	1	.004	3
	2	Sex*Age_Cat_Collapsed	10.883	4	.028	4
	3	SES*Age_Cat_Collapsed	53.842	4	.000	4
	4	Cribra_presence*Age_Cat_Collapsed	11.177	4	.025	3
5	Generating Class[b]	SES*Sex*Cribra_presence, Sex*Age_Cat_Collapsed, SES*Age_Cat_Collapsed, Cribra_presence*Age_Cat_Collapsed	14.761	16	.542	

a. At each step, the effect with the largest significance level for the Likelihood Ratio Change is deleted, provided the significance level is larger than .050.
b. Statistics are displayed for the best model at each step after step 0.
c. For 'Deleted Effect', this is the change in the Chi-Square after the effect is deleted from the model.

B.3 Linear Enamel Hypoplasia Presence, Full Sample (All Four Sites), Adult Individuals

Step[a]		Effects	Chi-Square[c]	df	Sig.	Number of Iterations
0	Generating Class[b]	SES*Sex*Age_Cat_Coll*EHpresence_LorR	.000	0	.	
	Deleted Effect 1	SES*Sex*Age_Cat_Coll*EHpresence_LorR	2.619	4	.623	3
1	Generating Class[b]	SES*Sex*Age_Cat_Coll, SES*Sex*EHpresence_LorR, SES*Age_Cat_Coll*EHpresence_LorR, Sex*Age_Cat_Coll*EHpresence_LorR	2.619	4	.623	
	Deleted Effect 1	SES*Sex*Age_Cat_Coll	.761	4	.944	3
	2	SES*Sex*EHpresence_LorR	.018	1	.893	2
	3	SES*Age_Cat_Coll*EHpresence_LorR	.532	4	.970	3
	4	Sex*Age_Cat_Coll*EHpresence_LorR	.431	4	.980	3
2	Generating Class[b]	SES*Sex*Age_Cat_Coll, SES*Sex*EHpresence_LorR, SES*Age_Cat_Coll*EHpresence_LorR	3.050	8	.931	
	Deleted Effect 1	SES*Sex*Age_Cat_Coll	.724	4	.948	3
	2	SES*Sex*EHpresence_LorR	.112	1	.738	2
	3	SES*Age_Cat_Coll*EHpresence_LorR	.474	4	.976	3
3	Generating Class[b]	SES*Sex*Age_Cat_Coll, SES*Sex*EHpresence_LorR, Age_Cat_Coll*EHpresence_LorR	3.524	12	.991	
	Deleted Effect 1	SES*Sex*Age_Cat_Coll	.715	4	.949	3
	2	SES*Sex*EHpresence_LorR	.152	1	.696	3
	3	Age_Cat_Coll*EHpresence_LorR	1.164	4	.884	2
4	Generating Class[b]	SES*Sex*EHpresence_LorR, Age_Cat_Coll*EHpresence_LorR, SES*Age_Cat_Coll, Sex*Age_Cat_Coll	4.239	16	.998	
	Deleted Effect 1	SES*Sex*EHpresence_LorR	.178	1	.673	3
	2	Age_Cat_Coll*EHpresence_LorR	1.189	4	.880	3
	3	SES*Age_Cat_Coll	26.194	4	.000	3
	4	Sex*Age_Cat_Coll	9.499	4	.050	3
5	Generating Class[b]	SES*Sex*EHpresence_LorR, SES*Age_Cat_Coll, Sex*Age_Cat_Coll	5.428	20	.999	
	Deleted Effect 1	SES*Sex*EHpresence_LorR	.133	1	.716	2
	2	SES*Age_Cat_Coll	26.581	4	.000	2
	3	Sex*Age_Cat_Coll	9.371	4	.052	2
6	Generating Class[b]	SES*Age_Cat_Coll, Sex*Age_Cat_Coll, SES*Sex, SES*EHpresence_LorR, Sex*EHpresence_LorR	5.561	21	1.000	

	Deleted Effect	1	SES*Age_Cat_Coll	26.581	4	.000	2
		2	Sex*Age_Cat_Coll	9.371	4	.052	2
		3	SES*Sex	.017	1	.895	2
		4	SES*EHpresence_LorR	.833	1	.362	2
		5	Sex*EHpresence_LorR	.131	1	.717	2
7	Generating Class[b]		SES*Age_Cat_Coll, Sex*Age_Cat_Coll, SES*EHpresence_LorR, Sex*EHpresence_LorR	5.578	22	1.000	
	Deleted Effect	1	SES*Age_Cat_Coll	26.658	4	.000	2
		2	Sex*Age_Cat_Coll	9.447	4	.051	2
		3	SES*EHpresence_LorR	.828	1	.363	2
		4	Sex*EHpresence_LorR	.127	1	.722	2
8	Generating Class[b]		SES*Age_Cat_Coll, Sex*Age_Cat_Coll, SES*EHpresence_LorR	5.705	23	1.000	
	Deleted Effect	1	SES*Age_Cat_Coll	26.651	4	.000	2
		2	Sex*Age_Cat_Coll	9.440	4	.051	2
		3	SES*EHpresence_LorR	.822	1	.365	2
9	Generating Class[b]		SES*Age_Cat_Coll, Sex*Age_Cat_Coll, EHpresence_LorR	6.526	24	1.000	
	Deleted Effect	1	SES*Age_Cat_Coll	26.651	4	.000	2
		2	Sex*Age_Cat_Coll	9.440	4	.051	2
		3	EHpresence_LorR	23.837	1	.000	2
10	Generating Class[b]		SES*Age_Cat_Coll, EHpresence_LorR, Sex	15.967	28	.966	

a. At each step, the effect with the largest significance level for the Likelihood Ratio Change is deleted, provided the significance level is larger than .050.
b. Statistics are displayed for the best model at each step after step 0.
c. For 'Deleted Effect', this is the change in the Chi-Square after the effect is deleted from the model.

B.4 Linear Enamel Hypoplasia Presence, Reduced Sample (London Sites), Adult Individuals

Step[a]		Effects	Chi-Square[c]	df	Sig.	Number of Iterations
0	Generating Class[b]	SES*Sex*EHpresence_LorR*Age_Cat_Coll	.000	0	.	
	Deleted Effect 1	SES*Sex*EHpresence_LorR*Age_Cat_Coll	7.997	4	.092	3
1	Generating Class[b]	SES*Sex*EHpresence_LorR, SES*Sex*Age_Cat_Coll, SES*EHpresence_LorR*Age_Cat_Coll, Sex*EHpresence_LorR*Age_Cat_Coll	7.997	4	.092	
	Deleted Effect 1	SES*Sex*EHpresence_LorR	.371	1	.543	2
	2	SES*Sex*Age_Cat_Coll	.617	4	.961	3
	3	SES*EHpresence_LorR*Age_Cat_Coll	4.176	4	.383	3
	4	Sex*EHpresence_LorR*Age_Cat_Coll	.535	4	.970	2
2	Generating Class[b]	SES*Sex*EHpresence_LorR, SES*Sex*Age_Cat_Coll, SES*EHpresence_LorR*Age_Cat_Coll	8.532	8	.383	
	Deleted Effect 1	SES*Sex*EHpresence_LorR	.823	1	.364	2
	2	SES*Sex*Age_Cat_Coll	.667	4	.955	3
	3	SES*EHpresence_LorR*Age_Cat_Coll	4.210	4	.378	3
3	Generating Class[b]	SES*Sex*EHpresence_LorR, SES*EHpresence_LorR*Age_Cat_Coll, Sex*Age_Cat_Coll	9.199	12	.686	
	Deleted Effect 1	SES*Sex*EHpresence_LorR	.801	1	.371	2
	2	SES*EHpresence_LorR*Age_Cat_Coll	4.248	4	.373	3
	3	Sex*Age_Cat_Coll	5.625	4	.229	2
4	Generating Class[b]	SES*Sex*EHpresence_LorR, Sex*Age_Cat_Coll, SES*Age_Cat_Coll, EHpresence_LorR*Age_Cat_Coll	13.447	16	.640	
	Deleted Effect 1	SES*Sex*EHpresence_LorR	.998	1	.318	3
	2	Sex*Age_Cat_Coll	5.823	4	.213	3
	3	SES*Age_Cat_Coll	41.135	4	.000	2
	4	EHpresence_LorR*Age_Cat_Coll	1.264	4	.868	3
5	Generating Class[b]	SES*Sex*EHpresence_LorR, Sex*Age_Cat_Coll, SES*Age_Cat_Coll	14.710	20	.793	
	Deleted Effect 1	SES*Sex*EHpresence_LorR	.864	1	.353	2
	2	Sex*Age_Cat_Coll	5.697	4	.223	2
	3	SES*Age_Cat_Coll	41.618	4	.000	2
6	Generating Class[b]	Sex*Age_Cat_Coll, SES*Age_Cat_Coll, SES*Sex, SES*EHpresence_LorR, Sex*EHpresence_LorR	15.575	21	.793	

	Deleted Effect	1	Sex*Age_Cat_Coll	5.696	4	.223	2
		2	SES*Age_Cat_Coll	41.617	4	.000	2
		3	SES*Sex	.043	1	.835	2
		4	SES*EHpresence_LorR	1.004	1	.316	2
		5	Sex*EHpresence_LorR	.008	1	.928	2
7	Generating Class[b]		Sex*Age_Cat_Coll, SES*Age_Cat_Coll, SES*Sex, SES*EHpresence_LorR	15.583	22	.836	
	Deleted Effect	1	Sex*Age_Cat_Coll	5.697	4	.223	2
		2	SES*Age_Cat_Coll	41.617	4	.000	2
		3	SES*Sex	.041	1	.839	2
		4	SES*EHpresence_LorR	.999	1	.317	2
8	Generating Class[b]		Sex*Age_Cat_Coll, SES*Age_Cat_Coll, SES*EHpresence_LorR	15.624	23	.871	
	Deleted Effect	1	Sex*Age_Cat_Coll	5.836	4	.212	2
		2	SES*Age_Cat_Coll	41.757	4	.000	2
		3	SES*EHpresence_LorR	.999	1	.317	2
9	Generating Class[b]		Sex*Age_Cat_Coll, SES*Age_Cat_Coll, EHpresence_LorR	16.623	24	.864	
	Deleted Effect	1	Sex*Age_Cat_Coll	5.836	4	.212	2
		2	SES*Age_Cat_Coll	41.757	4	.000	2
		3	EHpresence_LorR	16.239	1	.000	2
10	Generating Class[b]		SES*Age_Cat_Coll, EHpresence_LorR, Sex	22.459	28	.760	

a. At each step, the effect with the largest significance level for the Likelihood Ratio Change is deleted, provided the significance level is larger than .050.
b. Statistics are displayed for the best model at each step after step 0.
c. For 'Deleted Effect', this is the change in the Chi-Square after the effect is deleted from the model.

B.5 Periosteal New Bone Presence, Full Sample (All Four Sites), Adult Individuals

Step[a]			Effects	Chi-Square[c]	df	Sig.	Number of Iterations
0	Generating Class[b]		SES*Sex*PNB_presence*Age_Cat_Coll	.000	0	.	
	Deleted Effect	1	SES*Sex*PNB_presence*Age_Cat_Coll	3.507	4	.477	4
1	Generating Class[b]		SES*Sex*PNB_presence, SES*Sex*Age_Cat_Coll, SES*PNB_presence*Age_Cat_Coll, Sex*PNB_presence*Age_Cat_Coll	3.507	4	.477	
	Deleted Effect	1	SES*Sex*PNB_presence	.062	1	.804	3
		2	SES*Sex*Age_Cat_Coll	.575	4	.966	3
		3	SES*PNB_presence*Age_Cat_Coll	2.425	4	.658	3
		4	Sex*PNB_presence*Age_Cat_Coll	11.410	4	.022	3
2	Generating Class[b]		SES*Sex*PNB_presence, SES*PNB_presence*Age_Cat_Coll, Sex*PNB_presence*Age_Cat_Coll	4.082	8	.850	
	Deleted Effect	1	SES*Sex*PNB_presence	.059	1	.808	2
		2	SES*PNB_presence*Age_Cat_Coll	2.101	4	.717	3
		3	Sex*PNB_presence*Age_Cat_Coll	11.118	4	.025	3
3	Generating Class[b]		SES*PNB_presence*Age_Cat_Coll, Sex*PNB_presence*Age_Cat_Coll, SES*Sex	4.141	9	.902	
	Deleted Effect	1	SES*PNB_presence*Age_Cat_Coll	2.085	4	.720	3
		2	Sex*PNB_presence*Age_Cat_Coll	11.394	4	.022	3
		3	SES*Sex	.006	1	.940	2
4	Generating Class[b]		SES*PNB_presence*Age_Cat_Coll, Sex*PNB_presence*Age_Cat_Coll	4.147	10	.940	
	Deleted Effect	1	SES*PNB_presence*Age_Cat_Coll	2.117	4	.714	3
		2	Sex*PNB_presence*Age_Cat_Coll	11.424	4	.022	3
5	Generating Class[b]		Sex*PNB_presence*Age_Cat_Coll, SES*PNB_presence, SES*Age_Cat_Coll	6.264	14	.959	
	Deleted Effect	1	Sex*PNB_presence*Age_Cat_Coll	11.424	4	.022	3
		2	SES*PNB_presence	.155	1	.694	2
		3	SES*Age_Cat_Coll	36.006	4	.000	2
6	Generating Class[b]		Sex*PNB_presence*Age_Cat_Coll, SES*Age_Cat_Coll	6.419	15	.972	
	Deleted Effect	1	Sex*PNB_presence*Age_Cat_Coll	11.424	4	.022	3
		2	SES*Age_Cat_Coll	36.236	4	.000	2
7	Generating Class[b]		Sex*PNB_presence*Age_Cat_Coll, SES*Age_Cat_Coll	6.419	15	.972	

a. At each step, the effect with the largest significance level for the Likelihood Ratio Change is deleted, provided the significance level is larger than .050.
b. Statistics are displayed for the best model at each step after step 0.
c. For 'Deleted Effect', this is the change in the Chi-Square after the effect is deleted from the model.

B.6 Periosteal New Bone Presence, Reduced Sample (London Sites), Adult Individuals

Step[a]			Effects	Chi-Square[c]	df	Sig.	Number of Iterations
0	Generating Class[b]		SES*Sex*PNB_presence*Age_Cat_Coll	.000	0	.	
	Deleted Effect	1	SES*Sex*PNB_presence*Age_Cat_Coll	3.119	4	.538	5
1	Generating Class[b]		SES*Sex*PNB_presence, SES*Sex*Age_Cat_Coll, SES*PNB_presence*Age_Cat_Coll, Sex*PNB_presence*Age_Cat_Coll	3.119	4	.538	
	Deleted Effect	1	SES*Sex*PNB_presence	.778	1	.378	3
		2	SES*Sex*Age_Cat_Coll	1.353	4	.852	4
		3	SES*PNB_presence*Age_Cat_Coll	8.306	4	.081	4
		4	Sex*PNB_presence*Age_Cat_Coll	11.158	4	.025	3
2	Generating Class[b]		SES*Sex*PNB_presence, SES*PNB_presence*Age_Cat_Coll, Sex*PNB_presence*Age_Cat_Coll	4.472	8	.812	
	Deleted Effect	1	SES*Sex*PNB_presence	.584	1	.445	2
		2	SES*PNB_presence*Age_Cat_Coll	8.410	4	.078	4
		3	Sex*PNB_presence*Age_Cat_Coll	10.670	4	.031	3
3	Generating Class[b]		SES*PNB_presence*Age_Cat_Coll, Sex*PNB_presence*Age_Cat_Coll, SES*Sex	5.056	9	.829	
	Deleted Effect	1	SES*PNB_presence*Age_Cat_Coll	7.907	4	.095	4
		2	Sex*PNB_presence*Age_Cat_Coll	10.227	4	.037	4
		3	SES*Sex	.000	1	.990	2
4	Generating Class[b]		SES*PNB_presence*Age_Cat_Coll, Sex*PNB_presence*Age_Cat_Coll	5.056	10	.887	
	Deleted Effect	1	SES*PNB_presence*Age_Cat_Coll	7.929	4	.094	3
		2	Sex*PNB_presence*Age_Cat_Coll	10.283	4	.036	3
5	Generating Class[b]		Sex*PNB_presence*Age_Cat_Coll, SES*PNB_presence, SES*Age_Cat_Coll	12.985	14	.528	
	Deleted Effect	1	Sex*PNB_presence*Age_Cat_Coll	10.283	4	.036	3
		2	SES*PNB_presence	4.800	1	.028	2
		3	SES*Age_Cat_Coll	48.731	4	.000	2
6	Generating Class[b]		Sex*PNB_presence*Age_Cat_Coll, SES*PNB_presence, SES*Age_Cat_Coll	12.985	14	.528	

a. At each step, the effect with the largest significance level for the Likelihood Ratio Change is deleted, provided the significance level is larger than .050.
b. Statistics are displayed for the best model at each step after step 0.

c. For 'Deleted Effect', this is the change in the Chi-Square after the effect is deleted from the model.

APPENDIX C

ANALYSIS OF VARIANCE RESULTS

C.1 Tooth Size, 2- and 3-way ANOVA Results

R_Max_Pm_MD (2-way of Tooth*Age_Category*SES):

Levene's Test of Equality of Error Variances: F=1.082, df1=27, df2=135, Sig=0.370

Do not reject the null that dependent variable error variances equal across groups.

Tests of Between-Subjects Effects
Dependent Variable: R_Max_Pm_MD

Source	Type III Sum of Squares	df	Mean Square	F	Sig.
Corrected Model	6.566[a]	27	.243	1.118	.328
Intercept	2662.476	1	2662.476	12242.259	.000
SES	.583	1	.583	2.681	.104
Age_Category	3.117	15	.208	.956	.505
SES * Age_Category	2.556	11	.232	1.069	.391
Error	29.360	135	.217		
Total	6876.215	163			
Corrected Total	35.926	162			

a. R Squared = .183 (Adjusted R Squared = .019)

Notes: No significant effects or interactions.

R_Max_Pm_MD (3-way of Tooth*Age_Category*SES*Sex):

Levene's Test of Equality of Error Variances: F=1.483, df1=35, df2=116, Sig=0.062

Do not reject the null that dependent variable error variances equal across groups.

Tests of Between-Subjects Effects
Dependent Variable: R_Max_Pm_MD

Source	Type III Sum of Squares	df	Mean Square	F	Sig.
Corrected Model	8.484[a]	35	.242	1.145	.291
Intercept	2605.630	1	2605.630	12308.191	.000
SES	.489	1	.489	2.308	.131
Age_Category	1.697	11	.154	.729	.709
Sex	.181	1	.181	.854	.357
SES * Age_Category	1.131	9	.126	.593	.800
SES * Sex	.048	1	.048	.226	.636
Age_Category * Sex	2.317	8	.290	1.368	.218

SES * Age_Category * Sex	.840	3	.280	1.322	.271
Error	24.557	116	.212		
Total	6397.709	152			
Corrected Total	33.041	151			

a. R Squared = .257 (Adjusted R Squared = .033)

Notes: No significant effects or interactions.

R_Max_Pm_BL (2-way of Tooth*Age_Category*SES): removed due to unequal variances by age.

R_Max_Pm_BL (3-way of Tooth*Age_Category*SES*Sex): removed due to unequal variances by age.

L_Max_Pm_MD (2-way of Tooth*Age_Category*SES):

Levene's Test of Equality of Error Variances: F=1.007, df1=30, df2=154, Sig=0.464

Do not reject the null that dependent variable error variances equal across groups.

Tests of Between-Subjects Effects
Dependent Variable: L_Max_Pm_MD

Source	Type III Sum of Squares	df	Mean Square	F	Sig.
Corrected Model	6.745[a]	30	.225	1.272	.175
Intercept	2637.695	1	2637.695	14924.352	.000
SES	1.183	1	1.183	6.693	.011
Age_Category	3.570	17	.210	1.188	.280
SES * Age_Category	3.821	12	.318	1.802	.052
Error	27.218	154	.177		
Total	7763.226	185			
Corrected Total	33.963	184			

a. R Squared = .199 (Adjusted R Squared = .042)

Notes: The effect of SES is highly significant (Sig. = 0.011) and the interaction between SES*Age is moderately significant (Sig. = 0.052).
Direction of SES: smaller teeth in middle/low SES (slight CI overlap)
Direction of SES*Age: smaller teeth in Age_Cats 14, 19, 20, 22, 23, 26, 29, 30 in middle/low SES (all CIs overlap)
Note: No high SES estimates for Age_Cats 12, 17, 18, 24, or 31…
…so high SES teeth only smaller in Age_Cats 16, 21, 25, 27, 28

L_Max_Pm_MD (3-way of Tooth*Age_Category*SES*Sex):

Levene's Test of Equality of Error Variances: F=1.936, df1=39, df2=129, Sig=0.582

Do not reject the null that dependent variable error variances equal across groups.

Tests of Between-Subjects Effects
Dependent Variable: L_Max_Pm_MD

Source	Type III Sum of Squares	df	Mean Square	F	Sig.
Corrected Model	8.581[a]	39	.220	1.422	.074
Intercept	2698.969	1	2698.969	17444.603	.000
SES	.338	1	.338	2.185	.142
Age_Category	2.714	12	.226	1.462	.147
Sex	.021	1	.021	.137	.712
SES * Age_Category	2.394	10	.239	1.548	.130
SES * Sex	.637	1	.637	4.118	.044
Age_Category * Sex	2.201	10	.220	1.423	.177
SES * Age_Category * Sex	.329	4	.082	.532	.712
Error	19.958	129	.155		
Total	7084.539	169			
Corrected Total	28.539	168			
a. R Squared = .301 (Adjusted R Squared = .089)					

Notes: The interaction between SES*Sex is highly significant (Sig. = 0.044). Direction of SES*Sex: smallest teeth in low SES males (no CI overlap with largest teeth in high SES males, but CIs overlap with all other pairs)

L_Max_Pm_BL (2-way of Tooth*Age_Category*SES):

Levene's Test of Equality of Error Variances: F=1.141, df1=30, df2=160, Sig=0.295

Do not reject the null that dependent variable error variances equal across groups.

Tests of Between-Subjects Effects
Dependent Variable: L_Max_Pm_BL

Source	Type III Sum of Squares	df	Mean Square	F	Sig.
Corrected Model	15.476[a]	30	.516	1.366	.113
Intercept	4866.179	1	4866.179	12887.083	.000
SES	.154	1	.154	.409	.524
Age_Category	7.405	17	.436	1.154	.309
SES * Age_Category	9.627	12	.802	2.125	.018
Error	60.416	160	.378		
Total	14925.951	191			
Corrected Total	75.892	190			
a. R Squared = .204 (Adjusted R Squared = .055)					

Notes: The interaction between SES*Age is highly significant (Sig. = 0.018). Direction of SES*Age: smaller teeth in Age_Cats 14, 16, 19, 20, 22, 26, 27, 29, 30 in middle/low SES (all CIs overlap)
Note: No high SES estimates for Age_Cats 12, 17, 18, 24, or 31…
…so high SES teeth only smaller in Age_Cats 21, 23, 25, 28

L_Max_Pm_BL (3-way of Tooth*Age_Category*SES*Sex):

Levene's Test of Equality of Error Variances: F=1.261, df1=40, df2=134, Sig=0.166

Do not reject the null that dependent variable error variances equal across groups.

Tests of Between-Subjects Effects
Dependent Variable: L_Max_Pm_BL

Source	Type III Sum of Squares	df	Mean Square	F	Sig.
Corrected Model	19.146[a]	40	.479	1.330	.117
Intercept	5047.785	1	5047.785	14029.306	.000
SES	.409	1	.409	1.137	.288
Age_Category	6.769	12	.564	1.568	.108
Sex	1.506	1	1.506	4.186	.043
SES * Age_Category	7.485	10	.748	2.080	.030
SES * Sex	1.551	1	1.551	4.312	.040
Age_Category * Sex	2.909	10	.291	.809	.621
SES * Age_Category * Sex	.663	5	.133	.368	.869
Error	48.214	134	.360		
Total	13683.500	175			
Corrected Total	67.360	174			

a. R Squared = .284 (Adjusted R Squared = .071)

Notes: The effect of Sex is highly significant (Sig. = 0.043). The interactions between SES*Age (Sig. = 0.030) and SES*Sex (Sig. = 0.040) are highly significant.
Direction of Sex: smaller teeth in females (slight CI overlap)
Direction of SES*Age: smaller teeth in Age_Cats 19, 20, 22, 26, 29, 30 in middle/low SES (all CIs overlap)
Note: No high SES estimates for Age_Cats 24 or 31…
…so high SES teeth only smaller in Age_Cats 21, 23, 25, 27, 28
Direction of SES*Sex: smaller teeth in high SES females (substantial CI overlap with middle/low SES females or middle/low SES males, very little CI overlap with high SES males [who have the largest teeth])

R_Max_M_MD (2-way of Tooth*Age_Category*SES):
Levene's Test of Equality of Error Variances: F=1.796, df1=30, df2=142, Sig=0.012
Reject the null that dependent variable error variances equal across groups.

R_Max_M_MD (3-way of Tooth*Age_Category*SES*Sex):
Levene's Test of Equality of Error Variances: F=1.642, df1=35, df2=117, Sig=0.026
Reject the null that dependent variable error variances equal across groups.

R_Max_M_BL (2-way of Tooth*Age_Category*SES):
Levene's Test of Equality of Error Variances: F=1.276, df1=30, df2=141, Sig=0.174
Do not reject the null that dependent variable error variances equal across groups.

Tests of Between-Subjects Effects
Dependent Variable: R_Max_M_BL

Source	Type III Sum of Squares	df	Mean Square	F	Sig.

Corrected Model	16.795[a]	30	.560	1.096	.350
Intercept	7203.229	1	7203.229	14104.841	.000
SES	1.573	1	1.573	3.081	.081
Age_Category	11.010	18	.612	1.198	.271
SES * Age_Category	6.372	11	.579	1.134	.339
Error	72.008	141	.511		
Total	20309.523	172			
Corrected Total	88.802	171			
a. R Squared = .189 (Adjusted R Squared = .017)					

Notes: The effect of SES is moderately significant (Sig. = 0.081).
Direction of SES: smaller teeth in middle/low SES (CI overlap)

R_Max_M_BL (3-way of Tooth*Age_Category*SES*Sex):
Levene's Test of Equality of Error Variances: F=1.516, df1=35, df2=116, Sig=0.052
Do not reject the null that dependent variable error variances equal across groups.

Tests of Between-Subjects Effects **Dependent Variable: R_Max_M_BL**					
Source	**Type III Sum of Squares**	**df**	**Mean Square**	**F**	**Sig.**
Corrected Model	20.540[a]	35	.587	1.201	.233
Intercept	6400.295	1	6400.295	13099.739	.000
SES	.001	1	.001	.001	.972
Age_Category	3.808	11	.346	.709	.728
Sex	1.671	1	1.671	3.419	.067
SES * Age_Category	3.470	8	.434	.888	.529
SES * Sex	.632	1	.632	1.294	.258
Age_Category * Sex	5.024	8	.628	1.285	.258
SES * Age_Category * Sex	1.691	4	.423	.865	.487
Error	56.675	116	.489		
Total	17869.624	152			
Corrected Total	77.216	151			
a. R Squared = .266 (Adjusted R Squared = .045)					

Notes: The effect of Sex is moderately significant (Sig. = 0.067).
Direction of Sex: smaller teeth in females (CI overlap)

L_Max_M_MD (2-way of Tooth*Age_Category*SES):
Levene's Test of Equality of Error Variances: F=2.327, df1=30, df2=146, Sig=0.000
Reject the null that dependent variable error variances equal across groups.

L_Max_M_MD (3-way of Tooth*Age_Category*SES*Sex):
Levene's Test of Equality of Error Variances: F=2.037, df1=33, df2=118, Sig=0.003
Reject the null that dependent variable error variances equal across groups.

L_Max_M_BL (2-way of Tooth*Age_Category*SES):

Levene's Test of Equality of Error Variances: F=1.406, df1=30, df2=146, Sig=0.096

Do not reject the null that dependent variable error variances equal across groups.

Tests of Between-Subjects Effects
Dependent Variable: L_Max_M_BL

Source	Type III Sum of Squares	df	Mean Square	F	Sig.
Corrected Model	15.964[a]	30	.532	.919	.591
Intercept	6759.253	1	6759.253	11675.531	.000
SES	.586	1	.586	1.012	.316
Age_Category	9.749	18	.542	.936	.537
SES * Age_Category	4.974	11	.452	.781	.658
Error	84.523	146	.579		
Total	20810.402	177			
Corrected Total	100.487	176			

a. R Squared = .159 (Adjusted R Squared = -.014)

Notes: No significant effects or interactions.

L_Max_M_BL (3-way of Tooth*Age_Category*SES*Sex):

Levene's Test of Equality of Error Variances: F=1.252, df1=35, df2=115, Sig=0.189

Do not reject the null that dependent variable error variances equal across groups.

Tests of Between-Subjects Effects
Dependent Variable: L_Max_M_BL

Source	Type III Sum of Squares	df	Mean Square	F	Sig.
Corrected Model	18.833[a]	35	.538	.921	.599
Intercept	5456.930	1	5456.930	9338.393	.000
SES	.010	1	.010	.018	.894
Age_Category	1.816	11	.165	.283	.988
Sex	1.057	1	1.057	1.810	.181
SES * Age_Category	3.549	8	.444	.759	.639
SES * Sex	.004	1	.004	.006	.938
Age_Category * Sex	6.492	8	.811	1.389	.209
SES * Age_Category * Sex	.383	4	.096	.164	.956
Error	67.201	115	.584		
Total	17708.395	151			
Corrected Total	86.033	150			

a. R Squared = .219 (Adjusted R Squared = -.019)

Notes: No significant effects or interactions.

R_Mand_Pm_MD (2-way of Tooth*Age_Category*SES):

Levene's Test of Equality of Error Variances: F=1.058, df1=33, df2=209, Sig=0.390

Do not reject the null that dependent variable error variances equal across groups.

Tests of Between-Subjects Effects Dependent Variable: R_Mand_Pm_MD					
Source	**Type III Sum of Squares**	**df**	**Mean Square**	**F**	**Sig.**
Corrected Model	8.510[a]	33	.258	1.346	.111
Intercept	3834.199	1	3834.199	20012.706	.000
SES	.322	1	.322	1.681	.196
Age_Category	3.479	19	.183	.956	.515
SES * Age_Category	2.541	13	.195	1.020	.433
Error	40.042	209	.192		
Total	11626.202	243			
Corrected Total	48.552	242			
a. R Squared = .175 (Adjusted R Squared = .045)					

Notes: No significant effects or interactions.

R_Mand_Pm_MD (3-way of Tooth*Age_Category*SES*Sex):

Levene's Test of Equality of Error Variances: F=1.204, df1=44, df2=176, Sig=0.201

Do not reject the null that dependent variable error variances equal across groups.

Tests of Between-Subjects Effects Dependent Variable: R_Mand_Pm_MD					
Source	**Type III Sum of Squares**	**df**	**Mean Square**	**F**	**Sig.**
Corrected Model	10.325[a]	44	.235	1.259	.151
Intercept	4049.947	1	4049.947	21727.527	.000
SES	.675	1	.675	3.621	.059
Age_Category	2.277	12	.190	1.018	.434
Sex	.037	1	.037	.199	.656
SES * Age_Category	2.294	11	.209	1.119	.349
SES * Sex	.006	1	.006	.031	.861
Age_Category * Sex	2.218	11	.202	1.082	.379
SES * Age_Category * Sex	.234	7	.033	.179	.989
Error	32.806	176	.186		
Total	10578.193	221			
Corrected Total	43.130	220			
a. R Squared = .239 (Adjusted R Squared = .049)					

Notes: The effect of SES is moderately significant (Sig. = 0.059).

Direction of SES: smaller teeth in middle/low SES (CI overlap)

R_Mand_Pm_BL (2-way of Tooth*Age_Category*SES):

Levene's Test of Equality of Error Variances: F=1.192, df1=33, df2=210, Sig=0.230

Do not reject the null that dependent variable error variances equal across groups.

Tests of Between-Subjects Effects Dependent Variable: R_Mand_Pm_BL

Source	Type III Sum of Squares	df	Mean Square	F	Sig.
Corrected Model	10.595[a]	33	.321	1.238	.187
Intercept	5222.835	1	5222.835	20145.252	.000
SES	.222	1	.222	.858	.355
Age_Category	2.738	19	.144	.556	.933
SES * Age_Category	4.580	13	.352	1.359	.182
Error	54.444	210	.259		
Total	15608.005	244			
Corrected Total	65.039	243			

a. R Squared = .163 (Adjusted R Squared = .031)

Notes: No significant effects or interactions.

R_Mand_Pm_BL (3-way of Tooth*Age_Category*SES*Sex):

Levene's Test of Equality of Error Variances: F=1.315, df1=44, df2=177, Sig=0.110

Do not reject the null that dependent variable error variances equal across groups.

Tests of Between-Subjects Effects
Dependent Variable: R_Mand_Pm_BL

Source	Type III Sum of Squares	df	Mean Square	F	Sig.
Corrected Model	15.458[a]	44	.351	1.458	.046
Intercept	6009.823	1	6009.823	24942.789	.000
SES	.579	1	.579	2.404	.123
Age_Category	2.920	12	.243	1.010	.442
Sex	.828	1	.828	3.436	.065
SES * Age_Category	3.198	11	.291	1.206	.286
SES * Sex	.026	1	.026	.106	.745
Age_Category * Sex	2.581	10	.258	1.071	.387
SES * Age_Category * Sex	1.838	8	.230	.953	.474
Error	42.647	177	.241		
Total	14232.685	222			
Corrected Total	58.105	221			

a. R Squared = .266 (Adjusted R Squared = .084)

Notes: The effect of Sex is moderately significant (Sig. = 0.065).

Direction of Sex: smaller teeth in females (CIs overlap slightly)

L_Mand_Pm_MD (2-way of Tooth*Age_Category*SES):

Levene's Test of Equality of Error Variances: F=1.304, df1=31, df2=201, Sig=0.143

Do not reject the null that dependent variable error variances equal across groups.

Tests of Between-Subjects Effects
Dependent Variable: L_Mand_Pm_MD

Source	Type III Sum of Squares	df	Mean Square	F	Sig.

Corrected Model	10.857[a]	31	.350	1.599	.030
Intercept	3512.682	1	3512.682	16033.328	.000
SES	1.202	1	1.202	5.486	.020
Age_Category	5.565	18	.309	1.411	.129
SES * Age_Category	3.350	12	.279	1.274	.236
Error	44.036	201	.219		
Total	11142.642	233			
Corrected Total	54.893	232			
a. R Squared = .198 (Adjusted R Squared = .074)					

Notes: The effect of SES is highly significant (Sig. = 0.020).
Direction of SES: smaller teeth in middle/low SES (slight CI overlap)

L_Mand_Pm_MD (3-way of Tooth*Age_Category*SES*Sex):
Levene's Test of Equality of Error Variances: F=1.524, df1=43, df2=169, Sig=0.032
Reject the null that dependent variable error variances equal across groups.

L_Mand_Pm_BL (2-way of Tooth*Age_Category*SES):
Levene's Test of Equality of Error Variances: F=1.423, df1=31, df2=195, Sig=0.080
Do not reject the null that dependent variable error variances equal across groups.

Tests of Between-Subjects Effects
Dependent Variable: L_Mand_Pm_BL

Source	Type III Sum of Squares	df	Mean Square	F	Sig.
Corrected Model	8.840[a]	31	.285	1.039	.419
Intercept	4867.777	1	4867.777	17731.958	.000
SES	.046	1	.046	.166	.684
Age_Category	3.147	18	.175	.637	.868
SES * Age_Category	4.979	12	.415	1.511	.123
Error	53.531	195	.275		
Total	14409.498	227			
Corrected Total	62.371	226			
a. R Squared = .142 (Adjusted R Squared = .005)					

Notes: No significant effects or interactions.

L_Mand_Pm_BL (3-way of Tooth*Age_Category*SES*Sex):
Levene's Test of Equality of Error Variances: F=1.293, df1=43, df2=164, Sig=0.129
Do not reject the null that dependent variable error variances equal across groups.

Tests of Between-Subjects Effects
Dependent Variable: L_Mand_Pm_BL

Source	Type III Sum of Squares	df	Mean Square	F	Sig.
Corrected Model	14.181[a]	43	.330	1.268	.147
Intercept	5405.699	1	5405.699	20791.503	.000

SES	.163	1	.163	.627	.430
Age_Category	3.156	12	.263	1.012	.441
Sex	2.303	1	2.303	8.857	.003
SES * Age_Category	4.627	10	.463	1.780	.068
SES * Sex	.061	1	.061	.234	.629
Age_Category * Sex	2.612	10	.261	1.004	.442
SES * Age_Category * Sex	1.095	7	.156	.602	.754
Error	42.639	164	.260		
Total	13229.348	208			
Corrected Total	56.821	207			

a. R Squared = .250 (Adjusted R Squared = .053)

Notes: The effect of Sex is highly significant (Sig. = 0.003). The interaction between SES*Age is moderately significant (Sig. = 0.068).

Direction of Sex: smaller teeth among females (slight CI overlap)

Direction of SES*Age: smaller teeth among Age_Cats 19-22, 24, 27, and 28 in middle/low SES (all CIs overlap)

Note: No high SES estimate for Age_Cat 31…

…so high SES teeth only smaller in Age_Cats 23, 25, 26, 29, and 30

R_Mand_M_MD (2-way of Tooth*Age_Category*SES):

Levene's Test of Equality of Error Variances: F=1.344, df1=31, df2=169, Sig=0.122

Do not reject the null that dependent variable error variances equal across groups.

Tests of Between-Subjects Effects
Dependent Variable: R_Mand_M_MD

Source	Type III Sum of Squares	df	Mean Square	F	Sig.
Corrected Model	15.886[a]	31	.512	1.402	.092
Intercept	9651.471	1	9651.471	26400.659	.000
SES	1.709	1	1.709	4.676	.032
Age_Category	6.566	18	.365	.998	.465
SES * Age_Category	3.235	12	.270	.737	.713
Error	61.782	169	.366		
Total	23004.638	201			
Corrected Total	77.669	200			

a. R Squared = .205 (Adjusted R Squared = .059)

Notes: The effect of SES is highly significant (Sig. = 0.032).

Direction of SES: smaller teeth among middle/low SES (slight CI overlap)

R_Mand_M_MD (3-way of Tooth*Age_Category*SES*Sex):

Levene's Test of Equality of Error Variances: F=1.641, df1=40, df2=133, Sig=0.020

Reject the null that dependent variable error variances equal across groups.

R_Mand_M_BL (2-way of Tooth*Age_Category*SES):

Levene's Test of Equality of Error Variances: F=1.957, df1=31, df2=166, Sig=0.004
Reject the null that dependent variable error variances equal across groups.

R_Mand_M_BL (3-way of Tooth*Age_Category*SES*Sex):
Levene's Test of Equality of Error Variances: F=2.154, df1=40, df2=131, Sig=0.001
Reject the null that dependent variable error variances equal across groups.

L_Mand_M_MD (2-way of Tooth*Age_Category*SES): removed due to unequal variances by age.

L_Mand_M_MD (3-way of Tooth*Age_Category*SES*Sex): removed due to unequal variances by age.

L_Mand_M_BL (2-way of Tooth*Age_Category*SES):
Levene's Test of Equality of Error Variances: F=1.535, df1=31, df2=160, Sig=0.047
Reject the null that dependent variable error variances equal across groups.

L_Mand_M_BL (3-way of Tooth*Age_Category*SES*Sex):
Levene's Test of Equality of Error Variances: F=2.167, df1=36, df2=132, Sig=0.001
Reject the null that dependent variable error variances equal across groups.

C.2 Vertebral Neural Canal Size, 2- and 3-way ANOVA Results

TR_L1_inverse (2-way of VNC_inverse*Age_Category*SES):

Levene's Test of Equality of Error Variances: F=0.956, df1=28, df2=313, Sig=0.533

Do not reject the null that dependent variable error variances equal across groups.

Tests of Between-Subjects Effects
Dependent Variable: TR_L1_inverse

Source	Type III Sum of Squares	df	Mean Square	F	Sig.
Corrected Model	.001[a]	28	2.790E-5	2.318	.000
Intercept	.206	1	.206	17087.728	.000
SES	4.712E-5	1	4.712E-5	3.914	.049
Age_Category	.001	15	3.890E-5	3.232	.000
SES * Age_Category	.000	12	9.067E-6	.753	.698
Error	.004	313	1.204E-5		
Total	.705	342			
Corrected Total	.005	341			

a. R Squared = .172 (Adjusted R Squared = .098)

Notes: The effect of Age is highly significant (Sig. = 0.000), and the effect of SES is highly significant (Sig. = 0.049).

Direction of Age: TR_L1_inverse size highest with Age_Cat 25, but decreases obviously after Age_Cat 25 (all CIs overlap)

Direction of SES: TR_L1_inverse highest with high SES (CIs overlap)

TR_L1_inverse (3-way of VNC_inverse*Age_Category*SES*Sex):

Levene's Test of Equality of Error Variances: F=0.997, df1=46, df2=286, Sig=0.483

Do not reject the null that dependent variable error variances equal across groups.

Tests of Between-Subjects Effects
Dependent Variable: TR_L1_inverse

Source	Type III Sum of Squares	df	Mean Square	F	Sig.
Corrected Model	.001[a]	46	2.854E-5	2.562	.000
Intercept	.288	1	.288	25814.922	.000
SES	.000	1	.000	9.344	.002
Age_Category	.000	12	2.809E-5	2.522	.004
Sex	.000	1	.000	20.388	.000
SES * Age_Category	8.582E-5	11	7.801E-6	.700	.738
SES * Sex	3.681E-6	1	3.681E-6	.330	.566
Age_Category * Sex	.000	11	1.066E-5	.957	.486
SES * Age_Category * Sex	7.842E-5	9	8.713E-6	.782	.633
Error	.003	286	1.114E-5		
Total	.688	333			
Corrected Total	.004	332			

a. R Squared = .292 (Adjusted R Squared = .178)

Notes: The effects of SES (Sig. = 0.002), Age (Sig. = 0.004), and Sex (Sig. = 0.000) are all highly significant.

Direction of SES: TR_L1_inverse higher for high SES (CIs overlap)

Direction of Age: TR_L1_inverse higher for Age_Cats 19-22, 25-26 (most CIs overlap)

Direction of Sex: TR_L1_inverse higher for females (no CI overlap)

AP_L1_inverse (2-way of VNC_inverse*Age_Category*SES):

Levene's Test of Equality of Error Variances: F=1.531, df1=32, df2=308, Sig=0.037

Reject the null that dependent variable error variances equal across groups.

AP_L1_inverse (3-way of VNC_inverse*Age_Category*SES*Sex):

Levene's Test of Equality of Error Variances: F=1.465, df1=45, df2=274, Sig=0.035

Reject the null that dependent variable error variances equal across groups.

TR_L2_inverse (2-way of VNC_inverse*Age_Category*SES):

Levene's Test of Equality of Error Variances: F=0.668, df1=28, df2=324, Sig=0.901

Do not reject the null that dependent variable error variances equal across groups.

Tests of Between-Subjects Effects
Dependent Variable: TR_L2_inverse

Source	Type III Sum of Squares	df	Mean Square	F	Sig.
Corrected Model	.001[a]	28	1.927E-5	1.645	.023
Intercept	.229	1	.229	19535.735	.000
SES	2.784E-5	1	2.784E-5	2.376	.124
Age_Category	.000	15	2.794E-5	2.385	.003
SES * Age_Category	6.143E-5	12	5.119E-6	.437	.948
Error	.004	324	1.172E-5		
Total	.724	353			
Corrected Total	.004	352			

a. R Squared = .124 (Adjusted R Squared = .049)

Notes: The effect of Age is highly significant (Sig. = 0.003).

Direction for Age: TR_L2_inverse higher for Age_Cat 25, but decreases obviously after that) (all CIs overlap)

TR_L2_inverse (3-way of VNC_inverse*Age_Category*SES*Sex):

Levene's Test of Equality of Error Variances: F=0.818, df1=46, df2=297, Sig=0.793

Do not reject the null that dependent variable error variances equal across groups.

Tests of Between-Subjects Effects
Dependent Variable: TR_L2_inverse

Source	Type III Sum of Squares	df	Mean Square	F	Sig.
Corrected Model	.001[a]	46	2.220E-5	2.028	.000

Intercept	.318	1	.318	29082.464	.000
SES	4.435E-5	1	4.435E-5	4.053	.045
Age_Category	.000	12	2.120E-5	1.937	.030
Sex	.000	1	.000	18.627	.000
SES * Age_Category	5.909E-5	11	5.372E-6	.491	.908
SES * Sex	1.407E-5	1	1.407E-5	1.286	.258
Age_Category * Sex	.000	12	1.019E-5	.931	.516
SES * Age_Category * Sex	5.771E-5	8	7.214E-6	.659	.727
Error	.003	297	1.094E-5		
Total	.706	344			
Corrected Total	.004	343			
a. R Squared = .239 (Adjusted R Squared = .121)					

Notes: The effects of SES (Sig. = 0.045), Age (Sig. = 0.030), and Sex (Sig. = 0.000) are all highly significant.

Direction of SES: TR_L2_inverse higher for high SES (CIs overlap)

Direction of Age: TR_L2_inverse higher for Age_Cats 19-22, 25-26, but decreases obviously after Age_Cat 26 (all CIs overlap)

Direction of Sex: TR_L2_inverse higher for females (no CI overlap)

AP_L2_inverse (2-way of VNC_inverse*Age_Category*SES):

Levene's Test of Equality of Error Variances: F=1.227, df1=32, df2=313, Sig=0.192

Do not reject the null that dependent variable error variances equal across groups.

Tests of Between-Subjects Effects
Dependent Variable: AP_L2_inverse

Source	Type III Sum of Squares	df	Mean Square	F	Sig.
Corrected Model	.001[a]	32	1.682E-5	.602	.958
Intercept	.347	1	.347	12424.654	.000
SES	3.573E-7	1	3.573E-7	.013	.910
Age_Category	.000	18	2.023E-5	.724	.786
SES * Age_Category	.000	13	1.496E-5	.535	.902
Error	.009	313	2.794E-5		
Total	1.274	346			
Corrected Total	.009	345			
a. R Squared = .058 (Adjusted R Squared = -.038)					

Notes: There are no significant effects or interactions.

AP_L2_inverse (3-way of VNC_inverse*Age_Category*SES*Sex): removed because of unequal variances by sex

TR_L3_inverse (2-way of VNC_inverse*Age_Category*SES):

Levene's Test of Equality of Error Variances: F=1.087, df1=28, df2=320, Sig=0.352

Do not reject the null that dependent variable error variances equal across groups.

Tests of Between-Subjects Effects Dependent Variable: TR_L3_inverse					
Source	**Type III Sum of Squares**	**df**	**Mean Square**	**F**	**Sig.**
Corrected Model	.001[a]	28	2.422E-5	2.103	.001
Intercept	.219	1	.219	19002.890	.000
SES	3.525E-5	1	3.525E-5	3.060	.081
Age_Category	.001	15	3.486E-5	3.026	.000
SES * Age_Category	6.399E-5	12	5.332E-6	.463	.935
Error	.004	320	1.152E-5		
Total	.722	349			
Corrected Total	.004	348			
a. R Squared = .155 (Adjusted R Squared = .081)					

Notes: The effect of Age is highly significant (Sig. = 0.000), and the effect of SES is moderately significant (Sig. = 0.081)
Direction for Age: TR_L3_inverse higher for Age_Cats 20, 22, and 25, but decreases obviously after Age_Cat 25 (all CIs overlap)
Direction for SES: TR_L3_inverse higher for high SES

TR_L3_inverse (3-way of VNC_inverse*Age_Category*SES*Sex):
Levene's Test of Equality of Error Variances: F=1.009, df1=45, df2=295, Sig=0.462
Do not reject the null that dependent variable error variances equal across groups.

Tests of Between-Subjects Effects Dependent Variable: TR_L3_inverse					
Source	**Type III Sum of Squares**	**df**	**Mean Square**	**F**	**Sig.**
Corrected Model	.001[a]	45	2.318E-5	2.115	.000
Intercept	.320	1	.320	29244.502	.000
SES	7.558E-5	1	7.558E-5	6.898	.009
Age_Category	.000	12	2.783E-5	2.540	.003
Sex	.000	1	.000	10.033	.002
SES * Age_Category	7.793E-5	11	7.085E-6	.647	.788
SES * Sex	1.102E-5	1	1.102E-5	1.006	.317
Age_Category * Sex	9.769E-5	11	8.881E-6	.811	.630
SES * Age_Category * Sex	9.243E-5	8	1.155E-5	1.054	.395
Error	.003	295	1.096E-5		
Total	.706	341			
Corrected Total	.004	340			
a. R Squared = .244 (Adjusted R Squared = .129)					

Notes: The effects of SES (Sig. = 0.009), Age (Sig. = 0.003), and Sex (Sig. = 0.002) are all highly significant.
Direction of SES: TR_L3_inverse higher for high SES (no CI overlap)

Direction of Age: TR_L3_inverse higher for Age_Cats 19-22, 25-27, but decreases obviously after Age_Cat 27 (all CIs overlap)

Direction of Sex: TR_L3_inverse higher for females (no CI overlap)

AP_L3_inverse (2-way of VNC_inverse*Age_Category*SES):

Levene's Test of Equality of Error Variances: F=0.706, df1=31, df2=310, Sig=0.879

Do not reject the null that dependent variable error variances equal across groups.

Tests of Between-Subjects Effects
Dependent Variable: AP_L3_inverse

Source	Type III Sum of Squares	df	Mean Square	F	Sig.
Corrected Model	.001[a]	31	3.602E-5	.974	.510
Intercept	.473	1	.473	12793.775	.000
SES	4.502E-5	1	4.502E-5	1.217	.271
Age_Category	.001	17	3.917E-5	1.059	.394
SES * Age_Category	.001	13	4.347E-5	1.175	.296
Error	.011	310	3.699E-5		
Total	1.398	342			
Corrected Total	.013	341			

a. R Squared = .089 (Adjusted R Squared = -.002)

Notes: There are no significant effects or interactions.

AP_L3_inverse (3-way of VNC_inverse*Age_Category*SES*Sex): removed because of unequal variances by Sex

TR_L4_inverse (2-way of VNC_inverse*Age_Category*SES):

Levene's Test of Equality of Error Variances: F=1.012, df1=28, df2=308, Sig=0.452

Do not reject the null that dependent variable error variances equal across groups.

Tests of Between-Subjects Effects
Dependent Variable: TR_L4_inverse

Source	Type III Sum of Squares	df	Mean Square	F	Sig.
Corrected Model	.001[a]	28	2.868E-5	2.187	.001
Intercept	.208	1	.208	15869.798	.000
SES	1.760E-5	1	1.760E-5	1.342	.248
Age_Category	.000	15	2.886E-5	2.201	.006
SES * Age_Category	.000	12	1.313E-5	1.001	.447
Error	.004	308	1.311E-5		
Total	.689	337			
Corrected Total	.005	336			

a. R Squared = .166 (Adjusted R Squared = .090)

Notes: The effect of Age is highly significant (Sig. = 0.006).

Direction for Age: TR_L4_inverse higher for Age_Cat 25, but decreases obviously after that (all CIs overlap)

TR_L4_inverse (3-way of VNC_inverse*Age_Category*SES*Sex):

Levene's Test of Equality of Error Variances: F=1.199, df1=45, df2=282, Sig=0.192

Do not reject the null that dependent variable error variances equal across groups.

Tests of Between-Subjects Effects
Dependent Variable: TR_L4_inverse

Source	Type III Sum of Squares	df	Mean Square	F	Sig.
Corrected Model	.001[a]	45	2.501E-5	1.961	.001
Intercept	.274	1	.274	21475.506	.000
SES	8.053E-5	1	8.053E-5	6.314	.013
Age_Category	.000	12	2.501E-5	1.961	.028
Sex	.000	1	.000	12.006	.001
SES * Age_Category	.000	11	1.109E-5	.869	.571
SES * Sex	3.506E-6	1	3.506E-6	.275	.601
Age_Category * Sex	.000	11	9.405E-6	.737	.702
SES * Age_Category * Sex	6.376E-5	8	7.970E-6	.625	.757
Error	.004	282	1.275E-5		
Total	.671	328			
Corrected Total	.005	327			

a. R Squared = .238 (Adjusted R Squared = .117)

Notes: The effects of SES (Sig. = 0.013), Age (Sig. = 0.028), and Sex (Sig. = 0.001) are all highly significant.

Direction of SES: TR_L4_inverse higher for high SES (CI overlap)

Direction of Age: TR_L4_inverse higher for Age_Cats 19-22, 25-26, but decreases obviously after Age_Cat 26 (all CIs overlap)

Direction of Sex: TR_L4_inverse higher for females (CI overlap)

AP_L4_inverse (2-way of VNC_inverse*Age_Category*SES):

Levene's Test of Equality of Error Variances: F=1.015, df1=32, df2=290, Sig=0.450

Do not reject the null that dependent variable error variances equal across groups.

Tests of Between-Subjects Effects
Dependent Variable: AP_L4_inverse

Source	Type III Sum of Squares	df	Mean Square	F	Sig.
Corrected Model	.002[a]	32	7.468E-5	1.696	.013
Intercept	.427	1	.427	9706.921	.000
SES	.000	1	.000	3.324	.069
Age_Category	.002	18	9.977E-5	2.266	.003
SES * Age_Category	.001	13	7.426E-5	1.687	.063
Error	.013	290	4.403E-5		

Total	1.306	323			
Corrected Total	.015	322			
a. R Squared = .158 (Adjusted R Squared = .065)					

Notes: The effect of Age is highly significant (Sig. = 0.003), and the effect of SES is moderately significant (Sig. = 0.069). The interaction between SES and Age is moderately significant (Sig. = 0.063).

Direction of SES: AP_L4_inverse higher for middle/low SES (CI overlap)
Direction of Age: AP_L4_inverse higher for Age_Cats 11-14, 18, 25-27, 30 (all CIs overlap)
Direction of Age*SES: AP_L4_inverse high for Age_Cats 11-16, 18, 25-27, and 30 in middle/low SES; typically higher than high SES (except Age_Cats 14 and 20); (all CIs overlap)

AP_L4_inverse (3-way of VNC_inverse*Age_Category*SES*Sex):

Levene's Test of Equality of Error Variances: F=0.914, df1=44, df2=256, Sig=0.630

Do not reject the null that dependent variable error variances equal across groups.

Tests of Between-Subjects Effects
Dependent Variable: AP_L4_inverse

Source	Type III Sum of Squares	df	Mean Square	F	Sig.
Corrected Model	.002[a]	44	4.547E-5	.985	.504
Intercept	.600	1	.600	13003.305	.000
SES	.000	1	.000	2.499	.115
Age_Category	.001	11	7.191E-5	1.558	.112
Sex	5.863E-5	1	5.863E-5	1.270	.261
SES * Age_Category	.001	11	5.591E-5	1.211	.279
SES * Sex	3.798E-5	1	3.798E-5	.823	.365
Age_Category * Sex	.000	11	2.567E-5	.556	.863
SES * Age_Category * Sex	9.167E-5	8	1.146E-5	.248	.981
Error	.012	256	4.616E-5		
Total	1.210	301			
Corrected Total	.014	300			
a. R Squared = .145 (Adjusted R Squared = -.002)					

Notes: There were no significant effects or interactions.

TR_L5_inverse (2-way of VNC_inverse*Age_Category*SES):

Levene's Test of Equality of Error Variances: F=1.429, df1=28, df2=268, Sig=0.080

Do not reject the null that dependent variable error variances equal across groups.

Tests of Between-Subjects Effects
Dependent Variable: TR_L5_inverse

Source	Type III Sum of Squares	df	Mean Square	F	Sig.
Corrected Model	.001[a]	28	2.927E-5	1.736	.014
Intercept	.152	1	.152	8989.335	.000

SES	9.113E-6	1	9.113E-6	.540	.463
Age_Category	.000	15	2.832E-5	1.680	.055
SES * Age_Category	.000	12	2.260E-5	1.340	.196
Error	.005	268	1.686E-5		
Total	.502	297			
Corrected Total	.005	296			
a. R Squared = .153 (Adjusted R Squared = .065)					

Notes: The effect of Age is moderately significant (Sig. = 0.055).
Direction of Age: TR_L5_inverse size is higher for Age_Cats 25 and 26, but there is a pronounced decrease after Age_Cat 26 (CIs overlap)

TR_L5_inverse (3-way of VNC_inverse*Age_Category*SES*Sex):

Levene's Test of Equality of Error Variances: F=1.366, df1=43, df2=245, Sig=0.076

Do not reject the null that dependent variable error variances equal across groups.

Tests of Between-Subjects Effects
Dependent Variable: TR_L5_inverse

Source	Type III Sum of Squares	df	Mean Square	F	Sig.
Corrected Model	.001[a]	43	2.480E-5	1.474	.037
Intercept	.192	1	.192	11410.186	.000
SES	1.379E-6	1	1.379E-6	.082	.775
Age_Category	.000	12	2.277E-5	1.353	.190
Sex	5.913E-5	1	5.913E-5	3.514	.062
SES * Age_Category	.000	11	1.595E-5	.948	.495
SES * Sex	3.799E-6	1	3.799E-6	.226	.635
Age_Category * Sex	.000	11	1.943E-5	1.154	.320
SES * Age_Category * Sex	4.176E-5	6	6.960E-6	.414	.870
Error	.004	245	1.683E-5		
Total	.489	289			
Corrected Total	.005	288			
a. R Squared = .206 (Adjusted R Squared = .066)					

Notes: The effect of Sex is moderately significant (Sig. = 0.062).
Direction of Sex: TR_L5_inverse size is higher for females (CIs overlap)

AP_L5_inverse (2-way of VNC_inverse*Age_Category*SES):

Levene's Test of Equality of Error Variances: F=1.236, df1=31, df2=249, Sig=0.190

Do not reject the null that dependent variable error variances equal across groups.

Tests of Between-Subjects Effects
Dependent Variable: AP_L5_inverse

Source	Type III Sum of Squares	df	Mean Square	F	Sig.
Corrected Model	.002[a]	31	6.328E-5	1.244	.184
Intercept	.333	1	.333	6552.181	.000

SES	.000	1	.000	2.753	.098
Age_Category	.001	17	5.586E-5	1.098	.356
SES * Age_Category	.001	13	7.411E-5	1.457	.134
Error	.013	249	5.088E-5		
Total	1.035	281			
Corrected Total	.015	280			
a. R Squared = .134 (Adjusted R Squared = .026)					

Notes: The effect of SES is moderately significant (Sig. = 0.098). Direction of SES: AP_L5_inverse size is higher for middle/low SES (CIs overlap slightly)

AP_L5_inverse (3-way of VNC_inverse*Age_Category*SES*Sex):

Levene's Test of Equality of Error Variances: F=1.648, df1=42, df2=220, Sig=0.012

Reject the null that dependent variable error variances equal across groups.