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The Relationship of Childhood Stress to Adult Health and Mortality Among Individuals From Two U.S. Documented Skeletal Collections, Late 19th to Early 20th Centuries

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The Relationship of Childhood Stress to Adult Health and Mortality Among Individuals From Two U.S. Documented Skeletal Collections, Late 19th to Early 20th Centuries

by

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A dissertation submitted in partial fulfillment of the requirements for the degree of Doctor of Philosophy
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Paleopathology, Epidemiological Transition, Lifecourse, Developmental Origins of Health and Disease

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ABSTRACT

Although the association between social inequality and poor adult health is well established, the mechanisms by which inequality is translated into poor adult health are less clear. Increasingly, evidence suggests that many adult health problems and health disparities have their origins in early life; the developmental origins of health and disease (DOHaD) hypothesis provides an explanatory mechanism linking adverse early life conditions with permanent structural or functional changes that increase the risk for disease. This hypothesis is consistent with bioarchaeological research noting reduced lifespan among individuals exhibiting signs of childhood stress.

The principal aim of this dissertation is to contribute a bioarchaeological perspective to health disparities research by investigating how health disparities can be measured and understood in the past. This study focuses on early life conditions as a source of adult health disparity by examining a skeletal sample for the association between childhood stress and adult longevity; the relationship between childhood stress and the presence of adult health conditions; and sex, ancestry, and regional differences in these relationships. The study sampled 830 age-documented, U.S. born African American males and females and Euro-American males from the Terry and the
Hamann-Todd anatomical collections, representing socially-marginalized individuals from the late 19th- to early 20th centuries. Enamel hypoplasia, femoral length, and vertebral neural canal diameters represented childhood stress; skeletal fractures, tibial periostosis, and the diseased, missing, and filled tooth index represented adult health. Longevity was modeled with Kaplan-Meier survival curves and adult health relationships were modeled with logistic regression. Additionally, cause of death data from historic health department publications and the study sample morgue records were examined for disparity in the epidemiological transition from infectious to degenerative cause of death.

The study found mixed results for all analyses. There was no reduction in longevity for the presence of enamel hypoplasia, short femoral length, or reduced thoracic neural canal diameter. African American males had statistically significant reduced longevity for small lumbar vertebral neural canal diameters. African American males from the Hamann-Todd Collection and Euro-American males from both collections had significant relationships between vertebral neural canal diameters and adult conditions; these relationships varied among the groups but in most cases demonstrated reduced odds for having the adult condition for individuals with smaller canal diameters. African American females had no differential survival or relationships between variables over the lifecourse. All groups except for the Terry Collection Euro-American males continued to have more infectious disease deaths than degenerative
disease deaths. The study results contribute to disparities research by demonstrating that the consequences of childhood stress varied by sex and ancestry and by demonstrating within-population variation in timing of the epidemiological transition. Additionally, the study results support the contention of greater male sensitivity to environmental conditions and contributes evidence supporting the DOHaD hypothesis.
CHAPTER ONE: INTRODUCTION

The principal aim of this dissertation is to contribute a bioarchaeological perspective to health disparities research by investigating how health disparities can be measured and understood in the past. Health disparities are not simply a matter of health differences among groups; rather, health disparities are differences that are related to social inequality including racism, gender discrimination, and socioeconomic discrimination (Braveman, 2006). Inequality and poverty are powerful stressors and their correlation with poor health among living groups is well established (Marmot, 2001; Nguyen and Peschard, 2003). For example, African Americans have higher rates of all-cause mortality, infant mortality, cardiovascular disease, and low birthweight infants compared to Euro-Americans (Cutler et al., 2008; Shuey and Wilson, 2008; Burris and Collins, 2010). Individuals with lower socioeconomic status have higher morbidity, disability, and early mortality compared to individuals with higher socioeconomic status (Adler and Rehkopf, 2008; CDC, 2011). Gender-based health disparities present a more complex picture; almost universally, men die younger than women but women have higher levels of morbidity (Austad, 2006).
Over the last several decades, there is increasing evidence that many adult health conditions have their origins in early life. For example, all cause mortality, cause specific mortality, cardiovascular disease, stomach cancer, smoking related cancers, diabetes, obesity, alcoholic cirrhosis, periodontal disease, and depression all have demonstrated an association with childhood socioeconomic status (e.g. Braveman and Barclay, 2009: S165). Other researchers have focused on elucidating the mechanisms by which social and environmental factors in early life are translated into poor adult health, emphasizing factors encountered during critical periods of fetal or infant development (e.g. Barker and Osmond, 1986; Kuzawa and Sweet, 2009; Wells, 2010). Collectively, this latter body of research is referred to as the Developmental Origins of Health and Disease Hypothesis (DOHaD).

The DOHaD hypothesizes that adverse early life conditions result in permanent structural or functional changes that increase the risk for noncommunicable disease, especially obesity, heart disease, and diabetes (Ben-Shlomo and Kuh, 2002; Gluckman et al., 2007). DOHaD research encompasses a large body of multidisciplinary work with contributions from social epidemiology, biomedicine, anthropology, and animal studies. Bioarchaeological researchers have examined the association of childhood stress and mortality among archaeological populations using enamel defects (e.g. Cook and Buikstra, 1979; Steckel, 2005; Boldsen, 2007; Wilson, 2014); vertebral spinal stenosis (e.g. Watts, 2011; 2013a); and stature (e.g. Kemkes-Grottenthaler, 2005). However,
several researchers recently have published studies specifically examining the relationship between stress and mortality based on the DOHaD with results that provide support to the model (e.g. Armelagos et al., 2009; Weisensee, 2013; Temple, 2014; Miszkiewicz, 2015; see also Amoroso, 2014).

To date, bioarchaeological studies are lacking in significant investigations into the relationship of early life environmental stress to adult morbidity, mortality, and health disparity among historic U.S. populations. This line of research is important in light of the well-documented health disparities that continue, or even widen (e.g. Olshansky, et al., 2012), in the U.S, as well as health disparities that exist within and between developed and developing nations. Bioarchaeological researchers can investigate health disparities from a DOHaD perspective using data that are not easily observable in living humans. The current study also is important because it examines the relationship of child developmental stress not only to death, but also to the intervening period between childhood and death; this lifecourse approach, to date, is infrequently employed in published bioarchaeological research.

**Research Design**

The skeletal remains of 830 African American males and females and Euro-American males are evaluated for childhood stress using vertebral neural canal diameters, femoral lengths measurements, and enamel hypoplasia presence. Skeletal
conditions reflecting accumulated adult health were documented as well, consisting of
dental health represented by caries, antemortem tooth loss, and dental fillings; tibial
periosteal lesions; skeletal fractures; and cause of death data from morgue records.
Relationships were analyzed using Kaplan-Meier survival analysis, binary logistic
regression, and means and frequency comparisons. Historic mortality data from
national and city populations were analyzed graphically to discern disparity in cause of
death. The skeletal remains examined for this study were sampled from the Robert J.
Terry and Hamann-Todd collections, representing individuals who likely experienced
social inequality and poverty for at least a part of their lives. The years covered by this
study coincide with the epidemiological transition of the late 19th to early 20th century,
allowing evaluation of disparity in infectious disease mortality trends.

Research Questions

• Question One: In what ways and to what extent is childhood stress associated
  with reduced adult survival?

  o If individuals with signs of childhood stress died younger than those who
    lacked such signs, this suggests that the environmental conditions associated
    with that stress and the physiological reaction to it caused lasting harm.

  Based on previous studies that found reduced survival
associated with developmental stress lesions, it was expected that this association would be demonstrated for all variables.

• **Question Two: In what ways and to what extent is childhood stress related to adult health conditions?**

  o Skeletal lesions tend to accumulate with age. In settings of social inequality, lesions may accumulate due to the effects of continued exposure to adverse environmental circumstances, presence of underlying physical frailty, or both. A significant directional relationship between signs of childhood developmental stress and lesions acquired later in life suggest accumulation from adversity and frailty rather than chance. Based on the high levels of stress markers described for historic pauper cemetery samples, I expected to find directional relationships.

• **Question Three: Do the ratios of death from degenerative disease to death from infectious disease increase among the skeletal collections groups over time? How do those ratios compare to those of the local and national populations?**

  o This question addresses the epidemiological transition: degenerative disease (e.g. cardiovascular disease, diabetes) replaced infectious disease as the leading cause of death in the U.S. over the late 19th to early 20th century, largely due to improvements in living standards and sanitation. If the study groups show a continued greater proportion of infectious disease death relative to degenerative disease and their patterns differ from those of the local and national populations, it suggests the benefits of
those improvements were not equitable. Based on the historical literature regarding variation in timing of epidemiological transition, the study groups were not expected to show evidence of transition and will have higher infectious disease ratios than their counterparts.

• **Question Four:** *In what ways and to what extent do the results of Questions 1 through 3 (i.e., survival related to childhood stress, relationship between childhood stress and adult health, and cause of death trends) differ by sex, ancestry, or collection?*

  - Variation in these patterns would suggest several implications regarding population subgroups, including differential responses to stress, differential exposure to stress, and differential societal buffering of stress. Based on greater male sensitivity to environmental stress (Stinson, 1985), I expect sex differences will be found. Based on the current health disparity literature, I expected to find ancestry differences, particularly in a lower age-at-death for African American males. Regional differences are expected based on differences in population increases between the two regions, described in Chapter 4.

**Organization of the Dissertation**

Chapter 2 reviews the historical background and concepts relative to the study, using literature from bioarchaeology, medical anthropology, history, social
epidemiology, and biomedicine. Concepts of health and inequality in the past and the present, relevant bioarchaeological studies of health in the U.S., historic public health conditions, and the epidemiological transition are discussed to contextualize the circumstances in which the study individuals lived. Chapter 3 discusses the study samples and the sampling strategy, and provides context specific to the cities in which the study sample individuals died. Chapter 4 outlines the osteological and historical data collection methods and the data analysis methods. The study results are presented in Chapter 5, followed by discussion and interpretation of the results in Chapter 6. Conclusions are summarized in Chapter 7, followed by a discussion of study limitations and suggestions for continued research.
CHAPTER TWO: LITERATURE REVIEW

This chapter provides the theoretical framework and the historical context for the study, drawing from the relevant anthropological, historical, biomedical, and social epidemiology literature. First, as the word ‘health’ can have multiple meanings, the concepts of health in the past and the present are reviewed to define its meaning for the present study. Next, as the overarching interest of this study is health disparity, explanatory hypotheses are reviewed, as are the effects of inequality on social groups in the present. This is followed by an overview of the health context of the late 19th to early 20th centuries and reviews of the relevant bioarchaeological health literature.

Health

Defining Health

Health is a "fuzzy concept" according to Boldsen and Milner (2012:115), difficult to define and lacking a single definition. Notions of what constitutes good or bad health, how illness is produced, and the expected social roles of the afflicted vary by culture and change through time. For example, in the mid-nineteenth century U.S.,
tuberculosis was believed by many physicians and laypersons to be a hereditary condition, susceptibility to which was affected by one’s family background, ethnicity, and possibly the emotional state of the parents at time of conception (Ott, 1996). So-called “consumptives” were not stigmatized or institutionalized; rather, they were expected to participate in society, marry, and have children (Rothman, 1994). Later in the nineteenth century, however, as theories of disease causation increasingly turned toward pathogen theories, the condition of consumption was recast as the communicable infectious disease tuberculosis, and the afflicted were societally stigmatized and feared, often forcing them to hide their illness.

The World Health Organization (WHO, 1946:100) sought a universal definition of health that considered more than the physical aspect, defining it as "a state of complete physical, mental, and social well-being and not merely the absence of disease or infirmity." This definition is criticized in large part because the requirement that all of these domains of well-being be complete is unrealistic and unattainable; under these criteria, most people would be considered unhealthy (Huber et al., 2011:2). Within medical anthropology, health is considered a broad construct, which, similar to the WHO definition, occurs within physical, psychological, and social domains but also considers an individual’s ability to function in social roles (Sobo, 2011). The prevailing biomedical model prioritizes the corporal manifestation of health, viewing disease as “distinct, discrete…entities that exist (in theory) separate from other diseases and from
the social groups and contexts in which they are found” (Singer and Clair, 2003:424). In contrast, medical anthropologists generally make a distinction between disease and illness. Disease is a measurable anatomic or physiological irregularity, whereas illness is the personal, culturally, and socially constructed experience of being unwell (Kleinman, 1986:144-148; Sobo, 2011:15). The problem with these constructs of illness and disease is that they “recapitulate the mind-body dichotomy” (Sobo, 2011:15) so that disease, by virtue of being body-centered, is considered to be factual and objective while illness, associated with the mind, is considered to be subjective and thereby possibly not ‘real.’ Additionally, these constructs are criticized because they address health only at the individual level, failing to consider the role of political-economic forces in shaping health, particularly in producing and maintaining differential health among and within societies (Singer, 1998).

**Bioarchaeological definitions of health**

It must be noted that while health is one of the major focuses of bioarchaeological research, it infrequently has been defined in the bioarchaeological literature. Challenges complicating the evaluation of health in prehistoric populations famously were outlined in the influential “osteoarchaeological paradox” article (Wood et al., 1992), followed by a period of brisk discussion and debate (e.g. Jackes, 1993; Cohen, 1994; Wood and Milner, 1994). Recently, there has been a resurgence of interest in what is meant by the word ‘health’
and how, or if, it can be evaluated in skeletal groups (e.g. Reitsema and McIlvaine, 2014; Temple and Goodman, 2014; DeWitte and Stojanowski, 2015; Gowland, 2015).

For bioarchaeologists, the problem with the health concept lies not just with how to define it but also how to measure it using a very limited number of variables. Boldsen and Milner (2012:115) directly address the difficulty of defining health, noting that it occurs along a spectrum from good health to health so bad the individual is about to die. Disease is considered to be a condition identifiable in skeletal remains that is associated in some way with mortality (Boldsen and Milner, 2012:116). Angel (1969:430) also defined health by mortality, stating that because disease and accidental death determine longevity, longevity is usually the best measure of adult health. Wood (1998:104) defined well-being as “any aspect of individual health or physical condition that is either positively associated with the probability of childbirth or negatively associated with the risk of death.”

Not all researchers agree that mortality is the best definition or proxy measure for health, noting that poor health, even if minor, has social and cultural repercussions through decreased work capacity (e.g. Goodman, 1993). Additionally, not all conditions considered to reflect poor health cause early death. Ortner (2003) emphasizes the latter point, noting that while skeletal lesions are a clear indication that a pathological process occurred, the impact on the individuals’ ability to function and reproduce is less clear.
The predominant model used to explain health in archaeological populations is the nonspecific stress model (Goodman et al., 1984b; Goodman and Armelagos, 1988). In brief: 1) physiological stress responses occur within the context of a feedback loop consisting of environmental resources and constraints; sociocultural factors, resources and constraints; and individual biology; 2) the stressor and accompanying responses may or may not manifest in observable skeletal or dental changes; and 3) there may be differential buffering among groups within the population. (Goodman et al., 1984b; Goodman and Armelagos, 1988; Goodman and Martin, 2002). Stress markers thus are useful proxies for health (Reitsema and McIlvaine, 2014), but “should be evaluated in terms of mortality and survivorship” (Temple and Goodman, 2014: 5).

The present research, with a focus on the interrelatedness of social inequality and health disparity, considers health from a political-economic perspective but a more pragmatic definition of health is required when working with the relatively limited information that can be obtained from skeletal material. Health can be defined on a functional level as "the ability to adapt and self-manage" (Huber et al., 2011:2). (Note that this definition of functional health is in contrast to the Neo-Marxian notion of functional health as that which employers expect from their workers - i.e., that workers be able to perform the labor required for the job (Singer and Baer, 2007:78). This functional definition emphasizes resilience, which is the ability to cope with and recover from physical, social, and mental stressors (Hicks and Miller, 2011:89). The
concept of resilience contrasts with frailty, which represents decreased reserve and resistance to stressors and increased susceptibility to illness and death (Bergman et al., 2007; Milner et al., 2008). The concepts of frailty and resistance are pertinent to understanding the health patterns observed in living populations as well as cemetery samples, and in the present research will be observed by proxy through skeletal lesion and injury accumulation over the lifecourse relative to how long an individual survived.

*Turn of the 20th century health beliefs: emic and etic*

As discussed previously, medical anthropologists make a distinction between disease and illness; disease is considered to be the presence of a measurable infirmity or abnormality, and illness is considered to be the personal experience of an afflicted individual (Sobo, 2011). Beyond these categories of objective and subjective experience, an additional distinction is made between the etic and emic perspectives. The emic view is from within, referring to the perspective of a member of a particular society; the etic view is from without, referring to the perspective of an outsider to that society (Quinlan, 2011). The period of time under study – the late 19th to mid 20th centuries – is well documented with primary sources from physicians and public health reformers. For the largely middle-class public health reformers, the health and behavior of the poor were of great concern, including the impact their health and behavior may have on society in general; the public health literature of the time period reflects the author’s
opinions about how the poor should behave, live, and clean their homes. In contrast, the emic view is very difficult to find - how did the disadvantaged members of that society define health and what were their opinions regarding the measures taken on their ‘behalf’? I was unable to locate any primary sources specific to health that could be attributed to destitute individuals living at the turn of the 20th century. Instead, the discussion below is taken from the history of health and historical archaeology literatures, and from early to middle 20th century sociological research.

The health beliefs for the time period of study coincide with the transition from antebellum, miasma theories of health to a professionalized, germ theory of disease described further below. In the antebellum period, the general belief among Euro-Americans was that health was dependent on physiological balance, and that disruptions in balance could result not only from the natural environment but also from moral and mental influences (Fett, 2002). Antebellum notions of what constituted good health varied by locale. Nineteenth century urban, middle class health reformers viewed the New England farm woman as a model of “robust physical health” who was “vigorous” from fresh air and exercise (Borish, 1990:17). In contrast, rural health reformers believed that farmwomen were in poor health due to too much physical exertion from the heavy, grinding workload that farm life entailed (Borish, 1990). Physical degeneration was considered a medical, spiritual, and physiological problem; a strong will required a strong body, especially for males (Park, 2007:1543-1546). Notions
of morality, vigor, degeneration, and defectiveness later became entwined with notions of genetic inferiority in the early to middle 20th century eugenics programs (Patterson, 2001).

Later in the nineteenth century, as disease began to be associated first with unsanitary conditions and later with specific pathogens, notions of health became public health oriented. As the poor and immigrants often lived in crowded and substandard housing, they became the public face of disease and much attention went to controlling their behavior. Notions of who was deserving of help held class and morality components – for example, health reformers expended more effort on helping the so-called worthy poor than those considered unworthy for having brought their problems on themselves, for example, alcoholics and the homeless (Lerner, 1997). Some health worker’s attitudes toward the poor who did not follow medical recommendations are reflected in the words they used to describe them: “careless, irresponsible, and vicious” (Lerner, 1997:1424).

While information about the attitudes of health workers and reformers regarding the poor and working class is available, the view from within the working class and poor is difficult to locate; the little that is available is found within outsiders’ accounts rather than primary sources. Several authors have noted disagreement between the working class and the medical practitioners about what constituted an illness or being ill (e.g. Zamkin, 1940; Koos, 1954; Stoeckle et al., 1963). For example, laypersons might
have considered whooping cough to be a normal part of a child’s life, while physicians considered it a life threatening illness (Zamkin, 1940:394).

More insight may be gained from Koos’ (1954) study of 1940s health among 500 families in a small upstate New York town. This study will be discussed in some detail as the individuals in Koos’ study lived close to the time period of the present research and may have had similar health beliefs and practices. Koos found that health attitudes and practices differed based on socioeconomic status (SES). Compared to individuals from the highest SES (professionals and white collar workers), individuals in the middle SES (skilled and unskilled wage workers and their families) and the lowest SES (sporadically employed workers and their families) did not consider having a potentially serious symptom (e.g., blood in stool, unexplained weight loss, chest pain) to be a problem. Simultaneously, individuals in the lower and middle SES more often reported experiencing these symptoms on the health research survey. High SES individuals were more likely to seek medical care than low SES individuals; the lowest SES individuals were more likely to disregard a symptom, but when they did attend to a symptom they more often sought advice from druggists than from physicians. In part, the reluctance to seek medical care stemmed from mistrust of doctors but also from financial concerns and constraints. For example, a low SES woman suffering vaginal prolapse for two years explained that although she wished to seek corrective care, other things were “more important”, such as needing a new car, and whether or
not her husband remained employed (Koos, 1954:37). The lowest SES households also reported “exceptional stocking” (Koos, 1954:88) of liver, kidney, and stomach pills and tonics. The reliance on and appeal of these pills is succinctly summarized by a public health nurse interviewed for the study: “Anything that promises to make you feel like a million dollars for 30 cents is welcome if you aren’t getting much out of life” (Koos, 1954:89).

Finally, working class notions of health seem to be tied closely to the ability to work. As mentioned previously, employers viewed worker’s’ health from a functional standpoint, requiring that they be physically able to perform the work they were hired for (Singer and Baer, 2007). It seems reasonable to expect that workers and individuals with limited income may have shared this definition out of economic need. A detailed discussion of occupational health history is outside the scope of this study, but in brief, it is fair to say that rapid industrialization made the U.S. an increasingly dangerous place to work (Rosner and Markowitz, 1997); workers had few rights (Kraut, 1994); and most employers of the late nineteenth to early twentieth centuries did not go to great lengths to protect workers’ health. For those employers that did value workers’ health, their public position at times was at odds with the material evidence (Beaudry, 1993). For example, the planned industrial city of Lowell, Massachusetts was considered to have a high standard for worker health from its inception, yet archaeological excavations and historical research revealed that conditions at worker’s boardinghouses
deteriorated by the late 19th century, coinciding with management policy changes emphasizing profit (Mrozowski et al., 1989).

Accounts of nineteenth century workers indicate they valued their well-being and shared municipal concern over sanitation, adequate housing, and disease control, but from a standpoint of personal concern rather than public health – in other words, how these conditions affected their daily lives (Beaudry, 1993:90). From a pragmatic standpoint, the poor simply could not afford to be sick. In the latter nineteenth and early twentieth centuries, the working poor suffering from tuberculosis tended to ignore or hide their symptoms, in part out of lack of recognizing the severity of the symptoms but also because they had to work (Ott, 1996). Women working inside the home were affected as well; for example, a woman from the Regionville study stated:

“I wish I really knew what you meant about being sick. Sometimes I’ve felt so bad I could curl up and die, but had to go on because the kids had to be taken care of, and besides, we didn’t have the money to spend for the doctor – how could I be sick?...How do you know when you’re sick, anyway? Some people can go to bed most anytime with anything, but most of us can’t be sick – even when we need to be” (Koos, 1954:30).

**Health Disparity**

Inequality has been described as a “pathogenic biosocial spiral” (Nguyen and Peschard, 2003:448): social inequality adversely affects health, and in turn, poor health exacerbates social inequality. The flowing section defines and discusses the concepts of
social inequality and health disparity and reviews hypotheses regarding how social inequality is translated into poor health. In the present study, the term “social inequality” is used for all forms of inequality, not just socioeconomic. Social inequality is anything that may cause people to be discriminated against, marginalized, and to have restricted access to resources including ancestry, ethnicity, gender, social class, socioeconomic status, disability, sexual orientation, and religion. Inequality can be overt, covert, institutional, structural and or interpersonal (Krieger, 1999). The present research focuses on inequality related to ancestry, sex, and socioeconomic status due to the limitations of skeletal research and the accompanying demographic data.

**Defining Health Disparity**

The terms “health inequality” and “health disparity” appear often in the literature but without a discussion of their meaning. Not all differences in health are health inequalities or disparities; rather, disparities are a particular type of difference in which disadvantaged social groups experience elevated risk or worse health than more advantaged social groups (Braveman, 2006). Health disparities are differences that are “unnecessary…avoidable… unfair and unjust” (Adler and Stewart, 2010:6). A great deal of literature in the biomedical, anthropological, and other social sciences addresses health inequality in developed and developing countries; however, the following discussion on health inequality focus on the U.S. for purposes of space as well as for
relevance to the research topic. Health disparities in socioeconomic status, ancestry, and gender are discussed below.

**Health disparity and socioeconomic status**

The inverse relationship between socioeconomic status and health is well established and is widely corroborated by numerous studies. This link is evident not only for absolute deprivation but also for relative deprivation (Braveman and Gruskin, 2003). Individuals with lower socioeconomic status tend to have higher morbidity, disability, and risk for premature mortality than individuals with higher socioeconomic status (Power and Matthews, 1998). For example, multiple health conditions follow a strong, consistent, socioeconomic health gradient that disproportionately affects the poor, including: cardiovascular disease, diabetes, hypertension, tuberculosis, chronic pulmonary disease, gastrointestinal disease, accidental and violent deaths, adverse birth outcomes, the prevalence of having any chronic disease, and all-cause mortality (Adler and Ostrove, 1999:8). The same study also noted that the SES association with cancer is complicated and not consistent: breast cancer and malignant melanoma are more associated with higher SES, due in part to behavioral differences such as delayed childbirth and recreational tanning, respectively. Once diagnosed, however, lower SES individuals have shorter breast cancer survival times. The Center for Disease Control (National Center for Health Statistics, 2012:38-40) reports that compared to individuals living well above the poverty line, individuals living at or below the poverty line have
three times higher frequencies of depression; 3.5 times the frequency of edentulism in adults aged 45 – 64 years; and almost two times the frequency of having two or more chronic health conditions.

Health disparity also is found among differing educational levels. A higher education level is associated with better health and greater longevity (Elo, 2009). U.S. life expectancy in the year 2000 was seven years less for those with 12 or fewer years of education than it was for those with at least some college (Meara et al., 2008:313). The onset of cardiovascular disease, chronic pulmonary disease, and diabetes was five to 15 years earlier for individuals with eight years of education or less compared to those with 16 years (Elo, 2009:557). Higher levels of maternal education are associated with lower infant mortality and better child health. Poor health also is demonstrated to be related to living in disadvantaged neighborhoods: after controlling for occupation, education level, and income, individuals living in disadvantaged neighborhoods had higher incidences of coronary heart disease than those in higher SES neighborhoods (Diez-Roux et al., 1997).

Children’s health also follows an SES gradient. A review of large-scale child health studies from 1982 through 1996 found that as SES decreased, all-cause mortality increased regardless of parents’ education, occupation, or income level (Boyce et al., 2002). Lower SES children were more likely than higher SES children to die from chronic conditions such as asthma, congenital anomalies, and cancers, as well as from
acute conditions including flu, pneumonia, and injuries. Lower SES children experience
greater burdens of morbidity: they have more frequent hospitalizations for asthma and
other chronic conditions; increased numbers of visual and hearing disorders; higher
blood lead levels; higher rates of rheumatic fever; and higher injury rates (Boyce et al.,
2002).

**Health disparity and ancestry**

It is well established that African Americans experience worse health than Euro-
Americans. Disparities are found at all SES levels even after adjusting for education and
income level (Cutler et al., 2008). African Americans have shorter life expectancies:
current life expectancy from birth for African American males is 71.1 years versus 76.1
years for Euro-American males; and for African American females, it is 77.6 years
versus 81.2 years for Euro-American females (National Center for Health Statistics,
2012:10). African Americans have significantly higher rates of all-cause mortality as well
as mortality from cardiovascular disease, cancer, diabetes, stroke, and homicide (Cutler
et al., 2008; Shuey and Wilson, 2008). African American children have significantly
greater mortality in the first year of life and also are more likely to be have been a
preterm or low birthweight delivery (Cutler et al., 2008). Current infant death rates are
12.9 per 1000 live births for African Americans versus 5.6 per 1000 live births for Euro-
Americans (National Center for Health Statistics, 2012:10).
African Americans have a higher burden of infectious disease than Euro-Americans. For example, a seroprevalence study of select infectious diseases among individuals considered to be at high risk for developing the disease demonstrated that African Americans had significantly higher rates than Euro-Americans in five of the six diseases measured (hepatitis A, B, and C; herpes simplex 2; and H. pylori) (McQuillan et al., 2004). In a comparison of health measures by educational status, African Americans at the highest educational levels scored lower than Euro-Americans in all measures (infant mortality in women older than 20 years; life expectancy at age 25; sedentary behavior of adolescents; coronary heart disease; diabetes; and adult obesity (Braveman et al., 2010).

Additionally, although some health conditions have a genetic component, race-based health disparities largely are explained by social, environmental, and cultural factors, not genes (Gravlee, 2009; Marks, 2013). Racial discrimination is a primary factor in health disparity by creating differential access to resources, safe neighborhoods, and adequate health care, and by causing high levels of stress from workplace inequality, residential segregation, and efforts to support families using limited resources (Shuey and Wilson, 2008:216).

**Health disparity and gender**

Gender differences in health have long been observed but the disparities are not as clear-cut as they are for ancestry and SES related inequality. Women outlive men in
almost every country; in the few instances when they have not (e.g. Bangladesh), the male advantage in longevity is attributed to differential access to nutrition and healthcare (Austad, 2006). Women have a greater life expectancy at birth than males, currently at 80.4 versus 75.2 respectively for the U.S. (Read and Gorman, 2010:373).

Although women outlive men, they are susceptible to the same diseases: the three age-adjusted leading causes of death for both genders are heart disease, stroke, and cancer (Rieker and Bird, 2005; Read and Gorman, 2010). Men die at a higher rate at every age for virtually all causes of death, including heart disease, cancer, flu and pneumonia, cerebrovascular accidents, accidents, and chronic obstructive pulmonary disease (Austad, 2006). Men have higher rates of life-threatening chronic illness at younger ages including heart disease (Crimmins et al., 2010). Women are more likely to have degenerative conditions such as arthritis that are not of themselves life-threatening but do affect quality of life. Women also are more likely to have functional limitations than do men (Warner and Brown, 2011).

In contrast to their increased longevity, women have higher rates of morbidity (Warner and Brown, 2011). The gender differences in morbidity are not as clear as the mortality differences, with the “gender gap” (Read and Gorman, 2010:373) varying widely by study, disease and life stage. Elevated morbidity for women is attributed to their greater longevity, although overall women live for more years without disease than men.
Gender differences in mortality and morbidity are attributed to a combination of biological, behavioral, and social factors (Crimmins et al., 2010; Read and Gorman, 2010). Explanatory hypotheses for women’s greater longevity and biological robusticity include: 1) greater immunological activity for women; 2) the protective effects of estrogen, notably in maintaining favorable high density lipoprotein cholesterol to low density lipoprotein cholesterol balance and a better ability to counter oxidative stress; and 3) the ability to inactivate deleterious alleles on the active X-chromosome (Austad, 2006; Pinkhasov et al., 2010). Additionally, males show evidence of greater sensitivity to environmental stressors and changing environmental conditions. In a review of the literature regarding male buffering, Stinson (1985) found that under conditions of stress, males had higher late prenatal demise and greater intrauterine growth restriction than females, but also showed greater response in growth to maternal supplementation. Providing additional evidence for male sensitivity, Colombian boys and girls with protein-deficient diets had delayed skeletal maturation, but girls subsequently experienced catch-up growth while the boys did not (Stini, 1969). Guatelli-Steinberg and Lukacs (1999) reviewed the enamel hypoplasia literature to date and found that, while not consistent, males in stressed environments had a slight trend to exhibit more enamel defects.

Biological factors alone do not explain the gender gap variation in morbidity that has been observed by time and social group; instead, the higher morbidity is likely
influenced by SES differences, with women remaining overall economically disadvantaged in relation to men. When inequalities in SES are factored into studies, the effect of gender on health is diminished (Read and Gorman, 2010).

**Embodiment of social inequality**

While the links between social inequality and poor adult health are well established, the mechanisms by which inequality is translated into poor adult health are less clear. Distinguishing among contributions from social, environmental, and biological factors is difficult even for epidemiologists who, by virtue of working with living people, have access to a far greater number of explanatory variables than do bioarchaeologists. Several sociocultural explanations have been developed within epidemiology, for example the social cohesion hypothesis, which notes that social network density correlates positively with better health outcomes; neomaterialist hypotheses, linking poor health with level of investment in infrastructure and social services; and pathway models, in which poor circumstances in childhood influence social trajectories and subsequent socioeconomic status and health behaviors (Graham, 2002; Nguyen and Peschard, 2003). An example of the latter group of explanatory models is the cumulative advantage model, which hypothesizes that the early life relationship between socioeconomic status and health subsequently is magnified over the lifecourse; advantaged individuals have an increasing health advantage relative to others over time (Willson et al., 2007:1887). Likewise, the social and physical effects
from disadvantage experienced at different life stages accumulate throughout life, conferring increased risk for poor health during adulthood (Graham, 2002)

The strength of the sociocultural explanatory models lies with the examination of societal structures that produce and maintain social stratification and health inequality, but these models alone cannot explain the underlying mechanisms that translate social factors into poor health. Biocultural lifecourse approaches that examine the contributions and inter-relatedness of physiological mechanisms as well as societal mechanisms over time have the most promise to explain the embodiment of social inequality.

A large body of research has been directed toward that explanation in the last several decades. Although social science researchers and public health workers had long been aware that poor adult health is linked to poor childhood circumstances, biomedical interest in the subject was sparked with the publication of British research that found correlations between maternal diet, neonatal birthweight, and adult cardiometabolic disease (e.g. Hales and Barker, 1992; Barker and Osmond, 1986). In the last several decades, multidisciplinary research in this area has expanded from an emphasis on fetal origins of health to include postnatal conditions and generally is referred to as the Developmental Origins of Health and Disease (DOHaD) hypothesis. The DOHaD hypothesizes that adult health is related to early life environmental conditions, and in particular, that adverse conditions may result in permanent
structural or functional changes that increase the risk for noncommunicable disease, especially obesity, heart disease, and diabetes (Ben-Shlomo and Kuh, 2002; Gluckman et al., 2007). An organism’s capacity for change in response to environmental change is referred to as **plasticity**. More specifically, developmental plasticity refers to the organism’s ability to adapt phenotypically to environmental cues, due to a given gene’s ability to produce a range of phenotypes (Gluckman et al., 2010). Emphasis is placed on critical periods, defined as narrow time frames of development when environmental stimuli can have lasting effects on the structure or function of tissues or body systems (Ben Shlomo and Kuh, 2002). Critical periods generally are considered to occur during the antenatal and lactation-dependent stages (Kuzawa, 2013). Changes may become permanent or long-term as traits lose plasticity after childhood (Wells, 2010).

In brief, the DOHaD hypothesizes that the fetus or neonate adjusts its developmental trajectory in response to environmental cues; for example, in response to maternal malnutrition, a fetus may re-allocate resources to critical organs such as the brain by diverting them from organs or tissues with less critical metabolic needs (Gluckman et al., 2010). While doing so may be protective for the short term, there are potential long-term negative consequences due to alteration in the structure and function of other important systems. The long term consequences – particularly increased risk for noncommunicable disease – are hypothesized to develop along two primary pathways (Hanson and Gluckman, 2015). The first pathway involves plasticity
in response to cues from environments that are interpreted as poor but that lie within
the evolutionary experience of the species; the resulting phenotypic changes are
thought to promote survival to reproductive age in the event of continued exposure to
adverse circumstances. If the environmental cues were misread or if subsequent
circumstances are improved, the resulting mismatch between the individual and the
environment increases risk. The second pathway results from exposure to
circumstances that do not lie within the evolutionary experience of the species, for
example, inappropriate weaning, excessive use of infant formula, and tobacco smoke
(Hanson and Gluckman, 2015). A final point about the DOHaD is that the effects can
be transgenerational. For example, exposure to adverse circumstances encountered in
uterus affects not only the fetus but also, if the fetus is female, her children, because ova
are developed prior to birth (Haas, 2013).

While much of the DOHaD research focuses on the connection between early life
stress and degenerative disease, an increasing body of research investigates the effect of
early life pathogen exposure, toxin exposure, and stress on the developing immune
system in regard to susceptibility to infectious and autoimmune disease later in life. For
example, experimental research on rodents demonstrates that exposure to
polychlorinated biphenyls during early developmental stages subsequently is
associated with increased susceptibility to infection, and epidemiological studies
suggest that childhood exposure to polychlorinated biphenyls is correlated with recurrent otitis media and recurrent respiratory infections (DeWitt et al., 2012).

In brief, the immune system is a complicated network of dedicated cells, tissues and molecules that surveil for, recognize, and defend against threats to the host (O’Connor et al., 2014). The innate system, present at birth, provides the first line of defense through non-specific pattern recognition of invaders, serving to defend against pathogens until the adaptive immune system can respond. The external innate system (skin, mucosal tissue, body secretions) prevents pathogens from invading internal tissues. If the external system is breeched, the internal innate system initiates a rapid but standard response of physiological barriers (increased oxygen tension, increased temperature), phagocytosis (engulfment and destruction of pathogens by monocytes and macrophages), and inflammation (Williams, 2012). Inflammation is a series of vascular events that is initiated if tissues are injured or phagocytosis fails: the clotting cascade is initiated at the infection site, trapping pathogens within clots; increased blood flow brings more phagocytes to the affected area; and increased capillary permeability allows phagocytes to cross vessel walls into the infected tissue spaces (Keogan et al., 2006). The inflammatory response also initiates the adaptive immune response. The adaptive system is highly specific, functioning to destroy pathogens as well as toxins produced by a pathogen, principally through the T and B-lymphocytes. Distinguishing features of the adaptive immune system are the ability to differentiate
between classes of pathogens, to adapt the response to the pathogen, to differentiate
between self and non-self molecules, and to create pathogen-specific immunological
memory that confers immunity to subsequent exposure to that pathogen (Williams,
2012).

Knowledge about the relative impact of exposure during specific developmental
periods on long-term immune function is limited (Slopen et al., 2013). Recent
developmental immunotoxicology research indicates that effects on immune system
structure and function depend on when in the developmental process the exposure
occurs. Developmental disruption appears to have detrimental consequences to both
the innate and adaptive immune responses. Three of the five major immune system
developmental windows occur in utero: initiation of hematopoiesis; migration of stem
cells and progenitor cells; and establishment of bone marrow and the thymus as
primary hematopoiesis and lymphopoiesis sites, respectively (West, 2002; DeWitt et al.,
2012). The hypothesized impacts of disruptions during these periods include failure of
stem cell formation and partial to complete immune failure; impaired post-natal T-cell
function and impaired innate immunity; and increased risk of cancer and autoimmune
disease (West, 2002; DeWitt et al., 2012). The next critical phase, maturation to
immunocompetence occurs from birth to about one year of age; disruption is this phase
is hypothesized to result in increased incidence of childhood infections and reduced
response to vaccines. The fifth phase, establishment of immune memory, occurs from
about one year to 13 years of age; disruption during this phase is hypothesized to be linked with increased risk for common and opportunistic infections, cancer, and allergies (West, 2002; DeWitt et al., 2012).

Very recently, bioarchaeological researchers have sought to interpret the meaning of developmental stress markers in relation to the DOHaD concept. Interest in this line of inquiry appears to have begun with the publication of Armelagos’ and colleagues’ (2009) article outlining the use of tooth enamel defects as evidence in support of critical period stress resulting in early mortality. Subsequent to that publication, several researchers have published studies specifically testing hypotheses informed by DOHaD theory. Using an early 19th to middle 20th century documented skeletal sample from Lisbon, Weisensee (2013) found that individuals who died from degenerative diseases had significantly higher cranial fluctuating asymmetry scores than individuals who died from infectious diseases. Using the same Lisbon skeletal collection, Amoroso and colleagues (2014) found that individuals with at least one mandibular canine enamel defect died nine years earlier than those without enamel defects, but after controlling for socioeconomic status, year of birth, and cause of death category, the effect became nonsignificant. This indicates that early stress alone did not account for the early mortality and that later life experiences may have made a larger contribution, indicating support for the cumulative health hypothesis rather than of the DOHaD. Temple (2014) tested two alternate DOHaD hypotheses using tooth enamel
defects among the Jomon (4000 to 2300 BP); individuals with early formation times for enamel defects died younger and were more likely to have subsequent defects, suggesting that the best explanation for the presence of the lesions was phenotypic plasticity in response to stress (i.e., a predictive adaptive response). Miszkiewicz (2015) found, in a mixed socioeconomic status sample from medieval Canterbury, that individuals of lower socioeconomic status had higher frequencies of enamel defects as well as a significantly lower age-at-death.

The degenerative disease and developmental immunology literature provide an explanation for the mechanisms by which social inequality becomes embodied. Criticism of this line of research notes that much of the evidence is based on animal studies rather than human, and that to be useful for health policy, a better understanding is needed of how effects vary along the spectrum of adverse conditions (Haas, 2013).

The Health Context of the late 19th and Early 20th Centuries

Epidemiological Transition

The study years encompass two important secular trends: the demographic and epidemiological transitions. The *demographic transition* describes post-industrial revolution declines in fertility, mortality, and population growth and the subsequent
shift in population age distributions (Gage, 2005). The *epidemiological transition* models changing patterns of disease and causes of death (Omran, 1971). More specifically, the 19th to 20th century transition refers to degenerative disease replacing infectious disease as the leading cause of death.

Although the exact timing of the transition is debated, it generally is agreed that mortality decline began in the United States in the late 19th century (Elman and Myers, 1997; Clay and Troesken, 2006; Gage, 2005). The cause of the infectious disease decline remains somewhat under debate, but improvements in sanitation and other public health measures appear most likely to be the major contributing factor especially during the pre-antibiotic era (Haines, 2001; Gage, 2005; Clay and Troesken, 2006; Smith, 2009). It should be noted that transition is not a homogenous process and that it differs by urban or rural setting, social status, and geographic area. Omran’s original model of transition is criticized in relation to contemporary populations because: 1) the explanations for transition are speculative, relying on small sample sizes or unreliable data; 2) poverty and other important social factors may not be addressed; and 3) the theory does not adequately address differential change for different social subgroups (Zuckerman et al., 2014:3). Relevant to the present study, the transition in health that occurred over the middle 19th to middle 20th centuries is well documented when viewed broadly. This research directly addresses the third criticism by examining the differences in cause of death trends among the study sample and the larger population.
A central hypothesis of the present study is that transition lagged for individuals experiencing social inequality.

Prior to the turn of the 20th century, the U.S. was a high-mortality area with tuberculosis and other respiratory illnesses as the leading cause of death. Diphtheria, whooping cough, scarlet fever, enteric disorders, measles, smallpox, and malaria were endemic. Infant mortality was a major contributor to the overall death rate, with most infant deaths attributed to diarrhea, teething, summer fever, colic and convulsions (Duffy, 1997). For early 20th century African American infants, the mortality rate was 184.3 per 1000 live births as opposed to 99 per 1000 live birth for Euro-American infants (Fishback et al., 2001:95). Maternal mortality also was high, accounting for 36 percent of all African-American female deaths and 27 percent of all Euro-American female deaths (Lee, 1977:260). Overall mortality rates in the U.S. fell by nearly 40 percent between 1900 and 1940 (Cutler and Miller, 2004:1). There was a concurrent increase in life expectancy at birth between 1880 and 1940: life expectancy for males increased from 41.7 years to 67 years, and for females increased from 43.5 years to 73 years (Elman and Myers, 1997:943). Ancestry-specific life expectancy from birth at the turn of the 20th century was 51.08 for Euro-American females versus 35.04 for African American females, and 48.23 for Euro-American males versus 32.54 for African American males (U.S. Department of Health, 1977:5-4).
Epidemics added to the already high morbidity and mortality loads. National-level epidemics during these years include smallpox, diphtheria, polio, and flu. The most significant epidemic during the study years was the Spanish Flu epidemic of 1918-1919. The Spanish Flu killed an estimated 675,000 people in the U.S.; the highest mortality occurred among young adults aged 25-29 and lowest mortality occurred for adults over 60 (Taubenberger, 2005:61). Pregnant women were the most likely to die if infected.

Regional differences in mortality existed between the southern states and the northern/western states, and between rural and urban areas. The picture that emerges is that individuals living in the South had worse health than the rest of the country. Data from the 1900 census show excess southern mortality; all-cause mortality for the southern states was 2501.6/100,000, while for the northern and western states, the all-cause mortality was 1681.7/100,000 (Crimmins and Condron, 1983:33). Deaths from airborne infectious diseases (tuberculosis, pneumonia, diphtheria, and flu) in the South were 636.1/100,000, compared to 446.1/100,000 in the Northern and Western states. Water and food-borne diseases show a similar excess mortality in the South, with a rate of 274.2/100,000 versus 163.8 /100,000 for the Northern and Western states (Crimmins and Condron, 1983:33).

In addition to differences in geographic regions, a substantial “urban penalty” (Haines, 2001:1) in mortality existed until about 1920 compared to suburban areas
(Harris and Mercier, 2005) and until about 1940 compared to rural areas (Crimmins and Condron, 1983; Haines, 2001). This penalty likely was due to inadequate clean water and sewage disposal, greater density and crowding, a consistently higher degree of contaminated food and water, and larger influxes of newly arrived immigrants who served as carriers as well as vulnerable new victims (Haines, 2001). Suburbs on average were healthier than cities, except for those suburbs that were industrial in character or those in unincorporated fringe areas lacking piped water (Harris and Mercier, 2005). In a comparison of the infant mortality rates (IMR) for 1923, U.S. cities had an IMR of 80 - 85 per 1000 live births, while the residential suburbs had an IMR of 59-62/1000 live births (Harris and Mercier, 2005:772). In contrast, the IMR for the industrial suburbs was similar to that of the cities, at a rate of 83/1000 live births. Rural white males had a ten-year greater life expectancy than their urban counterparts; rural white females had a seven-year greater life expectancy (Cain and Hong, 2009:451). Urban child mortality in mid-to-late 1890s was estimated to be 22 percent greater than in rural areas, decreasing to about a 13 percent difference by 1910 (Haines 2001:2).

Developments in Public Health and Sanitation

The historic decline in mortality and infectious disease is largely attributed to public health measures including public works efforts to improve sanitation and the water supply. The sanitary reform movement - in large part stimulated by volunteer
and citizen’s organizations - began prior to the Civil War in response to the increasing filth of the rapidly growing and industrializing cites and in response to epidemics (Duffy, 1997; Porter, 1999). The prevailing theories of illness causation at the time were the miasma theory, in which some diseases were caused exposure to filth or foul air, for example odors emanating from rotting garbage and sewage, and contagion, in which some diseases were caused by contact with an individual suffering from the disease (Tesh, 1995). Public health measures for this period emphasized keeping cities and streets clean, a difficult endeavor in overcrowded cities: “Festering piles of garbage littered urban streets, dead animals lay where they fell, privies and cesspools over-ran into drainless, unpaved streets. Horses defecated indiscriminately” (Leavitt and Numbers, 1997:8). On the domestic front, sanitary authorities and popular women’s magazines emphasized the importance of good housekeeping (Tesh, 1995). A few cities had pre-war health departments, but many were temporary boards created in response to epidemics and often were disbanded after the epidemic ran its course (Porter, 1999).

By linking infectious disease with crowded, dirty conditions, and focusing on improving these conditions and on quarantines, the sanitary reform movement began to reduce the urban death rate and, unintentionally, created a framework that facilitated eventual acceptance of bacteriology theory (Burnham, 2015).

Sanitary reform in the federal Army during the Civil War provided a model for newly created city and state health departments after the war (Leavitt, 1992; Porter,
1999). The Sanitary Commission - formed in 1861 in response to pressure from a citizens’ group - found appalling conditions in Army camps and thousands of sick soldiers without access to food or medical care (Duffy, 1990). After the war, most major cities formed and funded permanent health departments in response to renewed concerns with public health. Also about this time, discoveries by Koch, Pasteur and others demonstrated links between specific microorganisms and disease; discoveries of specific pathogen links, as well as the availability of technologies such as x-ray, microscopes, stethoscopes, and labs contributed to an increasing acceptance of pathogen and disease models by physicians (Leavitt, 1992; Porter, 1999; Burnham, 2015). Replacement of the miasma theory with the germ theory of disease, however, was not a uniform process. The principle of a specific pathogen-disease model was met with resistance by sectarians and particularly by some physicians who clung to the idea of illness as an inherent part of individual constitution. It was, however, relatively better received among public health workers and some laypersons, whose previous and ongoing experience with sanitation principles were very similar to the new aseptic principles (Burnham, 2015).

**Health Care and Hospitals**

Prior to the Civil War, hospitals provided only a small proportion of urban medical care (Dowling, 1982). The sick stayed home if they could. Those who could
afford to do so convalesced at home under the care of private physicians. In the event an ill person could not stay home it likely was due to poverty: there was no home to stay in or no place to lie down if they had a home.

Instead of hospitals, the dominant model of care was in the form of free dispensaries. Dispensaries had first been established in the U.S. about the time of the Revolution and by the middle 19th century had become the primary means for providing care for the urban poor (Rosenberg, 1997). The typical dispensary was staffed by a druggist and by at least one resident physician. Conditions treated were relatively minor: lacerations, upper respiratory infections, dyspepsia, dental extractions, vaccinations, fracture reductions and minor surgeries. Individuals with chronic or degenerative ailments were not treated at the dispensaries but instead sent to almshouses (Rosenberg, 1997). Some dispensaries also had visiting physicians for those too sick to travel to the dispensary and starting around 1880, some also cooperated with visiting nurse associations to send trained nurses into the homes of the poor (Dock, 1921). Many dispensaries also functioned as milk stations in an effort to provide safe milk or formula to poor children (Markel, 2000).

The practice of treating the poor at dispensaries and those of higher income at home with private physicians points to an early disparity in health care. The usual treatment modality at the dispensaries was medication prescription; there was a “routine and exclusive dependence on drug therapy” (Rosenberg, 1997:311).
Unfortunately, many of the drug therapies at the time were of dubious efficacy if not outright harmful. The middle and upper classes also experienced these therapeutics, but in contrast, they benefitted from private physicians who recommended lifestyle adjustments and altered therapeutic regimes as needed (Knox et al., 1983).

Although they remained an important health care resource for the poor through the 19th century, by the 1920s the dispensaries were being phased out or incorporated into hospital outpatient centers (Rosenberg, 1997). Additionally, hospitals had become more desirable places to be after Lister discovered that use of phenol drastically reduced post-operative infection rates and deaths (Dowling, 1982). Attracted by improved infection control, increased professionalization, medical specialization, x-ray services, and laboratory services, the middle and upper classes began seeking hospital-based treatment (Knox et al., 1983). Between 1873 and 1923, the number of hospitals increased from about 170 institutions to more than 4,500 (Rosner, 1982:357). Government hospital beds serving the poor increased in number by 211 percent between 1873 and 1889, while private hospital beds, serving the middle and upper classes, increased by 283 percent (Dowling, 1982:27).
Bioarchaeology of the 19th-20th Century U.S.

Poorhouses and institutions

Historical bioarchaeological studies of health for U.S. samples are relatively few compared to the large body of prehistoric bioarchaeological literature. Several large 18th to 19th century cemeteries have been excavated and studied, however, most notably the Dunning Poorhouse in Chicago (Grauer and McNamara, 1995; Grauer et al., 1998); the Monroe County Poorhouse in Rochester, NY (Lanphear, 1988; Higgins and Sirianni, 1995); the Milwaukee County Institution Grounds (Milligan, 2010); the Albany Almshouse (Phillips, 2001); the Oneida County Asylum (Phillips, 2001); and the Eerie County Poorhouse Cemetery (Byrnes, 2015). Except for the Oneida County cemetery, these cemeteries were comprised of individuals who, at death, likely were among the poorest members of their communities.

Milligan (2010) analyzed skeletal health among 406 adult males and 57 adult females sampled from 1,646 burials excavated from the Milwaukee County Institutional Grounds cemetery (MCIG) in 1991-1992. The MCIG was in use from 1882 – 1925, serving as a pauper’s cemetery for residents of Milwaukee institutions, unclaimed individuals from morgues, and those unable to pay for burial. More than half of the adults of both sexes were aged 35-49 at death. Thirty-three percent of females and 40 percent of males had at least one hypoplasia; nine percent of females and six percent of males had tibial periostosis; and nine percent of females and ten percent of males had
signs of infectious disease. Cemetery burial records indicate that 12 percent of individuals died from tuberculosis and two percent died from syphilis. There were no significant differences in markers between males and female at MCIG in contrast to the expectation that, because more relief services were available for adult females than for males, females’ exposure to stressors prior to institutionalization would be reduced (Milligan, 2010).

Grauer and McNamara (1995) studied a sample of 61 adults from the Dunning County Poorhouse cemetery in Chicago. This cemetery was associated with the Cook County Poor, dates from 1851-1869, and was excavated in 1990. Fifty-six percent of the sample were female and 44 percent were male, a proportion that is at odds with the workhouse reports of far greater numbers of males admitted than females and is atypical for the other pauper cemeteries reviewed. The authors’ explanation for the unusual proportion is that while more males were admitted to the poorhouse, more females than males were dying in it.

Most of the individuals in the Dunning cemetery were aged between 25 and 54 years at death, and there were no significant difference in the age distributions. Twenty-six percent of females and 24 percent of males had periostosis; 22 percent of females and 24 percent of males had fractures; 32 percent of females and 44 percent of males had canine hypoplasia; and 44 percent of females and 56 percent of males had at least one caries (Grauer and McNamara, 1998:156). None of these differences were significant.
Milligan (2010) compared her sample to published results from the Monroe County Poorhouse (MCP) and the Dunning Poorhouse studies, concluding that overall the MCIG individuals appear healthier than the earlier cemetery samples, suggesting the public health measures enacted later in the 19th century may have limited their exposure to stressors. MCIG had significantly lower frequencies in all nutritional and developmental markers compared to the other cemeteries. Tibial lesions did not differ significantly.

Phillips (2001) compared the Oneida County Insane Asylum cemetery, located in Rome, N.Y., to the Albany Almshouse cemetery. Based on the presence of gold personal jewelry, gold dental fillings, and on the lack of infant and subadult burials, it is believed the Oneida cemetery represented middle-to-upper class inmates of the insane asylum rather than poorhouse residents. The ability to compare differences between the sites in terms of socioeconomic status, however, is confounded by institutionalization and the control exerted over the inmates lives.

Active dates for the Oneida cemetery were 1860 to the 1890s and it was excavated in 1988 (Phillips, 2001). The Albany Almshouse cemetery was in use from about 1880 to 1900, and was excavated in 1990. Both cemetery samples were a mix of individual and commingled burials, totaling 100 for Oneida and 30 for Albany. Compared to the Albany cemetery, individuals interred in the Oneida cemetery exhibited significantly higher frequencies of tuberculosis (29.1 percent and 14 percent,
respectively); fractures (30.5 percent and 35.7 percent, respectively); and periostosis (29.1 percent and 14.3 percent, respectively). Skeletal pathological conditions were not reported by sex, but dental pathology in the form of a diseased-missing teeth index (DMI) showed worse DMI for Albany females (64.2) than for males (39.1) but not for Oneida females (67.4) and males (74.6) (Phillips, 2001).

Lanphear (1988) examined the health of 125 adult males and 74 adult females from the Monroe County Poorhouse (MCP) cemetery in Rochester, N.Y. The cemetery was in use from 1826 – 1863 for individuals from the poorhouse, a workhouse, and other government institutions, and was partially excavated in 1984. Consistent with other pauper cemeteries, the sample exhibits considerable signs of developmental stress, infection, and nutritional stress. Significant differences by sex consist of caries, which seven percent of all male teeth and 12 percent of all female teeth exhibited, and antemortem tooth loss, which accounted for 46 percent of all female teeth and 40 percent of all male teeth. No significant differences occur for rickets (ten percent of females, 9.5 percent of males); tuberculosis lesions (5.5 percent of females, 7.2 percent of males); and tibial periostosis (60 percent of females, 56 percent of males) (Lanphear, 1988).

In a subsequent study, Higgins and Sirianni (1995) compared cemetery records for MCP women and subadults with cemetery records for the greater Rochester area women and subadults. Tuberculosis was the leading cause of death for poorhouse
infants and accounted for considerably more deaths among all-age poorhouse subadults than the Rochester sample. Gastrointestinal disease was the leading cause of death for Rochester subadults and the second leading cause for poorhouse subadults. There were no recorded typhus deaths for Rochester, but it was a significant contributor to older poorhouse children. Tuberculosis was the leading cause of death for both groups of adult females; the second leading cause for poorhouse females was typhus at 19 percent of all deaths, yet among the Rochester females typhus accounted for only one percent of the deaths (Higgins and Sirianni, 1995). Mortality for the two samples was similar, with most females from each group dying between 20 and 49 years of age (Higgins and Sirianni, 1995). The slightly lower frequency of gastrointestinal deaths at the poorhouse may be due to more reliance on breast-feeding at the poorhouse or for the presence of fresh, uncontaminated milk from the poorhouse dairy, yet is offset by the unsanitary conditions in the poorhouse. The presence of typhus deaths at the poorhouse but not in Rochester likewise is attributed to unsanitary conditions in the poorhouse (Higgins and Sirianni, 1995).

More recently, Byrnes (2015) examined a sample of 97 male and 49 female adults from the Eerie County Poorhouse cemetery, focusing on skeletal trauma. The cemetery is located in Buffalo, N.Y.; many of the individuals were German or Irish immigrants. The cemetery was in use from 1851 to 1913 and was excavated during 2008, 2009, and 2012. Males had significantly more occurrences of at least one fracture than females at
35 percent and 18 percent, respectively. Fractures increased with age for males, indicated by significantly higher fracture presence for middle aged to older males than for younger males, at 32.4 percent and 18.4 percent, respectively (Byrnes, 2015).

Collectively, these studies demonstrate that the individuals interred in the pauper cemeteries experienced high levels of childhood stress and other pathologies, but without a consistent pattern of significant differences by sex. The present study’s sample individuals, while having slightly later years of death, are expected to exhibit similar levels of stress.

Studies of health inequality

Inequality, especially as identified by status, has long been an interest in bioarchaeological research. Most of these studies have addressed health in relation to status in precontact New World groups, European expansion, and urbanization. Bioarchaeological studies of historical, industrialized societies have been less well represented until recently and it should be noted here that there are very few studies focused on within-population health inequality in the U.S. This is due to the scarcity in the U.S. of large church or municipal cemeteries that would include higher status individuals. Most of the large samples of historic skeletal material studied and published come from pauper cemeteries or anatomical collections consisting of the society’s destitute and indigent. As such, these studies address health within population
subgroups that experienced inequality within their given contexts. The bioarchaeology
of historic groups in the U.S. that experienced inequality is reviewed below.

American and Euro-American males, analyzing 651 individuals from the Montague
Cobb, Robert J. Terry, and Hamann Todd anatomical collections. These individuals died
in Washington, DC, St. Louis, and Cleveland, respectively, and were born between the
antebellum period through Reconstruction. Both ancestry groups represent low income
or indigent individuals. African American males had significantly higher frequencies of
syphilis than Euro-American males (4.2 percent and 2.1 percent, respectively) and
tuberculosis (6.4 percent and 1.7 percent, respectively) (de la Cova, 2011). Differences in
osteomyelitis (2.5 percent and 0.4 percent), rickets (4.8 percent and 3.1 percent), and
mean ages at death were not significant. Both groups had very high rates of fracture
presence (97.9 percent for Euro-American males, 87.1 percent for African American
males); the higher rate for Euro-American males is statistically significant (de la Cova,
2010).

Within-group comparisons are a way to examine relative health inequality.
Kelley and Angel (1987) compared a combined sample of 90-two enslaved adults from
25 sites spanning the 18th and 19th centuries. Of special interest is the difference between
individuals from the Catoctin Furnace iron-works and combined individuals from the
other 19th century sites. Historical accounts including letters from iron-works owners
indicated that iron-works owners recognized the productive value of providing better food and living conditions to workers. The study results support the hypothesis that ironworkers received better food, housing, or work conditions. Catoctin furnace males had a five-year greater mean age-at-death and greater stature than the 19th century males (Kelley and Angel, 1987). The advantages males experienced at this site do not seem to have been conferred to the women and children. Compared to the 19th century group, the Catoctin individuals had higher rates of tooth enamel disruption (26 percent and 46 percent, respectively) and higher cranial-base height measurements, indicating high stress incurred during their childhood years. Catoctin females had shorter mean stature than females in the 19th century groups, despite the Catoctin males having greater stature than their comparison group. This pattern of better health indicators for the Catoctin males but not for females and children points to an overall picture of preferential treatment of the male iron workers compared to other slaves, as well as compared to the women and children at the site. Caveats to the study include mixed, small samples and the genetic component of stature, but the results are an intriguing look at within-group health inequality.

A final study compares the health of enslaved individuals from the New York African Burial Ground (ABG) with mostly free African Americans from the First African Baptist Church (FABC) in Philadelphia (Barrett and Blakey, 2011). A study of the children in these samples demonstrated that the majority of the ABG children
experienced physiological stress in childhood including anemia and infectious disease (Blakey, 2001). The frequency of enamel defects among children with deciduous dentition was 30 percent higher than that of the children in the FABC; in contrast, enamel defects of the permanent dentition of adults were 20 percent lower in the ABG than in the FABC (Barrett and Blakey, 2011:224). The different stress pattern between the adults and children within the ABG sample is attributed to: 1) birthplace, with those who died in childhood born into slavery in New York with the resulting extreme physiological stress that slavery that entails; and 2) forced migration of many of the adults in the sample, who likely experienced better living conditions as children than did the children born into slavery in New York. The FABC sample generally displays better overall adult health than slave samples but with a very high infant mortality rate (Rankin-Hill, 1989).

As noted previously, studies comparing health between socioeconomic classes among historic U.S. samples are very few in number due to the paucity of skeletal data from the middle and upper classes. Comparisons of Euro-American and African American health also are few; de la Cova’s work demonstrates that health differences among Euro-America males or African American males is not a simple question of which group had better or worse overall health. Collectively, however, these studies of 19th century cemetery and anatomical samples consistently give an overall picture of
high stress, morbidity, and mortality among disadvantaged groups, and form the basis for the expectation of similar findings among the sample used with the present study.

This review demonstrates that links between social inequality and health disparity are well established; that explanatory models considering specific physiological responses to early life stress in conjunction with social theory offer the most promise for real-world application; and that socially marginalized individuals living in the public health context of the 19th and early 20th centuries exhibit high levels of skeletal pathology. The historical literature review illustrates the many social, environmental and economic factors that would have exposed individuals to stress in utero and over their lives. Additional historical background specific to the study sample individuals is provided in Chapter 4.
CHAPTER THREE: MATERIALS

The skeletal data used in this analysis comes from a sample of individuals in the Robert J. Terry (referred to hereafter as Terry) and the Hamann-Todd anatomical collections from St. Louis, Missouri and Cleveland, Ohio, respectively. Both collections consist primarily of unclaimed human remains from the early 20th century, representing some of the most disadvantaged residents of these industrial cities. The collections, sample selection, and the cities from which the collections were assembled are discussed in this chapter.

Why Use Anatomical Collections for Bioarchaeological Research?

The purpose of this study is to investigate how health disparities can be measured and understood within the realm of bioarchaeology. More specifically, it stems from an interest in how the concepts of health and inequality are interrelated, and how, within the limitations of osteological research, we can understand the consequences of that interrelationship for individuals in the past. To conduct research in longevity, it was necessary to have a reasonable degree of certainty about
chronological age-at-death. As of this writing, there are no large, excavated, historical skeletal collections from the U.S. that are accompanied by documentation that can be matched to individual burials. The Terry and Hamann-Todd collections instead are used because the skeletal remains are complete, well preserved, and accompanied by the crucial demographic information. While the individuals in these collections never were buried, they would have been buried by public funds had they not been retained for medical school use by the state anatomy boards, making the anatomical collections comparable to historic pauper cemetery samples. Although the anatomical collections are to some degree subject to selection bias, appropriately selected samples are considered to be a reasonable representation of the impoverished subpopulation, although not of the general population, for their respective cities (Meindl et al., 1990; Hunt and Albanese, 2005).

The Hamann-Todd Osteological Collection

The Hamann-Todd collection currently is housed at the Cleveland Museum of Natural History. This collection contains the remains of over 3,500 individuals obtained from Cleveland city hospitals and the Cuyahoga County Morgue, also located in Cleveland (Quigley, 2001). The collection was begun in 1911 by Carl Hamann, Chair of the Western Reserve University Anatomy Department, and was expanded by Thomas
Wingate Todd when he assumed chairmanship of the Anatomy department in 1912 (Cobb, 1935; Cobb, 1981). Active collection ceased in 1938 upon Todd’s death.

Todd implemented a detailed cadaver intake and processing protocol that is responsible for the large amount of anthropometric, demographic, and in some cases, medical information available for the individuals in the collection. When a cadaver became available, the city undertaker notified the lab prosector on duty shortly before transfer of the cadaver; the cadaver was “tagged, photographed, measured, and embalmed…within one hour from the moment the body reaches out portals” (Cobb, 1932:39). The cadavers were accompanied by death certificates from which the prosector transcribed the “essential data” (Cobb, 1932:40) during intake. In addition, as Todd’s primary research was skeletal aging, he reviewed each individual cadaver prior to autopsy and after maceration, recording his observations regarding accuracy of the given age (Meindl et al., 1990).

The Hamann-Todd Collection is unique as it was not comprised of selected individuals, but rather included the skeletons of all individuals who were sent to the medical school’s Anatomy Lab for dissection (Todd, 1927; Cobb, 1935). Cobb noted that in 1931, the collection represented about one percent of the deaths in the city from 1911 up to that year. Most of these individuals were from “the least stable elements of marginal economic groups in the living population...people who with few exceptions were without skilled occupations” (Cobb, 1935:157). In 1931, approximately 40 percent
of the Euro-American individuals and 99 percent of the African American individuals were of native birth (Cobb, 1935:158). Cobb noted that the majority of native-born Euro-Americans in the collection, up to 1931, were born in Ohio, Pennsylvania, or New York. This demographic finding is similar to that of the present study’s Hamann-Todd Euro-American sample in which the general Great Lakes area contributed most of the individuals, and within that area, Pennsylvania and Ohio accounted for the largest percentages (Figure 1). Cobb’s (1932) analysis of the 1931 data found that in contrast to the Euro-Americans, most of the African Americans in the collection came from Georgia, Alabama, and South Carolina. The present study’s sample again has similar states of origin, with the most of the males and females originating from Southeastern states and a few from the northern states.

The Robert J. Terry Anatomical Collection

The Terry Collection consists of 1,728 complete sets of remains of individuals from the St. Louis, Missouri area. It is on permanent loan to the National Museum of Natural History’s Anthropology Department and currently is housed at the Smithsonian Institution’s Museum Support Center in Suitland, Maryland.

Anatomy Department Chair Robert J. Terry founded this collection in 1910 at the Washington University School of Medicine. The majority of individuals in the collection were obtained from St. Louis hospitals and morgues (and, to a lesser extent, other areas
of Missouri) for use in the anatomy dissection lab; from these, Terry retained some remains with the intent to create a skeletal collection representing the full range of human variation (Hunt and Albanese, 2005). Terry had developed a detailed protocol for processing and documenting the remains based on several previous attempts at establishing a collection. In addition to collecting data from death certificates, Terry corresponded with coroners, hospitals, and other institutions to obtain or confirm the demographic data for the individuals in the collection (Hunt and Albanese, 2005). As a result of the collection protocol and the documentation efforts, the Terry Collection is extremely well preserved and the strong majority of remains are accompanied by demographic and anthropometric data.

Although Terry began the collection in 1910, most of the remains that currently are in the collection date after 1920 due to replacement of damaged remains over the years (Hunt and Albanese, 2005). After Terry’s retirement in 1947, skeletons continued to be added to the collection under the direction of new department chair Mildred Trotter. Trotter focused on increasing the numbers of females and younger individuals, and during her management willed individuals were added to the collection. The willed individuals potentially are of a higher socioeconomic status than the unclaimed individuals; however, these individuals fall outside the study dates and are not included in this research.
African Americans in the Terry Collection primarily were native born. Euro-Americans are represented by both native born and foreign born. Neither Terry nor Trotter published any analyses of the states or countries of origin for the collection; however, mapping of the known states of birth for the Terry individuals in the present study sample indicate some similarities and differences from the Hamann-Todd sample (Figure 1). The Hamann-Todd African American males and females predominantly are from the southeastern states; while the Terry Collection birth states also cover the southeast, the states with the highest contribution are centered along the Mississippi river. The Euro-American males in the Terry sample show a predominantly mid-western origin centered on Missouri, while the Hamann-Todd Euro-American male sample is centered within the Great Lakes and to a lesser extent the Northeast.

Study Sample and Selection Criteria

The study sample consists of 830 adult African American males and females and Euro-American males. Euro-American females were excluded from the research due to insufficient numbers available for study. Five-hundred twelve individuals are from the Terry Collection and 318 are from the Hamann-Todd Collection (Table 1). These numbers represent all individuals in each collection that met the sample selection criteria and that were available for study at the time of data collection. The criteria consisted of: 1) birth in the United States between 1880 and 1910; 2) death no later than
Figure 1: Maps of birth states for the Terry Collection and Hamann-Todd samples.
1941; and 3) recorded age of at least 18 years, with fused femoral epiphyses.

Additionally, only complete individuals were included; individuals represented only by a cranium or by postcrania were excluded from the sample.

The study was limited to native-born individuals to reduce, as much as possible, the variation in social and environmental conditions experienced during childhood. Birth years were restricted to the period between 1880 and 1910 to reflect early childhood experienced during the general health improvements of the epidemiological transition but prior to the onset of federal child welfare programs beginning with the creation of the U.S. Children’s Bureau in 1912 (McGowan, 2005). Additionally, the birth years were restricted to maintain as much similarity as possible in general living conditions and public health conditions for these individuals, while still allowing for an adequate sample size. The year of death was capped at 1941 to reflect the period when the epidemiological transition is generally considered to have been well underway, but prior to the onset of large-scale production and availability of penicillin that began during the U.S. engagement in World War II. It should be noted, however, that antibacterial sulfonamides were in use by the late 1930s.

Age-at-death for the final sample ranged from 18 - 61 years, providing a range of individuals who fell short of, met, or exceeded the life expectancy from birth for 1900-1902: 32.5 years for African American males; 35 years for African American females;
Table 1: Sex and ancestry frequencies (n = 830)

<table>
<thead>
<tr>
<th></th>
<th>African American Males</th>
<th>Euro-American Males</th>
<th>African American Females</th>
<th>Total</th>
</tr>
</thead>
<tbody>
<tr>
<td>Terry Collection</td>
<td>294</td>
<td>72</td>
<td>146</td>
<td>512</td>
</tr>
<tr>
<td>Hamann-Todd Collection</td>
<td>193</td>
<td>40</td>
<td>85</td>
<td>318</td>
</tr>
<tr>
<td>Totals</td>
<td>487</td>
<td>112</td>
<td>231</td>
<td>830</td>
</tr>
</tbody>
</table>

and 48.2 years for Euro-American males (U.S. Department of Health, 1977:5-4). The years of death ranged from 1913 to 1935 for the Hamann-Todd sample and from 1920 to 1941 for the Terry Collection.

As emphasized previously, the individuals in these collections, with the exception of those obtained in the latter years of the Terry Collection, were unclaimed from city or county morgues, hospitals, sanitariums, and other social and health institutions. As such, these individuals were among the most vulnerable and disadvantaged members of society at the time of their deaths. For both collections, the majority of unclaimed individuals were males, a situation also common to pauper cemeteries and institutional cemeteries. As mentioned previously, Cobb (1935) noted that most of the Hamann-Todd individuals were unskilled laborers. A review of the Terry Collection database indicates that the most frequent occupation cited for males
was laborer, and for females was domestic service or housewife. To better understand the context in which these individuals lived and the resources available to them, the social, economic and public health conditions in St. Louis and Cleveland are reviewed.

**St. Louis and Cleveland in the Late 19th to Early 20th Centuries**

The study years were politically and economically tumultuous, encompassing the Spanish American War and World War I; the Spanish flu epidemic; the Great Recession of 1893-1897; the Great depression of 1929-1939; and the economic upturn during the buildup to the U.S. entry into WWII. Additionally, the study years were a period of social tension related to the rapidly changing and growing U.S. population, marked by a series of immigration exclusion acts, and the beginning of the African American Great Migration.

St. Louis and Cleveland share similar histories as major industrial cities that peaked in prosperity in the early twentieth century with a subsequent economic decline and erosion of the urban core accelerating after the Great Depression and WWII. Industrialization had brought not just booming economies, immigration, and changing ethnic make-ups but also changes in local identity. Both cities started as far Western outposts with New England affinities. By the time of the study years, Cleveland had evolved into a modern northern city, considered one of the most socially progressive in the nation (Miller and Wheeler, 1997). St. Louis, conversely, post-Civil War was a
gateway city at the junction of the North, South, and West. Although Missouri had sided with the Union during the Civil War, the state, and particularly St. Louis, was far more divided than this suggests. Complicating these processes was the pull of manifest destiny: Missouri identified more with the West than with the “backward South” (Primm, 1998:231), and a strong faction within Missouri was less concerned with the North/South divide than with protecting their financial interests in the expanding western territories (Arenson, 2011; Primm, 1998).

On the other hand, pre-Civil War Cleveland had an “egalitarian and abolitionist spirit” (Cuban, 1967:300). While both cities had Jim Crow laws, Cleveland was a less restrictive place to live, including maintaining integrated public schools since before the Civil War, and electing African Americans to public office (Cuban, 1969). In St. Louis, racial tension escalated during the early decades of the 20th century. Prior to the war, schools, restaurants, hotels, and barbershops were segregated but overt racial conflict had been minimal (Primm, 1998). In 1916, segregationists pushed through an ordinance requiring whites and African Americans to live on separate blocks, and to have separate churches, schools, and entertainment venues (Vexler, 1974; Primm, 1998). Differences between the cities in racial tension are evident in the lack of racial violence in Cleveland post-WW1 (Cuban, 1967) and the 1917 race riot, a particularly bloody and violent attack on African Americans in East St. Louis, Illinois fueled in part by social and economic strain, particularly competition for jobs (Rudwick, 1964), but also, according to
Lumpkins (2008), an expression of racial hatred and an attempt by Euro-Americans to end African American’s quest for equality.

Creating a clean water supply and controlling sewage were constant, generally unsuccessful struggles for most of the years covered by this study. Both cities were important manufacturing centers as well as water- and rail-based trade hubs. Iron, steel, and oil were major industries and employers for both cities (Schroeder, 1997; Lewis, 2004). The large numbers of factories and processing plants provided jobs but also contributed to health hazards by heavily polluting the rivers and lakes, including the groundwater and the city water sources (Cleveland Hospital Council, 1920; Schroeder, 1997). Along with the jobs from the enormous industrial growth came increased use of steam engines, which required tremendous amounts of coal to power, compounding the constant stream of coal-based industrial smoke from heavy railroad traffic, utilities, and commercial sector (Hurley, 1997; Tarr and Zimring, 1997).

Additionally, while both cities suffered from poor air quality, that of St. Louis was notoriously bad due to heavy industrial use of a particularly toxic type of coal (Tarr and Zimring, 1997). In St. Louis, smoke was pervasive – it reduced the number of hours of daily sunlight, injured vegetation, blackened buildings, discolored clothing, and deposited grit and dirt everywhere, including in people’s homes. The local bituminous coal used in St. Louis was especially hazardous to health and the environment; it contained large amounts of impurities and when burned, produced toxic ash, carbon
dioxide, and sulphur dioxide, a key ingredient in toxic rain. Coal use continued to grow with the city and the industrial sector, so that by the 1920s, St. Louis city engineers calculated that 900 tons of solids per square mile were deposited in the city by smoke (Tarr and Zimring, 1997:201). Cleaning costs and other smoke-related issues cost the city about 15 million dollars per year.

Cleveland and St. Louis launched smoke control efforts in the late 1800s but for both cities, obtaining clean air lagged behind sewage control and clean water supply by several decades. In Cleveland, public health officials described the air quality in 1920 as “…unfit for human consumption just as their sewage-polluted water was a few years ago” (Cleveland Hospital Council, 1920:85). Additionally, Cleveland public health officials identified coal smoke as political-economic health hazard, noting that:

“nothing of a practical nature is being done or even attempted in the field of smoke prevention” (Cleveland Hospital Council, 1920); the plants held by a small group of men accounted for 80 percent of the smoke in the city; and that smoke was responsible for a large amount of illness every year.

For both cities, common turn of the 20th century health hazards included impure water, inadequate sewage control, and contaminated milk and food. In addition to these stressors common to the general population, marginalized individuals experienced crowded living quarters with subsequent exposure to infectious disease as well as sociocultural stressors including racism, negative societal attitudes toward the poor,
and differential access to health care. Limited incomes would have put people at risk for malnutrition. Other stressors the study individuals likely would have experienced include long work hours and strenuous, dangerous work conditions.

Both cities experienced rapid urbanization and population growth in the latter years of the 19th century. The population increase primarily was due to foreign migration, in-migration of Euro-Americans from nearby states, and in-migration of African Americans from the southern states, many of whom were drawn by the prospect for work (Cobb, 1935). While population growth was rapid and large for both cities, that experienced by Cleveland was extreme in comparison to St. Louis. Between 1880 and 1900, Cleveland’s population increased by 138 percent, and increased by another 109 percent between 1900 and 1920 (Table 2). During the same intervals, the St.

<table>
<thead>
<tr>
<th>Year</th>
<th>Cleveland Population</th>
<th>St. Louis Population</th>
</tr>
</thead>
<tbody>
<tr>
<td>1880</td>
<td>160,146</td>
<td>350,318</td>
</tr>
<tr>
<td>1900</td>
<td>381,768</td>
<td>575,238</td>
</tr>
<tr>
<td>1920</td>
<td>796,841</td>
<td>772,897</td>
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<tr>
<td>1930</td>
<td>900,429</td>
<td>821,960</td>
</tr>
<tr>
<td>1940</td>
<td>878,366</td>
<td>818,048</td>
</tr>
</tbody>
</table>

Table 2: Population change for St. Louis and Cleveland, 1880-1940

1 Source: Grabowski, 2015 ; 2 Source: St. Louis City Plan Commission, 1969.
Louis population increased by 64 percent and 34 percent. Both cities experienced a slowdown in the population increase after 1920 and a slight decline between 1930 and 1940.

Maintaining adequate housing, infrastructure, public health, and sanitation resources to accommodate the rapid population growth was a concern for both cities, as the increased population undoubtedly placed tremendous pressure on existing resources as well as on infrastructure and private charitable services. The massive increases Cleveland experienced may have resulted in even greater stress on its poorest citizens than that experienced by St. Louis. The Cleveland Board of Health, commenting on the state of public health in the city in 1910, noted that the city’s health program expenditures "had not kept pace with those in other major U.S. cities" (Alewitz, 1997). Later, a survey of the city’s health and hospital services noted: “The children of the parochial school, the children of pre-school age, the expectant mother, all lack the life-saving service which is offered at public expense in other cities” (Cleveland Hospital Council, 1920).

Both cities opened their first public hospitals (in the modern sense) in the middle 1800s with additional private hospitals and specialized government institutions added over the decades (Vexler, 1974; Alewitz, 1997). However, racial and economic discrimination and differential access to and quality of health care occurred in both cities. Although the public hospitals provided care for individuals unable to pay, a
slightly lower standard of care was considered acceptable for charity cases (Berg, 2003). For example, St. Louis’ City Hospital spent less per patient per day than the private hospitals. City Hospital was strictly segregated with separate wards for African Americans, some of which were in the basement (Berg, 2003). In Cleveland, a hospital bed shortage in the 1920s particularly affected African American patients as hospitals within the largely African American central area were closed and patients often were denied access to available beds in other parts of the city (Giffin, 1976). St. Louis opened City Hospital II in 1919 specifically for use by African Americans but administered and staffed by Euro-American physicians (Berg, 2003). Unfortunately, the care appears to have been dismal. It was described in a newspaper account as “a crowded firetrap with intolerable conditions…so crowded it had to mix tuberculosis patients with the general population” (Berg, 2003). The hospital was plagued with both inadequate infrastructure and inadequate funding: in 1932, City Hospital I (already considered to be of a lower standard) spent $3.22 per patient per day, while City Hospital II spent $1.55 (Berg, 2003:149). Crowding was so bad that it often was necessary to push two beds together to accommodate three patients. Conditions remained poor until a new facility was completed in 1937.

In addition to limited access to health care, the poor in both cities often suffered terrible housing. In 1907, a civic league housing committee inspection found, within the poorest areas of St. Louis, general disrepair and dilapidation, with few fire escapes, few
dwellings with running water, and blocked yard sink drains forming stagnant, garbage-choked pools around the water supply (Rumbold, 1908). Ninety-two percent of the inhabitants had access only to privies, usually located in the yard, usually in appalling condition, and usually shared by multiple families (Rumbold, 1908:17). Housing was crowded; dwellings generally were apartments in subdivided houses, with entire families sometimes occupying a one-room apartment. Of particular note regarding the health implications of this housing were the yards between the main houses and the alley houses:

“Crowded somehow between the front building, the rear building, sometimes the middle building, the shed, which may hold men or horses or junk, is the row of privy vaults, the piles of manure, ashes and garbage, the frequent dead rat...the old mattresses and bedsprings, the rags and rubbish, blood and feather of fowls, and in the midst of it the hydrant with its broken half-clogged sink, which furnishes the water, sometimes for all the people in half a block...Children live and play in such yards” (Rumbold, 1908:10-18).

Thirty years later, another St. Louis health survey noted that a 15-block area of obsolete housing accounted for two-thirds of the tuberculosis deaths, half of the infant deaths, three-fourths of the illegitimate children, and two-thirds of the “delinquents” in the city (Primm, 1998:458).

Similar conditions were noted in a survey of housing for Cleveland’s poor: “A mass of shacks not fit for cattle to be kept in. Spaces between buildings from six inches to two feet wide, packed with rotting accumulations thrown in from the alleys above. Privy vaults and sink holes that are not protected against leakage of fecal accumulation
to surrounding area, accessible to children and occupants of premises” (Cleveland Hospital Council, 1920:49). Substandard and hazardous conditions were not limited to tenement buildings and other family housing, but also were found in lodging houses for single men: “The cheapest lodging houses operated in the densest areas of New York’s lower East Side shine by comparison with Cleveland’s ‘flop-houses,’ which are a disgrace” (Cleveland Hospital Council, 1920:53).

It should be noted that while notions of cleanliness and morality often intertwined to place blame on the poor for creating or perpetuating these living conditions, the attitude was not universal as can be seen in this observation from a St. Louis public health surveyor: “About 50 percent of the houses in the Negro district should be declared unfit for habitation due to extreme dilapidation. In spite of this, the rooms are often pathetically clean” (Rumbold, 1908:63). Economic discrimination in housing also was noted; surveyors found that while the housing available to African Americans was in the worst condition, African Americans paid a higher average rent than Euro-Americans (Rumbold, 1908:57).

In Cleveland, public health officials placed responsibility for the continued hazardous living conditions in the tenements on the city government, stating: “it points to an astonishing indifference on the part of the Bureau of Sanitation” (Cleveland Hospital Council, 1920:48). This quote was made after noting that enforcements and
improvements for conditions in the tenements were not made despite repeated reports by district health and sanitation workers.

In sum, while all members of Cleveland and St. Louis society were exposed to the common stressors of polluted air, unclean water, and infectious disease, the poor and marginalized members of society faced additional social and economic stressors. Limited public services, differential access to health care, potentially dangerous work, and unsafe housing combined to place the Terry Collection and Hamann-Todd individuals in harm’s way for at least the latter part of their lives. Although African Americans faced discrimination in both cities, racial tensions appear to have been greater in St. Louis.

The primary limitations for the present study’s sample are age and ancestry estimations, and sample size. As described previously, both Terry and Todd placed a high priority on obtaining accurate demographic documentation, particularly for correct age. Despite this, both collections exhibit five-year age peaks suggesting that the persons supplying the demographic information may have estimated some ages.

To mitigate the effects of this issue, the present study used only individuals with a recorded age that was considered to be reliable by Terry or Todd. Additionally, during data collection, each skeleton was reviewed for concordance with the documented age. Ancestry was documented when the body was received by the medical school and thus reflects the race concepts of the time period (Hunt and
Albanese, 2005). These concepts may have varied over time and by the individual recording or estimating the ancestry. As ancestry was assigned at the time of collection, however, it seems very possible that the race assigned in death was similar to the ways the individual was perceived and treated by society in life. For this study, each skeleton was reviewed for concordance with the documented ancestry.

The final limitation was sample size. Although African American males and females are well represented, Euro-American males are less well represented, with only 40 available from the Hamann-Todd Collection. This has created an unbalanced sample design. To mitigate this issue, data analysis methods were chosen that are not affected by, or can compensate for, this imbalance.
CHAPTER FOUR: METHODS

The data collection and analytical methods required to investigate the research questions are outlined in this chapter. The description for each osteological variable includes a brief discussion of the relevant literature justifying the use of the variable for this study.

Methodological Considerations

Bioarchaeologists commonly use skeletal lesion frequencies and metric data to compare health status among groups. In the earlier decades of paleopathological research, researchers generally considered groups with low lesions counts to be healthier than those with high lesion counts. It has been noted, however, that estimations of population health derived through cemetery sample lesion frequencies may be inaccurate due to the ‘osteological paradox’ (Wood et al., 1992): healed lesions simultaneously are evidence of past illness as well as resilience, and cemetery samples are biased toward the least healthy members of that population due to selective mortality and to differential susceptibility to illness and early death (frailty). While
methodological issues remain under discussion, it generally is agreed that careful
attention to demographic structure, appropriate quantitative methods, contextually-
grounded analyses, and use of multiple lines of evidence help to mitigate the problem
(Goodman and Martin, 2002; Wright and Yoder, 2003; Baker and Pearson, 2006; Cohen
and Crane-Kramer, 2007b; Pinhasi and Bourbou, 2008; Knudson and Stojanowski, 2009;
Buikstra, 2010; Agarwal and Glencross, 2011; Boldsen and Milner, 2012; Ortner, 2012).
By testing the relationship of skeletal markers of childhood stress to adult health
markers and to age-at-death, the present study provides information regarding the
long-term consequences of the circumstances associated with the lesion formation.
Although the exact circumstances for any given individual are unknowable, the
consequences, if patterned, will indicate whether a childhood stress marker was a sign
of poor health or of resilience for the individuals living within the social, economic, and
public health contexts specific to the study years.

Another limitation to past health interpretation is that relatively few illnesses
leave evidence in bones and teeth (Ortner, 2003). Additionally, only a small percentage
of individuals experiencing an infectious disease will have a skeletal manifestation of
that disease. For example, skeletal lesions occur in only about five to seven percent of
individuals with tuberculosis (Aufderheide and Rodriguez-Martin, 1998); three to five
percent of individuals with leprosy (Ortner, 2003); and one to five percent of
individuals with treponemal disease. On the other hand, bones and teeth are sensitive
to systemic stress, with stress manifesting as growth disruption or by lesions indicating an infectious or immunocompromised state. For these reasons this research uses osteological stress markers rather than specific diagnoses to interpret health.

Another limiting factor for the present study is the probable homogeneity in socioeconomic status for the sample individuals. Ideally, a study concerned with health disparities would compare individuals from different socioeconomic strata, but the lack of sufficient numbers of high status individuals from this time period precludes that approach. Instead, this research looks for sex, ancestry, and regional differences in health.

The data required to test the project hypotheses consist of skeletal and dental lesions, postcranial metrics, demographic data, and cause of death data. The osteological variables were chosen to reflect stress and health conditions experienced over the lifecourse. To that end, three markers reflecting childhood developmental stress and three markers reflecting later life health experience were chosen. While the latter are intended to represent adult health experiences, they are used with the understanding that they overlap with later childhood years and thus reflect a cumulative health experience.

The osteological methods used required only macroscopic analysis or low-level magnification and standard osteometric tools. Except where indicated, all osteological
markers were recorded following the protocols in *Standards for Data Collection from Human Skeletal Remains* (Buikstra and Ubelaker, 1994).

**Osteological Methods: Childhood Developmental Stress**

**Linear Enamel Hypoplasia**

Linear enamel hypoplasias (LEH) are macroscopic horizontal defects in tooth crown enamel reflecting stress episodes that occurred during crown formation in childhood (Schultz et al., 1998)(Figure 2). LEH are particularly useful for evaluating childhood health experiences in the adult skeleton because, outside of crown loss, wear, or damage, they are permanent. Additionally, evidence suggests that tooth enamel formation is more sensitive to disruption than bone mineralization (May et al., 1993). LEH are referred to as ‘nonspecific’ because, despite multiple causes for the defects, the exact cause cannot be attributed to a particular lesion (Goodman and Martin, 2002:24).

Permanent tooth crowns begin forming during the first year of life after birth, with deposition of the enamel matrix beginning at the occlusal surface and progressing toward the cementoenamel junction (Larsen, 1997). Enamel deposition may slow or stop in response to a variety of causes, including malnutrition, major infections, fevers, local
Figure 2: Linear enamel hypoplasia of the mandibular teeth.

Photo credit: Walter Larrimore.
trauma, and hereditary anomalies (Larsen, 1997; Hillson, 2008). The disruption may appear as pits, spots of hypocalcification, or lines. The latter of these, varying in size from fine lines to wider furrows, usually are referred to as linear enamel hypoplasia and are the most commonly employed hypoplastic tooth defect in bioarchaeological studies of health.

LEH has been associated with malnutrition among living populations, for example in Mexico (Goodman et al., 1987), China (Zhou and Corruccini, 1998), and Brazil (Santos and Coimbra, 1999); and with social inequality in archaeological populations (e.g. Martin et al., 1984; Palubeckaitė et al., 2002; Miszkiewicz, 2015). LEH also have been associated with early mortality in archeological populations in North American (e.g. Cook and Buikstra, 1979; Goodman and Armelagos, 1989; Duray, 1996), Europe (e.g. Palubeckaitė et al., 2002; Boldsen, 2007), and Japan (e.g Temple, 2014) but the association is not consistent. For example, LEH was not associated with early mortality in a 19th century Canadian sample (Saunders, 1999) and varied over time and location within the prehistoric Illinois River Valley (Wilson, 2014). On an aggregate level, two or more LEH was associated with a 7.6 percent decreased chance for survival for North and South American populations spanning approximately 6000 years (Steckel, 2005).

LEH show no clear patterns of sex differences, with some groups showing higher frequency for males (e.g. Saunders, 1999), some for females (e.g. King et al.,
2005), or no differences (e.g. Lanphear, 1990; Slaus, 2008). For example, among an age-
documented 19th-20th century Lisbon sample, LEH was associated with early mortality
for males but the association became nonsignificant when controlled for socioeconomic
status (Amoroso et al., 2014). Younger age-at-death with LEH was found for Late
Antique to Early Medieval Croatian females with LEH but not for males e.g. (Slaus,
2008). These conflicting results indicate that LEH can be associated with early mortality
but expression of that association is highly context-specific.

The anterior teeth are considered best for LEH evaluation, in large part because
they are the most susceptible to disruption (Goodman and Rose, 1991). Goodman and
colleagues (1980) advocate a best-tooth analysis approach using two maxillary incisors
and two mandibular canines, while other studies successfully have relied on a single
tooth type (e.g. Boldsen, 2007). For the present research, the intent was to evaluate all
maxillary and mandibular anterior teeth to determine the maximum number of shared
LEH between at least two nonadjacent teeth. During exploratory data analysis,
however, it became apparent that these criteria resulted in high numbers of missing
values due to antemortem tooth loss as well as to postmortem loss and breakage
(discussed in Data Preparation). For this reason, analysis was limited to the maxillary
and mandibular canines, with the maximum number of LEH on any canine serving as
the variable. These criteria will reflect stress occurring from approximately the age of
six months to seven years (White and Folkens, 2005:367).
All anterior teeth were examined under a low level magnifying lamp. LEH were scored as present if a visible line was palpable by fingernail, and the maximum number of lines occurring per tooth was recorded. The study variable subsequently was coded as 0 (no LEH), 1 (one LEH), and 2 (two or more LEH).

Vertebral Neural Canal Size

The dimensions of the vertebral neural canals (VNC) are very sensitive to growth disruption from systemic stress and thus are useful markers of childhood health (Clark et al., 1986; Clark, 1988). The vertebral neural canal refers to the portion of the vertebrae composed of the posterior centrum and neural arches through which the spinal cord passes. Neural canals typically have a buffer of about one to three millimeters between the bone and the spinal cord (Tatarek, 2005). In cases of stunting, this space is diminished, increasing risk for local spinal insult.

The neural arches begin developing in utero, attaining 65 to 70 percent of their adult size at term (Clark et al., 1986; Larsen, 1997). At around four to five years of age, the neural arches fuse to the centrum, starting in the thoracic vertebrae and finishing in the lumbar vertebrae (Scheuer and Black, 2000:195-196), at which point growth in the anterior-posterior dimension is essentially complete (Clark, 1986). The transverse dimension continues to grow into the teens. Recently, Watts (2013b:7) found that the anterior-posterior dimension of the lumbar vertebrae stabilized at three to five years of
age while the transverse dimension increased until about age 15 to 17 in a British archaeological sample. Other researchers have found, however, that the L1 matures earlier than L2-L5, reaching full size by ten years of age (Papp et al., 1994).

VNC are useful indicators for health comparison because, unlike the long bones, their relatively short and rapid growth period renders them less prone to catch-up growth in the event of improved environmental circumstances. Importantly, studies have demonstrated that VNC stunting is independent of overall vertebral size and thus is not merely a reflection of dimorphism or body size differences (Watts, 2013a:96). Stunted VNC have been linked with early adult age-at-death in prehistoric and historic skeletal populations (Boldsen, 1998; Tatarek, 1999; Watts, 2011; Clark et al., 1986) and with increased adult morbidity in living populations (Porter, 1998).

VNC are not included in Standards, so this project utilizes the methods described in Clark (1986) and followed by Boldsen (1998), Tatarek (1999), and Watts (2011, 2013). The anterior-posterior and transverse canal diameters of the first thoracic and first lumbar vertebrae were measured to the nearest .01 mm using Mitutoyo Digimax digital calipers (Figure 3).

To reduce intra-observer error, each dimension was measured three times and the mean of each dimension was calculated and rounded to the nearest 0.1 mm. After Watts (2011, 2013), measurements were not taken on individuals with supernumerary vertebrae, scoliosis, or spina bifida as it is unknown how these conditions may affect the
canal size. Additionally, measurements were not taken on transitional vertebrae (e.g., lumbar vertebrae lacking transverse processes and exhibiting ribs facets) and those with fractures, lytic lesions, or unfused neural arches. Other vertebrae were unmeasurable due to postmortem damage or loss, or to enlarged foramina and/or spicules on the posterior vertebral body that interfered with correct caliper placement. These inclusion criteria resulted in large numbers of missing values. The anterior-posterior lumbar dimension was the most heavily affected with 20 percent of the sample missing, and was excluded from the study (discussed in Data Preparation). The first thoracic transverse diameter also was removed from the study due to a significant result on intra-observer error analysis. The other two vertebral dimensions were retained.

The quantitative analysis methods for this study required categorical data, so the measurements were categorized 1 for small (one or more standard deviations less than the mean); 2 for average (-0.99 to 0.99 standard deviations from the mean); and 3 for large (one or more standard deviations greater than the mean). As no significant differences were found between the collections, the means were based on pooled data from groups comprised of the same sex and ancestry.
Figure 3: Vertebral neural canal diameters. A: anterior-posterior diameter. B: transverse diameter. Photo credit: Walter Larrimore.
**Femoral Length**

Femoral length serves as a cumulative measure of stress incurred during childhood (Wright and Yoder, 2003). Lower extremity long bone lengths may be used as a proxy for stature (Trotter and Gleser, 1951; Jantz and Jantz, 1999); therefore, rather than calculating stature from long bone lengths, this project instead uses femoral length as a variable. Although stature estimation would work very well on these individuals – particularly as the formulas that would be used were developed in large part on the Terry Collection – one of the intents of this research was to provide comparative material for bioarchaeological studies of health. By using femoral length, the study results will provide a more comparable resource for researchers whose archaeological samples may lack an appropriate stature estimation formula.

Terminal adult stature results from the interaction of an individual’s genetic potential and the environmental conditions experienced during growth (Kemkes-Grottenthaler, 2005; Mielke et al., 2006). The relationship between adverse childhood circumstances (e.g. malnutrition, illness) and reduced terminal stature has been well documented in living and archaeological populations (Larsen, 1997; Goodman and Martin, 2002). Reduced stature is widely demonstrated to be associated with decreased longevity through analysis of archaeological populations and of historical anthropometric data (Kemkes-Grottenthaler, 2005; Steckel, 2008).
In the event of improved circumstances later in the growth period, individuals experiencing growth suppression during early childhood still may achieve full potential height through catch-up growth (Goodman and Martin, 2002). Human growth trajectories are “self-stabilizing” (Meindl and Russel, 1998:379) with subsequent catch-up rates as high as three times the normal growth rate after temporary disruptions. Catch-up ability may be overwhelmed, however, in the event of prolonged malnutrition, illness, or other stress (Meindl and Russell, 1998). If catch-up growth occurred for the study sample individuals, earlier childhood stress still may be detected by the presence of LEH or stunted VNC.

A potentially limiting factor for the utility of femoral length for this study was secular change, i.e., change over time due to a combination of environmental factors and genetics. Secular change in American stature and long bone length has been documented to occur within the period of the study years; some of this research has included individuals from the Terry Collection (Trotter and Gleser, 1951; Jantz and Jantz, 1999). In a pooled sample of Terry and Huntington Collection individuals, WWII casualties, and the Forensic Data Bank, all groups (male/female/African American/European American) showed change in stature from the mid-1800s to 1970 (Jantz and Jantz, 1999). To determine whether the study sample exhibited secular change in femoral length, the measured femoral lengths were regressed on year of birth;
a small positive effect was found for males, and these results are described further in Data Preparation.

The maximum length of both femora was measured to the nearest millimeter by standard osteometric board, with the left femoral measurement used as the study variable. Measurements were not taken on fractured femora or those with postmortem damage or pathological change affecting the articular surfaces. When the left femur was absent or unmeasurable, it was replaced with the right femoral measurement. The measurements were categorized 1 for small (one or more standard deviations less than the mean); 2 for average (-0.99 to 0.99 standard deviations from the mean); and 3 for large (one or more standard deviations greater than the mean). As no significant differences were found between the collections, the means were based on pooled data from groups comprised of the same sex and ancestry groups.

Osteological Methods: Adult Health

Diseased, Missing, and Filled Teeth Index

This research uses dental caries and antemortem tooth loss (AMTL) as a measure of later-life stress although it should be noted that the permanent teeth begin erupting around age six with the appearance of the first molar. Caries and AMTL reflect dietary composition and nutritional status and serve as general indicators of immunological
status (Larsen, 1997; Goodman and Martin, 2002). Both tend to increase with chronological age, complicating comparison of raw data counts between groups of differing age structures.

Figure 4 shows a caries, an amalgam filling, and AMTL co-occurring in a mandible. Caries refers to areas of demineralized tooth enamel and dentine caused by contact with by-products of carbohydrate fermentation (Larsen, 1997). Clinical studies have demonstrated caries to be linked to poor health and increased risk of death in living populations (Garcia et al., 1998; Jansson et al., 2002). Increased caries rates often are cited in bioarchaeological research focusing on changes in health related to changes in subsistence (e.g., Cohen and Armelagos, 1984.; Cohen and Crane-Kramer, 2007a). Clinical evidence indicates that malnutrition, undernutrition, and fasting promote caries formation and increase caries rates by changing oral ecology (Lukacs, 2012).

Pregnancy and malnutrition contribute to differential age and sex prevalence by altering saliva flow rate and composition. Additionally, variation in enamel protein gene loci may contribute to variations in cariogenesis by altered enamel structure (Lukacs, 2012). Comparing the caries burden among populations is complicated by the different methodologies researchers use for determining caries presence or absence (Liebe-Harkort et al., 2009). In their initial stages, caries present as white or brown spots
Figure 4: Mandible with caries, filling, and AMTL. A: caries; B: amalgam filling; C. Photo credit: Walter Larrimore.
in the enamel, later penetrating the enamel in sizes ranging from a small spot up to complete destruction of the tooth. Some researchers include the presence of these brown or white spots in their counts, while others require perforation of the enamel.

Additionally, it is important to consider the relationship between caries and antemortem tooth loss when attempting to determine their frequencies. Disregarding AMTL when determining caries prevalence means that some caries experience would not be accounted for, potentially causing caries underenumeration in a survey. Conversely, assuming that teeth are lost only because of caries potentially overenumerates the caries count. Teeth may be lost prior to death for many reasons including trauma, caries, and periodontal disease, and the reason for a loss cannot always be determined (Hillson, 2001). Regardless of the specific etiology, AMTL is a useful general indicator of adult health related to diet. Tooth loss may affect an individual’s nutritional status by limiting the ability to chew and by reducing food options (Goodman and Martin, 2002). Clinical studies have associated AMTL with increased morbidity in living populations (Jansson et al., 2001; Jansson et al., 2002; Padilha et al., 2008).

Studies utilizing caries and AMTL often conduct analysis at the population level, not the individual level, expressing rates as a percentage of the total number of teeth or resorbed sockets in the population, or as the percentage of individuals affected (Hillson, 2001). The present study’s lifecourse approach required a method allowing
caries and AMTL experience to be calculated at the individual level. To summarize oral health, I used the Decayed, Missing, and Filled Tooth Index (DMF-T) (Hillson, 2001). The DMF-T presents an overall state of the teeth in use at the time of death by considering the number of unhealthy teeth in relation to the number of teeth at risk.

The DMF-T and variants were developed, and continue to be used, for use in dental practice and dental epidemiology (Waldron, 2007). Variants of the DMF-T omitting the ‘filled’ category have been adopted for use in bioarchaeological studies (Moore and Corbett, 1971; Powell, 1991; Saunders et al., 1997; Caselitz, 1998). The DMF-T was useful for the present study because in addition to serving as a health marker, it also has functional implications. A high score may indicate a higher risk for inadequate micronutrient and caloric intake due to pain and to a reduced ability to chew, potentially exacerbating any underlying illnesses an individual experienced.

The DMF-T index is the ratio of the number of unhealthy teeth to the total number of tooth positions X 100. The numerator is the sum of carious teeth, filled teeth, and teeth lost antemortem. The denominator is sum of the numbers of extant teeth, teeth lost antemortem, and teeth lost postmortem. To maintain a consistent number of teeth for comparison across samples, some clinical epidemiological studies exclude the third molars in their surveys of permanent teeth. The present study, however, includes the third molars because it was not the actual rates of tooth disease but rather the overall susceptibility of an individuals’ dentition to disease that was of interest.
Excluding third molars can underestimate the index; in a comparative study of modern military dental data, Adams (2002) found that excluding third molars resulted in significantly lower DMF-T scores. Additionally, including third molars makes this research consistent with modern population research following the current World Health Organization (2013) recommendations to include all 32 permanent teeth.

For the present study, the maxillary and mandibular dentition was inventoried per Standards. Caries, fillings and antemortem loss were counted as present or absent for each tooth; severity was not scored. A tooth simultaneously exhibiting caries and a filling was counted only once, in the caries category, according to the WHO (2013) practice. The teeth and empty sockets were examined under a lighted, low-level magnifier and the assessment was aided by use of a dental probe as needed. Interproximal tooth surfaces were observed where possible but the ability to see these surfaces varied with tooth spacing. Staining and postmortem damage to many of the teeth precluded use of the earlier stages of caries formation, so crown caries were defined as dark-stained, irregular perforations of the enamel (Steckel et al., 2006; Buikstra and Ubelaker, 1994) corresponding at a minimum to Hillson’s (2005:298) caries score three. Root caries were counted as present or absent when, at a minimum, they presented as stained or unstained shallow cavities on the root or following the cement-enamel junction, corresponding to Hillson’s root caries score 5. Antemortem loss was identified by the presence of alveolar resorption. Empty sockets lacking signs of
resorption were counted as post mortem loss. Unerupted teeth or teeth not in occlusion (e.g. impacted third molars) were noted but were not included in the analysis.

Teeth with less than 50 percent visibility of the crown due to damage, calculus, or wear were considered unobservable. Additionally, individuals were excluded from the analysis if their combined postmortem loss and unobservable teeth exceeded 25 percent of the number of their total tooth positions. Several individuals had gold capped teeth with healthy appearing tooth roots; as these teeth were still present and the reason for the caps unknown, the teeth were scored as present but unobservable for pathology. The calculated DMF-T indices were used for comparison of group means. For testing the relationship between DMFT and child stress, DMFT was dichotomized using group-specific means: 0 (less than group mean) and 1 (equal to or exceeds group mean).

Tibial Periosteal Lesions

Bone is under a constant state of remodeling even after epiphyseal fusion marks the cessation of the more marked growth associated with childhood and adolescence. In the adult stage, the rate of remodeling slows but continues to add new bone at the periosteal surface and to resorb bone from the endosteum (i.e., the marrow cavity). In the event of an insult to the bone that causes inflammation or that causes the periosteum (the membrane covering the outer surface of bone) to lift from the bone
surface, the periosteum responds by forming a layer of bone at the site (Ortner, 2003; Weston, 2012). Thus, in contrast to the normal remodeling process of healthy bone, this process is considered pathological.

Etiologies are many, including trauma, local and systemic infections, vascular disease, venous stasis, metabolic conditions, and nutritional deficiencies (Cook, 2007; Weston, 2012); in cases where a specific etiology cannot be identified, however, the lesions are referred to as non-specific stress. Periosteal lesions begin as loosely organized woven bone on the bone surface; as it heals, the new bone formation becomes striated and more compact (Figure 5). Eventually, if the individual survives and remodeling continues, the lesion becomes smoother and denser, appearing as a raised or undulating area on the bone cortex.

Periosteal lesions are one of the most commonly occurring pathological conditions found within archaeological assemblages, and of the long bones, the tibia is a frequent site for the lesions to occur. The sensitivity of the tibia to lesion development is not fully understood, but likely is related to minimal overlying soft tissue, low vascularity, and the preference of some bacteria for cooler areas of bone (Ortner, 2003; Klaus, 2014). Tibial lesions frequently are used to compare health within and among populations, and also have been used to demonstrate change in health with change in subsistence (e.g. Goodman et al., 1984a; Larsen et. al, 2007). Generally speaking,
Figure 5: Healed tibial periosteal new bone formation.
Photo credit: Walter Larrimore.
periosteal lesions tend to increase in prevalence as environmental stress increases (Larsen, 1997).

The present study was concerned only with lesions representing systemic processes. Traumatic lesions can be distinguished from lesions representing systemic processes in several ways. First, periosteal lesions resulting from systemic conditions tend to be bilateral or involve multiple long bones, whereas periosteal lesions resulting from trauma are more likely to be unilateral. Additionally, traumatic lesions tend to be small, localized, and non-destructive (Ortner and Putschar, 1981).

Both tibiae were examined for the presence of periosteal lesions. Side, section, aspect, extent, and healing status of the lesions were recorded following Standards, and additionally, the lesions were assigned an aggregate score following the Global History of Health protocol for osteoperiostitis (Steckel et al., 2006); however, the present study used only the presence or absence of bilateral activity.

Tibial lesions were scored as “present” if at least one discrete patch of healed or active was present on both diaphyses. Bone formation at muscle attachment sites was not included, as this more likely reflects mechanical activity (Ortner, 2003). The research protocol required that each tibia had a minimum of 75 percent of the diaphysis present and 75 percent intact cortex, but postmortem damage to the diaphysis was minimal within the collections. Several individuals had only one tibia available for evaluation because of amputation or because the tibia was missing from the box. Of these cases,
individuals with only one tibia were included in the analysis if the available tibia was free of lesions, as this individual would be scored as ‘condition absent’ regardless of the activity on the other tibia. Tibiae with lesions that lacked the antimere for comparison were scored as unobservable.

**Skeletal Fractures**

Fractures are among the most commonly recorded traumatic lesions in archaeological skeletal assemblages. A bone’s susceptibility to fracture depends on its density, strength, and capacity to absorb energy, as well as the rate, direction, magnitude and duration of the applied force (Galloway, 1999). Whether accidental or intentional (i.e., interpersonal violence or self-inflicted) in origin, fractures provide insight into lifestyle, activity, environmental hazards, and violence. Antemortem skeletal fractures are included in this research because they may have significant consequences for the individual including impaired mobility or range of motion, bone infection, and secondary arthritis (Figure 6). Additionally, intrauterine stress may increase fracture risk by affecting peak bone mass (Cooper et al., 2006; Tandon et al., 2012).

The determination of whether or not a fracture occurred antemortem was based on evidence of healing in the dry bone. In brief, the healing process begins immediately upon injury with hematoma formation (Ortner, 2003). A callus begins to form within
and around the break about one week after the injury, and then begins to mineralize into woven bone about a week after it is formed (Galloway, 1999; Ortner, 2003). The callus mineralization stage continues for another four weeks to four months, depending on the type of bone and fracture characteristics (Galloway, 1999). The callus gradually remodels over the next several years, losing the woven appearance and consolidating into laminar bone, and eventually returning to its original form, depending on the severity, degree of displacement, and infection (Lovell, 1997; Galloway, 1999).

Fractures become visible on x-ray within ten to 14 days of injury (Galloway, 1999: 15) when resorption causes the necrotic bone in the break margins to look blurred. In practical terms of bioarchaeological assessment at the macroscopic level, fracture healing becomes detectable when the mineralizing callus bridges the fracture, at about three weeks after initial injury (Lovell, 1997:145). In contrast, perimortem fractures lack any evidence of healing, and so, in terms of dry bone assessment, perimortem refers to those fractures that occurred at the time of death or up to about three weeks prior. The perimortem interval also includes fractures that occurred after death while the bone is still “fresh” (Lovell, 1997: 145). Postmortem fractures occur in the context of deteriorating organic components, causing the bone to react differently to force than does fresh bone. Break characteristics indicating a postmortem occurrence are differential coloration of the break margins compared to the surrounding bone, smaller
Figure 6: Healed tibia and fibula fractures.
Photo credit: Walter Larrimore.
fragments, squared edges, and in the case of moisture-logged bone, crumbling (Galloway, 1999).

Childhood fractures were excluded from the study for several reasons. First, the present study was using antemortem fractures as an indicator of stress experienced after the childhood years. Second, depending on the age-at-death, evidence of fractures incurred in childhood may be obliterated by remodeling by the time of adult death (Glencross, 2011). Third, child fractures are found much less often than adult fractures, consistent with research indicating that within developing countries, children’s contribution to overall trauma rates was mostly minor injuries, while individuals in the “economically active” (Judd, 2004:35) ages of 20 to 40 years sustained the most injuries of all groups.

Although it can be difficult to estimate the timing of a well-healed fracture, the extent of remodeling, the fracture location, and alteration in bone shape in many cases aid in distinguishing those incurred in childhood from those incurred later (Glencross and Stuart-Macadam, 2000). Children’s bones are more cartilaginous, thinner, and more porous than those of adults, and thus are thus less likely to incur a complete fracture (Glencross and Stuart-Macadam, 2000). The most common forms of fracture in children are incomplete: greenstick, which is a fracture occurring only on one side of the bone; torus, which is buckling of the bone usually at the epiphyseal-metaphyseal junction; and plastic deformation, which causes an unusual bowing of the bone without an actual
fracture line (Lewis, 2007). In addition, certain other fractures particularly suspicious for childhood occurrence are supracondylar fractures of the distal humerus (Glencross, 2011) and fractures of the clavicle shaft (Glencross and Stuart-Macadam, 2000).

The cranium, mandible, clavicles, innominates, and all arm and leg long bones were evaluated for fractures. The approach to fracture count in this study should be considered conservative. Radiographs were precluded because of time and financial constraints. Only those elements in which fractures confidently could be diagnosed were included. Antemortem long bone, innominate, and clavicle fracture presence was evaluated by presence of any woven or remodeled callus, angular or rotational deformity, and antimere asymmetry (Lovell, 1997; Ortner, 2003). Cranial fractures were identified by depression of the external vault, plastic deformation, or lines radiating from an impact point or occurring concentrically (Figure 7) (Slaus et al., 2012).

Depressed fractures of the cranium were distinguished from soft-tissue or infectious lesions after Walker (Walker, 1989:313): absence of reactive bone; a well-delineated circular, ellipsoidal, or patterned shape; and a lack of multiple lesions on the cranium or elsewhere in the skeleton. Avulsion fractures of muscle attachments were not included as small examples of these can be difficult to discern from other activity at the site. Fractures likely to have occurred during childhood based on the previously described characteristics were noted but not included in the analysis. Limb amputations were
Figure 7: Fractured occipital with slight depression and concentric and radiating linear fractures. Photo credit: Walter Larrimore.
considered a form of fracture and included in the counts, as was sharp-force trauma to the cranium.

There are various approaches to quantifying fractures on an individual level, including counting injuries by body segment (i.e. torso, leg), by individual elements, or by injury. The present study questions whether adult fractures were linked with childhood stress; that is, were individuals with childhood stress more prone to injury later in life? To that end, the variable used for this study was the total number of fractures per person, rather than the number of fractured individual elements. Fractures involving paired elements that reasonably can be expected to have resulted from a single contact with force were counted as a single occurrence; for example, the tibia and fibula sometimes fracture together, as do the radius and ulna. Nasals that were contiguous with the adjacent portion of the maxilla were considered a single injury. An element was considered observable only when at least 75 percent complete. Individuals missing more than 25 percent of the cranium or 25 percent of the postcranial elements were excluded from the analysis.

**Mortality Data**

This study uses mortality data to examine whether the sample exhibits a pattern of decreasing proportion of deaths from infectious to degenerative disease over time (i.e., epidemiological transition), and whether the patterns were similar to those of the
contemporaneous local and national populations. Mortality data were obtained from a variety of sources. For the Terry Collection individuals, cause of death was obtained by inspecting scans of the original death certificates, available online as described previously in Chapter 3. For the Hamann-Todd sample, the cause of death was obtained from the collections manager; the cause of death for each individual was transcribed into lab records from the death certificate when the body arrived at Todd’s lab, as described previously in Chapter 3. St. Louis and Cleveland city-wide mortality data were obtained from annual reports of their respective local health departments, some copies of which were available in digital form, and others available on microform or original hardcopy at the National Library of Medicine in Bethesda, MD, and the New York Public Library in New York City. The national data were obtained from digital editions of the death-registration area annual mortality statistics reports published by the U.S. Department of Commerce.

Historic data sources have some limitations that affect research design as well as interpretation. For example, not all individual deaths may have been reported or recorded on the local or national level, and diseases may be classified differently by different examiners due to variation in knowledge and experience of the coroner or physician assigning the cause of death. Mortality trends over time may be affected by changing autopsy rates and use of biomedical technology (Armstrong et al., 1999).
Other important considerations for this research are the expansion of the death registration area from 26 states plus the District of Columbia in 1916, to all states in 1933, and the revision of the *International Classifications of Diseases* (ICD) over the study years (Moriyama et al., 2011). The ICD is a diagnostic and classification tool in use in the U.S. and several other countries since 1900. The mortality data used for this research cover three versions of the ICD, from ICD-2 (years 1910-1920) through ICD-4 (years 1930-1938). Changes in diagnostic criteria and coding over the revisions make some specific diagnoses difficult to follow. To overcome this problem, the present study uses broad categories to demonstrate change over time, as this method has been demonstrated to be effective despite some overlap in categories (Dunn and Shackley, 1944; Alter and Carmichael, 1996). Additionally, cause of death comparisons were stopped at 1937 to minimize the number of ICD revisions affecting the study.

One challenge with broadly categorizing the historical data is inconsistency among various authors as to what constitutes degenerative or infectious due to some overlap in categories. For example, Weisensee (2013), to acknowledge that some forms of cancer are infectious in origin, separates neoplastic disease from degenerative diseases while Gage (2005) combines these categories. I chose to follow Gage by including neoplasms with degenerative disease. I followed Armstrong and colleagues’ (1999) study of historic infectious disease mortality by placing any systemic or local condition that usually is infectious in origin into the infectious category except for the
diagnosis of chronic myocarditis, which was a common diagnosis for the collections individuals in this study. Although early use of the term tended to refer to any disease of the myocardium, by the early 1900s a clinical distinction was made between myocardial damage caused by vascular occlusion from that of other causes; after WWI, this increasingly was understood to be infectious. Silber (1979:3) notes that during the 1920s and 1930s, the term had fallen into such disfavor that physicians rarely used the term except in the presence of diphtheria or rheumatism. However, the chronic myocarditis category is absent from the St. Louis publications and some of the Cleveland publications, indicating this diagnosis as well as some other cardiac conditions possibly were collapsed into the “other diseases of the heart” classification. There is no way to discern the contribution of infectious chronic myocarditis to that category, so it was necessary to include chronic myocarditis in the degenerative category, following Gage (2005). This may result in a slight over-representation of degenerative disease relative to infectious disease, but as this protocol was applied to all groups and localities in the study, the comparison of the degenerative to infectious ratios among the groups was not affected. In other words, the degenerative to infectious ratios may be slightly inflated, but if so, they are inflated across the board.

As described previously, the cause of death for each individual from the collections sample was categorized as degenerative, infectious, and ‘other’ (Table 3). The Terry Collection and Hamann-Todd data are aggregates of five-year intervals
represented by the middle year; this approach was necessary due to the sometimes small numbers of individuals, particularly Euro-American males and African American females, entered into the collections in any given year. For the St. Louis, Cleveland, and national populations, the annual crude death counts for each diagnosis were categorized separately for African American males and females, and Euro-American males. It is important to note that the city and national data include some child deaths.

### Table 3: Disease classification system used for the study

<table>
<thead>
<tr>
<th>Classification</th>
<th>Examples</th>
</tr>
</thead>
<tbody>
<tr>
<td>Infectious</td>
<td>Tuberculosis (all forms), pneumonia, influenza, bronchitis, syphilis (all stages), meningitis, typhoid, malaria, septicemia, abscesses, gangrene, endocarditis, pericarditis, appendicitis, diarrhea, mastoiditis</td>
</tr>
<tr>
<td>Degenerative and chronic</td>
<td>Chronic myocarditis¹, coronary thrombosis, hypertension, mitral insufficiency and all other non-infectious valve conditions, all other non-infectious cardiac and vessel diseases, diabetes, chronic nephritis, renal failure, Parkinson’s disease, emphysema, stroke, all cancers</td>
</tr>
<tr>
<td>Other</td>
<td>Homicide (gunshots wounds, stabbings), suicide (hanging, drowning), accidental trauma (falls, drowning, industrial accidents), strangulated hernia, intestinal obstruction, alcoholism, morphine addiction, oxalic acid poisoning, epilepsy, pellagra, malnutrition, puerperal fever and all other pregnancy and childbirth related conditions</td>
</tr>
</tbody>
</table>

¹ See text for inclusion rationale

The publications varied by the type of information available. The National Department of Commerce publications specified numbers of deaths by sex, ancestry, and age, but none of the St. Louis data and only some of the Cleveland data had this
level of specificity. For some but not all years, the cities and national publications had in common the causes of death broken specified by sex and ancestry. To make comparisons among these locations and to maintain consistency across the study, it was necessary to use the all-ages data with diagnostic classes specific to infants and children excluded. The years tabulated and analyzed were chosen based on the limited availability of surviving copies of the city publications as well as by the years that included the above data specifications. Cleveland in particular has a short range of dates as their Department of Health stopped annual mortality data publication for the years 1916-1923.

The variable used for this analysis was the ratio of degenerative disease to infectious disease (D:I), expressed as a percent based on the crude death counts. The ratios then were graphed to observe for trends over time. This approach was taken instead of quantitative analysis due to the diagnostic issues and other challenges with the historical data outlined above. The approach taken for the present study was internally consistent, but the numerical results of this analysis should not be used for direct comparison with other studies.

Data Preparation

This section describes the data preparation and the analytical methods used to test the study hypotheses. All analyses were conducted in SPSS 22 (IBM, 2013).
Intra-observer Error

Intra-observer error analysis followed the test-retest method. The error sample consisted of a total of 62 crania and 52 sets of postcrania from the Hamann-Todd and Terry Collections. Each cranium or postcrania was scored twice following the original data collection protocol under similar conditions, with the re-test intervals ranging from one to 20 weeks. The data were examined for normality of distribution with the Shapiro-Wilk test and linearity assumptions for the correlation variables were examined by scatterplot; all assumptions were met. The measured scale data (femoral length and vertebral neural canal dimensions) were evaluated with paired t-tests; the frequency data (maximum canine hypoplasia, decayed/filled teeth, antemortem tooth loss, cranial fractures, facial fractures, postcranial fractures) were evaluated with Spearman’s rho; and the periosteal lesions were valuated with Cohen’s kappa.

The paired t-tests have the advantage of providing a correlation coefficient as well as a test of the means. With the exception of T1TR, all scale variables demonstrated strong correlation between the original and repeated measures ($r = 0.796$ to $0.998$, $p < .01$), and all lacked significant difference between the means ($p \geq 0.175$). These results indicate strong support for the reliability of the test-retest data, with the exception of the...
T1TR score. The T1TR scores were strongly correlated \( r = 0.998 \) and had a mean difference only 0.04 mm, but this amount of difference was enough to be significant \( t = 2.28, df = 43, p = 0.027 \). Due to the significant difference in means, I removed T1TR from the study.

Except for the tibial lesions, the test-retest values for the categorical variables all were strongly, positively correlated, with Spearman’s rho ranging from 0.738 to 0.996, and all variables were retained. Cohen’s kappa = 0.653 for the tibial periosteal lesion test-retest data. Although this score was considered good agreement (Altman, 1991) it was preferable to have a score greater than 0.7. Review of the raw data indicated that most of the disagreement between the original and retest observations occurred with a GHHP periosteal lesion score of two (Steckel et al., 2006), which is the minimum expression of periosteal activity. I repeated the Cohen’s kappa using only tibiae with a minimum lesion score of three with a resulting kappa of 0.834, which was considered very good (Altman, 1991). Based on these results, I set the minimum GHHP score at three for the condition to be considered “present” in the subsequent hypothesis testing and modeling. With the exceptions noted above, my repeat observations were strongly and significantly associated with my original observations, indicating that my observations were consistent through the data collection.
Missing Values Analysis

Upon completion of the error study, the primary dataset was inspected for missing values. Several variables showed high levels of missing data (Table 4). The vertebral neural canal dimensions had the highest numbers of missing values. There is disagreement on what percentage of missing values is too high, with opinions varying between five and 20 percent (Dzuria et al., 2013). L1AP had 20 percent total missing, and the number missing from the other neural canal dimensions ranged from 13 to 15 percent. As many of the quantitative measures used in this research require complete data sets, it was necessary to find a means to account for the missing values. The options were limited to deleting the cases with missing variables during analysis, discarding the affected variables from the study, or replacing the missing values. Each of these options has limitations. Case-wise or listwise deletion results in the loss of a large number of individuals for each analysis, reduces power, and introduces bias when data are not missing completely at random (MCAR) (Allison, 2009). Deletion of variables reduces the scope of the study. Successful imputation depends on an adequate number and type of conditioning variables.

Additionally, as the vertebral variables were the most affected by missing values, I conducted a Little’s MCAR test on these data; the Little’s MCAR test results ($\chi^2 = 46.34$, $df = 52$, $p = 0.695$) indicated that the vertebral measurements were missing completely at random. I attempted two methods of imputation for the vertebral data –
the expectation-maximization method and multiple imputation using the Missing Values module in SPSS. Both resulted in data sets with increased numbers of outliers and skew, possibly due to the limited number of variables available for the conditioning set.

Due to the poor outcome of the imputation attempts, I decided against imputation of the missing values and instead chose case-wise and list-wise deletion. Under conditions of MCAR, casewise deletion will not increase bias and listwise deletion of predictor variables in regression methods is robust as long as missingness among predictors is not related to the dependent variable (Allison, 2009). I opted to discard the L1AP variable as it had the highest number of missing cases at 20 percent but retained the others, with the recognition that each analysis will reflect different subsamples of the data.

After the vertebral variables, the variable with next highest percentage of missing data was LEH at 8.5 percent, which was reduced to 6.9 percent by modifying the criteria for inclusion. The original inclusion criteria of at least two observable anterior teeth selectively removed the older individuals from this important variable in large part due to the tendency to lose teeth with age. For example, the mean age-at-death was 46 years
Table 4: Percent of Missing Values by Variable

<table>
<thead>
<tr>
<th>Variable</th>
<th>n</th>
<th>Missing (%)</th>
<th>Total</th>
</tr>
</thead>
<tbody>
<tr>
<td>Femoral length</td>
<td>821</td>
<td>9 (1.1)</td>
<td>830</td>
</tr>
<tr>
<td>T1AP</td>
<td>721</td>
<td>109 (13.1)</td>
<td>830</td>
</tr>
<tr>
<td>L1AP</td>
<td>663</td>
<td>167 (20.1)</td>
<td>830</td>
</tr>
<tr>
<td>L1TR</td>
<td>703</td>
<td>127 (15.3)</td>
<td>830</td>
</tr>
<tr>
<td>LEH</td>
<td>773</td>
<td>57 (6.9)</td>
<td>830</td>
</tr>
<tr>
<td>Periosteal lesions</td>
<td>813</td>
<td>17 (2)</td>
<td>830</td>
</tr>
<tr>
<td>Total fractures</td>
<td>826</td>
<td>4 (.5)</td>
<td>830</td>
</tr>
<tr>
<td>DMFT Index</td>
<td>806</td>
<td>24 (2.9)</td>
<td>830</td>
</tr>
</tbody>
</table>

for the individuals retaining only one observable anterior tooth, in contrast to 37 years for individuals with two or more observable anterior teeth. Thus, removing from analysis the individuals who have retained only one anterior tooth would create a dataset biased toward the younger individuals. As age-at-death was one of the primary concerns of the study, it was necessary to re-evaluate the inclusion criteria to find a compromise that would allow retention of some of these older individuals while respecting the differential sensitivity to disruption of the anterior tooth types. The LEH inclusion criteria were modified to include any individual with at least one observable mandibular or maxillary canine, as described earlier in this chapter. The remaining variables had very low percentages of missing values, ranging from 3 percent to 0.5 percent. These all were retained without modification and analyzed using listwise or casewise deletion.
Femoral Secular Change Study

Due to previous researchers’ findings of femoral secular change in historic and modern U.S. skeletal samples (i.e. Jantz and Jantz, 1999), a linear regression was conducted to investigate whether secular change was affecting the present sample’s femoral lengths. Euro-American males had a birth interval slightly earlier than the African American males and females, with mean year of birth 1893 for African American Males, 1888 for Euro-American males, and 1895 for African American females. Normality and homoscedasticity assumptions were tested (not shown): the data were not normally distributed for any group but the scatterplots revealed linearity. The homoscedasticity assumption was met and there was no correlation between residuals. The data from each subgroup were trimmed ten percent to account for several outliers in the African American and Euro-American males groups. Femoral length served as the dependent variable and the birth year was the predictor variable.

Year of birth did not predict femur size for the African American females ($F[1,183]=.006, p = 0.937$) or Euro-American males ($F[1,88] = .007, p =0.933$) but does predict femur size for African American males ($F[1,381]= 14.51, p < 0.001$). This difference equates to an increase of 0.41 mm per year for African American males, which was greater than the 0.21 mm/year increase found for African American males born from 1800 through 1979 (Jantz and Jantz, 1999). It should be noted that although the model was significant, the contribution was small. Year of birth explains only 3.4
percent of the variation in femoral length for African American males \( (R^2 = .034) \). In other words, greater than 95 percent of the variation in femoral length, for this sample, was due to some variable(s) other than time. Based on these results, the femora were retained in the study with the caveat that, although the amount of variability explained was small, secular change does contribute to femoral length for the African American males in the sample.

Linear regressions for birth year also were conducted for L1TR and T1AP diameters using ten percent trimmed data for each sex and ancestry group; year of birth did not predict these measurements for any group.

**Data Analysis Methods**

Descriptive analyses were used to characterize the sample and to compare frequencies and means among the groups. The variables first were examined by sex and ancestry, then were divided into six groups to facilitate the within-sample comparisons of sex, ancestry, and regional differences. The six groups consist of Terry Collection African American Males; Terry Collection African American Females; Terry Collection Euro-American Males; Hamann-Todd Collection African American Males; Hamann-Todd Collection African American Females; and Hamann-Todd Collection Euro-American Males. Age, vertebral neural canal diameters, and femoral length means were compared with one-way analysis of variance. Kruskal-Wallis was used to

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compare group distributions for the DMF-T index. Pearson’s Chi-square analysis of independence and Fisher’s exact tests were used to evaluate counts of tibial periosteal lesions, LEH, and fractures.

Question One asks in what ways and to what extent childhood stress is associated with reduced adult survival. This question was interested in survival by lesion presence and how that may differ for males and females, or by ancestry or region. These questions were addressed with the Kaplan-Meier method (K-M) of survival analysis (Kaplan and Meier, 1958). Survival analysis, also referred to as hazards analysis and event history analysis, is an umbrella term referring to a group of methods that model the relationship between particular variables and time to occurrence of an event of interest (Kleinbaum and Klein, 2005). These methods are commonly used in the biomedical sciences and increasingly have been employed in paleopathological analysis (e.g. DeWitte and Wood, 2008; Temple, 2014; Wilson, 2014).

The K-M estimator is a univariate, nonparametric survival analysis method. K-M estimates the survival function, which is used to estimate the probability of surviving past a certain point in time; this is a cumulative measure based on the proportion of the sample surviving to that time (Kleinbaum and Klein, 2005). In addition to the survival function, the items of interest from the K-M method are the median survival times and significance tests for differences between survival functions of the variable levels. K-M is able to include censored data in estimating cumulative survival; censored data refers
to cases that have not experienced the event of interest by the end of the study period or that are lost from the study prior to the end. For the present research, this was relevant because the event of interest was death from natural causes; the study individuals who died from homicide or other trauma are censored in these circumstances.

The present study investigates whether survival was related to different levels of child stress variables. The intent for this research question was to use the Cox proportional hazards model (Cox, 1972) a multivariate survival method that would have allowed testing for between-group differences. The Cox proportional hazards method, however, requires hazards proportionality over time and the present study data were demonstrated to violate that assumption by crossing of the survival curves for many of the analyses (discussed further in Chapter 5: Results). K-M was chosen as a replacement because it does not assume proportionality, but survival differences among sex and ancestry groups and for different variable levels cannot be tested simultaneously. Instead, K-M can be stratified to compare survival within different subgroups by multiple levels of a factor. It is important to note, however, that the resulting stratified pairwise comparisons test the survival function differences of the variable levels within a group, not among the groups. For example, it tests whether a survival difference occurs between short and long femora for African American females, but not for a survival difference for short femora between African American females and African American males. Directly comparing survival functions between
groups was possible by conducting a separate analysis for each level of each variable, but the very high number of analyses and post-hoc comparisons required was unrealistic in terms of setting a significance level and would have an increased risk for generating Type I errors. Since the primary research concern was whether or not stress affected survival among this sample, the compromise was to focus significance testing on within-group differences and to infer between group differences qualitatively.

To that end, separate analyses were conducted for the childhood variables hypoplasia, T1AP, L1TR, and femoral length. As K-M requires a categorical dependent variable, the measured or counted data for each variable were converted into categories. The event of interest was death from natural cause. The survival time was represented by age-at-death. To gain the maximum amount of survival information while limiting the number of comparisons, the analysis for each variable was conducted using the following protocol:

- Each analysis was stratified by Sex/Ancestry groups with significance testing conducted for each stratum;
- If a significant difference in survival curves was detected for any of the Sex/Ancestry groups, pairwise comparisons for survival differences by levels of the variable were conducted only for the affected group, stratified by Sex/Ancestry/Collection group.

Significance for all analyses was based on Log-rank and Tarone Ware tests. Log-rank comparisons typically are used but Log-rank is unreliable when curves cross, a condition affecting many of the analyses in this study (Peat and Barton, 2005). Tarone Ware is less sensitive to crossing but places emphasis on early portions of the curve
(Mills, 2011). All decisions regarding significance were based on Tarone Ware but the Log-rank results also are reported by convention (Allison, 2010).

Question Two asks in what ways and to what extent childhood stress is related to adult health conditions. To answer this question, either binary logistic regression or Fisher’s exact test with Phi, depending on subgroup size, was conducted to evaluate the relationship between childhood variables and adult variables. Prior to hypothesis testing, each Sex/Ancestry and Sex/Ancestry/Collection group was evaluated for associations among variables specific to that group using Pearson’s Chi-square analyses, pairing each dichotomized adult variable with each childhood variable. All variable combinations with a $p < .05$ were included in the regressions. As this step was exploratory rather than hypothesis testing, I was not concerned about Type 1 error. The subsequent logistic regressions were conducted only at the Sex/Ancestry/Group level, because the Chi-square analyses revealed several associations at the Sex/Ancestry/Collections level that were not apparent at the aggregate group level. The Sex/Ancestry Chi-square results, however, are included with the results to illustrate these differences.

For the Chi-square and logistic regression analyses, DMF-T was dichotomized in relation to the specific group’s mean, into 0 (does not exceed the mean) and 1 (equals or exceeds the mean). Fractures were dichotomized into 0 (no fractures) and 1 (one or more fractures). PNB continued to be used as a dichotomized variable defined by 0
(absent) and 1 (present). For regressions, childhood variables were included at the three-levels categorical variable described earlier in this chapter. Age was included with each binary logistic regression as a continuous predictor variable to account for the tendency to accumulate pathology with age.

Question Three asks if the ratios of degenerative disease death to infectious disease death for the skeletal collections groups increase over time and compares their ratios to those of the local and national populations. To answer this question, the study uses cause of death data from the study collection individuals, as well as from the Cleveland, St. Louis, and national populations. Data from the publications were tabulated and examined graphically to observe for changes in the proportion of degenerative disease relative to infectious disease over time. The variable used was the ratio of degenerative disease to infectious disease (D:I), expressed as a percent, based on the crude death counts. A graphical rather than quantitative approach was used due to the limitations of the data as described previously in this chapter.

Question Four asks in what ways and to what extent the results of Questions 1 through 3 (i.e., survival related to childhood stress, relationship between childhood stress and adult health, and cause of death trends) differ by sex, ancestry, or collection. This question was addressed through discussion and summarization of the results by sex, ancestry, and collections.
CHAPTER FIVE: RESULTS

The study results are provided in this chapter. First, the sample is characterized through descriptive statistics, frequency comparisons, and means comparisons to evaluate the overall burden of morbidity and to identify group differences. Next, the analytical results are provided in order of the research questions:

1) Do individuals with childhood stress die younger than those without?
2) Does evidence of childhood stress predict presence of adult stress?
3) Are the patterns of degenerative and infectious causes of death for the sample similar to the contemporaneous local and national trends?
4) Do the results differ by sex, ancestry, or collection?

Descriptive Statistics

Prior to their use in hypothesis testing, the study variables were evaluated with descriptive statistics. Age, vertebral neural canal diameters, and femoral length means were compared among groups using one-way analysis of variance. Kruskal-Wallis was used to compare group distributions for the DMFT index. Chi-square, Fisher’s exact
tests and frequency distributions were used to compare group differences for
frequencies of count variables (hypoplasia, fractures, and tibial periosteal lesions).

**Age-at-Death**

The age-at-death for the sample overall ranges from 18 years to 61 years with a
mean age of 37.9 years (Figure 8 and 9, Table 5). As discussed in Chapter 3, some age
heaping does seem to be present at the five-year age marks, primarily between ages 30
and 50. At about five percent, age 40 has the highest percentage of the five-year age
marks, slightly less that that of the overall highest age, 38.

To compare means among groups, the data for each subgroup were trimmed ten
percent from each end; trimming is a robust method that reduces the influence of
outliers (Wilcox, 1998). Welch’s robust analysis of variance (ANOVA) was conducted on
the age data as it is the more accurate method in instances of heterogeneity of variances
(Field, 2005). Post-hoc tests were chosen in consideration of homoscedasticity and the
unbalanced sample sizes. The mean age-at-death for the sample overall and for the
individual groups falls within early middle age (Table 5). ANOVA and post-hoc tests
demonstrate that the trimmed mean age-at-death for Euro-American males was 5.7
years greater than that of African American males, and that the difference was
significant (Tables 6,7). African American females also had a significantly younger
mean age-at-death than Euro-American males, but the mean ages for African American
Figure 8: Age-at-death distribution for entire sample (n = 830)
Figure 9: Age-at-death boxplots by subgroup.
**Table 5: Descriptive statistics for age in years (n = 830)**

<table>
<thead>
<tr>
<th>Group</th>
<th>n</th>
<th>Min.</th>
<th>Max.</th>
<th>Mean</th>
<th>S.D.</th>
<th>Skew</th>
<th>Kurtosis</th>
<th>Statistic</th>
<th>df</th>
<th>Sig.</th>
<th>10% Trimmed Mean</th>
</tr>
</thead>
<tbody>
<tr>
<td>Sample</td>
<td>830</td>
<td>18</td>
<td>55</td>
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<td>-.952</td>
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<td>487</td>
<td>&lt; .001</td>
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<td>.029</td>
<td>.965</td>
<td>112</td>
<td>.227</td>
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</tr>
<tr>
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<td>231</td>
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<td>57</td>
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<td>9.4</td>
<td>.260</td>
<td>-.864</td>
<td>.972</td>
<td>231</td>
<td>&lt; .001</td>
<td>36.1</td>
</tr>
<tr>
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<td>294</td>
<td>18</td>
<td>60</td>
<td>38.8</td>
<td>9.7</td>
<td>-.047</td>
<td>-.96</td>
<td>.976</td>
<td>294</td>
<td>&lt; .001</td>
<td>38.8</td>
</tr>
<tr>
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<td>18</td>
<td>61</td>
<td>44.2</td>
<td>8.5</td>
<td>-.668</td>
<td>.96</td>
<td>.966</td>
<td>72</td>
<td>.047</td>
<td>44.7</td>
</tr>
<tr>
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<td>57</td>
<td>37.5</td>
<td>9.7</td>
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<td>-.911</td>
<td>.973</td>
<td>146</td>
<td>.006</td>
<td>37.4</td>
</tr>
<tr>
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<td>193</td>
<td>18</td>
<td>55</td>
<td>35.6</td>
<td>10.0</td>
<td>.066</td>
<td>-.972</td>
<td>.965</td>
<td>193</td>
<td>&lt; .001</td>
<td>35.3</td>
</tr>
<tr>
<td>HT White Males</td>
<td>40</td>
<td>24</td>
<td>55</td>
<td>40.2</td>
<td>8.2</td>
<td>.002</td>
<td>-.809</td>
<td>.972</td>
<td>40</td>
<td>.413</td>
<td>40.2</td>
</tr>
<tr>
<td>HT Black Females</td>
<td>85</td>
<td>20</td>
<td>54</td>
<td>34.5</td>
<td>8.6</td>
<td>.292</td>
<td>-.785</td>
<td>.965</td>
<td>85</td>
<td>.020</td>
<td>34.1</td>
</tr>
</tbody>
</table>

1Shapiro-Wilk test

**Table 6: Welch's ANOVA, age-at-death**

<table>
<thead>
<tr>
<th></th>
<th>n</th>
<th>Statistic</th>
<th>df1</th>
<th>df2</th>
<th>Sig.</th>
</tr>
</thead>
<tbody>
<tr>
<td>Sex/Ancestry¹</td>
<td>668</td>
<td>40.47</td>
<td>2</td>
<td>254.21</td>
<td>&lt; .001</td>
</tr>
<tr>
<td>Sex/Ancestry/Collection²</td>
<td>668</td>
<td>29.42</td>
<td>5</td>
<td>180.99</td>
<td>&lt; .001</td>
</tr>
</tbody>
</table>

¹Levene statistic = 5.98 (2,665), p = .003. ²Levene statistic = 3.75 (5, 662), p = .002
Table 7: Games-Howell post-hoc tests, age-at-death

<table>
<thead>
<tr>
<th>Group</th>
<th>Comparison Group</th>
<th>Mean Difference</th>
<th>Std. Error</th>
<th>Sig.</th>
</tr>
</thead>
<tbody>
<tr>
<td>Black Males¹</td>
<td>White Males</td>
<td>-5.7</td>
<td>.73</td>
<td>&lt; .001</td>
</tr>
<tr>
<td></td>
<td>Black Females</td>
<td>1.28</td>
<td>.66</td>
<td>.128</td>
</tr>
<tr>
<td>White Males¹</td>
<td>Black Females</td>
<td>6.93</td>
<td>.82</td>
<td>&lt; .001</td>
</tr>
<tr>
<td>TC Black Males²</td>
<td>TC White Males</td>
<td>-5.89</td>
<td>.82</td>
<td>&lt; .001</td>
</tr>
<tr>
<td></td>
<td>TC Black Females</td>
<td>1.48</td>
<td>.84</td>
<td>.490</td>
</tr>
<tr>
<td></td>
<td>HT Black Males</td>
<td>3.49</td>
<td>.78</td>
<td>&lt; .001</td>
</tr>
<tr>
<td></td>
<td>HT White Males</td>
<td>-1.32</td>
<td>1.18</td>
<td>.871</td>
</tr>
<tr>
<td></td>
<td>HT Black Females</td>
<td>4.69</td>
<td>.92</td>
<td>&lt; .001</td>
</tr>
<tr>
<td>TC White Males²</td>
<td>TC Black Females</td>
<td>7.37</td>
<td>.96</td>
<td>&lt; .001</td>
</tr>
<tr>
<td></td>
<td>HT Black Males</td>
<td>9.38</td>
<td>.91</td>
<td>&lt; .001</td>
</tr>
<tr>
<td></td>
<td>HT White Males</td>
<td>4.57</td>
<td>1.27</td>
<td>.008</td>
</tr>
<tr>
<td></td>
<td>HT Black Females</td>
<td>10.56</td>
<td>1.03</td>
<td>&lt; .001</td>
</tr>
<tr>
<td>TC Black Females²</td>
<td>HT Black Males</td>
<td>2.01</td>
<td>.92</td>
<td>.248</td>
</tr>
<tr>
<td></td>
<td>HT White Males</td>
<td>-2.8</td>
<td>1.28</td>
<td>.256</td>
</tr>
<tr>
<td></td>
<td>HT Black Females</td>
<td>3.21</td>
<td>1.04</td>
<td>.029</td>
</tr>
<tr>
<td>HT Black Males²</td>
<td>HT White Males</td>
<td>-4.81</td>
<td>1.24</td>
<td>.004</td>
</tr>
<tr>
<td></td>
<td>HT Black Females</td>
<td>1.2</td>
<td>.99</td>
<td>.837</td>
</tr>
<tr>
<td>HT White Males²</td>
<td>HT Black Females</td>
<td>6.0</td>
<td>1.33</td>
<td>&lt; .001</td>
</tr>
</tbody>
</table>

¹Significant at $p \leq .017$. ²Significant at $p \leq .003$. Significant results are in bold print.

males and females do not differ significantly from each other. These sex and ancestry patterns were consistent at the collections level with the exception of Hamann-Todd Euro-American and African American males, for whom the 4.8 years disadvantage for African American males does not reach Bonferonni-corrected significance. Across the collections, the only statistically significant age difference was found between the Hamann-Todd and Terry Collection African American males.
Linear Enamel Hypoplasia

Overall, there was a very high burden of LEH among the sample (Figure 10). Only 24 percent of all individuals were free of LEH (Table 8). Thirty-eight percent of the entire sample has one lesion and 38 percent of the entire sample has two or more lesions. Among the Sex/Ancestry groups, only 20 percent of Euro-Americans were unaffected by hypoplasia, while 29 percent of African American females were unaffected. African American males had the highest percentage of two or more lesions at 42 percent, while only 30 percent of African American females had two or more lesions.

Among the Sex/Ancestry/Collection groups, Hamann-Todd African American males had the largest percentage of individuals without lesions at 31 percent, while Terry Collection Euro-American males had the smallest percentage of individuals without lesions at 15.8 percent. Terry Collection males of both ancestry groups had the highest overall burden: they were the least likely to have no lesions and the most likely to have two or more. Euro-American males and African American females from the Hamann Todd Collection were the most often to have a single lesion, but the least often to have two or more lesions. Hamann-Todd African American males were close to evenly divided among zero, one, and two lesions.
Figure 10: LEH category by subgroup.
Table 8: LEH descriptive statistics (n = 773)

<table>
<thead>
<tr>
<th>Group</th>
<th>n</th>
<th>Absent (%)</th>
<th>Present (%)</th>
<th>1 (%)</th>
<th>≥2 (%)</th>
</tr>
</thead>
<tbody>
<tr>
<td>TC Black Males</td>
<td>280</td>
<td>46 (16)</td>
<td>234 (84)</td>
<td>110 (39)</td>
<td>124 (44)</td>
</tr>
<tr>
<td>TC White Males</td>
<td>57</td>
<td>9 (16)</td>
<td>48 (84)</td>
<td>21 (37)</td>
<td>27 (47)</td>
</tr>
<tr>
<td>TC Black Females</td>
<td>138</td>
<td>40 (29)</td>
<td>98 (71)</td>
<td>50 (36)</td>
<td>48 (35)</td>
</tr>
<tr>
<td>HT Black Males</td>
<td>183</td>
<td>57 (31)</td>
<td>126 (69)</td>
<td>56 (31)</td>
<td>70 (38)</td>
</tr>
<tr>
<td>HT White Males</td>
<td>36</td>
<td>10 (28)</td>
<td>26 (72)</td>
<td>17 (47)</td>
<td>9 (25)</td>
</tr>
<tr>
<td>HT Black Females</td>
<td>79</td>
<td>23 (29)</td>
<td>56 (71)</td>
<td>40 (51)</td>
<td>16 (20)</td>
</tr>
<tr>
<td>Totals</td>
<td>773</td>
<td>185 (24)</td>
<td>588 (76)</td>
<td>294 (38)</td>
<td>294 (38)</td>
</tr>
</tbody>
</table>

LEH data were dichotomized into presence and absence to evaluate differences among groups by Chi-square (Table 9). No significant difference in presence or absence exists among the three Sex/Ancestry groups ($\chi^2 = 4.45, df = 2, p = 0.110$). A significant difference was found at the collections level ($\chi^2 = 19.36, df = 2, p = 0.002$). Pairwise comparisons focusing on the research questions of sex, ancestry, and collections differences were conducted with Fisher’s exact tests with only two significant pairings found: 16 percent of Terry Collection African American males were without lesions compared to 31 percent of Hamann-Todd African American males (Table 9). Forty percent of Terry Collection African American females were free of lesions compared to 16 percent of Terry Collection African American males, but this difference did not reach Bonferroni-corrected significance (Table 9).
Femoral Length

Femoral lengths descriptive statistics and normality tests are presented in Table 10. The descriptive statistics show marked sexual dimorphism, with African American females having the lowest mean femoral length at 438 mm and African American males having the highest at 474 mm overall (Figure 11). This trend continues at the collections level. In terms of actual stature, the means do not indicate markedly dis-similar height between African American and Euro-American males, as the mean lengths for both groups was associated with approximately the same mean stature of about 5.6 inches based on stature regression equations (Bass, 1995:27-30). For African American females, the mean femoral length equates to a mean height of about 5.2 inches (Bass, 1995:31).
Figure 11: Femoral length (mm) boxplots by subgroup.
To reduce the influence of the outliers found in several subgroups, the distributions for each subgroup were trimmed ten percent from each end to conduct the ANOVAs. The ANOVAs indicate that significant differences exist among the Sex/Ancestry groups as well as at the collections level (Table 11,12). The variances were not equal among the Sex/Ancestry groups ($F[2,656] = 5.168, p = 0.006$) and the Sex/Ancestry/Collection groups ($F[5,653] = 3.624, p = 0.003$). Games-Howell was chosen for post-hoc comparisons as it is recommended for unequal groups sizes and unequal variances (Field, 2005).

Table 10: Femoral length (mm) descriptive statistics (n = 821)

<table>
<thead>
<tr>
<th>Group</th>
<th>n</th>
<th>Mean</th>
<th>Min.</th>
<th>Max.</th>
<th>S.D.</th>
<th>Skew</th>
<th>Kurtosis</th>
<th>Shapiro-Wilk</th>
<th>df</th>
<th>Sig.</th>
<th>10% Trimmed mean</th>
</tr>
</thead>
<tbody>
<tr>
<td>Black Males</td>
<td>480</td>
<td>474</td>
<td>367</td>
<td>562</td>
<td>26.69</td>
<td>-.063</td>
<td>.245</td>
<td>.998</td>
<td>478</td>
<td>.730</td>
<td>474</td>
</tr>
<tr>
<td>White Males</td>
<td>112</td>
<td>458</td>
<td>382</td>
<td>517</td>
<td>22.40</td>
<td>-.054</td>
<td>1.09</td>
<td>.994</td>
<td>110</td>
<td>.915</td>
<td>459</td>
</tr>
<tr>
<td>Black Females</td>
<td>229</td>
<td>438</td>
<td>364</td>
<td>503</td>
<td>24.37</td>
<td>-.082</td>
<td>-.045</td>
<td>.992</td>
<td>226</td>
<td>.293</td>
<td>439</td>
</tr>
<tr>
<td>TC Black Males</td>
<td>291</td>
<td>474</td>
<td>397</td>
<td>542</td>
<td>27.60</td>
<td>.014</td>
<td>.391</td>
<td>.996</td>
<td>291</td>
<td>.711</td>
<td>474</td>
</tr>
<tr>
<td>TC White Males</td>
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<td>461</td>
<td>414</td>
<td>504</td>
<td>19.53</td>
<td>-.302</td>
<td>1.62</td>
<td>.951</td>
<td>72</td>
<td>.007</td>
<td>461</td>
</tr>
<tr>
<td>TC Black Females</td>
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<td>438</td>
<td>382</td>
<td>494</td>
<td>25.01</td>
<td>-.079</td>
<td>-.482</td>
<td>.989</td>
<td>144</td>
<td>.293</td>
<td>439</td>
</tr>
<tr>
<td>HT Black Males</td>
<td>189</td>
<td>474</td>
<td>410</td>
<td>538</td>
<td>25.13</td>
<td>-.071</td>
<td>-.277</td>
<td>.996</td>
<td>189</td>
<td>.912</td>
<td>474</td>
</tr>
<tr>
<td>HT White Males</td>
<td>40</td>
<td>457</td>
<td>407</td>
<td>517</td>
<td>21.65</td>
<td>.281</td>
<td>.415</td>
<td>.983</td>
<td>40</td>
<td>.812</td>
<td>456</td>
</tr>
<tr>
<td>HT Black Females</td>
<td>85</td>
<td>438</td>
<td>364</td>
<td>503</td>
<td>23.38</td>
<td>-.089</td>
<td>.994</td>
<td>.987</td>
<td>85</td>
<td>.554</td>
<td>439</td>
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</table>
Table 11: Welch’s ANOVA, femoral length.

<table>
<thead>
<tr>
<th></th>
<th>n</th>
<th>Statistic</th>
<th>df1</th>
<th>df2</th>
<th>Sig.</th>
</tr>
</thead>
<tbody>
<tr>
<td>Sex/Ancestry</td>
<td>659</td>
<td>274.23</td>
<td>2</td>
<td>251.25</td>
<td>&lt; .001</td>
</tr>
<tr>
<td>Sex/Ancestry/Coll</td>
<td>814</td>
<td>110.17</td>
<td>5</td>
<td>459.04</td>
<td>&lt; .001</td>
</tr>
</tbody>
</table>

Table 12: Femoral length Games-Howell post-hoc tests.

<table>
<thead>
<tr>
<th>Comparison Group</th>
<th>Mean Difference</th>
<th>Std. Error</th>
<th>Sig.</th>
</tr>
</thead>
<tbody>
<tr>
<td>Black Males¹</td>
<td>White Males</td>
<td>14.92</td>
<td>1.76</td>
</tr>
<tr>
<td></td>
<td>Black Females</td>
<td>35.67</td>
<td>1.52</td>
</tr>
<tr>
<td>White Males¹</td>
<td>Black Females</td>
<td>20.75</td>
<td>1.92</td>
</tr>
<tr>
<td>TC Black Males²</td>
<td>TC White Males</td>
<td>13.38</td>
<td>2.89</td>
</tr>
<tr>
<td></td>
<td>TC Black Females</td>
<td>36.17</td>
<td>2.64</td>
</tr>
<tr>
<td></td>
<td>HT Black Males</td>
<td>.72</td>
<td>2.44</td>
</tr>
<tr>
<td></td>
<td>HT White Males</td>
<td>17.21</td>
<td>3.79</td>
</tr>
<tr>
<td></td>
<td>HT Black Females</td>
<td>36.67</td>
<td>2.74</td>
</tr>
<tr>
<td>TC White Males²</td>
<td>TC Black Females</td>
<td>22.80</td>
<td>3.13</td>
</tr>
<tr>
<td></td>
<td>HT Black Males</td>
<td>-12.65</td>
<td>2.97</td>
</tr>
<tr>
<td></td>
<td>HT White Males</td>
<td>3.83</td>
<td>4.14</td>
</tr>
<tr>
<td></td>
<td>HT Black Females</td>
<td>23.29</td>
<td>3.22</td>
</tr>
<tr>
<td>TC Black Females²</td>
<td>HT Black Males</td>
<td>-35.45</td>
<td>2.77</td>
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<td></td>
<td>HT White Males</td>
<td>-18.96</td>
<td>4.01</td>
</tr>
<tr>
<td></td>
<td>HT Black Females</td>
<td>.49</td>
<td>3.04</td>
</tr>
<tr>
<td>HT Black Males²</td>
<td>HT White Males</td>
<td>-16.48</td>
<td>3.88</td>
</tr>
<tr>
<td></td>
<td>HT Black Females</td>
<td>35.94</td>
<td>2.87</td>
</tr>
<tr>
<td>HT White Males²</td>
<td>HT Black Females</td>
<td>19.46</td>
<td>4.08</td>
</tr>
</tbody>
</table>

¹Statistical significance is \( p \leq .017 \). ²Statistical significance is \( p \leq .003 \). Significant results are in bold print.

Among the Sex/Ancestry groups, all pairs differ significantly. Among the Sex/Ancestry/Coll groups, all pairwise combinations were significantly different except for the group’s counterpart in the other collection; i.e., Terry Collection African
American males do not significantly differ from Hamann-Todd African American males; Terry Collection Euro-American males do not differ significantly from Hamann-Todd Euro-American males; and Terry Collection African American females do not differ significantly from Hamann-Todd African American females.

Vertebral Diameters

As mentioned in the data preparation section in Chapter 4, the first thoracic transverse neural canal diameter and first lumbar anterior-posterior neural canal diameter were removed from the study due to intra-observer measurement variation and excessive missing values, respectively. Similar to the femoral length, the T1AP and L1TR dimensions had a few outliers contributing to abnormal distributions (Figures 12,13). To reduce the influence of outliers on means testing, the data for each subgroup was trimmed 10 percent from each end. Analyses were conducted separately for each of the vertebral dimensions.

The T1AP measurements exhibit sexual dimorphism and ancestry differences in means. Euro-American males had the highest mean T1AP diameter at 15.2 mm, and African American females had the smallest at 14.2 mm (Table 13). On average, the diameter for Euro-American males was 0.5 mm larger than the diameter for African
Figure 12: T1AP diameter boxplots by subgroup.
Figure 13: L1TR diameter boxplots by subgroup.
Table 13: T1AP (mm) descriptive statistics (n = 716)

<table>
<thead>
<tr>
<th>Group</th>
<th>n</th>
<th>Mean</th>
<th>Min.</th>
<th>Max.</th>
<th>S.D.</th>
<th>Skew</th>
<th>Kurtosis</th>
<th>Statistic</th>
<th>df</th>
<th>Sig.</th>
<th>10%Trimmed Mean</th>
</tr>
</thead>
<tbody>
<tr>
<td>Black Males</td>
<td>419</td>
<td>14.7</td>
<td>11.5</td>
<td>18.3</td>
<td>1.27</td>
<td>.089</td>
<td>-.394</td>
<td>.994</td>
<td>419</td>
<td>.126</td>
<td>14.7</td>
</tr>
<tr>
<td>White Males</td>
<td>90</td>
<td>15.2</td>
<td>12.8</td>
<td>18.5</td>
<td>1.21</td>
<td>.151</td>
<td>-.159</td>
<td>.989</td>
<td>90</td>
<td>.647</td>
<td>15.2</td>
</tr>
<tr>
<td>Black Females</td>
<td>207</td>
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<td>11.5</td>
<td>17.1</td>
<td>1.07</td>
<td>.238</td>
<td>.575</td>
<td>.989</td>
<td>207</td>
<td>.106</td>
<td>14.2</td>
</tr>
<tr>
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<td>14.7</td>
<td>11.5</td>
<td>17.8</td>
<td>1.29</td>
<td>.024</td>
<td>-.586</td>
<td>.990</td>
<td>249</td>
<td>.101</td>
<td>14.7</td>
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<tr>
<td>TC White Males</td>
<td>60</td>
<td>15.3</td>
<td>13.0</td>
<td>18.5</td>
<td>1.20</td>
<td>-.087</td>
<td>-.303</td>
<td>.987</td>
<td>60</td>
<td>.766</td>
<td>15.3</td>
</tr>
<tr>
<td>TC Black Females</td>
<td>132</td>
<td>14.1</td>
<td>11.7</td>
<td>16.5</td>
<td>1.06</td>
<td>.069</td>
<td>.936</td>
<td>.988</td>
<td>132</td>
<td>.302</td>
<td>14.2</td>
</tr>
<tr>
<td>HT Black Males</td>
<td>170</td>
<td>14.7</td>
<td>11.6</td>
<td>18.3</td>
<td>1.25</td>
<td>.199</td>
<td>-.057</td>
<td>.994</td>
<td>170</td>
<td>.753</td>
<td>14.7</td>
</tr>
<tr>
<td>HT White Males</td>
<td>30</td>
<td>14.9</td>
<td>12.8</td>
<td>17.7</td>
<td>1.22</td>
<td>.331</td>
<td>.447</td>
<td>.979</td>
<td>30</td>
<td>.879</td>
<td>14.9</td>
</tr>
<tr>
<td>HT Black Females</td>
<td>75</td>
<td>14.2</td>
<td>11.5</td>
<td>17.1</td>
<td>1.09</td>
<td>.512</td>
<td>-.095</td>
<td>.955</td>
<td>75</td>
<td>.009</td>
<td>14.2</td>
</tr>
</tbody>
</table>

Shapiro-Wilk test results.

American males, and African American males were 0.5 mm larger than African American females.

Welch’s ANOVA indicates that the trimmed means for African American males and females and Euro-American males all differ from each other significantly, and these differences were maintained at the collections level (Tables 14,15). Euro-American males from both collections had the highest means and African American females from both collections had the lowest means. Similar to the femoral lengths, the measurements do not differ significantly between groups of the same sex and ancestry. All male-
female pairings differ significantly. Ancestry difference was present within the Terry Collection, where the mean diameter for African American males was 0.66 mm smaller than that of Euro-American males. The means for Hamann-Todd African American and Euro-American males do not differ significantly.

Table 14: Welch’s ANOVA, T1AP

<table>
<thead>
<tr>
<th></th>
<th>n</th>
<th>Statistic</th>
<th>df1</th>
<th>df2</th>
<th>Sig.</th>
</tr>
</thead>
<tbody>
<tr>
<td>Sex/Ancestry&lt;sup&gt;1&lt;/sup&gt;</td>
<td></td>
<td>45.4</td>
<td>2</td>
<td>193.3</td>
<td>&lt; .001</td>
</tr>
<tr>
<td>Sex/Ancestry/Collections&lt;sup&gt;2&lt;/sup&gt;</td>
<td></td>
<td>19.7</td>
<td>5</td>
<td>143.6</td>
<td>&lt; .001</td>
</tr>
</tbody>
</table>

Table 15: T1AP Games-Howell post-hoc tests.

<table>
<thead>
<tr>
<th>Group</th>
<th>Comparison Group</th>
<th>Mean Difference</th>
<th>Std. Error</th>
<th>Sig.</th>
</tr>
</thead>
<tbody>
<tr>
<td>Black Males&lt;sup&gt;1&lt;/sup&gt;</td>
<td>White Males</td>
<td>-0.52</td>
<td>0.11</td>
<td>&lt; .001</td>
</tr>
<tr>
<td></td>
<td>Black Females</td>
<td>0.51</td>
<td>0.08</td>
<td>&lt; .001</td>
</tr>
<tr>
<td>White Males&lt;sup&gt;1&lt;/sup&gt;</td>
<td>Black Females</td>
<td>1.03</td>
<td>0.12</td>
<td>&lt; .001</td>
</tr>
<tr>
<td>TC Black Males&lt;sup&gt;2&lt;/sup&gt;</td>
<td>TC White Males</td>
<td>-0.66</td>
<td>0.14</td>
<td>&lt; .001</td>
</tr>
<tr>
<td></td>
<td>TC Black Females</td>
<td>0.52</td>
<td>0.10</td>
<td>&lt; .001</td>
</tr>
<tr>
<td></td>
<td>HT Black Males</td>
<td>-0.01</td>
<td>0.10</td>
<td>&gt; .999</td>
</tr>
<tr>
<td></td>
<td>HT White Males</td>
<td>-0.26</td>
<td>0.18</td>
<td>.735</td>
</tr>
<tr>
<td></td>
<td>HT Black Females</td>
<td>0.5</td>
<td>0.12</td>
<td>&lt; .001</td>
</tr>
<tr>
<td>TC White Males&lt;sup&gt;2&lt;/sup&gt;</td>
<td>TC Black Females</td>
<td>1.17</td>
<td>0.14</td>
<td>&lt; .001</td>
</tr>
<tr>
<td></td>
<td>HT Black Males</td>
<td>0.65</td>
<td>0.14</td>
<td>&lt; .001</td>
</tr>
<tr>
<td></td>
<td>HT White Males</td>
<td>0.40</td>
<td>0.21</td>
<td>.421</td>
</tr>
<tr>
<td></td>
<td>HT Black Females</td>
<td>1.16</td>
<td>0.16</td>
<td>&lt; .001</td>
</tr>
<tr>
<td>TC Black Females&lt;sup&gt;2&lt;/sup&gt;</td>
<td>HT Black Males</td>
<td>-0.51</td>
<td>0.10</td>
<td>&lt; .001</td>
</tr>
<tr>
<td></td>
<td>HT White Males</td>
<td>-0.77</td>
<td>0.19</td>
<td>.003</td>
</tr>
<tr>
<td></td>
<td>HT Black Females</td>
<td>-0.01</td>
<td>0.12</td>
<td>&gt; .999</td>
</tr>
<tr>
<td>HT Black Males&lt;sup&gt;2&lt;/sup&gt;</td>
<td>HT White Males</td>
<td>-0.26</td>
<td>0.19</td>
<td>.751</td>
</tr>
<tr>
<td></td>
<td>HT Black Females</td>
<td>0.50</td>
<td>0.13</td>
<td>.002</td>
</tr>
<tr>
<td>HT White Males&lt;sup&gt;2&lt;/sup&gt;</td>
<td>HT Black Females</td>
<td>0.76</td>
<td>0.20</td>
<td>.007</td>
</tr>
</tbody>
</table>

<sup>1</sup> Statistical significance is p ≤ .017. <sup>2</sup> Statistical significance is p ≤ .003. Significant are results in bold print.
Descriptive statistics for the L1TR diameter are presented in Table 16; means comparisons in Tables 17 and 18. Among the Sex/Ancestry groups, there was a pattern of sexual dimorphism and ancestry differences. Euro-American males had the

Table 16: L1TR (mm) descriptive statistics (n = 700)

<table>
<thead>
<tr>
<th>Group</th>
<th>n</th>
<th>Mean</th>
<th>Min.</th>
<th>Max.</th>
<th>S.D.</th>
<th>Skew</th>
<th>Kurtosis</th>
<th>Statistic</th>
<th>df</th>
<th>Sig.</th>
<th>10% Trimmed Mean</th>
</tr>
</thead>
<tbody>
<tr>
<td>Black Males</td>
<td>405</td>
<td>21.7</td>
<td>17.6</td>
<td>26.4</td>
<td>1.71</td>
<td>.215</td>
<td>.504</td>
<td>.992</td>
<td>405</td>
<td>.035</td>
<td>21.6</td>
</tr>
<tr>
<td>White Males</td>
<td>91</td>
<td>23.1</td>
<td>20.5</td>
<td>26.3</td>
<td>1.45</td>
<td>.350</td>
<td>-.606</td>
<td>.971</td>
<td>91</td>
<td>.037</td>
<td>23.1</td>
</tr>
<tr>
<td>Black Females</td>
<td>204</td>
<td>20.6</td>
<td>17.0</td>
<td>24.7</td>
<td>1.81</td>
<td>.261</td>
<td>.172</td>
<td>.989</td>
<td>204</td>
<td>.102</td>
<td>20.6</td>
</tr>
<tr>
<td>TC Black Males</td>
<td>245</td>
<td>21.6</td>
<td>18.0</td>
<td>26.0</td>
<td>1.64</td>
<td>.499</td>
<td>-.912</td>
<td>.990</td>
<td>249</td>
<td>.228</td>
<td>21.6</td>
</tr>
<tr>
<td>TC White Males</td>
<td>60</td>
<td>23.1</td>
<td>20.5</td>
<td>26.0</td>
<td>1.35</td>
<td>.416</td>
<td>-.377</td>
<td>.987</td>
<td>60</td>
<td>.080</td>
<td>23.1</td>
</tr>
<tr>
<td>TC Black Females</td>
<td>131</td>
<td>20.6</td>
<td>17.0</td>
<td>24.6</td>
<td>1.60</td>
<td>.320</td>
<td>-.518</td>
<td>.988</td>
<td>132</td>
<td>.062</td>
<td>20.6</td>
</tr>
<tr>
<td>HT Black Males</td>
<td>160</td>
<td>21.8</td>
<td>17.6</td>
<td>26.4</td>
<td>1.82</td>
<td>.212</td>
<td>-.922</td>
<td>.994</td>
<td>170</td>
<td>.253</td>
<td>21.7</td>
</tr>
<tr>
<td>HT White Males</td>
<td>31</td>
<td>23.2</td>
<td>20.6</td>
<td>26.3</td>
<td>1.65</td>
<td>.262</td>
<td>-.561</td>
<td>.979</td>
<td>30</td>
<td>.265</td>
<td>23.1</td>
</tr>
<tr>
<td>HT Black Females</td>
<td>73</td>
<td>20.6</td>
<td>17.5</td>
<td>24.7</td>
<td>1.52</td>
<td>.136</td>
<td>-.774</td>
<td>.955</td>
<td>75</td>
<td>.892</td>
<td>20.6</td>
</tr>
</tbody>
</table>

Table 17: Welch's ANOVA, L1TR.

<table>
<thead>
<tr>
<th></th>
<th>n</th>
<th>Statistic</th>
<th>df1</th>
<th>df2</th>
<th>Sig.</th>
</tr>
</thead>
<tbody>
<tr>
<td>Sex/Ancestry1</td>
<td>563</td>
<td>136.55</td>
<td>2</td>
<td>191.51</td>
<td>&lt; .001</td>
</tr>
<tr>
<td>Sex/Ancestry/Collection2</td>
<td>563</td>
<td>55.02</td>
<td>5</td>
<td>140.78</td>
<td>&lt; .001</td>
</tr>
</tbody>
</table>

1 Levene’s test .353(2,560), p = .702. 2 Levene’s test .877(2, 557), p = .496.
Table 18: L1TR Hochberg’s GT2 post-hoc tests.

<table>
<thead>
<tr>
<th>Group</th>
<th>Comparison Group</th>
<th>Mean Difference</th>
<th>Std. Error</th>
<th>Sig.</th>
</tr>
</thead>
<tbody>
<tr>
<td>Black Males(^1)</td>
<td>White Males</td>
<td>-1.43</td>
<td>.14</td>
<td>&lt; .001</td>
</tr>
<tr>
<td></td>
<td>Black Females</td>
<td>1.04</td>
<td>.11</td>
<td>&lt; .001</td>
</tr>
<tr>
<td>White Males(^1)</td>
<td>Black Females</td>
<td>2.48</td>
<td>.15</td>
<td>&lt; .001</td>
</tr>
<tr>
<td>TC Black Males(^2)</td>
<td>TC White Males</td>
<td>-1.46</td>
<td>.18</td>
<td>&lt; .001</td>
</tr>
<tr>
<td></td>
<td>TC Black Females</td>
<td>.99</td>
<td>.13</td>
<td>&lt; .001</td>
</tr>
<tr>
<td></td>
<td>HT Black Males</td>
<td>-.11</td>
<td>.13</td>
<td>.999</td>
</tr>
<tr>
<td></td>
<td>HT White Males</td>
<td>-1.51</td>
<td>.23</td>
<td>&lt; .001</td>
</tr>
<tr>
<td></td>
<td>HT Black Females</td>
<td>1.02</td>
<td>.16</td>
<td>&lt; .001</td>
</tr>
<tr>
<td>TC White Males(^2)</td>
<td>TC White Females</td>
<td>2.44</td>
<td>.19</td>
<td>&lt; .001</td>
</tr>
<tr>
<td></td>
<td>HT Black Males</td>
<td>1.35</td>
<td>.19</td>
<td>&lt; .001</td>
</tr>
<tr>
<td></td>
<td>HT White Males</td>
<td>-.05</td>
<td>.27</td>
<td>&gt; .999</td>
</tr>
<tr>
<td></td>
<td>HT Black Females</td>
<td>2.48</td>
<td>.21</td>
<td>&lt; .001</td>
</tr>
<tr>
<td>TC Black Females(^2)</td>
<td>HT Black Males</td>
<td>-1.10</td>
<td>.14</td>
<td>&lt; .001</td>
</tr>
<tr>
<td></td>
<td>HT White Males</td>
<td>-2.5</td>
<td>.25</td>
<td>&lt; .001</td>
</tr>
<tr>
<td></td>
<td>HT Black Females</td>
<td>.03</td>
<td>.18</td>
<td>&gt; .999</td>
</tr>
<tr>
<td>HT Black Males(^2)</td>
<td>HT White Males</td>
<td>-1.40</td>
<td>.24</td>
<td>&lt; .001</td>
</tr>
<tr>
<td></td>
<td>HT Black Females</td>
<td>1.13</td>
<td>.17</td>
<td>&lt; .001</td>
</tr>
<tr>
<td>HT White Males(^2)</td>
<td>HT Black Females</td>
<td>2.53</td>
<td>.26</td>
<td>&lt; .001</td>
</tr>
</tbody>
</table>

\(^1\)Statistical significance is \(p \leq .017\). \(^2\)Statistical significance is \(p \leq 0.003\). Significant are results in bold print.

largest mean diameter at 23.1 mm, and African American Females the smallest at 20.6 mm. Euro-American males were on average 1.4 mm larger in diameter than African American males. African American males were 1 mm larger in diameter than African American females. These patterns were maintained with the trimmed means, and Welch’s ANOVA and Hochberg’s GT 2 post-hoc tests demonstrate these differences were statistically significant (Tables 17, 18). At the collections level, all sex and ancestry
combinations were similar to their counterparts in the other collection, with no significant differences. Otherwise, all pairwise comparisons were significantly different.

Decayed, Missing, and Filled Teeth Index

Overall, the sample exhibits moderate to high percentages of affected dentition (Figure 14). The descriptive statistics for the DMFT index are presented in Table 19. African American males had the lowest mean index at 27.7, which equates to nine to ten teeth either carious, filled, or lost antemortem. Euro-American males had the worst dentition on average with a mean index of 49, equating to about 50 percent of teeth affected by decay, loss, or fillings. All groups except Terry Collection Euro-American males had individuals with disease-free teeth, indicated by an index of zero. Kruskal-Wallis and post-hoc tests demonstrate that these differences were significant (Tables 20, 21).

African American males and females do not differ significantly, although African American females had a slightly higher mean DMFT at 31.7 compared to African American males at 27.7. Similar to the pattern observed with the femoral and vertebral measurements, groups of the same sex and ancestry combination do not differ significantly between the collections. Within the collections, Euro-American males
Figure 14: DMFT boxplots by subgroup.
Table 19: DMFT descriptive statistics and normality tests (n = 736).

<table>
<thead>
<tr>
<th>Group</th>
<th>n</th>
<th>Mean</th>
<th>Min.</th>
<th>Max.</th>
<th>S.D.</th>
<th>Skew</th>
<th>Kurtosis</th>
<th>Statistic¹</th>
<th>df</th>
<th>Sig.</th>
</tr>
</thead>
<tbody>
<tr>
<td>Black Males</td>
<td>477</td>
<td>27.71</td>
<td>0</td>
<td>100</td>
<td>21.14</td>
<td>.911</td>
<td>.593</td>
<td>.93</td>
<td>477</td>
<td>&lt; .001</td>
</tr>
<tr>
<td>White Males</td>
<td>107</td>
<td>49.06</td>
<td>0</td>
<td>100</td>
<td>26.0</td>
<td>.352</td>
<td>-.504</td>
<td>.97</td>
<td>107</td>
<td>.007</td>
</tr>
<tr>
<td>Black Females</td>
<td>222</td>
<td>31.74</td>
<td>0</td>
<td>100</td>
<td>21.87</td>
<td>.916</td>
<td>.534</td>
<td>.94</td>
<td>222</td>
<td>&lt; .001</td>
</tr>
<tr>
<td>TC Black Males</td>
<td>285</td>
<td>27.23</td>
<td>0</td>
<td>100</td>
<td>19.89</td>
<td>.907</td>
<td>.907</td>
<td>.94</td>
<td>285</td>
<td>&lt; .001</td>
</tr>
<tr>
<td>TC White Males</td>
<td>69</td>
<td>49.20</td>
<td>0</td>
<td>100</td>
<td>26.96</td>
<td>.240</td>
<td>-.533</td>
<td>.97</td>
<td>69</td>
<td>.044</td>
</tr>
<tr>
<td>TC Black Females</td>
<td>141</td>
<td>31.71</td>
<td>0</td>
<td>100</td>
<td>23.35</td>
<td>.975</td>
<td>.570</td>
<td>.92</td>
<td>141</td>
<td>&lt; .001</td>
</tr>
<tr>
<td>HT Black Males</td>
<td>192</td>
<td>28.43</td>
<td>0</td>
<td>100</td>
<td>22.91</td>
<td>.886</td>
<td>.556</td>
<td>.93</td>
<td>192</td>
<td>&lt; .001</td>
</tr>
<tr>
<td>HT White Males</td>
<td>38</td>
<td>48.80</td>
<td>9.38</td>
<td>100</td>
<td>24.44</td>
<td>.640</td>
<td>-.383</td>
<td>.94</td>
<td>38</td>
<td>.041</td>
</tr>
<tr>
<td>HT Black Females</td>
<td>81</td>
<td>31.81</td>
<td>0</td>
<td>100</td>
<td>19.16</td>
<td>.709</td>
<td>-.005</td>
<td>.95</td>
<td>81</td>
<td>&lt; .003</td>
</tr>
</tbody>
</table>

¹Shapiro-Wilk test for normal distribution.

continue to have the highest mean index and African American males the lowest, and these differences were statistically significant. While Euro-American males of both collections were significantly different from all other groups except for their counterparts, there were no significant differences among the pairings of the African American males and females.

Table 20: DMFT Kruskal-Wallis results.

<table>
<thead>
<tr>
<th>Group</th>
<th>n</th>
<th>X²</th>
<th>df</th>
<th>Asympt. Sig.</th>
</tr>
</thead>
<tbody>
<tr>
<td>Sex/Ancestry</td>
<td>806</td>
<td>63.72</td>
<td>2</td>
<td>&lt; .001</td>
</tr>
<tr>
<td>Sex/Ancestry/Colllection</td>
<td>806</td>
<td>64.16</td>
<td>5</td>
<td>&lt; .001</td>
</tr>
</tbody>
</table>
Table 21: DMFT Dunn's Bonferroni post-hoc tests.

<table>
<thead>
<tr>
<th>Group</th>
<th>Comparison Group</th>
<th>Mean Difference</th>
<th>Std. Error</th>
<th>Adjusted. Sig.</th>
</tr>
</thead>
<tbody>
<tr>
<td>Black Males</td>
<td>White Males</td>
<td>-198.46</td>
<td>25.90</td>
<td>&lt; .001</td>
</tr>
<tr>
<td></td>
<td>Black Females</td>
<td>-44.12</td>
<td>18.91</td>
<td>.059</td>
</tr>
<tr>
<td>White Males</td>
<td>Black Females</td>
<td>-154.40</td>
<td>27.40</td>
<td>&lt; .001</td>
</tr>
<tr>
<td>TC Black Males</td>
<td>TC White Males</td>
<td>-197.59</td>
<td>31.28</td>
<td>&lt; .001</td>
</tr>
<tr>
<td></td>
<td>TC Black Females</td>
<td>-39.93</td>
<td>24.0</td>
<td>&gt; .999</td>
</tr>
<tr>
<td></td>
<td>HT Black Males</td>
<td>-6.20</td>
<td>21.73</td>
<td>&gt; .999</td>
</tr>
<tr>
<td></td>
<td>HT White Males</td>
<td>-207.06</td>
<td>40.2</td>
<td>&lt; .001</td>
</tr>
<tr>
<td></td>
<td>HT Black Females</td>
<td>-58.25</td>
<td>29.31</td>
<td>&gt; .999</td>
</tr>
<tr>
<td>HT Black Males</td>
<td>TC White Males</td>
<td>191.35</td>
<td>32.67</td>
<td>&lt; .001</td>
</tr>
<tr>
<td></td>
<td>TC Black Females</td>
<td>33.72</td>
<td>25.82</td>
<td>&gt; .999</td>
</tr>
<tr>
<td></td>
<td>HT White Males</td>
<td>-200.86</td>
<td>41.32</td>
<td>&lt; .001</td>
</tr>
<tr>
<td></td>
<td>HT Black Females</td>
<td>-52.05</td>
<td>30.84</td>
<td>&gt; .999</td>
</tr>
<tr>
<td>TC Black Female</td>
<td>TC White Males</td>
<td>157.66</td>
<td>34.0</td>
<td>&lt; .001</td>
</tr>
<tr>
<td></td>
<td>HT White Male</td>
<td>-167.13</td>
<td>42.54</td>
<td>&lt; .001</td>
</tr>
<tr>
<td></td>
<td>HT Black Female</td>
<td>-18.33</td>
<td>32.5</td>
<td>&gt; .999</td>
</tr>
<tr>
<td>TC White Male</td>
<td>HT White Male</td>
<td>-9.47</td>
<td>47.02</td>
<td>&gt; .999</td>
</tr>
<tr>
<td>HT Black Females</td>
<td>TC White Males</td>
<td>139.33</td>
<td>38.13</td>
<td>&lt; .004</td>
</tr>
<tr>
<td></td>
<td>HT White Males</td>
<td>148.81</td>
<td>45.76</td>
<td>&lt; .017</td>
</tr>
</tbody>
</table>

Significant results are in bold.

**Tibial Periosteal Lesions**

As anticipated, the tibial lesion burden was high throughout the collection with 44 percent of the entire sample exhibiting bilateral periosteal lesions. At the Sex/Ancestry level, 50 percent of African American females had lesions compared to 42 percent of African American males and 46 percent of Euro-American males but these differences were not significant by Pearson Chi-square test (Table 22; Figures 15,16).
Figure 15: Tibial PNB frequency by sex/ancestry Groups
Among the Sex/Ancestry/Collection groups, all groups except the Terry Collection Euro-American males and Hamann-Todd African American females had a higher percentage of lesion absence than lesion presence. Hamann-Todd Euro-American males had the highest frequency of lesion absence at 64 percent, while Terry Collection Euro-American males had the highest percentage of lesion presence at 52 percent. The Pearson’s Chi-square for lesion presence among the groups was not significant, however, which was surprising considering that the Hamann-Todd Euro-American males exhibited more disparity in its presence than the other groups. This finding may in part be due to the small sample size of Hamann-Todd Euro-American males, but it is noted that none of the expected cell counts were less than five.

Table 22: PNB descriptive statistics and chi-square (n = 816)

<table>
<thead>
<tr>
<th>Group</th>
<th>Absent (%)</th>
<th>Present (%)</th>
<th>Total</th>
<th>Chi-square</th>
<th>df</th>
<th>Sig.</th>
</tr>
</thead>
<tbody>
<tr>
<td>Entire Sample</td>
<td>446 (54)</td>
<td>367 (44)</td>
<td>813</td>
<td>7.47</td>
<td>2</td>
<td>.188</td>
</tr>
<tr>
<td>Sex/Ancestry Groups</td>
<td>-</td>
<td>-</td>
<td>-</td>
<td>7.47</td>
<td>2</td>
<td>.188</td>
</tr>
<tr>
<td>Black Males</td>
<td>274 (58)</td>
<td>202 (42)</td>
<td>476</td>
<td></td>
<td></td>
<td></td>
</tr>
<tr>
<td>White Males</td>
<td>59 (54)</td>
<td>51 (46)</td>
<td>110</td>
<td></td>
<td></td>
<td></td>
</tr>
<tr>
<td>Black Females</td>
<td>113 (50)</td>
<td>114 (50)</td>
<td>227</td>
<td></td>
<td></td>
<td></td>
</tr>
<tr>
<td>Sex/Ancestry/Collection Groups</td>
<td>-</td>
<td>-</td>
<td>-</td>
<td>3.84</td>
<td>5</td>
<td>.147</td>
</tr>
<tr>
<td>TC Black Males</td>
<td>172 (59)</td>
<td>120 (41)</td>
<td>292</td>
<td></td>
<td></td>
<td></td>
</tr>
<tr>
<td>TC White Males</td>
<td>34 (48)</td>
<td>37 (52)</td>
<td>71</td>
<td></td>
<td></td>
<td></td>
</tr>
<tr>
<td>TC Black Females</td>
<td>74 (51)</td>
<td>70 (49)</td>
<td>144</td>
<td></td>
<td></td>
<td></td>
</tr>
<tr>
<td>HT Black Males</td>
<td>102 (55)</td>
<td>82 (45)</td>
<td>184</td>
<td></td>
<td></td>
<td></td>
</tr>
<tr>
<td>HT White Males</td>
<td>25 (64)</td>
<td>14 (36)</td>
<td>39</td>
<td></td>
<td></td>
<td></td>
</tr>
<tr>
<td>HT Black Females</td>
<td>39 (47)</td>
<td>44 (53)</td>
<td>83</td>
<td></td>
<td></td>
<td></td>
</tr>
</tbody>
</table>
Figure 16: PNB frequency by subgroups.
Fractures

The number of fractures among the sample ranges from zero to seven (Figure 17, Tables 22,23). Fifty-five percent of the entire sample had at least one fracture, and of individuals with any fracture, most (34 percent) had only one, and 14 percent had two. Only 6.5 percent of the entire sample had three or more fractures. African American males had a maximum of seven fractures, while Euro-American males and African American females had a maximum of five fractures (Figure 17). The occurrence of high numbers of fractures was somewhat misleading, as the actual number of individuals affected consist of only one individual with seven fractures (0.1% of entire sample), two individuals (0.2%) with five fractures, and nine individuals (1.1%) with four fractures.

The scarcity of individuals with high numbers of fractures complicates frequency comparisons among the sample subgroups because of the resulting empty cells and low expected cell counts, so to test for differences among the groups, the variable was dichotomized into presence and absence for Fisher’s exact tests (Table 24). The variable was categorized into placed into categories of 0, 1, and ≥2 for subsequent survival analysis.

Euro-American males had the highest percentage of fracture presence at 62 percent compared to a low of 48 percent for African American females. Chi-squares were conducted to determine if there were differences in the distributions of fracture
Figure 17: Fracture frequency by sex/ancestry/collection group.
Table 23: Fracture descriptive statistics (n = 826)

<table>
<thead>
<tr>
<th>Group</th>
<th>Absent n(%)</th>
<th>Present(%)</th>
<th>1 Fracture n(%)</th>
<th>≥2 Fractures n(%)</th>
<th>Total</th>
</tr>
</thead>
<tbody>
<tr>
<td>Black Males</td>
<td>211 (43)</td>
<td>274 (57)</td>
<td>179 (37)</td>
<td>95 (20)</td>
<td>485</td>
</tr>
<tr>
<td>White Males</td>
<td>42 (38)</td>
<td>68 (62)</td>
<td>37 (34)</td>
<td>31 (28)</td>
<td>110</td>
</tr>
<tr>
<td>Black Females</td>
<td>120 (52)</td>
<td>111 (48)</td>
<td>68 (29)</td>
<td>43 (19)</td>
<td>231</td>
</tr>
<tr>
<td>TC Black Males</td>
<td>137 (47)</td>
<td>156 (53)</td>
<td>110 (37)</td>
<td>46 (16)</td>
<td>293</td>
</tr>
<tr>
<td>TC White Males</td>
<td>26 (36)</td>
<td>46 (64)</td>
<td>21 (30)</td>
<td>25 (34)</td>
<td>72</td>
</tr>
<tr>
<td>TC Black Females</td>
<td>76 (52)</td>
<td>68 (48)</td>
<td>44 (30)</td>
<td>26 (18)</td>
<td>146</td>
</tr>
<tr>
<td>HT Black Males</td>
<td>74 (39)</td>
<td>118 (51)</td>
<td>69 (36)</td>
<td>49 (25)</td>
<td>192</td>
</tr>
<tr>
<td>HT White Males</td>
<td>16 (42)</td>
<td>22 (58)</td>
<td>16 (42)</td>
<td>6 (16)</td>
<td>38</td>
</tr>
<tr>
<td>HT Black Females</td>
<td>44 (52)</td>
<td>41 (48)</td>
<td>24 (28)</td>
<td>17 (20)</td>
<td>85</td>
</tr>
<tr>
<td>Sample Total</td>
<td>373 (45)</td>
<td>453 (55)</td>
<td>284 (34)</td>
<td>169 (21)</td>
<td>826</td>
</tr>
</tbody>
</table>

Table 24: Fisher’s exact tests, fractures

<table>
<thead>
<tr>
<th>Groups Compared</th>
<th>Chi-square</th>
<th>df</th>
<th>Sig.</th>
</tr>
</thead>
<tbody>
<tr>
<td>Black Males – White Males</td>
<td>1.04</td>
<td>1</td>
<td>.337</td>
</tr>
<tr>
<td>Black Males - Black Females</td>
<td>4.49</td>
<td>1</td>
<td>.037</td>
</tr>
<tr>
<td>Black Females – White Males</td>
<td>5.66</td>
<td>1</td>
<td>.02</td>
</tr>
</tbody>
</table>

Statistical significance is Bonferroni corrected to $p \leq .017$.

presence among the Sex/Ancestry groups and among the Sex/Ancestry/Collection groups. A significant difference was found among the Sex/Ancestry groups ($\chi^2 = 6.70, df = 2, p = 0.03$); subsequent pairwise Fisher’s exact comparisons, however, do not meet the Bonferroni corrected statistical significance of $p \leq .017$. Terry Collection Euro-American males had the highest percentage of fracture presence among the collections subgroups, and African American females from both collections were tied for the lowest. There
were no significant differences found, however, in the distributions at the collections level ($\chi^2 = 10.52, df = 5, p = 0.062$).

**Cause of Death Categories**

The most common cause of death for all groups was infectious disease (Table 25). Except for Hamann-Todd Euro-American males, infectious disease accounted for greater than 50 percent of the deaths for all groups. For Hamann-Todd males, the other etiology category matched infectious disease at 40 percent of the total deaths for that group. Hamann-Todd African American males and females had the highest occurrence

<table>
<thead>
<tr>
<th>Group</th>
<th>Degenerative Disease (%)</th>
<th>Infectious Disease (%)</th>
<th>Other (%)</th>
<th>n</th>
</tr>
</thead>
<tbody>
<tr>
<td>Black Males</td>
<td>125 (25)</td>
<td>305 (63)</td>
<td>57 (12)</td>
<td>487</td>
</tr>
<tr>
<td>Black Females</td>
<td>61 (26)</td>
<td>129 (56)</td>
<td>41 (18)</td>
<td>231</td>
</tr>
<tr>
<td>White Males</td>
<td>29 (26)</td>
<td>54 (48)</td>
<td>29 (26)</td>
<td>112</td>
</tr>
<tr>
<td>TC Black Males</td>
<td>99 (34)</td>
<td>171 (58)</td>
<td>24 (8)</td>
<td>294</td>
</tr>
<tr>
<td>TC Black Females</td>
<td>50 (34)</td>
<td>76 (52)</td>
<td>20 (14)</td>
<td>146</td>
</tr>
<tr>
<td>TC White Males</td>
<td>21 (29)</td>
<td>38 (52)</td>
<td>13 (18)</td>
<td>72</td>
</tr>
<tr>
<td>HT Black Males</td>
<td>26 (14)</td>
<td>134 (69)</td>
<td>33 (17)</td>
<td>193</td>
</tr>
<tr>
<td>HT Black Females</td>
<td>11 (13)</td>
<td>53 (62)</td>
<td>21 (25)</td>
<td>85</td>
</tr>
<tr>
<td>HT White Males</td>
<td>8 (20)</td>
<td>16 (40)</td>
<td>16 (40)</td>
<td>100</td>
</tr>
<tr>
<td>Totals</td>
<td>215 (26)</td>
<td>488 (59)</td>
<td>127 (15)</td>
<td>830</td>
</tr>
</tbody>
</table>
of infectious death at 69 percent and 62 percent respectively. Terry Collection African American males and females had the highest contribution from degenerative death at 34 percent each. Hamann-Todd Euro-American males had the highest contribution from other etiologies. Additional cause of death results are presented under Question 3.

Overall, the descriptive statistics results were notable for the tendency for sex and ancestry groups to exhibit similar means in the size variables with their counterparts across the collections, indicating ancestry differences and sexual dimorphism. The presence of LEH, fractures, and PNB was high in the overall sample, and statistically significant differences occur for LEH and fractures but not for PNB. Euro-American males had especially poor dental health as indicated by high DMFT scores. These results will be discussed further in Chapter 6.

**Question One Results**

This question asks in what ways and to what extent childhood stress is associated with reduced survival. To answer this question, I conducted Kaplan-Meier survival analyses on each of the childhood stress variables: LEH, femoral size, T1AP dimension, and L1TR dimension. Each variable was stratified by Sex/Ancestry group, and if any significant differences were found, pairwise comparisons were conducted only within the affected group as described in Chapter 4. Statistical significance was Bonferroni-corrected to \( p < 0.017 \). All groups were examined for censoring and those
results are discussed by variable when applicable. Note that for all analyses in this section, time begins at zero years although the earliest possible entry into the analysis occurs at 18 years.

**Linear Enamel Hypoplasia**

For this analysis, three hypoplasia categories were created consisting of 0 (no LEH), 1 (one LEH), and 2 (two or more LEH). Within the groups, the pattern of censored cases across survival time was generally similar but there was variation in the percentages of censoring which may affect results, and in particular, Euro-American males were more heavily censored and their results should be interpreted with caution (Table 26). The median survival time is the point at which the probability of survival is 50 percent. It was anticipated that individuals without hypoplasia would have a better (i.e., longer) median survival time than individuals with hypoplasia. This expectation

<table>
<thead>
<tr>
<th>Group</th>
<th>Level</th>
<th>Median Survival Time</th>
<th>95% Confidence Interval</th>
<th>Censored</th>
</tr>
</thead>
<tbody>
<tr>
<td></td>
<td></td>
<td></td>
<td>Lower Bound</td>
<td>Upper Bound</td>
</tr>
<tr>
<td>Black Males</td>
<td>0</td>
<td>36</td>
<td>34.2</td>
<td>27.8</td>
</tr>
<tr>
<td></td>
<td>1</td>
<td>38</td>
<td>36.6</td>
<td>39.4</td>
</tr>
<tr>
<td></td>
<td>2</td>
<td>39</td>
<td>37.1</td>
<td>40.9</td>
</tr>
<tr>
<td>White Males</td>
<td>0</td>
<td>41</td>
<td>35.9</td>
<td>46.1</td>
</tr>
<tr>
<td></td>
<td>1</td>
<td>45</td>
<td>42.2</td>
<td>47.8</td>
</tr>
<tr>
<td></td>
<td>2</td>
<td>44</td>
<td>38.4</td>
<td>49.6</td>
</tr>
<tr>
<td>Black Females</td>
<td>0</td>
<td>38</td>
<td>34.2</td>
<td>41.8</td>
</tr>
<tr>
<td></td>
<td>1</td>
<td>34</td>
<td>29.9</td>
<td>38.1</td>
</tr>
<tr>
<td></td>
<td>2</td>
<td>38</td>
<td>36.7</td>
<td>39.3</td>
</tr>
</tbody>
</table>
was not met for any of the groups. For African American females, the shortest median survival time of 34 years occurred with a single LEH, but the survival time of 38 years for individuals without LEH was the same as for individuals with two LEH.

Neither of the male groups met the expectation of longer survival for individuals lacking LEH. For both groups, individuals without hypoplasia died younger than those with lesions. Median survival for African American males without hypoplasia was two years less than for those with one lesion and three years less than for those with two or more lesions. Median survival for Euro-American males without hypoplasia was four years less than for those with one lesion and three years less than for those with two or more lesions.

There was a great deal of overlap in the confidence intervals among the different LEH categories; this overlap suggests that the survival function differences were significant, and that was confirmed by the survival curves and significance testing (Figures 18 - 20). The differences in the distributions of the variable levels were not statistically significant for any Sex/Ancestry group and thus no pairwise comparisons were conducted. Overall among the groups, the cumulative survival curves were very similar in shape and run close together with no clear advantages among the levels of the variable. An exception occurs with African American females, where the lines for one and two LEH diverge at about age 25 and remain separated until about age 38 when the three lines converge, but the differences were not statistically significant (Figure 19).
Figure 18: LEH survival functions for Black males.
Figure 19: LEH survival functions for Black females.
Figure 20: LEH survival functions for White males.
Femoral Length

Analyses were conducted for each of the Sex/Ancestry groups. The femoral measurements were divided into categories of 1 (short), 2 (average), and 3 (long) as described in Chapter 4. Censored percentages were low for African American females (2.1% - 6.7%) and African American males (3.8% - 5.3%) (Table 27). Euro-American males had unbalanced censoring percentages among the variable levels, ranging from zero percent to 11.8 percent, indicating their results should be interpreted with caution.

Table 27: Femoral length median survival times and censoring

<table>
<thead>
<tr>
<th>Group</th>
<th>Level</th>
<th>Survival Time</th>
<th>Median Survival Time</th>
<th>95% Confidence Interval</th>
<th>Censored</th>
</tr>
</thead>
<tbody>
<tr>
<td></td>
<td></td>
<td></td>
<td>Lower Bound</td>
<td>Upper Bound</td>
<td>n</td>
</tr>
<tr>
<td>Black Males</td>
<td>1</td>
<td>40</td>
<td>38.1</td>
<td>42.0</td>
<td>80</td>
</tr>
<tr>
<td></td>
<td>2</td>
<td>39</td>
<td>37.7</td>
<td>40.1</td>
<td>306</td>
</tr>
<tr>
<td></td>
<td>3</td>
<td>35</td>
<td>31.8</td>
<td>38.2</td>
<td>94</td>
</tr>
<tr>
<td>White Males</td>
<td>1</td>
<td>45</td>
<td>32.4</td>
<td>57.6</td>
<td>15</td>
</tr>
<tr>
<td></td>
<td>2</td>
<td>45</td>
<td>43.0</td>
<td>47.1</td>
<td>80</td>
</tr>
<tr>
<td></td>
<td>3</td>
<td>38</td>
<td>34.3</td>
<td>41.6</td>
<td>17</td>
</tr>
<tr>
<td>Black Females</td>
<td>1</td>
<td>36</td>
<td>33.0</td>
<td>40</td>
<td>38</td>
</tr>
<tr>
<td></td>
<td>2</td>
<td>38</td>
<td>35.9</td>
<td>40.1</td>
<td>146</td>
</tr>
<tr>
<td></td>
<td>3</td>
<td>36</td>
<td>32.1</td>
<td>39.8</td>
<td>45</td>
</tr>
<tr>
<td>TC Black Males</td>
<td>1</td>
<td>40</td>
<td>36.1</td>
<td>43.9</td>
<td>50</td>
</tr>
<tr>
<td></td>
<td>2</td>
<td>40</td>
<td>38.2</td>
<td>41.8</td>
<td>181</td>
</tr>
<tr>
<td></td>
<td>3</td>
<td>35</td>
<td>31.2</td>
<td>38.8</td>
<td>60</td>
</tr>
<tr>
<td>HT Black Males</td>
<td>1</td>
<td>40</td>
<td>32.7</td>
<td>47.3</td>
<td>30</td>
</tr>
<tr>
<td></td>
<td>2</td>
<td>38</td>
<td>36.1</td>
<td>39.9</td>
<td>125</td>
</tr>
<tr>
<td></td>
<td>3</td>
<td>35</td>
<td>31.2</td>
<td>38.8</td>
<td>34</td>
</tr>
</tbody>
</table>
For African American males and Euro-American males, the lowest median survival times were associated with the longest femoral lengths. African American males with short femora had the longest survival time for their group. Euro-American males showed no survival difference between short and average length femora. Among African American females, individuals with average length femora had a slight advantage over those with short or long femora, which were tied for the lowest median survival time. Relative to individuals with short or average femora, long-femora individuals had a survival disadvantage by four to five years for African American males and seven years for Euro-American males. At each level of the variable, Euro-American males had the highest median survival.

The trends noted in the descriptive statistics table were evident in the survival curves. Trends can be seen most clearly for African American males where the long femur line diverges from the other lines at approximately the 82 percent cumulative survival point (age middle to late 20s), and maintains a lower cumulative survival probability throughout (Figure 21). For Euro-American males, the pattern was more mixed, with small femora disadvantaged early in adulthood and long femora disadvantaged at midlife (Figure 24). The curves for African American females mostly were converged, with a slight advantage for short femora from ages 30 to 40 (Figure 25).

The survival distribution differences were significant only for the African American males: between long and short femora (Log Rank: $\chi^2 = 5.54, df = 2, p = 0.019$;
Figure 21: Femoral length survival functions for Black males.
Figure 22: Femoral length survival functions for TC Black males.
Figure 23: Femoral length survival functions for HT Black males.
Figure 24: Femoral length survival functions for White males.
Figure 25: Femoral length survival functions for Black females.
Tarone-Ware: $\chi^2 = 6.5, df = 2, p = 0.011$); and between long and average femora (Log Rank: $\chi^2 = 6.8, df = 2, p = 0.009$; Tarone-Ware: $\chi^2 = 7.02, df = 2, p = 0.008$). To explore this survival difference further, separate pairwise comparisons were conducted for levels of the variable within the Terry Collection and Hamann-Todd African American males groups (Figures 22,23).

As suggested by the survival curves, the difference in survival distribution by femoral length was significant for Terry Collection African American males but not for Hamann-Todd African American males. More specifically, the survival time for long femora differs from the survival time for short femora (Log Rank $\chi^2 = 4.64, df = 2, p = 0.031$; Tarone-Ware $\chi^2 = 6.19, df = 2, p = 0.013$) and for average femora (Log Rank $\chi^2 = 4.7, df = 2, p = 0.030$; Tarone-Ware $\chi^2 = 6.19, df = 2, p = 0.014$). The survival times for short and average femora do not differ from each other.

As the results of this analysis were opposite of the expectations – earlier death for shorter femora - several measures were undertaken to explore it further. First, the raw data were reviewed to determine if data entry or categorization errors accounted for the results, but no errors were found. Second, as secular change was noted to affect the African American male sample, the years of birth for the longer-femur individuals were graphed to determine if there was heaping at the latter half of the birth year interval.
(graphs not shown). For the combined longer-femur group, the birth years were well distributed across the study interval, with 52 percent of individuals born before the study midpoint of 1895. When graphed by Sex/Ancestry/Collection group, however, the graph for the Terry Collection African American males shows that the majority were born after 1895. Terry Collection African American females also had more individuals born after 1895.

A third measure investigated the possibility that reduced survival for African American males reflected secular change by repeating the Kaplan-Meier analysis of femoral lengths categories stratified by decade of birth (results not shown). If longer femora were associated with reduced survival irrespective of secular change, the effect should remain despite decade of birth. The resulting survival functions, however, exhibited converged curves and a lack of significant differences in mean age-at-death among the size categories for all three decades. Based on the graphs and the stratified K-M, the femoral length survival functions for the African American males likely were reflecting secular change. This factor is discussed further in Chapter 6.

In sum, the general pattern for the femora indicates that longer femora were associated with lower survival for both male groups, but was statistically significant only for the African American males. Within the African American male group, only the Terry Collection reaches significance, for the differences between long and short and long and average femoral length.
First Thoracic Vertebral Neural Canal Diameter

For Kaplan-Meier analysis, the T1AP dimension was converted from measurement data to category data as described in Chapter 4: Category 1 (small), Category 2 (average), and Category 3 (large). For all groups, the categories were relative to the specific group mean, not to the overall sample mean. Median survival times and censoring information are provided in Table 28. The percentage of censoring was low.

Table 28: T1AP median survival times and censoring

<table>
<thead>
<tr>
<th>Group</th>
<th>Level</th>
<th>Median Survival Time</th>
<th>95% Confidence Interval</th>
<th>Censored</th>
<th>n</th>
<th>n(%)</th>
</tr>
</thead>
<tbody>
<tr>
<td>Black Males</td>
<td>1</td>
<td>37</td>
<td>33.8 - 40.2</td>
<td>72</td>
<td>3</td>
<td>(4.2)</td>
</tr>
<tr>
<td></td>
<td>2</td>
<td>40</td>
<td>38.2 - 41.8</td>
<td>268</td>
<td>15</td>
<td>(5.6)</td>
</tr>
<tr>
<td></td>
<td>3</td>
<td>35</td>
<td>32.7 - 37.3</td>
<td>79</td>
<td>3</td>
<td>(3.8)</td>
</tr>
<tr>
<td>White Males</td>
<td>1</td>
<td>45</td>
<td>39.0 - 51.0</td>
<td>13</td>
<td>3</td>
<td>(23.1)</td>
</tr>
<tr>
<td></td>
<td>2</td>
<td>44</td>
<td>42.1 - 45.9</td>
<td>64</td>
<td>4</td>
<td>(6.3)</td>
</tr>
<tr>
<td></td>
<td>3</td>
<td>46</td>
<td>41.0 - 51.0</td>
<td>14</td>
<td>2</td>
<td>(14.3)</td>
</tr>
<tr>
<td>Black Females</td>
<td>1</td>
<td>38</td>
<td>29.5 - 46.4</td>
<td>27</td>
<td>0</td>
<td></td>
</tr>
<tr>
<td></td>
<td>2</td>
<td>37</td>
<td>35.0 - 40.0</td>
<td>150</td>
<td>6</td>
<td>(4.0)</td>
</tr>
<tr>
<td></td>
<td>3</td>
<td>33</td>
<td>24.4 - 41.6</td>
<td>34</td>
<td>0</td>
<td></td>
</tr>
<tr>
<td>TC Black Males</td>
<td>1</td>
<td>39</td>
<td>36.4 - 41.6</td>
<td>46</td>
<td>0</td>
<td></td>
</tr>
<tr>
<td></td>
<td>2</td>
<td>42</td>
<td>40.0 - 44.0</td>
<td>155</td>
<td>3</td>
<td>(1.9)</td>
</tr>
<tr>
<td></td>
<td>3</td>
<td>35</td>
<td>38.4 - 41.6</td>
<td>48</td>
<td>1</td>
<td>(2.1)</td>
</tr>
<tr>
<td>HT Black Males</td>
<td>1</td>
<td>35</td>
<td>30.7 - 39.3</td>
<td>26</td>
<td>3</td>
<td>(11.5)</td>
</tr>
<tr>
<td></td>
<td>2</td>
<td>39</td>
<td>37.3 - 40.7</td>
<td>113</td>
<td>12</td>
<td>(10.6)</td>
</tr>
<tr>
<td></td>
<td>3</td>
<td>36</td>
<td>32.2 - 39.8</td>
<td>31</td>
<td>2</td>
<td>(6.5)</td>
</tr>
</tbody>
</table>
for African American females and males, but the Euro-American males had high censoring percentages ranging from 6.3 percent to 23.1 percent. The Euro-American male results are reported but should be considered with caution. Euro-American males had the highest median survival times at all levels. African American females had the lowest survival times for average and large T1AP, but African American males had the lowest survival time for small T1AP. Similar to the survival pattern seen with the femora, large T1AP diameters were associated with the lowest median survival times for African American males; African American females also had lowest survival for large canals. Euro-American males had the highest survival for individuals with large T1AP.

The age patterns described above were observed in the survival curves most clearly for African American males, in which all three curves separate in the early thirties, with the large category maintaining a slightly lower cumulative survival than that of the average category (Figure 26). Small and large re-converge at about year 40. The survival functions for Euro-American males primarily were converged, and for African American females, they were converged except for decreased survival for larger T1AP between years 30 to 40 and a slight advantage for small T1AP between 40 and 50 years (Figures 27,28).

None of the Sex/Ancestry groups reached Bonferroni-corrected statistical significance for different survival among the variable levels. For African American
males, however, average T1AP has a five year advantage over large T1AP that approaches significance (Log Rank $\chi^2 = 3.38, df = 2, p = 0.066$; Tarone Ware: $\chi^2 = 5.47 df = 2, p = 0.019$).

Pairwise comparisons within the African American group indicate a pattern similar to that of the femur in which the Terry Collection males show a greater divergence among the variable levels than do the Hamann-Todd males (Figures 29,30), but the pairwise comparisons for neither group were significant by Log Rank or Tarone Ware. The most marked divergence occurs among Terry males between large and average T1AP, with the large T1AP distribution diverging from the other variable levels around the 30-year mark.

In sum for the T1AP dimension, African American males and females exhibit a similar pattern in which, against expectations, large neural canals exhibit a trend toward reduced survival; however, as expected, the average sized canals had longer survival times than small canals for African American males. Euro-American males had only a one-year difference in median survival between small and large T1AP. None of the differences were found to be statistically significant. Unlike the situation with the femora, the linear regression of the T1AP and year of birth conducted during exploratory data analysis did not demonstrate secular change.
Figure 26: T1AP survival functions for Black males.
Figure 27: T1AP survival functions for White males.
Figure 28: T1AP survival functions for Black females.
First Lumbar Vertebral Neural Canal Diameter

The first lumbar transverse dimensions were placed into size categories of 1 (small), 2 (average), and 3 (large). Placement into categories was based on specific group mean, not on overall sample mean, as described in Chapter 4. For this variable, no groups had extreme censoring but African American Females and Euro-American males did have some variation in percentage of censoring among levels (Table 29).

Consistent with the other childhood variables, Euro-American males had the highest median survival for each level of the variable. African American females had the youngest median survival for medium and large sized L1TR. African American males had the lowest median survival for small L1TR diameters, at 35 years compared to 37 years for African American females. The expectation for this variable was that individuals with small L1TR would have the lowest survival time. The general trend was consistent with this expectation for African American males (Figure 29), who had a median survival time of 35 years for small L1TR compared to 40 years for average and 38 years for large, and for Euro-American males (Figure 32), for whom small and medium size canals were tied for the shortest median survival at 44 years.

For African American females (Figure 33) and Euro-American males, the highest median survival time was associated with large canal dimensions, while for African American males, the average sized canals had the highest survival time. The only group
Table 29: L1TR median survival times and censoring

<table>
<thead>
<tr>
<th>Group</th>
<th>Level</th>
<th>Survival Time</th>
<th>Median</th>
<th>95% Confidence Interval</th>
<th>Censored</th>
</tr>
</thead>
<tbody>
<tr>
<td></td>
<td></td>
<td></td>
<td>Lower Bound</td>
<td>Upper Bound</td>
<td>n</td>
</tr>
<tr>
<td>Black Males</td>
<td>1</td>
<td>35</td>
<td>31.0</td>
<td>39.0</td>
<td>63</td>
</tr>
<tr>
<td></td>
<td>2</td>
<td>40</td>
<td>38.6</td>
<td>41.4</td>
<td>287</td>
</tr>
<tr>
<td></td>
<td>3</td>
<td>38</td>
<td>36.0</td>
<td>40.0</td>
<td>57</td>
</tr>
<tr>
<td>White Males</td>
<td>1</td>
<td>44</td>
<td>41.5</td>
<td>46.5</td>
<td>17</td>
</tr>
<tr>
<td></td>
<td>2</td>
<td>44</td>
<td>41.6</td>
<td>46.3</td>
<td>60</td>
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<tr>
<td></td>
<td>3</td>
<td>47</td>
<td>42.3</td>
<td>51.7</td>
<td>14</td>
</tr>
<tr>
<td>Black Females</td>
<td>1</td>
<td>36</td>
<td>32.1</td>
<td>39.9</td>
<td>33</td>
</tr>
<tr>
<td></td>
<td>2</td>
<td>36</td>
<td>33.7</td>
<td>38.3</td>
<td>132</td>
</tr>
<tr>
<td></td>
<td>3</td>
<td>38</td>
<td>36.0</td>
<td>40.0</td>
<td>40</td>
</tr>
<tr>
<td>TC Black Males</td>
<td>1</td>
<td>37</td>
<td>30.9</td>
<td>43.1</td>
<td>39</td>
</tr>
<tr>
<td></td>
<td>2</td>
<td>40</td>
<td>38.4</td>
<td>41.6</td>
<td>176</td>
</tr>
<tr>
<td></td>
<td>3</td>
<td>36</td>
<td>32.9</td>
<td>39.1</td>
<td>32</td>
</tr>
<tr>
<td>HT Black Males</td>
<td>1</td>
<td>32</td>
<td>23.9</td>
<td>40.1</td>
<td>24</td>
</tr>
<tr>
<td></td>
<td>2</td>
<td>38</td>
<td>34.8</td>
<td>41.2</td>
<td>111</td>
</tr>
<tr>
<td></td>
<td>3</td>
<td>42</td>
<td>38.4</td>
<td>45.6</td>
<td>25</td>
</tr>
</tbody>
</table>

that reaches statistically significant difference was the African American males group for small and large diameters (Log Rank: $\chi^2 = 6.41, df = 2, p = 0.001$; Tarone-Ware: $\chi^2 = 6.5, df = 2, p = 0.011$), and for small and average (Log Rank $\chi^2 = 10.62, df = 2, p = 0.001$; Tarone-Ware: $\chi^2 = 9.02, df = 2, p = 0.003$). Pairwise comparisons for Terry Collection African American males (Figure 30), indicate the significant difference in survival distributions occurs between small and average sized L1TR dimensions (Mantel-Cox Log Rank: $\chi^2 = 8.79, df = 1, p = 0.003$; Tarone-Ware: $\chi^2 = 7.46, df = 1, p = 0.006$), with 37
Figure 29: L1TR survival functions for Black males.
Figure 30: L1TR Survival functions for TC Black males.
Figure 31: L1TR survival functions for HT Black males.
Figure 32: L1TR survival functions for White males.
Figure 33: L1TR survival functions for Black females.
years median survival for small and 40 years median survival for average. For 
Hamann-Todd African American males (Figure 33), the small and large sized canal 
median survival times differ significantly (Log Rank: $\chi^2 = 6.30, df = 1, p = 0.012$; Tarone-
Ware: $\chi^2 = 7.46, df = 1, p = 0.006$), at 32 years for small and 42 years for large. This 
difference was very clear on the survival curve in which the survival functions among 
the variable levels diverge in the mid-twenties, with the function for large L1TR 
consistently remaining above that of average and small and the function for small L1TR 
consistently remaining less than average and large.

In sum, the L1TR dimension presents a slightly more mixed pattern than those of 
the other size variables but overall meets the expectation for a trend of reduced survival 
for smaller to average canal sizes. It was met in part for Euro-American males, with 
small and average canals tied for the lowest survival time. African American females 
had similar median survival and functions for all three levels, but did exhibit a slight 
advantage for small L1TR between 30 and 40 years. Only African American males 
reached statistical significance for reduced survival with small L1TR canal diameters; 
this reduced survival was particularly evident in the functions for small and large size 
diameters within the Hamann-Todd African American male group.

Overall, the survival analyses for the childhood variables demonstrate a trend for 
Euro-American males to have the highest median survival for each level of all four 
variables, and for African American females to have the lowest, but most of these
differences were not significant. African American males had significantly reduced survival for small L1TR canal size; otherwise, the expectation that small sized vertebral dimensions, short femora, and LEH presence would be associated with reduced survivorship for the most part was not met. Conversely, long femora were associated with reduced survivorship in Terry Collection African American males. The results are discussed in Chapter 6.

**Question Two Results**

This question asks in what ways and to what extent childhood stress is associated with adult health. To answer this question, exploratory Chi-square analyses first were conducted to evaluate the predictive ability of the child variables for the later life variables, as described in Chapter 4 (Table 30). Significant associations were identified only for Terry Collection Euro-American males, Hamann-Todd African American males, and Hamann-Todd Euro-American males. Binary logistic regressions were conducted on the associated variables for the Terry Collection Euro-American males and Hamann-Todd African American males. Age was added as a covariate to all regressions. For Hamann-Todd Euro-American males, logistic regression could not be performed on the associated variables due to an insufficient number of cases per variable; instead, the association between the variables was evaluated with Fisher’s exact test and Phi, and a binary logistic regression of the predicted variable and age.
### Table 30: Predictor variables for logistic regression analyses

<table>
<thead>
<tr>
<th>Group</th>
<th>Predicted Variable</th>
<th>Predictor Variable</th>
<th>Pearson’s Χ²</th>
<th>df</th>
<th>Asymp. Sig¹</th>
</tr>
</thead>
<tbody>
<tr>
<td>Black Males</td>
<td>DMFT</td>
<td>L1TR</td>
<td>6.36</td>
<td>2</td>
<td>.041</td>
</tr>
<tr>
<td></td>
<td>Fractures</td>
<td>L1TR</td>
<td>6.69</td>
<td>2</td>
<td>.035</td>
</tr>
<tr>
<td></td>
<td>Periosteal lesions, Cause of death</td>
<td>None Applicable</td>
<td></td>
<td></td>
<td></td>
</tr>
<tr>
<td>White Males</td>
<td>DMFT</td>
<td>T1AP</td>
<td>6.44</td>
<td>2</td>
<td>.040</td>
</tr>
<tr>
<td></td>
<td>Fractures, Periosteal lesions, Cause of death</td>
<td>None applicable</td>
<td></td>
<td></td>
<td></td>
</tr>
<tr>
<td>Black Females</td>
<td>DMFT, Fractures, Periosteal lesions, Cause of death</td>
<td>None applicable</td>
<td></td>
<td></td>
<td></td>
</tr>
<tr>
<td>TC Black Males</td>
<td>DMFT, Fractures, Periosteal Lesions, Cause of death</td>
<td>None Applicable</td>
<td></td>
<td></td>
<td></td>
</tr>
<tr>
<td>TC White Males</td>
<td>Periosteal Lesions</td>
<td>T1AP</td>
<td>7.124</td>
<td>2</td>
<td>.028</td>
</tr>
<tr>
<td></td>
<td>DMFT</td>
<td>T1AP</td>
<td>7.144</td>
<td>2</td>
<td>.028</td>
</tr>
<tr>
<td></td>
<td>Fractures, Cause of death</td>
<td>None applicable</td>
<td></td>
<td></td>
<td></td>
</tr>
<tr>
<td>TC Black Females</td>
<td>DMFT, Fractures, Cause of Death, Periosteal lesions</td>
<td>None applicable</td>
<td></td>
<td></td>
<td></td>
</tr>
<tr>
<td>HT Black Males</td>
<td>DMFT</td>
<td>T1AP</td>
<td>7.270</td>
<td>2</td>
<td>.026</td>
</tr>
<tr>
<td></td>
<td>Fractures</td>
<td>L1TR</td>
<td>9.31</td>
<td>2</td>
<td>.017</td>
</tr>
<tr>
<td></td>
<td>Periosteal lesions, Cause of death</td>
<td>L1TR</td>
<td>10.681</td>
<td>2</td>
<td>.005</td>
</tr>
<tr>
<td></td>
<td></td>
<td>None applicable</td>
<td></td>
<td></td>
<td></td>
</tr>
<tr>
<td>HT White Males</td>
<td>Fractures</td>
<td>L1TR</td>
<td>6.251</td>
<td>2</td>
<td>.044</td>
</tr>
<tr>
<td></td>
<td>DMFT, Cause of Death, Periosteal lesions,</td>
<td>None applicable</td>
<td></td>
<td></td>
<td></td>
</tr>
<tr>
<td>HT Black Females</td>
<td>DMFT, Fractures, Periosteal lesions</td>
<td>None Applicable</td>
<td></td>
<td></td>
<td></td>
</tr>
</tbody>
</table>

¹ Significance for p value is set at .05; results are not Bonferroni adjusted.

The vertebral dimensions supplied all of the predictor variables; hypoplasia and femoral length had no significant associations with any of the later-life variables.

African American females as a group and as collections-based groups had no
associations between adult and childhood variables and thus no predictors with which to run regressions. Logistic regressions were conducted at the collections level rather than the Sex/Ancestry level because differences were found between collections groups during the preliminary analysis. Age was included with each regression as a continuous predictor variable to account for the tendency to accumulate skeletal pathology with age.

The assumption of no multicollinearity of the variables was met for each regression through inspection of VIF values, all of which were less than 1.6. Standardized residuals, Cooks distances, leverage values, and DF Beta values were examined for each regression; all were within accepted range except for leverage values, which will be discussed with the regressions to which they are specific.

**Terry Collection Euro-American Males**

Separate binary logistic regressions were conducted to examine the relationships between DMFT, T1AP, and age, and between PNB, T1AP and age. The first regression evaluated the effects of the T1AP size and age on the likelihood that an individual would have a DMFT index greater than the group mean (Table 31). During inspection of the residuals, 24 percent (n=14) of the leverage values were found to slightly exceed three times the average leverage. As all other residuals were within normal range, the leverages were noted but should not be cause for concern (Field, 2005). The affected
cases were inspected and no unusual values were found among the independent variables.

When age and T1AP size were considered together, they significantly predict whether or not an individual has a DMFT index greater than the mean, $\chi^2 = 8.04$, $df = 3$, $p = 0.045$. The model correctly classified 69 percent of the cases, an increase from the baseline of 53.4 percent. Sensitivity was 80.6 percent and specificity was 55.6 percent. The Hosmer and Lemeshow test ($\chi^2 = 6.238$, $df = 8$, $p = 0.621$) indicates an acceptable model fit. Despite the acceptable fit and the model significance, none of the variables in the equation achieve significance independent from each other.

Table 31: DMFT binary logistic regression, TC White males

<table>
<thead>
<tr>
<th>Variable</th>
<th>n</th>
<th>B</th>
<th>S.E.</th>
<th>Wald</th>
<th>df</th>
<th>Sig.</th>
<th>Odds Ratio</th>
<th>95% CI for Odds Ratio</th>
</tr>
</thead>
<tbody>
<tr>
<td>Age</td>
<td>58</td>
<td>0.03</td>
<td>0.036</td>
<td>0.674</td>
<td>1</td>
<td>0.412</td>
<td>1.03</td>
<td>0.960 – 1.106</td>
</tr>
<tr>
<td>Large T1AP</td>
<td>9</td>
<td>4.90</td>
<td>2.086</td>
<td></td>
<td></td>
<td></td>
<td></td>
<td></td>
</tr>
<tr>
<td>Small T1AP</td>
<td>11</td>
<td>0.63</td>
<td>1.03</td>
<td>0.376</td>
<td>1</td>
<td>0.540</td>
<td>1.88</td>
<td>0.251 – 14.0</td>
</tr>
<tr>
<td>Medium T1AP</td>
<td>38</td>
<td>1.72</td>
<td>0.90</td>
<td>3.678</td>
<td>1</td>
<td>0.055</td>
<td>5.60</td>
<td>0.963 – 32.55</td>
</tr>
<tr>
<td>Constant</td>
<td>-2.43</td>
<td>1.65</td>
<td>2.16</td>
<td></td>
<td></td>
<td></td>
<td>0.142</td>
<td>0.088</td>
</tr>
</tbody>
</table>

The lack of significance for the predictors may be due to sample size; at a total of 58 cases, the sample meets the requirement of 10 to 15 cases per variable but was limited by relatively few cases falling outside of medium T1AP. It was also possible an
interaction occurred between age and the vertebral measurement that was not detected by the multicollinearity check. Another possibility was that the exploratory chi-square result was a Type 1 error.

The second logistic regression for Terry Collection Euro-American males was conducted to evaluate the effects of the T1AP size on likelihood of having tibial periosteal lesions (Table 32). When both predictors were considered together, the resulting model was significant ($\chi^2 = 12.51, df = 3, p = 0.006$). The model fit was adequate per Hosmer and Lemeshow test ($\chi^2 = 11.5, df = 7, p = 0.117$).

Table 32: PNB binary logistic regression, TC White males

<table>
<thead>
<tr>
<th>Variable</th>
<th>n</th>
<th>B</th>
<th>S.E.</th>
<th>Wald</th>
<th>df</th>
<th>Sig.</th>
<th>Odds Ratio</th>
<th>95% CI for Odds Ratio</th>
</tr>
</thead>
<tbody>
<tr>
<td>Age</td>
<td>59</td>
<td>.085</td>
<td>.04</td>
<td>4.04</td>
<td>1</td>
<td>.044</td>
<td>1.09</td>
<td>1.00</td>
</tr>
<tr>
<td>Large T1AP</td>
<td>9</td>
<td>7.18</td>
<td>2</td>
<td></td>
<td></td>
<td></td>
<td>2.09</td>
<td>1.00</td>
</tr>
<tr>
<td>Small T1AP</td>
<td>11</td>
<td>-3.18</td>
<td>1.19</td>
<td>7.10</td>
<td>1</td>
<td>.008</td>
<td>.042</td>
<td>.004</td>
</tr>
<tr>
<td>Medium T1AP</td>
<td>39</td>
<td>-1.99</td>
<td>.97</td>
<td>4.199</td>
<td>1</td>
<td>.040</td>
<td>.136</td>
<td>.020</td>
</tr>
<tr>
<td>Constant</td>
<td></td>
<td>-2.00</td>
<td>1.78</td>
<td>1.26</td>
<td>1</td>
<td>.262</td>
<td>.135</td>
<td></td>
</tr>
</tbody>
</table>

Sixty-two percent of the cases were classified correctly, improved from a baseline of 52.5 percent. Sensitivity was 64.3 percent, specificity was 61.3 percent. All variables were significantly related to PNB. When controlling for other variables in the model: 1) the odds of having PNB increase by a factor of 1.09 with every unit increase in age; 2)
the odds of having PNB were decreased by a factor of 0.042 if an individual has a small T1AP compared to an individual with a large T1AP; 3) the odds of having PNB decrease by a factor of 0.136 if an individual has a medium sized T1AP compared to an individual with a large T1AP. The effect of increasing age for PNB was small, but the effects for small and average T1 were large (Monson, 1980). It was noted however that while adequate for the number of cases, this sample size was small.

Hamann-Todd African American Males

Separate binary logistic regressions were conducted to evaluate the relationships among DMFT, L1TR, T1AP, and Age, and among Fractures, L1TR, and Age. The first regression examined the effects of age, T1AP size, and L1TR size on the likelihood of having a DMFT index that exceeds the group mean (Table 33). On examination of the residuals, six leverage values were found to slightly exceed three times the average leverage. Cases were inspected and there were no unusual values in the data. As all other residuals were within normal range, the leverages were not of concern (Field, 2005). When all variables were included, the model was significant ($\chi^2 = 32.54, df = 5, p < 0.001$). The Hosmer and Lemeshow test indicates the model was an adequate fit ($\chi^2 = 4.04, df = 8, p = 0.853$). The model correctly classified 69.5 percent of cases, improved from 57 percent at baseline, with a sensitivity of 52.3 percent and a specificity of 82.6
percent. Age, large T1AP, medium T1AP, and small L1TR all were significant. When controlling for other variables in the model: 1) the odds of having a DMFT score exceeding the mean increase by a factor of 1.08 for every unit increase in age; 2) for individuals with an average sized T1AP, the odds of having a DMFT score exceeding the mean decrease by a factor of 0.220 compared to an individual with a large T1AP; and 3) for individuals with a small L1TR, the odds of having a DMFT score that exceeds the group mean decrease by a factor of 0.200 compared to an individual with a large L1TR. The age effect was small, but the average T1 and small L1 effects were strong.

Table 33: DMFT binary logistic regression, HT Black males

<table>
<thead>
<tr>
<th>Variable</th>
<th>n</th>
<th>B</th>
<th>S.E.</th>
<th>Wald</th>
<th>df</th>
<th>Sig.</th>
<th>Odds Ratio</th>
<th>CI for Odds Ratio</th>
</tr>
</thead>
<tbody>
<tr>
<td>Age</td>
<td>151</td>
<td>.073</td>
<td>.02</td>
<td>13.11</td>
<td>1</td>
<td>&lt;.001</td>
<td>1.08</td>
<td>1.03</td>
</tr>
<tr>
<td>Large T1AP</td>
<td>31</td>
<td>10.53</td>
<td>2</td>
<td>.005</td>
<td></td>
<td></td>
<td></td>
<td></td>
</tr>
<tr>
<td>Small T1AP</td>
<td>24</td>
<td>-.55</td>
<td>.63</td>
<td>.76</td>
<td>1</td>
<td>.38</td>
<td>.58</td>
<td>.17</td>
</tr>
<tr>
<td>Average T1AP</td>
<td>96</td>
<td>-1.51</td>
<td>.50</td>
<td>9.30</td>
<td>1</td>
<td>.002</td>
<td>.22</td>
<td>.08</td>
</tr>
<tr>
<td>Large L1TR</td>
<td>24</td>
<td>4.62</td>
<td>2</td>
<td>.100</td>
<td></td>
<td></td>
<td></td>
<td></td>
</tr>
<tr>
<td>Small L1TR</td>
<td>24</td>
<td>-1.61</td>
<td>.75</td>
<td>4.57</td>
<td>1</td>
<td>.033</td>
<td>.20</td>
<td>.05</td>
</tr>
<tr>
<td>Average L1TR</td>
<td>103</td>
<td>-.76</td>
<td>.52</td>
<td>2.11</td>
<td>1</td>
<td>.15</td>
<td>.47</td>
<td>.17</td>
</tr>
<tr>
<td>Constant</td>
<td>103</td>
<td>-1.14</td>
<td>.91</td>
<td>1.56</td>
<td>1</td>
<td>.212</td>
<td>.32</td>
<td></td>
</tr>
</tbody>
</table>

185
The second binary logistic regression for the Hamann-Todd African American males was conducted to evaluate the effect of L1TR size on the likelihood of having at least on skeletal fracture (Table 34). Skeletal fractures were dichotomized as 0 (no fractures) and 1 (at least one fracture). The L1TR variable was categorized into 1 (small), 2 (average), and 3 (large).

Table 34: Fracture binary logistic regression, HT Black males

<table>
<thead>
<tr>
<th>Variable</th>
<th>n</th>
<th>B</th>
<th>S.E.</th>
<th>Wald</th>
<th>df</th>
<th>Sig.</th>
<th>Odds Ratio</th>
<th>95% CI for Odds Ratio</th>
</tr>
</thead>
<tbody>
<tr>
<td>Age</td>
<td>159</td>
<td>.04</td>
<td>.02</td>
<td>5.66</td>
<td>1</td>
<td>.017</td>
<td>1.04</td>
<td>1.01</td>
</tr>
<tr>
<td>Large L1TR</td>
<td>25</td>
<td>6.21</td>
<td>2</td>
<td>.045</td>
<td></td>
<td></td>
<td></td>
<td></td>
</tr>
<tr>
<td>Small L1TR</td>
<td>110</td>
<td>-2.11</td>
<td>.86</td>
<td>6.10</td>
<td>1</td>
<td>.014</td>
<td>.12</td>
<td>.02</td>
</tr>
<tr>
<td>Medium L1TR</td>
<td>25</td>
<td>-1.75</td>
<td>.77</td>
<td>5.17</td>
<td>1</td>
<td>.023</td>
<td>.17</td>
<td>.04</td>
</tr>
<tr>
<td>Constant</td>
<td></td>
<td>.76</td>
<td>1.01</td>
<td>.57</td>
<td>1</td>
<td>.450</td>
<td>2.14</td>
<td></td>
</tr>
</tbody>
</table>

When age and L1TR were considered together, the model was significant ($\chi^2 = 18.5, df = 3, p < 0.001$). The Hosmer and Lemeshow test indicates the model was an acceptable fit ($\chi^2 = 4.94, df = 8, p = 0.764$). Seventy percent of cases were classified correctly by the model compared to 65.4 percent of the cases correctly classified by the baseline model. Sensitivity was 90 percent and specificity was 33 percent. Age and all three levels of L1TR were significant. When controlling for the effects of the other variables: 1) the odds of having a fracture increase by a factor of 1.04 with each unit
increase in age; 2) for individuals with a small L1TR, the odds of having a fracture decrease by a factor of 0.12 compared to individuals with a large L1TR; and 3) for individuals with an average L1TR, the odds of having a fracture decrease by a factor of 0.17 compared to individuals with a large L1TR. These effects were moderate to large, as the strength of the effect increases the further the value falls from 1 (Monson, 1980).

Hamann-Todd Euro-American Males

As described previously, the association between L1TR and fractures was evaluated with Fisher’s exact test and Phi, using fracture presence/absence, and L1TR small/not small as variables (Table 35). Fisher’s exact test confirms that L1TR diameter was associated with presence or absence of a fracture: individuals with larger L1TR diameters were less often fractured than expected, while individuals with small diameters were more often fractured than expected ($\chi^2 = 6.15, df = 1, p = 0.02$). The effect

<table>
<thead>
<tr>
<th></th>
<th>Fractures Absent (%)</th>
<th>Fractures Present (%)</th>
<th>Total</th>
<th>$X^2$</th>
<th>df</th>
<th>sig</th>
<th>Phi</th>
</tr>
</thead>
<tbody>
<tr>
<td>L1TR Small</td>
<td>0 (0)</td>
<td>6 (100)</td>
<td>6</td>
<td>6.15</td>
<td>1</td>
<td>.02</td>
<td>.46</td>
</tr>
<tr>
<td>L1TR Not Small</td>
<td>13 (56)</td>
<td>10 (44)</td>
<td>23</td>
<td></td>
<td></td>
<td></td>
<td></td>
</tr>
<tr>
<td>Total</td>
<td>13 (45)</td>
<td>16 (55)</td>
<td>29</td>
<td></td>
<td></td>
<td></td>
<td></td>
</tr>
</tbody>
</table>
was moderate to strong ($\phi = 0.460$, $p = 0.02$). Binary logistic regression demonstrated that presence or absence of a fracture was not predicted by age: $\chi^2 (1) = 2.94$, $p = 0.086$.

Overall, the childhood variables found to have a predictive relationship with adult variables were limited to the vertebral dimensions. African American females and Terry Collection African American males had no predictors identified with exploratory Chi-square. The predictive relationships that were significant by logistic regression and Fisher’s exact tests tended to have strong to moderate effects. Similar to the survival analysis results, larger sizes tend to have a disadvantage as the results predict reduced odds of having a condition for smaller or average sizes, except for L1TR, which showed less association with fracturing. These results are discussed further in Chapter 6.

**Question Three Results**

This question asks if the ratios of degenerative disease death to infectious disease death for the skeletal collections groups increase over time, and how their ratios compare to those of the local and national populations. This question addresses health inequality by examining whether the study sample individuals show the epidemiological transition in mortality; specifically, a decrease in infectious disease death and an increase in degenerative disease deaths. Trends were examined graphically to observe for changes in the proportion of degenerative disease relative to infectious disease. The variable used was the ratio of degenerative disease to infectious
disease (D:I), expressed as a percent, based on the crude death counts as described in Chapter 4.

Figures 34 and 35 first illustrate the distribution in cause of death categories for the years 1914 – 1929 and 1930 – 1941, respectively. Overall in the 1914-1929 interval, the infectious disease rate was high for all groups relative to the degenerative and other categories except for the Hamann-Todd Euro-American males, who had a proportion of other deaths only slightly lower than the infectious proportion. The degenerative disease proportion was particularly low for Hamann-Todd Euro-American males. A change in proportions was evident in the next chart, with all groups showing an increased proportion of deaths due to degenerative disease, most markedly among Terry Collection African American females and Hamann-Todd Euro-American male. This change in relative proportion, however, was in part due to the decrease in the proportions of the ‘other’ causes of death, occurring most dramatically among the Hamann-Todd Euro-American males.

The Kaplan Meier curves in Figures 36 - 38 illustrate the trend for a younger age-at-death for infectious causes than for degenerative causes for all sex and ancestry groups. This finding was expected as degenerative diseases manifest later in life; failing to meet this expectation would have indicated a potential problem with the broad disease categories. The median age-at-death deficit for infectious disease
Figure 34: Cause of death proportions by subgroup, 1914-1929.
Figure 35: Cause of death proportions by subgroup, 1930-1941.
compared to degenerative was seven years for African American males, five years for Euro-American males, and four years for African American females.

Cause of death trends within the samples and within the local and national populations were observed using the ratio of degenerative causes to infectious (D:I) causes. Figures 39 - 44 plot the D:I cause of death variable described above. The results were expected to show that in contrast to the local and national populations, the Terry and Hamann-Todd Collection individuals would begin with a low D:I and maintain a low ratio over the study years. This expectation was met with the exception of the Euro-American males. Results for individual groups are described below.

Terry Collection African American females show very little change over time, beginning with a D:I of 0.5 and ended at 0.6, indicating that more of their deaths were from infectious causes than degenerative (Figure 39). The Terry females’ ratio consistently remained less than that of the national and St. Louis African American females. On the national level, there was a trend for increasing numbers of degenerative disease to infectious but infectious deaths continue to outnumber degenerative. There was a marked increase in the number of degenerative deaths for St. Louis females, beginning with a ratio of 0.4 but ending with a ratio of 1.35.
Figure 36: Death category survival curves for Black males.
Figure 37: Death category survival curves for White males.
Figure 38: Death category survival curves for Black females.
Terry Collection African American males show an increase in the D:I ratio but infectious deaths continued to outnumber degenerative deaths over the course of the study (Figure 40). A slight decrease in the ratio toward the end of the study was shared with the St. Louis male population. The Terry Collection Black males’ ratio remains less than those of the national and St. Louis populations, but they approach the D:I ratio of the national level near the end of the study. The national population was notable for very little change over time. In contrast, the St. Louis African American males begin with a D:I of about 0.7 but degenerative diseases surpass infectious diseases and they end with a ratio of about 1.1.

The Terry Collection Euro-American males began the interval with death only from infectious disease, and infectious disease deaths continued to outnumber degenerative until the mid-1930s, when the ratio rose sharply from about 0.4 to 2.5. They remained well below the ratio for national and St. Louis Euro-American males in the early years of the study, but surpassed them toward the end. The national males started with more infectious deaths than degenerative but ended with a ratio of about 1.7. The St. Louis Euro-American males exhibit the same strong increase in the D:I seen with the St. Louis African American males and females. The St. Louis Euro-American males began with about equal numbers of degenerative and infectious deaths 1, plateauing at about 1.4 in the mid-1920s.
Figure 39: Death category ratios, TC Black females (n = 105).

* St. Louis data compiled from Starkloff (1916); Starkloff and Haines (1928); Bredek (1937). National data compiled from U.S. Department of Commerce (1917, 1927, and 1938).
Figure 40: Death category ratios, Terry Collection Black males (n = 245)

*St. Louis data compiled from Starkloff (1916); Starkloff and Haines (1928); Bredek (1937). National data compiled from U.S. Department of Commerce (1917, 1927, and 1938).
Figure 41: Death category ratios, Terry Collection White males (n = 48)

*St. Louis data compiled from Starkloff (1916); Starkloff and Haines (1928); Bredek (1937). National data compiled from U.S. Department of Commerce (1917, 1927, and 1938).
Figure 42: Death category ratios, Hamann-Todd Black females (n = 64)

*Cleveland data compiled from Rockwood (1926); Green (1938). National data compiled from U.S. Department of Commerce (1917; 1927; 1938).
Hamann-Todd African American females exhibit minimal change in the D:I ratio, ending only slightly higher than they began (Figure 42). Infectious diseases continued to outnumber degenerative deaths through the study years. The ratio peaked at slightly greater than 0.2 in the mid 1920s. At all times, the ratio stayed well below the ratios for the Cleveland and national populations, both of which increased over time. In contrast to the Haman-Todd females, the Cleveland females exhibited a sharp increase, ending with degenerative diseases deaths outnumbering infectious with a ratio of about 1.1. The national African American female ratio increased slightly over time but remained less than 1.

Hamann-Todd African American males exhibited very little change over time, with infectious disease deaths outnumbering degenerative over the entire course of the study and remaining less than the ratios for the Cleveland and national populations (Figure 43). The Hamann-Todd African American males’ ratio remained flat and less than 0.2 until the end of the study when it increased to about 0.3. In contrast, degenerative disease deaths for the Cleveland African American males showed a definite increase over time but infectious disease deaths continued to outnumber degenerative. The national population showed only a slight increase over the time period and remained less than 1. The Hamann-Todd Euro-American males differ from the other groups by exhibiting a decrease in degenerative to infectious disease deaths
Figure 43: Death category ratios, Hamann-Todd Black males (n = 159).

*Cleveland data compiled from Rockwood (1926); Green (1938). National data compiled from U.S. Department of Commerce (1917; 1927; 1938).
Figure 44: Death category ratios, Hamann-Todd White males (n = 23).

*Cleveland data compiled from Rockwood (1926); Green (1938). National data compiled from U.S. Department of Commerce (1917; 1927; 1938).
between the beginning and end of the study years (Figure 44). The trend line fluctuates, possibly affected by small sample sizes. Except for the early 1920s, infectious disease deaths continued to outweigh degenerative disease deaths. In contrast, the Cleveland Euro-American males show a very sharp increase over the study years, beginning with a ratio of slightly less than 1.0 but ending with a ratio of 2.5. On the national level, degenerative disease deaths surpass infectious deaths by the mid 1920s, and by the end of the study the ratio has increased to 1.8.

Overall, the graphs support the expectation that the individuals from the anatomical collections would continue to die primarily from infectious causes. This expectation was supported by D:I ratios remaining less than 1. The exception to this was Terry Collection Euro-American males, who exhibit a sharp increase in the ratio to 2.5 in the latter years of the study. Although the ratios do remain less than 1, most collections samples exhibited a slight increase in the ratio over time, which was unexpected. The results were notable for the disparity between the sample groups and their counterparts at the city-level, with the city populations demonstrating the transition by an increase in the ratio over time. These results will be discussed further in Chapter 6.
Question Four Results

This question examines how patterns of health inequality differ among disadvantaged groups. To answer this question, Questions 1, 2, and 3 were reviewed and summarized in relation to sex, ancestry and collection. Differences in lesion frequencies and means also will be reviewed. Due to the absence of Euro-American females in the study, sex was compared between African American males and females, and Ancestry was compared between Euro-American males and African American males. Collection serves as a proxy for geographical region and was compared between same Sex/Ancestry pairs. Means and frequency results are summarized in Table 36.

Sex Differences

The most notable sex difference found was that African American males show significant differences in survival functions for femoral length, T1AP and L1TR while African American females show no significant differences for any variable. Overall, African American males had higher median survival times than African American females for all variables and levels except for small T1AP, which has a non-significantly lower time for males than for females. Neither sex shows a significant difference for survival associated with the presence of hypoplasia or T1AP. Among the African American males, significant differences in the femur survival functions were limited to the Terry Collection males: the median survival for long femora was seven years lower
that it was for medium and short. For the L1TR diameters, Terry Collection males with small L1TR had a three-year lower median survival time than those with an average L1TR. For Hamann-Todd males, small L1TR has a ten-year median survival deficit compared to large L1TR.

African American females combined and as collections groups had no associations between childhood stress variables and later life health variables. In contrast, African American males as a combined group had a significant association between DMFT and L1TR and between fracture presence and L1TR. On further analysis, this difference was found to be limited to the Hamann-Todd African American males and to include the T1AP diameter. Hamann-Todd African American males with average T1AP or small sized L1TR were less likely to have a DMFT index exceeding the group mean than individuals with large T1AP or L1TR. Compared to individuals with a large T1AP, Hamann-Todd African American males with a small L1TR were less likely to have at least one fracture and those with a medium L1TR were more likely to have at least one fracture.

Terry Collection and Hamann-Todd Collection African American males and females were similar in the relative lack of change in degenerative to infectious disease ratio over the study years. Although there was slight variation in the ratio over time, all groups start and end with a ratio less than 0.5. For females from both collections, this occurs in stark contrast to the ratios for the St. Louis and Cleveland female populations,
which start less than 1 and climb sharply, ending above 1 by the middle to late 1930s. The collections females also remain well below that of the national female population, although the national ratio plateaus around 0.8. African American males from the collection start low and remain less than 0.5, in contrast to the local city populations which trend upward slightly over the time period. The ratios for collections males remain well below that of the national population, which rises from 0.6 to 1.3 over the study years.

Neither the African American males nor the African American females exhibit a clear difference in terms of overall lesion burden. For LEH, there were no statistically significant differences between African American males and females as a group or at the collections level, although the 29 percent difference for LEH absence for Terry Collection African American males and females approaches Bonferroni-corrected significance. There were no statistically significant sex differences in DMFT, PNB, or presence of fractures. Femoral lengths and vertebral measurements all differ significantly by sex, reflecting sexual dimorphism (Table 36). African American females had the highest percentages of individuals with periosteal lesions and a higher mean DMFT index, neither of which were significant. Females as a group and at the collections level had a slightly lower mean age-at-death at, but it was not statistically significant.
Table 36: Summary comparison of variables among subgroups

<table>
<thead>
<tr>
<th>Group</th>
<th>LEH</th>
<th>Mean Femur</th>
<th>Mean T1AP</th>
<th>Mean L1TR</th>
<th>Fracture Lesions % Present</th>
<th>Fracture %</th>
<th>Mean DMFT</th>
<th>Mean Age</th>
</tr>
</thead>
<tbody>
<tr>
<td>Sex Difference</td>
<td></td>
<td></td>
<td></td>
<td></td>
<td></td>
<td></td>
<td></td>
<td></td>
</tr>
<tr>
<td>Black Males</td>
<td>22</td>
<td>78</td>
<td>474</td>
<td>14.7</td>
<td>21.7</td>
<td>42</td>
<td>44</td>
<td>27.7</td>
</tr>
<tr>
<td>Black Females</td>
<td>29</td>
<td>71</td>
<td>439</td>
<td>14.2</td>
<td>20.6</td>
<td>50</td>
<td>52</td>
<td>31.7</td>
</tr>
<tr>
<td>HT Black Males</td>
<td>31</td>
<td>69</td>
<td>474</td>
<td>14.7</td>
<td>21.7</td>
<td>45</td>
<td>38</td>
<td>28.4</td>
</tr>
<tr>
<td>HT Black Females</td>
<td>29</td>
<td>71</td>
<td>439</td>
<td>14.2</td>
<td>20.6</td>
<td>53</td>
<td>52</td>
<td>31.8</td>
</tr>
<tr>
<td>TC Black Males</td>
<td>16</td>
<td>84</td>
<td>474</td>
<td>14.7</td>
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<td>41</td>
<td>47</td>
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<tr>
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<td>71</td>
<td>439</td>
<td>14.2</td>
<td>20.6</td>
<td>49</td>
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<td>31.7</td>
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<tr>
<td>Ancestry Difference</td>
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<tr>
<td>Black Males</td>
<td>22</td>
<td>78</td>
<td>474</td>
<td>14.7</td>
<td>21.7</td>
<td>42</td>
<td>44</td>
<td>27.7</td>
</tr>
<tr>
<td>White Males</td>
<td>20</td>
<td>80</td>
<td>459</td>
<td>15.2</td>
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<td>46</td>
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<td>49.1</td>
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<tr>
<td>HT Black Males</td>
<td>31</td>
<td>69</td>
<td>474</td>
<td>14.7</td>
<td>21.7</td>
<td>45</td>
<td>38</td>
<td>28.4</td>
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<tr>
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<td>72</td>
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<td>474</td>
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<td>41</td>
<td>47</td>
<td>27.2</td>
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<tr>
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<td>84</td>
<td>461</td>
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<td>52</td>
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<td>474</td>
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<tr>
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<tr>
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<td>439</td>
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<td>53</td>
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<tr>
<td>TC White Males</td>
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<td>84</td>
<td>461</td>
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<td>52</td>
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<td>49.2</td>
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<tr>
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<td>28</td>
<td>72</td>
<td>457</td>
<td>14.9</td>
<td>23.1</td>
<td>34</td>
<td>38</td>
<td>48.8</td>
</tr>
</tbody>
</table>

Ancestry Differences

Overall, Euro-American males had higher median survival times than do African American males at all levels of the variables. Euro-American males had no significant differences in survival functions among variable levels, while African American males...
do have different survival for levels of femur size and L1TR with some variation by collection, as described previously.

African American males as a combined group had a significant association between DMFT and L1TR and between fracture presence and L1TR. On further analysis, the associations for African American males were found to be limited to Hamann-Todd and to include the T1AP diameter for the DMFT association. Euro-American males as a combined group had an association between T1AP and DMFT index that on further analysis was found to be limited to the Terry Collection; T1AP also was found to be associated with periosteal lesions. Additionally, further analyses uncovered an association between L1TR and fracture presence for Hamann-Todd Euro-American males.

As described previously, Hamann-Todd African American males with average T1AP or small L1TR were less likely to have a DMFT-index exceeding the group mean than individuals with large T1AP or L1TR. Compared to individuals with a large L1TR, Hamann-Todd African American males with a small or average L1TR were less likely to have at least one fracture than those with a large L1TR. Terry Collection Euro-American males with a small or average sized T1AP had decreased odds of having periosteal lesions compared to individuals with large T1AP; however, in a separate analysis, the T1AP size was not significantly related to DMF-T in the absence of age. Hamann-Todd Euro-American males showed a positive association between L1TR and
fracture presence on Fisher’s exact test, but had no relationship with age by Logistic Regression.

The primary difference in mortality by ancestry was a higher D:I ratio for Euro-American males than African American males. Terry Collection African American males also increase slightly over time but end at a D:I of only 0.5. In the Hamann-Todd Collection, African American males increase over time while the Euro-American males fluctuate, but Euro-American males end with a higher D:I than African American males; both however were less than 0.5. For both Euro-American collections, these ratios stay well below the ratios for their respective city populations. African American males from the collections start low and remain less than 0.5 in contrast to the city populations which trend upward slightly over the time period. The ratios for collections males remain well below that of the national population, which rises from 0.6 to 1.3 over the study years.

There were few statistically significant differences in pathology between African American and Euro-American males. There were no significant differences in LEH, PNB, and fracture presence. African American males had significantly lower mean DMFT scores than Euro-American males overall and at the collections level. African American males had significantly younger ages at death than Euro-American males overall and within the Terry Collection; within Hamann-Todd, the difference, with a $p$-value of 0.004, fails to meet the Bonferroni level of $p \leq 0.003$. African American males
had significantly longer femoral lengths than Euro-American males when compared at the combined Sex/Ancestry level and within the collections. Euro-American males had significantly higher mean T1AP and L1TR neural canal diameters than African American males at the combined Sex/Ancestry level and within the collections, with the exception of Hamann-Todd males, who had no significant difference in T1AP diameter.

**Collections Differences**

African American females and Euro-American males from neither collection exhibit survival difference by level of any variable. As described previously, African American males do differ by collection in their patterns of survival by child variable. Terry Collection males had survival differences between femoral lengths but Hamann-Todd African American males do not. Both groups had differences in L1TR diameters: between small and large L1TR for Hamann-Todd and between small and average for the Terry Collection. There were no differences in survival patterns for T1AP for either African American group at the collection level.

As described previously, African American females from neither collection had significant associations between child variables and later health variables. Terry Collection Euro-American males differed from Hamann-Todd Euro-American males by variable pairings. For the Terry Collection Euro-American males, T1AP was associated with DMFT by Chi-square but did not exhibit predictive ability by logistic regression.
Terry Collection Euro-American males also had a Chi-square association between T1AP and periosteal lesions, which was found to be predictive, along with age, by logistic regression. For Hamann-Todd Euro-American males, Fisher’s exact tests and Phi indicate large L1TR was associated with fracture presence with a moderate to strong effect. Logistic regression shows that fracture presence was not predicted by age. The African American male groups differed between collections as well. The Terry Collection group had no associations between lesions, while for Hamann-Todd, L1TR and T1AP were associated with DMFT index, and L1TR was associated with fracture presence.

The African American female and male groups show little difference between the collections, in all cases beginning and ending with a ratio less than 0.5 while the ratios for their respective cities increase. All stay less than the ratio for the respective national populations. Euro-American males do exhibit different trends in the ratios between collections: while both collections start near zero, the Terry Collection ratio increases to 1.3 while Hamann-Todd stays less than 1. The difference may in part be due to the earlier end for Hamann-Todd collection years, which ends three years earlier than Terry Collection. Ratios for both groups stay less than the ratios for their respective city populations.

There were few statistically significant differences by collection. There were no differences between Hamann-Todd and Terry Collection African American females,
and between Hamann-Todd and Terry Collection African American males, in the PNB, fractures or in mean DMFT, femoral length, or L1TR canal diameters. Hamann-Todd African American males had a significantly younger mean age-at-death than Terry Collection African American males. Terry Collection African American males had a statistically significant higher frequency of LEH presence than Hamann-Todd African American males.

Overall, the study results reflect a mix of met and unmet expectations. The Kaplan-Meier analyses demonstrate that femoral length and L1TR diameters had significant differences in survival among variable levels, but of these, only the L1TR demonstrates the expected reduction for smaller size. The logistic regressions and Fisher’s exact tests results show that vertebral neural canal size was related to several adult conditions for males independent of age. The cause of death analyses demonstrate that African American males and females did not undergo the epidemiological transition but do exhibit slight increase in D:I over the study years. Sex, ancestry, and collections differences exist with African American males in particular exhibiting more significant results than other groups. These results are discussed in Chapter 6.
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CHAPTER SIX: DISCUSSION

Several childhood stress variables were related to survivorship and to health later in life, but statistically significant relationships were limited to males and the direction was not always as expected. The results lend support to the notion that males are more sensitive to environmental effects than females, and lend support to the DOHaD. As expected, the individuals in the tested sample exhibit high skeletal lesion frequencies overall and they more often died from infectious diseases relative to degenerative diseases than did their local and national counterparts. Several sex, ancestry, and regional differences were found for survival, associations of child stress with adult health markers, and lesion frequency. The implications of these results are discussed throughout this chapter. In the following discussion, the term ‘environment’ refers to the overall milieu and so was inclusive of social and physical environmental factors.
Childhood Stress and Survival

This question examined in what ways and to what extent childhood stress was associated with reduced adult survival. Among African American males from the Hamann-Todd Collection, those individuals who exhibited childhood developmental stress in the form of small lumbar canal diameters died ten years earlier than those with large canal diameters. The inverse relationship was found femoral lengths: African American males with longer femoral lengths died younger than individuals with shorter femoral lengths. No differential survival occurred with LEH and T1AP canal diameters.

Based on other studies showing reduced longevity associated with LEH (e.g. Steckel, 2005), stature (e.g. Kemkes-Grottenthaler, 2005), and vertebral canal diameters (e.g. Watts, 2013), and considering the increased physiological stress and disease exposure associated with the adverse housing options available to lower socioeconomic groups, I expected to find clear differences in survival as the frail members of society were selected out of the population. While the literature review demonstrates that an association for early mortality with LEH was not consistent, I anticipated that given the environmental circumstances, individuals exhibiting higher numbers of stress events (i.e., at least two LEH) would die younger than those with zero or one LEH. There are several possible explanations for these results. First, it was possible that the recording method employed in this research was too blunt. As this study recorded only the
number of defects and not the location on the tooth crown, the timing of the defect formation could not be considered. The canines are among the teeth most sensitive to disruption, so while the presence of the defects indicates physiological stress occurred for the sample, the stress may not have occurred during critical developmental windows or may not have been sufficient to cause lasting damage from disruption of other systems.

LEH presence was very high among the individuals in this study; 76 percent of the sample had at least one defect, which was higher than the 33 percent females/40 percent males found for the Milwaukee Country Institutional Grounds cemetery (Milligan, 2010); the 32 percent females/44 percent males for the Dunning Poorhouse Cemetery (Grauer and McNamara, 1995); and 50 percent for the overall sample within de la Cova’s (2008) study of 19th century males. However, it was similar to the overall 73 percent hypoplasia presence Lanphear (1990) found for the Monroe County Poorhouse. With such a high lesion frequency, survival differences may have been better discerned using defect timing instead of defect presence or counts. For example, in a sample with 100 percent LEH presence, use of LEH formation-time methods revealed that individuals who formed enamel defects at earlier ages died younger and had greater mortality risk compared to individuals who formed defects at later ages (Temple, 2014). Employing a time-of-formation method also may have helped to identify LEH formed during the weaning period, which would have implications for
cultural differences in practices and differences in practices between rural and urban areas.

Second, there is evidence suggesting that some enamel defects may represent stress associated with a protective effect rather than a deleterious effect, perhaps representing strengthened immunity. For example, weak expressions of accentuated striae (microscopic enamel defects) that formed during childhood were associated with a lower risk for dying between ages seven and 30 in a medieval Danish population (Thomas, 2003). Since LEH was so common among the present sample, noise from mixed ‘good’ and adverse cases of defect formation may have obscured patterns. Arguing for this interpretation was the tendency for a younger median age-at-death for African American and Euro-American males without defects compared to those with one or two defects. In a related scenario, the canines may have been too sensitive to disruption in this population, possibly causing LEH from minor stress to swamp the effect from the fewer lesions that would develop from greater stress.

The T1AP results show a trend for African American males and females with larger diameters to die five years younger than those with smaller diameters, but the results were not statistically significant. The lack of significant association between a small first T1AP diameter and earlier death was consistent with several other studies involving the T1. Clark (1986), found that younger ages at death were associated with small AP diameters for seven of the 12 thoracic vertebrae but not for T1. The present
study results also were consistent with Watts (2011) who found no reduced longevity for small AP diameters of the tenth through twelfth thoracic vertebrae, and with Tatarek’s (1999) analysis of combined data from the Terry and Hamann-Todd collections, which found no reduced age-at-death for small neural canal diameters for Euro-American males or African American males and females, but instead found several weak inverse relationships. Tatarek’s study, however, included individuals from a broad time interval outside the birth and death dates for the present study, and her Euro-American sample appears to represent a mix of native- and foreign-born individuals.

As expected, small L1TR diameters were associated with lower survival functions. The statistically significant result for both African American male groups reinforces the observation that growth disruption in childhood has negative long-term health consequences. The reduced longevity associated with the L1TR was consistent with results from other studies which found younger ages at death to be associated with decreased lumbar transverse neural canal diameters (e.g. Clark, 1986; Watts, 2011). This result lends support to the concept of inherent male vulnerability and greater susceptibility to environmental disruption (Stini, 1969; Stinson, 1985; Guatelli-Steinberg and Lukacs, 1999).

The negative relationship of the femoral lengths to age-at-death was unexpected given the preponderance of bioarchaeological and health literature linking reduced
longevity with shorter stature (Kemkes-Grottenthaler, 2005). For African American and Euro-American males, individuals with longer femoral lengths had the youngest median age-at-death, although it was statistically significant only for Terry Collection African American males.

What might explain the conflicting results from the L1 and femur survival analyses and what might it mean in terms of early life stress and health? There are several possible explanations for the inverse association found for femoral length and survival. First, secular change explains this trend in African American males at least in part, as indicated by the linear regression of femoral length on the year of birth conducted during exploratory data analysis. Examining the implications of the Kaplan Meier results further, a graph of the birth years for the Terry Collection African American males (the only group for whom the survival difference was statistically significant) demonstrated that more individuals with long femora were born over the latter half of the study birth years than the first half. This finding lends weight to a secular change interpretation. The femoral secular change study conducted prior to data analysis, however, demonstrated that the contribution from year of birth explained only about three percent of the variation in length for African American males. The large amount of variation unexplained by passage of time leaves room for additional sociocultural or environmental factors that may have contributed to survival difference.
Second, terminal adult stature is a complicated mix of genetic and environmental factors, so that not all short stature will represent malnutrition or other stress and not all short stature will be associated with reduced longevity. For example, among Japanese-American males older than 70, taller stature was associated with greater risk for death, higher fasting insulin levels, and with lower frequencies of a variant of the FOXO3 gene which is associated with increased longevity (He et al., 2014). While that study was limited to elderly individuals whose ages fall outside the limits of bioarchaeological estimation, it reinforces the point that mechanisms for within-population stature variation should be evaluated by context.

A third possibility for the inverse association of the femur with survival may be compensatory (i.e. catch up) growth. Compensatory growth refers to growth acceleration following periods of stress, accomplished by reallocation of energy from other developmental systems (Hales and Ozanne, 2003). As described in Chapter 2, decreased longevity and health risks are well-documented consequences associated with fetal and neonatal growth disruption and catch-up growth. Trajectories of compensatory growth, however, are variable. If and when the adverse environmental circumstances improve and adequate nutrition is available after a period of disruption, the individual’s growth may resume at a ‘normal’ rate starting from the small-for-age size small body size; growth may accelerate immediately following the period of adversity; growth may accelerate at a later developmental stage; or growth may
overshoot that of individuals who did not experience growth disruption (Mangel and Munch, 2005). As a result of differential compensatory growth timing and trajectories, terminal stature achieved after growth disruption may be stunted, average, or tall relative to the population. Growth disruption from exposure to adverse circumstances during development therefore will not always result in stunted adult stature.

Although much of the compensatory growth literature is based on animal models, there is evidence for reduced longevity associated with rapid catch-up growth and taller size for humans as well. Among a cohort of Finnish boys who were tall at age seven, boys who were smaller and lighter at birth than predicted by maternal BMI had significantly shorter adult longevity than their peers who had the expected size at birth (Barker et al., 2011). Barker and colleagues (2011:415) suggest this pattern of compensatory growth may be common among African Americans due to higher rates of low birthweight deliveries. This literature underscores the point that although the association between stunting and reduced longevity is well documented geographically and temporally, within-population stature variation may be misleading in regard to estimating health.

Relevant to the present study, the amount of femoral size variability unexplained by change over time leaves room for the possibility that some of the taller individuals who died early may represent those who experienced rapid compensatory growth that masked the earlier growth disruption. The conflicting results of reduced survival for
small L1 and femora may represent different timing of stress and/or compensatory growth. In the study of Finnish boys mentioned above (Barker et al., 2011), the authors were able to identify infancy as the period of increased growth by analyzing maternal and child health records. For the present study, no similar documentation is available but the different timing of fusion for each of the skeletal elements suggests different pathways to lower survival. The lumbar canal is essentially complete by age ten (Papp et al., 1994), while the femur continues to grow until about age 14 to 19 years.

Additionally, to further examine the effects of secular change on the femur survival analysis, I conducted Kaplan Meier curves after dividing the sample into three ten-year cohorts covering the birth years for the study. The difference in median survival decreased and lost statistical significance, and the curves were converged. If the longer femur’s association with survival was not an artifact of secular change, it stands to reason that the relationship would remain after division into the cohorts. More notable, however, was that the curves did not show a divergence for small femora – the small femora remained tightly converged with the other survival functions, suggesting that if secular change was occurring, it was not masking the expected effect of lower survival with shorter stature. Additional exploration along this line falls outside the scope of the present research, but the conflicting femur and L1 patterns suggest investigating survival differences among differing size variable mismatches as a means of discerning compensatory growth.
Finally, it is possible that the results may be explained by chance or may reflect a sample bias. Thirteen percent of the thoracic vertebrae and 15 percent of the lumbar vertebrae had missing values. Casewise deletion for individuals with missing data resulted in different samples for each analysis. It is possible that the conflicting results merely reflect these different samples, although as discussed in Chapter 4, casewise deletion will not increase bias under conditions of MCAR (Allison, 2009). The similarity of the present study’s vertebral results to those of Watts (2011) and Tatarek (1999) suggest the results are more likely to support a biological explanation than to support chance.

The statistically significant associations for small L1TR sizes with reduced survival support the literature linking childhood development to adult longevity. The direction for the femur was the inverse of the research expectation. This finding has implications for bioarchaeological interpretations of adult health, for which it is common to equate shorter stature with poor health and taller stature with good health. If the femur results in this study were due solely to secular change, the rapidity with which it occurred should be of concern to researchers working with long time-scale samples, and especially for researchers working with 19th to 20th century industrial samples. If the femur results were in part due to adverse effects related to compensatory growth, the present study highlights the need to explore within-group variation with the understanding that the relationship between stature and longevity is not consistent.
Childhood Stress and Adult Health

This research examined whether or not individuals who experienced childhood developmental stress were more likely to have accumulated additional signs of poor health. Directional relationships would suggest that childhood stress resulted in greater susceptibility to additional health problems; that individuals were exposed to ongoing adverse environmental circumstances; or both. Significant relationships were found only for Hamann-Todd African American males and Euro-American males from both collections, and only for the vertebral dimensions as predictor variables. While few significant relationships were found, those that were found were strong. Additionally, these results are interesting because they demonstrate the expected and common increase in dental problems, tibial lesions, and fractures found with age, yet simultaneously demonstrate that, among the affected groups, the odds of having these pathologies are greater for large vertebral canal sizes independent of age.

Independent of age, Hamann-Todd African American males with average T1AP or small L1TR had strongly decreased odds for a DMFT greater than their group mean, and individuals with small or average L1TR had strongly decreased odds of having skeletal fractures. These results conflict with the L1 survival results that demonstrated that Hamann-Todd African male individuals with small L1TR canal diameters died younger than those with larger canals. On one hand, these findings suggest better resilience for the individuals with smaller L1 or T1 relative to individuals with large
VNC. However, these findings also possibly are a paradoxical situation in which the individuals with smaller diameters are dying prior to greater expression of or accumulation of fractures and dental disease. This relationship remains unclear; both conditions also were weakly, positively associated with age, but the reductions in odds were significant independent of age. Additionally, reduced odds for fracture for small sizes, independent of age, conflicts with evidence that intrauterine growth disruption may increase fracture risk by affecting peak bone mass (Cooper et al., 2006). The reasons for the contradictory L1TR results are unclear. Perhaps larger sized individuals were more often selected for strenuous or dangerous labor than smaller sized individuals.

Hamann-Todd Euro-American males had a significant association between L1TR diameter size and fracture presence when tested by Fisher’s exact test. Individuals with large L1 had fewer than expected fractures. Age had no effect on present or absence of fractures, which is contrary to the basic idea of injury accumulation over time. Additionally, the association of large L1 with fewer fractures is in contrast to the Hamann-Todd African American males, who had decreased odds for fracture with small L1. In this case of the Euro-American males, the relationship was more consistent with the expectation that larger L1 indicates less childhood stress and better skeletal health.

Independent of age, Terry Collection Euro-American males had strongly decreased odds for having periosteal lesions if they had small or average T1AP canals.
Age, independent of canal diameter size, had a weak positive effect on presence of periosteal lesions. This relationship is interesting; although the Terry Collection Euro-American males had only small, non-significant differences in survival functions for T1AP, they did exhibit the general trend for larger T1AP to have reduced median age-at-death. Decreased odds for periosteal lesions for small and average T1AP are consistent with the trend for better survival with small and average T1AP.

A DMFT score greater than the group mean also was significantly related to T1AP diameter for Terry Collection Euro-American males, but none of the T1AP diameters were significant independent of age. This suggests that for DMFT, age and T1 had an affect but the two may be too correlated for significance or there may be some other variable confounding the results. The regression coefficients were positive, indicating that T1AP size and age combined were increasing the odds for higher DMF-T scores. Worse DMFT scores related to increased age are unsurprising as caries and antemortem tooth loss tend to increase with age.

There is little published research that specifically tests relationships between child developmental stress variables and other skeletal lesions, so my expectation that individuals with developmental stress would be more susceptible to other conditions (i.e., frailty) was based in large part on the DOHaD and accumulation literature, and on the bioarchaeological literature demonstrating overall high lesion counts for pauper and institution cemeteries. Studies that have examined associations between child stress and
other lesions had varied results. In medieval London, periosteal lesions were not related to femoral length (DeWitte and Bekvalac, 2011). Cribra orbitalia (a sign of childhood nutritional or parasite stress) was positively correlated with periosteal lesions for medieval and early modern Croatians (Novak et al., 2009). Tatarek’s (1999) vertebral canal study of a Terry Collection and Hamann-Todd combined sample found very few significant associations between specific vertebrae and numerous skeletal pathologies, but found that individuals with smaller neural canal diameters had fewer associations with pathology than did individuals with average to large canals. Tatarek’s (1999) study demonstrating a tendency for individuals with smaller canal sizes to have fewer associated skeletal pathologies is consistent with the present study results showing decreased odds for later life pathological conditions for individuals with smaller or average canal diameters.

The category of cause of death demonstrated no significant associations for any of the child stress variables during the exploratory Pearson’s Chi-square analysis, which was surprising; I had anticipated that degenerative disease would be associated with child stress variables based on DOHaD literature. Since hypoplasia demonstrated no significant survival differences in the Kaplan-Meier analyses, the lack of significant relationships to adult health conditions was not surprising. The lack of a significant association of infectious disease for shorter femora was against expectations that individuals with shorter stature would have worse health and more pathology;
however, it is important to remember that it is unknown whether or not the individuals with short stature reached their genetic potential for height.

In sum, several relationships were found among the childhood stress variables and adult health variables. Significant results were again limited to males, although for these analyses, both Euro-American male groups are represented and only the Hamann-Todd African American males are represented. When a relationship between variables was present, the analyses show a general pattern of decreased odds for having an adult health condition with smaller or average sizes relative to large.

Cause of Death Trends

This research question examined cause of death data to determine if the individuals in the anatomical collections showed evidence of epidemiological transition; i.e., a decrease in infectious disease deaths relative to degenerative disease deaths. The research expectation was that a lack of evidence for transition would be found for the anatomical collection groups, and their degenerative to infectious disease ratios would be lower in comparison to their local and national counterparts. A caveat to this analysis is the small number of Euro-American and African American females available for each interval represented on the graphs.

The expectation that the sample individuals would maintain infectious disease as their primary cause of death was met for all groups except for Terry Collection Euro-
American males. The D:I ratios for the other five Sex/Ancestry/Collection groups stayed less than 1. Despite the continued dominance of infectious disease deaths, however, all groups except Hamann-Todd Euro-American males and Hamann-Todd African American females had a higher D:I ratio at the end of the study years than they did at the beginning. While the continued low D:I ratio indicates that the transition had not occurred for most of the study sample, the slight upward trend over time suggests that some change was occurring. The change over time, though, was not uniformly upward: most of the study sample groups demonstrated fluctuations, most notably the Euro-American male groups. The Hamann-Todd Euro-American males’ D:I ended lower than it began, but the final interval was increased from the previous interval. Hamann-Todd African American females demonstrated little improvement in the D:I, remaining flat. The Terry Collection Euro-American males were the exception to the continued low D:I. This group experienced a marked D:I spike at the latter interval of the study years, increasing from less than 0.5 to about 2.5. The increase is suspicious because it is extreme in relation to the trend over the previous intervals. Lending support to an increased D:I, however, was the higher mean age-at-death Terry Euro-American males had relative to the other anatomical collections groups in the study, indicating they had more time for degenerative disease to manifest.

As expected, the collections individuals maintained lower D:I than those of the local and national populations. The national, Cleveland, and St. Louis populations all
exhibited increased D:I over the course of the study years, in most cases exceeding a D:I of 1. The city populations generally experienced the steepest increases. The trajectories of change over time are particularly interesting when comparing the collections individuals to the local population. As discussed in Chapter 4, historic mortality data can be difficult to compare due to regional and temporal differences in knowledge and qualifications of coroners or medical examiners, and to changes in the ICD over time. These problems are mitigated somewhat for comparisons of the collections with their respective cities because they shared a pool of examiners. Some of the study groups share a similar trajectory to their city population while others do not. African American females from both collections did not experience the steep increase in D:I found for the city populations. In contrast, the change for study sample females was so slight that it almost was flat. Terry Collection African American males had a similar, gradual trajectory to the St. Louis African American males, although the D:I for both groups stays 1. After an initial plateau, the Hamann-Todd African American males share a similar level of increase with the Cleveland African American male group. The national population D:I exhibits a gradual increase for all groups.

As described in Chapter 2, Omran’s (1971) original model for epidemiological transition is criticized for presenting a uniform process, failing to account for differential change by demographic subgroups, including failure to account for heterogeneity within subgroups (Zuckerman et al., 2014). The D:I differences between
the collections groups and their respective cities are evidence in support of heterogeneity of transition within populations. The collections groups comprise a very specific subgroup of the city data. These individuals most likely were very poor at least at the end of their lives, and remaining unclaimed from a morgue has implications for precarious social networks as well. In this regard, they likely were among the most vulnerable residents of their cities. In light of the literature reviewed in Chapter 2 regarding the contribution of improved living standards and public health measures to the transition, the disparity in the D:I changes suggests continued poor circumstances and pathogen exposure for these collections individuals in contrast to the improving circumstances for their sex and ancestry counterparts in the city as a whole.

A contributor to the continued dominance of infectious disease mortality for the study sample individuals may be related to rural-to-city migration. As can be seen from the state of birth maps in Chapter 3, many of the individuals in the study sample moved to St. Louis and Cleveland from other states. Individuals moving between geographic locations may encounter new pathogens or pathogen strains not previously experienced, particularly when living in crowded and unsanitary housing, and thus may lack an effective adaptive immune response to them.

Another contributor to the continued high infectious disease rate may be exposure to coal smoke. Prolonged exposure to coal smoke causes significant health problems for children and adults. In children, coal smoke has been shown to adversely
affect lung development, causing clinically significant reductions in pulmonary function including forced expiratory volume and lung capacity (Fullerton et al., 2008). Low birthweight, prenatal mortality, and intrauterine growth retardation also are associated with coal smoke. Indoor smoke exposure also has detrimental health effects, linked with lung cancer, chronic obstructive pulmonary disease, cardiovascular events, acute carbon monoxide toxicity, decreased pulmonary function, pneumonia, impaired immune function, and cataracts (Zhang and Smith, 2007; Fullerton et al., 2008).

Relevant to the high continued rate of infectious disease for the study sample individuals, coal smoke also has been linked with increased risk for tuberculosis; the tuberculosis bacillus attacks the alveolar macrophages, which are damaged by smoke inhalation, thereby reducing an individual’s ability to resist the invading pathogens (Fullerton et al., 2008). Although all individuals living in the Cleveland or St. Louis would have been exposed to the ambient air pollution, the poor faced compounded risk from indoor air pollution from use of wood or coal burning stoves. For example, in a poor St. Louis city neighborhood, 80 percent of the homes relied on coal stoves (Primm, 1998:446). For individuals with decreased immunocompetence, the combination of increased potential exposure to the TB mycobacterium due to crowded housing, and decreased ability to destroy the mycobacterium after exposure due to the effects of smoke inhalation would have increased risk for the developing the disease. Cigarette smoke potentially could have played a role as well.
The mortality analysis indicates that African American males and females in the collections did not undergo the transition by end of the study years and that their D:I stayed less than that of the local and national populations. On the local level, the city populations experienced steeper increases. On the national level, although African American males and females had a gradual increase in the D:I over time, their D:I also remained less than 1, indicating that they, too, did not undergo the transition by the end of the study years. This was in contrast to the Terry Collection, national, and city Euro-American males, who experienced marked transitions over the study years. Hamann-Todd Euro-American males fluctuated over time and ended with a decreased D:I relative to the beginning of the study.

**Sex, Ancestry, and Regional Differences**

The fourth research question sought to determine if there were any differences in survival, adult health, and epidemiological transition among the groups. There were no significant differences in the numbers of skeletal lesions or DMFT. All size variables differ significantly, reflecting sexual dimorphism. Neither sex underwent the epidemiological transition, with D:I remaining less than 1 and with little change over time. The most striking finding regarding sex differences was that for African American males, childhood development was associated with differential survival and adult health conditions; for African American females, child stress was not associated with
differential survival or adult health conditions. The associations for African American males occur with the femoral and the vertebral diameters, vary by region, and were statistically significant.

The pattern of significant results occurring only for African American males and not for African American females was consistent with the contention that males exhibit greater frailty, or that females are more biologically robust. Males are more susceptible to viral, bacterial, fungal, and parasitic infections than females (Klein, 2000:628). Differences in susceptibility are explained by differential effects of the sex hormones on the immune system - androgens suppress immunocompetence while estrogen enhances it - and by differential effects of hormones on disease resistance genes (Klein, 2000:629-632). More specifically, females have higher immunoglobulin levels and a stronger immune response to pathogen exposure (Verthelyi, 2001). As noted in Chapter 2, however, the pattern of greater male sensitivity was not consistent. For example, Watts (2013) found reduced longevity for shorter femora for one group of British archaeological males but not for another, and found that females with craniofacial fluctuating asymmetry had lower ages at death but males did not. Conversely, Weisensee (2013) found that 19th century Lisbon males had higher rates of fluctuating asymmetry than did their female counterparts.

The slightly higher age-at-death for African American males than for females in the present study was not surprising as the 1900-1902 life expectancies showed little
difference in life expectancy from birth, age 1, and 20 years. I had anticipated that the collections females may trend slightly lower than the males due to complications of pregnancy and childbirth, but only two individuals had causes of death attributed to pregnancy. The paucity of significant lesion count differences was similar to the patterns found in the 19th century poorhouse and institutional cemeteries reviewed in Chapter 2.

The review of ancestry differences between males revealed that while African American and Euro-American males both had relationships between child development markers and adult health conditions, only African American males had differential survival for child development variables. This finding suggests greater environmental sensitivity for African American males. Lesion frequency differences were limited to worse teeth for Euro-American males, which was not surprising based on de la Cova’s (2008) finding of greater frequency of caries in Euro-American males. Neither African American males nor Hamann-Todd Collection Euro-American males underwent the epidemiological transition; Terry Collection Euro-American males may have but their results are suspect.

The other major ancestry difference was age-at-death. Euro-American males outlived African American males by an average of five years based on the mean ages of the samples under study. While neither of these groups could be said to exhibit good overall health, having differential survival for levels of a developmental stress variable
and earlier mean age-at-death lends weight to the concept of greater vulnerability for African American males than for Euro-American males. If both groups represented the poor and marginalized members of their societies, why might this be so?

There are several possible explanations. First, African American males may have been at greater risk for fetal developmental disruption, and subsequent weakened immunological systems, than Euro-American males. For example, African American women currently have two times as many preterm and small for gestational age deliveries as Euro-American women (Kuzawa and Sweet, 2009). This disparity has been documented for several decades; given the well-documented historical environmental stressors and as there is evidence low birthweight can be intergenerational, low birth weight likely was a risk for African American mothers at the turn of the 20th century as well (Jasienska, 2009).

Second, some of the African Americans in the collections may have had decreased resistance to disease from pre-existing conditions. As demonstrated in the birth-state maps in Chapter 4, many of the African Americans in the sample migrated into Cleveland and St. Louis from Southern States while most of the Euro-American males migrated from Northeastern or Great Lakes states. Pellagra, hookworm, and malaria were common among rural southern farmworkers, so it is possible that some of the migrating African Americans experienced that burden (Humphreys, 2009). The poor health status of migrant African Americans was noted by health officials
throughout the North, citing high rates of adult, infant, and child morbidity and mortality for the newcomers and characterizing their health as worse than African Americans who had not migrated north (Meckel, 1997). Cobb (1935:161) noted that many of the African Americans in the Hamann-Todd Collection were new migrants, and that few of them were of older age.

Economic discrimination also likely contributed to the age-at-death disparity. African Americans were disproportionately affected by the Great Depression, having higher rates of unemployment than Euro-Americans (Sundstrom, 1997). Economic disparity likely would have contributed to increased vulnerability through malnutrition and psychological stress. The combinations of accumulated damage, environmental and living condition hazards, undernutrition, and new stressors would have challenged the immune system of and contributed to infectious disease susceptibility. Finally, any findings related to age must consider that some age heaping is present in both collections; this pattern could affect the present study results, although it is noted that the Hamann-Todd collection is deemed reliable under careful sampling selection (Meindl et al, 1990).

Statistically significant regional differences were few. No significant differences exist between the African American female groups. African American males from both collections have differential survival for a child developmental stress variable, but only the Hamann-Todd group has significant predictive relationships between vertebral
variables and adult health conditions. Euro-American males from both groups had associations between a vertebral variable and adult health variables, but neither group has an association between child stress and reduced survival. Cause of death data for the Terry Collection Euro-American males suggests they underwent the epidemiological transition while Euro-American Hamann-Todd males did not.

There was striking similarity in measurement variables and DMFT between same sex groups across collections. For pathologies, the only significant difference was between Euro-American males: Terry Collection Euro-American males had more LEH. Non-significant lesion frequency differences between regions were not entirely unexpected based on de la Cova’s (2008) previous research showing few significant differences in lesion frequencies among the 19th century males.

While statistically few significant differences between the collections were found, the overall pattern that emerges from the results suggests that African American males from the Hamann-Todd experienced worse health than their Terry Collection counterparts. Hamann-Todd African American males had associations between vertebral canal diameter size, oral health and fractures that suggest an influence of child developmental factors on the odds of having an adult health condition. Additionally, the Hamann-Todd group had a striking ten-year reduction in median age of survival associated with a small lumbar canal transverse diameter; Terry Collection African American males had a three-year reduction.
The differences in study results for the collections lends some support to the research expectation that Hamann-Todd individuals would have worse health due to environmental stress from the extreme population increase over the late nineteenth and early twentieth centuries. As discussed in Chapter 3, while both cities had large and rapid population increases, Cleveland’s population increased by 138 percent between 1900 and 1920, compared to a 64 percent increase in St. Louis. This extreme population increase had affects on clean water and sewer control and also contributed to increased crowding in housing. If the study sample individuals had reduced immunocompetence, exposure to new pathogens as well as increased pathogen load from crowded housing and environmental factors may have resulted in greater susceptibility to infectious disease.

Other evidence for worse health for Hamann-Todd African American males was found in the D:I ratios. The cause of death analysis shows that the Hamann-Todd African American males maintained a very low D:I of about 0.3 while Terry Collection group increased to about 0.7; infectious diseases had a lower mean age-at-death. Additionally, the trends for the overall city populations differed: the D:I for St. Louis African American males increased to 1 while that of Cleveland maxed at 0.7. While this indicating a greater continued contribution to death from infectious disease for the Cleveland African American males, the increased D:I for the St. Louis city data suggests that conditions in that city were improving for African American males. Further
evidence comes from Cobb’s (1935:161) analysis of the Hamann-Todd collection, in which he noted that: 1) many of the African American individuals in the collection were newly arrived migrants, based on lab documentation; and 2) the African American individuals had a mean age-at-death of 37 years, which he described as “an unusually early peak” that was not shared by Euro-Americans.

The study results were a mix of met and unmet expectations, with some seemingly paradoxical situations. The few significant differences in survival times based on levels of stress variables must be considered in relation to selective mortality. For any sample of adults, it is important to remember that selective mortality operates on heterogeneity in frailty – a society’s most frail infants and children do not survive childhood and thus are not represented among the adults (Wood et al., 1992; DeWitte and Stojanowski, 2015). For this sample of unclaimed individuals, most of whom died younger than their predicted life expectancy, there may be too little heterogeneity in frailty for differential survival results to be discerned and the indicators chosen may not be adequate measures of frailty in this sample.

The clearest and least contradictory results show that: 1) African American males from both collections had the expected result of lower survival when they had smaller first lumbar transverse canal diameters; 2) the vertebral canal diameters were related to several adult health conditions that vary by group but in most cases the odds of having a negative health condition decreased for small or for average size relative to large; 3)
females exhibit less sensitivity to environmental disruption than males, as evidenced by no reduction in survival associated with stress variables, and no relationships of stress variables to adult health; and 4) with the possible exception Terry Collection Euro-American males, the sample groups did not undergo the epidemiological transition as defined by a persistence of greater incidence of infectious cause of death relative to degenerative, and in all cases show worse ratios than the city overall.

The survival disadvantage for African American males with small lumbar neural canal diameters, particularly for the Hamann-Todd group, lends support to the concept of early life origins for health disparities. All groups except the Terry Collection Euro-American males maintained disparity in cause of death, continuing to die more often from infectious diseases than their local and national counterparts.

The primary expectation for this study was that a pattern of worse health over the lifecourse would be found for individuals with evidence of childhood stress – small vertebral canals, shorter femora, or hypoplasia presence would be associated with lower survival as well as with increased odds of having adult health conditions. Although none of the variables in this study met the expectation of a consistent association with worse health, the first lumbar vertebra was demonstrated to have differential survival for different canal sizes as well as a strong predictive relationship to adult health conditions for African American males. This pattern suggests that the lumbar canals may be a more sensitive indicator of early life stress and of differential
frailty than the femora and hypoplasia in this population. The lack of similar patterns for African American females, and restriction of the vertebral patterns to adult conditions for Euro-American males suggests gender and social groups difference in expression. The reduced survival with longer femora for Terry Collection African American males suggests that differential compensatory growth trajectories may be exhibiting in effect in this population. The mortality data contributes to this lifecourse analysis by highlighting the disparity in infectious disease deaths for most of the study sample groups and their city- and national-level cohorts.
CHAPTER SEVEN: CONCLUSION

There is increasing evidence that many adult health problems have their origins in childhood (e.g. Braveman and Barclay, 2009; Dowd, 2009; Benyshek, 2013; Thayer and Kuzawa, 2014). When health problems result from adverse circumstances – for example, material deprivation and physiological stress from discrimination, poverty, exclusion – or are linked more closely with disadvantaged groups than advantaged groups, health problems and health differences are disparities.

This dissertation sought to better understand the expression of adult health disparities in skeletal samples – how differences in survival, pathology load, and child growth and development might reflect societal contexts, and what, if any, consequences a given lesion may have in that context. To that end, this study used a lifecourse approach to examine the effects of adverse childhood circumstances, via skeletal data proxies, on adult health and longevity. More specifically, this research questioned whether child stress was linked with a shortened lifespan; whether certain pathological conditions accumulated in life were related to child stress; whether individuals experiencing social inequality, as suggested by unclaimed status, died from infectious disease in rates similar to those of the general population; and whether any of these
patterns exhibited disparity by sex, ancestry, and region. These relationships were explored in a late 19th to early 20th century skeletal sample of 830 African American males and females, and Euro-American males, who were unclaimed from St. Louis and Cleveland city morgues.

The study found that childhood developmental stress, indicated by small lumbar neural canal diameters, was linked with a shortened lifespan for African American males. The reduction was particularly marked for African American males from the Cleveland morgues, whose lifespan was ten years shorter than their cohorts with large canal diameters. This result was consistent with other studies that have demonstrated decreased longevity with smaller vertebral canal diameters (e.g. Clark, 1986; Watts, 2011, 2013). Terry Collection African American males also had shortened lifespans associated with smaller lumbar canal diameters, but in comparison to average diameters, not large diameters, and the age difference was 3 years. African American females had no reduction in lifespan associated with small vertebral canals. The lumbar spine results were important because they support the concepts of greater male sensitivity to adverse environmental circumstances and of greater female robusticity (Stini, 1969; Stinson, 1985; Guatelli-Steinberg, 1999). Additionally, this result points to a health disparity compared to Euro-American males, who had no reduction in lifespan with smaller canals, in either city.
Two results from the study were unexpected. First, no group had a shortened lifespan associated with the presence of enamel hypoplasia. This was surprising, because while the relationship between hypoplasia and longevity varies among studies (e.g. Palubeckaite, 2002; Wilson, 2014), the high infectious disease rates, environmental pollutants, and dangerous housing documented during this time would seem likely to select out the more frail members of society. The explanations for the lack of reduced longevity for individuals with hypoplasia include: the presence/absence method was too blunt an instrument; the canines are too sensitive to disruption, with high lesion frequencies masking an effect; hypoplasias can result from episodic stress, such as febrile illnesses, and episodic stress may not cause lasting physiological damage to the extent that chronic stress, such as malnutrition does (Vercellotti, 2014).

The second surprising result was the lack of a reduced lifespan for individuals with reduced stature, as evidenced by shorter femora. No groups had a significant decrease; rather, Terry Collection African American males had decreased longevity associated with longer femora. Reduced longevity for individuals exhibiting growth stunting is very well documented among living and archaeological populations, in many locations of the world and through time (e.g Kemkes-Grottenthaler, 2005; Steckel, 2008). A linear regression of femoral lengths with year of birth indicated secular change was occurring for African American males, but explained only about three percent of the variation; this suggests that some other processes were involved. Explanations
include: 1) rapid catch-up growth, which has a similar association with biological damage as growth disruption (Barker et al., 2011); and 2) terminal adult stature is a complicated mix of genetic and environmental factors, such that not all short stature will represent adverse circumstances or stunting and not all tall stature will represent good health (e.g. He et al, 2014; Vercellotti, 2014). Additionally, reduced longevity for shorter members of a group is not a universal finding in bioarchaeological studies and reduced longevity with shorter stature may vary within populations (e.g. DeWitte and Hughes-Morey, 2012; Watts, 2013).

Another interesting result, in terms of what it means for sex differences, was that while African American females had no relationships between child stress markers and adult accumulated health conditions, three of the four male groups did and in each case, it involved one of the vertebral canal diameters. This finding again points to greater male sensitivity to environmental circumstances and highlights the sensitivity of the vertebral canals as indicators of stress; a point made here because they are not often used in published bioarchaeological research. Similar to the inverse association with femoral length and longevity, the significant result involving the vertebral canals usually demonstrated a possibly ‘better’ outcome for individuals with smaller or average sizes, for example, less odds of having a fracture or bad oral health related to having small canal diameters for Hamann-Todd African American males, independent of age. The relationships between the vertebral canal diameters and adult health
conditions may be paradoxical, reflecting death prior to accumulation of these
conditions, considering they also had reduced lifespans associated with small canal
diameters. Conversely, for Hamaan-Todd Euro-American males, large vertebral neural
canal diameters were associated with fewer fractures; this finding was an interesting
ancestry difference and was in line with the study expectations.

Collectively, the association of the lumbar vertebra with reduced survival and
the significant relationships of differing vertebral diameters to adult health conditions
lends weight not only to the sensitive-male concept but also to the DOHaD. As the first
lumbar vertebral canal achieves approximately 65 percent of its growth by birth (Clark,
1986) and essentially is complete by age 10 (Papp et al., 1994), it encompasses many
critical or sensitive developmental periods and has less opportunity for catch-up
growth than the femur. Stunted neural canals are linked with reduced
immunocompetence. The developing vertebrae follow a growth curve similar to that of
the thymolymphatic tissue and respond to many of the same hormones, and thus
should display similar developmental disruption in settings of deprivation (Clark,
1986). In other words, if the vertebrae are stunted, some immune function likely is
damaged as well.

For the African American males in the study, the association of vertebral
diameters with shorter survival time and with adult health conditions is especially
pertinent as they also showed a very flat degenerative to infectious disease ratio over
time, not experiencing the decline in infectious disease deaths that their national and local counterparts did. Increased susceptibility to infectious disease would help explain the younger ages at death for the small diameter individuals, particularly in light of the previous discussion on increased susceptibility to tuberculosis and other pulmonary diseases related to smoke exposure, as well as to increased infectious disease exposure in crowded living conditions. This point especially is salient for the Hamann-Todd African American males; as described in Chapter 2, Cleveland experienced an extreme population increase in a short amount of time, putting pressure on housing, water, and sewage supplies and likely worsening the already terrible housing described by public health workers (Cleveland Hospital Council, 1920).

This study also provides insight into differential experiences within the epidemiological transition. A clear increase in degenerative disease deaths relative to infectious disease deaths can be seen over time for most of the national and city data. With the exception of the Terry Collection Euro-American males, none of the study groups experienced the transition over the course of the study years, highlighting a health disparity between unclaimed and their local and national counterparts. In terms of other heath disparities noted in the study, Euro-American males had a significantly higher age-at-death compared to African American males and females, possibly an effect of the younger ages of African American males and females migrating north during the Great Migration of the early to middle 1900s. Another important finding of
this study was that in addition to lending support to the concept of male sensitivity to environmental adversity, the study lends support to the concept of greater female resilience to environmental adversity, even in light of their young ages at death. The African American females exhibited no survival differences related to any child stress variable and had no associations between a child stress variable and later health.

The cause of death information demonstrated that infectious disease remained the major cause of death for the study sample African Americans, indicating they did not undergo the epidemiological transition over the study period. Euro-American males from the Terry Collection did markedly spike in degenerative disease, but the large increase over a few years was in great contrast to the more gradual trend of the local and national data, possibly suggesting sample bias. African American males and females from the study sample maintained lower degenerative to infectious ratios than the African American male and female general populations of their respective cities. This finding indicates a subgroup health disparity, wherein the study sample represents the most vulnerable members among the African American population.

Finally, this study demonstrates that research using historic data and skeletal collections can contribute to understanding health disparity. The consistency with which African American males demonstrated significant relationships involving the vertebral canals compared to African American females supports the literature indicating greater male sensitivity to environmental circumstances during growth and
development (Stinson, 1985). The reduced longevity for African American males with small L1 but not for Euro-American males with small L1, despite the poverty and adverse circumstances both groups likely faced, suggests African American males may have had greater frailty resulting from childhood stress; may have accumulated additional stress and pathogen exposure related to differential access to resources; or both. Further research using combinations of child growth and development variables may help pinpoint differential health effects based on timing of disruption and inform child health policy particularly in relation to cautions on interpreting growth standards.

**Study Limitations and Future Research**

The current study found fewer significant relationships than expected, including a lack of the well-documented relationship between shorter stature and reduced longevity. Several challenges in the research design and data analysis were encountered that may have reduced the ability to detect relationships between signs of child developmental stress and longevity, and between child developmental stress and adult health. Foremost was the problem of missing values, particularly for the vertebral diameters. The use of casewise deletion essentially created different samples for each analysis; casewise deletion does not increase bias in conditions of MCAR, but it was possible that the percentages missing were too large for this data set. Additionally, despite the large overall sample size, the small number of Euro-American males relative
to African American males and females created an imbalance that limited choice in data analysis methods. Combined with the problem of missing values, the small number of Euro-American males possibly reduced the ability to detect relationships by logistic regression and limited the number of variables that could be included in the regressions.

It is possible that the expected stress, longevity, and health condition relationships were difficult to detect due to relative homogeneity in overall poor health status. The individuals that entered the collections died relatively young and the sample had a high overall pathology burden, reinforcing the point that many of these unclaimed individuals had experienced long-term if not life-long adverse circumstances. More relationships may have been found in a group more heterogeneous in terms of social inequality. Conversely, there also could have been too much heterogeneity. Many of the individuals in the collections died during the depression; it is possible that some of these individuals had a very different socioeconomic status in childhood than they did shortly before death.

The racial categories used for the study – African American and Euro-American - also potentially were a limiting factor. Racial category was assigned at death to these individuals by the morgue or lab personnel, so it was possible that some individuals may have been classified differently in death than they were in life (Hunt and Albanese, 2005). Additionally, these individuals represent a wide U.S. population; they traveled to
their respective cities from different states of birth and represent many ethnic backgrounds and thus, even within their respective sex/ancestry subgroups, they represent different genetic backgrounds. This point is especially relevant for the femoral length data, as designations of short or long may not be reflecting differences in childhood environmental conditions but may instead be reflecting differences in genetic potential. Placing the femoral and vertebral measurements into broad size categories could have compounded that problem and it is recommended that additional investigations retain the measured data. However, the trend for a lack of significant differences in means for postcranial metrics, DMFT scores, and age-at-death between the same sex/ancestry groups lends support to treating them as a single population for this study.

The study results suggest several additional lines of inquiry into developmental stress, adult health, and survival. The most compelling finding for this study was that despite the small number of significant relationships found over the lifecourse for child stress, when they occurred they most often involved African American males and the vertebral dimensions. This finding suggests that other effects of disruption may be exhibited among the African American males in the sample. In particular, based on evidence in the recent literature (e.g., Jasienska, 2009; Kuzawa and Sweet, 2009; Barker et al., 2011), it is possible that different within-group patterns of compensatory growth may be discernable among this sample. Different trajectories of catch-up growth, and
their relationships to health and survival, can be investigated using combinations of
developmental variables, for example by identifying individuals with small lumbar
vertebral canals that have long femora. For the latter studies, the third lumbar vertebra
may be particularly useful as it shows evidence of greater susceptibility to in-utero
disruption (Papp et al., 1997; et al., 2003).

Other research suggested by the results of this study include investigating the
relationships between specific cause of death and child stress by examining the
relationships between developmental disruption variables and particular diseases
including cancer and tuberculosis. Additional research aimed at identifying within- and
between-group variation in skeletal manifestations of growth disruption may help
identify sources of health disparity by examining the differential effects based on timing
of exposure. Bioarchaeologists, having the advantage of direct dry bone observation
and measurement and access to large 19th to 20th century skeletal collections in the U.S.,
Europe, China, and Africa, are well situated to contribute to this research.


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APPENDIX A: PERMISSIONS

The photographs in Chapter Four (Figures 2 through 7) were taken by Walter Larrimore specifically for use in this dissertation and are included with his permission as indicated in the email message below.

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From: Larrimore, Walter E.
Sent: Saturday, December 05, 2015 2:02 PM
To: Coolidge, Rhonda
Subject: RE: Permission for photos

Rhonda Coolidge has my permission to use the photographs described below without limits or restrictions.

Walter Larrimore
SI Contract Photographer

Walter@LarrimorePhoto.com (non SI email)

---
From: Coolidge, Rhonda
Sent: Friday, December 04, 2015 2:15 PM
To: Larrimore, Walter E.
Subject: Permission for photos

Hi Walter,

Do I have your permission to use the photos of the enamel hypoplasia, tibial periostosis, tibia/fibula fracture, mandibular teeth, cranial fracture, and vertebra that you took of Terry Collection individuals on November 4, 2015?

According to the graduate office, in order to include in my dissertation the photos you took for me, I need your written permission. The permission can be in the form of email to be included in the dissertation appendix, hence this message.

Thank you,

Rhonda