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The Effects of Racialization on European American Stress in the Nineteenth and Twentieth Centuries

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The Effects of Racialization on European American Stress in the Nineteenth and Twentieth Centuries

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ABSTRACT

This dissertation explores disparities in stress among European Americans (EA) and between EA and African Americans (AA) in racialized communities in the nineteenth and twentieth centuries. Comparisons among EA and between EA and AA are conducted to understand the biological consequences of racialization. Racialization is the process of assigning people to hierarchical categories for purposes of political, social, and economic discrimination. This dissertation investigates how racialization might have affected childhood stress using biocultural theory and facets of critical archaeology theory. Indicators of stress from skeletonized individuals in the William M. Bass Donated Skeletal Collection, Hamann-Todd Osteological Collection, and the Robert J. Terry Anatomical Skeletal Collection are used in this study. These indicators represent non-specific childhood stress and include measures of the anteroposterior (AP) and transverse (TR) diameters of the ventral neural canals (VNC) of the five lumbar vertebrae as well as linear enamel hypoplasia (LEH) frequency data from the maxillary central incisors and mandibular canines. Historical sources contextualize this investigation.

The results of the finite mixture analysis (FMA) suggest that at least three phenotypically distinct groups of EA existed between 1828 and 1984. This study was not able to determine with certainty whether these EA groups represented particular racialized groups. Multiple analysis of variance (MANOVA) tests found a significant race effect with regard to late childhood /adolescent stress during the Early (1828-1881) period between EA and AA. AA had significantly smaller TR VNC diameters, suggesting they also experienced significantly more late childhood/adolescent stress. MANOVA tests also found significant sex effects during the Intermediate (1914-1945) and Late (1946-1984) periods.

One-way analysis of variance (ANOVA) tests showed that early childhood stress, as demonstrated by AP VNC diameter and LEH decreased over time. ANOVA tests also showed that late childhood/adolescent stress, as demonstrated by TR VNC diameter, increased over time. The findings in this study suggest that explorations into the possible effects of racialization on population heterogeneity and stress heterogeneity are warranted and should also consider the intersection of various other identities such as sex, gender, class, language, religion, and nationality.

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Chapter 1

INTRODUCTION

This study examines skeletal indicators of childhood and adolescent stress in the United States (U.S.) among racialized European Americans (EA) and African Americans (AA) in the early nineteenth to the late twentieth centuries. Comparisons among racialized groups are conducted to assess whether racialization may have led to disparities in stress. Racialization is the process of using skin color, religion, language, nationality, and other biophysical and cultural factors to assign people to hierarchical categories for the purpose of politico-social and economic discrimination (Smedley, 1998; Delgado, 2001; Martinot, 2003; Murji and Solomos, 2005; Orser, 2007). European American males, who gained hegemony in the U.S. during the seventeenth and eighteenth centuries, used racialization to claim white racial superiority and to justify political, social, and economic discrimination of inferiorly defined races (Babson, 1990; Epperson, 1996; Orser, 1998; Martinot, 2003; Agbe-Davies, 2015). However, history shows us that racial categories are not immutable, but instead experience bouts of fluidity (Roediger, 1992, 2005; Parrillo, 2000; Brander et al., 2001; Painter, 2010; Pinder, 2012, 2015). The perceived racial status of an individual affected his or her class status by circumscribing access to political, social, and economic resources (Smedley, 1998; Orser, 2007), many of which have been shown to influence disparities in childhood and adolescent stress among racialized groups in modern populations (Oliver and Muntaner, 2005; Jacobs et al., 2009; Krieger, 2010; Kershaw et al., 2011).

The fluidity of racial identity is brought to light through changes in the assignment of individuals to either the white ethnic or white racial category over the last few centuries. White

ethnic identity has been assigned -- historically and in the present-day -- to all individuals perceived to be of European descent because they were thought to belong to one of the four Caucasian or white ethnic divisions: non-Aryan, Aryan, Semitic, and Euskaric (Dillingham et al., 1911; McDermott and Samson, 2005). Nordic groups, English, Scots-Irish, and some old (individuals who migrated to the U.S. before the nineteenth century) Germans were classified as racially white from the seventeenth to the nineteenth centuries (Parrillo, 2000; Black, 2003). A white racial identity entailed the perception that one was biologically and intellectually superior to all other white ethnics and nonwhites. In fact, white ethnics were considered nonwhites racially. Nonwhite European descent groups were non-Protestants, Southern and Eastern Europeans, and were gradually assimilated into the white race through the process of racialization during the nineteenth and first half of the twentieth century (Roediger, 1992, 2005; Black, 2003), with some groups assimilating more quickly than others. The rate of assimilation depended on the political, social, and economic environment in which these nonwhites immigrated and their acceptance of old immigrant Protestant Euro-American (i.e., English, Scottish, Scots-Irish, Protestant Germans) cultural norms (Parrillo, 2000; Roediger, 2005). Knowles and Prewitt (1969) also suggest that assimilation was possible because these groups had gradual access to naturalization, citizenship education, political power, suffrage, and ultimately the right of self-determination. Assimilation came with increasing access to political, economic, and social resources, all of which are related to health disparities (Jacobs et al., 2009; Oliver and Muntaner, 2005; Krieger, 2010; Kershaw et al., 2011).

The unacknowledged assumption in most current/modern research is that European immigrants and U.S.-born European Americans from all over Europe were considered white racially. The nuanced racial classification among these groups at various times in U.S. history is

not mentioned or methodologically explored in many studies using collections comprised of people of European descent (e.g., Jurmain, 1977; El-Najjar et al., 1978; de la Cova, 2011). Consequently, European descent has been analytically used as synonymous with "white" racial status and/or health status, resulting in the erroneous conflation of all European descent groups into the white racial category.

The initial exclusion and gradual inclusion into the white race of nonwhite Europeans might have affected the health of these populations through resource discrimination. Yet, this variation is masked by the common practice of putting nonwhites into the white category with little consideration of their actual racial status as historically documented. Thus, the inclusion of these groups with white racial groups might portray a picture of white racial health that is far from accurate, especially prior to WWII and before "white" became an all-inclusive racial category for anyone of European descent (Roediger, 2005). To better document health patterns among European immigrants and their descendants, it is necessary to account for changing and diverse racial statuses among these groups as health disparities fall largely along racial lines. Studies like Dillingham et al. (1911) and Edgar (2009) demonstrate that rates of assimilation among groups shift through time, and that folk conceptualizations of race (which conflate biology with ethnicity) impact mating choice and thus gene flow. How cultural conceptualizations of race impact economic opportunities and health over time, however, remains largely unexplored in biological anthropology.

This dissertation explores the contribution of heterogeneous groups of racialized European Americans to health disparities at various times in U.S. history. This dissertation suggests that more diversity existed among European Americans in racial categorization in the nineteenth and twentieth centuries than in the present. European Americans, who have typically been

categorized as “white” racially by modern anthropologists (e.g., Jurmain, 1977; El-Najjar et al., 1978; de la Cova, 2011) might in fact include several racialized groups of European Americans with unique experiences. Groups perceived as racially distinct would have been isolated through the enforcement of racist structures (laws, policies, cultural norms, customs, and beliefs) meant to ensure political, economic, social, and even reproductive segregation (Pascoe, 1996; Roseman, 2014). Reproductive segregation was enforced through custom and miscegenation laws (Pascoe, 1996; Martinot, 2003; Agbe-Davies, 2015). Roseman (2014) argues that the regulation of reproduction by racisms (referred here as racist structures) in historic and contemporary populations has shaped genetic and possibly phenotypic variation in ways that correlate with racialized groups.

To better capture the childhood and adolescent stress experiences among racialized groups in the nineteenth and twentieth centuries, possibly hidden racialized breeding groups amongst European American samples must be found and examined separately. To this end, non-metric cranial traits from European American skeletal samples are examined using finite mixture analysis (FMA) to illuminate phenotypically distinct groups that quite possibly represent distinct racialized groups. This method, as explained in more detail in Chapter Four, categorizes individuals into groups based on the variation in morphology. This method does not force groups; if only one group exists, FMA will only identify one group. This study assumes that racialization created structure in mate choice and reproduction, and thus phenotypic or morphological variation. For more on regulations on reproduction and genetic and phenotypic variation in racialized communities see Chapter Three.

It is important that diverse racial statuses not be ignored because perceived racial status affected the access of individuals to resources (Smedley, 1998; Orser, 2007), resources known to

influence health disparities in stress (Oliver and Muntaner, 2005; Jacobs et al., 2009; Krieger, 2010; Kershaw et al., 2011). Health disparities are differences in morbidity, mortality, and access to health care among populations (Dressler et al., 2005). For almost 200 years after the U.S. became a sovereign nation, health disparities between racial groups were considered a function of intrinsic differences in biology (Charatz-Litt, 1992; Pernick, 1997; Goodman, 2000; Bryd and Clayton, 2001; Weiss and Lambert, 2011; Krieger, 2011). Systematic and systemic cultural prejudices classified racial groups into a hierarchy where white races (i.e., Nordic groups, English, Scots-Irish, and Germans) were seen as racially superior to all nonwhites, namely non-Protestants, Southern and Eastern Europeans, Native Americans, African Americans, Asian Americans, and Latin Americans as well as Africans and Asians (Blakey, 1988; Jones and Carter, 1996; Dinnerstein et al., 2003; Pinder, 2012). In conformity with these prejudices, the apparent better health of whites simply reflected a superior biology, whereas the poor health of nonwhites was due to their inferior genes and overall inability to cope with their environment (Krieger, 2011). Such conclusions perpetuated the use of biology to distinguish racial groups, despite the confounding fact that discrepancies in the quality of the social, economic (occupational), political, and biological environments of racialized groups co-varied with apparent distinctions in resilience (Lucas, 1974; Richardson et al., 1997; Gravlee, 2009; Krieger, 2010; Williams et al., 2010; Kershaw et al., 2011; Asada et al., 2013; Leatherman and Jernigan, 2014).

"Biology," then, was (and continues to be) used to create, perpetuate, and maintain a form of structural racism comprised of laws, policies, norms, and practices that served to reflect, maintain, and perpetuate racial inequalities (Jones, 1972; Stern, 2005; Weiss and Lambert, 2011; Pinder, 2012). This use of biology to structure inequalities resulted in the concept of a white

racial superiority. To support structural racism, cultural racism was propagated by scientists and educators to ensure that groups deemed to be "nonwhites" would be perceived as having an inferior and subordinate culture and status within mainstream white America (Jones and Carter, 1996; Orser, 2007; Painter, 2010). Thus, government policies were devised to maintain political, social, and economic inequality instead of affording all individuals equal opportunities. This inequality along racial lines has persisted into modern-day United States society (Brundage, 1993; Franklin and Moss, 1994; Christian, 1995; Davidson, 2004; Ward, 2005; Krieger, 2011).

There is extensive documentation of health disparities between those who we contemporarily classify as African Americans (perceived as black racially) and European Americans (perceived as white racially) (Lucas, 1974; Robinson, 1984; Richardson et al., 1997; Friedman-Jimenez and Claudio, 1998; Loomis and Richardson, 1998; McCarthy, 2000; Davidson et al., 2002; Oliver and Muntaner, 2005, Chung-Bridges et al., 2008; Krieger, 2010; Kershaw et al., 2011). These studies demonstrate that the perpetuation of structural and cultural racism has contributed greatly to the persistence of racial health disparities through the twentieth and into the twenty-first centuries. Racial distinctions are reinforced by structures set in place by dominant groups to maintain environmental inequality, and this systematic inequality has biological consequences that affect health and stress (Krieger, 2001, 2011) in dominant and non-dominant groups. Structure (norms, institutions, laws, roles, and policies) organizes, mediates, constrains, and/or enables human agency (Shanks and Tilley, 1987; Cockerham, 2005; Saitta, 2007). Thus, structure may severely restrain choices in groups deemed inferior due to their racialization. As the human body interacts with its external environment it embodies health determinants related to the peculiar social, political, economic, and biological structures of that environment (Krieger, 2011). In short, cultural perceptions about biological differences between perceived racial groups

have created disparities in stress that distinguish them on account of the environments in which they live.

Despite the established relationship between structural and cultural racism and health disparities, scholars continued to insist that racial classifications could be supported with biological evidence. Garn (1971) attempted to define race in humans using the definition of "race" commonly employed in the non-human biological disciplines. He regarded races as major to minor subdivisions of *Homo sapiens* that included geographical, local, and micro subdivisions. However, Garn's (1971) description of races concealed the evolutionary relationships between populations (a point made by Wahsburn [1964]) and did not account for the fact that no isolated population conforms to any particular racial group. Consequently, no set of traits are fixed in a given population; there is instead continuity between groups (Montague, 1964). Furthermore, differences within races have been shown to be greater than the average differences between races and individuals have been shown to differ continuously between racial groups (Osborne, 1971; Lewontin, 1972; Templeton, 1999). Thus, the ultimate unit is the species, and subdivisions made within the human species are arbitrary because there is too much continuity and local variance to divide groups into subspecies (Howells, 1971; Goodman, 2000).

However, although genetic variation does not support the existence of race, genetic variation has been affected by the existence of structural racism over the last two centuries in communities with extensive histories of racialization (Roseman, 2014). Roseman (2014) argues that racisms [racist structures] regulated and continue to regulate gene flow by restricting mating choice and maintaining status heritability through customs and statutes. This regulation has produced correlations between genetic and phenotypic variation and racially defined groups. Accordingly, structural racism not only produces disparities in stress, but it also produces genetic variation

along racial lines in some communities, both products of which are biological consequences of racialization (Gravlee, 2009; Edgar, 2009; Roseman, 2014; see Chapters 2 and 3).

This study problematizes modern conceptions of whiteness by attempting to illuminate evidence of racial heterogeneity among European Americans in historic and contemporary skeletal samples. Non-specific markers of childhood and adolescent stress (i.e., markers of stress with several possible etiologies) are then explored and compared between distinct groups of racialized European Americans and African Americans. This study uses a biocultural model of stress to situate patterns of stress in their social, political, and economic context. A biocultural model holds that the cultural environment is the both the source of stress and of resources that avert stress (Goodman and Leatherman, 1998). Non-specific indicators of stress (e.g., vertebral neural canal size variation and linear enamel hypoplasias) are argued to be biological responses to short or long term stressors (social, political, and economic events) that cause inequalities and/or limit resources.

Whiteness is thus problematized in two ways, first by examining possible heterogeneity amongst historically racialized European Americans, and second by examining possible heterogeneity in stress experiences amongst these groups. Not all European Americans were racialized into the white race in the nineteenth and twentieth centuries. Thus, European American experiences should be critically examined and not blindly examined under the modern shroud of "whiteness."

Racialization acted to segregate and discriminate along multiple facets of identity including religion, language, nationality, race, and class. The intersection of these various identities mutually shaped (Walby et al., 2014) the experiences of racialized groups. This study does not make the assumption that one identity is more relevant than any other. Rather, racialization is

addressed in this research because it inherently incorporates multiple identities by using them (i.e., religion, language, and/or nationality) to place populations into racial identity groups (Martinot, 2003; Agbe-Davies, 2015) with predetermined class (identity) restrictions. Gender is also addressed as environmental and physiological stress might have been experienced differently amongst racialized males and females. The aim of this study is not to determine which groups were considered white or nonwhite European Americans, but to examine as many racialized experiences as a possible with the hope of illuminating alternative childhood and adolescent experiences. This study aims for a more holistic analysis of inequality on human experience by investigating, rather than masking, racially distinct European American experiences that have been traditionally subsumed into dominant white narratives and minimally addressed by biological anthropologists. Highlighting these alternative experiences creates a more inclusive history, one that strives to more accurately depict diverse race affiliations among European American populations.

As stated above, structural racism, one of the instruments employed in the process of racialization, can produce genetic variation and disparities in stress along racial lines in racialized communities (Gravlee, 2009; Edgar, 2009; Roseman, 2014). In this dissertation, I search for these biological products of racialization in historic and contemporary populations. I initially employed discriminant function analysis (DFA) to determine whether a heterogeneous group of European Americans existed from 1828-1984. However, DFA performed so poorly that finite mixture analysis (FMA) was employed instead to determine group membership. The groups identified by FMA were used in the analysis of childhood and adolescent stress in this study. Lumbar vertebral neural canal diameters (VNC) were measured and linear enamel hypoplasias (LEH) were counted in order to asses stress among groups. VNC diameters are good

indicators of neural and immune development during early childhood and late childhood/adolescence (Clark et al., 1986; Porter et al., 1987; Clark et al., 1989; Jeffrey et al., 2003). LEH represent deficiencies in enamel secretion (i.e., amelogenesis) during development (Guatelli-Steinberg et. al., 2012). They usually appear as horizontal pits or grooves along the surface of enamel.

In an effort to explore whether a European Americans were heterogeneous through time and whether certain groups experienced different levels of stress, I ask the following questions:

1. Are there distinguishable groups based on cranial trait variation within the sample of European Americans? What role might racialization play in shaping these distinctions?
2. Do these groups remain constant in size and proportion over time? If not, how do they change? What might explain these changes?
3. Do phenotypically distinct groups of European Americans have similar distributions of childhood and adolescent stress (LEH and VNC size) temporally?
4. How do trends in stress among racialized European American groups relate to trends in stress among African Americans? What might explain these relationships?

The samples in the study are divided into four birth cohorts that represent differing levels of racial tension. These cohorts are Early Period (1828-1881), Middle Period (1882-1913), Intermediate Period (1914-1945), Late Period (1946-1984). The Early Period (1828-1881) begins with the earliest individuals' birth date in the collection. This period represents a time when Europeans primarily migrated from Western Europe (Dillingham et al., 1911). With the exception of the Irish who migrated to the United States during this period (Orser 2007; Brighton 2008, 2009), there were few resource threats from other European groups considered nonwhite, so this period is considered a period of minimal racial tension among Europeans and European

Americans. During the Middle Period (1882-1913), from 1882 to 1902, there was a decrease in immigrants from Western Europe by 75% and an increase in immigrants from Southern and Eastern Europe by 475% (Dillingham et al., 1911). Furthermore, 70% of the 13 million Southern and Eastern Europeans migrating to the US between 1886 and 1925 arrived between 1901 and 1915 (Roediger, 2005). By ending the Middle Period in 1913, I capture a large part of this migration while simultaneously leaving out the fluctuations in migration and acceptance beginning with World War I in 1914. This period is considered a time of increasing racial tension as white European Americans began to compete more frequently with nonwhite Europeans and European Americans for resources. The Intermediate Period (1914-1945) starts at the beginning of World War I, spans the Great Depression, and ends at the end of World War II. This period represents a time of changing acceptance as white for certain European and European American groups in light of rivalries during the wars and resources stress during the depression. Racial tension likely fluctuated during this time period. The Late Period (1946-1984) begins with the end of World War II and ends with the latest individual birth date in the skeletal sample used in this dissertation. This period represents a time of decreasing racial tension and increasing acceptance as white for all Europeans and European Americans (Knowles and Prewitt, 1969; Clark and O'Donnell, 1999; Roediger, 2005).

I have five expectations in this study that relate to the above questions. First, I expect EA in the Early Period (1828-1881) to be more homogenous or mostly consist of one group as the great migration of Eastern and Southern Europeans did not begin until 1882 (Dillingham et al., 1911). Second, I expect to see three distinct European American groups during the Middle Period (1882-1913) because of the migration of millions of Southern and Eastern Europeans during this period (Dillingham et al., 1911; Parrillo, 2000; Roediger, 2005). Third, I expect to see a decrease

in some groups and in increase in others during the Intermediate Period (1914-1945) because this was a time when acceptance of Southern and Eastern European migrants was burgeoning (Roediger, 2005). My fourth expectation is that European Americans will be mostly comprised of one group during the Late Period (1946-1984) due to "white" becoming an umbrella term for all individuals of European descent (Knowles and Prewitt, 1969; Clark and O'Donnell, 1999; Roediger, 2005). Last, I expect to see significant differences in the temporal distribution of indicators of childhood and adolescent stress among racialized European American and African American groups as racial classification mediates access to resources that affect health.

The following chapter outlines race as a solely socially constructed phenomenon with real political, social, and economic consequences. Chapter Three demonstrates how the process of racialization also affects human biology, particularly genetic variation and physiological stress. Chapters Four and Five outline the materials, methods, and results of this study. Findings are discussed in light of the experiences of racialized groups in the U.S. in Chapter Six. I also point out the limitations of these findings. I conclude by discussing the implications of this study for studies looking to problematize whiteness and/or address disparities in stress in racialized communities. I also discuss future endeavors and questions that might enhance and extend studies of this sort.

Chapter 2

THE ANTHROPOLOGY OF RACE AND RACIALIZATION: SOCIOECONOMIC CONSEQUENCES

To understand the effects of racial identity on disparities in health, we must first establish the processes that shape and maintain racial categorization, and their sociocultural consequences. This chapter provides a general overview of the history behind the establishment of the race concept, racial groups, and racialization. While this research is undertaken from an anthropological perspective, which is often focused on the cultural and biological definitions of race, it is important also to realize that social policy enacted on account of racial beliefs in turn reifies those cultural and biological definitions. I review the interaction of racialization with political and social policies. I also discuss how race is defined, and how groups identified other races from the late seventeenth century to the twentieth century. Problems determining race and defining race are also explored in light of biological anthropology and archaeology.

Defining Race

Humans have been aware of similarities in hair form, eye color, skin color, and face shape within various groups for millennia (Painter, 2010). There is no doubt that understanding differences and similarities among past and present populations is important in the reconstruction of prehistoric human interactions and migration patterns (Brues, 1990). Racial classification, however, has been primarily used by various groups to segregate and rank populations, rather than understand their evolution (Babson, 1990; Smedley, 1998). Race as a classifying and ranking agent emerged between the fifteenth and eighteenth centuries when ranking and

typology were at their height in the largely descriptive sciences (Marks, 2010; Brace, 2010). The term "race," however, flourished when Samuel George Morton swapped *varieties* for *races* in his translation of Johann Friedrich Blumenbach's 1775 dissertation "On the Natural Varieties of Mankind" (Brace, 2010). Blumenbach, however, did not consider his varieties as races (with the purpose of segregating and ranking populations), but rather as arbitrary divisions of the human species. Morton, in establishing the American School of Anthropology (as well as helping to found American invertebrate and vertebrate paleontology) promoted the existence of discrete races, with each race having distinct physical and mental capabilities (Stanton, 1960; Abrahams, 1966; Brace, 2010). The racism inherent in this school was most infamously used to justify slavery in the U.S. (Brace, 2010).

Smedley (1998:690) defines race as a social construct that "emerged as the dominant form of identity in those societies where it functions to stratify the social system." Although biological correlates to race are mutable and superficial, and are subject to evolutionary convergence in different groups, the term "race" proposes that one's racial status can be determined simply by looking at physical and physiological features and culture. Racial identity ties an individual's physical characteristics to social meaning meant to marginalize along racial lines. Racial identification therefore rests in social, political, and economic tensions that create iniquities. As Smedley (1998) emphasized, culture is learned behavior. It follows that racializing people is also learned behavior and contributes to the continual reification of race across generations. Racial worldviews prompted by very powerful social, political, and economic forces have perpetuated the idea that biological heredity corresponds with racial categories.

Race is not to be confused with ethnicity. Ethnic groups are defined as demarcated social entities that ascribe restricted membership to individuals, which in turn allows them to confine

their interactions with other members of the group who share similar worldviews, lifestyles, languages, and history distinct from other groups (Orser, 1991; Smedley, 1998). Though both ethnicity and race act to separate groups, they do not serve the same purpose within a society and they affect individuals differently (Orser, 1991, 1998, 2007a). The key distinction between ethnicity and race is that ethnicity is self-imposed—it is a product of within-group identity. Ethnicity establishes belonging and camaraderie among individuals with authentic cultural associations. Race, on the other hand, is an imposed category, most often created by Europeans to delineate groups that could be readily identifiable by phenotypic features (e.g., skin color) and/or cultural practices, religion, traditions, and ethnicity (Orser, 1991, 2007a; Epperson, 1996). Race is destructive; it divides individuals based on perceived biological and cultural differences. Other cultures, such as the Japanese categorization of Koreans or Ainu, also practiced forms of racialized hierarchies (as argued by Hudson, 1999). The European act of racial categorization had far-reaching impacts across the globe over centuries, and is the subject of this dissertation's study. As the phenotypic and cultural markers used to identify racial groups change, the racist paradigm incorporates those changes in order to maintain the social hierarchy (Orser, 2007).

Race therefore ranks groups on a hierarchical scale for the purpose of ascribing a subhuman status to groups deemed inferior (Babson, 1990; Smedley, 1998). Racial categories are inherently ranked, and are not simply ways to demarcate groups as "other." Both Epperson (1996) and Orser (2007) argue that races were created to establish a system of domination, prescribing sociocultural power relations. As this study focuses on the impact of the European promulgation of race, we cannot separate racial categorization from colonialism, capitalism, and imperialism. The establishment of hierarchies was essential to the operation of these economic processes (Nassaney, 2007).

Thus, categories of race trace their roots to European colonialism and Western practices of naturalizing systems of hierarchy and domination (Epperson, 1996; Martinot, 2003; Brace, 2005; Nassaney, 2007) through reference to physical traits (Orser, 1991; Smedley, 1998). Races were distinguished by the presumably high frequency of certain arbitrary traits in one population as opposed to another population. The assumptions were that these traits were largely genetically founded (as opposed to environmentally founded) and that they did not group themselves independently. The implication of these assumptions was that racial groups were fixed, allowing for total genetic discontinuity between races and consequently the existence of pure races (Goodman, 2000; Templeton, 2013).

Race in Biological Anthropology

Race at the Beginnings of the Discipline

Late eighteenth- and early nineteenth-century physical anthropologists (a discipline later recast as "biological" anthropology) distinguished themselves and the field by their focus on the origins of human populations, the relationships between populations, and human variation (Cravens, 1978; Little, 2010; Giles, 2010; Ortner, 2010; Marks, 2010; Caspari, 2009). That race would play a prominent role among anthropologists in the United States and Europe is not surprising. Anthropologists inconsistently partitioned mankind into typologies or races to explain relationships and human variation (Brace, 2010; Painter, 2010; Marks, 2010), ultimately tying these ideas to human evolution. A typology is a classification system used to "sort entities into mutually exclusive categories" (Adams, 1988:43). The obsession with racial typologies amongst anthropologists in the U.S. was in part due to the fact that they lived, trained, and worked in a society economically, politically, and socially structured along racial lines and deeply grounded

in racist ideologies supporting white supremacy beginning with slavery and continuing through the Civil Rights movement. Growing support of Galton's Eugenics movement (discussed in detail below) as well as nationalist racism in Europe in the late nineteenth- and early twentieth-century made race a central focus among European (and U.S.) anthropologists (Marks, 2010; Weiss and Lambert, 2011). Many anthropologists dedicated their energies to confirming racist ideologies and supporting racist agendas (Brace, 2010). Most also believed (with the exception of Franz Boas and a few others) that races were originally pure and dissimilar and that any gradations between alleged pure races were assumed to be mixtures of previously pure races and not consequences of human variation across geography (Cravens, 1978).

Both Aleš Hrdlička, the founder of the *American Journal of Physical Anthropology*, and Earnest Hooton, the first anthropologist to produce PhDs in physical anthropology (Giles, 2010; Caspari, 2009), believed in the existence of pure biological human races. They were racial formalists and thus overly concerned with constructing racial typologies and assessing racial morphologies (as opposed to studying evolutionary models or growth and development as sources of variation) (Allen, 1989). Hrdlička believed that the study of human racial groups was necessary for understanding human variation, primarily among inferiorly perceived races. Like most biological scientists in that era, both Hrdlička and Hooton were eugenicists who believed in using anthropology to advance the human species (Marks, 2010; Caspari, 2009). Eugenics, a termed coined by Sir Francis Galton and professionalized as a field and movement by Karl Pearson and Ethel M. Elderton in the late nineteenth- and early twentieth-centuries, was a means by which perceived superior races, through social policy, could ensure their genetic dominance in future generations by selectively limiting the ability of races perceived as inferior to contribute genetically to ensuing generations (Black, 2003; Largent, 2008; Weiss and Lambert, 2011).

Professionals trained in the humanities, biology, genetics, and statistics, both in the United Kingdom and in the United States, promoted eugenic research (Black, 2003; Weiss and Lambert, 2011). The intended result of eugenic research was to develop policies that aided the rapid and continual advancement in health, fitness, intelligence, and culture of the human species through racial purification (Cravens, 1978; Black, 2003; Larson, 2004; Ortner, 2010).

Not all early physical anthropologists were eugenic hardliners. Unlike Hrdlička and most eugenicists, Hooton did not adhere to intellectual determinism as a component of the inherent biological determinism of eugenics. To most eugenics researchers, this determinism insisted that genetics determined biological, intellectual, and cultural achievement, as well as one's particular inclinations (Cravens, 1978; Larson, 2004; Ortner, 2010). Instead, Hooton believed mental capacity was not diminished by the interbreeding of the races and that no one race had an intellectual advantage over another (Giles, 2012). Hooton's eugenic stance was unique, but he fell in line with most negative eugenicists who targeted the biologically unfit—based largely on culture and intellect—for segregation and sterilization (Giles, 2012). Positive eugenics based fitness on racial status, looking for methods to encourage certain racial groups to have more offspring than others.

Franz Boas, on the other hand, challenged racial categorization and opposed eugenics (Goldstein, 1948; Cravens, 1978; Caspari, 2009). He is best known as the anthropologist who advocated for the four-field approach to anthropology which included archaeology, as well as cultural, linguistic (with an emphasis on folklore), and physical anthropology (Cravens, 1978; Little, 2010). His research contributed to the critique of biological determinism, when he studied the impact of heredity and environment on human variation. With the financial backing of the U.S. Immigration Commission, from 1908 to 1910, Boas and his team set out to measure head

shape among immigrants and their immigrant- and U.S.-born offspring (Boas, 1912; Gravlee et al., 2003a, 2003b; Caspari, 2009). Boas' study found that changes in the cephalic index (skull width divided by skull length) and facial shape correlated with changes in environment. His findings demonstrated that one of the most prominent traits (i.e., the cephalic index) for discriminating races was subject to plasticity or changes resulting from environmental influences across generations. If the physical determinants of race could change, then it followed that the categorizations of races were also mutable. In short, Boas' findings challenged biological determinism by suggesting that races were not fixed (Boas, 1912; Gravlee et al., 2003a; Little, 2010). Yet, it was not until advancements in molecular genetics and statistics that hypotheses concerning the genetic and biological bases for races could be tested.

Revisiting Race in the Mid- to Late Twentieth Century

Starting in the mid-twentieth century, geneticists and, later, molecular anthropologists began to theorize about race and apply genetics to assess whether the concept of race is supported biologically. Much of the foundational arguments that still drive biological anthropologists were set in the 1960s. Livingstone (1962) suggested that races do not exist, only clines. Clinal theory argues that differences in a measurable genetic character or trait within groups are often correlated with gradients in climate, geography, and ecology. According to Livingstone, races are no more than statistical combinations of adaptive traits that have geographical gradients of skin tone, hair texture, face shape, and so on. He stressed the fact that there is great genetic variation within perceived racial groups and overlapping variation between groups. This variation is due to natural selection acting on individuals in their geographical environments, and

that many traits ascribed to races in fact arose in multiple groups by convergence. Thus, according to Livingston (1962) perceived races represent local limitations in human variation.

Montagu (1964) further asserted that race in the light of genetics was meaningless. "Race" was purely arbitrary when used by eighteenth-century naturalists Georges-Louis Buffon and Carl Linnaeus to distinguish between geographic groups of people. In Montagu's (1964) view, the anthropological belief that races are composed of individuals that collectively have characteristics that distinguish them from other individuals is unlikely. Racial groups vary within themselves and in comparison to other groups with regard to gene frequency, yet there is always continuity between groups. Characters are neither fixed nor transmitted as complexes but behave as expressions of many independent units. Therefore, it is absurd to establish a race by averaging characteristics, generalizing groups, and suppressing variation by only focusing on certain characteristics. Montagu concluded that it is obvious that differences exist between groups, but the concept of race is still very artificial; it does not agree with biological facts and leads to confusion and perpetuation of error. Furthermore, race carries with it physical type, heredity, blood (as a cultural measure), culture, nation, personality, intelligence and achievement, many of which are not biological traits, and all of which can result in discrimination (Montagu, 1964). This is the reason that, in the 1990s and 2000s, some scholars argued that the term race should be thrown out altogether (Brown and Armelagos, 2001).

Taking a contrary position, Washburn (1964) asserted that races do exist as a result of human evolution and local forces. However, races are insignificant in light of the evolution of the human species as a whole, and it is impossible to study human races without addressing human culture. Washburn (1964) argued that races result from thousands of years of genetic mutations, natural selection, migration and genetic drift, all of which, except for mutation, are in some way

affected by culture. Migration and gene flow, with respect to racial differences, act to reduce differences between groups. Genetic drift, on the other hand, acts to create differences between groups while establishing more similarity within groups. The strength of selection relies on reproductive success, which among all populations is linked to culture and social systems regarding mating habits. Migration depends on culture, and drift depends on population size, which in turn depends on culture. Washburn (1964) suggested that selection, over a long period of time, is the primary source of racial differences. Thus, the origin of races must depend on adaptations, and the differences between groups must have been adaptive at some point in their evolutionary past.

He further noted that typology and classification must be removed from our thinking in order for our studies and culture to progress. There are no primary or major races and the idea of such races stems from eighteenth- and nineteenth-century typology. The perception of race would change if it were addressed with selection, migration, and drift in mind. Furthermore, because races are open systems, the number of races should depend on the purpose of classification (Washburn, 1964). Washburn (1964) argued that there was no purpose for the biological classification of races. If one insisted on classifying races he or she should have to give important reasons for doing so, such as in examining the effects of racial classification on biology; such studies are narrow compared with the breadth of misapplications of race in other topics, including analyses of intelligence (Cravens, 1978). Also, racial classification tells us very little about evolutionary conditions and relationships. Understanding the role of evolution in shaping human variation, instead of documenting races, should be the focus of physical anthropology. He pointed to the fact that “Mongoloids” were once thought to have a very cold-adapted face when in fact many of them lived in tropical environments. In summary, Washburn

(1964) asserted that the classification of races is based on several misunderstandings and misinterpretations of how traits arise in populations. Given his influence on the discipline (Washburn, 1951; Stini, 2010), Washburn's perspectives, along with Livingstone's arguments for clines, guided the entire next generation of biological anthropologists in their perspectives on race and its relationship with biology.

Howells (1971), in his own attempt to describe race, also focused on explanations in light of genetics. He argued that every individual is made up of a unique combination of existing genes, and that, following selection, genes favored by the environment will become more frequent in the gene pool. Every population differs in some aspect of their gene pool. So, the genetic structure and gene pools of different populations are distinct in gene frequencies and gene combinations, both of which are displayed through the visible features of their members (Howells, 1971). Though Howells attempted to describe diversity within and between populations, he makes it clear that the lowest unit of analysis among humans is the species. Subdivisions within the human species are arbitrary constructions because too much continuity and local variance exist to divide groups into subspecies.

Dobzhansky (1971) further asserted that individual populations of Africans or Asians differ just as much, if not more, among themselves than the populations of Europe. And although he did believe that race was a valid category, he reemphasized that dividing peoples into races suppresses the variation anthropologists seek to explain. He then pointed out that biological species are genetically closed systems and races are genetically open systems. Gene flow as well as phenotypic and developmental plasticity can blur race boundaries (Dobzhansky, 1971).

Perhaps races are breeding populations that differ from other populations in the frequency of one or more genetic traits (Livingstone, 1964). An issue arises from this perspective: at what

point do you establish that group “A” is a different race than group “B”? What traits are regarded as important traits when trying to establish races? These questions point to the fact that it is up to the observer to determine the importance of some traits over other traits (Brown and Armelagos, 2001). Such subjectivity is on display in the work of later twentieth-century anthropologists.

Garn (1971) regarded races as major to minor subdivisions of the species *Homo sapiens* in his attempt to define race in a way that would seem biologically sound. Geographical race, a major subdivision, was defined as a collection of populations with common physical features resulting from a common ancestry which extended over a geographically definable area. He would consider the “Negroids” of Africa and the “Caucasoids” of Europe as geographical races. Examples of local races, or minor subdivisions within geographical races, include the "Pygmies" and the "Alpines." Following and within local races are micro-races, the smallest subdivision of race, which represent tribes or villages. Of all racial groupings, geographical races were used until the 1990s, mostly because they were the easiest to distinguish. However, Garn’s (1971) description of the races did not attempt to reveal evolutionary relationships between groups. He only classified groups based on typological and geographical similarities. Under his classification system, Native Americans and Eastern Asian populations would be deemed different geographical races regardless of the fact that Native Americans descend from ancient Asian populations, and thus are genetically nested within Asian variation. Moreover, this perspective—on large geographical races—inherently undermines the focus on variation, as it homogenizes and stereotypes morphological characteristics.

Geographical races, according to Howells (1971), equate to the zoological term "subspecies." Each subspecies should be distinguishable from another subspecies by the possession of certain distinctive hereditary traits (Montagu, 1964). According to Howells (1971), for a population to

be deemed a subspecies it must differ from another group within the same species by 75%. Yet, this percentage is an arbitrary standard. Moreover, there are no standardized traits to choose from when attempting to delineate races among humans (Templeton, 2013; Goodman, 2000). For instance, Australian Aborigines and Southern Indians all have a dark skin color that is typically attributed to sub-Saharan Africans. This may be due not only to the similar environments shared between these populations, but also to the fact that no populations exist in complete isolation and have a shared evolutionary history. Consequently, no traits or set of traits are fixed to a given population. When populations come in contact with one another they not only exchange knowledge and goods, but genes as well. This continuous gene flow between populations makes it very difficult to define races (Goodman, 2000; Hunley et al., 2016).

Does the biological definition of subspecies equate to Garn's geographical race? Studies continue to show that differences within races are greater than the average differences between races for many characteristics, and individuals have been shown to differ continuously between races (Osborne, 1971; Lewontin, 1972; Ryman et al., 1983; Dean et al., 1994; Templeton, 1999; Goodman, 2000; Brown and Armelagos, 2001; Templeton, 2013; Hunley et al., 2016). Lewontin (1972) supported the argument against biological races. He analyzed the distribution of 17 genes within 7 populations deemed racial groups (Black Africans, Caucasians, Oceanians, South Asian Aborigines, Native Americans, Mongoloids and Australian Aborigines) and found that the mean proportion of the total species variation that is accounted for within populations is 85.4%, with a maximum of 99.7% and a minimum of 63.6% (Lewontin, 1972). Thus, only about 14.6% of all human genetic variation is between perceived racial groups. He also noted that differences between populations within a race only explained 8.3% of the 14.6%; the other 6.3% was accounted for by racial classification.

Templeton (1999) pointed to further evidence supporting the assertion that races or subspecies in human populations do not exist. He examined the F_{st} statistic from 16 human populations to determine whether differentiation between human races fit the widely accepted requirement for other animals of an F_{st} from 0.25 to 0.30 (Smith et al., 1997). He found the F_{st} value for humans to be 0.156, which is not high enough to establish human subspecies along the same lines as other animal subspecies are assessed. Accordingly, about 15.6% of all human genetic diversity is between perceived racial groups, leaving 84.4% of all genetic diversity to be accounted for within groups. Templeton followed up this study with another in 2013 using 52 populations. Using the F_{st} threshold again, he examined the five major geographical groups of humans to assess if those would be recognized as subspecies in other animals. He found only 4.3% of genetic variation between groups, well below the accepted 25-30% subspecies threshold. It must be noted that the selection of traits to determine whether subspecies exist is arbitrary. Different traits might produce different F_{st} values, hence the debate over whether "subspecies" are valid categorical or evolutionary units (Smith et al., 1997). Nonetheless, Templeton (1999; 2013) showed that human populations that might be divided into races (i.e., subspecies) based on geography or appearance still prove to be more alike than different.

However, some scholars point to Rosenberg and colleagues' (2002) study to refute Templeton's (1999; 2013) findings. Rosenberg and colleagues (2002), using several genetic markers from 52 human populations, discovered that when they sought to sort humans into five populations, they sorted in groups corresponding to geographical races. Yet, when they sought to divide humans into more than five groups they were able to distinguish between smaller local populations. So are there five, ten, or twenty races? Rosenberg and colleagues' (2002) study

points to the fact that given an extensive sample of genetic markers, humans can be divided into many discrete units representing breeding populations from the geographical to the local level.

The Persistence of Race in Biological Anthropology

Despite all of the evidence supporting the arbitrariness of race, as well as calls by some biological anthropologists to abandon the use of race as a concept altogether, some geneticists and anthropologists (Nei and Roychoudhury, 1982; Harpending et al., 1996; Miele and Sarich, 2005) continue to use race and deem it important in the study of the human variation. These studies might demonstrate the application of new methods to the scientific racism that anthropologists argued against in the 1960s and 1970s. For instance, though Nei and Roychoudhury (1982) acknowledged the problems with the concept of race, they divide human populations into three major races (Caucasoid, Negroid, and Mongoloid) in their study of human racial evolution and genetic distance. They analyzed the degree of genetic differentiation between these populations to acquire a better understanding of the time at which populations diverged and the amount of gene flow that might have occurred since that time. They argued that their findings supported two hypotheses regarding human racial evolution: the Negroid group and Caucasoid-Mongoloid group diverged and then after thousands of years the Caucasoid and Mongoloid groups evolved and diverged. Alternatively, they argued that these races diverged at the same time, but that there was more gene flow between Caucasoids and Mongoloids than between the Negroids and either Caucasoids or Mongoloids (Nei and Roychoudhury, 1982). This study acts to reify race as a biological construct, and, unfortunately, perpetuates the sense of hierarchy among human races based on their divergences.

Harpending and colleagues (1996) also used race in their analysis of human diversity, combining multiple methods to reevaluate the arguments made in studies like Nei and Roychoudhury (1982). They emphasized that understanding human origins means understanding the demographic history of populations, including population size, extent of gene flow, and isolation. In looking at genetic diversity in Africans, they found diversity to be higher in mitochondrial DNA (mtDNA) sequences, craniometrics, and in short tandem repeat (STR) polymorphisms, STRs having high mutation rates (Weber and Wong, 1993; Bendall et al., 1996; Hapending et al., 1996). Diversity was no higher among Africans than other geographical races in classical markers such as blood groups, which have low mutation rates (Nei, 1987; Hapending et al., 1996). This evidence, they suggested, supports an African divergence earlier than the diversions of all other races as traits with high mutation rates are believed to show greatest diversity in groups diverging at earlier time periods. This study depicts human variation in light of isolation and gene flow, and establishes a temporal hierarchy among the divergences of human groups. Unlike Nei and Roychoudhury (1982), this and other studies reinforce that populations ought to replace races, because genetic studies have already shown that geographical races or subspecies of humankind do not exist (Lewontin, 1972; Templeton, 1999; Templeton, 2013).

Brown and Armelagos (2001) echoed Livingstone (1962) and Montague (1964) in their assertion that race is arbitrary and that the boundaries between races depend on the particular characteristics used and the classifier's cultural norms. Individuals who insist on classifying races cannot help but select traits that support their preconceived notions of race, rather than look at the gamut of traits, many of which do not distinguish groups. Brown and Armelagos (2001) further suggest that similarities among groups are likely an expression of underlying diversity

present prior to humans leaving Africa, which were then shaped by evolutionary forces. Such similarities might also be due to microevolution attributable to parallel environmental pressures.

The concept of race as a biological reality continues to be central in arguments made in the anthropological literature, though it more typically is found on the fringes and not among mainstream works. Works like Philip Rushton's *Race, Evolution and Behavior* (2000), Nicholas Wade's *A Troublesome Inheritance* (2014), and Gregory Cochran's and Henry Harpending's *The 10,000 Year Explosion* (2010) all reflect anthropological and genomic approaches to explaining group differences based on biology, reifying racial categories in the process. Their existence alone speaks to the fact that, more than four decades since Lewontin's arguments, and over half a century since Montagu declared that races are genetically meaningless constructs, the idea of biologically identifiable races still persists. It is arguable that social policies that restricted and controlled human reproduction by restricting the environments in which groups could live actually created the very biological characteristics these more recent studies claim as the evidence for hierarchical biological races (Du Bois, 1975; Gravlee, 2009; Reverby, 2010; Roseman, 2014), a concept on which I now focus my discussion.

To Die or Not to Die

So, why hasn't the concept of race died already? The answer is arguably because race was never a solely scientific endeavor, but instead developed and matured in tandem with political and social agendas (Du Bois, 1975; Martinot, 2003; Painter, 2010). In fact, the establishment of the National Research Council (NRC) in 1916 and its Committee on Anthropology in 1917 represented the merger of political and anthropological interest in race science. Charles Davenport, an ardent eugenicist who founded the Eugenics Record Office in 1910 and the

Eugenics Research Association in 1912, was a member of the committee (Steggerda, 1944; Pernick, 1997; Caspari, 2009; Painter, 2010). This committee had the decidedly eugenic goal of ensuring the improvement of humankind by regulating the influx of "undesirable" races to the U.S. and their contribution to future progeny (Caspari, 2009), as it formed in the wake of an enormous influx of immigrants from these undesirable groups to U.S. shores (Dillingham et al., 1911). The eugenic efforts of this committee and others like it in the early twentieth century (e.g., Race Betterment Foundation) (Pernick, 1997) undoubtedly influenced the Immigration Acts of 1921, 1924, and 1929, which favored the British, Germans, and the Irish (Clark and O'Donnell, 1999). These acts effectively limited the migration of undesirable races to the U.S. (Clark and O'Donell, 1999; Painter, 2010; Weiss and Lambert, 2011; Brace, 2010) and helped shape U.S. population history.

Law and policy makers also sought the approval of the scientific community to withhold resources from and even forcibly sterilize criminals, the mentally ill, and presumably weaker and/or degenerate races (Black, 2003; Painter, 2010; Weiss and Lambert, 2011). Earnest Hooton and Aleš Hrdlička, both honored as founders of American biological anthropology, were among the eugenicists who were quite outspoken on the need for policy to regulate undesirables (Caspri, 2009; Giles, 2012). In addition to state compulsory sterilization laws, the infamous *Buck v Bell* 1927 Supreme Court decision paved the way for eugenics policy, allowing the forced sterilization of perceived degenerates through the removal of reproductive organs (Pernick, 1997; Largent, 2008; Painter, 2010). California led the way in sterilization programs until the 1970s, when many sterilization policies were deemed violations of human rights (Largent, 2008; Mooney, 2010).

Nevertheless, race continues to influence immigration policy and has yet to be removed from the U.S. census. The persistence of race on the U.S. census demonstrates the government's obsession with race and documentation of "otherness." It not only persists in politics, but is constantly forced into the public purview by social media as much as it was in the past within eugenics propaganda (Klein, 2012). Thus, race continues to be an engrafted part of social life and a social reality for everyday people; racial policy constructed social boundaries, thereby mediating gene flow, and reinforced the associations of socioeconomic factors with culturally bound groups. It is no wonder that it has not been an easy task to convince the public that races are socially constructed. This is also perhaps due to the visual nature of perceived races. How do you convince people to ignore the variation they see on an everyday basis when scientists, who study variation and speak publicly with authority, continue to erroneously explain variation using racial categories (e.g., Nei and Roychoudhury, 1982; Harpending et al., 1996; Cochran and Harpending, 2010)?

Racial categories do not explain variation, but racism might in fact explain genetic variation in the last two centuries in racialized communities (Roseman, 2014). Roseman (2014) points out that there are two types of variation that scientists attempt to explain using race as a guide: 1) prehistoric human variation and 2) historic and contemporary human variation. Prehistoric human variation is variation that occurred before the written record. Historic variation occurred during the written record, but more than 50 years ago, and contemporary variation occurred within the last 50 years. Historic variation and contemporary variation are combined. It has already been well established in previous sections above that race does not explain human variation in prehistory. But race often explains human variation in historic and contemporary times because its inception brought with it racist political, economic, and social forces (Du Bois,

1975) capable of shaping genetic variation by regulating mating choice and ensuring the inheritance of racial status. Thus, mating is not random in racialized communities, but regulated and restricted by social policy and norms. These racist policies and norms affect biology by determining the pattern of human variation. “Thus, the allele frequency differences that we see between racialized groups as people understand and experience them today are in part the product of racism acting to shape genetic variation” (Roseman, 2014). The correlation between perceived races, genetic variation, and phenotype results from racist policies and norms.

Furthermore, the existence of race in social, political, and economic interactions often leads to biological differences in health, particularly physiological stress (Oliver and Muntaner, 2005; Jacobs et al., 2009; Krieger, 2010; Kershaw et al., 2011) among perceived races, making race a reality with impacts on welfare. This is strongly reflected when analyzing the social, political, and economic reality as well as the biological reality of race in the U.S. in the nineteenth and twentieth centuries. I discuss these realities below, but beforehand I review historical and archaeological exploration of racial disparities through the study of material culture, as material evidence in the form of artifacts and structures has also been used to explore the effects of racialization on people groups. Although these studies focus on the material correlates of racialization, they offer insight into how one might investigate the health (biological) correlates of racialization as economic status (indicated by material possession) is often correlated with health status (Oliver and Muntaner, 2005; Jacobs et al., 2009; Krieger, 2010; Kershaw et al., 2011).

Race in Archaeology

Critical Archaeology

A critical approach is concerned with the research process and the way knowledge is produced. It is also concerned with the impact research endeavors and findings have on populations (Agbe-Davies, 2015). Critical archaeology investigates the political, social, and economic relationships between groups to illuminate the foundations and mutability of material inequality, power relations, domination, resistance, and racisms, among other overlapping power dynamics. Present inequities have changed over time, which forces communities to realize that inequity is not natural but mutable and can be challenged. Critical archaeology makes available for deployment this information in the struggle for political, social, and economic equality (Wilkie and Bartoy, 2000; Franklin, 2001b; Moore, 2006; Agbe-Davis, 2007; Leone, 2010), and is capable of generating warranted social change.

Critical archaeology challenges nationally accepted histories by providing alternative histories (of previously ignored groups) that debunk spurious myths concerning groups, expose the detrimental effects of dominant ideologies on subordinate groups, and expose the beneficiaries of harmful dominant ideologies (Blakey, 2001; Agbe-Davis, 2007; Leone, 2010). Thus, critical archaeology illuminates histories that have been ignored or only minimally acknowledged in officially sanctioned histories around the globe. Critical histories broaden the American experienceto include groups and events commonly ignored because of their identification as other or inferior. Critical archaeology illuminates these alternative histories to bring about unity, a sense of belonging, empowerment, and social change to the groups that helped to create and are most affected by these narratives. This approach also challenges the very nature of the dominance of certain histories in light of others, as well as the truth and infallibility

of conventional histories. Conventional histories are the product of dominant groups that recognized the political, social, and economic power of history as a legitimizing force for warranted change or maintenance of the status quo.

Critical archaeology targets studies concerning women, children, the poor, lower-class, racially disenfranchised, and workers because it incorporates an all-inclusive approach to archaeology/history and it is concerned with present-day social and political contexts (McDavid, 1997; Franklin, 1997). Critical archaeology is reflexive, in this regard, in that it considers the impacts of its findings and interpretations on living, usually descendent populations (Sandlin and Bey, 2006). It also considers the particular interests such interpretations might serve and its beneficial or damaging effects, as well as potential to stimulate social change. Because critical archaeology is always politically situated, archaeologists are advised to practice self-reflexivity, that is to reflect on their own biases and the impact their biases might have on their research and on descendent communities (Orser, 1998; Wilkie, 2004).

Critical Archaeology, Race, and Inequality

Critical race theory is a key component to addressing race through archaeological evidence in critical archaeology. Critical race theory burgeoned in the 1970s as legal scholars and activists became aware of the link between the law and public policy and racial disparities (Delgado and Stefancic, 2001). This theory has since become ingrained in many disciplines, including education, sociology, history, political science, and anthropology (Ford, 1997; Delgado and Stefancic, 2001; Epperson, 2004). It holds that race is socially constructed, racism is normal and benefits (both materially and psychologically) races deemed to be superior, racialization affects groups differently temporally, race intersects with other identities (such as gender), and the

racially oppressed offer better insights into race and racism (Harris, 1997; Delgado and Stefancic, 2001; Epperson, 2004) The primary goals of critical race theory are to illuminate structures in society that contribute to racial inequality and then change relationships between race and power that create, maintain, and perpetuate this inequality. The latter goal points to the activist dimension of this theory. Critical race theory demands that proponents actively seek out change that enhances the lives of populations marginalized because of their racial status (Delgado and Stefancic, 2001).

Critical race theory literature, as well as the critical archaeologists who employ this literature, exposes the origins and facets of structural racism, racial ideologies, and white hegemony by exposing their material correlates (Harris, 1997; Agbe-Davis, 2007). Until the late 1980s, race was not a research interest amongst archaeologists (Orser, 1998, 2007). However, the simultaneous development of post-processual and critical archaeology in the 1980s, and the birth of the World Archaeological Congress (WAC) in 1986—which was mainly concerned with apartheid in South Africa, indigenous rights, and with making archaeology relevant to contemporary populations—established issues of race and racialization as valid and pursuable topics of archaeological inquiry (Orser, 2007). The WAC took notice that inequality didn't just appear but "had historical roots that could be archaeologically investigated" (Orser, 2007:19). Prior to this realization and confrontation, archaeologists tended to tell dominant narratives of the past that contributed to white hegemony, in effect making archaeology yet another tool of oppression and control (Orser, 2007).

People organize the material world in hierarchies that by their nature reify inequalities (Nassaney, 2007). Consequently, the integration of both historical documents and archaeologically recovered material remains might elucidate the process by which groups were

assigned racial designations (i.e., the process of racialization) (Nassaney, 2007). This integration can also aid in investigating the maintenance of racial categories and the effects of such categorization on individuals (Smedley, 1998).

The trinity of race, class, and gender reemerged in the 1990s as promising concepts of identity formation for archaeologists shortly after the establishment of the WAC and the recognition of race issues as valid problems. Their interpretations were increasingly critical and complex (Meskell and Preucel, 2004). Race and racialization emerged as the most prominent concepts of identity formation in African American archaeological discourse (Orser, 2007). Race continues to dominate as the most explored identity concept in the field (Nassaney, 2007).

Finding Race in the Archaeological Record

The mutability of race combined with the fact that populations rarely represent distinct cultural entities make it difficult for archaeologists to investigate race with immutable material and spatial archaeological correlates (Orser, 1998). No direct line exists from material objects to culture or status (Howson, 1990). Orser (1998) suggests that the best way to address racial inequality is to view the material culture of Africans/African Americans in light of market capitalism, since it was/is within this market that whites used race to impose conditions of material inferiority among African Diaspora. He then points out that the rise of mercantilism in North America brought large numbers of mass-produced objects to everyone in America, regardless of ancestry or cultural background. This, in effect, homogenized material culture across perceived racial boundaries. With this in mind, archaeologists turned their attention to consumption patterns and consumer tastes to assess the racial correlates of material inequality

(Orser, 1998; Mullins, 1999). Archaeologists also sought to determine the various meanings of similar artifacts to distinct groups (Orser, 1998).

On a theoretical note, Babson (1990) and Singleton (2006) recommend that we not reify racial identities by assigning certain artifacts to these categories. By designating race to artifacts, we ignore the fact that racial identities stemmed from a racist system designed to promote hegemony and material inequalities. Researchers must emphasize race as a social ideological construct created in order to establish, maintain, and perpetuate political, social, and economic inequity. The interaction, however, between so-called racial groups should be traceable archaeologically because racist ideologies determined the boundaries of this interaction. The effects of racial classification (racialization) are real (Orser, 2007). Racially segregated groups share common experiences, some of which leave traces in the material record. Such traces are due to the fact that the process of racialization generates a struggle for material objects as well as space between racially designated groups. The dominant group in racially structured societies typically wins this struggle, allotting to themselves greater access to goods and services. This disparity in consumption patterns creates a bridge between race and material culture (Orser, 2007).

Critical Archaeology and Racialization

Critical archaeologists have taken the lead among anthropologists in deconstructing the processes and results of racialization in past societies. Using literature pertaining to critical race theory, critical archaeologists attempt to expose the origins and facets of structural racism, racial ideologies, and white hegemony by exposing their material correlates (Agbe-Davis, 2007). Critical archaeologists like Carol McDavid (1997), Paul Mullins (1999; 2011), Charles Orser

(2007), Dean Saitta (2007), and Mark Leone (2010), and those working alongside biological anthropologists like Cheryl La Roche and Michael Blakey (1997) have attempted to trace inequalities (in power, social, political, and economic relationships) diachronically and synchronically. They also set out to demonstrate how such inequalities have changed, shifted, or continued in contemporary societies, allowing them to connect past and present and create a dialogue between the two.

Charles Orser (2007), for example, integrated historical and archaeological evidence to elucidate the process by which Irish immigrants and Irish Americans were assigned and responded to racialization in nineteenth-century New York. Stephen Brighton (2009) extended this work by including archaeological evidence from New Jersey and Ireland. To interpret material correlates of Irish racialization, both authors linked the past status and material negotiations of the Irish within Ireland's class structure, and as a marginalized group in Ireland under the British, to material negotiations as a marginalized group in the U.S. Their studies showed that past and present experiences of racialization shaped Irish material culture. These studies also demonstrated the link between racialization and race-based class oppression.

Although culturally constructed, racial classifications and the inequality that they entailed have biological consequences that affect genetic variation and health in racialized populations (Gravlee, 2009; Gravlee et al., 2009; Roseman, 2014). My study explores racial diversity among European Americans in the U.S. from the nineteenth and twentieth centuries in order to construct a more inclusive picture of the effects of racialization on childhood and adolescent stress among European and African Americans through time. To this end, this study draws from critical archaeology theory in its efforts to trace the mutability of race and create a dialogue between past and present experiences of racialization. This research thus seeks to understand the

environmental context that enabled the descendants of nonwhite European Americans and immigrants to become white, and to interrogate whether their stress levels changed in accordance with changes in definitions of whiteness. Identifying the factors that put all European Americans after 1945 in the white racial category and thus on the more fortunate side of the health disparity gap might provide insight into the most prominent factors influencing racial disparities in health today.

Socioeconomics and Racial Experiences of Europeans, European American, and African Americans in the 19th-century and 20th-century U.S.

Although this section is primarily concerned with racial interactions in the nineteenth and twentieth centuries, I want to take some time to unravel the burgeoning of whiteness in colonial America, with particular focus on Virginia. Virginia was the heart of English mainland colonialism in the seventeenth century and thus is considered the best place to start when considering the foundations of whiteness. For most of the seventeenth century, English settlers referred to themselves as Christian (Martinot, 2003). An individual's Englishness or Christian status was assumed as the norm and thus not frequently referenced (Agbe-Davies, 2015). Other statuses were often referenced in terms of religion and nationality (non-Christians and/or non-English). Native Americans were considered "other" and inferior to the English in appearance and ability. They were considered primitive and savage. Native American status as other and/or nonwhite primarily centered on the English or Christian belief that they had inferior social and cultural systems (Parrillo, 2000; Pinder, 2012). By accepting the English and/or Christian social and cultural systems, Native Americans could rise on the social ladder, but almost never to the status of the English or white (Pinder, 2012).

African, Negro, and Mulatto were used in narratives, wills, certificates, and colonial law to refer to people of African descent. Negro and Mulatto were used to identify a persons' racial and economic status. Mulatto was also used in some cases to simply refer to a brown skinned individual (Stephens, 1999; King and Chaney 2010). Martinot (2003) argues that the term Negro after 1662 became synonymous with slave and thus entailed an economic status as opposed to a racial status. However, indentured servants of European descent were also described as slaves. Agbe-Davies (2015) references Negro and Mulatto in both ethnic and racial terms. However, Negro and Mulatto did not entail the power relationship inherent in racial identities in the early part of the seventeenth century (Martinot, 2003). Nonetheless, distinctions existed in how Negros and Mulattos were treated in the colony. For instance, free Negros and Mulattos were either constrained to certain locations or forced to leave the colony later in the seventeenth century (Agbe-Davies, 2015).

Beginning in the 1660s, the migration of indentured servants from Europe to Virginia began to decline. This decline was followed by an increase, in the 1670s, in bound labor from Africa (Agbe-Davies, 2015). Success in tobacco farming required large amounts of land and cheap labor. By exploiting cheap labor from Africa and tightening the hold on laborers by switching from term to perpetual and inherited bondage, planters ensured economic and political success for themselves and their progeny. To further secure their economic and political domination, planters enacted laws meant to restrict mating choice (Agbe-Davies, 2015). According to Martinot (2003), these laws were meant to establish a purity condition that would separate European colonists (and the power they enjoyed) from slaves (the majority of whom were of African descent). This purity condition stipulated that only a person born to both European descent parents could be considered white. As the purpose of this self-identification was to

distinguish ones' economic and political position from others, "white" then came to entail a sense of supremacy over all other groups excluded from the white category. Thus, the shift from the English/Negro to the white/black dichotomy occurred at the end of the seventeenth century.

A distinction was later made in the nineteenth and twentieth centuries between white ethnic and white racial status as large groups of Europeans from non-Protestant, Southern and Eastern Europeans began to muddle established definitions of whiteness. As stated in the Introduction, white ethnic identity has been assigned—historically and in the present-day—to all individuals perceived to be of European descent because they were thought to belong to one of the four Caucasian or white ethnic divisions: non-Aryan, Aryan, Semitic, and Euskaric (Dillingham et al., 1911; McDermott and Samson, 2005; Painter, 2010). Nordic groups, English, Scots-Irish, and Germans were classified as *racially* white (Parrillo, 2000; Black, 2003). A white racial identity entailed the perception that one was biologically and intellectually superior to all other white ethnic groups and nonwhites. In fact, these other ethnically white groups—non-Protestants, Southern and Eastern Europeans—were considered Nonwhite Europeans (Roediger, 1992, 2005).

Individuals of Asian descent also migrated to the U.S. in large numbers and found themselves confronted with racism and racist structures. Parrillo (2000) points out that the Chinese, who migrated in large scales to the U.S. during the first half of the nineteenth century, were also considered nonwhite and even diseased. They were composed primarily of skilled laborers, farmers, exiles, and refugees. They migrated to escape famine and political unrest, although the California gold rush also motivated many to seek refuge in the U.S.

Nonwhites in the nineteenth and early twentieth centuries interacted with one another and with whites in a complex and ever-changing manner guided by structures of racialization.

European American males, who had gained dominance in the U.S. during the colonial period, used racialization to claim white racial superiority and to justify political, social, and economic discrimination of inferiorly defined races (Babson, 1990; Orser, 1998; Epperson, 1996). Thus, racialization unequally affected access to resources including employment, sufficient income, and property ownership, structuring class and class oppression (Orser, 2007; Smedley, 1998). Class herein refers to ones' socioeconomic position (Paynter and McGuire, 1991) and is often measured by assessing individual or group income or resources. Race-based class oppression occurs when races designated as inferior have limited access to political, social, and economic resources (Orser, 2007), many of which have been shown to influence health disparities in childhood stress among social races in contemporary populations (Jacobs et al., 2009; Oliver and Muntaner, 2005; Krieger, 2010; Kershaw et al., 2011). In this section, I discuss these two concepts in light of the racial and racialized experiences of European and African Americans in the nineteenth- and early twentieth-centuries in the U.S.

Nonwhite European American and Immigrant Experience

Historians, political scientists, and sociologists have investigated the burgeoning of the concept of race, definitions of whiteness and otherness, and how European immigrants and their progeny were racialized (Omi and Winant, 1986; Parrillo, 2000; Martinot, 2003; Ward, 2005; Painter, 2010; Pinder, 2012, 2015). As discussed above, their findings suggest that white as a race was established to first separate Europeans (predominately the English) from Africans and Native Americans (Martinot, 2003). White racial status in subsequent centuries was used to separate old immigrants and northern Europeans from non-Protestant, Eastern, and Southern Europeans to ensure the continued political, social, and economic superiority of the former

groups (Jones and Carter, 1996; Dinnerstein et al., 2003; Painter, 2010; Pinder, 2012). White socioeconomic and political superiority was maintained by the establishment of laws and policies that privileged whites and defined social relations between constructed racial groups (Martinot, 2003; Roediger, 2005; Goldberg, 2005; Pinder, 2012). These studies also showed how racialization continues to affect groups and social policy in the U.S. (Omi and Winant, 1986; Hero, 2010; Saperstein et al., 2013).

The populations examined in this dissertation were active participants in the process of racialization. White Americans, mostly of German, English, Scottish, and Scots-Irish descent, experienced anxiety over the influx of non-Protestant, Eastern and Southern European immigrants and concomitant competition over resources during the nineteenth and twentieth centuries (Jones and Carter, 1996; Parrillo, 2000; Dinnerstien et al., 2003; Roediger, 2005; Painter, 2010; Pinder, 2012). As a result of this anxiety, powerful whites used the process of racialization to marginalize non-Protestant, Eastern and Southern Europeans while simultaneously allowing for the rapid assimilation of Northern Europeans (Horne, 1996; Dinnerstien et al., 2003; Roediger, 2005; Painter, 2010).

Ignatiev (1995), Orser (2007), and Brighton (2009) showed that Catholic Irish immigrants and Irish Americans were also at times not considered white. The Irish, having begun their migration to the U.S. in the late 1840s because of the Irish Potato Famine, were often depicted as apes and monkeys with white skin in cartoons and propaganda in the nineteenth and early twentieth centuries (Orser, 2007; Painter, 2010). Italians and Jews were also considered nonwhite (Jacobson, 1998; Brodkin, 1998; Painter, 2010) and most Southern and Eastern Europeans were considered undesirable nonwhites (Dillingham et al., 1911). These undesirable nonwhite immigrants migrated to the U.S. in response to the Immigration Acts of 1882, 1884

1886, and 1888, which excluded the Chinese, but allowed millions of immigrants, predominately from Southern and Eastern Europe, to come to the U.S. (Dillingham et al., 1911; Roediger, 2005; Pinder, 2012).

Yet, what sets nonwhite European Americans apart from other minorities in the history of U.S. race relations is their ability, albeit at different times, to transition from an inferior to a superior racial status (Knowles and Prewitt, 1969; Painter, 2010). Knowles and Prewitt (1969) suggested that this transition was possible because these groups had gradual access to naturalization, citizenship, education, political power, suffrage, and ultimately the right of self-determination. This access was in an effort to prevent conflict between EA and Europeans whose ancestral country or country of origin was at war during World War I and World War II (Roediger, 2005). African Americans, Native Americans, and Asian Americans have not been able to change their racial status and so remain among the dominated racial groups in the U.S.

White racial identity, therefore, was and is constantly being redefined in light of the changing makeup of the American populace. It is situational, grounded in social and economic privilege, and responds to political, social, economic, and cultural shifts (Omi and Winant, 1986; Painter, 2000; McDermott and Samson, 2005; Ward, 2005; Pinder, 2015). In this study, I employ four time periods to reflect these cultural shifts and the differences in how groups were racially classified: Early (1828-1881), Middle (1882-1913), Intermediate (1914-1945), and Late (1946-1984) periods. These reflect period of racial tension and immigration patterns, and so are important for approaching the questions this dissertation asks about the effects of racialization on genetic variation and childhood and adolescent stress (Dillingham et al., 1911; Horne, 1996; Clark and O'Donnell, 1999; Dinnerstein et al., 2003; Roediger, 2005).

Early Period (1828-1881)

The Early Period (1828-1881) begins with the earliest individuals' birth date in the collection. The Naturalization Act of 1790 required that an individual be white to become a citizen of the U.S. This act implied that a distinct racial category of white existed early in U.S. history and that the opposite—nonwhite—existed as well, including Native Americans and African Americans. Subsequent naturalization acts and laws as well as immigration acts favored old immigrants (Germans, English, Scottish, and Scots-Irish) over non-Protestant, Eastern, and Southern Europeans (Roediger, 2005; Pinder, 2012). The Irish, in particular, were marginalized and exploited because of their identity as Catholic and poor, despite having white ethnicity (Gilroy, 2003). They began migrating to the American colonies in the seventeenth century in small numbers. It wasn't until the Potato Famine of the 1840s that thousands of Irish immigrants landed on U.S. shores (Brighton, 2008; Linn, 2010). These immigrants were relegated to the most menial jobs and poorest working and living conditions. They worked and lived in unsanitary and disease ridden environments that resulted in them been labeled as disease prone and a health threat to whites (McCaffery, 1997; Gallman, 2000). Their status also resulted in them receiving marginalized health care. So, not only did Catholic Irish experience greater rates of morbidity and mortality during the early years of their migration (Ernst, 1949; Keneally, 1998), but they were further exposed to greater risk by inadequate treatment. Differences in stress among racialized European American populations in the Early Period might reflect Irish marginalization.

Until 1882 Europeans primarily migrated from Western Europe (Dillingham et al., 1911; Painter, 2010). These old immigrants made up the white racial category (Painter, 2010). White citizenship entailed voting and property rights, higher wages, and better jobs for men (Roediger,

2005; Painter, 2010). With the exception of the Irish who migrated to the United States beginning in the 1840s (Orser 2007; Brighton 2008, 2009), there were few resource threats from other European groups considered nonwhite as there was only a small number of Eastern and Southern European immigrants in the U.S. prior to 1882 (Dillingham et al., 1911; Painter, 2010). Accordingly, this period is considered a period of minimal racial tension among Europeans and European Americans.

Middle Period (1882-1913)

The Middle Period begins with the large scale migration of Southern and Eastern Europeans to the U.S. According to Dillingham and colleagues (1911), the nationality composition of European immigrants in the U.S. changed after 1880 from consisting of primarily Western Europeans to being comprised of Southern and Eastern Europeans (i.e., Italian, Jewish, and Slavic peoples). In fact, from 1882 to 1902 there was a decrease in immigrants from Western Europe by 75% and an increase in immigrants from Southern and Eastern Europe by 475% (Dillingham et al., 1911). Furthermore, 70% of the 13 million Southern and Eastern Europeans migrating to the U.S. from 1886 to 1925 arrived from 1901 to 1915 (Roediger, 2005). The Immigration Acts of the 1880s opened the door for these immigrants to come to the U.S.

These new immigrants were not immediately accepted as racially white. Nonwhite Europeans were gradually assimilated into the white race, with some groups assimilating more quickly than others (Painter, 2010). The rate of assimilation depended on the political, social, and economic environment in which these nonwhites immigrated and their acceptance of old immigrant Protestant Euro-American cultural norms including attire, the English language, and Protestantism (Parrillo, 2000; Roediger, 2005; Painter, 2010). The Naturalization Act of 1906

demonstrates the pervasiveness of anti-new immigrant attitudes as it required anyone seeking citizenship to learn English. This requirement was not lifted until 1940 (Weinberg and Crosswell, 1967).

By ending the Middle Period in 1913, I capture most of the large scale migration of Southern and Eastern Europeans while simultaneously leaving out the fluctuations in migration and acceptance beginning with World War I in 1914. This period is considered a time of increasing racial tension as white European Americans began to compete more frequently with nonwhite Europeans and European Americans for resources.

Intermediate Period (1914-1945)

The Intermediate Period (1914-1945) starts at the beginning of World War I, spans the Great Depression, and ends at the end of World War II. During World War I (1914-1918) white European Americans aimed to unify all European Americans in an effort to reduce conflict on American soil between whites and nonwhites whose countries of origin were at war with one another. Assimilation was accelerated through education in order to achieve unification. These efforts primarily benefited British, German, and Scots-Irish immigrant populations in the United States because they were already considered to be part of the white race (Horne, 1996). Immigration laws passed in 1921, 1924, and 1929 also favored British, German, and Irish peoples (Clark and O'Donnell, 1999).

The effects of the Great Depression (1929-1939) on resource stress cannot be underestimated. The depression affected people from all racial groups and social classes, albeit differentially (Cochran, 1968; McElvaine, 1984). Nonwhites continued to experience resource marginalization even during this time of limited resources. In fact, African, Native, Mexican, and

Asian Americans were subject to higher rates of wage discrimination and unemployment rates at times exceeding 50% (Cochran, 1968, Philp, 1977; Starr, 1996). It is possible, then, that nonwhite European Americans were also subject to more intense resource stress than white European Americans.

During World War II (1939-1945), the Nationality Act of 1940 was passed. This act was pivotal in ameliorating racial tensions between white and nonwhite European Americans and immigrants because it removed the English language requirement and declared that individuals were citizens based on birth or naturalization (Weinberg and Crosswell, 1967). Moreover, during and after U.S. involvement in World War II (1941-1945), racism became problematic in U.S. politics along with antisemitism, anti-Catholicism, and anti-new immigrant. The war began the process of ameliorating the institutional and cultural factors that marginalized nonwhite Europeans as it brought all people of European ancestry together in the wake of anti-Nazi and antifascist mobilization (Roediger, 2005).

The favoring of Irish citizens in the 1920s immigration legislation, the Nationality Act of 1940, and the beginning of the decline of antisemitism, anti-Catholicism, and anti-new immigrant attitudes during World War II designates this period as a time of gradual acceptance of nonwhite European Americans in white America. Including the years from 1940 to 1945 in this period offers a better understanding of how changing attitudes toward non-Protestants, Southern and Eastern Europeans initially impacted gene flow.

The intermediate time period (1914-1945) accounts for the lag I expect between nonwhite European Americans becoming racially white and an improvement in their stress status. This thirty-year period represents a time when whites were becoming more inclusive of all individuals of European ancestry. Thus, this period represents a time of changing acceptance as white for

some European and European American groups during the world wars and resources stress during the depression. This period also accounts for the varying rates in which groups were accepted because it spans a time when different European groups were accepted as white at different times, for different reasons, and during different political, social, and economic environments. Racial tension likely fluctuated during this time period.

Late Period (1946-1984)

The Late Period (1946-1984) begins with the end of World War II and ends with the latest individual birth date in the skeletal sample used in this dissertation. Racial tension between white and previously nonwhite European American and European immigrants during this period continued to decrease. With citizenship made possible for all Europeans by the Nationality Act of 1940, these groups were allotted voting and property rights, higher wages, and better jobs (Roediger, 2005). Although laws cannot legislate away racism, such acts reflected acceptance of nonwhite European Americans by white European Americans with enough support to enact legislation. Accordingly, by the 1950s the income gap between previously nonwhite European Americans and European immigrants and white European Americans disappeared (Clark and O'Donnell, 1999). This period denotes a time when 'white' became an umbrella term for all individuals of European descent (Roediger, 2005).

The above laws and events demonstrate how both institutional and cultural racism marginalized nonwhite Europeans and pressured them to assimilate so that they could attain a better status for themselves and their children. Assimilation entailed the loss of language, culture, and even patriotism toward one's country of origin. It also entailed the adoption of the English language, American patriotism, a capitalistic economic system, and bigotry (Roediger, 2005,

Kendall, 2006; Parillo 2000; Painter 2010). This may not seem like a fair trade, but with whiteness came power, privilege, and wealth, which in turn permitted access to better health care and healthier environments (Kendall, 2006). This period represents a time of decreasing racial tension and increasing acceptance as white for all Europeans and European Americans (Knowles and Prewitt, 1969; Clark and O'Donnell, 1999; Roediger, 2005).

African American Experience

Although African Americans were not considered immigrants, they were considered nonwhite and were marginalized from the late seventeenth century well into the twentieth century via polices, statutes, and laws (Parrillo, 2000; Martinot, 2003). The Three-fifths Compromise, vagrant's ordinances, and the grandfather clause were all aimed at marginalizing African Americans and maintaining white hegemony (Parrillo, 2000; Marinot, 2003; Pinder, 2012). The Three-fifths Compromise stated that African Americans were only counted as three-fifths of a person, vagrant ordinances were meant to set curfews and geographical boundaries on African Americans, while the grandfather clause stated that a person could vote if his or her grandfather voted. Vagrant ordinances and the grandfather clause were part of a network of racists polices, known as Jim Crow laws, that were set in place to marginalized and intimidate African Americans beginning in the late 1870s. Throughout the U.S., but especially in the South, ideologies of racial hierarchy and white supremacy argued that blacks were inferior in mental, social, and physical capabilities (Fredrickson, 1971; Ely and Bodenhamer, 1984; Painter, 2010). Blacks were a primitive form of human and incapable of being civilized. Their supposedly savage tendencies made them a danger to society and effectively stereotyped them as innately criminal. The purity and virtue of white women became the core of racist ideologies and the

driving force for discrimination and violence against blacks beginning in the late seventeenth century (Fredrickson, 1971; Wyatt-Brown, 1982; Rable, 1984; Martinot, 2003). As a result of prevailing racial ideologies, legal segregation, disenfranchisement, and violence meted out toward blacks became the norm until the late 1960s (Rable, 1984; Brundage, 1993; Bergeron et al., 1999; Parrillo, 2000). Entire communities stigmatized, discriminated against, and collectively marginalized African Americans (Christian, 1995; Crouch, 1992; Davidson, 2004; Sitton and Conrad, 2005; Phillips, 1996; Morton, 2000).

In the South and North, from the colonial period to the early twentieth century, blacks were subject to very hard labor and the most menial of jobs with very little pay. Racialization also forced them to survive under consistently impoverished circumstances (Johannsen, 1970; Martinot, 2003; Parrillo, 2010; Foner, 2014). Most worked as field hands, sharecroppers, custodians, cooks, laundry women, nannies, shoe shiners, or in the mills and mines (Drago, 1982; Schultz, 1994; Phillips, 1996; Bergeron et al., 1999; Davidson, 2004; Parillo, 2010).

Early Period (1825-1881)

For most of the colonial and pre-Civil War periods, the southern economy primarily benefited and was governed by European Americans, most of whom were considered white. The southern economy enjoyed the fruits and control of slave labor (Franklin, 1956; Ely and Bodenhamer, 1984; Morris, 1984). The inhumane treatment of slaves during this period was due primarily to the fact that they were considered chattel property with the sole purpose of carrying out the demands of their masters (Wyatt-Brown, 1982). Slaves needed to be watched, instructed, and coerced by their white superiors, or else they might slip into an existence of idleness, starvation, or worse: heathenism (Franklin, 1956; Wyatt-Brown, 1982; Schwarz, 1996).

However, as the Civil War (1861-1865) waned plantations were gradually abandoned as slaves fled to contraband camps, military service, or Black shanty towns across Union lines (Holt, 1982; Bergeron, et al., 1999). Those who remained on plantations required payment for their labor, reasonable hours, and land to farm for themselves (Bergeron et al., 1999). This demand for compensation fueled anger in white southerners who still believed that the proper place of blacks was under their fist and in their fields.

The Freedmen's Bureau was established by Congress in 1865 to oversee the transition of slaves to freedmen and maintain order in the South (Holt, 1982; Crouch, 1992; Davidson 2004). Bureau agents oversaw labor contracts between black employees and white employers and settled disputes between the two (Holt, 1982; Bergeron et al., 1999). The Bureau was not established to aid blacks in obtaining equality but instead to give them a chance at competing in the market economy (Holt, 1982).

Freedmen sought equal rights under the law, and whites were forced to compete economically and politically with blacks. Consequently, whites established Jim Crow laws to structure white interactions with freedmen and maintain the latter's subordinate position socially, politically, and economically (Rable, 1984; Parrillo, 2000). Even judicial decisions beginning in the early and continuing through the late period gave preference to whites in contract disputes or in cases concerning white on black and black on white crime (Wilson, 1965; Fredrickson, 1971; Ely and Bodenhamer, 1984; Huges, 2007). Convicted whites were given capricious fines or warnings; blacks, on the other hand, faced serious jail time and summary punishment from crimes committed against whites (Ely and Bodenhamer, 1984). Torture and cruel punishment existed in the South well into the twentieth century (Franklin, 1956; Bruce, 1979; Brundage, 1993; Ely and Bodenhamer, 1984). In fact, in the South, blacks made up 85% of the lynching

victims between 1880 and 1930, while whites comprised 83% of lynching victims outside the South for the same time period (Brundage, 1993). This study does not mention whether "whites" meant all ethnic whites or particular European American or immigrant groups.

Although racial tension existed prior to the Early Period (1828-1882), this period reflects ever increasing racial tensions toward African Americans. During this time African American status changed from enslaved to freedman and voting citizen in light of the 14th Amendment (1866). Yet, after the Civil War and during Reconstruction (~1863-1877) white European Americans acted to undo the success and independence African Americans had acquired during Reconstruction (Crouch, 1992) by increasing racist structures (Sitton and Conrad, 2005; Franklin and Moss, 1994). Whites eschewed African American men's 14th Amendment (1866) right to suffrage by enacting the grandfather clause, which restricted voters to men whose grandfather had the right to vote. Laws/clauses were also enforced that required land ownership, and literacy as pre-requisites for voting, none of which were prevalent among African Americans, as well as poll taxes that many African Americans could not afford to pay (Franklin and Moss, 1994). Laws were further enacted in twelve southern states from 1852 to 1884 prohibiting interracial marriage and miscegenation between whites and blacks (Pascoe, 1996; Hollinger, 2003; Edgar, 2009). By denying African American the right to vote, dominant whites ensured that blacks would remain underrepresented and thus in the lowest rungs of society.

Middle Period (1882-1913)

The *Plessy v. Ferguson* 1896 verdict made legal separate but equal public accommodations for African Americans and European Americans, ensuring all but equal segregation for non-whites. It also helped to intensify structural racism (political, economic, and social) during the

Jim Crow Era (Parrillo, 2000; Davidson, 2004). During the Jim Crow Era, racist structures were heightened and legalized, and individuals were cruelly penalized for seeking to act outside of the restrictions imposed on them by such structures (Fredrickson, 1971; Rable, 1984; Brundage, 1993; Christian, 1995; Bergeron et al., 1999; Davidson, 2004). The Jim Crow Era was a period of legalized political, economic, and social marginalization and marks a period of heighten racial tension toward African Americans. Jim Crow reigned until it was successfully challenged by the *Brown v. Board of Education* 1954 decision to desegregate schools. In the wake of this decision, the Civil Rights movement gained momentum and began to successfully strip away Jim Crow legal and social structures.

Intermediate Period (1914-1945)

Discrimination under the Jim Cow Era in the South and similar discrimination in the North continued to intensify and become the norm for African Americans throughout the intermediate period (Kennedy, 1959; Newby, 1965; Parrillo, 2000; Hughes, 2007). However, the increased restrictions on immigration during and after World War I (1914-1918) from Southern and Eastern Europe, as well as increased employment opportunities brought about during World War II established African Americans as the largest, most opportune, and affordable labor source (Phillips, 1996). The relief brought about by World War I did not continue until the end of World War II, but was interrupted by the Great Depression (1929-1939). In fact, resource marginalization toward African Americans was so intense during the depression that one would have been hard pressed to find one employed African American if there were employable European Americans (Cochran, 1968; McElvaine, 1984). These ten years of depression make the Intermediate period one of the most racist and stressful periods for African Americans. Perhaps

unique patterns of stress will appear in this group in light of fluctuating access to jobs while still under Jim Crow during this time.

Late Period (1946-1984)

Although *Brown v. Board of Education* 1954 and the Civil Rights legislation in the 1960s repealed Jim Crow laws, it did not immediately repeal informal racial discrimination. Informal racial discrimination includes the social and cultural structures that, in combination with continued white hegemony, allow for persistent occupational, federal, commercial, and residential discrimination, all of which are positively correlated with increased incidences of stress and reduced overall health (Lucas, 1974; Baron, 1983; Robinson, 1984; Richardson et al., 1997; Loomis and Richardson, 1998; Friedman-Jimenez and Claudio, 1998; McCarthy, 2000; Davidson et al., 2002; Dressler et al., 2005; Oliver and Muntaner 2005, Chung-Bridges et al., 2008; Krieger, 2010; Kershaw et al., 2011). The perpetuation of this informal social and cultural discrimination is rooted in racism.

In conclusion, races are the product of racialization. They reflect biological distinctions created by social policies that imposed restrictions on groups. These restrictions may not always be visible, based on recorded history or the analysis of biology, but their effects shaped human genetic variation and health disparities nonetheless. While this chapter reviewed some of the evidence for these arguments, it is not exhaustive. However, this perspective on the relationship between race, racialization, and socioeconomic conditions is essential to unraveling the relationship biology, in terms of genetic variation and stress, has with racialization.

Chapter 3

RACE, STRUCTURE, AND STRESS

This chapter details how race is connected to social structure—and how this structure is linked to individual stress. I review the literature to show this relationship, and how racism imposes restrictions on groups through these structures. I then address how childhood stress is a manifestation of structural racism, and review indicators of childhood stress in the skeleton. Limitations and considerations when analyzing and interpreting stress, including the osteological paradox and issues pertaining to the etiology of stress, are also discussed.

Race, Structure, and Structural Racism

Racial ideologies and white hegemony enabled and continue to empower the racialization processThis interaction also has led to the establishment of racist political, economic, and social structures to maintain the hierarchical order of humankind. Society thus functioned and continues to function under an oppressive system of structural racism.

Structural racism is the use of structures to establish and perpetuate inequity between socially defined racial groups (Jones, 1972; Stern, 2005; Weiss and Lambert, 2011; Pinder, 2012). This occurs when race acts to structure social relationships in such a way that one racial group achieves an elevated status at the expense of another. These structures ensure that discrimination and marginalization play integral roles in the nonwhite American experience, which, as reviewed in Chapter Two, shifted as definitions of who qualified as "white" changed. This experience includes those of African Americans, Latin/Hispanic Americans, Native Americans, Asian Americans, and nonwhite European Americans, all of whom have encountered structural racism

((Baron, 1983; Parrillo, 2000; Byrd and Clayton, 2001; Painter, 2010; Williams et al., 2010; Asada et al., 2013).

Structural racism represents a shameful truth in the history of race relations in the United States. This experience was pervasive. It influenced—and in many ways continues to influence—the right to vote, the freedom to choose a marriage partner, have a fair trial, hold public office, access certain spaces, live a healthy lifestyle, obtain wealth, and choose a residence (Franklin and Moss, 1994; Christian, 1995; Phillips, 1996; Orser, 2007; Brighton, 2009).

The connection between structural racism and racialization cannot be understated. It is because certain groups were designated as inferior races during the process of racialization that they then encountered racist structures. Racialization therefore ensured that racially segregated groups would share common experiences, some of which leave traces in the skeleton. I extend Orser's (2007) assertion that material traces of inequity result from the struggle racialization generates over space and resources between racially designated groups—a struggle in which the dominate group typically wins—by including physiological traces of inequality as yet another outcome of the same struggle. Victories in the struggle for space, resources, and prominent roles by the dominant groups led to the successful development and implementation of racist structures. These structures manifested themselves as Jim Crow South statues, Eugenics legislation (e.g., sterilization), occupational discrimination, residential discrimination, resource marginalization, and discriminatory health care practices (Christian, 1995; Williams and Rucker, 2000; Sitton and Conrad, 2005; Smith, 2008; Largent, 2008; Mooney, 2010; Kershaw et al., 2011). Many of these structures have been shown to correlate with racial disparities in stress (Charatz-Litt, 1992; Williams and Rucker, 2000; Williams et al., 2010).

I discuss the measurements of stress in more detail below, but it is important to provide a definition of stress at the outset before discussing the interaction of race and structure on stress. According to the National Institute of Mental Health, stress is the brain's and body's reaction to short- and long-term changes in one's environment. Stress can have positive and negative effects on individual health. Positive stress is usually short-term and occurs when the body releases chemicals and hormones (a metabolic effect) that benefit the individual in certain situations like dangerous situations. During such situations the brain increases its oxygen intake as well as activity to increase the chance of survival. When this type of stress persists because the source of or reaction to the stress is long-term, however, it can suppress immunity, digestion, reproduction, and other bodily functions. This type of negative stress is called chronic stress. A person experiencing chronic stress is more susceptible to illness. Sources of chronic stress might include continual exposure to discriminatory health practices, unsanitary environments, unsafe working conditions, and impoverished conditions.

Anthropologists have likewise described stress as resulting from external factors and stimuli, which in turn affect individual physiology and homeostasis (Goodman et al., 1984, 1988; Goodman and Armelagos, 1989; Goodman, 1991; Temple and Goodman, 2014). Nutrition, pathogen exposure, occupation, activity types and levels, individual status, resource access, and genetic factors all combine in Goodman's models to yield stress that, in turn, has effects on metabolism. Any negative, chronic effect on homeostasis will in turn result in bone disease (in adults) or variation in bone development (in juveniles). Ultimately, then, it is the biocultural interaction of these many factors, which are driven by cultural perceptions of race and sex, that differentially result in stress among groups. While stress is referred to as both a physical and experienced (i.e., perceived) experience (Temple and Goodman, 2014; Reitsema and McIlvaine,

2014), I use the term stress in my analyses to refer specifically to physiological stress, as effects on mental health or perceptions do not manifest on the skeleton.

Theories That Address Race and Structure

There are multiple approaches to modeling the interaction of social structure and stress, which embody theoretical approaches from anthropology, sociology, and cultural studies. Here I review three that are used in this study to interpret the signs of stress as interpreted from skeletal markers: biocultural models, health lifestyle models, and ecosocial theory. The lattermost blends together key concepts from the first two models. These are not exhaustive of all models available, but they are appropriate given the discussion of structural racism above.

Biocultural Model

Biological anthropologists use the biocultural model to situate disparities in stress within stressful transitions that produce inequalities among groups (Larsen, 1994; Steckel and Rose, 2002; Davidson, 2008). The biocultural model of stress situates patterns of stress (as measured in health status, or, in the case of skeletal remains, signs of metabolic stress or arrested growth) in their cultural environment—the social, political, and economic context. This model views the cultural environment as the source of stress and of the resources that avert stress (Goodman et al., 1988; Goodman and Leatherman, 1998). Indicators of stress that result from growth insults (e.g., vertebral neural canal diameters and linear enamel hypoplasias) are the biological response to short or long term stressors that cause inequalities and/or limit resources. Stressors can be resisted through cultural systems (Goodman et.al., 1988). In children, the presence of these

indicators shows that cultural factors that might have ameliorated the effects of stressors were inadequate.

I use this model to investigate whether social factors produced by structural racism affected physiological stress among white European Americans, nonwhite European Americans, and African Americans, as reflected in their skeletal remains. There is a possibility that access to certain resources, namely economic disparities or cultural relief, averted stressful experiences for some groups and not others. Evidence of intergroup and intragroup variation in skeletal markers will signify exposure to different types of and/or longevity of stressors.

Health Lifestyle Model

Sociologists use a different but related model for the relationship between stress and structure. Cockerham's (2005) health lifestyle model considers both structure and agency in health outcomes. This model links individual behavior and tendencies, as culturally restricted, to the contours created by the power of structural conditions (Cockerham, 2005). He argues that though individuals are able to choose between actions that benefit or harm their physical health, their options are not infinite but constrained by structures that mediate social interactions and resources.

Four structural variables are capable of molding daily lifestyle practices that influence health outcomes: "(1) class circumstances, (2) age, gender, race/ethnicity, (3) collectivities, and (4) living conditions" (Cockerham, 2005:56). These variables establish (by constructing relationships) structure, represented in this study as individual agency (i.e., choice) contingent on social structures (i.e., opportunities). Life choices eventually result in practices or actions;

consistently reproduced actions produce health lifestyles (i.e., daily routine practices that influence health).

Ecosocial Theory

Ecosocial theory is premised on the ecological argument that the human body interacts with its external environment in such a way that it embodies health determinants related to the particular social, political, economic, and biological facets of that environment. These environments denote our ecological context. Nancy Krieger (2010, 2011) defined and has refined this theory over the last two decades. Ecosocial theory incorporates interactions between societal and ecological context, levels of analysis, time (life-course and historical generation), space, individuals and populations, and social inequality to determine who and what produces health disparities (Krieger, 2011). Krieger (2011) argues for the exploration of the impact of power dynamics on resource distributions as well as social and biological production and reproduction. She sets out four core constructs of ecosocial theory:

1. Embodiment: the claim that our experiences and interactions with our social and physical environment are embodied in our biological makeup.
2. Pathways of embodiment are varied and contribute differently to the distribution of disease. Pathways include, but are not limited to, exposure to economic and social deprivation, pathogens, hazardous conditions, discrimination, and inadequate health care.
3. There is a "cumulative interplay of exposure, susceptibility, and resistance to pathways, at multiple levels, across the life-course, in relation to historical generation" (Krieger, 2011:222-223).

4. People and institutions act with some level of power and agency, and therefore be held accountable for their actions. Macro-level agency most often restricts and directs micro-level action.

Together, these core constructs allow for an interpretive approach to disparities that addresses several components contributing to stress between groups. In many aspects, this theoretical approach unites concepts from the biocultural and health lifestyle models.

Yet, while the health lifestyle model includes structure in its analysis of health, it is broad in its analysis of health lifestyles and practices primarily because it was formulated for living populations. The ecosocial model is even more comprehensive in its analysis of health, as it also touches on issues of susceptibility based on structure. As I am using skeletal data in this study, I am not able to adequately address varying levels of susceptibility, individual risk of exposures, and/or length of exposures, concepts addressed in the Osteological Paradox (Wood et al., 1992), which is reviewed at the end of this chapter. Consequently, I employ the biocultural model because it more directly addresses physiological stress, was designed with skeletal populations in mind, and situates stress in its biocultural environment, which includes structural racism.

Evidence Supporting the Relationship between Race, Structure, and Stress

Among European Americans

Research comparing the health of historic European immigrants, first-generation U.S.-born groups, and established Americans of European descent (e.g., English, Germanic, French and Nordic)—referred to as "native" born—has demonstrated patterns of similarity and distinction in mortality and morbidity (Pearl, 1921; Winslow and Wang, 1931; Magath, 1937). These studies did not consider racism as a factor, and were not embedded within any of the models or

structural theories noted above. As they were conducted early in the twentieth century, they provide a window into the demographic patterns that existed among different groups during that period.

Pearl (1921) examined U.S. Census data on stillbirths from white U.S. born and foreign-born women for the year 1918. He compared U.S. women to women from Canada, Ireland, the United Kingdom, Italy, Russian, Austria, Germany, Scandinavia, Poland, and Hungary. He found that the rate of stillbirths between U.S. born women and women from Scandinavia, Germany, Poland, and Hungary were compatible, while all other foreign-born women had higher rates of stillbirth. Pearl (1921) also examined mortality rates among three groups of European-descended individuals: native born of native parents, native born of foreign or a combination of foreign and native parentage, and foreign born individuals. He included population and mortality data from the year 1910 from six states, and found that the mortality rates among those who were native born and had native parents was lower than all other groups for individuals age 5 to 94. A conclusion to be drawn from his study is that native born individuals experienced lower rates of stillbirth and lived longer than those of foreign parentage or birth.

Winslow and Wang (1931) set out to determine whether immigrant groups were contributing positively to the rising overall mortality rates of individuals 45 and older. They analyzed U.S. Census mortality data from six states from 1890 to 1920. Groups were divided into native born/native parentage, native born/foreign parentage, and foreign born. They found that mortality rates lowered over time for all age groups up to 49 years. Most importantly, however, the native born/native parentage group curve pattern corresponded with the pattern for the population as a whole. This led them to conclude that the influx of foreign born groups to the general population did not significantly affect mortality rates for older individuals.

Magath (1937) followed up Winslow and Wang's study, and investigated trends in echinococcus (hydatid) disease in the U.S. and Canada from 1882 to 1936. Echinococcus disease is caused by a tapeworm infection that sometimes produces cysts in the lungs and liver (https://www.cdc.gov/parasites/echinococcosis/). Data for this study were obtained from previous studies and the Mayo Clinic. Magath found that the disease was twice as likely to occur among males and that most patients acquired the disease abroad. Regarding birth place, he found that the highest number of cases were among immigrants as opposed to U.S. born individuals. Pearl (1921), Winslow and Wang (1931), and Magath (1937), in addition to other researchers (Howard, 1921; Deporte, 1925), examined the relationship between health and U.S. born versus foreign born or foreign parentage. These studies are themselves historic stories and provided an interesting vantage to understand how researchers categorized people and addressed issues of health. These methods of categorization have since fallen into disuse; very rarely do researchers examine differences between groups as individuals defined them historically.

Yet, considering the dearth of studies comparing health indicators among individuals of European descent, one study looked at individuals living in the eighteenth century, but did not compare among ancestries. Woods (1996) used linear enamel hypoplasias (LEH) to investigate the impact of the social and economic environment on childhood stress among 44 individuals (mostly males) held captive during the French and Indian War. This sample primarily represented colonial-born individuals of Dutch, Irish, German, Norwegian, and possibly Scottish or English descent from early eighteenth century New England and New York State. They were also amont the first few generations of European settlers in the northern American colonies. Her analysis demonstrated a low prevalence of LEH for this mixed sample. She argued that the low prevalence of LEH in this population reflected the abundance of resources, lack of

overcrowding, and lack of social stratification in the early eighteenth-century frontier. Although individuals in this study were not segregated into their various descent groups and compared, Wood's (1996) mention of social stratification is important. The low LEH among all individuals in this group points to a period when race and racialization played no role or a minimal role in social, political, and economic interactions among Europeans; race, therefore, did not greatly influence social stratification among individuals of European descent (Martinot, 2003). Race did, however, play a prominent role in interactions between Europeans and Indians and Europeans and Africans (Agbe-Davies, 2015).

Among African and European Americans

In contrast to the limited focus on comparing health among European Americans, studies comparing stress between African Americans and European Americans abound in anthropology and epidemiology journals. This focus has merit, as the largest contribution to modern health disparities in the U.S. are those that exist between European and African American populations (Dressler et al., 2005). Health disparity denotes differences in morbidity, mortality, and access to health care among populations (Dressler et al., 2005). African Americans have not been as successful as previously marginalized European Americans in obtaining a level of power, privilege, and wealth conducive to access to better health care and environments, because racist political, social, and economic structures present throughout the eighteenth and early twentieth centuries were only partially ameliorated by Civil Rights legislation during the Civil Rights movement (Dowling, 1982; Greenberg, 1990; Williams and Rucker, 2000; Byrd and Clayton, 2001). These structures, in combination with continued white hegemony, allow for persistent occupational, federal, commercial, and residential discrimination (i.e., economic and political

discrimination), all of which have been shown to be positively correlated with increased incidence of stress (Lucas, 1974; Robinson, 1984; Richardson et al., 1997; Friedman-Jimenez and Claudio, 1998; Loomis and Richardson, 1998; McCarthy, 2000; Davidson et al., 2002; Oliver and Muntaner, 2005, Chung-Bridges et al., 2008; Krieger, 2010; Kershaw et al., 2011).

After the Civil War, African Americans continued to encounter structures that determined their status in society and level of employment. Work reserved for African Americans—"Negro work"—was an explicit norm in the United States well into the twentieth century (Baron, 1983). Structures constrained the occupational choices of African Americans which in turn restrained their finances and access to other resources (Taylor, 1974). Lucas (1974) examined the 1967 Survey of Economic Opportunity and the Dictionary of Occupational Titles, which details the aptitudes, training time, temperaments, physical demands, relationship to people, and working conditions of 13,778 occupations. He found that African Americans most often occupied jobs with greater physical demands than did European Americans. Higher African American employment in physically demanding jobs echoes the racist ideologies behind "Negro Work," which held that "Negros" were suited for hard labor, at best, and expendable at worst (Baron, 1983).

Ten years later, Robinson (1984) demonstrated that African Americans with the same educational experience as their European American counterparts continued to be unequally exposed to harsh and hazardous working environments that were conducive to greater illness and injury likelihoods. This study, along with more recent studies (Chun-Bridges et al., 2008; Krieger, 2010; Okechukwu et al., 2014), confirmed the persistence of occupational discrimination and its relationship to stress.

Friedman-Jimenez and Claudio (1998) showed that African Americans were given the least desirable and most hazardous jobs while drilling through a mountain for the Gauley Bridge in West Virginia in the first half of the twentieth century. The mountain, made mostly of silica, was drilled using the more dangerous dry drilling technique as opposed to the safer wet drilling technique. African Americans comprised 80% of the tunnel workers (those most exposed to silica dust) despite the fact that the European Americans comprised 80% of the local population. This apparent task discrimination resulted in African American drillers making up 76% of the 700 deaths due to acute silicosis between 1930 and 1935.

Occupational discrimination was not the only form of disparity for African Americans. Great differences in living conditions also demarcate the African American experience from "white" groups. Kershaw and colleagues (2011) showed that African Americans were not only disproportionately residing in highly segregated impoverished communities, but they also were 2.74 times more likely to experience hypertension than whites. Further analysis showed that the association between hypertension and race was greatest amongst African Americans in highly segregated neighborhoods. This study points to the relationship between residential discrimination and stress.

Even when health care is available, treatment inequality severely impacts African Americans. Haskins and colleagues (2013) investigated racial disparities in the survival of motor vehicle accident victims from 2000 to 2008. They obtained their data from the National Automobile Sampling System Crashworthiness Data System. A sample of 5,861 individuals, 15 years and older, were included in their analyses. They found that African Americans and whites were given the same service and treated in hospital trauma centers. However, African Americans were 50% more likely to die than whites within the first 30 days they were hospitalized. This difference

was maintained when severity of injury and other factors were controlled. This disparity in survival is striking because it only relates to hospitalized victims and their survival rate. It is suggested, though not proven, that African Americans might have received poorer treatment or that they were treated at lower quality hospitals.

Kramer and colleagues (2015) extended this evidence when they examined the pattern of heart disease among African Americans and whites in the U.S. from 1973 to 2010. They obtained their data from the National Center for Health Statistics. Their sample included information on race, sex, birth cohort, and period-specific differences for 23.2 million individuals ages 35 and up who died from any type of heart disease. They found that there was an overall decline in heart disease mortality over time, and that 50% of this decline resulted from technical and medical advances. Nonetheless, they found a slower decline among African Americans and that the gap between African Americans and whites in heart disease mortality increased beginning in the 1980s. Their results suggest that a possible difference in health care among groups existed, but that differences were more likely due to differences in exposure to stressors related to social systems and the environment.

From these and other studies, we may conclude that, throughout the twentieth century, the perpetuation of structural racism ensures that African Americans occupied the most arduous and hazardous occupations (Morton, 2000; Boardman and Field, 2002), lived in the poorest communities, and received the least aid from government agencies. The above studies demonstrate how structural racism contributed greatly to the persistence of racial disparities in stress in the twentieth, and now into the twenty-first, centuries.

Childhood Stress

Although stress manifests itself in the skeleton by many pathways throughout life, I focus on indicators of childhood stress in this study. Children are more susceptible to environmental perturbations than adults and thus present a more complete reflection of environmental stressors (Scrimshaw and Behar, 1965). Thus, stress indicators associated with growth reflect childhood vulnerabilities. These vulnerabilities arise from multiple sources, but often reflect the experiences of parents. Childhood stress is indirectly influenced in utero by maternal stress (Leviton et al., 2016), as well as the structural racism experienced by parents. The ability of parents, for example, to obtain resources could be the difference between a malnourished or healthy child (Campo et al., 2008; Cheng et al., 2008). Growing up in an unsanitary environment and not having access to health care also influence childhood stress and development (Li and Daling, 1991; Oliveti et al., 1996; Ribas-Fitó et al., 2006).

This likelihood of risk is borne out in studies of groups subject to racial discrimination. Prelow and colleagues (2004), for example, investigated the impact of exposure to stressful events, impoverished neighborhoods, and discrimination on urban African and European American youths. They found that all three factors independently led to elevated risk of depression. This study points to the negative effects of stress, impoverished neighbors, and discrimination on mental health among U.S. youths.

Osypuk and Acevedo-Garcia (2008) investigated the effects of residential racial segregation on preterm births among whites and African Americans in 237 metropolitan areas, 22 of which were highly segregated. They used natality data from 1,944,703 births from the year 2000 to conduct their study. The results of their analyses showed that preterm births were more prevalent among African Americans than white living in hyper-segregated communities. The disparity was

less among African Americans and whites in less segregated communities. This study provides a structural explanation for preterm births, which have been shown to lead to a higher risk of disease later in life (Crump et al., 2011; Sipola-Leppänen et al., 2015).

Catov and colleagues (2016) followed up on this study by investigating disparities in the birth weight of their infants among African American and white mothers from 1997 to 2011 using prenatal hospital registry data from the Magee Obstetric Medical and Infant Database. The women included in their study delivered when they were at 37 to 41 weeks of gestation. They found that birth weight decreased over time for the children of both African American and white women. They also found, however, that African American women experienced greater reductions in thebirth weight of their children than did white women. This racial disparity in birth rate puts the children of African American women at greater risk of childhood leukemia later in life (Hjalgrim et al., 2003). This study, combined with the other studies cited above, provide evidence that racialized children are also affected by racist structures.

In this study, I focus on two indicators of childhood stress: vertebral neural canal (VNC) diameters and linear enamel hypoplasias (LEH). These are considered in more detail below.

Vertebral Neural Canal Diameters

Lumbar vertebral neural canal (VNC) diameters relate to childhood stress. VNC are used as indicators of good or poor neural and immune development during early growth (Clark et al., 1986; Porter et al., 1987; Clark et al., 1989; Jeffrey et al., 2003), as the timing of their fusion and their growth relate to childhood metabolism and stress. A reduced VNC represents an environment in which malnutrition and/or disease stunted growth (Clark et al., 1986; Watts, 2011). Measurements include anteroposterior (AP) and transverse (TR) diameters (Figure 1). In

general, lumbar AP diameters mature at around 3-5 years of age, while lumbar TR diameters mature at around 11-17 years of age (Hinck et al., 1966; Clark, 1988; Watts, 2013; Newman and Gowland, 2015). Little variation in age was found in maturation age between lumbar AP diameters (Watts, 2013). However, TR diameters for lumbar vertebra 1-4 matured by age 15, while the fifth lumbar TR diameter matured by 11 years of age (Watts, 2013). Thus, we may interpret AP diameter as indicative of infant and early childhood stress, while TR diameter is indicative of later childhood/adolescent stress.

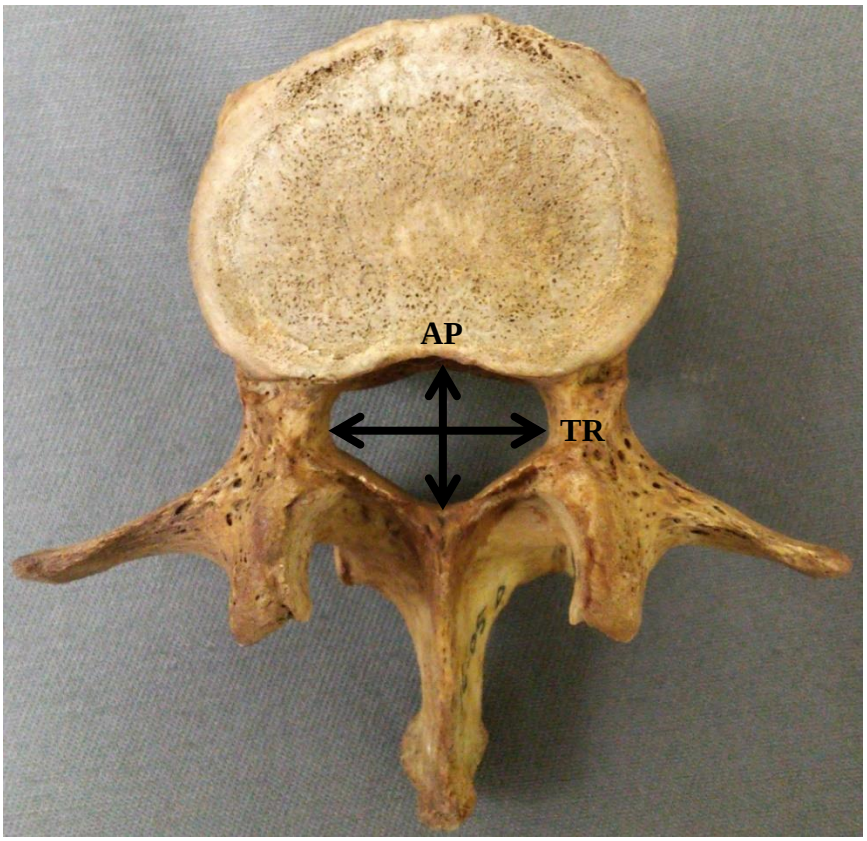

Figure 1. VNC anteroposterior (AP) and transverse (TR) diameter measurements, UT16-05D.

In addition to being indicators of childhood and adolescent stress, VNC diameters are useful because once their maturation stage is complete their diameters remain the same (except in cases where deformities of the spine occur) (Porter et al., 1980). So, unlike vertebral body height or long bone lengths, VNC diameters do not experience catch-up growth (Clark, 1988), which tends to confound indicators of childhood and adolescent stress. VNC are also more vulnerable to growth disruptions than dental and other skeletal tissues (Platt and Stewart, 1962).

Clark and colleagues (1986) sought to determine whether poor growth in utero and during early childhood affected development later in life, susceptibility to illness and disease, and life-span. Small VNC diameters represented poor early growth. They measured the thoracic and lumbar VNC AP and TR diameters of ninety skeletons from Dickson Mounds. They also recorded vertebral wedging as an indicator of decreased adult health. They found that small VNC diameters were significantly and inversely related to increased wedging and significantly and positively related to decreased life-span. They concluded that small VNC diameters predict poor adult health. This relationship was also demonstrated in another study by Clark and colleagues (1988).

Newman and Gowland (2015) compared VNC TR diameters and LEH in a sample of 96 sub-adults and 40 adults from post-medieval sites in England. They found that individuals with a higher frequency of LEH also demonstrated deficiencies in TR diameter. Watts (2015) also compared LEH and VNC diameters to ascertain childhood and adolescent stress. She looked solely at lumbar vertebra and included both TR and AP diameters. Her sample consisted of 941 skeletons from Late and Post Medieval London. Watts (2015) also compared these measures to age-at-death to determine whether individuals who experience LEH and/or reduced VNC died earlier than individuals without LEH and reduced VNC.

Watts (2015) found that LEH was not associated with VNC. Also LEH and AP VNC were not associated with adult mortality or future stress episodes indicated by stunted TR VNC diameters. That is to say that stress in early childhood as indicated by LEH and stunted AP VNC diameters did not have a lasting impact on adult longevity or health. Stress during later childhood/adolescence, indicated by reduced TR VNC diameters, was associated with increased risk of mortality.

Comparisons of LEH frequency and VNC (Newman and Gowland, 2015; Watts, 2015) reveal that LEH and VNC are not always associated. Employed separately they may paint different pictures of childhood and adolescent stress. Employed together, however, they may demonstrate a more nuanced picture of non-specific childhood and adolescent stress.

Linear Enamel Hypoplasias

Dental enamel is the hardest tissue in the human body (Goodman and Rose, 1990; Hillson, 1996). Enamel is deposited incrementally during dental development by enamel producing cells called ameloblasts during a process known as amelogenesis (Hillson, 1996; Reid and Dean, 2006). Two aspects make enamel suitable for use in studies investigating physiological stress in past populations, 1) it develops incrementally and 2) once deposited it does not remodel (Hillson, 1996). The incremental deposition of enamel has been shown to occur in a circadian fashion that enables researchers to estimate crown formation in day intervals (Reid and Dean, 2000; Reid and Dean, 2006). In addition, enamel's inability to remodel ensures that linear enamel hypoplasias (e.g. Figure 2) leave permanent markers indicative of nonspecific childhood stress and morbidity on the crown surface, markers that researchers can employ to reconstruct childhood life histories (Guatelli-Steinberg, 2004; Reid and Dean, 2006).

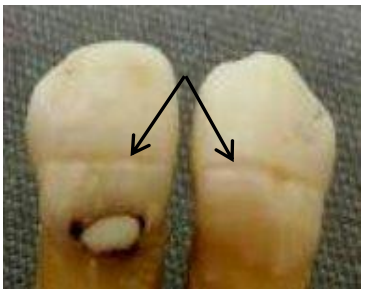

Figure 2. Linear enamel hypoplasia on mandibular canine antimeres, UT10-01D.

Linear enamel hypoplasias (LEH) represent deficiencies in enamel secretion (i.e., amelogenesis) during development (Guatelli-Steinberg et. al., 2012). They occur when ameloblasts endure stress that surpasses an unknown threshold leading to a disruption in enamel deposition (Goodman and Rose, 1990; Hillson and Bond, 1997). These disruptions manifest themselves on the crown surface as furrow/line defects, single or multiple pit defects, or exposed-plan defects (Goodman and Rose, 1990; Hillson, 1992; Hillson and Bond, 1997; Guatelli-Steinberg, 2004). Furrow defects appear as horizontal grooves on the tooth surface, while pits appear as horizontally oriented circular depressions. Exposed-plan defects occur when one or several planes of brown striae are exposed demonstrating a complete absence of enamel; this defect is also known as enamel aplasia (Goodman and Rose, 1990; Hillson and Bond, 1997). Furrow defects are the most common form, while exposed-plane defects are the most distinct and observable (Hillson and Bond, 1997).

The association of LEH with childhood stress derives from its association with Wilson bands or accentuated brown striae of Retzius in incisors, canines, and premolars (Goodman and Rose, 1990). In fact, Goodman and Rose (1990) found that 80% of the LEH found in 19 canines and incisors were associated with Wilson bands. LEH association with Wilson bands and the association of Wilson bands with physiological/systemic stress has deemed LEH one of the most

employed defects in investigations concerning the environmental stress experienced by humans and non-human primates in prehistoric, historic, and contemporary populations (Goodman and Rose, 1990; Guatelli-Steinberg, 2004; Skinner, 2012; Temple et al., 2013; Wilson, 2014; Gerber, 2014; Temple, 2014; Kierdorf et al., 2015; Żądzińska et al., 2015, Clark et al., 2014). LEH provide insight into varying stress experiences, exposures, and susceptibility (Newell et al., 2006). Susceptibility is related to and possibly caused by environmental stressors weakening the immune system (Temple, 2010), genetic predisposition, and/or cultural behaviors (Goodman and Rose, 1990). Morbidity reflects susceptibility to stress.

Most researchers contend that the majority of LEH defects are due to systemic stress resulting from malnutrition and/or disease or weaning stress (Goodman and Rose, 1990; Hillson and Bond, 1997, Guatelli-Steinberg et al., 2012; Turner and Armelagos, 2012; Sanberg et al., 2014). LEH in deciduous and permanent teeth can also be used to provide insight into the prenatal health of the child and the mother (Blakey and Armelagos, 1985; Storey, 1988; Goodman and Rose, 1990). However, LEH occurring in a localized or isolated manner provide insight into non-systemic stress (e.g., trauma) (Goodman and Rose, 1990; Guatelli-Steinberg et al., 2012). Goodman and Rose (1990) suggest that changes in LEH frequency between groups might be evidence that genetics is not the primary determinant of LEH manifestations (Goodman and Rose, 1990; Hillson and Bond, 1997). However, Goodman and Rose (1990) also note that changes in genetic makeup and environmental exposures to stress inducers occur over time and affect LEH frequency. In short, genetics cannot be ruled out as a factor influencing the distribution of LEH.

Lanphear (1990) examined the maxillary central incisors and mandibular canines of 296 individuals from the nineteenth-century Monroe County Poorhouse in Rochester, New York.

This population consisted mostly of foreign-born Europeans with a low socioeconomic status (SES) from the beginning of the industrial period. This population experienced higher levels of stress and an earlier weaning period compared to hunter/gatherer and agricultural populations. Lanphear (1990) found no significant difference in the distribution of LEH between males and females. Her findings suggest that the cultural transition from an agricultural to an industrial society had a negative effect on children with the lowest SES.

Temple (2010) investigated the effects of transitioning from a foraging to an agriculturally rice-based subsistence on two Jamon prehistoric populations from western and eastern Japan using LEH and cribra orbitalia (CO). He then compared these findings to the prehistoric Yayoi population responsible for introducing wet rice farming to Japan. The results of this analysis revealed that the subsistence transition significantly affected the distribution of LEH among Western Jamon, while the Eastern Jamon and Yayoi maintain similar LEH distributions after the introduction of wet rice agriculture. He argued, using the archaeological record, that the Eastern Jamon and Yayoi had annually stable and more nutritious foods economies than the Western Jamon before the transition. Consequently, the Western Jamon were expected to demonstrate the most significant change in LEH distribution, while little change was expected and reflected between the Easter Jamon and Yayoi.

Turner and Armelagos (2012) investigated the relationship between LEH, environment, and diet in an archaeological population from Machu Picchu, Peru. The results of this study revealed that local environment partially influenced LEH distribution on molar teeth. Diet and residential origin were both significantly associated with LEH. Guatelli-Steinberg and colleagues (2012) also examined the relationship between LEH distribution and environmental conditions in the mandibular canines of non-human primates (e.g., great apes). They argued that LEH distribution

was possibly associated with seasonality conditions such as rainfall, food or resource availability, and temperature. Their results provided mixed support for an association between seasonality and LEH.

The above studies reveal that one's physical environment is related to stress during development. Severe stress, in concert with genetics and individual susceptibility, can disrupt development and produce LEH of varying types, durations, and severity. The significance of each factor can fluctuate during development as a result of ecological or cultural changes (Goodman and Rose, 1990; Turner and Armelagos, 2012). The multitude of interactions and fluctuations produce varying morbidity rates across and within populations. How researchers interpret these rates depends on knowledge of possible etiologies of stress and the process through which stress results in LEH as well as how accurately, completely, and reliably data are obtained to test hypotheses.

Most studies employing LEH as an indicator of stress record LEH frequency, location on the crown surface, width, and age at stress onset. The primary goal of these endeavors is to obtain more information regarding LEH distribution, duration, and average age of stress in populations. While histological analyses are ideal for obtaining the most accurate and reliable information, they are simply too destructive to employ in every study. However, another means by which to obtain more accurate information concerning LEH distribution, duration, and age at stress onset (and ending) is to employ microscopic analysis of perikymata on the crown surface (Hillson and Bond, 1997; Reid and Dean, 2006).

Limitations and Considerations When Analyzing and Interpreting Stress

The Osteological Paradox

Anthropologists seek to reconstruct the life-ways of archaeological and contemporary populations from osteological remains. However, Wood and colleagues (1992) assert that demographic non-stationarity, selective mortality, and hidden heterogeneity limit analysis of morbidity (susceptibility to stress) and mortality in archaeological populations, which I extend to include most osteological samples. These three factors, which I explain in more detail below, are referred to collectively as the osteological paradox.

Demographic nonstationarity occurs when a population is no longer in a stationary state — "a state characterized by closure to migration, constant age-specific fertility and mortality, zero growth rate, and an equilibrium age distribution" (Wood et al., 1992:344). The main issue this factor highlights is that fertility influences age-at-death, while mortality has little to no effect on age-at-death (Coale, 1957; Keyfitz, 1985). Thus, life expectancy and age-at-death statistics are more likely measures of fertility and not mortality. Wood and colleagues (1992) acknowledged the longstanding awareness of demographic non-stationarity among anthropologists, but choose to spend the rest of their study describing the limitations of selective mortality and hidden heterogeneity.

Selective mortality points to the fact that samples do not represent the entire population at risk of disease or death at every age. That is to say that data pertaining to a particular type of stress (e.g., VNC diameters or LEH) in 20 year olds only pertains to individuals who died at 20 years of age in the skeletal sample. These data are incomplete because they do not include individual morbidity and mortality data from all individuals in the population who reached the age of 20 because they died at later years and are thus analyzed as members belonging to an

older cohort. Moreover, stress data collected for any given age group "is highly selective for lesions that increase the risk of death at that age" (Wood et al., 1992:344). Also, because all individuals who reached a given age cannot be observed, the stress that is observed is likely an overestimate of the actual amount of stress for that age group in the population.

The authors also note that some individuals in a sample may have experienced stressful events but died before the event was able to manifest itself in their skeleton and our data. The individuals with lesions may have been healthier than those without lesions because they survived the stress. Thus, a population with high levels of stress might demonstrate better overall health than one with low levels of stress. In addition, individuals with healed lesions might be healthier and less frail/susceptible because they survived than individuals with active lesions at the time of death. The authors argue that a highly selective sample is unavoidable and inherent in skeletal data.

Hidden heterogeneity in risk is the last factor of the osteological paradox Wood et al. (1992) mention. This factor points to the fact that individuals with varying frailty/susceptibility to disease and/or death compose osteological samples. Varying frailty/susceptibility might reflect differences in genetics, access to resources, or a number of other environmental changes. Heterogeneity in risk leads to a disconnect between population statistics and individuals' risk because researchers cannot interpret population rates of mortality and morbidity in terms of individual mortality and morbidity as one can never know for certain individual risk exposures and length of exposures. However, although anthropologists cannot determine with certainty individual risk of illness or death, they do and should continue to postulate reasonable theories and models of population structure, morbidity, and mortality with the understanding that there are only high probabilities and no certainties. These theories and models of stress should include

what epidemiologists, pathologists, clinicians, doctors, and medical anthropologists have learned from the interaction between environment and stress among modern populations. These studies provide the necessary backdrop against which to confidently theorize about the distribution as well as possible etiology of stress in historic and prehistoric populations.

Some studies (Goodman and Martin, 2002; Steckel and Rose, 2002) and recent methodological and technological advances led Wright and Yoder (2003) to assert that there has been progress in addressing the osteological paradox. Yet, no matter how advanced statistical models (as in Konigsberg and Holman, 1999), demographic models, population genetic models, and DNA techniques have become, anthropologists to date have not addressed all the components of the osteological paradox with 100% certainty. As the osteological paradox is difficult and perhaps impossible to address, many authors simply make mention of or ignore it altogether (Goodman and Martin, 2002). It is not only important to be aware of these challenges, but also to state them in the limitations of one's research. This is important because it keeps the issue in the purview of researchers and thus ensures the continual advancement of models and techniques that will aid in removing the limitations the osteological paradox presents.

Etiology of Stress

A complicated relationship exists between biological indicators of stress and particular activities and lifestyles. Populations have always been subject to varying environments, genetics, and stressors. No two populations are identical and the factors acting on different populations are neither identical nor proportional. The lack of identical populations is evident in the array of studies showing that what holds true for one population does not necessarily hold true for another (Moskowitz, 1984; Bridges, 1992, 1993; Larsen, 1997; Jurmain, 1999). The observation

that dinosaurs, an array of animals, and hominoids have all demonstrated signs of osteoarthritis and other indicators of metabolic disorders and stress for millions of years **(**Moodie, 1923; Wells 1964**)** only implies that stressors have coexisted with such groups for millennia. It says nothing of the possible etiology as etiological factors have undoubtedly changed over the millennia.

Nonetheless, etiology is what anthropologists attempt to determine when they encounter biological indicators of stress in the skeleton. Correlations existing between biological indicators of stress and specific activities (e.g., occupations or athletic) or lifeways (e.g., hunter-gather, agricultural, or industrial) that inform anthropologists of the activities and lifeways a group might have been involved in historically or prehistorically (Hawkey and Merbs, 1995; Kelley and Angel, 1987; Molnar, 2007). Such correlations also give anthropologists an idea of the possible causes (e.g., traumatic, infectious, or systemic) of certain lesions in the skeleton.

Even so, anthropologists must keep in mind that the etiological factors contributing to skeletal lesions are varied and possibly additive; some cannot be known with certainty (Johnson, 1965). In addition, different environmental factors can impact the skeleton in the same way, as bone can only react by adding more bone, subtracting bone, or a combination of the two (Steinbock, 1976; Mann and Murphy, 1990; Ortner, 1991; Lian and Stein, 1999). Third, comparing stress among two or more populations increases the number of possible etiologies contributing to skeletal lesions because the impact of factors such as genetics, hormones, metabolism, and environment from each population must be considered (Goodman et al., 1984, 1988; Krieger, 2011). Therefore, every study of stress and its possible etiology is population specific and should thus be examined critically (Leatherman and Jernigan, 2015).

Concluding Remarks

While publications regarding white-black/white-other comparisons abound, very few have explored variation within the white ethnic category and the effects of changing racialization processes on these groups. In fact, many studies (Jurmain, 1977; Aufderheide et al., 1985; Hershkovitz et al., 1999; de la Cova, 2011) continue to reflect the tradition of conflating all European groups at all historical time periods into the white racial category without regard to the process of racialization and their racial status as historically documented. That the nineteenth and twentieth centuries experienced several episodes of social, political, and economic unrest in the United States (Grimshaw, 1969; Drago, 1982; Crouch, 1992; Brundage, 1993; Bryant, 1994; Christian, 1995; Bergeron, et al. 1999; Painter, 2010) makes nonwhite European Americans and African Americans ideal samples for studying the effects of racialization on physiological stress. My study foregrounds this endeavor.

In no way does this research attempt to reify race by making it appear that distinct racial groups exist. Identity is often fluid and constantly being reinvented for better or worse (Meskell and Preucel, 2004). Yet, under "white" hegemony, race was a powerful instrument of oppression for people of African descent (Babson, 1990; Smedley, 1998). Perhaps race was also a powerful instrument of oppression for nonwhite European Americans as well.

The enormity and longevity of racial disparities in stress demand investigations into their maintenance. Examining the effects of racialization on stress in racialized European American historic samples offers the best opportunity to investigate racial disparities, as white European Americans initiated while nonwhite European Americans negotiated and resisted the process of racialization. As racial markers changed, the racist paradigm incorporated those changes to maintain the social hierarchy (Charatz-Litt, 1992; Bryd and Clayton, 2001; Orser, 2007).

Racialization cannot be ignored, not only because it produced social inequality, but also because it may have impacted stress and population health overall (Williams and Rucker, 2000; Gravlee, 2009; Williams et al, 2010). Therefore, this study explores whether changes associated with racialization impacted stress distributions using biocultural theory.

Chapter 4

MATERIALS AND METHODS

In this chapter, I discuss the skeletal samples used in this study and the analytical methods used to address the expectations presented in Chapter 1. Details about the groups represented by the skeletal sample and their context are provided in the Materials section below. Sampling strategies and techniques are also presented. In the Methods section, specific data collected by which to assess metabolic stress, the measurements taken from the skeletal remains for analyses, and statistical techniques are described.

Materials

Skeletal Samples

Three collections were employed in this study, the Hamann-Todd Osteological Collection, Robert J. Terry Anatomical Skeletal Collection, and William M. Bass Donated Skeletal Collection. All individuals included in this study were U.S.-born and of known sex, age, and year of birth. Individuals in the Hamann-Todd and Terry collections were born in states spanning the U.S. (Table A-1, Appendix I). Individuals in the Bass collection were primarily born in the Southeastern U.S. While there are individuals from different regions in the total sample, I combined the regions in this study to add statistical power to the analyses. The individuals in this sample are placed into one of four cohorts based on their dates of birth. These birth cohorts are Early (1828-1881), Middle (1882-1913), Intermediate (1914-1945), and Late (1946-1984). They reflect racial tension and immigration patterns in the nineteenth and twentieth centuries (see

Chapter 2) (Dillingham et al., 1911, Horne 1996; Clark and O'Donnell 1999; Dinnerstein et al. 2003; Roediger 2005). Table 1 shows the demographic distribution of the sample by birth cohort.

The selection criteria for including skeletons in this study restricted the total number of appropriate skeletons in these three collections. To satisfy the requirements for the discriminant function analysis (DFA) and finite mixture analysis (FMA), all individuals were required to have undamaged crania with all or the majority of their cranial elements present. Because a number of individuals in the Hamann-Todd, Terry, and Bass collections were autopsied, cranial traits were not observable on all crania (see *Nonmetric Traits* below). I scored all observable traits, and noted the absence of all unobservable traits. The nature of the samples, being primarily older individuals with low socioeconomic status, was such that defects on one or several lumbar vertebrae were common. Thus, it was not mandatory that all individuals have all vertebrae present to be included in this analysis. Individuals were included if they had at least one lumbar vertebra with at least one measurable vertebral canal diameter with no sign of disease (outside osteoarthritis that did not affect measurements of the vertebral canal), as the average diameter per vertebrae is treated as a separate unit of measure and compared to averages across cohorts. Because of dental wear and orthodontic disease, as well as storage and handling damage, most

Table 1. Sample composition: number of individuals by ancestry, time period, and sex.

Ancestry	Time Period												
	Early (1828-1881)			Middle (1882-1913)			Intermediate (1914-1945)			Late (1946-1984)			Sample
	M	F	Total	M	F	Total	M	F	Total	M	F	Total	Total
European American	82	35	117	35	29	64	97	37	134	40	40	80	395
African American	46	70	116	39	60	99	25	13	38	25	5	30	283

individuals were missing teeth or their teeth were damaged and not observable. Thus, like the cranial nonmetric traits, I examined teeth when possible for enamel hypoplasia, but observations were extremely limited. The standards for inclusion in the sample were not as strict to ensure that enough individuals were present from each birth cohort, particularly the early and late cohorts, to perform statistical analyses with a reasonable amount of power.

The Hamann-Todd collection is located in the Department of Physical Anthropology at the Cleveland Museum of Natural History in Cleveland, OH. This collection is comprised of 3,592 skeletons and represents one of the largest anatomical collections in the world (Hunt and Albanese, 2005). The skeletons in the collection were retrieved between 1910 and 1940. Dr. Carl August Hamann began collecting skeletons in the early 1900s from the Cleveland Cuyahoga County Morgue while professor of anatomy at Western Reserve University. In 1912, T. Wingate Todd assumed Hamann's position at Western Reserve University. It was Todd who greatly expanded the collection from a little over a hundred skeletons to well over 3,300 skeletons (Quigley, 2001; Hunt and Albanese, 2005). In this study's sample, the Hamann-Todd collection (n=142) contributed 13 European American females, 70 European American males, 29 African American females, and 30 African American males. These group designations were attributed to donors posthumously by Hamann and by Todd, and so do not reflect the nuances of self-identification sought in this study. This sub-sample includes African and European American males and females with birth years ranging from 1842 to 1913. Individuals from this sample are divided into Early (1828-1881) and Middle (1882-1913) birth cohorts.

The Terry collection is located in the Department of Anthropology at the Smithsonian's National Museum of Natural History in Washington, D.C. This collection was acquired through the joint efforts of Robert J. Terry and Mildred Trotter. The Terry Collection is composed of

1,728 individuals, including 436 European American males, 303 European American females, 508 African American males, and 355 African American females with known age and sex. Like the Hamann-Todd collection, racial identities of donors did not reflect ethnic or more specific group identity. The individuals in this collection died between 1910 and 1966 in St. Louis, Missouri (Hunt and Albanese, 2005). According to Hunt and Albanese (2005), individuals who died before the 1950s in this collection represent low socioeconomic status at the time of death. Individuals collected after the passing of the Willed Body Law of Missouri in 1955-1956 primarily represent the middle and upper classes (Quigley 2001). The Terry collection (n=251) contributed 45 European American females, 40 European American males, 108 African American females, and 58 African American males to my study sample. This sub-sample includes African and European American males and females with birth years ranging from 1828 to 1933. Individuals from this sample are divided into Early (1828-1881), Middle (1882-1913), and Intermediate (1914-1945) birth cohorts.

The Bass collection is located in the Department of Anthropology at the University of Tennessee, Knoxville. This collection is a contemporary collection of skeletal remains of individuals primarily from East Tennessee and the southeastern U.S. The Bass collection was initiated in 1981 by Dr. William M. Bass. It is mainly comprised of donated individuals and unclaimed individuals from Medical Examiner Offices. In 2011 there were over 1,200 individuals, of whom 91% were European American (~1,092) while 7% were African American (~84) (Shirley et al., 2011). Males comprised 70% and females 30% of the sample (Shirley et al., 2011). These numbers are constantly changing as the collection grows by about 90 individuals per year (Maijanen, 2014); the current number is close to 1,700 individuals. The Bass collection (n=285) contributed 83 European American females, 144 European American males, 11 African

American females, and 47 African American males. This sub-sample includes African and European American males and females with birth years ranging from 1902 to 1984. Individuals from this sample are divided into Middle (1882-1913), Intermediate (1914-1945), and Late (1946-1984) birth cohorts. No other sizable collection in the United States has individuals who were in the Late birth cohort; the next largest collection is the University of New Mexico's Maxwell Museum Donated Collection, which has approximately 300 individuals.

A key component of this study is determining the degree of biological affinity among individuals in the European American sample. As noted above, in none of the three collections used in this study is the European ancestry for individuals sufficiently and reliably recorded, and so how individuals might have been racially classified during their lifetime remains unknown. While subject to a number of assumptions (discussed below), one solution is to use Ossenberg's (2013) data to establish a discriminant function by which to ascribe European regional affinities to the skeletons in my study, using her nonmetric cranial traits. The ascribed racial statuses reflect how these groups would have been historically racialized, assuming their traits are reliably associated with ancestry-specific traits, and *not* how they self-identified.

A sample of 359 Europeans from Ossenberg's (2013) online database was employed in this study to establish the comparative sample for discriminant function analysis (DFA) (Table 2). Among these data, the British sample is comprised of individuals from St. Thomas Church. They represent nineteenth-century (1821-1873) British Canadians from Belleville, Ontario. The Bavarian, French, German, Czechoslovakian, and Russian samples are primarily historic anatomical samples from the University Medical Museum at the University of Tokyo. The Italian sample is also from a historic anatomical collection housed in the Department of Anatomy,

Table 2. Samples employed in this study from Ossenberg (2013).

European Region	Nationality	Males	Females	Total
Western	Bavarian	4	3	7
	British	126	105	231
	French	1	0	1
	German	5	2	7
Southern	Italian	44	43	87
Eastern	Czechoslovakian	10	3	13
	Russian	13	0	13
Total		203	156	359

University of Siena. These data were employed in this study because they represented an easily accessible sample of nonmetric cranial data from Western, Southern, and Eastern Europeans on which to base DFA approaches to assigning the research sample to European regions. Ossenberg's (2013) online database also provided a detailed description of the methods she used to score all of her traits. This was paramount in ensuring that I gathered data in a consistent manner.

Methods

Both nonmetric and metric data were measured from the skeletal sample. Here I provide a description of these traits and measurement methods, including those traits that reflect signs of metabolic stress encountered during development (see Chapter 3). I also discuss the reliability of my measurements, and the statistical approaches used for analysis. I did not utilize craniometric traits in this study because I was aware beforehand of the large number of individuals with autopsied skulls in my collection samples. I instead sought to maximize my time in each collection by employing cranial nonmetric traits to examine the degree of biological affinity. I was also aware of the lack of and poor preservation of teeth in two of my samples, so I did not

consider dental nonmetric traits, apart for signs of metabolic stress, or odontometrics as viable alternatives.

Cranial Nonmetric Traits

This study employed 13 cranial nonmetric traits described in Ossenberg (2013). See Table 3 for a list of all the traits and their corresponding figure numbers. These numbers reference pictures of the traits provided in Appendix II. All traits were scored as either absent (0), present (1), or indeterminate (9); indeterminate included unobservable traits. Although I initially recorded gradations in some traits, I chose to simplify my coding scheme due to a lack of confidence in the compatibility of my grades with Ossenberg's (2013). Therefore, I simplified Ossenberg's (2013) coding scheme to match mine for comparison. Accordingly, traits with minimal to extreme levels of expression (on one and/or both sides for bilateral traits) were scored as present. Traits with no level of expression (on both sides for bilateral traits) were scored as absent. Indeterminate referred to incidences where traits were unobservable bilaterally or absent on one side and unobservable on the other. Traits were unobservable when the area of their expression was fragmentary, autopsied, damaged by wear, fused, and/or obscured by debris or cartilage. For detailed trait descriptions and scoring methods see Appendix II).

I observed cranial nonmetric traits from European Americans in the Bass, Hamann-Todd, and Terry collections. All traits were observed with the aid of hand-held lighting for greater visibility. Traits were scored as present even if I only observed them on one side of the crania because there is still a genetic basis for these traits (Donlon, 2000).

As I noted above, these cranial nonmetric traits were used as markers of biological affinity between European Americans. Cranial nonmetric traits are considered polygenic traits

Table 3. Nonmetric traits employed in this study, based on Ossenberg (2013).

Name	Abbreviation	*Figure #
Postcondylar canal	POS	A-1
Lateral pterygoid plate foramen	LPF	A-2
Supraorbital foramen	SOF	A-3
Frontal grooves	FRG	A-4
Mental foramen double	MEN	A-5
Transverse fissure of basi-occiput	TRFS	A-6
Hypoglossal canal bridged or double	HYP	A-7
Foramen spinosum and/or ovale wall deficient	FSP	A-8
Intermediate condylar canal	ICC	A-9
Pterygospinous bridge (foramen Civinini)	CIV	A-10
Pterygobasal bridge	PTB	A-11
Trochlear spur	TRS	A-12
Mylohyoid bridge	MHB	A-13

(*) = Figure numbers correspond to figures in Appendix II.

(Releford and Lees, 1982). Polygenic traits are quantitative in that they are normally distributed because they are simultaneously influenced by multiple genes and environmental influences (Relethford and Lees, 1982; Buikstra, 1990). Researchers who employ polygenic traits in their analyses do so considering several assumptions. They assume that the environment has no impact or only randomly impacts polygenic traits, that such traits are moderate to highly heritable (i.e., genetic factors explain most of the traits' phenotypic variance), and that inheritance occurs by way of an equal and additive genetic model (Berry and Berry, 1967; Saunders and Popovich, 1978; Cheverud and Buikstra, 1981; Relethford and Lees, 1982; Cheverud, 1982, 1988; Ossenberg, 1984; Hauser and De Stefano, 1989; Donlon, 2000; Tyrrell, 2000; Relethford, 2003; Carson, 2006a,2006b; Marínez-Abadías et al., 2009). The equal and additive genetic model assumes that phenotypes are the result of equal contributions from specific alleles (Relethford and Lees, 1982). Lastly, researchers assume that morphological trait frequencies reflect allele frequencies (Hertzog, 1968; Cheverud and Buikstra, 1981;

Hallgrimsson et al., 2004) and that morphological distance reflects genetic distance (Hallgrimsson et al., 2004).

Wijsman and Neves (1986), however, revealed that nonmetric cranial data and genetic data are not always correlated, and so morphological distance is not always a good reflection of genetic distance. Moreover, the environment has been shown to affect morphology (Grüneberg, 1952; Riga, 2013); although the degree of this effect is unknown (Larsen, 1997). Thus, studies must take into account the potential impact of nutrition, disease, and infection on morphology (Bryd, 2014). The effects of geographic and cultural norms cannot be ignored either (Relethford and Lees, 1982; Edgar, 2009; Nikita et al., 2012). It is possible that the cranial nonmetric traits were affected by the environment in ways that changed their morphology over the timeframe of this study (1828-1984). Despite these findings, cranial nonmetric traits have been quite successful in correctly assigning known individuals (Prowse and Lovell, 1995; Christensen, 1998; Donlon, 2000; Hanihara et al. 2003) and so are used often to address questions concerning to relatedness (Hallgrímsson et al., 2004; Nikita et al., 2012; Mcllvaine et al. 2014). I will discuss the possible effects of the environment on nonmetric cranial traits in more detail in the Discussion.

Researchers should also consider the possibility that the traits they employ to discriminate populations might be traits that evolve at different rates, and may be subject to rapid changes in frequency in a population. This becomes an issue when the temporal gap between one's study sample and reference sample is large (Roseman and Weaver, 2004; Spradley et al., 2008). Consequently, it is possible that Ossenberg's data are inappropriate as a training sample because lengthy time gaps may exist between the common ancestors of her groupings (modern Europeans) and those groups that migrated to the United States. If traits that evolve rapidly, in

part due to founder effects or reduced mating pool sizes, are present among those I observed, it might not be possible to discriminate individuals in this study's sample using Ossenberg's data as a training sample.

Statistical Methods for Grouping Using Cranial Nonmetric Traits

Discriminant function analysis (DFA) is employed in this study to determine how the European Americans in this study will discriminate using a predefined sample of Europeans from Ossenberg's (2013) online database. The function produced by the DFA will predict group membership (Field, 2013). Unlike other statistics, DFA does not identify groups and is subject to poor performance when the individuals being assigned to a group membership are not included in the training sample used to develop the discriminant functions (Konigsberg et al., 2009; Kramer and Konigsberg, 1999). The use of this method assumes that individuals represent a majority of ancestry from one region of Europe, and not a mix of regions, which assumes traits are more strongly associated with one region over another and have high genetic heritability. DFA also assumes that sex ratios, sexual dimorphism, and morphology are the same between the training sample and unknown sample (Kramer and Konigsberg, 1999). This method also assumes that the environment (through gene-by-environment interactions) has little impact on the expression of the nonmetric cranial traits employed in this study. A potential danger of such an approach is that it reinforces typology over genetic variation in understanding human variation.

Another solution with fewer assumptions involves employing finite mixture analysis (FMA). Finite mixture analysis, unlike DFA, does not need a predefined training sample to predict group membership. FMA instead allows variance within a sample to discriminate among groups, parsing affinities among individuals into as many parsimonious groups as necessary based on the

traits employed (Kramer and Konigsberg, 1999). Moreover, it does not have to assume that sex ratios, sexual dimorphism, and morphology are the same between the training sample and unknown sample. FMA is also more accurate than other clustering analyses like k-means. FMA more accurately assigns unknown samples than k-means when using large sample sizes and complete datasets and when using small samples with missing data patterns (Kramer and Konigsberg, 1999).

One disadvantage in using FMA is that it cannot accommodate missing data. It is for this reason that the number of nonmetric cranial traits was limited to 13 traits. These 13 traits best discriminate the sample and allow for the most power with regard to sample size per group. These thirteen traits were chosen from the initial sample of twenty-five observed traits. Metopism, occipito-mastoid bone, asterionic bone, parietal notch bone, and squamo-parietal synostosis were removed as discriminatory traits because the study sample is composed of primarily older individuals and I did not want to include scores that were perhaps obliterated because of suture closure occurring with aging and not due to a lack of trait expression. Os japonicum, squamous style, accessory optic canal, and odonto-occipital articulation were eliminated because they were unobservable in100 or more individuals. I chose not to impute scores because, ultimately, imputed data is not an adequate substitute for actual observed data, so traits missing for 100 or more individuals were eliminated from the study. The retromolar foramen was eliminated because it was difficult to determine if some foramen resulted from porosity or were actually retromolar foramen. The pharyngeal fossa was present in 83% of the sample and absent in 8% of the sample. Tympanic dehiscence was present in 87% of the sample and absent in 10% of the sample. Thus, both the pharyngeal fossa and tympanic dehiscence had very little discriminatory power and were eliminated from the sample.

Vertebral Neural Canal (VNC) Size

The anteroposterior (AP) and transverse (TR) diameters neural canal diameters of every available lumbar vertebra from all 678 individuals in my sample were measured. I used lumbar vertebrae in this study because they are indicators of stress during development (Clark et al., 1986; Clark et al., 1989; Jeffrey et al., 2003; Newman and Gowland, 2015; see Chapter 3). A reduced VNC represents an environment in which malnutrition and/or disease stunted growth (Clark et al., 1986; Watts, 2011).

Measurements were not taken from one diameter and/or the other if vertebra were missing, presented trauma, were compressed, or were deformed. Measurements were also omitted if vertebral neural canals were sectioned, not fused, severely damaged by erosion, or obscured by osteophytes. A Mitutoyo digital caliper was used to measure each diameter to the nearest 0.01mm. The AP diameter represented the farthest distance from the posterior wall of the vertebral body to the "apex of a slight anterior bulge" opposite this measure on the neural arch (Eisenstein, 1983:189) (Figure 3). The TR diameter represented the widest interpedicular diameter or the greatest distance between the left and right pedicles (Eisenstein, 1983) (Figure 3).

Eisenstein (1977, 1983) endeavored to perform one of the first and most comprehensive analyses of VNC diameters between groups of European and African ancestry. He measured the lumbar AP and TR diameters of 78 white males, 35 white females, 256 South African black males, and 116 South African black females to determine mean diameters for European and African descent groups. These individuals are included in the Raymond Dart Collection in the Department of Anatomy at the University of Witwatersrand; they died between the 1920s and 1980s.

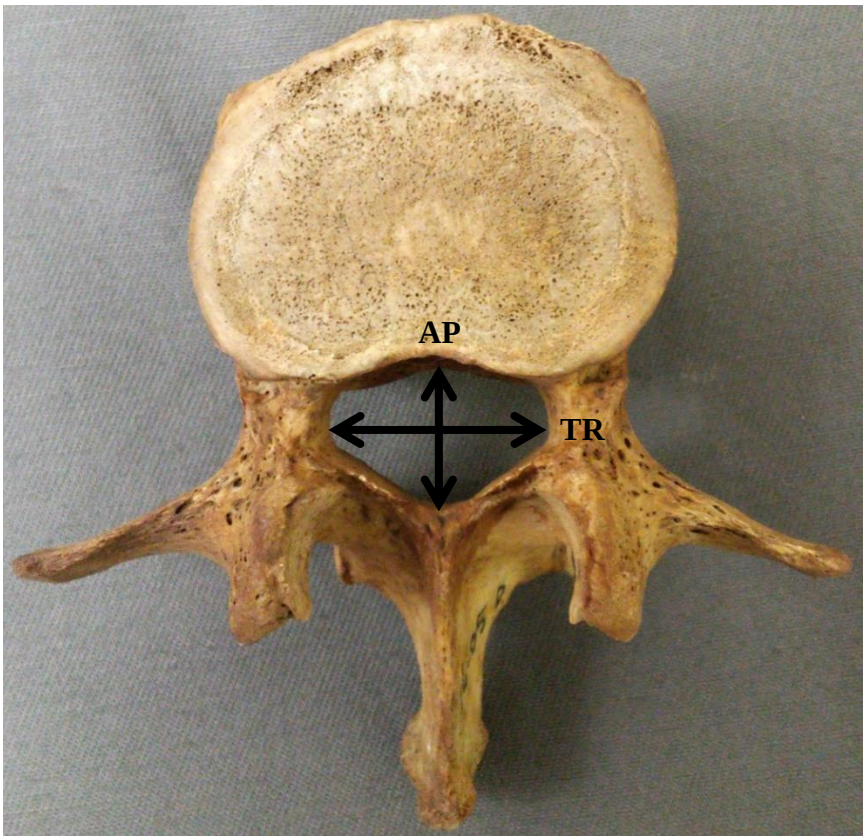

Figure 3. VNC anteroposterior (AP) and transverse (TR) diameter measurements, UT16-05D.

Eisenstein's (1977, 1983) means have become the standard in studies interested in spinal stenosis and normal variation in the lumbar spine (Porter and Pavitt, 1987; Clark, 1988; Papp et al., 1994; Jeffrey et al., 2003; Watts, 2013, 2015). He found the lower limits of his sample to be 10mm for anteroposterior diameter and 17mm for transverse diameter. His means are similar to those in previous studies (Verbiest, 1977; Ulrich et al., 1980) attempting to determine the normal range of lumbar canal variation. Verbiest (1977) analyzed lumbar vertebral canals from 92 patients from a follow-up study that lasted 27 years. He found that an anteroposterior diameter less than 10mm was pathological. Ulrich and colleagues (1980) used the CT scans from 60 individual's lumbar vertebral neural canals. They found the lowest limit of normal variation of

the anteroposterior diameter of the lumbar spine to be 11.5mm and the lowest limit of normal variation for the transverse diameter to be 16mm. The fact that these studies stem from different populations from different time periods under different environments suggest that a lower normal limit of 10mm for anteroposterior diameter and 16mm for transverse diameter is reasonable. Therefore, I employ these lower limits to interpret whether anteroposterior and transverse diameters fall below the normal range of variation for lumbar VNC diameters.

Linear Enamel Hypoplasia (LEH)

LEH were recorded from the permanent maxillary central incisors (UCI) and mandibular canines (LC) of all 678 individuals in my sample with suitable teeth present. These tooth classes were selected because they tend to be most affected by LEHs (Goodman and Rose, 1990). LEH defects were identified macroscopically as horizontal furrows or successive rows of pits along the labial or buccal surface of the tooth crown (Goodman and Rose 1990; Hillson 1992; Hillson and Bond 1997) (Figure 4). LEH defect was recorded as present (1) or absent (0). Minimally worn teeth with at least 70% of crown height intact, or all of the mid crown and cervical region were analyzed because LEH defects appear more prominent in these regions (Hillson and Bond 1997). I matched LEH defects across antimeres (right and left corresponding teeth, e.g., right and left maxillary central incisor) because matching establishes these defects as the result of a systemic etiology (i.e., an ailment affecting the whole body) and not the result of localized trauma (Hillson and Bond, 1997; King et al., 2005; Hubbard et al., 2009). Teeth with extensive calculus deposition, wear, staining, and/or dental work were excluded from the sample. Teeth were not cleaned or manipulated in any way to make LEH more apparent; the nature of the teeth was such that they were too fragile to endure manipulation.

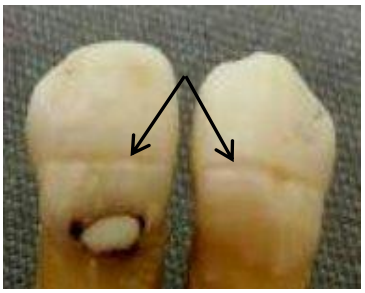

Figure 4. Linear enamel hypoplasia on mandibular canine antimeres, UT10-01D.

Intraobserver Error

To determine intraobserver repeatability, the two VNC diameters for each lumbar vertabra, nonmetric cranial data, and linear enamel hypoplasia (LEH) data from the maxillary central incisors and mandibular canines of a small sample of individuals were gathered for a second time at least a day after the initial data were collected. The Cronbach's α statistic was used to determine intraobserver replicability for vertebral neural canal (VNC) anteroposterior (AP) and transverse (TR) diameters. A total of 39 AP and 39 TR measurements were retaken more than a day after the original scores were obtained. These measurements show non-significant error and are highly repeatable; Cronbach's α equals 0.987 for AP diameter measurements, and Cronbach's α equals 0.981 for TR diameter measurements. These results indicate that 99% of variance is not due to measurement error for AP diameters and 98% of variance is not due to measurement error for TR diameters. In sum, both sets of data were gathered consistently throughout the study and the data are thus reliable.

I used chi-squared tests to determine whether I scored each nonmetric trait consistently during both scoring periods. The criteria for significance was a p-value less than or equal to 0.05 and an expected count or sample size (n) greater than five. The results are presented in Table 4. The results of the chi-squared reveal that there were significant differences between my first and

Table 4. Chi-Squared replicability results for nonmetric traits.

Trait	N	χ^2	df	*p*-value
UCI	7	7.000	1	0.143
LC	10	0.104	1	1.000
POS	19	8.972	1	0.105
LPF	19	2.554	1	0.178
SOF	19	15.354	1	<0.001
FRG	19	12.058	1	0.001
MEN	19	11.377	1	0.002
TRFS	19	0.101	1	1.000
HYP	19	12.436	1	0.001
FSP	18	9.360	1	0.012
ICC	18	10.811	1	0.003
CIV	19	19.000	1	0.053
PTB	19	15.240	1	<0.001
TRS	19	-	-	-
MHB	19	19.000	1	0.001

N = number of individuals; χ^2 = chi-squared; df = degrees of freedom.

second scoring trial for seven traits: SOF, FRG, MEN, FSP, ICC, PTB, and MHB. However, when I examine these data my scores changed from the first to the second trail no more than two times for each trait with a significant p-value. Upon further inspection of my statistics, I noticed that 50% or more of my cells had expected counts less than five for all of the nonmetric measures, including UCI and LC. This is a major violation of chi-squared tests because it drastically reduces test power (Field, 2013). So, I interpret both the significant and nonsignificant tests with caution. In the future I will collect more second trial data to ensure that none of my expected counts fall below five. TRS could not undergo chi-squared analyses because there was at least one incidence where there was only one expression of presence or absence for both trials combined. Chi-squared requires two categorical variables (e.g., POSfirst and POS) and two

expressions for both categorical variables (e.g., presence, absence); when there is only one expression the tests cannot be used (Field, 2013).

Statistical Analysis

Data were recorded in Microsoft Excel. Statistics were performed using R for Windows (R Core Team 2013) and IBM SPSS Statistics version 24.

Before proceeding with any analyses, I needed to determine whether traits were significantly sexual dimorphic, which would determine whether I performed these analyses with sexes combined or separated. Chi-squared analyses on categorical LEH data from the maxillary central incisors and mandibular canines of European and African American males and females were conducted to determine whether sexual dimorphism was present at the $p \leq 0.05$ significance level. The Shapiro-Wilks statistic was employed to determine whether VNC diameters were normally distributed among European and African American groups. There were a few instances where certain diameters were not normally distributed. Consequently, I conducted Mann-Whitney U tests on all VNC data to determine whether sexual dimorphism existed at the $p \leq 0.05$ significance level. Sexual dimorphism existed in VNC data, and warranted males and females being analyzed separately in this study for all data.

The nonmetric cranial data from the European American (EA) sample underwent DFA and FMA to establish distinct European American groups. Once individuals were assigned to a group they were then placed in either the Early (1828-1881), Middle (1882-1913), Intermediate (1914-1945), or Late (1946-1984) cohort depending on the year they were born. In this study I consider each EA group produced by the statistical analyses an EA racial group. This study assumes these groups depict isolated breeding populations that likely represented different racial groups. It is

not known which groups might have been identified as white or nonwhite. The use of the term race in this study is ambiguous.

Two-factor MANOVAs were employed to determine whether sex or racial differences existed among groups within each cohort for the ten VNC AP and TR diameters. Shapiro-Wilk's tests of normality were again performed for each EA racial group and African Americans by sex, VNC diameter, and birth cohort (e.g., white males L1 AP, Early cohort) to determine whether VNC diameters were normally distributed within each group combination. MANOVAs decrease Type I error by decreasing the number of times statistical tests are run on the same data. This method decreases the probability of attaining a significant result when there is no significant effect. MANOVAs also work well when there are several dependent variables, in this case five AP diameters and five TR diameters. They are also useful for determining whether groups differ along a combination of variables. Thus, I used MANOVAs to determine whether groups in one cohort significantly differed by sex, ascribed race, or a combination of sex and race. Pillai-Bartlett's trace ($p \leq .05$) was used to determine the significance of any sex, race, or interaction (sex and race) effect in each MANOVA, as it is the most resistant to slight deviations from normality in the data. Each MANOVA was followed up with box-plots to visually assess any significant race and interaction effects. Effects of sex were expected because of sexual dimorphism and only minimally addressed in this study.

One-way Analysis of Variance (ANOVA) tests, followed by the Games-Howell post-hoc tests, were used to determine whether group combinations (e.g., group 2 female L3 TR) significantly differed in VNC diameters from one cohort to the next; they were thus used to examine temporal patterns within sex and racial groups. Games-Howell post hoc tests were employed because they are powerful, accurate when sample sizes are unequal, and are not

sensitive to differences in population variances (Field, 2013). Significance was set at $p \leq 0.05$. One-way ANOVAs were used to highlight temporal trends in early/late childhood and adolescent stress. Kruskal-Wallis tests were performed on group combinations (e.g., African American male L1 AP) that did not meet the Shapiro-Wilk's test for normality requirement in at least one of the cohorts. Kruskal-Wallis tests were used to determine differences between VNC diameters from one cohort to the next for these groups. Thus, they are useful for determining trends in early/late childhood and adolescent stress when data are not normally distributed in one or more groups.

Fisher's exact tests were used to determine if LEH presence/absence data differed between groups in each cohort and within groups from one cohort to the next. The criteria for significance was a p-value less than or equal to 0.05. This test was employed because LEH sample sizes were often under five and this test can accommodate extremely small samples and maintain accuracy (Field, 2013).

The purpose of the above analyses is to answer the following questions, introduced in Chapter 1:

1. Are there distinguishable groups based on cranial trait variation within the sample of European Americans? What role might racialization play in shaping these distinctions?
2. Do these groups remain constant in size and proportion over time? If not, how do they change? What might explain these changes?
3. Do phenotypically distinct groups of European Americans have similar distributions of childhood and adolescent stress (LEH and VNC size) temporally?
4. How do trends in stress among racialized European American groups relate to trends in stress among African Americans? What might explain these relationships?

To review from Chapters 1 and 2, the samples in the study are divided into four birth cohorts that represent differing levels of racial tension. These cohorts are Early Period (1828-1881), Middle Period (1882-1913), Intermediate Period (1914-1945), Late Period (1946-1984). I have five expectations in this study that relate to the above questions and cohorts. First, I expect EA in the Early Period (1828-1881) to be more homogenous as the great migration of Eastern and Southern Europeans did not begin until 1882 (Dillingham et al., 1911). Second, I expect to see three distinct European American groups during the Middle Period (1882-1913) because of the migration of millions of Southern and Eastern Europeans during this period (Dillingham et al., 1911; Parrillo, 2000; Roediger, 2005). Third, I expect to see a drastic decrease in some groups and in increase in others during the Intermediate Period (1914-1945) because this was a time when acceptance of Southern and Eastern European migrants was burgeoning (Roediger, 2005). My fourth expectation is that European Americans will be mostly comprised of one group during the Late Period (1946-1984) due to "white" becoming an umbrella term for all individuals of European descent (Knowles and Prewitt, 1969; Clark and O'Donnell, 1999; Roediger, 2005). Last, I expect to see significant differences in the temporal distribution of indicators of childhood and adolescent stress among racialized European American and African American groups as racial classification mediates access to resources that affect health.

Chapter 5

RESULTS

This chapter presents the results of the statistical analyses described in Chapter 4. The limitations of the analytical approaches used were presented in that chapter, as well as intraobserver error. This chapter starts by demonstrating the predictability of the discriminant function analysis (DFA) using Ossenberg's European sample. In light of the DFA results, finite mixture analysis (FMA) is employed to assign individuals from the three osteological collections to subgroups that presumably represent different genetic and therefore possibly racialized groups. While, as stated in the Methods, it would be ideal to know the actual racial identities of individuals in my samples, they were not recorded and so require using a discriminating method as an alternative approach to identify groups in the presumably mixed European American sample within each time period. Subsequently, this chapter presents results of MANOVAs, ANOVAs, and Fisher's exact tests described under the "Statistical Analysis" section of Chapter 4. While I provide some limited discussion of result implications, I reserve my interpretations of results for the Discussion in Chapter 6.

Discriminant Function Analysis and Finite Mixture Analysis

The nonmetric cranial data from Ossenberg's (2013) sample were used to create a discriminant function to separate the European American individuals into Western, Eastern, and Southern affinity groups. The results of the function show that Ossenberg's traits do not strongly discriminate among individuals in her original data. The discriminate function analysis could correctly predict Ossenberg's Western Europeans 47.0% (n=134) of the time. Southern

Europeans follow in predictability with 48.6% (n=70). The discriminant function analysis performed the best when predicting Eastern Europeans; it predicted affinity 62.5% (n=8). The Eastern European group also had the smallest sample size, so this undoubtedly affected predictability power. In general the discriminant functions did not perform well (Table 5). Thus, the application of a discriminant function from this DFA to the individuals in this study from the United States would result in a high probability of error. Given the aforementioned problem of population structure differences in Ossenberg's data being different from those in the European American study sample, and temporal gaps between samples, the DFA results are not well suited for use in discriminating groups in this study.

Table 5. Discriminant Function Predictability for Ossenberg's (2013) Sample.

European Affinity	Predicted Group Membership in Percents		
	Western	Southern	Eastern
Western	47.0%	27.6%	25.4%
Southern	27.1%	48.6%	24.3%
Eastern	12.5%	25.0%	62.5%

The poor predictability of the DFA warranted the use of finite mixture analysis (FMA) to assess group membership. FMA allows groups to form based on the structure of trait variance within the sample without being forced by a predefined training sample, and thus does not require Ossenberg's data to predict group membership. FMA divided the sample of European Americans into four distinct groups, rejecting the first group as unparsimonious for a maximum likelihood best fit, and retaining three groups. These groups are designated as EA racial group two, three, and four. This study assumes these groups depict isolated breeding populations that likely represented different racial groups. It is not known which groups might have been

identified as white or nonwhite. The groups produced by the FMA are in Table 6. Notice that far fewer European Americans are assigned to racial group four in the United States sample. This is perhaps an artifact of the traits used to discriminate groups in the FMA. It is also possible that a different set of traits or more traits might discriminate into more or fewer groups.

Table 6. Sample distribution by time period and sex following finite mixture analysis.

Racial Group	Early Period			Middle Period			Intermediate Period			Late Period		
	Males	Females	Total	Males	Females	Total	Males	Females	Total	Males	Females	Total
Two EA	29	18	47	16	13	29	34	19	53	19	21	40
Three EA	48	16	64	18	12	30	55	15	70	20	16	36
Four EA	5	1	6	1	4	5	8	3	11	1	3	4
African American	46	70	116	39	60	99	25	13	38	25	5	30
Total	128	105	233	74	89	163	122	50	172	65	45	110

VNC Comparisons across Sex and Racial Group within Birth Cohorts

Once European Americans were separated into racial groups two, three, and four (Table 6), Shapiro-Wilk's tests of normality were performed for each affinity, including African Americans, by sex and VNC diameters (see Tables A-6 through A-13, see Appendix I). Out of 296 tests, 18 (6%) tests did not meet normality. MANOVAs, however, were still employed to determine differences among groups within each cohort for the ten VNC AP and TR diameters because they are resilient to mild departures from normality and decrease Type I error (Field, 2013). Pillai-Bartlett's trace ($p \leq .05$) was used to determine significance in each MANOVA.

As a follow-up to these comparisons, one-way ANOVAs were used to determine whether diameters changed within racial groups and sexes from one cohort to the next for all group VNC combinations that met the normality standard. The 18 tests that did not meet the Shapiro-Wilk's normality standard represented 16 race, sex, and VNC diameter combinations. Kruskal-Wallis

tests were performed on these 16 combinations to determine whether there were differences between VNC diameters from one cohort to the next, as this test does not assume normality.

The number, mean, and standard deviation for all of the individuals from each cohort employed in the MANOVA analyses are presented in Tables 7a and 7b, and the ranges are presented in Tables 8a and 8b. AP diameters in group two males, with the exception of L4 AP, increase from the early to the late cohort, while the TR diameters decrease from early to late cohort. The lowest range of L4 TR is 15.5mm. This measure falls below the normal range of variation for TR diameters (Verbiest, 197; Ulrich et al., 1980; Eisenstien, 1983). This suggests that group two males experienced late childhood or adolescent stress during the intermediate period (1914-1945). Group three males' TR diameters, except for L4 TR, decrease from the early to the late cohort. Group four males have very small sample sizes for all cohorts ($n \leq 8$). The middle and late cohorts have a sample size of less than or equal to one. L1- L3 AP diameters increase from early to intermediate, while L4 and L5 AP diameters decrease from early to intermediate. Group four TR diameters decrease from the early to intermediate cohort. These data, however, should be interpreted with caution as the small sample might be comprised of individuals on the extreme ends of AP and TR diameter variation in a larger sample. AP and TR diameters for African American males show no pattern between cohorts, yet the lowest range for L3 AP diameter is 9.76mm, which is below the normal range of variation for AP diameters (Verbiest, 1977; Ulrich et al., 1980; Eisenstien, 1983). This finding suggests that African American males experienced early childhood stress in the middle period (1882-1913).

Group two female AP diameters for L1-L4 showed a clear pattern as diameters decreased from the early to the middle cohort, increased from the middle to the intermediate cohort and then decreased again from the intermediate to the late cohort. L4 AP diameters displayed a

Table 7a. Mean lumbar vertebral neural canal (VNC) diameters by birth cohort for males. Birth cohorts are abbreviated as follows: ER = Early; MD = Middle; IN = Intermediate; LT = Late. Lumbar vertebral (L1-L5) canal diameters are taken from two dimensions: AP = Anteroposterior diameter; TR = Transverse diameter. Parenthetical figures are sample size and standard deviations to the nearest tenth millimeter.

Race	Cohort	L1 AP	L1 TR	L2 AP	L2 TR	L3 AP	L3 TR	L4 AP	L4 TR	L5 AP	L5 TR
Group two Male	ER	16.6 (26,1.9)	23.6 (29,1.7)	15.6 (26,1.8)	23.5 (29,1.5)	15.1 (26,1.8)	24.1 (26,1.5)	16.4 (21,2.2)	25.3 (21,1.8)	17.2 (21,2.4)	27.8 (17,2.7)
	MD	16.7 (16,1.1)	23.8 (15,1.0)	16.6 (16,1.5)	24.0 (16,1.2)	15.7 (14,1.3)	24.4 (14,1.3)	15.9 (14,1.5)	24.5 (14,1.8)	17.1 (14,2.2)	27.5 (6,2.7)
	IN	17.6 (33,1.5)	23.8 (33,1.7)	16.7 (34,1.6)	23.8 (34,1.9)	15.7 (33,1.9)	24.0 (34,2.1)	16.2 (34,2.6)	24.4 (33,2.9)	17.5 (29,2.6)	28.0 (26,2.5)
	LT	17.4 (19,1.2)	22.4 (19,1.1)	16.1 (19,1.2)	22.6 (19,0.9)	15.3 (19,1.3)	23.1 (19,1.2)	15.8 (19,2.0)	24.1 (19,2.0)	17.5 (18,2.6)	27.2 (18,3.0)
Group three Male	ER	16.7 (46,1.5)	23.8 (47,1.8)	16.1 (44,1.7)	23.7 (47,1.7)	15.4 (41,2.0)	23.6 (45,1.5)	15.6 (37,2.6)	23.7 (42,1.9)	17.3 (32,2.5)	26.5 (23,2.5)
	MD	16.5 (17,1.3)	22.7 (17,1.5)	15.2 (18,1.5)	22.7 (18,1.3)	14.2 (18,1.6)	22.8 (18,1.5)	15.3 (16,2.1)	23.8 (13,1.5)	16.6 (10,2.5)	27.2 (8,2.1)
	IN	17.4 (53,1.6)	23.4 (53,1.8)	16.1 (52,1.6)	23.4 (54,1.7)	15.2 (52,1.7)	23.7 (53,1.9)	15.9 (50,2.4)	24.2 (51,1.8)	17.3 (46,2.5)	27.5 (46,2.8)
	LT	17.7 (20,1.1)	22.8 (20,1.7)	15.9 (19,1.4)	22.5 (19,1.6)	15.1 (18,1.7)	22.8 (19,1.6)	16.1 (20,2.3)	23.9 (20,2.0)	17.2 (17,2.0)	28.0 (19,3.2)
Group four Male	ER	16.7 (4,2.0)	25.1 (4,2.6)	16.3 (5,2.2)	25.2 (5,2.8)	14.5 (4,2.6)	25.5 (4,2.9)	16.7 (5,2.0)	25.7 (5,2.4)	19.4 (3,4.9)	27.4 (3,1.5)
	MD	18.6 (1,-)	22.1 (1,-)	16.5 (1,-)	23.5 (1,-)	-	-	15.1 (1,-)	24.0 (1,-)	-	28.2 (1,-)
	IN	16.8 (7,0.8)	23.1 (8,1.6)	16.5 (8,1.1)	23.7 (8,1.5)	15.9 (8,1.1)	24.0 (8,1.1)	16.0 (8,2.0)	24.2 (8,1.8)	15.8 (6,2.9)	27.2 (6,2.7)
	LT	15.8 (1,-)	22.9 (1,-)	14.2 (1,-)	22.2 (1,-)	13.8 (1,-)	22.5 (1,-)	13.7 (1,-)	25.0 (1,-)	-	-
African American Male	ER	16.5 (45,1.3)	22.4 (45,2.0)	15.6 (43,1.5)	23.1 (44,1.9)	15.2 (44,1.8)	23.6 (44,2.1)	15.7 (39,2.0)	24.5 (34,1.9)	17.3 (30,2.8)	26.8 (23,3.0)
	MD	16.7 (39,1.6)	21.7 (39,1.7)	15.7 (38,1.5)	22.3 (37,1.6)	14.8 (37,1.9)	22.8 (38,1.7)	15.5 (36,2.5)	23.8 (35,2.0)	16.8 (34,2.2)	26.5 (29,2.9)
	IN	17.6 (23,1.0)	21.8 (23,1.2)	16.2 (22,1.1)	22.1 (22,1.5)	15.3 (24,1.5)	23.2 (24,1.9)	15.9 (25,2.1)	24.1 (23,2.3)	16.9 (22,2.2)	27.5 (18,2.5)
	LT	17.1 (23,1.2)	21.9 (23,1.4)	15.8 (23,1.5)	22.1 (23,1.6)	15.0 (24,1.7)	22.8 (24,1.6)	15.8 (25,2.1)	23.8 (25,1.8)	17.0 (22,2.4)	25.8 (21,1.9)

Table 7b. Mean lumbar vertebral neural canal (VNC) diameters by birth cohort for females. Birth cohorts are abbreviated as follows: ER = Early; MD = Middle; IN = Intermediate; LT = Late. Lumbar vertebral (L1-L5) canal diameters are taken from two dimensions: AP = Anteroposterior diameter; TR = Transverse diameter. Parenthetical figures are sample size and standard deviations to the nearest tenth millimeter.

Race	Cohort	L1 AP	L1 TR	L2 AP	L2 TR	L3 AP	L3 TR	L4 AP	L4 TR	L5 AP	L5 TR
Group two Female	ER	16.8 (15,1.7)	22.9 (17,1.7)	16.2 (17,1.6)	23.0 (16,1.5)	15.7 (16,1.4)	23.5 (17,1.2)	16.2 (17,2.0)	24.1 (16,1.5)	17.0 (12,1.1)	26.6 (13,2.4)
	MD	16.2 (10,1.1)	22.6 (11,2.3)	15.7 (11,1.3)	22.5 (12,2.4)	14.4 (11,1.5)	22.2 (12,1.9)	15.8 (11,1.8)	23.1 (12,1.6)	17.3 (8,2.3)	26.1 (9,2.4)
	IN	17.5 (14,1.5)	21.8 (15,1.6)	17.0 (15,1.8)	22.3 (17,1.5)	16.3 (18,2.4)	23.2 (18,1.5)	16.3 (18,2.6)	23.9 (17,1.9)	17.2 (17,2.6)	27.1 (17,2.8)
	LT	17.1 (19,1.1)	20.8 (20,1.7)	16.0 (20,1.2)	21.2 (20,1.6)	15.0 (21,1.5)	21.8 (21,1.7)	15.5 (20,1.6)	22.4 (20,1.9)	16.1 (18,1.9)	24.8 (20,2.7)
Group three Female	ER	16.0 (13,1.5)	22.9 (15,1.9)	15.8 (13,1.2)	22.9 (14,1.8)	15.5 (13,1.4)	23.4 (14,1.8)	15.3 (13,1.4)	23.7 (13,1.7)	16.4 (12,2.1)	26.8 (6,2.9)
	MD	17.0 (9,1.1)	21.8 (10,2.4)	16.0 (11,1.2)	23.1 (12,2.2)	15.3 (11,1.5)	23.3 (12,1.8)	15.9 (8,1.5)	23.8 (10,1.7)	17.2 (10,1.5)	26.3 (7,1.5)
	IN	17.5 (12,1.5)	22.6 (12,1.1)	16.8 (14,1.7)	23.0 (14,1.2)	16.1 (15, 2.2)	22.8 (15,1.3)	16.2 (14,1.8)	23.4 (14,1.7)	16.5 (15,2.4)	25.2 (12,1.5)
	LT	17.4 (16,1.4)	22.8 (16,1.6)	16.3 (16,1.7)	22.6 (16,1.4)	15.6 (15,1.4)	22.6 (16,1.6)	15.5 (15,1.9)	23.4 (16,2.0)	16.2 (12,1.5)	25.6 (12,2.6)
Group four Female	ER	16.8 (1,-)	23.8 (1,-)	-	23.6 (1,-)	15.5 (1,-)	23.3 (1,-)	14.6 (1,-)	24.1 (1,-)	13.1 (1,-)	27.9 (1,-)
	MD	15.7 (3,1.7)	22.3 (4,1.7)	15.2 (4,2.1)	22.9 (4,2.0)	15.5 (4,3.3)	23.3 (4,2.2)	16.3 (4,3.6)	24.3 (4,2.4)	16.4 (3,2.7)	26.4 (2,2.6)
	IN	18.1 (3,0.5)	21.6 (3,1.1)	16.5 (4,0.4)	22.7 (4,1.2)	15.9 (4,1.0)	22.4 (4,1.3)	16.1 (4,1.1)	23.2 (4,0.9)	16.8 (4,1.5)	25.5 (3,1.8)
	LT	16.7 (3,1.6)	20.8 (3,0.5)	15.2 (3,1.3)	20.8 (3,1.1)	13.6 (2,1.4)	21.8 (3,1.8)	14.9 (3,1.9)	22.8 (3,1.9)	16.3 (2,0.7)	27.4 (2,2.6)
African American Female	ER	16.7 (62,1.4)	21.5 (65,2.0)	16.2 (62,1.4)	21.8 (65,1.8)	15.6 (59,1.6)	22.4 (67,1.8)	16.1 (57,2.1)	23.4 (55,1.9)	16.9 (36,2.0)	26.6 (34,2.6)
	MD	16.9 (56,1.5)	20.7 (56,1.7)	16.1 (58,1.6)	21.2 (59,1.8)	15.4 (59,1.9)	22.0 (59, 2.0)	15.9 (56,2.6)	23.0 (56,2.2)	16.2 (44,2.5)	25.7 (46,2.5)
	IN	17.1 (12,1.5)	20.9 (12,1.8)	16.4 (11,1.6)	21.5 (11,1.8)	16.0 (13,1.8)	22.4 (13,2.0)	16.5 (11,1.4)	23.6 (10,2.2)	15.8 (8,1.6)	25.3 (7,2.1)
	LT	18.4 (5,2.0)	20.5 (5,2.4)	17.1 (5,1.8)	21.0 (5,2.4)	17.0 (5,1.6)	21.9 (5,2.5)	17.4 (5,2.3)	22.8 (5,2.0)	18.0 (5,2.5)	25.3 (5,2.4)

Table 8a. Ranges of lumbar vertebral neural canal (VNC) diameters by birth cohort for males. Birth cohorts are abbreviated as follows: ER = Early; MD = Middle; IN = Intermediate; LT = Late. Lumbar vertebral (L1-L5) canal diameters are taken from two dimensions: AP = Anteroposterior diameter; TR = Transverse diameter.

Race	Cohort	L1 AP	L1 TR	L2 AP	L2 TR	L3 AP	L3 TR	L4 AP	L4 TR	L5 AP	L5 TR
Group two Male	ER	12.29-20.59	20.67-27.02	11.95-19.01	20.02-26.23	12.15-17.87	19.88-26.04	11.78-20.96	18.94-27.79	13.72-21.54	20.08-31.76
	MD	14.71-18.34	22.04-25.53	14.46-19.75	22.08-26.17	13.89-18.10	22.41-26.22	13.61-18.62	21.30-26.77	13.40-21.43	24.14-31.04
	IN	15.15-21.02	20.49-27.48	12.28-19.64	20.97-28.12	11.96-20.05	20.18-28.49	12.08-23.40	15.50-32.73	13.12-23.50	23.47-34.39
	LT	14.48-19.48	20.52-24.68	14.00-18.82	21.13-24.13	12.94-18.31	20.97-25.23	12.96-21.09	20.14-29.53	12.41-21.64	23.06-34.27
Group three Male	ER	13.89-19.97	20.09-28.42	12.82-19.44	20.63-27.60	10.97-19.29	20.24-27.11	11.39-21.19	19.69-26.40	11.92-21.64	21.92-30.60
	MD	14.36-18.37	21.00-26.63	12.77-17.96	21.19-26.21	12.09-17.19	20.27-25.58	11.15-19.45	20.97-26.13	13.51-21.53	24.44-30.85
	IN	13.47-20.97	19.31-26.84	12.28-20.35	20.02-27.51	12.29-20.68	20.13-28.03	11.57-22.61	20.00-28.48	12.14-23.53	21.64-32.48
	LT	15.60-20.42	18.68-26.00	13.33-18.29	19.19-24.92	12.52-18.03	19.57-25.99	12.54-20.07	20.75-29.11	13.04-20.13	22.43-34.86
Group four Male	ER	14.59-19.48	22.85-28.77	13.79-19.49	23.49-30.13	12.43-18.02	22.48-29.32	14.43-19.12	23.30-29.31	14.12-23.72	26.09-29.00
	MD	-	-	-	-	-	-	-	-	-	-
	IN	15.79-17.85	21.21-25.48	15.00-18.01	21.53-25.90	13.69-17.13	22.43-25.64	12.43-19.00	21.92-26.43	12.77-20.92	23.71-29.95
	LT	-	-	-	-	-	-	-	-	-	-
African American Males	ER	14.01-20.45	18.23-27.95	13.07-18.63	19.00-27.80	11.42-20.79	19.16-28.09	11.52-21.10	20.35-28.20	11.12-22.77	21.10-32.52
	MD	10.55-20.30	18.06-25.64	10.05-18.37	19.56-25.22	9.76-18.27	19.71-26.48	10.07-21.64	19.66-27.91	12.98-22.58	21.89-32.54
	IN	15.83-19.08	19.37-23.89	14.28-17.88	19.22-25.04	12.64-18.42	19.96-27.43	12.49-20.84	20.25-28.68	13.72-22.82	23.76-32.17
	LT	14.69-19.77	19.22-24.62	13.39-18.18	19.25-25.04	11.38-18.10	19.26-25.61	10.40-19.19	20.36-27.07	10.85-20.64	23.20-29.82

Table 8b. Ranges of lumbar vertebral neural canal (VNC) diameters by birth cohort for females. Birth cohorts are abbreviated as follows: ER = Early; MD = Middle; IN = Intermediate; LT = Late. Lumbar vertebral (L1-L5) canal diameters are taken from two dimensions: AP = Anteroposterior diameter; TR = Transverse diameter.

Race	Cohort	L1 AP	L1 TR	L2 AP	L2 TR	L3 AP	L3 TR	L4 AP	L4 TR	L5 AP	L5 TR
Group two Female	ER	14.69-20.70	20.23-25.80	14.23-19.77	21.02-26.13	13.15-18.33	21.37-25.47	12.73-19.74	22.04-27.85	14.07-18.20	23.58-32.10
	MD	14.29-18.01	20.02-26.49	13.34-18.30	20.21-27.27	11.51-17.06	20.12-26.32	13.14-19.41	21.07-26.34	14.60-21.71	22.72-29.78
	IN	14.61-19.71	19.06-24.88	12.06-18.96	18.98-24.53	10.48-21.81	19.26-25.02	9.27-21.55	19.94-27.45	11.43-22.23	23.13-33.86
	LT	14.07-18.86	17.17-23.51	13.02-17.69	18.41-24.40	10.50-17.89	18.68-25.06	10.92-17.72	19.32-25.60	12.85-19.65	20.51-29.30
Group three Female	ER	14.05-18.72	20.89-26.45	13.41-17.80	20.05-25.98	12.59-17.07	20.04-26.84	12.81-17.48	21.35-26.37	12.94-19.75	23.13-29.81
	MD	15.65-18.89	19.36-25.92	13.13-17.31	20.09-26.67	13.05-18.24	20.25-25.80	13.48-18.75	20.76-26.26	15.53-19.54	24.60-28.55
	IN	14.53-19.66	20.95-23.94	13.80-19.00	20.75-24.77	12.51-20.07	19.99-25.10	13.51-20.24	20.33-26.72	12.05-22.47	21.50-27.62
	LT	15.13-20.02	19.40-25.32	13.22-18.98	20.00-25.08	13.52-18.87	20.01-25.60	11.49-18.86	19.36-26.84	13.05-18.59	20.82-30.72
Group four Female	ER	-	-	-	-	-	-	-	-	-	-
	MD	13.81-17.17	20.27-24.36	13.05-17.46	20.33-24.65	12.51-18.88	20.76-25.70	13.00-20.25	22.12-27.35	14.76-19.52	24.54-28.18
	IN	17.48-18.51	20.25-22.31	16.15-16.90	21.15-23.73	14.81-16.76	21.30-24.24	14.52-16.97	22.17-24.13	14.78-18.31	23.49-27.04
	LT	14.92-17.68	20.20-21.19	13.77-16.38	19.62-21.57	12.62-14.53	19.84-23.43	12.68-16.16	21.01-24.76	15.80-16.75	25.52-29.24
African American Female	ER	12.87-19.91	17.27-27.36	12.43-18.80	17.37-25.38	12.46-18.82	17.79-26.12	11.68-22.21	19.94-27.29	12.60-20.47	21.40-31.15
	MD	13.47-20.07	17.39-26.28	12.78-19.46	17.75-25.16	11.70-21.33	18.43-27.96	11.35-22.70	19.70-29.83	11.27-20.81	21.63-31.16
	IN	15.01-19.49	17.98-23.46	13.94-18.83	18.73-24.97	13.05-18.84	19.79-26.38	14.10-19.21	21.52-27.34	12.90-17.59	22.21-28.84
	LT	15.54-20.91	17.49-23.44	14.98-19.63	17.85-23.66	14.72-18.84	18.70-25.48	13.53-19.38	20.03-25.50	15.32-21.43	21.30-27.95

minimal range of 9.27mm. This range is below the normal range of variation for AP diameters (Verbiest, 1977; Ulrich et al., 1980; Eisenstien, 1983) and suggests that group two females experienced stress during the intermediate period. TR diameters decreased from the early to the late cohort. Group three females demonstrated an increase in L1-L4 AP diameters from the early to the late cohort. TR diameters decreased from the early to the late cohort. Among females in group four AP diameters from the middle to the intermediate cohort increased for L1-L3 and L5, while L1-L4 TR diameters decreased from the middle to the late cohort. With small numbers of individuals in each cohort for group four females ($n \leq 4$), these data do not provide meaningful interpretations. AP diameters among African American females increase from the early to the late cohort. TR diameters decrease from the early to the late cohort among African American females. However, due to the small sample size (n=5) for African American females in the late cohort, results from the AP and TR diameters in this cohort cannot be trusted.

The results of the MANOVAs are displayed in Table 9. These were performed within each birth cohort, comparing the sexes and racial groups. The MANOVA for the early cohort (1828-1881) showed that there was a significant race effect (Pallai's Trace = 0.666, $F(30, 183) = 1.740$, $p = 0.015$) for the transverse diameters in L1-L2; racial groups were significantly different in the transverse diameters of L1 ($p = 0.025$) and L2 ($p = 0.023$). Boxplots of L1 and L2 transverse diameters for the early cohort are shown in Figures 5 and 6. The race effect is apparent in the distinction between European American group two and group three from the European American group four and African Americans with regard to L1 transverse diameters. Group four appeared to have significantly larger L1 TR diameters than all the other groups, while African Americans have significantly smaller L1 TR diameters. However, the L2 transverse diameters among all three European American groups are similar and set apart from African Americans, who again

Table 9. Results of MANOVAs comparing VNC diameters within cohorts between sexes and racial groups. Bold text indicates statistically significant Pallai's Traces for each MANOVA.

	Males	Females	Total	Sex Effect	Race Effect	Sex*Race Interaction
Birth Cohort	*N*	*N*	*N*	P-value	P-value	P-value
Early	43	32	75	0.234	**0.015**	0.636
Middle	39	43	82	0.564	0.095	0.987
Intermediate	78	25	103	**0.028**	0.068	0.145
Late	51	32	83	**0.022**	0.195	0.365

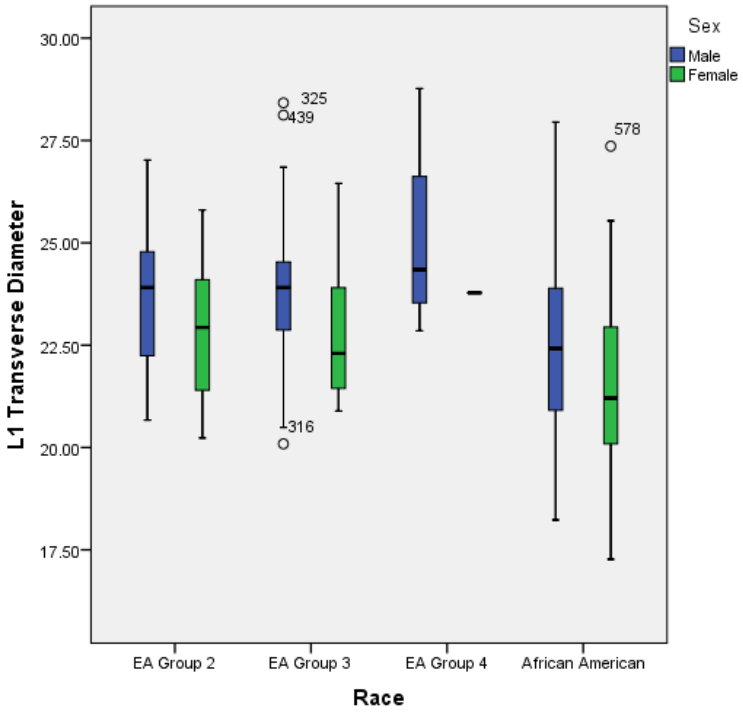

Figure 5. Early Cohort (1828-1881) L1 Transverse Diameter among Race/Sex Groups.

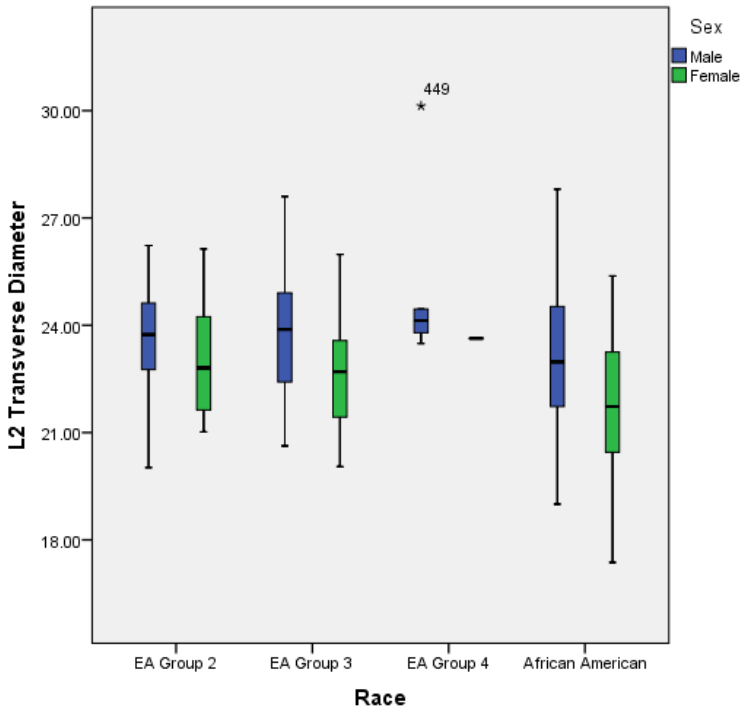

Figure 6. Early Cohort (1828-1881) L2 Transverse Diameter among Race/Sex Groups.

have significantly smaller diameters. These results suggest that adolescent stress among at least European American groups two and three was similar during the early (1828-1881) period, while European Americans in group four and African Americans experienced different levels of stress. It must be noted, however, that the sample size for group four males was less than five and for group four females was one or zero (Table 7a and 7b). Thus, the significant race effect driven by group four during the early cohort for L1 TR is not reliable as the sample size is too small. There was no sex main effect or interaction effect in the early cohort. Among individuals in the middle cohort (1882-1913) there was no sex main effect, race main effect, or interaction effect (see Appendix III, Figures A-24 through A-33).

The MANOVA for the intermediate cohort (1914-1945) showed that there was a significant sex effect (Pallai's Trace = 0.201, $F(10, 86) = 2.159$, $p = 0.028$). Sex groups were significantly different in the transverse diameters of L1 ($p = 0.032$) and L3 ($p = 0.050$). Boxplots of L1 and L3

transverse diameters for the intermediate cohort are shown below (Figures 7 and 8). There was also a significant sex effect (Pallai's Trace = 0.255, $F(10, 67) = 2.299$, $p = 0.022$) for transverse diameters in the late cohort (1946-1984). Sexes were significantly different in transverse diameters of L1 ($p = 0.028$), L4 ($p = 0.025$), and L5 ($p = 0.009$). Boxplots of L1, L4, and L5 transverse diameters for the late cohort are presented in Figures 9, 10, and 11. Sex effects are expected, and reflect adolescent sexual dimorphism when they occur in the transverse diameter because this diameter matures between 11 and 17 years of age (Hinck et al., 1966; Clark, 1988; Watts, 2013; Newman and Gowland, 2015). There was no race main effect or interaction effect on VNC diameters in the intermediate or late cohorts.

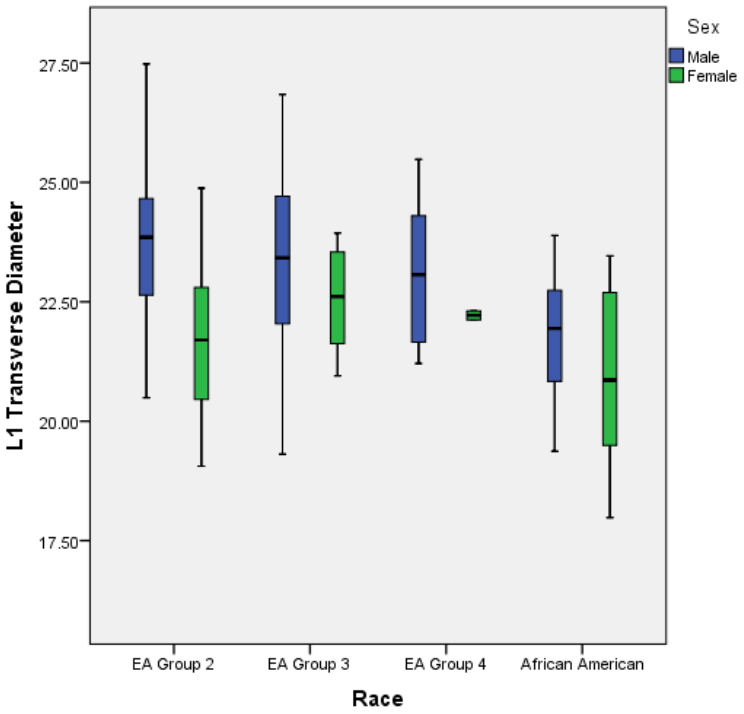

Figure 7. Intermediate Cohort (1914-1944) L1 Transverse Diameter among Race/Sex Groups.

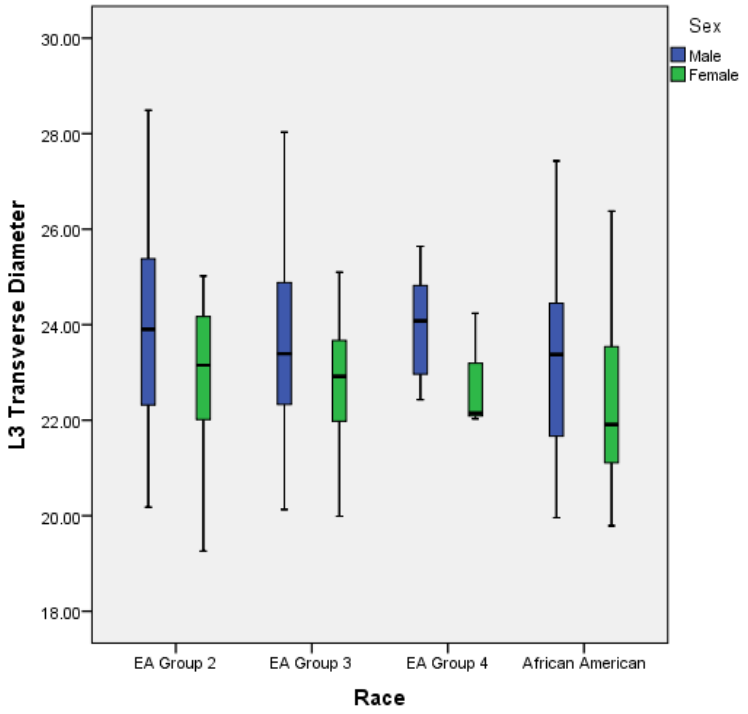

Figure 8. Intermediate Cohort (1914-1944) L3 Transverse Diameter among Race/Sex Groups.

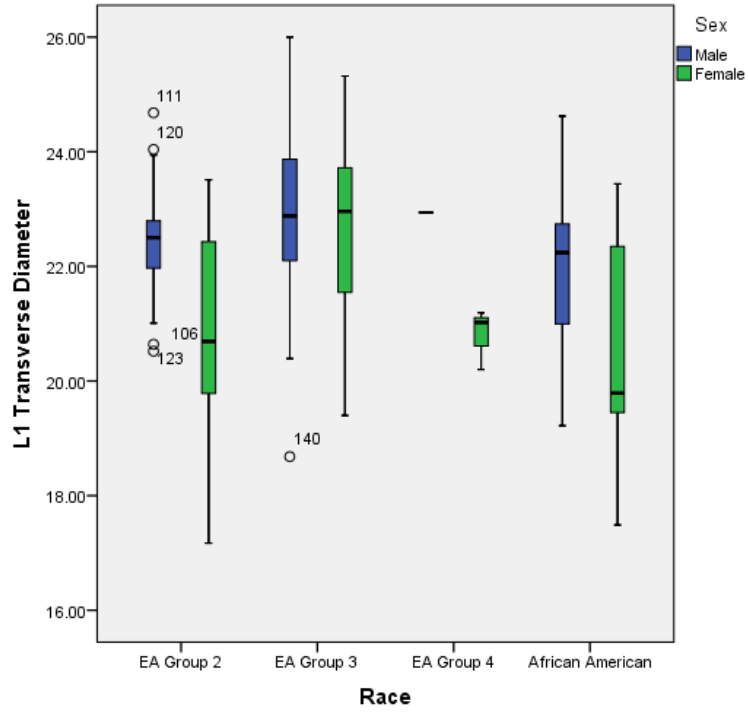

Figure 9. Late Cohort (1945-1984) L1 Transverse Diameter among Race/Sex Groups.

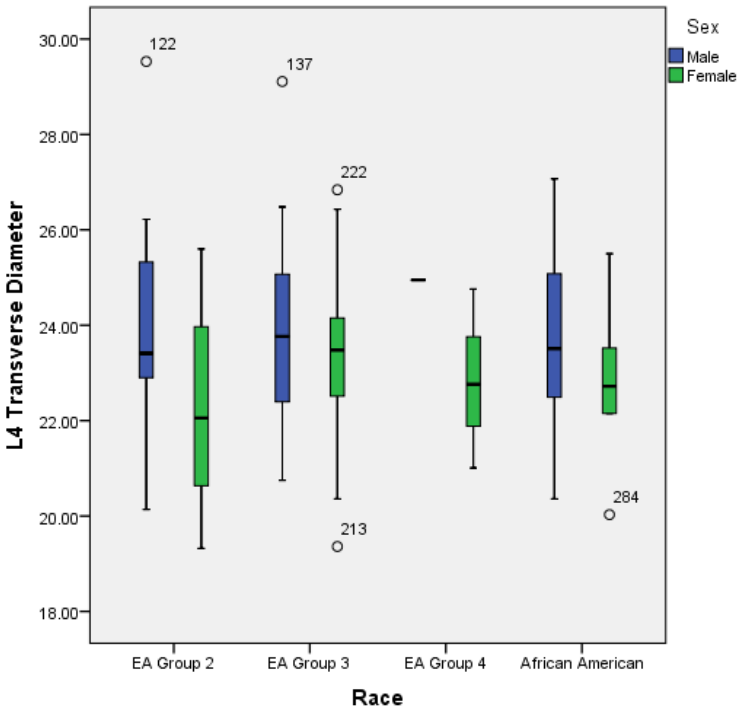

Figure 10. Late Cohort (1945-1984) L4 Transverse Diameter among Race/Sex Groups.

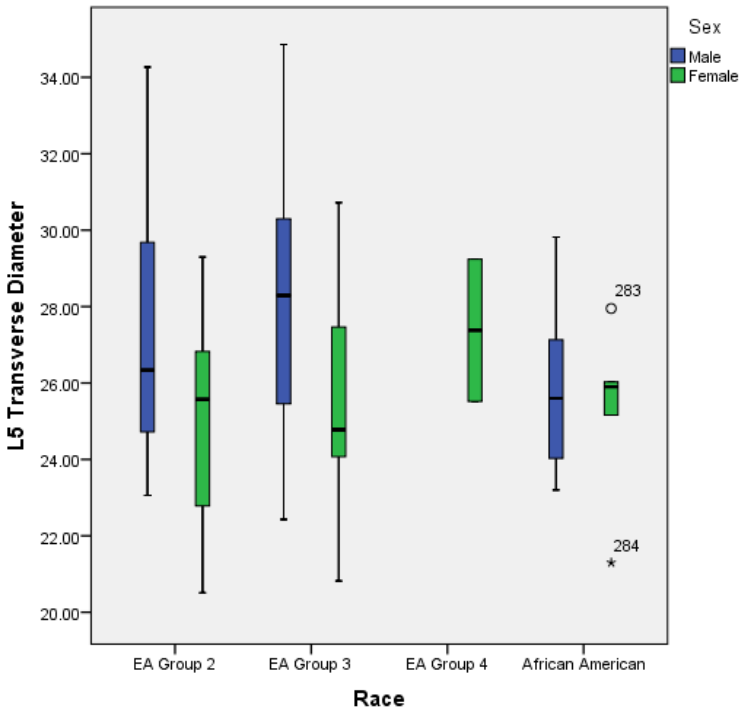

Figure 11. Late Cohort (1945-1984) L5 Transverse Diameter among Race/Sex Groups.

VNC Comparisons across Birth Cohorts within Sex and Race

One-way analysis of variance (ANOVA) tests were employed to determine whether AP and TR diameters changed significantly from one cohort to the next within each sex and race group (e.g. group two males, African American females, etc.). For reference, the number, mean, and standard deviation for VNC diameters by cohort for European American group two males are shown in Table 7 above. There is a significant effect of time period on L1 AP diameter for group two males ($F(3, 90) = 2.704$, $p = 0.050$). However, the Games-Howell post-hoc results show no significant change in L1 AP diameters from one cohort to the next despite the significant effect of time overall (Table 10). There is a significant effect of time period on L1 TR diameter, ($F(3, 92) = 3.952$, $p = 0.011$). L1 TR diameters significantly decreased from the early to late ($p = 0.026$), middle to late ($p = 0.003$), and intermediate to late ($p = 0.004$) cohorts (Table 11). There is a significant effect of time period on L2 TR diameter, ($F(3, 94) = 3.535$, $p = 0.018$). L2 transverse diameters also significantly decreased from the early to late ($p = 0.037$), middle to late ($p = 0.003$), and intermediate to late ($p = 0.011$) cohorts (Table 12). The Kruskal-Wallis statistic was used to detect a change through time in L3 TR diameter for group two males because one of the cohort groups did not meet normality. The Kruskal-Wallis statistic showed that there was a significant effect of time period on L3 TR diameter ($H(3) = 8.076$, $p = 0.044$) (Table 13). However, the pairwise comparison results show that none of the adjusted p-values fall below the $p = 0.05$ significance level (Table 14). Despite the significant overall effect of time, the results of the pairwise comparisons indicate that there is no significant difference in L3 TR diameter from one cohort to the next. The significant increase in L1 AP diameter from the early to late cohort might represent a decrease in early childhood stress over time for group two males. The

Table 10. ANOVA *p*-values for L1 anteroposterior diameters for group two males, multiple cohort comparisons. Bolded numbers indicate significant results. Letters next to numbers indicate the change from the last cohort: d = decrease in canal size; i = increase in canal size.

Cohort	Early	Middle	Intermediate
Early			
Middle	0.999		
Intermediate	0.137	0.095	
Late	0.398	0.360	0.895

Table 11. ANOVA *p*-values for L1 transverse diameters for group two males, multiple cohort comparisons. Bolded numbers indicate significant results. Letters next to numbers indicate the change from the last cohort: d = decrease in canal size; i = increase in canal size.

Cohort	Early	Middle	Intermediate
Early			
Middle	0.972		
Intermediate	0.970	1.000	
Late	**0.026d**	**0.003d**	**0.004d**

Table 12. ANOVA *p*-values for L2 transverse diameters for group two males, multiple cohort comparisons. Bolded numbers indicate significant results. Letters next to numbers indicate the change from the last cohort: d = decrease in canal size; i = increase in canal size.

Cohort	Early	Middle	Intermediate
Early			
Middle	0.638		
Intermediate	0.904	0.971	
Late	**0.037d**	**0.003d**	**0.011d**

Table 13. Kruskal-Wallis results for group combinations that did not meet normality. Abbreviations: H = test statistics; df = degrees of freedom; TR = Transverse; AP = Anteroposterior; Adjusted *p*-value = the *p*-value from the pairwise comparisons.

Sex	Race	Vertebra	Diameter	N	H	Df	Adjusted *p*-value
Male	Group two	L3	TR	93	8.076	3	0.044
		L4	TR	87	6.453	3	0.092
		L5	TR	67	2.349	3	0.503
Male	Group three	L2	TR	138	9.561	3	0.023
		L3	AP	129	5.681	3	0.128
		L4	TR	126	1.099	3	0.777
Male	Group four	L2	TR	15	2.166	3	0.539
Male	African American	L1	AP	130	12.664	3	0.004
		L2	AP	126	3.399	3	
		L5	AP	108	1.016	3	
Female	Group two	L2	AP	63	8.237	3	0.041
		L2	TR	65	10.416	3	0.015
		L5	AP	55	4.174	3	0.243
Female	Group three	L1	TR	53	3.355	3	0.340
Female	African American	L4	AP	129	3.782	3	
		L4	TR	126	2.436	3	

Table 14. Kruskal-Wallis adjusted *p*-values for L3 transverse diameters for group two males, pairwise cohort comparisons. Bolded numbers indicate significant results. Letters next to numbers indicate the change from the last cohort: d = decrease in canal size; i = increase in canal size.

Cohort	Early	Middle	Intermediate
Early			
Middle	1.000		
Intermediate	1.000	1.000	
Late	0.076	0.093	0.355

significant decrease in L1-L2 TR diameters over time might represent an increase in late childhood and adolescent stress over time for group two males.

The number, mean, and standard deviation for VNC diameters by cohort for group three males are shown in Table 7 above. The ANOVA results show that there is a significant effect of time period on L1 AP diameter ($F(3, 132) = 3.778$, $p = 0.012$). L1 AP diameters significantly increased from the early to late cohort (p=0.033) and middle to late cohort (p=0.023) (Table 15). The Kruskal-Wallis statistic also showed that there was a significant effect of time period on L2 TR diameter, H (3) = 9.561, $p = 0.023$) (Table 13). However, the pairwise comparison results show that none of the adjusted p-values fall below the $p = 0.05$ significance level (Table 16). Despite the significant overall effect of time, the results of the pairwise tests indicate that there is no significant difference in L2 TR diameter from one cohort to the next.

The increase in L1 AP diameter signifies a decrease in early childhood stress over time. There is no significant effect of time on AP and TR diameters among group four males. This lack of significance is perhaps due to the small sample size for group four males ($n \leq 8$) across cohorts. Thus, any results for group four males are not reliable.

The Kruskal-Wallis statistic was used to detect a change through time in L1 AP diameter for African American males because one of the cohort groups did not meet normality. L1 AP diameters significantly differ over time ($H(3) = 12.664$, $p = 0.005$) (Table 13). Pairwise comparisons with adjusted p-values show a significant increase in L1 AP diameters from the early to intermediate (p=0.004) cohort (Table 17). It appears that African American males experienced a decrease in early childhood stress through time.

The number, mean, and standard deviation for VNC diameters by cohort for group two females are shown in Table 8. There is a significant effect of time period on L1 TR diameter for

Table 15. ANOVA *p*-values for L1 Anteroposterior diameters for group three males, multiple cohort comparisons. Bolded numbers indicate significant results. Letters next to numbers indicate the change from the last cohort: d = decrease in canal size; i = increase in canal size.

Cohort	Early	Middle	Intermediate
Early			
Middle	0.891		
Intermediate	0.155	0.087	
Late	**0.033i**	**0.023i**	0.828

Table 16. Kruskal-Wallis adjusted *p*-values for L2 transverse diameters for group three males, pairwise cohort comparisons. Bolded numbers indicate significant results. Letters next to numbers indicate the change from the last cohort: d = decrease in canal size; i = increase in canal size.

Cohort	Early	Middle	Intermediate
Early			
Middle	0.157		
Intermediate	1.000	0.712	
Late	0.054	1.000	0.312

Table 17. Kruskal-Wallis adjusted *p*-values for L1 anteroposterior diameters for African American males, pairwise cohort comparisons. Bolded numbers indicate significant results. Letters next to numbers indicate the change from the last cohort: d = decrease in canal size; i = increase in canal size.

Cohort	Early	Middle	Intermediate
Early			
Middle	1.000		
Intermediate	**0.004i**	0.058	
Late	0.403	1.000	1.000

group two females ($F(3, 59) = 4.547$, $p = 0.006$). The multiple comparison results show that L1 TR diameter significantly decreased from the early to late ($p=0.005$) cohort (Table 18). There is a significant effect of time period on L3 AP diameter ($F(3, 62) = 3.226$, $p = 0.028$). L3 AP diameters, however, did not show a significant change from one cohort to the next (Table 19). L3 TR diameters showed a significant effect of time ($F(3,64) = 4.965$, $p = 0.004$). L3 TR diameter significantly decreased from the early to the late cohort ($p = 0.006$) and from the intermediate to the late cohort ($p = 0.031$) (Table 20). L4 TR diameters showed a significant effect of time ($F(3,61) = 3.770$, $p = 0.015$. L4 TR diameter significantly decreased from the early to the late cohort ($p = 0.020$) among group two females (Table 21).

The Kruskal-Wallis statistic was used to detect a change through time in L2 AP and TR diameters for group two females because one of the cohort groups did not meet normality. L2 AP diameters significantly differ over time, ($H(3) = 8.237$, $p = 0.041$) (Table 13). Pairwise comparisons with adjusted p-values show that despite the overall effect of time, there was no significant change in diameters from cohort to cohort (Table 22). L2 TR diameters significantly differ over time, ($H(3) = 10.416$, $p = 0.015$) (Table 13). Pairwise comparisons with adjusted p-values show a significant decrease in L2 TR diameters from the early to late cohort ($p=0.011$) (Table 23).

There is a significant effect of time period on L1 AP diameter for group three females ($F(3, 46) = 2.931$, $p = 0.043$). However, the Games-Howell post-hoc results show no significant change in L1 AP diameters from one cohort to the next despite the significant effect of time overall (Table 24). There is no significant effect of time on AP and TR diameters among group four females. The results for females follow the trend of decreasing TR diameters with time. Late childhood/adolescent stress appears to increase with time among European American females.

Table 18. ANOVA *p*-values for L1 transverse diameters for group two females, multiple cohort comparisons. Bolded numbers indicate significant results. Letters next to numbers indicate the change from the last cohort: d = decrease in canal size; i = increase in canal size.

Cohort	Early	Middle	Intermediate
Early			
Middle	0.983		
Intermediate	0.283	0.788	
Late	**0.005d**	0.169	0.300

Table 19. ANOVA *p*-values for L3 anteroposterior diameters for group two females, multiple cohort comparisons. Bolded numbers indicate significant results. Letters next to numbers indicate the change from the last cohort: d = decrease in canal size; i = increase in canal size.

Cohort	Early	Middle	Intermediate
Early			
Middle	0.128		
Intermediate	0.810	0.060	
Late	0.487	0.678	0.224

Table 20. ANOVA *p*-values for L3 transverse diameters for group two females, multiple cohort comparisons. Bolded numbers indicate significant results. Letters next to numbers indicate the change from the last cohort: d = decrease in canal size; i = increase in canal size.

Cohort	Early	Middle	Intermediate
Early			
Middle	0.195		
Intermediate	0.963	0.367	
Late	**0.006d**	0.926	**0.031d**

Table 21. ANOVA *p*-values for L4 transverse diameters for group two females, multiple cohort comparisons. Bolded numbers indicate significant results. Letters next to numbers indicate the change from the last cohort: d = decrease in canal size; i = increase in canal size.

Cohort	Early	Middle	Intermediate
Early			
Middle	0.326		
Intermediate	0.982	0.588	
Late	**0.020d**	0.680	0.084

Table 22. Kruskal-Wallis adjusted *p*-values for L2 anteroposterior diameters for group two females, pairwise cohort comparisons. Bolded numbers indicate significant results. Letters next to numbers indicate the change from the last cohort: d = decrease in canal size; i = increase in canal size.

Cohort	Early	Middle	Intermediate
Early			
Middle	1.000		
Intermediate	0.271	0.058	
Late	1.000	1.000	0.135

Table 23. Kruskal-Wallis adjusted *p*-values for L2 transverse diameters for group two females, pairwise cohort comparisons. Bolded numbers indicate significant results. Letters next to numbers indicate the change from the last cohort: d = decrease in canal size; i = increase in canal size.

Cohort	Early	Middle	Intermediate
Early			
Middle	0.922		
Intermediate	1.000	1.000	
Late	0.011d	1.000	0.182

Table 24. ANOVA *p*-values for L1 anteroposterior diameters for group three females, multiple cohort comparisons. Bolded numbers indicate significant results. Letters next to numbers indicate the change from the last cohort: d = decrease in canal size; i = increase in canal size.

Cohort	Early	Middle	Intermediate
Early			
Middle	0.282		
Intermediate	0.107	0.860	
Late	0.086	0.890	0.999

The number, mean, and standard deviation for VNC diameters by cohort for African American females are shown in Table 8. There were no significant effects of time period on AP and TR diameters for African American females.

In summary, anteroposterior vertebral canal diameters increased through time for EA group three males and African American males. It appears that these two groups might have experienced a decrease in early childhood stress through time. The opposite trend appears for transverse vertebral canal diameters, which tend to decrease over time among EA group two males and females. These groups appear to experience an increase over time in late childhood and adolescent stress. With the exception of EA group two males' L4 TR diameters and EA group two females' L4 AP diameters from the intermediate cohort, as well as African American males' L3 AP diameters from the middle cohort, all other vertebral neural canals were within the range of normal variation seen in previous studies. These ranges suggest that only three groups definitely experienced stress in childhood or adolescence. However, as no previous study comes from regionally and temporally contemporaneous populations, it is possible that the ranges might be slightly different among these groups. For instance, if the lower range for AP diameters was set at less than 11.5mm (Eisensteins, 1983 diameter), African American males in the intermediate and late cohorts, EA group three males from the early cohort, and EA group two females from the intermediate and late cohorts would have all had ranges below the lowest range for normal AP diameters.

Comparisons of Frequencies of Linear Enamel Hypoplasia

Fisher's Exact tests were employed to determine within-cohort group differences in LEH in the maxillary central incisor and mandibular canine. The tests were also used to determine

differences in LEH for each group over time. These parallel the analytical approach used to examine VNC. The only significant and reliable Fisher's Exact results were for African American males, which showed a significant decrease in LEH frequency from the early to late cohort ($p = 0.023$) and from the middle to late cohort ($p = 0.016$) (Table 25). Childhood stress appears to have decreased among African American males through time. Results from all the other LEH Fisher's Exact tests were not significant or unreliable because most of the teeth in these groups were missing, worn, fragmented, or obscured (Tables 26-27). Unfortunately, this significantly lowered the number of teeth that could be scored, and left only subjective interpretation of changes in LEH frequencies over time.

Overall, the patterns of LEH presence and absence suggest that the incidence of LEH was highest among group three males, group two females, and African American males and females. African American males and females had the highest incidences of LEH. African American males have more or equal incidences of LEH when compared to African American females for all cohorts except the intermediate cohort when females have more than males. The occurrence of LEH among group two and four males and group three and four females is too small to detect any trend. In general, LEH occurs more frequently on mandibular canines than maxillary central incisors. This might be due to their longer maturation rate and thus longer exposure time to environmental stressors (Dean and Reid, 2001).

In summary, the results of the MANOVA analyses show that there is significant sexual dimorphism in vertebral canal size in the latter two cohorts, but in only the early cohort is there a significant difference between the racial groups. ANOVA results for the vertebral canal data show that early childhood stress was decreasing with time, while late childhood and adolescent stress was increasing with time. EA group three males and African American males showed a

Table 25. Fisher's Exact *p*-values for mandibular canine LEH frequencies compared among African American male birth cohorts. Letters next to numbers indicate the change from the last cohort: d = decrease in LEH presence; i = increase in LEH presence.

Cohort	Early	Middle	Intermediate
Early			
Middle	-		
Intermediate	-	-	
Late	**0.023d**	**0.016d**	0.107

Table 26. Distribution of LEH among males. UCI = maxillary central incisors; LC = mandibular canine.

Racial Group	Cohort	N	Present		Absent		Missing	
			UCI	LC	UCI	LC	UCI	LC
Group two males	10	29	1	0	0	0	28	29
	11	16	0	1	0	0	16	15
	12	34	2	3	0	0	32	31
	13	19	3	1	0	2	16	16
Group three males	10	48	5	6	0	0	43	42
	11	18	1	2	0	0	17	16
	12	55	2	1	0	0	53	54
	13	20	2	7	0	0	18	13
Group four males	10	5	0	1	0	0	5	4
	11	1	0	0	0	0	1	1
	12	8	0	2	0	0	8	6
	13	1	0	0	0	0	1	1
African American	10	46	9	17	0	0	37	29
males	11	39	5	19	0	0	34	20
	12	25	5	8	0	0	20	17
	13	25	3	8	0	3	22	14

Table 27. Distribution of LEH among females. UCI = maxillary central incisors; LC = mandibular canine.

Group	Cohort	N	Present		Absent		Missing	
			UCI	LC	UCI	LC	UCI	LC
Group two females	10	18	0	4	0	0	18	14
	11	13	0	2	0	0	13	11
	12	19	2	2	0	2	17	15
	13	21	1	3	0	2	20	16
Group three females	10	16	0	1	0	0	16	15
	11	12	0	0	1	0	11	12
	12	15	1	4	0	0	14	11
	13	16	0	3	0	0	16	13
Group four females	10	1	0	0	0	0	1	1
	11	4	0	1	0	0	4	3
	12	4	0	1	0	0	4	3
	13	3	1	1	0	0	2	2
African American	10	70	2	17	1	1	67	52
females	11	60	11	28	0	2	49	30
	12	13	2	4	0	0	11	9
	13	5	2	2	1	1	2	2

significant decrease in childhood stress over time, while EA group two males and females showed a significant increase in late childhood and adolescent stress over time. Only group two males and females as well as African American males had ranges that fell below of normal variation seen in previous studies, suggesting that most of the population in this study did not experience childhood or adolescent stress or that they experienced normal levels of stress. There were no overall trends in LEH except for its higher occurrence on mandibular canines among all groups.

Chapter 6

DISCUSSION AND CONCLUSIONS

This study set out to assess whether a heterogeneous group of racialized European Americans (EA) existed among a sample of EA born between 1828 and 1984. This study also set out to determine whether phenotypically distinct groups of EA experienced different levels of childhood and adolescent stress over time. The results of this study show that a heterogeneous group of EA existed in the U.S. between 1828 and 1984. These groups likely represent racialized breeding groups of EA as mating choice was and continues to be restricted by racist structures (Roseman, 2014). There are also differences in the distribution of stress indicators among racialized EA and African Americans (AA) within the early, middle, and intermediate cohorts. In this chapter, I review these results in light of racist structures as well as other components of the political, social, and economic environment in which these populations lived. First, I address the limitations of the discriminant function analysis (DFA) and finite mixture analysis (FMA) as well as possible solutions to these limitations. Second, I review the utility of vertebral neural canal diameters to reveal the occurrence of childhood and adolescent stress in light of the results presented in chapter 5. Third, I discuss how the results address the questions and expectations presented in Chapters 1 and 4. I also discuss future endeavors pertaining to this research. I conclude by summarizing this dissertation and its findings.

Limitations: Discriminant Function Analysis (DFA) and Finite Mixture Analysis (FMA)

Before considering what the analytical results indicate about the fundamental questions asked in this study, it is important to consider the limitations in the research design, especially the use

of discriminant function analysis (DFA) and finite mixture analysis (FMA) to identify phenotypically distinct groups of EA. As I explained in the previous chapters, I initiated this study using 25 of Ossenberg's (2013) nonmetric cranial traits. Ossenberg's (2013) methods of trait analysis were employed because this study sought to determine whether a discriminant function based on Ossenberg's (2013) Western, Southern, and Eastern European sample (Table 2, see Chapter 4) would potentially assign individuals within my sample of EA to a Western, Eastern, or Southern European affinity group. My intentions were to associate individuals from the EA study sample, using discriminant scores, with the Western group into the "white western" racial category, individuals who most resembled Southern Europeans into the "nonwhite southern" racial category, and individuals who most resembled Eastern Europeans into the "nonwhite eastern" racial category. These assignments were intended to separate groups into the white or nonwhite status they *might* have been assigned prior to the Second World War; these assignments do not reflect how individuals self-identified.

As explained in Chapter 4, DFA develops functions, based on a predefined sample, that predict group membership. Yet DFA is subject to poor performance when the individuals being assigned to a group membership do not originate from populations in the predefined sample used to develop the discriminant functions (Konigsberg et al., 2009; Roseman, 2014). The DFA created using Ossenberg's (2013) nonmetric traits did not discriminate well among the three European regions (Table 5, see Chapter 5). The poor discriminatory power of the DFA suggests that there would be a high probability of error in assignments given to individuals in the European American sample (Table 5, see Chapter 5), and so for this reason the original method for discriminating among European American groups in this study's sample was revised to employ finite mixture analysis (FMA).

Kramer and Konigsberg's (1999) FMA was used to identify EA groups, if any. FMA does not make assumptions about the number of groups present in the data. Instead, FMA uses the apportionment of variance in the sample to determine the most likely number of groups present. Also, the fact that this method is atheoretical and model-free makes it extremely useful in exploratory endeavors like this dissertation. FMA also does not assume, as the DFA does, that EA individuals must belong to some pure European counterpart. Instead, FMA discriminates EA individuals into groups without prior assumptions about the unique evolutionary experiences in the U.S.

FMA, however, also has limitations. First, FMA cannot be performed when data are missing from the sample. Imputation, marginalization, and algorithm methods are used to address missing data in many clustering analyses like FMA (Wagstaff, 2004; Wilson, 2015). However, imputation and algorithm methods are never better than using observed data, so I chose marginalization or to ignore missing data in this study. Missing data were prevalent in the EA sample, and so I removed traits with large numbers of missing cases. I also removed traits likely to be scored erroneously (see Chapter 4 for more on trait removal and selection). This removal and selection process resulted in a smaller sample of EA (n=395) and 13 morphological traits being used to determine whether heterogeneity existed among EA. The smaller sample sizes among group four males and females in this study were likely the result of a smaller overall EA sample after the removal and selection process. The smaller number of morphological traits also might have increased the probability of highly heritable traits being removed from the study. The removal of highly heritable traits would limit discriminatory power if the assumption holds that the phenotypic traits used in this study can indeed distinguish groups in the EA sample.

Despite the shortcomings, the FMA still yielded three distinguishable groups as determined from the data. Whether these three groups correspond with distinctions in European regional descent, or with traits that are reflecting differences in factors related to racialization in the United States, remains indeterminate without knowing how individuals in the sample self-identified. Nevertheless, I proceed here to address each question and expectation in light of the results, with reference to the historical context and racial environment in each birth cohort.

Vertebral neural canal diameters (VNC) as measures of childhood and adolescent stress

Before proceeding to contextualize the results, it is worth briefly considering the justification for using vertebral neural canal diameters (VNC) as measures of childhood and adolescent stress. Small VNC diameters have been shown to correlate with other measures of childhood stress and adolescent stress. Porter and Pavitt (1987) measured the transverse (TR) and anteroposterior (AP) diameters of Anglo-Saxon and Romano-British archaeological populations from Great Britain. They found that small TR diameters significantly correlated with the presence of linear enamel hypoplasias (LEH). They also found that small AP diameters were significantly correlated with Harris lines. Harris lines result from infection and malnutrition during the maturation of long bones (Huges et al., 1996; Geber, 2014). Jeffrey and colleagues (2003) took MRI scans of the VNC of 161 U.S. children born between 1988 and 1989. Low birth weight is associated with small TR and AP diameters. Low birth weight is inversely associated with neonatal and childhood stress (Martorell and González-Cossío, 1987; Cameron and Demerath, 2002). These two studies show that small VNC diameters significantly correlate with various indicators of early/late childhood and adolescent stress.

A second reason VNC diameters are good measures of stress centers around their relationship to longevity. Clark and colleagues (1986) found that small TR diameters were associated with increased risk of adult mortality in the 15-25 year old group among prehistoric (950 A.D.-1300 A.D.) individuals from Dickson Mounds (n=2,060). Watts (2015) also found that small TR diameters were associated with increased adult mortality among a London sample (n=941) that dated from 1117 A.D. to 1853 A.D. These two studies suggest that small TR diameters might predict late adolescent and adult health and that this prediction is not restrained by time. These findings are important because they suggest that highly stressful events occurred during late childhood and adolescence. These findings also suggest that this stress has long-term and negative effects on adolescent and adult health. The above studies, taken together, show that small VNC correlate with increased morbidity and mortality in childhood, adolescence, and adulthood.

The question remains: What measures represent pathologically small TR or AP diameters (i.e., diameters that signify unusually high levels of stress)? Some researchers suggest a VNC below the normal range of variation, that is to say a VNC AP diameter of roughly less than 10mm and a VNC TR diameter of roughly less than 16mm (Verbiest, 1977; Ulrich et al., 1980; Eisenstein, 1983). These extremely small diameters undoubtedly represent childhood and adolescent stress, but are not the sole numeric measures capable of representing stress. Porter and Pavitt (1987) had small mean TR diameters between 21.28-25.66mm and AP diameters between 13.53-15.85mm. Jeffrey and colleagues (2003) had small mean TR diameters between 22.6-26.0mm and AP diameters between 18.8-20.8mm. Watts (2015) had small mean TR diameters for males between 22.33-24.91mm and for females between 21.49-24.47mm. Clark and colleagues (1986) had a small mean TR diameter of 21.85mm. Not one of the means in the above

studies fall below 10mm for AP diameters and 16mm for TR diameters. These studies did not provide ranges, but do suggest that the average mean diameters associated with poor childhood, adolescent, and even adult heath are well within what is considered the "normal" range of variation for VNC diameters. In essence, small diameters associated with higher levels of stress are not outside the "normal" range of variation. Perhaps the best way to assess what represents pathologically small VNC in any population, however, would be to compare it to another form of childhood or adolescent stress or even mortality data. This study attempted to compare VNC findings with LEH, but the LEH data were largely missing (Tables 26 and 27, see Chapter 5). Nonetheless, it can be argued that significant changes in diameters, whether they are within the reported "normal" range of variation or not, likely point to significant changes in stress. This study assumes the latter.

Question 1 Discussion: Are there distinguishable groups based on cranial trait variation within the sample of European Americans? What role might racialization play in shaping these distinctions?

Yes, there are distinguishable groups based on cranial trait variation within the sample of EA. The FMA identified three distinct groups of EA. These results suggest that a more heterogeneous group of EA existed in the past and continues to exist today. It is argued in this study that the process of racialization likely influenced the formation of these groups as racist structures would have restricted mating choice along perceived racial lines (Roseman, 2014). It is not known which groups would have been perceived as white, but the distinction among groups warrants exploration into group experiences. Such experiences might at least point to the group that encountered higher levels of stress and thus was perhaps most marginalized. It is possible,

however, that these groups represent three marginalized EA populations who would have all been considered nonwhite. Other identities would then explain the divisions among these groups. These identities might have centered on class, language, religion, nationality, and/or cultural practices/norms and could have further restricted mating choice in an already racially constrained group.

Question 2 Discussion: Do these groups remain constant in size and proportion over time? If not, how do they change? What might explain these changes?

I asked this question in order to investigate whether different levels of racial tension affected EA population heterogeneity during different cohorts. Moreover, knowing how groups discriminated over time might affect disparities in stress as each cohort might have a different number of EA groups. The number and size (which determines statistical power) of groups could determine whether heterogeneity in phenotype or stress exists. Despite the uncertainties associated with discriminating among groups, results nevertheless suggest changes in the group sizes as distinguished phenotypically in the EA sample between the early nineteenth and the late twentieth centuries. As a caveat, it is worth pointing out that these shifts may have to do with sampling biases or regional effects that cannot be taken into account with the available information about each skeletal collection. Table 6 in Chapter 5 shows that there were more group three than group two males among the EA racial groups in the Early and Intermediate birth cohorts. Yet, in the Middle and Late cohorts these groups are nearly equally distributed among males. Overall, there are more group three males in all cohorts. Group four males have an extremely small sample ($n \leq 8$) for all cohorts.

The opposite is true of females, there are consistently fewer group three than group two females from the Early to the Late cohort. These groups are nearly equally distributed in the Early and Middle cohorts. The contribution of group four females to the EA sample remains too small ($n \leq 4$) to speculate for all cohorts.

It appears that the change in phenotypic composition is primarily due to more equal contributions of group two and three males in the Middle and Late cohorts and less equal contributions of group two and three females in the Intermediate and Late cohorts. Group four males and females appear to have no impact on phenotypic composition. Yet, the disproportionately fewer assignments to the group four sample is likely an artifact that resulted from decreased sample sizes in the elimination of large amounts of missing data from the FMA. Thus, these small samples might not reflect the lack of group four EA in the broader population. With these caveats in mind, I address the four expectations made in association with this second question below.

Expectation I

I expect EA in the Early Period (1828-1881) to be more homogenous as the great migration of Eastern and Southern Europeans did not begin until 1882 (Dillingham et al., 1911).

This expectation is confirmed in the male sample as group three males dominate the sample in the Early cohort. This finding also might reflect differences in migration patterns among group two and three males. Perhaps the ancestors of group three males were more likely to migrate before females to find employment and lodging before the rest of their family arrived (Comité, 1986; Green, 2012) than males from group two and four. Group three males might also represent

a group of males who experienced more marginalization than group two or four males and thus were more likely to experience financial hardship, leaving them unable to afford proper burial. Thus, this group could represent a nonwhite group of EA.

It is also possible that group three represents individuals that would have been considered white (i.e., those of Western or Northern European descent). Most of the European Americans in the Early period were of Western European descent (German, English, Scottish, Scots-Irish) (Dillingham et al., 1911; Roediger, 2005; Painter, 2010; Pinder, 2012). The small number of Southern and Eastern immigrants resulted in a minimal amount of racial tension. Moreover, Early period populations, although racialized, were largely lower class (Agbe-Davies, 2015). There were few middle and upper class individuals in the sample regardless of racial status. Thus, group three males might represent lower class whites, especially given the demographics of body acquisition in the Hamann-Todd collection and Terry collection. The intersection of race and class cannot be ignored as class might have had a greater impact on status and mating restrictions than race during the Early period. Conversely, these data could accurately depict population demographics during the Early period.

Although there are more EA group two females overall in the early cohort, group three females make a comparable contribution. Accordingly, there is no homogenous sample of EA females in the Early cohort. This is not in line with what I expected. The lack of homogeneity points to three possibilities: 1) females had different migration patterns than males, leading to equal contributions of females from at least two populations during the Early period; or 2) females were more likely, no matter what group they came from, to experience harsh sex discrimination (Deitch, 1993), making them more likely to endure hardship and enter the skeletal

collection; 3) females represent increasing gene flow between groups as they are just as likely to be assigned to group two or three.

Expectation II

I expect to see three distinct European American groups during the Middle Period (1882-1913) because of the migration of millions of Southern and Eastern Europeans during this period (Dillingham et al., 1911; Parrillo, 2000; Roediger, 2005).

There are still three distinct groups in the Middle period, yet much changed in the sample composition from the Early to the Middle period. There are more equally distributed males and females, which provide evidence for the fact that migration from 1882 to 1902 decreased from Western Europe by 75% and increased by 475% from Southern and Eastern Europe (Dillingham et al., 1911). The increase in Southern and Eastern Europeans possibly caught up with the proportion of descendants from Western Europe, hence a more equal distribution among sexes from group two and three is reflected in the sample composition of the Middle cohort. The comparable equal distribution of males and females could also represent increased gene flow between groups as they are just as likely to be assigned to group two or three.

One might also expect to see a disproportionate increase in one, two, or all the EA groups within the sample during the Middle period because the influx of immigrants led to increased anxiety over resources among white European Americans (Jones and Carter, 1996; Parillo, 2000; Dinnerstien et al., 2003; Painter, 2010). There is an increase in the proportion of group two males to the sample as they comprised 35% of individuals in the Early cohort and 46% in the Middle cohort. Group three males decreased in proportion from 59% to 51% from the Early to the Middle period. Given the historical data, and assuming the FMA grouping represent real

geographic distinctions among Europeans, it is likely that group two males represent the influx of either Southern or Eastern Europeans. The anxiety this influx produced in whites would have led to increased racial tensions and marginalization of Southern and Eastern European immigrants and Americans (Horne, 1996; Dinnerstien et al., 2003; Roediger, 2005; Painter, 2010). Marginalization would have subjected these groups more frequently to a lower SES and greater likelihood of not being able to afford proper burial.

Expectation III

I expect to see a drastic decrease in some groups and in increase in others during the Intermediate Period (1914-1945) because this was a time when acceptance of Southern and Eastern European migrants was burgeoning (Roediger, 2005).

This expectation is met in the Intermediate period. There is an increase in group three and a decrease in group two proportions for males. There is also an increase in the proportion of group four males. The opposite is observed for females. There is an increase in the proportion of group two females and a decrease in group three females' proportions. There is also a decrease in the proportion of group four females. These findings suggest increased variation, which point to a time of increased acceptance of all EA.

Expectation IV

European Americans will demonstrate an amalgamation of the Western, Southern, and Eastern European phenotype in the Late Period (1946-1984) due to white becoming an umbrella term for all individuals of European descent (Knowles and Prewitt 1969; Clark and O'Donnell 1999; Roediger, 2005).

The Late period might actually reflect increased gene flow as males are almost just as likely to be assigned to group two as they are to group three. This would be in line with decreased racial tensions and increased acceptance in the years after the First and Second World Wars (Weinberg and Crosswell, 1967; Clark and O'Donnell, 1999; Roediger, 2005). During the Late period, there is a slight increase in the likelihood of females being assigned to group two and three. Moreover, groups two and three are not comparable. This might reflect a lag in increased gene flow and acceptance from the previous cohort. Thus, these data might represent females with distinct European ancestry.

In general, males in the Early and Intermediate cohorts are more likely to be assigned to group three. In the Middle and Late cohort they are almost equally likely to be assigned to groups two and three. Females are more likely to be assigned to group two in the Early, Intermediate, and Late cohort, while in the Middle cohort they are equally likely to be assigned to either group two or three.

Questions 3 and 4 Discussion: Do phenotypically distinct groups of European Americans have similar distributions of childhood and adolescent stress (LEH and VNC size) temporally? How do trends in stress among racialized European American groups relate to trends in stress among African Americans? What might explain these relationships?

Expectation V

I expect to see significant differences in the temporal distribution of indicators of childhood and adolescent stress among racialized European American and African American groups as racial classification mediates access to resources that affect health.

The only significant race effect detected by the MANOVA occurred during the Early period (1828-1882). Racial groups were significantly different in the transverse diameters of L1 and L2 in the early cohort. Boxplots of these diameters (Figures 5 and 6 in Chapter 5) demonstrate that the transverse diameters among EA groups two and three are similar for L1 and L2, but set apart from African American males and females. This suggests that late childhood and adolescent stress amongst groups two and three was similar during the Early (1828-1882) period. No conclusions can be made concerning group four males and females because of their extremely small sample size ($n \leq 5$).

The Early period represents a time of minimal racial tension toward all nonwhite EA groups because of the small number of Europeans present in the United States that would have been considered nonwhite (i.e., Southern and Eastern Europeans and Americans) (Dillingham et al., 1911; Painter, 2010). It is expected, because of decreased racial tensions, that there would be little differences in stress among EA. The lack of significant differences in VNC diameters among EA group two and three suggest they experienced similar levels of stress. The race effect is apparent in the distinction between the EA racial groups as a whole, and African American males and females. The TR diameters of African American males and females during the Early period are significantly smaller than those of the EA racial groups suggesting they experienced greater late childhood and adolescent stress. The establishment of Jim Crow laws immediately after Reconstruction decreased the availability of resources among AA. They would have experienced more stress and significantly smaller VNC diameters, as is evident among L1-L2 TR diameters in the Early cohort.

The only other incidences of possible racial differences are seen in the Middle and Intermediate cohorts. The lowest numeric range for L3 AP diameters (9.76mm) among AA

males in the Middle cohort falls below 10mm (i.e., the lowest normal range of variation seen in other studies). This extremely low range suggests that African American males indeed experienced stress during the Middle cohort. This stress was not detected by the MANOVA because the VNC diameters during this period were not significantly different among any of the populations. Perhaps the perception of nonwhite EA as higher status than African Americans on the racial inferiority ladder by the dominant white hegemony during the Early and Middle period (Dillingham et al., 1911; Fredrickson 1971; Ely and Bodenhamer 1984; McDermott and Samson, 2005; Painter, 2010) ensured that African Americans would encounter more racist structures than nonwhite EA during the Middle period. However, differences between African American males and females and EA racial groups is not a trend and is only seen during the Early and Middle period, after which there is a decrease in the stress disparity gap that is maintained in the latter two cohorts. This decrease is expected over time as it is a common occurrence in literature pertaining to disparities in stress between European and African Americans (Dressler et al., 2005; Williams et al., 2010; Asada et al., 2013).

It is likely that these results reflect a period of hyper-racism in the wake of the rights afforded African Americans during Reconstruction (~1863-1877), including the right to vote, in the Earlier period (Crouch, 1992; Franklin and Moss, 1994; Sitton and Conrad, 2005). Jim Crow intensified during this era and was only further ignited by the Plessy vs. Ferguson 1896 verdict that made legal separate-but-equal accommodations for European and African Americans in the Middle period (Rable, 1984; Bergeron et al., 1999; Parillo, 2000; Davidson, 2004). This hyper-racism appears to be reflected in the decreased L1-L2 TR diameters during the Early cohort among African American males and females and the below normal range of L3 AP diameters among African American males in the Middle cohort. Decreased TR diameters reflect

significantly more stress during late childhood and early adolescence in the Early period. It was during late childhood and early adolescence, ages 11-17, that children began working to contribute to their and their families' livelihood (Hindman, 2002; Rosenberg, 2013); they were thus less buffered from the detrimental effects of racist structures. The lower than normal range in L3 AP diameters in the Middle cohort suggest higher levels of early childhood stress in this group and the inability of African Americans to buffer male children between the ages of three and five from environmental stressors.

As with the Middle cohort, there is no significant race effect evidenced by the MANOVA in the Intermediate cohort. Yet, during the Intermediate cohort, the L4 TR diameter among group two males has a low range (15.50mm) that is below the normal range of variation for TR diameters. These findings suggest that EA group two males experienced late childhood and/or early adolescent stress levels higher than all the other populations in the Intermediate cohort. Also, during the Intermediate period, EA group two females have L4 AP diameters with a low range (9.27mm) that falls below the normal range of variation for AP diameters. Group two females experienced higher levels of early childhood stress during the Intermediate period. It appears that group two males and females experienced higher levels of stress than all other groups during the Intermediate cohort. The immigration laws passed in 1921, 1924, and 1929 as well as the Nationality Act of 1940 designate this period as a time of gradual acceptance of nonwhite EA as white (Clark and O'Donnell, 1999; Weinberg and Crosswell,1967). This is the only time period when EA groups two and three do not experience similar levels of stress. In fact, EA group two individuals experienced higher levels of stress than all other groups including African American males and females. This finding suggests that distinct EA groups experienced different distributions of childhood and adolescent stress through time. Perhaps group two

individuals experienced higher levels of resource marginalization during the depression in the Intermediate period. As the Intermediate period is also meant to represent a 30-year lag in acceptance of all EA as white, the experiences of group two males and females might represent white EA resistance to this group during this transition. Yet, the fact that males and females experienced different types of stress suggest sex differences that would have led to different levels of exposure to stressors during early childhood, late childhood, and adolescence. Both race and sex appear to influence exposure to stress.

The lack of significant race effects in the MANOVA for the Middle, Intermediate, and Late cohorts does not mean that differences in stress did not occur as Middle cohort African American males and Intermediate cohort EA group two males and females had VNC diameters below the normal range of variation. Their lower than normal ranges indicate higher levels of stress among these groups in relation to other groups. Thus, the only cohort that did not experience differences in stress among racial groups is the Late cohort. It is expected that during the Late period racial tensions should be minimal for all EA groups because white is an umbrella term for all individuals of EA ancestry. The lack of significant differences in stress between all racial groups suggests that resource marginalization along racial lines declined to a point where EA and AA groups experienced similar levels of stress. It is also possible that nonwhite EA racial groups and African Americans employed some sort of cultural buffering that ultimately exposed them to levels of stress comparable to white EA. In essence, similar levels of stress might not represent acceptance by the white hegemony. It could also be that FMA created EA racial groups when there were none, and so the EA sample might represent one EA group of nonwhite or white EA. All racial groups in the Late cohort might have simply been equally stressed. One must also

consider the possibility that VNC diameters might not be the best indicators of childhood and adolescent stress in the Late cohort.

The ANOVA results demonstrate significant decreases in early childhood stress (as indicated by AP diameters) for group three males and African American males. That only males experienced decreases in early childhood stress suggest positive changes in diet, including improvements in weaning practices. Reliable conclusions from the results of group four males and females cannot be drawn given their extremely small sample sizes.

There are no significant changes in childhood and adolescent stress for group three females. This finding suggests that stress among group three females remained constant over time or cultural buffering aided in maintaining constant levels of stress experiences over time. Moreover, African American females do not demonstrate significant changes in childhood and adolescent stress over time. This might reflect the better treatment of African American boys than girls as African American males not only showed a significant decrease in early childhood stress over time in AP diameters, but they are also the only group to demonstrate a significant decrease in LEH experienced in early childhood. Belachew and colleagues (2011) reported that during times of food insecurity or shortage in southwest Ethiopia adolescent girls experienced higher rates of food insecurity and these same girls were more like to report health problems than boys. They suggest that this is because girls are seen as subordinate and an economic liability, whereas boys are seen as providers and defenders of the family. The preferential treatment of boys in southwest Ethiopia due to perceived gender roles might have occurred among African American Americans historically as well and, if so, would explain the decrease in early childhood stress among males and not females over time. Casey and colleagues (2005), however, found that African American male children in food insecure households in the U.S. had significantly poorer

health than African American females. This study included data taken during the year 2000 from 399 children born in the late twentieth century. This study did not consider changes in health over time. Taken together, Belachew and colleagues (2011) and Casey and colleagues (2005) might simply point to the possibility that females either experienced less stress overall or were better able to buffer stress over time than males. And, perhaps the decrease in early childhood stress among African American reflects males being less exposed to stress or better able to buffer, though not to the point of achieving stress levels comparable to the lower levels seen in African American females. After all, the lowest range for L3 AP diameter is 9.76mm for African American males in the Middle period. Such low range is not seen among AA females and is below the normal range of variation for AP diameters (Verbiest, 1977; Ulrich et al., 1980; Eisenstien, 1983). Moreover, African American female AP mean diameters are higher or equal to that of males in all cohorts with few exceptions.

The lack of any significant change in LEH over time in all the other race/sex groups is more an artifact of the small dental sample (Table 26, 27) than a reflection of a lack of change in the occurrence of stressful episodes during childhood. The higher occurrence of LEH on mandibular canines is likely due to their longer maturation rate and thus longer exposure time to environmental stressors (Dean and Reid, 2001). The lack of significant changes in childhood and adolescent stress among African American females must be tempered considering the small sample sizes in the Intermediate and Late birth cohorts.

Regarding late childhood and early adolescent stress, as indicated by TR diameters, there are increases in stress over time for EA group two males and females. These data show a significant increase in stress from the Early to the Late period. These data reveal a general trend of increasing stress among EA in group two. The comparable trend between males and females

suggest a lack of sex bias. However, I am not certain what could have caused this trend over time, other than a lack of cultural buffering during late childhood and early adolescence over time amongst group two EA. It might also be that group two EA were more impacted by racist structures during late childhood and adolescence.

There are also significant sex effects in the Intermediate and Late cohorts. Though sex effects are expected, sex was not a factor in the Early and Middle cohorts which suggest more severe sex discrimination in the latter two cohorts, discrimination capable of affecting late childhood and adolescent stress. The treatment of males and females must have changed during the latter two periods when they reached late childhood and adolescence. Females experienced decreased transverse diameters suggesting increased stress during the Intermediate and Late cohorts. Males might have also received more and better food because they were considered future leaders, defenders, and providers.

A Summary of Findings and Future Considerations

The FMA in this study is not meant to reify race. Groups are identified using variation in morphological traits. It is unknown how these individuals self-identified. This research can only speculate on whether identified groups represent different racial groups, one racial group, or groups clustering along class, language, religion, or nationality (all of which restrict mating choice). It is also possible that the nonmetric cranial traits in this study allow FMA to identify groups that might not have been identified using a different set of traits. More EA group three males in the Early and Intermediate cohorts, followed by comparable representation of group two and three males in the Middle and Late cohorts could be a result of sampling biases, though there is no documentation to assume this would be the case. The change in group two and three males'

representation over time might signify increased gene flow between all Europeans and EA in the Middle period and increased gene flow in the Late period as acceptance into one white race increased for all European descent groups. This trend is also present during the Early and Middle cohorts for females as morphologically they are just as likely to identify with EA group two as they are with group three. This trend is not present among females in the Intermediate and Late cohorts.

The results of the MANOVAs show that there is a significant race effect demonstrated by increased late childhood and adolescent stress amongst African American males and females in the Early cohort that may be related to their limited resources during slavery or increased racial tension against African Americans after the benefits allotted them during the Reconstruction Era. African American males experience higher levels of stress in early childhood in the Middle cohort as evidenced by smaller than normal AP diameters. Smaller than normal TR diameters suggest that EA group two males experienced higher levels of late childhood and adolescent stress in the Intermediate cohort. The smaller than normal AP diameters in EA group two females from the Intermediate cohort suggest they experienced higher levels of early childhood stress.

ANOVA results for the VNC data show that early childhood stress was decreasing with time for group three males and African American males. These two groups appear to be equally buffered against early childhood stress through time. They might have also had similar weaning patterns that assisted in this cultural buffering. There was no decrease in early childhood stress for any other racial group or for the females. Race and sex appear to influence the distribution of early childhood stress. Perhaps with the inclusion of more group four males and females and African American females to the sample, this trend might extend to other racial groups and

across sexes. Late childhood and adolescent stress increased with time for group two males and females. Group three males and females as well as African American males did not display any significant increase in adolescent stress over time. Group four males and females did not display any trend, but sample size was small for all cohorts. African American females did not display any significant trend, but sample size was small for the last two cohorts. There appears to be a difference between group two males and females and all other groups regarding adolescent stress over time. These other groups appear to be buffered against adolescent stress or have greater resilience against adolescent stress over time. This finding was not expected and is worth exploring deeper in the future.

Unfortunately, this study is not able to identify specific environmental stressors related to VNC diameters and LEH, as both are due to non-specific stress. However, I have applied the biocultural model (Chapter 3) to situate patterns of stress in their social, political, and cultural context with emphasis on racial tensions during each period. This model assumes that the cultural environment is the source of stress and of the resources that avert stress (Goodman et al., 1988; Goodman and Leatherman, 1998). By incorporating facets of critical archaeology theory, I am better able to problematize whiteness and expose possible racial heterogeneity and stress heterogeneity among European Americans from 1828 to 1984. Moreover, by thinking critically about the impact of racialization and racist structures on mating choice and access to resources that influence stress, I have contextualized two indicators of biological stress within the concept of racialization. This brings biological consequences to the forefront in questions concerning the effects of race on mating and stress in racialized and minimally acknowledged communities. My hope is that this study will encourage others to illuminate distinct groups within EA populations

and to examine their experiences separately in light of historical and contemporary contexts as well as similar experiences.

This study undoubtedly could be improved with more data. By adding to the EA sample and diversifying the traits used in future analyses, I might obtain more statistical and discriminatory power. In future analyses of EA heterogeneity I might also consider the fact that fair skinned individuals of African descent often passed for white by choosing to live in communities where their African ancestry was unknown (Nix and Qian, 2015). The act of passing implies that white status could be taken by force and that it was a choice for some people. I could also problematize African American by searching for phenotypically distinct subgroups among African Americans through time. The varied racial terms used to describe people of African (Stephens, 1999; King and Chaney, 2010; Agbe-Davies, 2015)descent might have affected mating choices and access to resources, which in turn might paint a varied picture of stress among AA subgroups. On the other hand, I might also consider comparing LEH data from a more complete sample to VNC data to determine if any relationship exists between these two measures of stress, and between both with age and mortality because they have been shown to vary in association with childhood and adolescent stress (Clark et al., 1986; Watts, 2015; Newman and Gowland, 2015). This would allow for a more nuanced interpretation of the analysis of stress indicators than was afforded in this study. In a future study, I might also assess the possibility that there is a relationship between nonmetric cranial traits and LEH or VNC measures; in this study I assume there is no relationship. Future considerations regarding how the intersection of race and sex affect stress in also warranted as both appear to impact stress differently over time in this study.

Conclusions

Racialization cannot be ignored, not only because of its longevity in the American experience and because it produces social inequality, but also because it had biological consequences (Williams and Rucker, 2000; Gravlee, 2009; Williams et al, 2010; Roseman, 2014). It affected gene flow and mate choice, and reified group distinctions that became inherited biological traits. This effect was not experienced equally between the sexes, as hinted at by my results; the experience of racialization was not the same between women and men. The restrictions placed on racialized communities may not always be visible in recorded history, but their effects shaped human variation and disparities in stress nonetheless. To this end, this study explores whether racialization produced or maintained heterogeneity in EA and impacted stress distributions in racialized communities using biocultural theory and facets of critical archaeology theory.

The phenotypic composition of what was considered white from the early nineteenth to the twentieth centuries has changed through time but has remained heterogeneous even as "white" became an umbrella term for all individuals of European descent. It is not known how European descent groups contributed phenotypically to the sample used in this study. DNA analyses would provide more insight into ancestry, which would then allow better speculations regarding racial status over time.

The results show that in the Early cohort there is a significant difference between the racial groups, with African American males and females having significantly smaller transverse diameters. This finding suggests that African Americans experienced significant amounts of late childhood and adolescent stress during this period. There is also evidence of higher levels of stress in the Middle and Intermediate cohorts among African American males and group two EA, respectively. These findings suggest that there is a racial disparity in childhood and adolescent

stress through time. Two EA groups—group two males and group three females—experienced more late childhood and adolescent stress over time than other EA racial groups and African American males and females. Group three males and African American males showed a decrease in early childhood stress over time. There is also significant sexual dimorphism in VNC size in the Intermediate and Late cohorts. The findings in this study suggest that race alone did not influence stress, but sex also influenced stress, reflecting sex discrimination across time. Weaning practices and other cultural practices likely influenced stress over time as well.

The application of the biocultural model and facets of critical archaeology theory to situate phenotypic heterogeneity and patterns of stress in their social, political, and economic context among European Americans between 1828 and 1984 has exposed nuances in the relationship between racialization and biology. Even though sample limitations do not allow for as detailed an analysis of the link between racial identity and biology as could be hoped, this study foregrounds research seeking to understand the effects of racialization on human variation and biological stress among distinct European American groups in racialized communities. My hope is that this study will encourage others to examine the experiences of previously ignored European Americans as they relate to others in mainstream America.

LIST OF REFERENCES

Abrahams HJ. 1966. Extinct medical schools of nineteenth-century Philadelphia. Philadelphia: University of Pennsylvania Press.

Adams WY. 1988. Archaeological classification: Theory versus practice. Antiquity 61:40-56.

Agbe-Davies A. 2007. Practicing African American archaeology in the Atlantic World. In: Ogundiran A, Falola T, editors. Archaeology of Atlantic Africa and the African diaspora. Indiana University Press, Bloomington. p 413-425.

Agbe-Davies A. 2015. Tobacco, pipes, and race in colonial Virginia: Little tubes of mighty power. New York: Routledge.

Allen JS. 1989. Franz Boas's physical anthropology: The critique of racial formalism revisited. Current Anthropol, 30(1):79-84.

Asada Y, Yoshida Y, Whipp AM. 2013. Summarizing social disparities in health. The Milbank Quarterly

Aufderheide AC, Angel L, Kelley J, Outlaw AC, Outlaw MA, Rapp G, Jr., Wiwers LE, Jr. 1985. Lead in bone III: prediction of social correlates from skeletal lead content in four colonial American populations (Catoctin Furnace, College Landing, Governor's Land, and Irene Mound). Am J of Phys Anthropol 66:353-361.

Babson DW. 1990. The archaeology of racism and ethnicity on southern plantations. Hist Archaeol 24(4):20-28.

Baron HM. 1983. The demand for black labor: Historical notes on the political economy of racism. In: Green J, editors. Workers' struggles past and present. Philadelphia, Pa: Temple University Press. p 25-61.

Belachew T1, Hadley C, Lindstrom D, Gebremariam A, Lachat C, Kolsteren P. 2011 Food insecurity, school absenteeism and educational attainment of adolescents in Jimma Zone Southwest Ethiopia: a longitudinal study. Nutr J 10:29.

Bendall KE, Macaulay VA, Baker JR, Sykes BC. 1996. Heteroplasmic point mutations in the human mtDNA control region. Am J Hum Genet 59:1276–1287.

Bergeron PH, Ash SV, Keith J. 1999. Tennesseans and their history. Knoxville, TN: The University of Tennessee Press

Bermudez de Castro JM. 1989. The Carabelli trait in human prehistoric populations of the Canary Istlands. Hum Biol 61:117-131.

Berry AC, Berry RJ. 1967. Epigenetic variation in the human cranium. J Anatomy 101(2):361-379.

Black E. 2003. War against the weak: Eugenics and America's campaign to create a master race. New York: Four Walls Eight Wonders.

Blakey ML, Armelagos GJ. 1985. Deciduous enamel defects in prehistoric Americans from Dickson Mounds: Prenatal and postnatal stress. Am J Phys Anthropol 66:371-380.

Blakey ML 1988. Racism through the looking glass: An Afro-American perspective. World Archaeol Bulletin 2:46-56.

Blakey ML 2001. Bioarchaeology of the African diaspora in the Americas: Its origins and scope. Ann Rev Anthropol 30: 387-422.

Boardman JD, Field SH. 2002. Spatial mismatch and race differential in male joblessness: Cleveland and Milwaukee, 1990. The Sociol Quart 43(2):237-255.

Boas F 1912. Changes in the Bodily Form of Descendents of Immigrants. Am Anthropol 14:530-562.

Brace CL. 1995. Region does not mean "race" –reality versus convention in forensic anthropology. J Forensic Sci 40(2):171-175.

Brace CL. 2005. "Race" is a four-letter word: the genesis of the concept. Oxford: Oxford University Press, Inc.

Brace CL. 2010. Physical anthropology at the turn of the last century. In: Little MA, Kennedy KAR, editors. Histories of American physical anthropology in the twentieth century. Lanham, MD: Lexington Books. p 25-53.

Bridges PS. 1992. Prehistoric arthritis in the Americas. Ann. Rev. Anthropol. 21: 67–91.

Bridges PS. 1993. The effect of variation in methodology on the outcome of osteoarthritic studies. Inter J Osteoarchaeol 3:289-295.

Brighton S. 2008. Degrees of alienation: The material evidence of the Irish and Irish American experience,1850-1910. Hist Archaeol 42(4):132-153.

Brighton S. 2009. Historical archaeology of the Irish diaspora: a transnational approach. Knoxville, TN: The University of Tennessee Press.

Brodkin K. 1998. How Jews became white folks and what that says about race in America. New Brunswick, NJ: Rutgers University Press.

Brown RA, Armelagos GJ. 2001. Apportionment of racial diversity: A review. Evol Anthropol 10:34-40.

Bruce DD, Jr. 1979. Violence and culture in the Antebellum South. Austin, TX: University of Texas Press.

Brues AM. 1990. The once and future diagnosis of race. In: Gill GW, Rhine S, editors. Skeletal attribution of race. Albuquerque: Maxwell Museum of Anthropology. p 1-7.

Brundage WF. 1993. Lynching in the New South: Georgia and Virginia, 1880-1930. Chicago: University of Illinois Press.

Bryant JM 1994. "We have no chance of justice before the courts": The freedman's struggle for power in Greene County, Georgia, 1865-1874. In: Inscoe JC, editor. Georgia in black and white: explorations in the race relations of a Southern State, 1865-1950, Athens, Georgia: The University of Georgia Press. p 13-37.

Buikstra JE, Frankenberg SR, Konigsberg LW. 1990. Skeletal biological distance studies in American physical anthropology: Recent trends. Am J Phys Anthropol 82:1-7.

Byrd WM, Clayton LA. 2001. Race, medicine, and health care in the United States: A historical Survey. J Natl Med Assoc 93(3) suppl:11S-34S.

Byrd RM. 2014. Phenotypic variation of transitional forager-farmers in the Sonoran Desert. Am J Phys Anthropol 155:579-590.

Cameron N, Demerath EW. 2002. Critical periods in human growth and their relationship to diseases of aging. Am J Phys Anthropol 119(S35):159–184.

Carson EA. 2006a. Maximum likelihood estimation of human craniometrics heritabilities. Am J Phys Anthropol 131:169-180.

Carson EA. 2006b. Maximum likelihood variance components analysis of heritabilities of cranial nonmetric traits. Hum Biol 78:383-402.

Casey PH, Szeto KL, Robbins JM, Stuff JE, Connell C, Gossett JM, Simpson PM. 2005. Child health-related quality of life and household food security. Arch Pediatr Adolesc Med 159(1):51-56.

Caspari R. 2009. 1918: Three perspectives on race and human variation. Am J Phys Anthropol 139:5-15.

Catvo JM, Lee MJ, Roberts JM, Xu J, Simhan HN. 2016. Race disparities and decreasing birth weight: Are all babies getting smaller? Am J Epidemiol 183 (1):15-23.

Charatz-Litt C. 1992. A chronicle of racism: The effects of the white medical community on black health. J Natl Med Assoc 84:717-725.

Cheng NF, Han PZ, Gansky SA. 2008. Methods and software for estimating health disparities: the case of children's oral health. Am J Epidemiol 168(8):906-914.

Cheverud JM, Buikstra JE. 1981. Quantitative genetics for skeletal nonmetric traits in the rhesus macaques on Cayo Santiago I: Single trait heritabilities. Am J Phys Anthropol 54: 43-49.

Cheverud JM, Buikstra JE. 1982. Quantitative genetics for skeletal nonmetric traits in the rhesus macaques on Cayo Santiago III: Relative heritability of skeletal nonmetric and metric traits. Am J Phys Anthropol 59: 151-155.

Cheverud JM. 1988. A comparison of genetic and phenotypic correlations. Evolution 42:958-968.

Christensen AF. 1998. Skeletal evidence for familial interments in the Valley of Oaxaca, Mexico. Homo 49:273-288.

Christian GL. 1995. Black soldiers in Jim Crow Texas, 1899-1917. College Station, Texas: Texas A&M University Press.

Chung-Bridges K, C Muntaner, LE Fleming, DJ Lee, KL Arheart, WG LeBlanc, SL Christ, KE McCollister, AJ Caban, and EP Davila 2008. Occupational segregation as a determinant of US worker health. Am J Industrial Med 51:555-567.

Clark GA, NR Hall, GJ Armelagos, GA Borkan, MM Panjabi, FT Wetzel 1986. Poor early growth prior to childhood: decreased health and life-span in the adult. Am J Phys Anthropol 70:145-160.

Clark GA. 1988. New method for assessing changes in growth and sexual dimorphism in paleoepidemiology. Am J Phys Anthropol 77:105-116.

Clark GA, Hall NR, Aldwin CM, Harris JM, Borkan GA, Srinivasan M. 1988. Measures of poor early growth are correlated with lower adult levels of thymosin-α_1: Results from the normative aging study. Hum Biol 60(3):435-451.

Clark GA, Aldwin CM, Hall, NR, Spiro A, Goldstein A. 1989. Is poor early growth related to adult immune aging?: A follow-up study. Am J Hum Biol 1:331-337.

Clark C, O'Donnell J. 1999. Rearticulating a racial identity: Creating oppositional spaces to fight for equality and social justice. In Clark C, O'Donnell, editors. Becoming and unbecoming white: owning and disowning a racial identity. Westport, Connecticut: Bergin & Garvey. p 1-9.

Clark AL, Tayles N, Halcrow SE. 2014. Aspects of health in prehistoric mainland Southeast Asia: Indicators of stress in response to the intensification of rice agriculture. Am J Phys Anthropol 153:484-495.

Coale AJ. I957. How the age distribution of a human population is determined. Cold Spring Harbor Symposia on Quantitative Biology 22:83-88.

Cochran TC. 1968. The Great Depression and World War II, 1929-1945. Glenview, Illinois: Scott, Foresman

Cochran G, Harpending H. 2010. The 10,000 year explosion: How civilization accelerated human evolution. New York: Basic Books

Cockerham C. 2005. Health lifestyle theory and the convergence of agency and structure. J Health Soc Behav 46:51-67.

Coon CS. 1962. The origin of races. New York: Knopf.

Cravens H. 1978. Omnis cultura ex cultura. In Cravens H. The triumph of evolution: American scientists and the heredity-environment controversy 1900-1941. P 89-120.

Crouch BA 1992. The Freedman's Bureau and black Texas. Austin, Texas: The University of Texas Press.

Crump C, Winkleby MA, Sundquist K, Sundquist J. 2011. Risk of hypertension among young adults who were born preterm: A Swedish national study of 636,000 births. Am J Epidemiol 173(7):797-803.

Davidson JM, Rose JC, Gutmann M, Haines M, Condon C, and Condon K. 2002. The quality of African-American life in the Old Southwest near the turn of the 20th century. In: Steckel R and Rose JC, editors. The Backbone of History: Health and Nutrition in the Western Hemisphere. Cambridge University Press. p 226-277.

Davidson JM. 2004. Mediating race and class through the death experience: Power relations and resistance strategies of an African-American Community, Dallas, Texas (1869-1907), PhD Dissertation, Department of Anthropology, University of Texas at Austin.

Davidson JM. 2008. Identity and violent death: contextualizing lethal gun violence within the African American community of Dallas, Tx (1900-1907). J Soc Archaeol 8(3):320-354.

Dean MC, Reid DJ. 2001. Perikymata spacing and distribution on hominid anterior teeth. Am J Phys Anthropol 116:209-215.

Dean M, Stephens C, Winkler C, Lomb DA, Ramsburg M, Boaze R, Stewart C, Charbonneau L, Goldman D, Albough BJ, Goedert JJ, Beasley P, Hwang L-V, Buchbinder S, Weedon M, Johnson PA, Eichelberger M, O'Brian SJ. 1994. Polymorphic admixture typing in human ethnic populations. Am J Hum Genet 55:788–808.

Deitch C. 1993. Gender, race, and class politics and the inclusion of women in Title VII of the 1964 Civil Rights Act. Gender and Society 7(2):183-203.

de la Cova C. 2011. Race, health, and disease in 19th-century-born males. Am J of Phys Anthropol 144:526–537.

Delgado R, Stefanic J. 2001. Critical race theory: An introduction. New York: New York University Press.

Deporte JV. 1925. Inter-racial variation in infant mortality. *Am J Epidemiol* 5(4):454-496.

Di Comité L. 1986. Aspects of Italian emigration, 1881-1915. In: Glazier IA, De Rosa L, editors. Migration across time and nations: population mobility in historical contexts. International Congress on economic history. New York: Holmes & Meier.

Dillingham WP, D Folkmar, EC Folkmar 1911. Dictionary of races or peoples. United States, Immigration Commission (1907-1910), Washington, Govt. Print. Off.

Dinnerstein L, Nichols RL, Reimers DM. 2003. Natives and strangers: a multicultural history of Americans, 4th edition. New York: Oxford University Press

Dobzhansky T. 1971. Race equality. In: Osborne R, editor. The biological meaning of race. ed. San Francisco: W.H. Freeman and Company. p 13-24.

Donlon DA. 2000. The value of infracranial nonmetric variation in studies of modern Homo s apiens: An Australian focus. Am J Phys Anthropol 113:349-368.

Dowling HF. 1982. City hospitals: The undercare of the underprivileged. Cambridge, MA: Harvard University Press.

Dressler WW, Oths KS, Gravlee CC. 2005. Race and ethnicity in public health research: Models to explain health disparities. Ann Rev Anthropol 34:231-252.

Drago EL 1982. Black politicians and Reconstruction in Georgia: A splendid failure. Baton Rouge: Louisiana State University Press.

Du Bois WEB. 1975. Dusk to dawn: An essay toward an autobiography of a race concept. Kraus-Thomson Organization Limited: Millwood, N.Y.

Edgar HJH. 2009. Biohistorical approaches to "race" in the United States: Biological distances among African Americans, European Americans, and their ancestors, Am J Phys Anthropol 139:58-67.

Eisenstein S. 1977. The morphometry and pathological anatomy of the lumbar spine in South African Negroes and Caucasoids with specific reference to spinal stenosis. J Bone Joint Surg Br 59:173-180.

Eisenstein S. 1983 Lumbar vertebral canal morphometry for computerized tomography in spinal stenosis. Spine 8:187-191.

El-Naijar MY, Desanti MV, Ozabek L. 1978. Prevalence and possible etiology of dental enamel hypoplasia. Am J Phys Anthropol 48(2):185-192.

Ely JW, Jr., Bodenhamer DJ. 1984. "Regionalism and the legal history of the South." In: Bodenhamer DJ, Ely JW, editors. Ambivalent legacy: A legal history of the South. Jackson, MI: University Press of Mississippi. p 3-29.

Epperson TW 1996. The politics of 'race' and cultural identity at the African Burial Ground Excavations, New York. World Archaeol Bull 7:108-117.

Ernst R. 1949. Immigrant life in New York City, 1825-1863. New York: King's Crown Press.

Field A. 2013. Discovering statistics using IBM SPSS statistics, 4th edition. London: Sage.

Foner E. 2014. Reconstruction: America's unfinished revolution, 1863-1877. New York: Harper Perennial 2014.

Ford RT. 1997. The boundaries of race: Political geography in legal analysis. In: Gates EN, editor. Critical race theory: Essays on the social construction and reproduction of "race." New York: Garland Publishing, Inc. p 213-291.

Franklin JH. 1956. The militant South: 1800-1861. Cambridge, MA: The Belknap Press of Harvard University Press.

Franklin M. 1997. 'Power to the people': Sociopolitics and the archaeology of black Americans. Hist Archaeol 31(3):36-50.

Franklin M. 2001b. A black feminist-inspired archaeology? J Soc Archaeol 1(1):108–125.

Franklin JH, Moss AA, Jr. 1994. From slavery to freedom: A history of African Americans, 7th edition. New York: McGraw-Hill Inc.

Fredrickson GM 1971. The black image in the white mind: The debate on Afro-American character and destiny, 1817-1914. New York: Harper & Row, Publishers.

Friedman-Jimenez G, Claudio L. 1998. Environmental justice. In: Rom WN, editor. Environmental and occupational medicine, 3rd editon. Philadelphia, Pa: Lippincott-Raven Publishers. p 1731-1740.

Gallman JM. 2000. Receiving Erins children: Philadelphia, Liverpool, and the Irish famine migration, 1845-1855. Chapel Hill: University of North Carolina Press.

Garn SM. 1971. Human races, 3rd editon. Springfield: Thomas.

Geber J. 2014. Skeletal manifestation of stress in child victims of the Great Irish Famine (1845-1852): Prevalence of enamel hypoplasia, harris lines, and growth retardation. Am J Phys Anthropol 155:149-161.

Giles E. 2010. Principle figures in physical anthropology before and during World War II. In: Little MA, Kennedy KAR, editors. Histories of American physical anthropology in the twentieth century. Lanham, MD: Lexington Books. p 141-154.

Giles E. 2012. Two faces of Earnest A. Hooton. Yr Phys Antropol 55:105-113.

Gilroy P. 2003. The Black Atlantic as counterculture to modernity. In: Braziel JE, Mannur A, editors. Theorizing diaspora. Oxford, England, UK: Blackwell Publishing. p. 49-82.

Goldberg DT. 2005. Racial Americanization. In: Murji K, Solomos J, editors. Racialization studies in theory and practice. Oxford: Oxford University Press. p 87-102.

Goldstein MS. 1948. Franz Boas' contributions to physical anthropology. Am J Phys Anthropol 4:145-161.

Goodman AH, Martin DL, Armelagos GJ, Clark G. 1984. Indicators of stress from bones and teeth. In: Cohen MN, Armelagos GJ, editors. Paleopathology at the origins of agriculture. New York: Academic Press, Inc. p 13-49.

Goodman AH. 2000. Why genes don't count (for racial differences in health). Am J Public Health 90:1699–1702.

Goodman AH, Thomas RB, Swedlund AC, Armelagos GJ. 1988. Biocultural perspectives on stress in prehistoric, historical, and contemporary population research. Y Phys Anthropol 31:169-202.

Goodman AH, Rose JC. 1990. Assessment of systemic physiological perturbations from dental enamel hypoplasias and associated histological structures. Year Phys Anthropol 33:59-110.

Goodman AH, Leatherman TL. 1998. Traversing the chasm between biology and culture: an introduction. In: Goodman AH, Leatherman TL, editors. Building a new biocultural synthesis: political-economic perspectives on human biology. Ann Arbor, The University of Michigan Press. p 3-41.

Goodman AH, Martin DL. 2002. Reconstructing health profiles from skeletal remains. In: Steckel RH, Rose JC, editors. The backbone of history: health and nutrition in the western hemisphere. New York: Cambridge University Press. p 11-60.

Gravlee CC, Bernard HR, Leonard WR. 2003a. New answers to old questions: did Boas get it right?: heredity, environment, and cranial form: a reanalysis of Boas's immigrant data. Am Anthropol 105(1):125-138.

Gravlee CC, Bernard HR, Leonard WR. 2003b. Exchange across difference: did Boas get it right or wrong?: Boas's changes in bodily form: the immigrant study, cranial plasticity, and Boas's physical anthropology. Am Anthropol 105(2):326-332.

Gravlee CC, Non AL, Mulligan CJ. 2009. Genetic ancestry, social classification, and racial inequalities in blood pressure in southeastern Puerto Rico. PLoS One 4:e6821.

Gravlee CC 2009. How Race Becomes Biology: Embodiment of Social Inequality. Am J Phys Anthropol 139:47-57.

Green NL. 2012. Changing paradigms in migration studies: From men to women to gender. Gender and History: 24(3):782–798.

Greenberg DS. 1990. Black health: Grim statistics. Lancet 355:780-781.

Grimshaw AD. 1969. Racial violence in the United States. Chicago: Aldine Publishing Company.

Grüneberg H. 1952. Genetical studies on the skeleton of the Mouse IV: Quasi-continuous variation. J Genet 51:95-114.

Guatelli-Steinberg D. 2004. Analysis and significance of linear enamel hypoplasia in Plio-Pleistocene hominins. Am J Phys Anthropol 123:199-215.

Guatelli-Steinberg D, Ferrell RJ, Spence J. 2012. Linear enamel hypoplasia as an indicator o physiological stress in great apes: Reviewing the evidence in light of enamel growth variation. Am J Phys Anthropol 148:191-204.

Hallgrimsson B, BO Donnabhain, GB Walters, DML Cooper, D Guobjartsson, K Stefansson 2004. Composition of the founding oopulation of Iceland: Biological distance and morphological variation in Early Historic Atlantic Europe. Am J Phys Anthropol 124:257-274.

Hanihara T, Ishida H, Dodo Y. 2003. Characterization of biological diversity through analysis of discrete cranial traits. Am J Phys Anthopol 121:241-251.

Harpending H, Relethford JH, and Sherry ST. 1996. Methods and models for understanding human diversity. In: Boyce AJ, Mascie-Taylor CGN, editors. Molecular biology and human diversity. Cambridge: Cambridge University Press. p 283-299.

Harris CI. 1997a. Whiteness as property. In: Gates EN, editor. Critical race theory: Essays on the social construction and reproduction of "race." New York: Garland Publishing, Inc. p 1-83.

Harris AP. 1997b. Foreword: The jurisprudence of Reconstruction. In: Gates EN, editor. Critical race theory: Essays on the social construction and reproduction of "race." New York: Garland Publishing, Inc. p 347-391.

Haskins AE, Clark DE, Travis LL. 2013. Racial disparities in survival among injured drivers. Am J Epidemiol 177(5):380–387.

Hauser G, De Stefano GF. 1989. Epigenetic variants of human skull. Stuttgart: Schweizerbart.

Hawkey DE, Merbs CF. 1995. Activity-induced musculoskeletal stress markers (MSM) and subsistence strategy changes among ancient Hudson Bay Eskimos. Inter J Osteoarchaeol 5:324-338.

Hero RE. 2010. Immigration and social policy in the United States. Annu Rev Polit Sci 13:445-468.

Hershkovitz I, Greenwald C, Rothschild BM, Latimer B, Dutour O, Jellema LM, Wish-Baratz. 1999. Hyperostosis frontalis interna: an anthropological perspective. Am J of Phys Anthropol 109:303-325.

Hertzog K. 1968 Association between discontinuous cranial traits. Am J Phys Anthropol 29:397-404.

Hillson S. 1992. Impression and replica methods for studying hypoplasia and perikymata on human tooth crown surfaces from archaeological sites. Int J Osteoarchaeol 2:65-78.

Hillson S. 1996. Dental anthropology. Cambridge University Press.

Hillson S, Bond S. 1997. Relationship of enamel hypoplasia to the pattern of tooth crown growth: A discussion. Am J Phys Anthropol 104:89-103.

Hinck VC, Clark WM, Hopkins CE. 1966. Normal interpediculate distances (minimum and maximum) in children and adults. Am J Roentgenol 97:141-153.

Hindman HD. 2002. Child labor: An American history. Armonk, N.Y. : M.E. Sharpe.

Hjalgrim LL, Westergaard T, Rostgaard K, Schmiegelow K, Melbye M, Hjalgrim H, Engels EA. 2003. Birth weight as a risk factor for childhood leukemia: A meta-analysis of 18 epidemiologic studies. Am J Epidemiol 158 (8):724-735.

Hollinger DA 2003. Amalgamation and hypodescent: The question of ethnoracial mixture in the history of the United States. Am. Hist. Rev. 108:1363-1390.

Holt TC. 1982. “An empire over the mind”: Emancipation, race, and ideology in the British West Indies and the American South. In: Kousser JM, McPherson JM, editors. Region, race, and Reconstruction: Essays in honor of C. Vann Woodward. New York: Oxford University Press. p 283-313.

Horne G. 1996. Race for the globe: U.S. foreign policy and racial interests. In: Bowser BP, Hunt

RG, editors. Impacts of racism on white Americans, 2nd edition. Thousand Oaks, California: SAGE Publications, Inc. p 88-112.

Howard WT, Jr. 1921. The real risk-rate of death to mothers from causes connected with childbirth. *Am J Epidemiol* 1(2):197-233.

Howells W. 1971. The meaning of race. In: Osborne R, editor. The biological meaning of race. San Francisco: W.H. Freeman and Company. p 3-12.

Howson JE 1990. Social relations and material culture: A critique of the archaeology of plantation slavery. *Historical Archaeology* 24(4):78-91.

Hubbard A, Guatelli-Steinberg D, Sciulli PW. 2009. Under restrictive conditions, can the widths of linear enamel hypoplasias be used as relative indicators of stress episode duration? Am J Phys Anthropol 138 (2):177-189.

Hudson M. 1999. The ruins of identity: ethnogenesis on the Japanese islands. Honolulu: University of Hawaii Press.

Hughes C, Heylings DJA, Power C. 1996. Transverse (Harris) lines in Irish archaeological remains. Am J Phys Anthropol 101(1):115-131.

Hughes L. 2007. Jim Crow's last stand. Whitefish, MN.: Kessinger.

Hunley KL, Cabana GS, Long JC. 2016. The apportionment of human diversity revisited. Am J Phys Anthropolo 160:561-569.

Hunt DR, J Albanese 2005. History and demographic composition of the Robert J. Terry Anatomical Collection. Am J Phys Anthropol 127:406-417.

Ignatiev N 1995. How the Irish became white. New York: Routledge.

Jacobs DE, Wilson J, Dixon SL, Smith J, Evans A. 2009. The relationship of housing and population health: a 30-year restrospective analysis. Environ Health Perspec 117(4): 597-604.

Jacobson MF. 1998. Whiteness of a different color: European immigrants and the alchemy of race. Cambridge, MA: Harvard University Press.

Jeffrey JE, Campbell DM, Golden MHN, Smith FW, Porter RW. 2003. Antenatal factors in the development of the lumbar vertebral canal. Spine 28(13):1418-1423.

Johnson LC. 1965. Discussion. In: Jarcho S, editor. Human palaeopathology. New Haven and London: Yale University Press. p 68-81.

Johannsen RW. 1970. Reconstruction, 1865-1877. New York: Free Press.

Jones JM. 1972. Prejudice and racism. Reading, MA: Addison-Wesley.

Jones JM, Carter RT. 1996. Racism and white racial identity: Merging realities. In: Bowser BP, Hunt RG, editors. Impacts of racism on white Americans, 2nd edition. Thousand Oaks, California: SAGE Publications, Inc. p 1-23.

Jurmain RD. 1977. Stress and the etiology of osteoarthritis. Am J of Phys Anthropol 46:353-366.

Jurmain RD. 1999. Stories from the skeleton: Behavioral reconstruction in human osteology. Amsterdam: Gordon and Breach.

Kelley JO, Angel JL. 1987. Life stresses of slavery. Am J Phys Anthropol 74:199-211.

Kendall FE. 2006. Understanding white privilege: Creating pathways to authentic relations across race. New York: Routledge Taylor & Francis Group.

Keneally T. 1998. The great shame: A story of the Irish in the old world and the new. London: Chatto & Windus.

Kennedy S. 1959. Jim Crow guide to the U. S. A.: The laws, customs, and etiquette governing the conduct of nonwhites and other minorities as second-class citizens. London: Lawrence & Wishart.

Kershaw KN, Diex Roux AV, Burgard SA, Lisabeth LD, Mujahid MS, Schulz AJ. 2011. Metropolitan-level racial residential segregation and black-white disparities in hypertension. Am J Epidemiol 174(5): 537-545.

Keyfitz N. I985. Applied mathematical demography, 2nd edition. Berlin: Springer-Verlag.

Kierdorf H, Witzel C, Kierdorf U, Skinner MM, Skinner MF. 2015. “Missing perikymata”-fact or fiction? A study on chimpanzee (Pan troglodytes verus) canines. Am J Phys Anthropol 157:276-283.

King JA, Chaney EE. 2010. Passing for Black in seventeenth-century Maryland. In: Interpreting the early modern world: Transatlantic perspectives. Beaudry M.C., Symonds J, editors. Boston: Springer. p 87-112.

King T, Humphrey LT, Hillson S. 2005. Linear enamel hypoplasias as indicators of systemic physiological stress: evidence from two known age-at-death and sex populations from postmedieval London. Am J Phys Anthropol 128:547-559.

Klein A. 2012. Slipping racism into the mainstream: A theory of information laundering. Commun Theory 22:427–448.

Konigsberg L, Holman DJ. 1999. Estimation of age at death from dental emergence and implications for studies of prehistoric somatic growth. In: Hoppa RD and Fitzgerald CM,

editors. Human growth in the past: Studies from bones and teeth. Cambridge: Cambridge University Press. p 264-289.

Konigsberg LW, Algee-Hewitt BFB, Steadman DW. 2009. Estimation and evidence in forensic anthropology: Sex and race. Am J Phys Anthropol 139:77-90.

Knowles LL, Prewitt K. 1969. Institutional racism in America. Englewood Cliffs, New Jersey: Prentice-Hall, Inc.

Kramer A, Konigsberg LW. 1999. Recognizing species diversity among large-bodied hominoids: a simulation test using missing data finite mixture analysis.

Kramer MR, Valderrama AL, Casper ML. 2015. Decomposing black-white disparities in heart disease mortality in the United States, 1973–2010: An age-period-cohort analysis. Am J Epidemiol 182(4):302-312.

Krieger N. 2001. Theories for social epidemiology in the 21st Century: An ecosocial perspective. Intl J of Epidemiol 30(4):668–677.

Krieger N 2010. Workers Are People Too: Societal aspects of occupational health disparities—An ecosocial perspective. Am J Industrial Med 1-12.

Krieger N. 2011. Epidemiology and the people's health: Theory and context. Oxford: Oxford University Press.

Lanphear KM. 1990. Frequency and distribution of enamel hypoplasias in a historic skeletal sample. Am J Phys Anthropol 81:35-43.

Largent M. 2008. Breeding contempt: The history of coerced sterilization in the United States. New Brunswick, NJ: Rutgers University Press

La Roche C, Blakey M. 1997. Seizing intellectual power: The dialogue at the New York African Burial Ground. Hist Archaeol 31:84-106.

Larsen CS. 1994. In the wake of Columbus: native population biology in the postcontact Americas. Yr Phys Anthropol 37:109–154.

Larsen CS. 1997. Bioarchaeology. Cambridge: Cambridge University Press.

Larson EJ. 2004. Genetics enters the picture. In: Larson EJ. Evolution: the remarkable history of a scientific theory. New York: The Modern Library. p 151-174.

Leatherman T, Jernigan K. 2015. Introduction: Biocultural contributions to the study of health disparities. Annals Anthropol Pract 38(2):171-186.

Leone MP. 2010. Critical historical archaeology. Walnut Creek, CA: Left Coast Press, Inc.

Leviton A, Allred EN, Kuban KCK, O'Shea TM, Paneth N, Onderdonk AB, Fichorova RN, Dammann O. 2016. The development of extremely preterm infants born to women who had genitourinary infections during pregnancy. Am J Epidemiol 183(1):28-35.

Lewontin R. 1972. The apportionment of human diversity. Evolutionary Biology 6: 381-398.

Li D-K, Daling JR. 1991. Maternal smoking, low birth weight, and ethnicity in relation to sudden infant death syndrome. Am J Epidemiol 134 (9):958-964.

Lian JB, Stein, GS. 1999. The cells of bone. In: Seibel MJ, Robins SP, Bilezikian JP, editors. Dynamics of bone and cartilage metabolism. New York: Academic Press. p 165-185.

Little MA. 2010. Franz Boas's place in American physical anthropology and its institutions. In: Little MA, Kennedy KAR, editors. Histories of American physical anthropology in the twentieth century. Lanham, MD: Lexington Books. p 55-85.

Linn MB. 2010. Elixir of emigration: Soda water and the making of Irish Americans in nineteenth-century New York City. Historical Archaeology 44(4):69-109.

Livingstone FB. 1962. On the nonexistence of human races. Curr Anthropol 3:279-281.

Livingstone FB. 1964. On the nonexistence of human races. In Montagu A, editor. The concept of race. London: The Free Press of Glencoe. p 46-60.

Loomis D, Richardson D. 1998. Race and the risk of fatal injury at work. Am J Pub Health 88:40-44.

Lucas REB. 1974. The distribution of job characteristics. The Review of Economics and Statistics 56:530-540

Magath TB. 1937. Hydatid (echinococcus) disease in Canada and the United States. Am J Epidemiol 25(1): 107-134.

Maijanen HMK 2014. The prevalence of pathological conditions in a modern skeletal collection: implications for forensic anthropology. Doctoral Dissertation; University of Tennesseee

Mann RW, Murphy SP. 1990. Regional atlas of bone disease: A guide to pathologic and normal variation in the human skeleton. Springfield: Charles C Thomas Publisher.

Marks J. 2010. The two 20th-century crises of racial anthropology. In: Little MA, Kennedy KAR, editors. Histories of American physical anthropology in the twentieth century. Lanham, MD: Lexington Books. p 187-206.

Martínez-Abadías N, Esparza M, Sjovold T, González-José R, Santos M, Hernández M. 2009. Heritability of human cranial dimensions: comparing the evolvability of different cranial regions.

Martinot S. 2003. The rule of racialization: class, identity, governance. Philadelphia: Temple University Press.

Martorell R, González-Cossío T. 1987. Maternal nutrition and birth weight. Am J Phys Anthropol 30(S8):195–220.

McCaffrey LJ. 1997. The Irish-Catholic Diaspora in America. Washington, D.C.: Catholic University Press.

McCarthy M. 2000. Social determinants and inequalities in urban health. Reviews on Environ Health 15(1-2):97-108.

McDavid C. 1997. Descendants, decisions, and power: The public interpretation of the archaeology of the Levi Jordan Plantation. Hist Archaeol 31:114-31.

McDermott M, FL Samson 2005. White racial and ethnic identity in the United States. Annu Rev Sociol 31:245-261.

McElvaine RS. 1984. The Great Depression : America, 1929-1941. New York, N.Y.: Times Books

Mcllvaine BK, LA Schpartz, CS Larsen, PW Sciulli 2014. Evidence of long-term migration on the Balkan Peninsula using dental and cranial nonmetric data: Early interaction between Corinth (Greece) and its Colony at Apollonia (Albania), Am J Phys Anthropol 153:236-248.

Meskell L, Preucel R. 2004. Identities. In: A companion to social archaeology, Meskell L, Preucel R, editors. Oxford, U.K.: Blackwell Publishing. p 121-141.

Miele F, Sariach V. 2005. Race: The reality of human differences. Boulder, CO: Westview Press.

Molnar P. 2006. Tracing prehistoric activities: Musculoskeletal stress marker analysis of a stone-age population on the Island of Gotland in the Baltic sea. Am J Phys Anthropol 129(1):12–23.

Montagu A. (1964). The concept of race. London: The Free Press of Glencoe.

Moodie RL. 1923. Paleopathology: An introduction to the study of ancient evidences of disease. Urbana, Illinois: University of Illinois Press.

Mooney JEP. 2010. Re-visiting histories of modernization, progress, and (unequal) citizenship rights: Coerced sterilization in Peru and in the United States. University of Arizona: History Compass.

Moore LE. 2006. Toward a still and quiet conscience: A study in reflexive archaeology. North Am Archaeol 27(2):149-174.

Morris TD. 1984. ““Society is not marked by punctuality in the payment of debts”: The Chattel Mortgages of Slaves.” In: Bodenhamer DJ, Ely JW, Jr., editors. Ambivalent Legacy: A Legal History of the South. Jackson, MI: University Press of Mississippi. p 147-170.

Morton MJ. 2000. Institutionalizing inequalities: Black children and child welfare in Cleveland, 1859-1998. J Soc Hist 34(1):141-162.

Moskowitz RW 1984. Introduction. In: Moskowitz RW, Howell DS, Goldberg VM, Mankin HJ, editors. Osteoarthritis: Diagnosis and management. Philadelphia: W.B. Saunders Company. p 1-6.

Mullins PR, Jones L. 2011. Archaeologies of race and urban poverty: The politics of slumming, engagement, and the color line. Hist Archaeol 45(1) 33-50.

Mullins PR. 1999. Race and affluence: An archaeology of African America and consumer culture. New York: Kluwer Academic.

Murji K, Solomos J, 2005. Introducton: Racialization in theory and practice. In: Murji K, Solomos J, editors. Racialization: studies in theory and practice. New York: Oxford University Press. p 1-28.

Nassaney MS. 2007. Forward. In: Orser CE., Jr., editor. The archaeology of race and racialization in historic America: the American experience in archaeological perspective. Gainesville: University of Florida Press, xi-xiii.

National Institute of Mental Health. 5 things you should know about stress (No. OM 16-4310). Retrieved from https://www.nimh.nih.gov/health/publications/stress/index.shtml

Nei M, Roychoudhury AK. 1982. Evolutionary relationships and evolution of human races. Evol Biol 14:1–59.

NeiM. 1987. Molecular Evolutionary Genetics. New York: Columbia University Press.

Newby IA. 1965. Jim Crow's defense; anti-Negro thought in America, 1900-1930. Baton Rouge: Louisiana State University Press.

Newell EA, Guatelli-Steinberg D, Field M, Cooke C, Feeney RNM. 2006. Life history, enamel formation, and linear enamel hypoplasia in the Ceboidea. Am J Phys Anthropol 131:252-260.

Newman SL, Gowland RL. 2015. Brief communication: The use of non-adult vertebral dimensions as indicators of growth disruption and non-specific health stress in skeletal populations. Am J Phys Anthropol 00:00-00.

Nikita E, Mattingly D, Lahr MM. 2012. Sahara: Barrier or corridor? Nonmetric cranial traits and

biological affinities of North African Late Holocene populations. Am J Phys Anthropol 147:280-292.

Nix E, Qian N. 2015. The fluidity of race:"Passing" in the United States, 1880-1940 (No. w20828). National Bureau of Economic Research.

O'Campo P, Burke JG, Culhane J, Elo IT, Eyster J, Holzman C, Messer LC, Kaufman JS, Laraia BA. 2008. Neighborhood deprivation and preterm birth among non-hispanic black and white women in eight geographic areas in the United States. Am J Epidemiol 167(2):155-163.

Okechukwu CA, Souza K, Davis KD, de Castro AB. 2014. Discrimination, harassment, abuse, and bullying in the workplace: Contribution of workplace injustice to occupational health disparities. Am J Industrial Med 57:573–586

Oliver NM, Muntaner C. 2005. Researching health inequities among African Americans: The imperative to understand social class. Intl J Health Serv 35(3):485-498.

Oliveti JF, Kercsmar CM, Redlin S. 1996. Pre- and perinatal risk factors for asthma in inner city African-American children. Am. J. Epidemiol 143(6):570-577.

Omi M, Winant H. 1986. Racial formation in the United States from the 1960s to the 1980s. New York: Routledge & Kegan Paul.

Orser CE., Jr. 1991. The archaeological search for ethnicity in the historic United States. Archaeologia Polona 29:109-121.

Orser CE, Jr. 2007. The archaeology of race and racialization in historic America: The American experience in archaeological perspective. Gainesville: University of Florida Press.

Orser CE, Jr. 1998. The challenge of race to american historical archaeology. Am Anthropol 100:661-668.

Orser CE, Jr. 1998. The archaeology of the African diaspora. Ann Rev Anthropol 27:63-82.

Ortner DJ. 1991. Theoretical and methodological issues in paleopathology. In: Ortner DJ, Aufderheide AC, editors. Human paleopathology: Current syntheses and future options. Washington: Smithsonian Institution Press. p 5-11.

Ortner DJ. 2010. Aleš Hrdlička and the founding of the American journal of physical anthropology: 1918. In: Little MA, Kennedy KAR, editors. Histories of American physical anthropology in the twentieth century. Lanham, MD: Lexington Books. p 87-104.

Osborne R. 1971. Races and the future of Man. In: Osborne R, editor. The biological meaning of race. San Francisco: W.H. Freeman and Company. p 149-160.

Osypuk TL, Acevedo-Garcia D. 2008. Are racial disparities in preterm birth larger in hypersegregated areas? Am J Epidemiol 167(11):1295-1304.

Ossenberg NS. 1984. Within and between race distances in population studies based on discrete traits of the human skull. Am J Phys Anthropol 45:701-716.

Ossenberg NS. 2013. Cranial nonmetric trait database. Kingston, Ontario, Canada: Nancy S. Ossenberg (producer). Queen's University Library, Data and Government Information Centre (distributor).

Painter NI. 2010. The history of white people. New York: W.W. Norton and Company.

Papp T, Porter RW, Aspden RM. 1994. The growth of the lumbar vertebral canal. Spine 19:2770-2773.

Parrillo VN. 2000. Strangers to these shores: Race and ethnic relations in the United States, 5th ed. Boston, MA: Allyn and Bacon.

Paynter R, McGuire R. 1991. The archaeology of inequality: material culture, domination, and resistance. In: McGuire R, Paynter R, editors. The archaeology of inequality, Oxford, U.K.: Basil Blackwell. p 1-27.

Pascoe P 1996. Miscegenation law, court cases, and ideologies of "race" in twentieth-century America. J Am Hist 83:44-69.

Pearl R. 1921. The vitality of the peoples of America. *Am J Epidemiol* 1(5):592-674.

Pernick MS. 1977. Public health then and now: Eugenics and public health in American history. Am J Pub Health 87:1767-1772.

Phillips KL 1996. "But it is a fine place to make money": Migration and African-American families in Cleveland, 1915-1929. J Soc Hist 30(2):393-413.

Philp KR. 1977. John Collier's crusade for Indian reform, 1920-1954. Tucson: University of Arizona Press.

Pinder SO. 2012. Whiteness and racialized ethnic groups in the United States: the politics of remembering. Lanham, Maryland: Lexington Books

Pinder SO. 2015. Colorblindness, post-raciality, and whiteness in the United States. New York: Palgrave Macmillan.

Platt BS, Stewart RJC. 1962. Transverse trabeculae and osteoporosis in bones in experimental protein-calorie deficiency. Br J Nutr 16:483-495.

Porter RW, Hibbert CS, Wellman R. 1980. Backache and the lumbar spinal canal. Spine 8:99-105.

Porter RW, Drinkall JN, Porter DE, Thorp L. 1987. The vertebral canal: II. Health and academic status, a clinical study. Spine 12(9):907-911.

Porter RW, Pavitt D. 1987. The vertebral canal: I. Nutrition and development, an archaeological study. Spine 12(9):901-906

Prelow HM, S Danoff-Burg, RR Swenson, and D Pulgiano 2004. The Impact of Ecological Risk and Perceived Discrimination on the Psychological Adjustment of African American and European American Youth. *Journal of Community Psychology* 32(4):375-389.

Prowse TL, Lovell NC. 1995. Biological continuity between the A- and C-Groups in Lower Nubia: evidence form cranial nonmetric traits. Int J Osteoarchaeol 5:103:114.

Quigley C 2001. Skulls and skeletons: human bone collections and accumulations. Jefferson, North Carolina: McFarland & Company, Inc. Publishers.

R Core Team. 2013. R: A Language and Environment for Statistical Computing. R Foundation for Statistical Computing, Vienna, Austria. URL http://www.R-project.org/.

Rable GC. 1984. But there was no peace: The role of violence in the politics of Reconstruction. Athens, GA: The University of Georgia Press.

Rasmussen BB, Klinenberg E, Nexica IJ, Wray M. 2001 Introduction. In: Rasmussen BB, editor. The making and unmaking of whiteness. Durham N.C. : Duke University Press. p 1-24.

Reid DJ, Dean MC. 2000. Brief communications: The timing of linear hypoplasias on human anterior teeth. Am J Phys Anthropol 113:135-139.

Reid DJ, Dean MC. 2006. Variation in modern human enamel formation times. J Hum Evol 50:329-346.

Relethford JH. 2003. Reflections of our past: how human history is revealed in our genes. Boulder, CO: Westview Press.

Relethford JH, Lees FC. 1982. The use of quantitative traits in the study of human population structure. Yr Phys Anthropol 25:113-132.

Reverby SM. 2010. Invoking "Tuskegee": Problems in health disparities, genetic assumptions, and history. J Health Care Poor Underserved 21:26-34.

Richardson D, D Loomis, SH Wolf, E Gregory 1997. Fatal agricultural injuries in North Carolina by race and occupation, 1977-1991. Am J Industrial Med 31:452-458.

Ribas-Fitó N, Torrent M, Carrizo D, Muñoz-Ortiz L, Júlvez J, Grimalt JO, Sunyer J. 2006. In utero exposure to background concentrations of DDT and cognitive functioning among preschoolers. Am J Epidemiol 164(10):955-962.

Riga A, Belcastro MG, Moggi-Cecchi J. 2014. Environmental stress increases variability in the expression of dental cusps. Am J Phys Anthropol 153:397-407

Rightmire GP 1999. Dental variation and human history. Rev Archaeol 20:1-3.

Robinson JC. 1984. Racial Inequality and the Probability of Occupation-Related Injury or Illness. Milbank Mem Fund Q Health Soc. 62:567-590.

Roediger D 1992. The wages of whiteness: race and the making of the American working class. London: Verso.

Roediger DR. 2005. Working toward whiteness: How America's immigrants became white, the strange journey from Ellis Island to the suburbs. New York: Basic Books.

Roseman CC, Weaver TD. 2004. Multivariate apportionment of global human craniometrics diversity. Am J Phys Anthropol 125:257-263.

Roseman CC. 2014. Troublesome reflection: Racism as the blind spot in the scientific critique of race. Hum Biol 86(3):233-240.

Rosenberg CM. 2013. Child labor in America: A history. Jefferson, North Carolina: McFarland & Company, Inc., Publishers.

Rosenberg NA, Pritchard JK, Weber JL, Cann HM, Kidd KK, Zhivotovsky LA, Feldman MW. 2002. Genetic structure of human populations. Science 298:2381–2385.

Rushton JP. 2000. Race, evolution, and behavior: a life history perspective. Port Huron, MI : Charles Darwin Research Institute.

Ryman N, Chakraborty R, Nei M. 1983. Differences in the relative distribution of human genetic diversity between electrophoretic and red and white cell antigens. Hum Heredity 33:93–102.

Saitta DJ. 2007. The archaeology of collective action: the American experience in archaeological perspective. Gainesville: University Press of Florida

Sandlin JA, Bey 3rd GJ. 2006. Trowels, trenches and transformation: A case study of archaeologists learning a more critical practice of archaeology. J Social Archaeol 6(2):255–276.

Sandberg PA, Sponheimer M, Lee-Thorp J, Van Gerven D. 2014. Intra-tooth stable isotope analysis of dentine: A step toward addressing selective mortality in the reconstruction of life history in the archaeological record. Am J Phys Anthropol 155:281-293.

Saperstein A, Penner AM, Light R. 2013. Racial formation in perspective: connecting individuals, institutions, and power relations. Annu Rev Sociol 39:359-378.

Saunders SR, Popovich F. 1978. A family study of two skeletal variants: atlas bridging and clinoid bridging. Am J Phys Anthropol 49:193-203.

Sauer NJ. 1992. Forensic anthropology and the concept of race: If races don't exist, why are forensic anthropologists so good at identifying them? Soc. Sci. Med. 34(2):107-111.

Schwarz PJ. 1996. Slave laws in Virginia. Athens: University of Georgia Press.

Schultz MR. 1994. Interracial kinship ties and the emergence of a rural black middle class: Hancock County, Georgia, 1865-1920. In: Inscoe JC, editor. Georgia in black and white: Explorations in the race relations of a southern state, 1865-1950. Athens, Georgia: The University of Georgia Press. p 141-172.

Scrimshaw NS, Behar M. 1965. Malnutrition in underdeveloped countries. N. Engl. J. Med. 271:137–144.

Shanks M, Tilley C. 1987. Social theory and archaeology. Cambridge, UK: Polity Press.

Sipola-Leppänen M, Vääräsmäki M, Tikanmäki M, Matinolli H-M, Miettola S, Hovi P, Wehkalampi K, Ruokonen A, Sundvall J, Pouta A, Eriksson JG, Järvelin M-R, and Kajantie E. 2015. Cardiometabolic Risk Factors in Young Adults Who Were Born Preterm. Am. J. Epidemiol. 181(11):861-873.

Shirley NR, RJ Wilson, LM Jantz 2011. Cadaver use at the University of Tennessee's Anthropological Research Facility. Clin Anat 24:372-380.

Singleton T. 2006. African diaspora archaeology in dialogue. In: Yelvington KA, editor. Afro-Atlantic dialogues, anthropology in the diaspora. Santa Fe, New Mexico: SAR Press. p 249-287.

Sitton T, Conrad JH. 2005. Independent Black Texans in the Time of Jim Crow. Austin, Texas: University of Texas Press.

Skinner MF, Pruetz JD. 2012. Reconstruction of periodicity of repetitive linear enamel hypoplasia from perikymata counts on imbricational enamel among dry-adapted chimpanzees (Pan troglodytes versus) from Fongoli, Senegal. Am J Phys Anthropol 149:468-482.

Smedley A 1998. 'Race' and the construction of human identity. Am Anthropol 100:690-702.

Smith HM, Chiszar D, Montanucci RR. 1997. Subspecies and classification. Herpetological Rev 28:13-16.

Smith CF 2008. Here lies Jim Crow: Civil rights in Maryland. Baltimore, Maryland: The Johns Hopkins University Press.

Spradley MK, Jantz RL, Robinson A, Peccerelli F. 2008. Demographic change and forensic identification: Problems in metric identification of Hispanic skeletons. J Forensic Sci 53:21–28.

Stanton WR. 1960. The leopard's spots: Scientific attitudes toward race in America, 1815-1859. Chicago: University of Chicago Press.

Starr K. 1996. Endangered dreams : the Great Depression in California. New York: Oxford University Press.

Steckel RH, Rose JC. 2002. Patterns of health in the western hemisphere. In: Steckel RH, Rose JC, editors. The backbone of history: health and nutrition in the western hemisphere. New York: Cambridge University Press. p 563-582.

Steggerda M. 1944. Charles Benedict Davenport (1866-1944); The man and his contributions to physical anthropology. Am J Phys Anthropol 2(2):167-185.

Steinbock TR. 1976. Paleopathological diagnosis and interpretation: Bone disease in ancient human populations. Springfield: Charles C. Thomas Publisher.

Stephen TM. 1999. Dictionary of Latin American racial and ethnic terminology, 2nd ed. Gainesville: University Press of Florida.

Stern AM. 2005. Sterilized in the name of public health: race, immigration, and reproductive control in modern California. Am J Pub Health 95(7):1128-38.

Stini WA. 1969. Nutritional stress and growth: Sex differences in adaptive responses. Am J Phys Anthropol 31:417–426.

Storey R. 1988. Prenatal enamel defects in Teotihuacan and Copan. Am J Phys Anthropol 75:275-276.

Taylor C, Jr. 1974. A case study of the consequences of displacement caused by urban renewal and highway construction on minority businesses in the City of Knoxville, Tennessee. A thesis presented for the master of science in planning degree: The University of Tennessee, Knoxville.

Temple DH. 2010. Patterns of systemic stress during the agricultural transition in prehistoric Japan. Am J Phys Anthropol 142:112-124.

Temple DH. 2014. Plasticity and constraint in response to early-life stressors among late/final Jomon period foragers from Japan: Evidence for life history trade-offs from incremental microstructures of enamel. Am J Phys Anthropol 155:537-545.

Temple DH, McGroarty JN, Guatelli-Steinberg D, Nakatsukasa M, Matsumura H. 2013. A comparative study of stress episode prevalence and duration among Jomon period foragers from Hokkaido. Am J Phys Anthropol 152:230-238.

Templeton AR. 1999. Human races: A genetic and evolutionary perspective. Am Anthropol 100(3):632-650.

Templeton AR. 2013. Biological races in humans. Stud Hist Philos Biol Biomed Sci 44(3):262-271.

Turner BL, Armelagos GJ. 2012. Diet, residential origin, and pathology at Machu Picchu, Peru. Am J Phys Anthropol 149:71-83.

Tyrrell A. 2000. Skeletal non-metric traits and the assessment of inter-and intra-population diversity: past problems and future potential. In: Cox M, Mays S, editors. Human osteology in archaeology and forensic science. London: Greenwich Medical Media. p 289-306.

Ullrich CG, Binet EF, Sanecki MG, Kieffer SA. 1980. Quantitative assessment of the lumbar spinal canal by computed tomography. Radiol 134:137-143.

Verbiest H. 1977. Results of surgical treatment of idiopathic developmental stenosis of the lumbar vertebral canal. A review of twenty-seven years' experience. Joint Surg 59B:181-188.

Wade N. 2014. A troublesome inheritance: Gene, race, and human history. New York: Penguin Books.

Wagstaff K. 2004. Clustering with missing values: No imputation required. In: Classification, clustering, and data mining applications. Proceedings of the meeting of the international federation of classification societies, p. 649-658.

Walby S, Armstrong J, Strid S. 2014. Intersectionality: Multiple inequalities in social theory. Sociol 46(2):224-240.

Ward DE. 2005. The white welfare state: The racialization of U.S. welfare policy. Ann Arbor, MI: The University of Michigan Press.

Washburn SL, 1951. The new physical anthropology. Transactions of the New York Academy of Sciences, Series II 13:298-304.

Washburn SL. 1964. The study of race. In Montagu A, editor. The concept of race. London: The Free Press of Glencoe. p 242-260.

Watts R 2011. Non-specific indicators of stress and their association with age at death tin medieval York: using stature and vertebral neural canal size to examine the effects of stress occurring during different periods of development. Int J Osteoarchaeol 21:568-576.

Watts R. 2013. Lumbar vertebral canal size in adults and children: observations from a skeletal sample from London, England. J Comp Human Biol 64:120-128.

Watts R 2015. The long-term impact of developmental stress. Evidence from Later Medieval and Post-Medieval London (AD1117-1853), Am J Phys Anthropol 00:00-00.

Weber JL, Wong C. 1993. Mutation of human short tandem repeats. Hum Mol Genet *2:*1123–1128.

Weinberg RD, Crosswell CM. 1967. Eligibility for entry to the United States of America, 3rd edition. Dobbs Ferry, N.Y.: Oceana Publications.

Weiss KM, Lambert BW. 2011. When the time seems ripe: Eugenics, the annals, and the subtle persistence of typological thinking. Ann Hum Genet 75(3):334–343.

Wijsman EM, Neves WA. 1986. The use of nonmetric variation in estimating human population admixture: A test case with Brazilian blacks, whites, and mulattos. Am J Phys Anthropol 70:395-405.

Wilkie Al, Bartoy KM. 2000. A critical archaeology revisited. Curr Anthropol 41(5):747-777.

Wilkie LA 2004. Considering the future of African American archaeology. Hist Archaeol 38(1):109-123.

Wilson SE. 2015. Methods for clustering data with missing values. Mathematical Institute: Universiteit Leiden, Netherlands.

Wells C. 1964. Bones, bodies, and disease: Evidence of disease and abnormality in early man. New York: Praeger.

Williams DR, Rucker TD. 2000. Understanding and addressing racial disparities in health care. Health Care Financing Rev 21(4):75-89.

Williams DR, Mohammed SA, Leavell J, Collins C. 2010. Race, socioeconomic status, and health: Complexities, ongoing challenges, and research opportunities. Ann N.Y. Acad Sci 1186:69-101.

Wilson JB. 1965. The black codes of the South. University: University of Alabama Press.

Wilson JJ. 2014. Paradox and promise: Research on the role of recent advances in paleodemography and paleoepidemiology to the study of "health" in Precolumbian societies. Am J Phys Anthropol 155:268-280.

Winslow CEA, Wang PL. 1931. The relation between changes in nationality stock and increasing death rates in adult life. Am J Hygiene 14:79-88.

Wood JW, Milner GR, Harpending HC, Weiss KM, Cohen MN, Eisenberg LE, Hutchinson DL, Jankauskas R, Cesnys G, Česnys G, Katzenberg MA, Lukacs JR, McGrath JW, Roth EA, Ubelaker DH, Wilkinson RG. 1992. "The osteological paradox: problems of inferring prehistoric health from skeletal samples [and comments and reply]." Curr Anthropol 33:343-370.

Woods L. 1996. Frequency and chronological distribution of linear enamel hypoplasia in a North American colonial skeletal sample. Am J Phys Anthropol 100:247-259.

Wright LE, Yoder CJ. 2003. Recent progress in bioarchaeology: Approaches to the osteological paradox. J Archaeol Res 11:43-70.

Wyatt-Brown B. 1982. Southern honor: Ethics and behavior in the Old South. Oxford: Oxford University Press.

Żądzińska E, Lorkiewicz W, Kurek M, Borowska-Strugińska B. 2015. Accentuated lines in the enamel of primary incisors from skeletal remains: A contribution to the explanation of early childhood mortality in a Medieval population from Poland. Am J Phys Anthropol 157:402-410.

APPENDICES

APPENDIX I

Table A-1. Sample demographics. Sex is abbreviated as follows: 0 = Male; 1 = Female. Ancestry is abbreviated as follows: EA = European American; AA = African American.

Collection	ID	Sex	Age	Ancestry	Birth Year	Cohort	Birth Place
Bass	01-02D	0	96	EA	1905	Middle	USA
Bass	04-01D	0	90	EA	1909	Middle	USA
Bass	12-87D	0	82	EA	1905	Middle	USA
Bass	16-92D	0	86	EA	1905	Middle	USA
Bass	21-94D	0	89	EA	1905	Middle	USA
Bass	22-90D	0	78	EA	1912	Middle	USA
Bass	3-98D	0	88	EA	1909	Middle	USA
Bass	27-99D	0	88	EA	1911	Middle	USA
Bass	2-96D	0	87	EA	1908	Middle	USA
Bass	1-81D	0	72	EA	1908	Middle	USA
Bass	10-88D	0	49	EA	1938	Intermediate	USA
Bass	10-96D	0	67	EA	1929	Intermediate	USA
Bass	11-02D	0	76	EA	1925	Intermediate	USA
Bass	12-89D	0	63	EA	1926	Intermediate	USA
Bass	12-90D	0	52	EA	1937	Intermediate	USA
Bass	14-92D	0	56	EA	1935	Intermediate	USA
Bass	16-91D	0	46	EA	1944	Intermediate	USA
Bass	1-82D	0	55	EA	1927	Intermediate	USA
Bass	20-99D	0	55	EA	1943	Intermediate	USA
Bass	21-99D	0	63	EA	1935	Intermediate	USA
Bass	23-01D	0	80	EA	1921	Intermediate	USA
Bass	24-05D	0	59	EA	1945	Intermediate	USA
Bass	26-02D	0	63	EA	1938	Intermediate	USA
Bass	2-88D	0	61	EA	1927	Intermediate	USA
Bass	2-94D	0	72	EA	1921	Intermediate	USA
Bass	30-04D	0	59	EA	1945	Intermediate	USA
Bass	34-02D	0	58	EA	1944	Intermediate	USA
Bass	37-05D	0	62	EA	1942	Intermediate	USA
Bass	3-83D	0	63	EA	1920	Intermediate	USA
Bass	3-93D	0	56	EA	1935	Intermediate	USA
Bass	4-00D	0	57	EA	1943	Intermediate	USA
Bass	43-01D	0	59	EA	1942	Intermediate	USA
Bass	43-02D	0	66	EA	1936	Intermediate	USA
Bass	43-04D	0	72	EA	1932	Intermediate	USA
Bass	4-89D	0	51	EA	1937	Intermediate	USA
Bass	51-05D	0	65	EA	1940	Intermediate	USA
Bass	56-03D	0	70	EA	1932	Intermediate	USA
Bass	5-98D	0	67	EA	1931	Intermediate	USA
Bass	7-93D	0	64	EA	1928	Intermediate	USA
Bass	8-89D	0	66	EA	1922	Intermediate	USA
Bass	8-94D	0	63	EA	1930	Intermediate	USA
Bass	9-97D	0	56	EA	1940	Intermediate	USA
Bass	07-02D	0	59	EA	1942	Intermediate	USA

Table A-1. Continued.

Collection	ID	Sex	Age	Ancestry	Birth Year	Cohort	Birth Place
Bass	09-02D	0	63	EA	1938	Intermediate	USA
Bass	10-89D	0	50	EA	1938	Intermediate	USA
Bass	10-90D	0	59	EA	1930	Intermediate	USA
Bass	10-95D	0	56	EA	1939	Intermediate	USA
Bass	11-00D	0	55	EA	1945	Intermediate	USA
Bass	11-89D	0	53	EA	1935	Intermediate	USA
Bass	12-88D	0	47	EA	1940	Intermediate	USA
Bass	12-97D	0	69	EA	1928	Intermediate	USA
Bass	14-98D	0	61	EA	1936	Intermediate	USA
Bass	16-01D	0	61	EA	1939	Intermediate	USA
Bass	16-04D	0	85	EA	1917	Intermediate	USA
Bass	16-98D	0	58	EA	1940	Intermediate	USA
Bass	18-93D	0	78	EA	1915	Intermediate	USA
Bass	19-05D	0	71	EA	1933	Intermediate	USA
Bass	19-99D	0	83	EA	1916	Intermediate	USA
Bass	20-02D	0	65	EA	1937	Intermediate	USA
Bass	20-92D	0	71	EA	1921	Intermediate	USA
Bass	21-90D	0	68	EA	1921	Intermediate	USA
Bass	22-00D	0	57	EA	1942	Intermediate	USA
Bass	23-05D	0	61	EA	1943	Intermediate	USA
Bass	23-93D	0	76	EA	1916	Intermediate	USA
Bass	23-94D	0	67	EA	1927	Intermediate	USA
Bass	27-90D	0	54	EA	1936	Intermediate	USA
Bass	2-84D	0	65	EA	1919	Intermediate	USA
Bass	29-99D	0	61	EA	1937	Intermediate	USA
Bass	30-03D	0	71	EA	1932	Intermediate	USA
Bass	31-02D	0	73	EA	1929	Intermediate	USA
Bass	31-03D	0	88	EA	1915	Intermediate	USA
Bass	32-02D	0	64	EA	1938	Intermediate	USA
Bass	32-93D	0	73	EA	1920	Intermediate	USA
Bass	35-03D	0	61	EA	1941	Intermediate	USA
Bass	35-99D	0	70	EA	1929	Intermediate	USA
Bass	36-04D	0	83	EA	1921	Intermediate	USA
Bass	38-01D	0	70	EA	1931	Intermediate	USA
Bass	39-04D	0	60	EA	1944	Intermediate	USA
Bass	3-91D	0	68	EA	1923	Intermediate	USA
Bass	44-02D	0	60	EA	1942	Intermediate	USA
Bass	45-05D	0	83	EA	1921	Intermediate	USA
Bass	45-93D	0	73	EA	1920	Intermediate	USA
Bass	46-05D	0	60	EA	1944	Intermediate	USA
Bass	47-05D	0	65	EA	1939	Intermediate	USA
Bass	48-05D	0	61	EA	1943	Intermediate	USA
Bass	4-87D	0	55	EA	1931	Intermediate	USA

Table A-1 Continued.

Collection	ID	Sex	Age	Ancestry	Birth Year	Cohort	Birth Place
Bass	49-04D	0	64	EA	1939	Intermediate	USA
Bass	4-96D	0	55	EA	1940	Intermediate	USA
Bass	4-99D	0	57	EA	1941	Intermediate	USA
Bass	50-03D	0	62	EA	1941	Intermediate	USA
Bass	53-04D	0	83	EA	1921	Intermediate	USA
Bass	62-05D	0	63	EA	1942	Intermediate	USA
Bass	6-96D	0	67	EA	1929	Intermediate	USA
Bass	6-98D	0	56	EA	1941	Intermediate	USA
Bass	7-87D	0	57	EA	1929	Intermediate	USA
Bass	7-89D	0	50	EA	1938	Intermediate	USA
Bass	8-95D	0	56	EA	1939	Intermediate	USA
Bass	10-94D	0	67	EA	1926	Intermediate	USA
Bass	14-05D	0	63	EA	1941	Intermediate	USA
Bass	14-88D	0	55	EA	1932	Intermediate	USA
Bass	19-88D	0	46	EA	1941	Intermediate	USA
Bass	36-93D	0	55	EA	1937	Intermediate	USA
Bass	5-93D	0	53	EA	1939	Intermediate	USA
Bass	65-04D	0	82	EA	1922	Intermediate	USA
Bass	08-04D	0	57	EA	1946	Late	USA
Bass	12-98D	0	45	EA	1952	Late	USA
Bass	13-03D	0	48	EA	1954	Late	USA
Bass	14-93D	0	32	EA	1960	Late	USA
Bass	16-05D	0	43	EA	1961	Late	USA
Bass	17-91D	0	26	EA	1964	Late	USA
Bass	24-02D	0	52	EA	1950	Late	USA
Bass	24-99D	0	49	EA	1949	Late	USA
Bass	25-01D	0	48	EA	1952	Late	USA
Bass	28-03D	0	54	EA	1949	Late	USA
Bass	30-93D	0	46	EA	1946	Late	USA
Bass	31-00D	0	48	EA	1952	Late	USA
Bass	31-99D	0	45	EA	1953	Late	USA
Bass	38-04D	0	53	EA	1951	Late	USA
Bass	42-05D	0	42	EA	1962	Late	USA
Bass	58-05D	0	53	EA	1952	Late	USA
Bass	59-04D	0	48	EA	1956	Late	USA
Bass	8-87D	0	24	EA	1962	Late	USA
Bass	9-00D	0	43	EA	1956	Late	USA
Bass	08-05D	0	55	EA	1949	Late	USA
Bass	10-05D	0	46	EA	1958	Late	USA
Bass	1-00D	0	40	EA	1959	Late	USA
Bass	12-01D	0	50	EA	1950	Late	USA
Bass	13-91D	0	33	EA	1957	Late	USA
Bass	15-05D	0	53	EA	1951	Late	USA

Table A-1. Continued.

Collection	ID	Sex	Age	Ancestry	Birth Year	Cohort	Birth Place
Bass	19-92D	0	27	EA	1965	Late	USA
Bass	22-91D	0	43	EA	1948	Late	USA
Bass	3-00D	0	43	EA	1957	Late	USA
Bass	32-05D	0	54	EA	1951	Late	USA
Bass	37-03D	0	43	EA	1959	Late	USA
Bass	3-87D	0	36	EA	1951	Late	USA
Bass	3-90D	0	43	EA	1946	Late	USA
Bass	44-05D	0	51	EA	1953	Late	USA
Bass	52-04D	0	50	EA	1953	Late	USA
Bass	54-03D	0	54	EA	1949	Late	USA
Bass	57-03D	0	54	EA	1949	Late	USA
Bass	59-05D	0	53	EA	1951	Late	USA
Bass	64-05D	0	51	EA	1954	Late	USA
Bass	99-09D	0	53	EA	1955	Late	USA
Bass	61-04D	0	50	EA	1954	Late	USA
Bass	112-08D	1	97	EA	1911	Middle	USA
Bass	17-04D	1	91	EA	1912	Middle	USA
Bass	21-93D	1	82	EA	1910	Middle	USA
Bass	01-83D	1	79	EA	1903	Middle	USA
Bass	20-93D	1	81	EA	1911	Middle	USA
Bass	33-01D	1	93	EA	1908	Middle	USA
Bass	33-99D	1	94	EA	1905	Middle	USA
Bass	46-09D	1	95	EA	1913	Middle	USA
Bass	6-93D	1	80	EA	1912	Middle	USA
Bass	94-06D	1	93	EA	1913	Middle	USA
Bass	10-01D	1	75	EA	1925	Intermediate	USA
Bass	11-90D	1	67	EA	1922	Intermediate	USA
Bass	12-99D	1	72	EA	1926	Intermediate	USA
Bass	13-02D	1	69	EA	1932	Intermediate	USA
Bass	13-97D	1	71	EA	1925	Intermediate	USA
Bass	14-01D	1	78	EA	1922	Intermediate	USA
Bass	1-88D	1	71	EA	1916	Intermediate	USA
Bass	20-98D	1	63	EA	1935	Intermediate	USA
Bass	21-02D	1	85	EA	1916	Intermediate	USA
Bass	23-00D	1	81	EA	1919	Intermediate	USA
Bass	27-01D	1	73	EA	1927	Intermediate	USA
Bass	28-90D	1	45	EA	1945	Intermediate	USA
Bass	2-92D	1	62	EA	1929	Intermediate	USA
Bass	30-05D	1	69	EA	1935	Intermediate	USA
Bass	32-04D	1	73	EA	1930	Intermediate	USA
Bass	45-03D	1	73	EA	1930	Intermediate	USA
Bass	6-95D	1	69	EA	1925	Intermediate	USA
Bass	04-02D	1	60	EA	1941	Intermediate	USA

Table A-1. Continued.

Collection	ID	Sex	Age	Ancestry	Birth Year	Cohort	Birth Place
Bass	08-02D	1	79	EA	1922	Intermediate	USA
Bass	10-98D	1	69	EA	1929	Intermediate	USA
Bass	16-99D	1	82	EA	1916	Intermediate	USA
Bass	24-00D	1	73	EA	1927	Intermediate	USA
Bass	26-99D	1	74	EA	1925	Intermediate	USA
Bass	28-01D	1	61	EA	1940	Intermediate	USA
Bass	50-05D	1	68	EA	1936	Intermediate	USA
Bass	57-04D	1	81	EA	1923	Intermediate	USA
Bass	5-97D	1	67	EA	1929	Intermediate	USA
Bass	6-92D	1	62	EA	1929	Intermediate	USA
Bass	7-92D	1	64	EA	1927	Intermediate	USA
Bass	9-95D	1	65	EA	1930	Intermediate	USA
Bass	23-88D	1	59	EA	1929	Intermediate	USA
Bass	26-93D	1	62	EA	1929	Intermediate	USA
Bass	47-01D	1	56	EA	1945	Intermediate	USA
Bass	107-10D	1	52	EA	1958	Late	USA
Bass	11-04D	1	54	EA	1950	Late	USA
Bass	12-02D	1	49	EA	1952	Late	USA
Bass	27-05D	1	58	EA	1946	Late	USA
Bass	27-91D	1	38	EA	1953	Late	USA
Bass	30-09D	1	31	EA	1977	Late	USA
Bass	39-01D	1	30	EA	1964	Late	USA
Bass	40-10D	1	61	EA	1948	Late	USA
Bass	42-06D	1	56	EA	1950	Late	USA
Bass	44-10D	1	62	EA	1947	Late	USA
Bass	51-07D	1	44	EA	1962	Late	USA
Bass	55-07D	1	51	EA	1955	Late	USA
Bass	56-07D	1	57	EA	1949	Late	USA
Bass	61-05D	1	55	EA	1950	Late	USA
Bass	77-07D	1	36	EA	1971	Late	USA
Bass	78-06D	1	49	EA	1957	Late	USA
Bass	7-97D	1	32	EA	1964	Late	USA
Bass	81-08D	1	46	EA	1961	Late	USA
Bass	82-07D	1	31	EA	1975	Late	USA
Bass	85-11D	1	46	EA	1964	Late	USA
Bass	89-06D	1	50	EA	1956	Late	USA
Bass	100-06D	1	57	EA	1949	Late	USA
Bass	100-09D	1	50	EA	1959	Late	USA
Bass	10-07D	1	50	EA	1957	Late	USA
Bass	102-10D	1	62	EA	1948	Late	USA
Bass	113-10D	1	45	EA	1965	Late	USA
Bass	12-09D	1	58	EA	1950	Late	USA
Bass	15-06D	1	58	EA	1947	Late	USA

Table A-1. Continued.

Collection	ID	Sex	Age	Ancestry	Birth Year	Cohort	Birth Place
Bass	20-03D	1	44	EA	1959	Late	USA
Bass	31-05D	1	51	EA	1953	Late	USA
Bass	33-03D	1	51	EA	1952	Late	USA
Bass	43-08D	1	55	EA	1953	Late	USA
Bass	47-08D	1	58	EA	1950	Late	USA
Bass	68-06D	1	54	EA	1951	Late	USA
Bass	73-08D	1	60	EA	1948	Late	USA
Bass	73-10D	1	59	EA	1951	Late	USA
Bass	85-05D	1	46	EA	1959	Late	USA
Bass	30-12D	1	38	EA	1974	Late	USA
Bass	35-02D	1	55	EA	1946	Late	USA
Bass	64-10D	1	50	EA	1960	Late	USA
Bass	15-93D	0	90	AA	1903	Middle	USA
Bass	2-91D	0	81	AA	1909	Middle	USA
Bass	06-02D	0	77	AA	1924	Intermediate	USA
Bass	08-10D	0	71	AA	1938	Intermediate	USA
Bass	15-89D	0	56	AA	1933	Intermediate	USA
Bass	15-91D	0	60	AA	1930	Intermediate	USA
Bass	1-92D	0	55	AA	1936	Intermediate	USA
Bass	19-94D	0	54	AA	1940	Intermediate	USA
Bass	23-03D	0	68	AA	1934	Intermediate	USA
Bass	23-06D	0	70	AA	1935	Intermediate	USA
Bass	30-01D	0	64	AA	1936	Intermediate	USA
Bass	31-01D	0	77	AA	1924	Intermediate	USA
Bass	31-93D	0	68	AA	1925	Intermediate	USA
Bass	35-93D	0	61	AA	1931	Intermediate	USA
Bass	3-89D	0	49	AA	1939	Intermediate	USA
Bass	41-06D	0	71	AA	1934	Intermediate	USA
Bass	6-00D	0	71	AA	1928	Intermediate	USA
Bass	6-87D	0	69	AA	1916	Intermediate	USA
Bass	71-05D	0	72	AA	1932	Intermediate	USA
Bass	8-91D	0	70	AA	1921	Intermediate	USA
Bass	96-10D	0	79	AA	1931	Intermediate	USA
Bass	9-89D	0	43	AA	1945	Intermediate	USA
Bass	100-07D	0	59	AA	1948	Late	USA
Bass	11-98D	0	49	AA	1948	Late	USA
Bass	12-05D	0	56	AA	1948	Late	USA
Bass	15-90D	0	36	AA	1953	Late	USA
Bass	17-00D	0	35	AA	1965	Late	USA
Bass	17-09D	0	60	AA	1948	Late	USA
Bass	17-88D	0	20	AA	1968	Late	USA
Bass	18-90D	0	27	AA	1963	Late	USA

Table A-1. Continued.

Collection	ID	Sex	Age	Ancestry	Birth Year	Cohort	Birth Place
Bass	19-07D	0	53	AA	1953	Late	USA
Bass	21-92D	0	25	AA	1967	Late	USA
Bass	25-04D	0	40	AA	1963	Late	USA
Bass	40-04D	0	47	AA	1956	Late	USA
Bass	45-06D	0	43	AA	1962	Late	USA
Bass	46-03D	0	23	AA	1980	Late	USA
Bass	48-04D	0	46	AA	1957	Late	USA
Bass	4-95D	0	43	AA	1951	Late	USA
Bass	53-05D	0	43	AA	1961	Late	USA
Bass	54-06D	0	43	AA	1962	Late	USA
Bass	5-94D	0	46	AA	1947	Late	USA
Bass	74-07D	0	55	AA	1952	Late	USA
Bass	75-06D	0	47	AA	1958	Late	USA
Bass	79-10D	0	63	AA	1947	Late	USA
Bass	81-07D	0	49	AA	1957	Late	USA
Bass	8-99D	0	43	AA	1955	Late	USA
Bass	98-06D	0	47	AA	1959	Late	USA
Bass	18-05D	1	99	AA	1905	Middle	USA
Bass	05-01D	1	59	AA	1941	Intermediate	USA
Bass	117-09D	1	71	AA	1937	Intermediate	USA
Bass	1-96D	1	66	AA	1929	Intermediate	USA
Bass	20-11D	1	66	AA	1944	Intermediate	USA
Bass	36-06D	1	73	AA	1932	Intermediate	USA
Bass	03-11D	1	39	AA	1971	Late	USA
Bass	2-86D	1	39	AA	1947	Late	USA
Bass	62-06D	1	54	AA	1951	Late	USA
Bass	6-89D	1	40	AA	1949	Late	USA
Bass	78-07D	1	24	AA	1983	Late	USA
Hamann-Todd	HTH1095	0	47	EA	1877	Early	USA
Hamann-Todd	HTH1174	0	54	EA	1870	Early	USA
Hamann-Todd	HTH1290	0	45	EA	1880	Early	USA
Hamann-Todd	HTH1326	0	82	EA	1844	Early	Ohio
Hamann-Todd	HTH1436	0	78	EA	1848	Early	USA
Hamann-Todd	HTH1527	0	85	EA	1842	Early	Ohio
Hamann-Todd	HTH206	0	63	EA	1851	Early	New York
Hamann-Todd	HTH212	0	57	EA	1857	Early	Washington DC
Hamann-Todd	HTH239	0	61	EA	1853	Early	USA
Hamann-Todd	HTH2603	0	58	EA	1876	Early	California
Hamann-Todd	HTH28	0	53	EA	1858	Early	Ohio
Hamann-Todd	HTH359	0	40	EA	1852	Early	Ohio
Hamann-Todd	HTH364	0	45	EA	1869	Early	New York
Hamann-Todd	HTH374	0	45	EA	1870	Early	Ohio

Table A-1. Continued.

Collection	ID	Sex	Age	Ancestry	Birth Year	Cohort	Birth Place
Hamann-Todd	HTH389	0	38	EA	1877	Early	USA
Hamann-Todd	HTH390	0	55	EA	1867	Early	New York
Hamann-Todd	HTH499	0	55	EA	1862	Early	Tennessee
Hamann-Todd	HTH537	0	55	EA	1862	Early	Ohio
Hamann-Todd	HTH924	0	60	EA	1862	Early	New York
Hamann-Todd	HTH991	0	59	EA	1863	Early	Ohio
Hamann-Todd	HTH1029	0	50	EA	1873	Early	Missouri
Hamann-Todd	HTH1031	0	57	EA	1866	Early	USA
Hamann-Todd	HTH1076	0	44	EA	1879	Early	Ohio
Hamann-Todd	HTH1081	0	45	EA	1878	Early	Ohio
Hamann-Todd	HTH1111	0	59	EA	1865	Early	Ohio
Hamann-Todd	HTH1129	0	50	EA	1874	Early	USA
Hamann-Todd	HTH1143	0	70	EA	1854	Early	USA
Hamann-Todd	HTH1151	0	57	EA	1867	Early	Michigan
Hamann-Todd	HTH1193	0	60	EA	1864	Early	USA
Hamann-Todd	HTH1225	0	80	EA	1845	Early	USA
Hamann-Todd	HTH1234	0	45	EA	1865	Early	USA
Hamann-Todd	HTH1248	0	40	EA	1870	Early	USA
Hamann-Todd	HTH189	0	43	EA	1870	Early	USA
Hamann-Todd	HTH1958	0	74	EA	1856	Early	USA
Hamann-Todd	HTH1985	0	68	EA	1862	Early	USA
Hamann-Todd	HTH1989	0	75	EA	1855	Early	USA
Hamann-Todd	HTH2029	0	59	EA	1871	Early	New York
Hamann-Todd	HTH2257	0	68	EA	1864	Early	Ohio
Hamann-Todd	HTH244	0	38	EA	1874	Early	USA
Hamann-Todd	HTH2549	0	60	EA	1874	Early	Pennsylvania
Hamann-Todd	HTH2599	0	63	EA	1871	Early	Tennessee
Hamann-Todd	HTH26	0	40	EA	1871	Early	USA
Hamann-Todd	HTH2663	0	59	EA	1875	Early	Ohio
Hamann-Todd	HTH286	0	45	EA	1847	Early	New York
Hamann-Todd	HTH301	0	45	EA	1881	Early	Ohio
Hamann-Todd	HTH309	0	55	EA	1849	Early	New York
Hamann-Todd	HTH318	0	25	EA	1868	Early	West Virginia
Hamann-Todd	HTH328	0	38	EA	1877	Early	Pennsylvania
Hamann-Todd	HTH344	0	50	EA	1866	Early	USA
Hamann-Todd	HTH393	0	56	EA	1860	Early	Wisconsin
Hamann-Todd	HTH550	0	39	EA	1878	Early	USA
Hamann-Todd	HTH686	0	65	EA	1854	Early	New York
Hamann-Todd	HTH841	0	50	EA	1870	Early	USA
Hamann-Todd	HTH910	0	47	EA	1874	Early	Pennsylvania
Hamann-Todd	HTH942	0	45	EA	1877	Early	Ohio
Hamann-Todd	HTH951	0	57	EA	1865	Early	Ohio

Table A-1. Continued.

Collection	ID	Sex	Age	Ancestry	Birth Year	Cohort	Birth Place
Hamann-Todd	HTH661	0	43	EA	1876	Early	Illinois
Hamann-Todd	HTH676	0	54	EA	1879	Early	New York
Hamann-Todd	HTH836	0	50	EA	1870	Early	Illinois
Hamann-Todd	HTH1331	0	27	EA	1899	Middle	USA
Hamann-Todd	HTH2763	0	35	EA	1900	Middle	Michigan
Hamann-Todd	HTH1769	0	24	EA	1904	Middle	Ohio
Hamann-Todd	HTH2318	0	46	EA	1886	Middle	Pennsylvania
Hamann-Todd	HTH2474	0	39	EA	1894	Middle	Massachusetts
Hamann-Todd	HTH2618	0	48	EA	1886	Middle	New York
Hamann-Todd	HTH2842	0	44	EA	1891	Middle	Ohio
Hamann-Todd	HTH469	0	28	EA	1888	Middle	USA
Hamann-Todd	HTH823	0	33	EA	1887	Middle	Pennsylvania
Hamann-Todd	HTH826	0	38	EA	1882	Middle	USA
Hamann-Todd	HTH950	0	35	EA	1887	Middle	Massachusetts
Hamann-Todd	HTH1256	1	55	EA	1870	Early	USA
Hamann-Todd	HTH2025	1	49	EA	1881	Early	Massachusetts
Hamann-Todd	HTH355	1	49	EA	1866	Early	Ohio
Hamann-Todd	HTH421	1	38	EA	1878	Early	New York
Hamann-Todd	HTH552	1	36	EA	1881	Early	West Virginia
Hamann-Todd	HTH1191	1	78	EA	1846	Early	USA
Hamann-Todd	HTH1647	1	63	EA	1865	Early	USA
Hamann-Todd	HTH1760	1	70	EA	1858	Early	New York
Hamann-Todd	HTH53	1	35	EA	1876	Early	Pennsylvania
Hamann-Todd	HTH1238	1	19	EA	1906	Middle	West Virginia
Hamann-Todd	HTH1900	1	27	EA	1902	Middle	Pennsylvania
Hamann-Todd	HTH886	1	32	EA	1889	Middle	Ohio
Hamann-Todd	HTH929	1	31	EA	1891	Middle	Pennsylvania
Hamann-Todd	HTH1235	0	45	AA	1880	Early	USA
Hamann-Todd	HTH182	0	32	AA	1868	Early	North Carolina
Hamann-Todd	HTH327	0	35	AA	1856	Early	New Jersey
Hamann-Todd	HTH416	0	30	AA	1878	Early	Rhode Island
Hamann-Todd	HTH451	0	35	AA	1881	Early	South Carolina
Hamann-Todd	HTH540	0	45	AA	1872	Early	USA
Hamann-Todd	HTH558	0	41	AA	1876	Early	Alabama
Hamann-Todd	HTH574	0	46	AA	1871	Early	New York
Hamann-Todd	HTH682	0	39	AA	1880	Early	Alabama
Hamann-Todd	HTH718	0	39	AA	1880	Early	Tennessee
Hamann-Todd	HTH777	0	45	AA	1875	Early	North Carolina
Hamann-Todd	HTH811	0	40	AA	1880	Early	Maryland
Hamann-Todd	HTH831	0	47	AA	1873	Early	Columbus
Hamann-Todd	HTH835	0	41	AA	1879	Early	Mississippi
Hamann-Todd	HTH880	0	40	AA	1881	Early	USA

Table A-1. Continued.

Collection	ID	Sex	Age	Ancestry	Birth Year	Cohort	Birth Place
Hamann-Todd	HTH1097	0	18	AA	1906	Middle	Maryland
Hamann-Todd	HTH1134	0	21	AA	1903	Middle	Georgia
Hamann-Todd	HTH1763	0	23	AA	1905	Middle	South Carolina
Hamann-Todd	HTH190	0	26	AA	1887	Middle	Virginia
Hamann-Todd	HTH1974	0	18	AA	1912	Middle	Georgia
Hamann-Todd	HTH2341	0	22	AA	1911	Middle	USA
Hamann-Todd	HTH2776	0	26	AA	1909	Middle	South Carolina
Hamann-Todd	HTH290	0	33	AA	1882	Middle	Virginia
Hamann-Todd	HTH523	0	24	AA	1893	Middle	Alabama
Hamann-Todd	HTH563	0	19	AA	1888	Middle	Washington DC
Hamann-Todd	HTH588	0	19	AA	1899	Middle	Alabama
Hamann-Todd	HTH695	0	18	AA	1901	Middle	West Virginia
Hamann-Todd	HTH738	0	25	AA	1894	Middle	South Carolina
Hamann-Todd	HTH744	0	22	AA	1897	Middle	Arkansas
Hamann-Todd	HTH854	0	19	AA	1902	Middle	USA
Hamann-Todd	HTH1022	1	46	AA	1877	Early	Tennessee
Hamann-Todd	HTH1070	1	42	AA	1881	Early	Ohio
Hamann-Todd	HTH1275	1	50	AA	1875	Early	Washington DC
Hamann-Todd	HTH1321	1	52	AA	1874	Early	Alabama
Hamann-Todd	HTH1397	1	33	AA	1873	Early	Mississippi
Hamann-Todd	HTH1781	1	48	AA	1880	Early	South Carolina
Hamann-Todd	HTH1860	1	55	AA	1874	Early	Alabama
Hamann-Todd	HTH2278	1	53	AA	1879	Early	USA
Hamann-Todd	HTH2380	1	58	AA	1875	Early	Virginia
Hamann-Todd	HTH2404	1	60	AA	1873	Early	West Virginia
Hamann-Todd	HTH2517	1	55	AA	1879	Early	West Virginia
Hamann-Todd	HTH2520	1	58	AA	1876	Early	Kentucky
Hamann-Todd	HTH2706	1	54	AA	1881	Early	Alabama
Hamann-Todd	HTH685	1	45	AA	1874	Early	Virginia
Hamann-Todd	HTH1161	1	24	AA	1900	Middle	Mississippi
Hamann-Todd	HTH1208	1	23	AA	1901	Middle	USA
Hamann-Todd	HTH1294	1	28	AA	1897	Middle	Ohio
Hamann-Todd	HTH1418	1	21	AA	1905	Middle	Michigan
Hamann-Todd	HTH1558	1	24	AA	1903	Middle	South Carolina
Hamann-Todd	HTH1949	1	19	AA	1911	Middle	South Carolina
Hamann-Todd	HTH2065	1	19	AA	1912	Middle	Georgia
Hamann-Todd	HTH2093	1	24	AA	1907	Middle	Florida
Hamann-Todd	HTH226	1	29	AA	1885	Middle	Pennsylvania
Hamann-Todd	HTH2838	1	22	AA	1913	Middle	Georgia
Hamann-Todd	HTH545	1	25	AA	1892	Middle	Alabama
Hamann-Todd	HTH576	1	16	AA	1902	Middle	Alabama
Hamann-Todd	HTH704	1	29	AA	1890	Middle	Michigan

Table A-1. Continued.

Collection	ID	Sex	Age	Ancestry	Birth Year	Cohort	Birth Place
Hamann-Todd	HTH933	1	28	AA	1894	Middle	Kentucky
Hamann-Todd	HTH954	1	24	AA	1898	Middle	South Carolina
Terry	5	0	67	EA	1869	Early	Illinois
Terry	109	0	46	EA	1876	Early	Texas
Terry	259	0	52	EA	1873	Early	Virginia
Terry	262	0	72	EA	1853	Early	Ohio
Terry	268	0	62	EA	1863	Early	Missouri
Terry	314	0	65	EA	1860	Early	Alabama
Terry	332	0	50	EA	1876	Early	Ohio
Terry	361	0	67	EA	1859	Early	Missouri
Terry	413	0	55	EA	1872	Early	Michigan
Terry	195	0	49	EA	1875	Early	Missouri
Terry	207	0	45	EA	1879	Early	Indiana
Terry	258	0	64	EA	1861	Early	Illinois
Terry	274	0	45	EA	1880	Early	Missouri
Terry	307	0	72	EA	1854	Early	New York
Terry	315	0	48	EA	1877	Early	Wisconsin
Terry	317	0	84	EA	1841	Early	Missouri
Terry	363	0	65	EA	1861	Early	Missouri
Terry	396	0	75	EA	1852	Early	Ohio
Terry	501	0	77	EA	1851	Early	Illinois
Terry	521	0	67	EA	1861	Early	New York
Terry	772	0	56	EA	1874	Early	Texas
Terry	282	0	52	EA	1872	Early	Pennsylvania
Terry	758	0	73	EA	1857	Early	Illinois
Terry	599	0	43	EA	1886	Middle	Illinois
Terry	747	0	45	EA	1884	Middle	Iowa
Terry	849	0	47	EA	1884	Middle	Missouri
Terry	895	0	47	EA	1884	Middle	Missouri
Terry	1126	0	38	EA	1895	Middle	Kentucky
Terry	1194	0	48	EA	1885	Middle	Missouri
Terry	1226	0	48	EA	1886	Middle	Alabama
Terry	546	0	40	EA	1888	Middle	Kentucky
Terry	755	0	36	EA	1893	Middle	New Jersey
Terry	756	0	47	EA	1882	Middle	New York
Terry	828	0	48	EA	1883	Middle	Missouri
Terry	846	0	48	EA	1883	Middle	New York
Terry	1089	0	43	EA	1889	Middle	Texas
Terry	1318	0	43	EA	1891	Middle	Missouri
Terry	1598	0	33	EA	1928	Intermediate	Missouri
Terry	187R	0	29	EA	1917	Intermediate	Missouri
Terry	311R	0	27	EA	1923	Intermediate	Missouri

Table A-1. Continued.

Collection	ID	Sex	Age	Ancestry	Birth Year	Cohort	Birth Place
Terry	373	1	55	EA	1871	Early	Missouri
Terry	451	1	72	EA	1854	Early	Missouri
Terry	481	1	67	EA	1859	Early	Missouri
Terry	791	1	66	EA	1864	Early	Indiana
Terry	854	1	72	EA	1859	Early	Tennessee
Terry	934	1	62	EA	1875	Early	Kansas
Terry	1058	1	68	EA	1864	Early	Kentucky
Terry	1201	1	65	EA	1868	Early	Ohio
Terry	1405	1	67	EA	1868	Early	Indiana
Terry	1432	1	84	EA	1851	Early	Illinois
Terry	1560	1	63	EA	1878	Early	Missouri
Terry	1565	1	85	EA	1873	Early	Illinois
Terry	1630	1	79	EA	1880	Early	Kansas
Terry	217	1	66	EA	1878	Early	Illinois
Terry	218	1	56	EA	1868	Early	Pennsylvania
Terry	686	1	78	EA	1852	Early	Missouri
Terry	736	1	57	EA	1872	Early	Missouri
Terry	925	1	71	EA	1860	Early	Missouri
Terry	1069	1	70	EA	1862	Early	Missouri
Terry	1103	1	74	EA	1858	Early	Missouri
Terry	1389	1	84	EA	1851	Early	Missouri
Terry	1435	1	66	EA	1869	Early	Connecticut
Terry	1463	1	65	EA	1871	Early	Illinois
Terry	1542	1	67	EA	1874	Early	Missouri
Terry	1586	1	84	EA	1875	Early	Missouri
Terry	611	1	65	EA	1864	Early	Missouri
Terry	680	1	30	EA	1898	Middle	Kentucky
Terry	847	1	39	EA	1892	Middle	Indiana
Terry	1199	1	45	EA	1888	Middle	Arkansas
Terry	1476	1	51	EA	1886	Middle	Illinois
Terry	1562	1	46	EA	1912	Middle	Missouri
Terry	1583	1	70	EA	1889	Middle	Missouri
Terry	1625	1	60	EA	1899	Middle	Missouri
Terry	983	1	30	EA	1902	Middle	Illinois
Terry	1568	1	62	EA	1896	Middle	USA
Terry	1576	1	69	EA	1889	Middle	Missouri
Terry	1592	1	54	EA	1906	Middle	Kansas
Terry	1120	1	24	EA	1909	Middle	Missouri
Terry	1571	1	55	EA	1903	Middle	Missouri
Terry	1587	1	76	EA	1885	Middle	California
Terry	1626	1	66	EA	1893	Middle	Missouri
Terry	1563	1	29	EA	1933	Intermediate	Washington DC

Table A-1. Continued.

Collection	ID	Sex	Age	Ancestry	Birth Year	Cohort	Birth Place
Terry	1582	1	46	EA	1915	Intermediate	Missouri
Terry	1566	1	33	EA	1926	Intermediate	Kentucky
Terry	1572	1	45	EA	1914	Intermediate	Missouri
Terry	119	0	73	AA	1863	Early	Mississippi
Terry	130	0	58	AA	1878	Early	Alabama
Terry	182	0	43	AA	1880	Early	Arkansas
Terry	226	0	47	AA	1877	Early	Alabama
Terry	309	0	44	AA	1881	Early	Alabama
Terry	336	0	48	AA	1878	Early	Missouri
Terry	340	0	67	AA	1859	Early	Kentucky
Terry	345	0	58	AA	1867	Early	Arkansas
Terry	368	0	56	AA	1871	Early	Alabama
Terry	403	0	52	AA	1875	Early	Missouri
Terry	443	0	46	AA	1880	Early	Mississippi
Terry	445	0	66	AA	1860	Early	Missouri
Terry	473	0	57	AA	1869	Early	Mississippi
Terry	500	0	68	AA	1860	Early	Missouri
Terry	507	0	57	AA	1870	Early	Louisiana
Terry	515	0	62	AA	1865	Early	South Carolina
Terry	524	0	49	AA	1877	Early	Mississippi
Terry	539	0	59	AA	1869	Early	Missouri
Terry	556	0	60	AA	1868	Early	Virginia
Terry	609	0	75	AA	1854	Early	Pennsylvania
Terry	638	0	55	AA	1881	Early	Louisiana
Terry	646	0	50	AA	1879	Early	North Carolina
Terry	725	0	57	AA	1872	Early	Alabama
Terry	742	0	79	AA	1850	Early	Missouri
Terry	788	0	65	AA	1865	Early	Illinois
Terry	790	0	77	AA	1853	Early	Missouri
Terry	801	0	65	AA	1866	Early	Kentucky
Terry	829	0	58	AA	1873	Early	Virginia
Terry	890	0	60	AA	1876	Early	Tennessee
Terry	941	0	59	AA	1872	Early	Oklahoma
Terry	980	0	63	AA	1869	Early	Mississippi
Terry	84	0	20	AA	1890	Middle	Alabama
Terry	90	0	18	AA	1903	Middle	Texas
Terry	128	0	17	AA	1899	Middle	Missouri
Terry	138	0	49	AA	1887	Middle	Illinois
Terry	204	0	24	AA	1900	Middle	Alabama
Terry	222	0	20	AA	1904	Middle	Tennessee
Terry	251	0	33	AA	1892	Middle	Texas
Terry	331	0	27	AA	1898	Middle	Louisiana

Table A-1. Continued.

Collection	ID	Sex	Age	Ancestry	Birth Year	Cohort	Birth Place
Terry	385	0	22	AA	1903	Middle	Mississippi
Terry	401	0	43	AA	1884	Middle	West Virginia
Terry	458	0	41	AA	1885	Middle	Missouri
Terry	465	0	23	AA	1904	Middle	Louisiana
Terry	466	0	31	AA	1895	Middle	Arkansas
Terry	471	0	38	AA	1888	Middle	Louisiana
Terry	492	0	31	AA	1895	Middle	Kansas
Terry	504	0	40	AA	1887	Middle	Arkansas
Terry	518	0	41	AA	1886	Middle	North Carolina
Terry	540	0	40	AA	1888	Middle	Missouri
Terry	578	0	32	AA	1897	Middle	Kentucky
Terry	579	0	17	AA	1912	Middle	Missouri
Terry	581	0	30	AA	1898	Middle	Mississippi
Terry	592	0	25	AA	1908	Middle	Alabama
Terry	960	0	17	AA	1914	Intermediate	Alabama
Terry	1466	0	22	AA	1914	Intermediate	Texas
Terry	1503	0	22	AA	1915	Intermediate	Illinois
Terry	1539	0	23	AA	1918	Intermediate	Arkansas
Terry	1573	0	33	AA	1928	Intermediate	Missouri
Terry	85	1	66	AA	1866	Early	Missouri
Terry	114	1	43	AA	1879	Early	Missouri
Terry	134	1	53	AA	1867	Early	Virginia
Terry	154	1	52	AA	1868	Early	Missouri
Terry	159	1	70	AA	1858	Early	North Carolina
Terry	256	1	60	AA	1865	Early	Tennessee
Terry	272	1	49	AA	1875	Early	Texas
Terry	286	1	50	AA	1874	Early	Tennessee
Terry	484	1	86	AA	1840	Early	Missouri
Terry	491	1	73	AA	1854	Early	Kentucky
Terry	528	1	64	AA	1863	Early	Missouri
Terry	530	1	57	AA	1870	Early	Texas
Terry	532	1	71	AA	1857	Early	Virginia
Terry	535	1	95	AA	1833	Early	Mississippi
Terry	538	1	65	AA	1863	Early	Louisiana
Terry	559	1	59	AA	1868	Early	Missouri
Terry	587	1	61	AA	1868	Early	Tennessee
Terry	640	1	70	AA	1860	Early	Tennessee
Terry	653	1	62	AA	1865	Early	Tennessee
Terry	661	1	75	AA	1853	Early	Kentucky
Terry	699	1	70	AA	1858	Early	Mississippi
Terry	761	1	80	AA	1850	Early	Missouri
Terry	769	1	70	AA	1860	Early	Tennessee

Table A-1. Continued.

Collection	ID	Sex	Age	Ancestry	Birth Year	Cohort	Birth Place
Terry	775	1	101	AA	1828	Early	Mississippi
Terry	819	1	65	AA	1865	Early	Missouri
Terry	894	1	75	AA	1862	Early	Tennessee
Terry	957	1	56	AA	1875	Early	Georgia
Terry	961	1	63	AA	1868	Early	Tennessee
Terry	1000	1	59	AA	1878	Early	Mississippi
Terry	1063	1	54	AA	1878	Early	Alabama
Terry	1071	1	70	AA	1862	Early	Kentucky
Terry	1078	1	80	AA	1852	Early	Tennessee
Terry	1104	1	85	AA	1847	Early	West Virginia
Terry	1135	1	62	AA	1871	Early	Tennessee
Terry	1145	1	56	AA	1877	Early	Missouri
Terry	1160	1	79	AA	1854	Early	Kentucky
Terry	1227	1	75	AA	1859	Early	Kentucky
Terry	1290	1	59	AA	1878	Early	Illinois
Terry	1314	1	61	AA	1873	Early	Kentucky
Terry	1329	1	73	AA	1861	Early	Kentucky
Terry	1338	1	84	AA	1850	Early	Missouri
Terry	1343	1	64	AA	1870	Early	Arkansas
Terry	1358	1	65	AA	1870	Early	Missouri
Terry	1403	1	57	AA	1880	Early	Tennessee
Terry	1406	1	63	AA	1872	Early	Mississippi
Terry	1410	1	56	AA	1879	Early	Mississippi
Terry	1416	1	70	AA	1865	Early	Arkansas
Terry	1419	1	54	AA	1881	Early	Missouri
Terry	1461	1	80	AA	1856	Early	Mississippi
Terry	1483	1	95	AA	1841	Early	Arkansas
Terry	1494	1	81	AA	1855	Early	Alabama
Terry	1504	1	58	AA	1879	Early	Missouri
Terry	1524	1	64	AA	1877	Early	Mississippi
Terry	1526	1	69	AA	1872	Early	Missouri
Terry	1536	1	71	AA	1870	Early	Mississippi
Terry	1585	1	83	AA	1876	Early	Missouri
Terry	95	1	35	AA	1886	Middle	Louisiana
Terry	255	1	22	AA	1902	Middle	Mississippi
Terry	280	1	24	AA	1901	Middle	Arkansas
Terry	304	1	20	AA	1905	Middle	Tennessee
Terry	323	1	23	AA	1903	Middle	Missouri
Terry	330	1	38	AA	1887	Middle	Mississippi
Terry	472	1	35	AA	1891	Middle	Virginia
Terry	511	1	34	AA	1894	Middle	Tennessee
Terry	529	1	37	AA	1889	Middle	Georgia

Table A-1. Continued.

Collection	ID	Sex	Age	Ancestry	Birth Year	Cohort	Birth Place
Terry	561	1	22	AA	1907	Middle	Louisiana
Terry	562	1	17	AA	1912	Middle	Mississippi
Terry	568	1	27	AA	1902	Middle	Arkansas
Terry	632	1	27	AA	1902	Middle	Arkansas
Terry	655	1	35	AA	1892	Middle	Tennessee
Terry	738	1	45	AA	1884	Middle	Mississippi
Terry	840	1	32	AA	1904	Middle	Missouri
Terry	844	1	26	AA	1905	Middle	Kentucky
Terry	886	1	23	AA	1908	Middle	Tennessee
Terry	887	1	34	AA	1893	Middle	Missouri
Terry	891	1	39	AA	1897	Middle	Arkansas
Terry	906	1	22	AA	1909	Middle	Missouri
Terry	913	1	27	AA	1904	Middle	Mississippi
Terry	921	1	38	AA	1893	Middle	Mississippi
Terry	926	1	23	AA	1908	Middle	Mississippi
Terry	949	1	24	AA	1907	Middle	Georgia
Terry	952	1	41	AA	1890	Middle	Arkansas
Terry	959	1	46	AA	1885	Middle	Illinois
Terry	978	1	46	AA	1886	Middle	Louisiana
Terry	994	1	30	AA	1902	Middle	Colorado
Terry	1004	1	42	AA	1890	Middle	Arizona
Terry	1006	1	27	AA	1905	Middle	Tennessee
Terry	1015	1	41	AA	1891	Middle	Mississippi
Terry	1032	1	21	AA	1911	Middle	Arkansas
Terry	1034	1	48	AA	1889	Middle	Tennessee
Terry	1064	1	33	AA	1899	Middle	Ohio
Terry	1092	1	36	AA	1896	Middle	Missouri
Terry	1105	1	22	AA	1910	Middle	Louisiana
Terry	1122	1	28	AA	1905	Middle	Arkansas
Terry	1129	1	50	AA	1883	Middle	Alabama
Terry	1143	1	41	AA	1892	Middle	Louisiana
Terry	1150	1	39	AA	1894	Middle	Arkansas
Terry	1163	1	41	AA	1896	Middle	Tennessee
Terry	1164	1	27	AA	1906	Middle	Kentucky
Terry	1173	1	38	AA	1895	Middle	Tennessee
Terry	723	1	22	AA	1914	Intermediate	Mississippi
Terry	822	1	16	AA	1914	Intermediate	Arkansas
Terry	1507	1	23	AA	1914	Intermediate	Mississippi
Terry	1544	1	23	AA	1918	Intermediate	Tennessee
Terry	1551	1	25	AA	1916	Intermediate	Tennessee
Terry	1589	1	47	AA	1915	Intermediate	Missouri
Terry	1590	1	38	AA	1923	Intermediate	Alabama
Terry	1600	1	26	AA	1933	Intermediate	Mississippi

Table A-2. Linear enamel hypoplasia Chi-squared tests for sexual dimorphism. Teeth are abbreviated as follows: UCI = Maxillary central incisor; LC = Mandibular canine. X^2 = chi-squared; df = degrees of freedom.

Ancestry	Tooth	N	X^2	df	*p*-value
European American	UCI	22	2.794	1	0.273
European American	LC	52	0.754	1	0.668
African American	UCI	41	2.435	1	0.209
African American	LC	110	0.153	1	1.000

Table A-3. Shapiro-Wilk's tests of normality prior to sexual dimorphism tests. Lumbar vertebral (L1-L5) canal diameters from two dimensions: AP = Anteroposterior diameter; TR = Transverse diameter.

VNC Diameter	Ancestry	Sex	N	*p*-value
L1AP	European American	Male	243	0.885
	European American	Female	118	0.766
	African American	Male	130	**0.003**
	African American	Female	135	0.220
L1TR	European American	Male	247	0.130
	European American	Female	127	0.253
	African American	Male	130	0.332
	African American	Female	138	**0.022**
L2AP	European American	Male	243	0.933
	European American	Female	128	0.637
	African American	Male	126	**0.017**
	African American	Female	136	0.660
L2TR	European American	Male	251	**0.005**
	European American	Female	133	0.260
	African American	Male	126	0.053
	African American	Female	140	0.136
L3AP	European American	Male	234	0.161
	European American	Female	131	0.491
	African American	Male	129	0.606
	African American	Female	136	0.546
L3TR	European American	Male	241	0.238
	European American	Female	137	0.770
	African American	Male	130	0.401
	African American	Female	144	0.156
L4AP	European American	Male	226	0.060
	European American	Female	128	0.400
	African American	Male	125	0.500
	African American	Female	129	0.082
L4TR	European American	Male	228	**0.002**
	European American	Female	130	0.813
	African American	Male	117	0.345
	African American	Female	126	**0.014**
L5AP	European American	Male	196	0.217
	European American	Female	114	0.330
	African American	Male	108	**0.020**
	African American	Female	93	0.532
L5TR	European American	Male	173	0.904
	European American	Female	104	0.679
	African American	Male	91	0.146
	African American	Female	92	0.114

Table A-4. Mann-Whitney U tests results for sexual dimorphism among European Americans. Lumbar vertebral (L1-L5) canal diameters are taken from two dimensions: AP = Anteroposterior diameter; TR = Transverse diameter.

VNC Diameter	N	*p*-value
L1AP	361	0.438
L1TR	374	**<0.001**
L2AP	371	0.526
L2TR	384	**<0.001**
L3AP	365	0.104
L3TR	378	**<0.001**
L4AP	354	0.881
L4TR	358	**<0.001**
L5AP	310	**0.039**
L5TR	277	**<0.001**

Table A-5. Mann-Whitney U tests results for sexual dimorphism among African Americans. Lumbar vertebral (L1-L5) canal diameters are taken from two dimensions: AP = Anteroposterior diameter; TR = Transverse diameter.

VNC Diameter	N	*p*-value
L1AP	265	0.986
L1TR	268	**<0.001**
L2AP	262	**0.018**
L2TR	266	**<0.001**
L3AP	265	**0.025**
L3TR	274	**<0.001**
L4AP	254	0.336
L4TR	243	**0.001**
L5AP	201	0.383
L5TR	183	0.145

Table A-6. Shapiro-Wilk's tests of normality for males in the early cohort. Lumbar vertebral (L1-L5) canal diameters are taken from two dimensions: AP = Anteroposterior diameter; TR = Transverse diameter.

VNC Diameter	Race	N	*p*-value
L1AP	Group two	26	0.911
	Group three	46	0.240
	Group four	4	0.542
	Black	45	0.182
L1TR	Group two	29	0.576
	Group three	47	0.457
	Group four	4	0.251
	Black	45	0.737
L2AP	Group two	26	0.823
	Group three	44	0.512
	Group four	5	0.914
	Black	43	0.226
L2TR	Group two	29	0.305
	Group three	47	0.393
	Group four	5	**0.005**
	Black	44	0.451
L3AP	Group two	26	0.203
	Group three	41	0.738
	Group four	4	0.318
	Black	44	0.308
L3TR	Group two	26	**0.047**
	Group three	45	0.320
	Group four	4	0.796
	Black	44	0.857
L4AP	Group two	21	0.978
	Group three	37	0.344
	Group four	5	0.623
	Black	39	0.085
L4TR	Group two	21	**0.002**
	Group three	42	**0.046**
	Group four	5	0.602
	Black	34	0.301
L5AP	Group two	21	0.154
	Group three	32	0.648
	Group four	3	0.644
	Black	30	0.982
L5TR	Group two	17	**0.015**
	Group three	23	0.574
	Group four	3	0.725
	Black	23	0.529

Table A-7. Shapiro-Wilk's tests of normality for males in the middle cohort. Lumbar vertebral (L1-L5) canal diameters are taken from two dimensions: AP = Anteroposterior diameter; TR = Transverse diameter.

VNC Diameter	Race	N	*p*-value
L1AP	Group two	16	0.360
	Group three	17	0.403
	Black	39	**0.004**
L1TR	Group two	15	0.398
	Group three	17	0.051
	Black	39	0.561
L2AP	Group two	16	0.858
	Group three	18	0.369
	Black	38	**0.006**
L2TR	Group two	16	0.495
	Group three	18	**0.035**
	Black	37	0.116
L3AP	Group two	14	0.534
	Group three	18	0.163
	Black	37	0.775
L3TR	Group two	14	0.116
	Group three	18	0.917
	Black	38	0.485
L4AP	Group two	14	0.763
	Group three	16	0.759
	Black	36	0.619
L4TR	Group two	14	0.126
	Group three	13	0.913
	Black	35	0.912
L5AP	Group two	14	0.755
	Group three	10	0.485
	Black	34	0.090
L5TR	Group two	6	0.600
	Group three	8	0.835
	Black	29	0.330

Table A-8. Shapiro-Wilk's tests of normality for males in the intermediate cohort. Lumbar vertebral (L1-L5) canal diameters are taken from two dimensions: AP = Anteroposterior diameter; TR = Transverse diameter.

VNC Diameter	Race	N	*p*-value
L1AP	Group two	33	0.448
	Group three	53	0.948
	Group four	7	0.283
	Black	23	0.099
L1TR	Group two	33	0.918
	Group three	53	0.426
	Group four	8	0.570
	Black	23	0.813
L2AP	Group two	34	0.737
	Group three	52	0.976
	Group four	8	0.502
	Black	22	0.377
L2TR	Group two	34	0.194
	Group three	54	0.133
	Group four	8	0.853
	Black	22	0.743
L3AP	Group two	33	0.966
	Group three	52	**0.029**
	Group four	8	0.454
	Black	24	0.815
L3TR	Group two	34	0.593
	Group three	53	0.189
	Group four	8	0.797
	Black	24	0.544
L4AP	Group two	34	0.195
	Group three	50	0.469
	Group four	8	0.915
	Black	25	0.453
L4TR	Group two	33	**0.030**
	Group three	51	0.999
	Group four	8	0.142
	Black	23	0.890
L5AP	Group two	29	0.467
	Group three	46	0.497
	Group four	6	0.430
	Black	22	**0.002**
L5TR	Group two	26	0.490
	Group three	46	0.248
	Group four	6	0.079
	Black	18	0.450

Table A-9. Shapiro-Wilk's tests of normality for males in the late cohort. Lumbar vertebral (L1-L5) canal diameters are taken from two dimensions: AP = Anteroposterior diameter; TR = Transverse diameter.

VNC Diameter	Race	N	*p*-value
L1AP	Group two	19	0.765
	Group three	20	0.412
	Black	23	0.800
L1TR	Group two	19	0.718
	Group three	20	0.807
	Black	23	0.886
L2AP	Group two	19	0.446
	Group three	19	0.642
	Black	23	0.364
L2TR	Group two	19	0.292
	Group three	19	0.701
	Black	23	0.713
L3AP	Group two	19	0.625
	Group three	18	0.374
	Black	24	0.812
L3TR	Group two	19	0.869
	Group three	19	0.996
	Black	24	0.469
L4AP	Group two	19	0.148
	Group three	20	0.513
	Black	25	0.596
L4TR	Group two	19	0.267
	Group three	20	0.661
	Black	25	0.666
L5AP	Group two	18	0.312
	Group three	17	0.621
	Black	22	0.332
L5TR	Group two	18	0.152
	Group three	19	0.778
	Black	21	0.274

Table A-10. Shapiro-Wilk's tests of normality for females in the early cohort. Lumbar vertebral (L1-L5) canal diameters are taken from two dimensions: AP = Anteroposterior diameter; TR = Transverse diameter.

VNC Diameter	Race	N	*p*-value
L1AP	Group two	15	0.152
	Group three	13	0.275
	Black	62	0.654
L1TR	Group two	17	0.714
	Group three	15	**0.030**
	Black	65	0.353
L2AP	Group two	17	0.204
	Group three	13	0.968
	Black	62	0.586
L2TR	Group two	16	0.316
	Group three	14	0.641
	Black	65	0.371
L3AP	Group two	16	0.993
	Group three	13	0.083
	Black	59	0.526
L3TR	Group two	17	0.842
	Group three	14	0.982
	Black	67	0.509
L4AP	Group two	17	0.249
	Group three	13	0.681
	Black	57	0.745
L4TR	Group two	16	0.164
	Group three	13	0.279
	Black	55	0.447
L5AP	Group two	12	**0.039**
	Group three	12	0.478
	Black	36	0.433
L5TR	Group two	13	0.405
	Group three	6	0.198
	Black	34	0.515

Table A-11. Shapiro-Wilk's tests of normality for females in the middle cohort. Lumbar vertebral (L1-L5) canal diameters are taken from two dimensions: AP = Anteroposterior diameter; TR = Transverse diameter.

VNC Diameter	Race	N	*p*-value
L1AP	Group two	10	0.908
	Group three	9	0.667
	Group four	3	0.580
	Black	56	0.322
L1TR	Group two	11	0.117
	Group three	10	0.052
	Group four	4	0.911
	Black	56	0.073
L2AP	Group two	11	0.961
	Group three	11	0.126
	Group four	4	0.376
	Black	58	0.706
L2TR	Group two	12	**0.012**
	Group three	12	0.511
	Group four	4	0.466
	Black	59	0.578
L3AP	Group two	11	0.953
	Group three	11	0.739
	Group four	4	0.162
	Black	59	0.346
L3TR	Group two	12	0.230
	Group three	12	0.461
	Group four	4	0.932
	Black	59	0.192
L4AP	Group two	11	0.819
	Group three	8	0.900
	Group four	4	0.320
	Black	56	**0.026**
L4TR	Group two	12	0.326
	Group three	10	0.840
	Group four	4	0.468
	Black	56	**0.014**
L5AP	Group two	8	0.404
	Group three	10	0.109
	Group four	3	0.053
	Black	44	0.565
L5TR	Group two	9	0.281
	Group three	7	0.714
	Black	46	0.170

Table A-12. Shapiro-Wilk's tests of normality for females in the intermediate cohort. Lumbar vertebral (L1-L5) canal diameters are taken from two dimensions: AP = Anteroposterior diameter; TR = Transverse diameter.

VNC Diameter	Race	N	*p*-value
L1AP	Group two	14	0.940
	Group three	12	0.228
	Group four	3	0.409
	Black	12	0.430
L1TR	Group two	15	0.813
	Group three	12	0.063
	Group four	3	0.160
	Black	12	0.639
L2AP	Group two	15	**0.026**
	Group three	14	0.126
	Group four	4	0.093
	Black	11	0.770
L2TR	Group two	17	0.197
	Group three	14	0.684
	Group four	4	0.585
	Black	11	0.911
L3AP	Group two	18	0.559
	Group three	15	0.483
	Group four	4	0.195
	Black	13	0.775
L3TR	Group two	18	0.072
	Group three	15	0.999
	Group four	4	0.294
	Black	13	0.134
L4AP	Group two	18	0.277
	Group three	14	0.787
	Group four	4	0.207
	Black	11	0.955
L4TR	Group two	17	0.982
	Group three	14	0.638
	Group four	4	0.517
	Black	10	**0.020**
L5AP	Group two	17	0.916
	Group three	15	0.461
	Group four	4	0.709
	Black	8	0.426
L5TR	Group two	17	0.434
	Group three	12	0.262
	Group four	3	0.617
	Black	7	0.921

Table A-13. Shapiro-Wilk's tests of normality for females in the late cohort. Lumbar vertebral (L1-L5) canal diameters are taken from two dimensions: AP = Anteroposterior diameter; TR = Transverse diameter.

VNC Diameter	Race	N	*p*-value
L1AP	Group two	19	0.341
	Group three	16	0.524
	Group four	3	0.067
	Black	5	0.960
L1TR	Group two	20	0.583
	Group three	16	0.771
	Group four	3	0.308
	Black	5	0.759
L2AP	Group two	20	0.224
	Group three	16	0.578
	Group four	3	0.694
	Black	5	0.945
L2TR	Group two	20	0.866
	Group three	16	0.774
	Group four	3	0.216
	Black	5	0.812
L3AP	Group two	21	0.118
	Group three	15	0.624
	Black	5	0.933
L3TR	Group two	21	0.782
	Group three	16	0.690
	Group four	3	0.651
	Black	5	0.884
L4AP	Group two	20	0.128
	Group three	15	0.966
	Group four	3	0.206
	Black	5	0.235
L4TR	Group two	20	0.295
	Group three	16	0.416
	Group four	3	0.927
	Black	5	0.975
L5AP	Group two	18	0.947
	Group three	12	0.789
	Black	5	0.483
L5TR	Group two	20	0.217
	Group three	12	0.903
	Black	5	0.387

Table A-14. Fisher's Exact *p*-values for maxillary central incisor LEH frequencies compared among races within the early cohort.

		Male				Female		
	Race	Group two	Group three	Group four	Black	Group two	Group three	Group four
Male	Group two							
	Group three	-						
	Group four	-	-					
	Black	-	-	-				
Female	Group two	-	-	-	-			
	Group three	-	-	-	-	-		
	Group four	-	-	-	-	-	-	
	Black	1.000	0.375	-	0.250	-	-	-

Table A-15. Fisher's Exact *p*-values for mandibular canine LEH frequencies compared among races within the early cohort.

		Male				Female		
	Race	Group two	Group three	Group four	Black	Group two	Group three	Group four
Male	Group two							
	Group three	-						
	Group four	-	-					
	Black	-	-	-				
Female	Group two	-	-	-	-			
	Group three	-	-	-	-	-		
	Group four	-	-	-	-	-	-	
	Black	-	1.000	1.000	1.000	1.000	1.000	-

Table A-16. Fisher's Exact *p*-values for maxillary central incisor LEH frequencies compared among races within the middle cohort.

		Male				Female		
	Race	Group two	Group three	Group four	Black	Group two	Group three	Group four
Male	Group two							
	Group three	-						
	Group four	-	-					
	Black	-	-	-				
Female	Group two	-	-	-	-			
	Group three	-	-	-	0.167	-		
	Group four	-	-	-	-	-	-	
	Black	-	-	-	-	-	0.083	-

Table A-17. Fisher's Exact *p*-values for mandibular canine LEH frequencies compared among races within the middle cohort.

		Male				Female		
	Race	Group two	Group three	Group four	Black	Group two	Group three	Group four
Male	Group two							
	Group three	-						
	Group four	-	-					
	Black	-	-	-				
Female	Group two	-	-	-	-			
	Group three	-	-	-	-	-		
	Group four	-	-	-	-	-	-	
	Black	1.000	1.000	-	0.515	1.000	-	1.000

Table A-18. Fisher's Exact *p*-values for maxillary central incisor LEH frequencies compared among races within the intermediate cohort.

		Male				Female		
	Race	Group two	Group three	Group four	Black	Group two	Group three	Group four
Male	Group two							
	Group three	-						
	Group four	-	-					
	Black	-	-	-				
Female	Group two	-	-	-	-			
	Group three	-	-	-	-	-		
	Group four	-	-	-	-	-	-	
	Black	-	-	-	-	-	-	-

Table A-19. Fisher's Exact *p*-values for mandibular canine LEH frequencies compared among races within the intermediate cohort.

		Male				Female		
	Race	Group two	Group three	Group four	Black	Group two	Group three	Group four
Male	Group two							
	Group three	-						
	Group four	-	-					
	Black	-	-	-				
Female	Group two	0.429	1.000	0.467	0.091			
	Group three	-	-	-	-	0.429		
	Group four	-	-	-	-	1.000	-	
	Black	-	-	-	-	0.429	-	-

Table A-20. Fisher's Exact *p*-values for maxillary central incisor LEH frequencies compared among races within the late cohort.

		Male				Female		
	Race	Group two	Group three	Group four	Black	Group two	Group three	Group four
Male	Group two							
	Group three	-						
	Group four	-	-					
	Black	-	-	-				
Female	Group two	-	-	-	-			
	Group three	-	-	-	-	-		
	Group four	-	-	-	-	-	-	
	Black	-	-	-	1.000	1.000	-	1.000

Table A-21. Fisher's Exact *p*-values for mandibular canine LEH frequencies compared among races within the late cohort.

		Male				Female		
	Race	Group two	Group three	Group four	Black	Group two	Group three	Group four
Male	Group two							
	Group three	0.067						
	Group four	-	-					
	Black	0.505	-	-				
Female	Group two	-	0.152	-	1.000			
	Group three	-	-	-	1.000	0.464		
	Group four	1.000	-	-	1.000	1.000	-	
	Black	-	-	-	1.000	1.000	1.000	1.000

Table A-22. Fisher's Exact p-values for maxillary central incisor LEH frequencies for Group two males across cohorts.

Cohort	Early	Middle	Intermediate
Early			
Middle	-		
Intermediate	-	-	
Late	-	-	-

Table A-23. Fisher's Exact p-values for mandibular canine LEH frequencies for Group two males across cohorts.

Cohort	Early	Middle	Intermediate
Early			
Middle	-		
Intermediate	-	-	
Late	-	1.000	0.400

Table A-24. Fisher's Exact p-values for maxillary central incisor LEH frequencies for Group three males across cohorts.

Cohort	Early	Middle	Intermediate
Early			
Middle	-		
Intermediate	-	-	
Late	-	-	-

Table A-25. Fisher's Exact p-values for mandibular canine LEH frequencies for Group three males across cohorts.

Cohort	Early	Middle	Intermediate
Early			
Middle	-		
Intermediate	1.000	1.000	
Late	1.000	1.000	1.000

Table A-26. Fisher's Exact p-values for maxillary central incisor LEH frequencies for Group four males across cohorts.

Cohort	Early	Middle	Intermediate
Early			
Middle	-		
Intermediate	-	-	
Late	-	-	-

Table A-27. Fisher's Exact p-values for mandibular canine LEH frequencies for Group four males across cohorts.

Cohort	Early	Middle	Intermediate
Early			
Middle	-		
Intermediate	-	-	
Late	-	-	-

Table A-28. Fisher's Exact p-values for maxillary central incisor LEH frequencies for black males across cohorts.

Cohort	Early	Middle	Intermediate
Early			
Middle	-		
Intermediate	-	-	
Late	-	-	-

Table A-29 (also Table 32). Fisher's Exact p-values for mandibular canine LEH frequencies for black males across cohorts.

Cohort	Early	Middle	Intermediate
Early			
Middle	-		
Intermediate	-	-	
Late	**0.050d**	**0.041d**	0.228

Table A-30. Fisher's Exact p-values for maxillary central incisor LEH frequencies for Group two females across cohorts.

Cohort	Early	Middle	Intermediate
Early			
Middle	-		
Intermediate	-	-	
Late	-	-	-

Table A-31. Fisher's Exact p-values for mandibular canine LEH frequencies for Group two females across cohorts.

Cohort	Early	Middle	Intermediate
Early			
Middle	-		
Intermediate	0.429	0.467	
Late	0.444	1.000	1.000

Table A-32. Fisher's Exact p-values for maxillary central incisor LEH frequencies for Group three females across cohorts.

Cohort	Early	Middle	Intermediate
Early			
Middle	-		
Intermediate	-	1.000	
Late	-	-	-

Table A-33. Fisher's Exact p-values for mandibular canine LEH frequencies for Group three females across cohorts.

Cohort	Early	Middle	Intermediate
Early			
Middle	-		
Intermediate	-	-	
Late	-	-	-

Table A-34. Fisher's Exact p-values for maxillary central incisor LEH frequencies for Group four females across cohorts.

Cohort	Early	Middle	Intermediate
Early			
Middle	-		
Intermediate	-	-	
Late	-	-	-

Table A-35. Fisher's Exact p-values for mandibular canine LEH frequencies for Group four females across cohorts.

Cohort	Early	Middle	Intermediate
Early			
Middle	-		
Intermediate	-	-	
Late	-	-	-

Table A-36. Fisher's Exact p-values for maxillary central incisor LEH frequencies for black females across cohorts.

Cohort	Early	Middle	Intermediate
Early			
Middle	0.214		
Intermediate	1.000	-	
Late	1.000	0.214	1.000

Table A-37. Fisher's Exact p-values for mandibular canine LEH frequencies for black females across cohorts.

Cohort	Early	Middle	Intermediate
Early			
Middle	1.000		
Intermediate	1.000	1.000	
Late	0.271	0.256	0.429

APPENDIX II

Trait descriptions taken directly from Ossenberg's (2013): Cranial Nonmetric Trait Database User Guide

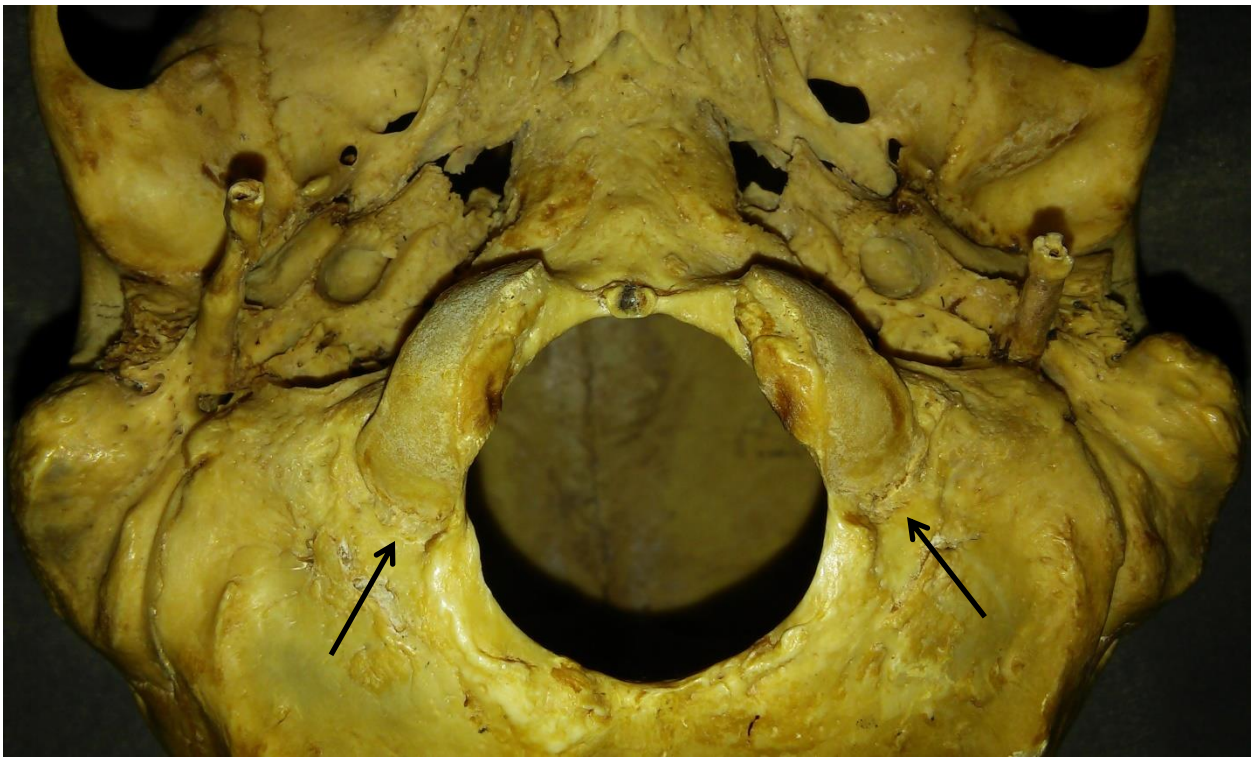

Figure A-1. Postcondylar canal (POS) absent, UT27-91D.

POS Postcondylar canal

A *postcondylar canal* which in life transmits a vein communicating between the sigmoid sinus and the suboccipital plexus is usually present bilaterally. A right or left side in which a canal or foramen of any size pierces the condylar fossa is scored **0;** absence of the postcondylar canal on that side is scored **1.**

Figure A-2. Lateral pterygoid plate foramen (LPF) present, UT12-09D.

LPF Lateral pterygoid plate foramen

This is a round or oval foramen 1-2 mm in diameter piercing the lateral pterygoid plate close to its posterior border and roughly at its mid-point or, more superiorly, near the roof of the infratemporal fossa. During life it transmits the mandibular nerve branch and/or vascular structures supplying the medial pterygoid muscle.

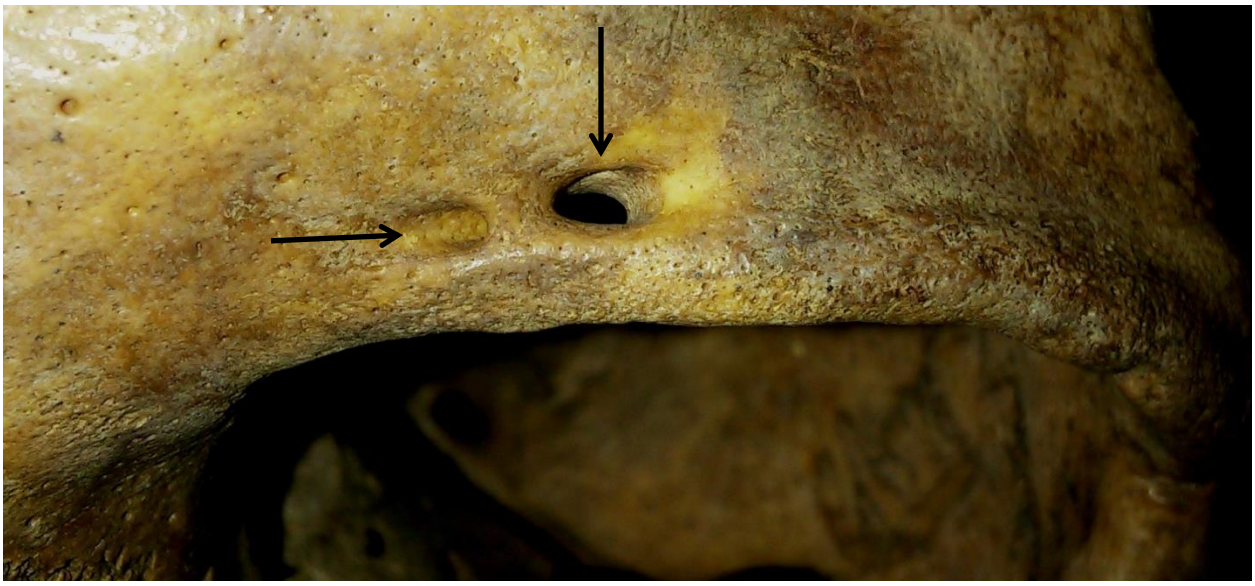

Figure A-3. Supraorbital foramen (SOF) present, UT12-09D.

SOF Supraorbital foramen

A branch of the frontal nerve and associated vessels supplying the skin of the forehead and scalp in some crania exit the orbit through a bony foramen or canal piercing the superior orbital margin. In some cases the canal is deep, its external opening as much as 15 mm above the orbital margin; deepest canals tend to occur in the lateral, rather than in the middle or supratrochlear portion of the margin. More commonly the feature is a foramen seemingly formed when spicules of bone growing from the edges of a deep notch meet; these tend to occur in the middle portion of the margin or towards its medial end. Any such canal or foramen which communicates between the roof of the orbit and the external surface of the frontal bone is scored **1.** Two or more such features on the same side are scored **2.** A deep notch in the orbital margin – even where the spicules of bone *almost* meet – is scored **0.** Openings for diploic veins are scored **0.**

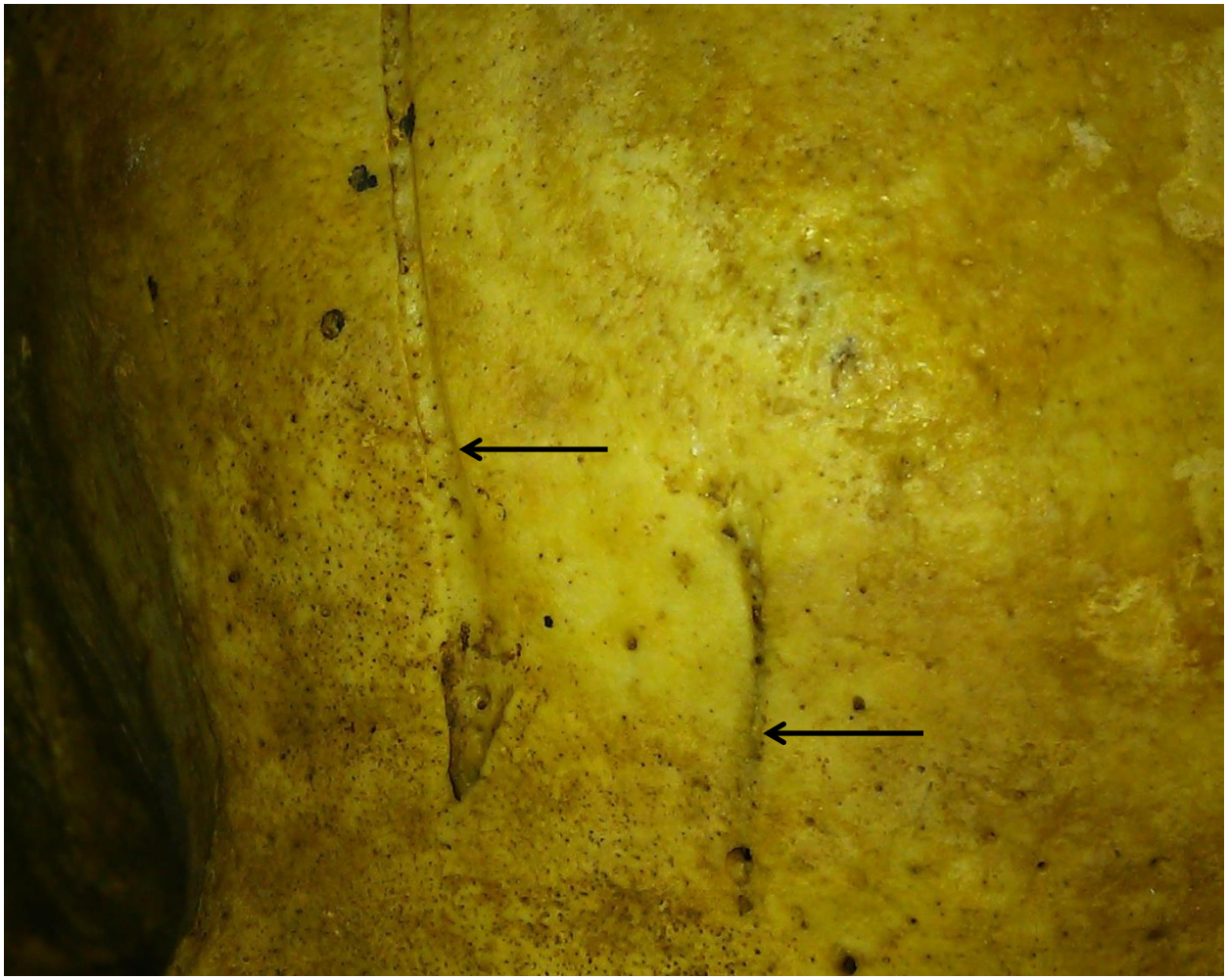

Figure A-4. Frontal grooves (FRG) present, UT37-01D.

FRG Frontal grooves

Frontal grooves scored **1** are usually single, sometimes multiple, grooves impressed into the lateral portion of the frontal bone by branches of the supraorbital nerve and/ or vessels running upwards from the orbital margin to enter the skin of the forehead and scalp. Frontal grooves often occur in association with a deep supraorbital canal, but they also occur independently of the presence of a canal or foramen. In some cases, vague meandering grooves run transversely on the frontal bone; such cases are scored **0.**

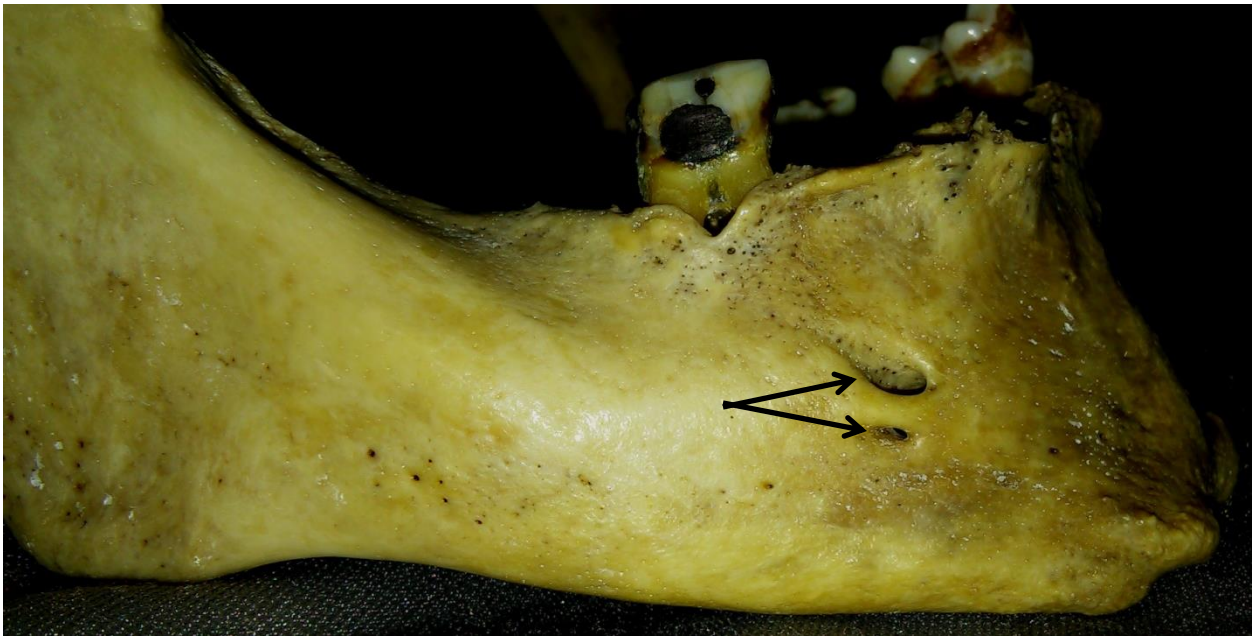

Figure A-5. Mental foramen double or more (MEN) present, UT12-97D.

MEN Mental foramen double

The mental nerve and associated vessels arise in the mandibular canal and exit the mandible via the mental foramen to supply the skin of the lip and chin region. Usually there is a single mental foramen, scored **0.** The case of two or more foramina is scored **1.**

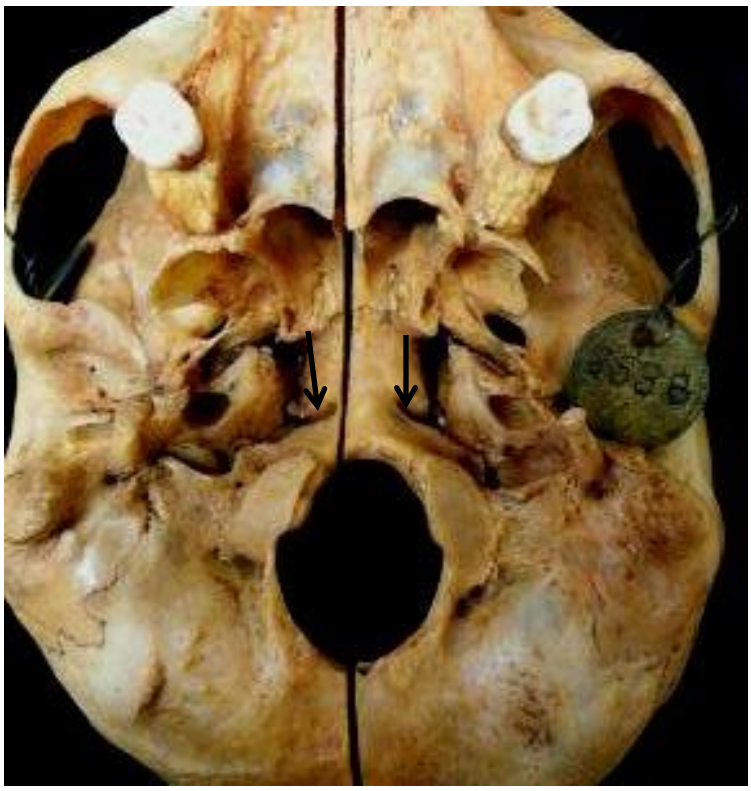

Figure A-6. Transverse fissure of basi-occiput (TRFS) present, HTH 2638.

TRFS Transverse fissure of basiocciput

This rare anomaly is a transversely oriented slit or dehiscence penetrating the basiocciput on one or both sides. Sometimes the slit is isolated within the occipital bone; more often it extends laterally to the petro-occipital synchondrosis giving the basiocciput a “waisted” apprearance. Any such feature is scored **1.**

Figure A-7. Hypoglossal canal bridged or double (HYP) present, UT115-07D.

HYP Hypoglossal canal bridged or double

The hypoglossal (anterior condylar) canal gives passage to cranial nerve XII and occasional vascular structures. It is usually single and undivided. The presence of two hypoglossal canals; or a case where the canal is partly occluded by a bony bridge – whether at the internal or the external aperture or anywhere within the canal - is scored **1.** Any partial expression of a bridge (spurs) is scored **0.** This trait is one of the manifestations of the occipital vertebra theoretically resulting from incomplete coalescence of the occipital somites; i.e. cranial border shift.

Figure A-8. Foramen spinosum and/or ovale wall deficient (FSP) present, UT12-09D.

FSP Deficient wall of the foramen spinosum and / or ovale

The roots of the sphenoid greater wing in the region of foramen rotundum and the pterygoid canal are preformed in cartilage; the other portions of the greater wing ossify in membrane. Early in the human fetus neither the foramen spinosum nor foramen ovale are differentiated: the mandibular nerve, middle meningeal artery and associated structures make their exit from, or entry to, the middle cranial fossa through the foramen lacerum medium as in the adult forms of other mammals. Subsequently, bone encroaches on and surrounds the neurovascular structures thereby separating the foramen ovale, foramen spinosum (and occasionally an emissary foramen of Vesalius anterior to foramen ovale) from each other and from the sphenopetrous fissure. Various expressions of arrested morphogenesis in this region are recognized: foramen ovale and spinosum are confluent, either foramen communicates with the sphenopetrous fissure, both foramina open into the fissure, or any combination of these deficiencies. The communications vary from the merest suture-like slit, to large deficiencies in the walls. Foramen spinosum confluent with sphenopetrous fissure is the trait most commonly seen. Any such variant is scored **1.**

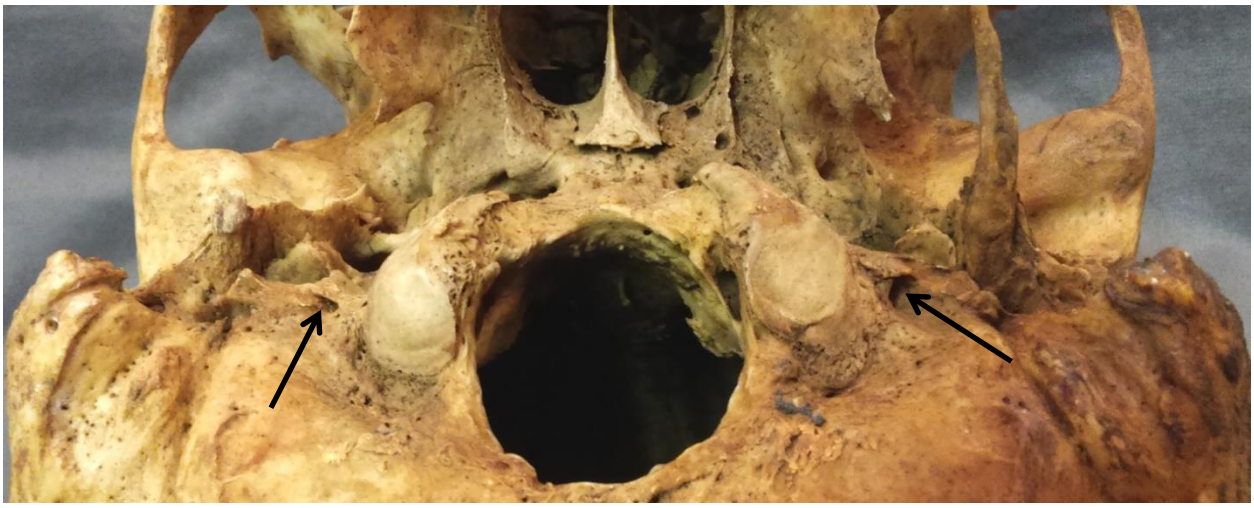

Figure A-9. Intermediate condylar canal (ICC) present, UT45-05D.

ICC Intermediate (lateral) condylar canal

A small vein commonly connects the beginning of the internal jugular and the anterior condylar (hypoglossal) emissary vein with the postcondylar emissary vein or suboccipital plexus. This vein runs backwards in a groove lateral to the base of the occipital condyle. In some crania a bony crest from the lateral lip of the groove grows medially to fuse with the base of the condyle whereby the groove for all of its length - or more commonly for a short portion - is converted to a canal one to two mm in diameter. Any such canal is scored **1.** A spur or crest which fails to fuse with the condyle is scored **0.** Because of the orientation of the ICC its openings especially in the case of a long canal of small diameter easily escape notice; the skull should therefore be tilted slightly to an oblique position to make the observation.

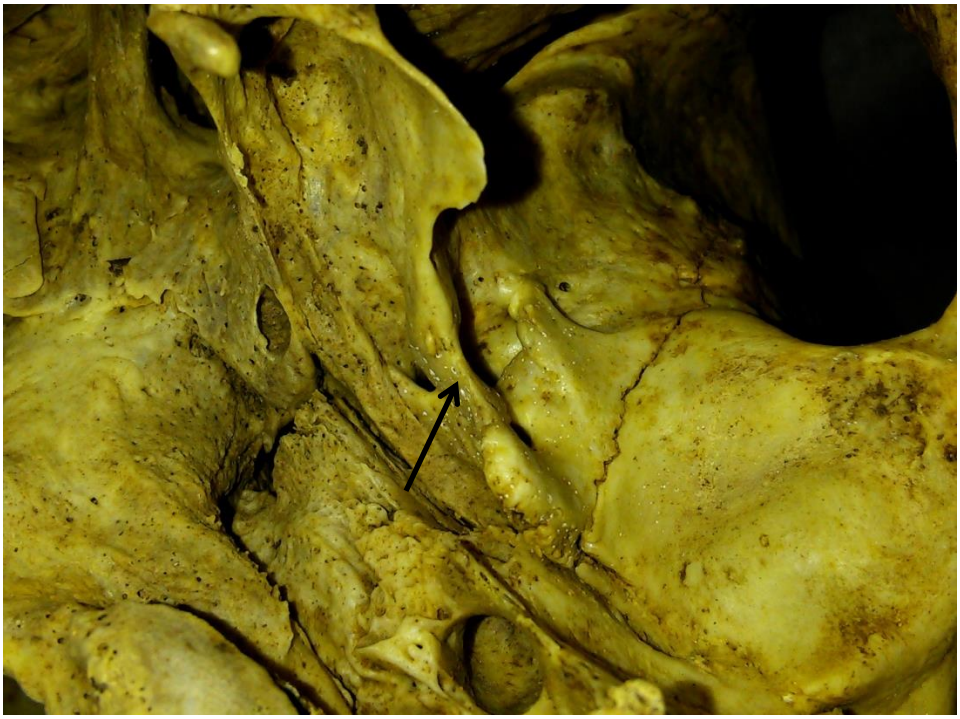

Figure A-10. Pterygospinous bridge (foramen Civinini) (CIV) present, UT23-88D.

CIV Pterygospinous bridge (Foramen of Civinini)

The pterygospinous ligament stretches from a point near the middle of the posterior border of the lateral pterygoid plate to, or to some point near, the spine of the sphenoid. The variant scored **1** is complete ossification of this ligament. A case where spurs extend towards each other but do not actually join is scored **0.** The *Pterygospinous bridge* forms a foramen, more or less sagittally oriented, and situated below and medial to the foramen ovale. This anomalous bony foramen varies in size and shape according to the extent of ossification of the structures forming its margins (i.e. the plate, ligament and spine) and may be subdivided into two or more apertures, completely, or partially by means of bony spurs. The trait LPF (*Lateral pterygoid plate foramen)* often occurs with a *Pterygospinous bridge,* but can also occur independently.

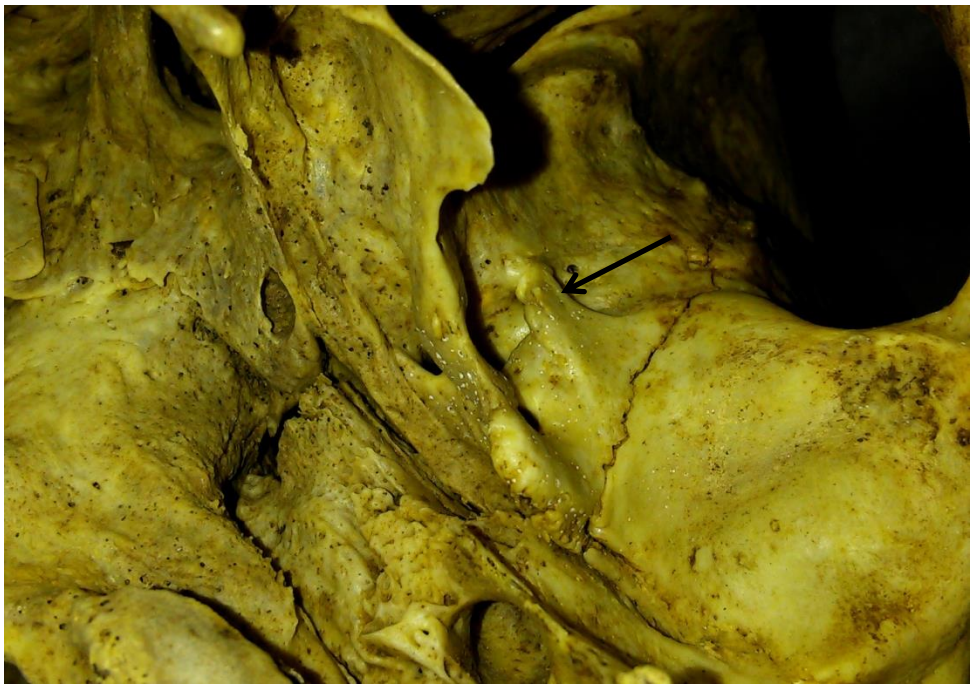

Figure A-11. Pterygobasal bridge (PTB) present, UT23-88D.

PTB Pterygobasal bridge

A ligament commonly stretches from the posterior border of the lateral ptyerygoid plate near its root, to a point on the greater wing of sphenoid lateral to the foramen ovale. The ligament likely gives attachment to fibres of the upper head of the lateral pterygoid muscle, and stretches below and protects the masseteric and deep temporal branches of the mandibular nerve. Occasionally, as they course laterally from the foramen ovale on the greater wing of sphenoid these nerves for a short distance lie in an approximately 5 mm wide shallow sulcus. Independent of the presence or not of a sulcus, the ligament may ossifiy completely or partially. Minimal expression scored **1**, is a tiny sharp forward- pointing spur on the greater wing lateral to the foramen ovale; also scored **1** is the case where a shallow sulcus is present and deepened slightly by a bony spur or crest seemingly pinched up from its posterior margin. Full expression scored **3** is either complete ossification of the ligament or spurs that *almost* connect, with only a slit-like gap between them.

An expression larger than minimal yet not sufficient to merit a **3** is scored **2.** Though situated close to each other on the roof of the infratemporal fossa *Pterygospinous bridge*, situated medial to the foramen ovale, cannot be confused with *Pterygobasal bridge* which lies lateral to the foramen; they are distinct and independent traits.

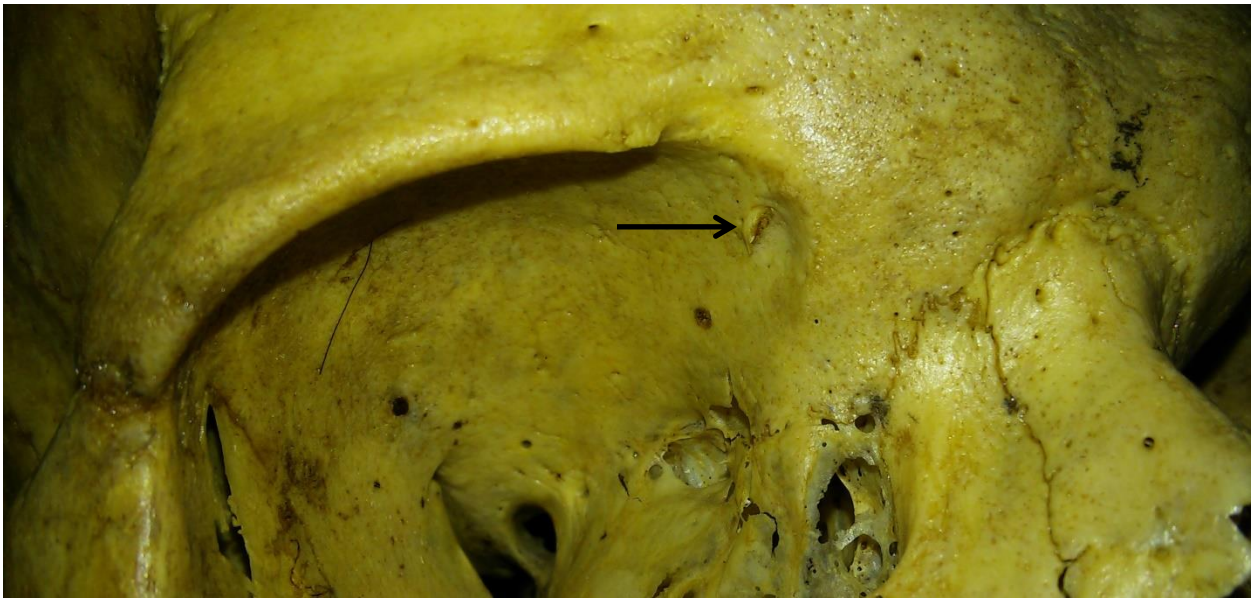

Figure A-12. Trochlear spur (TRS) present, UT10-89D.

TRS Trochlear spur

This variant is a small spine on the upper medial wall of the orbit at the site of attachment of the fibrocartilaginous pulley for the tendon of the superior oblique muscle of the eyball. It represents ossification into one of the two ligaments –most commonly the ligament of the posterior-superior horn - attaching the cartilaginous arc of the pulley to the frontal bone. The bony spur varies from barely perceptible to well-developed: any expression is scored **1.**

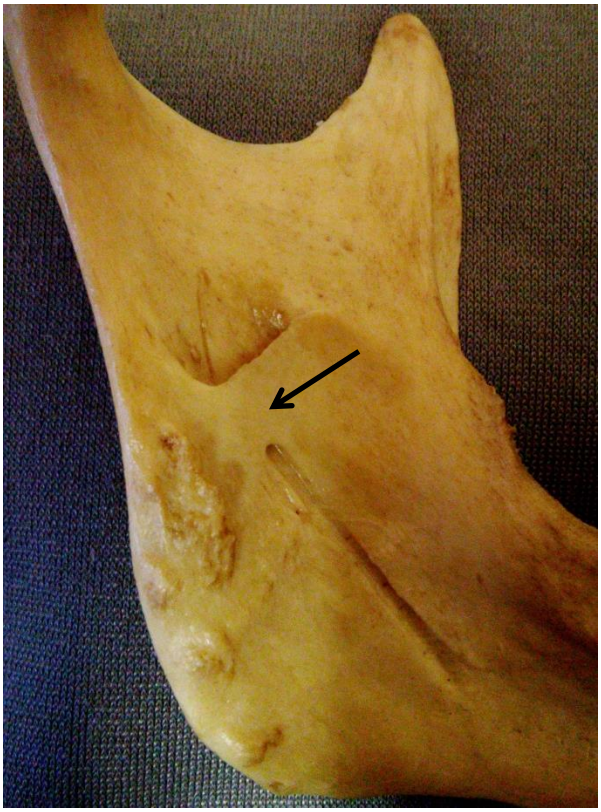

Figure A-13. Mylohyoid bridge (MHB) present, UT07-89D

MHB Mylohyoid bridge

Ossification of the sphenomandibular ligament at its insertion on the medial surface of the mandibular ramus converts the mylohyoid groove to a bony canal enclosing the mylohyoid nerve and vessels, a variant scored **1.** The mylohyoid canal varies in length from 2 to 25 mm and may be interrupted into two or more segments. Rarely, the mylohyoid canal opens superiorly at the level of the mandibular foramen. In this case its opening is often shielded by an extension backwards of the lingula (the extension also representing ossification into the sphenomandibular ligament). Such high-opening mylohyoid canals, especially if they are long ones, can easily be overlooked. Mylohyoid bridges starting at the level of the mandibular foramen were noted separately on my scoring sheets but included with the other MHB variants scored **1** in the tables.

APPENDIX III

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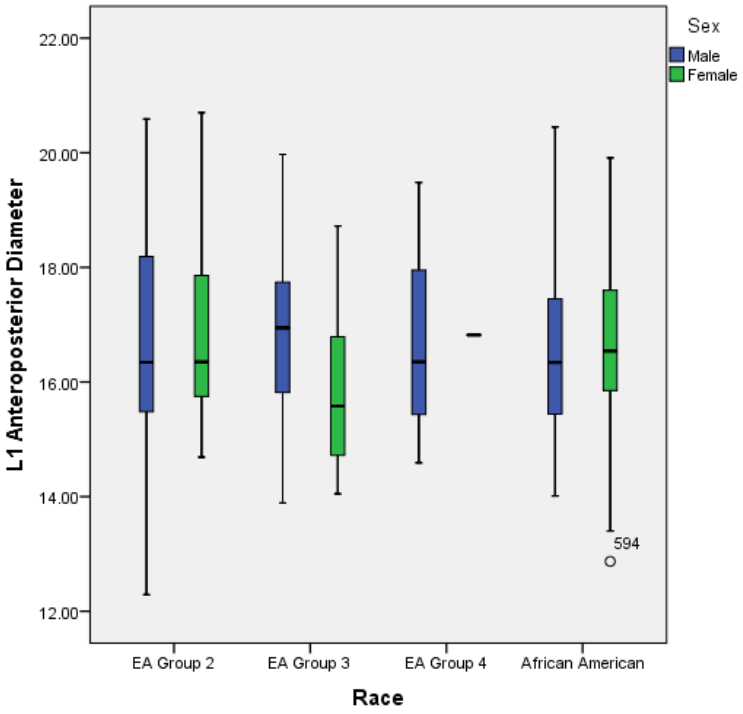

Figure A-14. Early cohort (1828-1881) L1 anteroposterior diameter among race/sex groups.

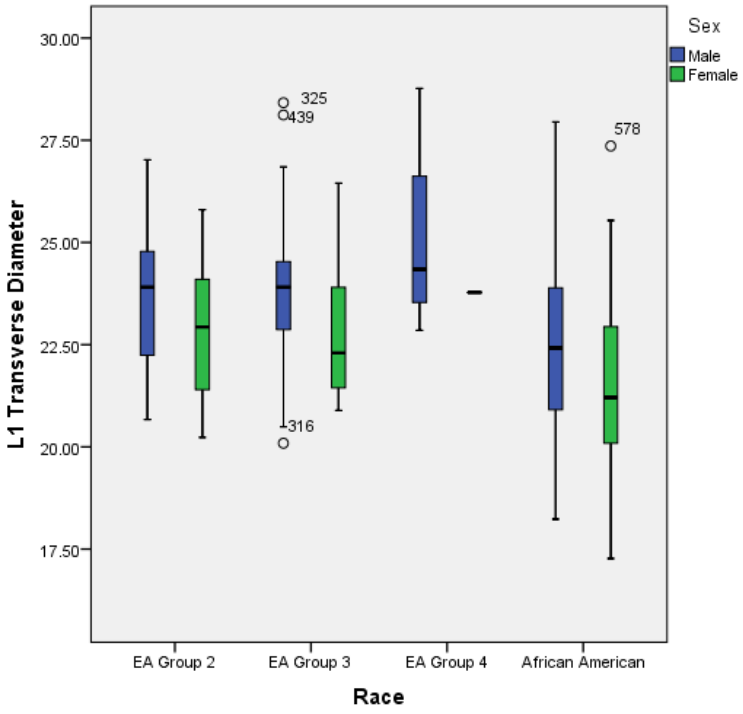

Figure A-15. Early cohort (1828-1881) L1 transverse diameter among race/sex groups.

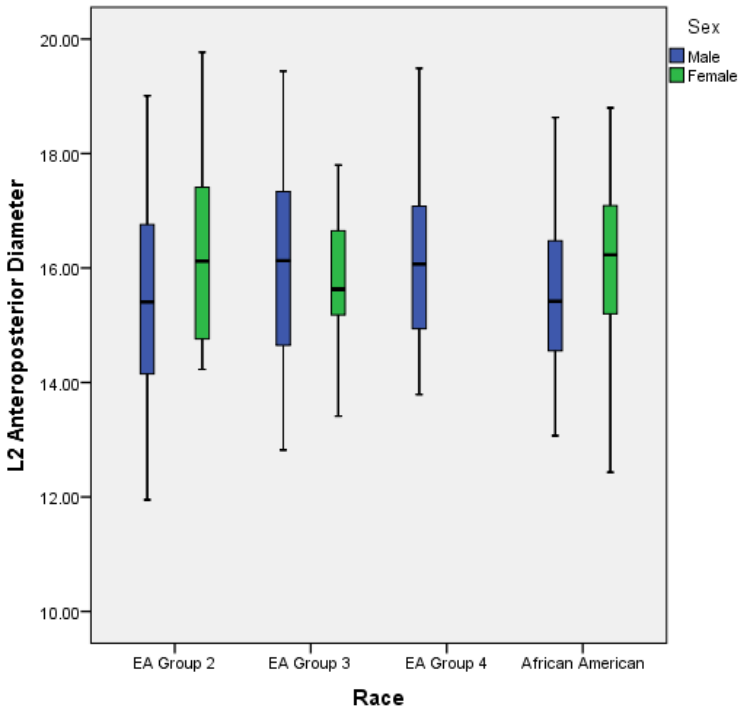

Figure A-16. Early cohort (1828-1881) L2 anteroposterior diameter among race/sex groups.

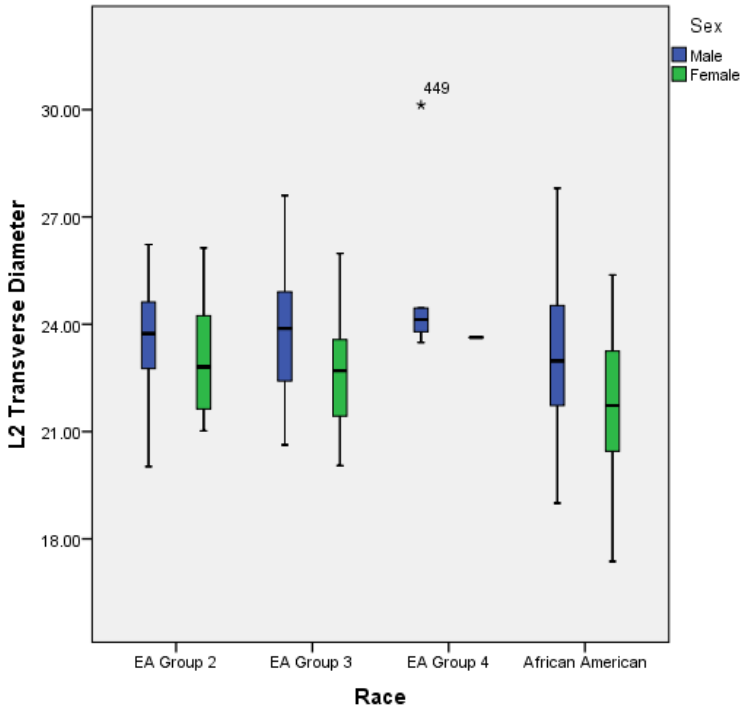

Figure A-17. Early cohort (1828-1881) L2 transverse diameter among race/sex groups.

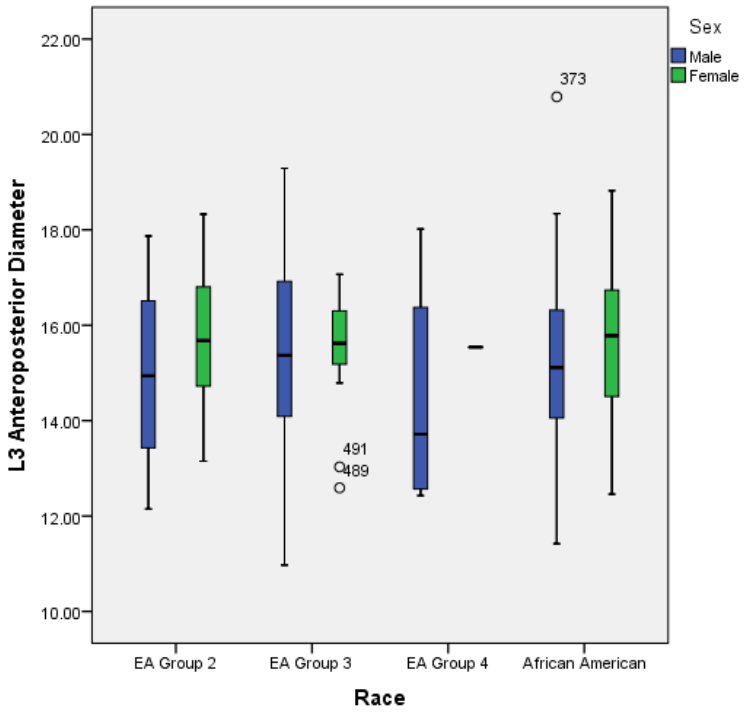

Figure A-18. Early cohort (1828-1881) L3 anteroposterior diameter among race/sex groups.

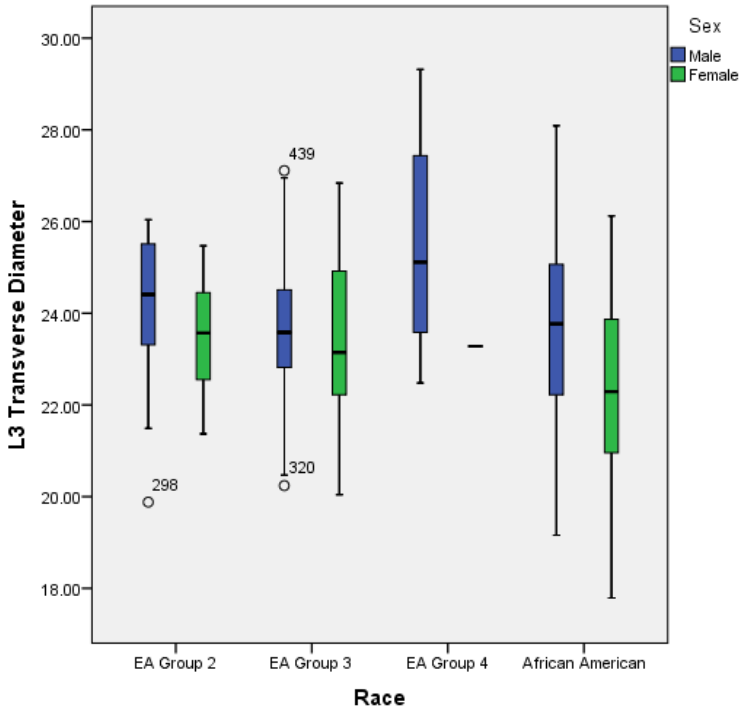

Figure A-19. Early cohort (1828-1881) L3 transverse diameter among race/sex groups.

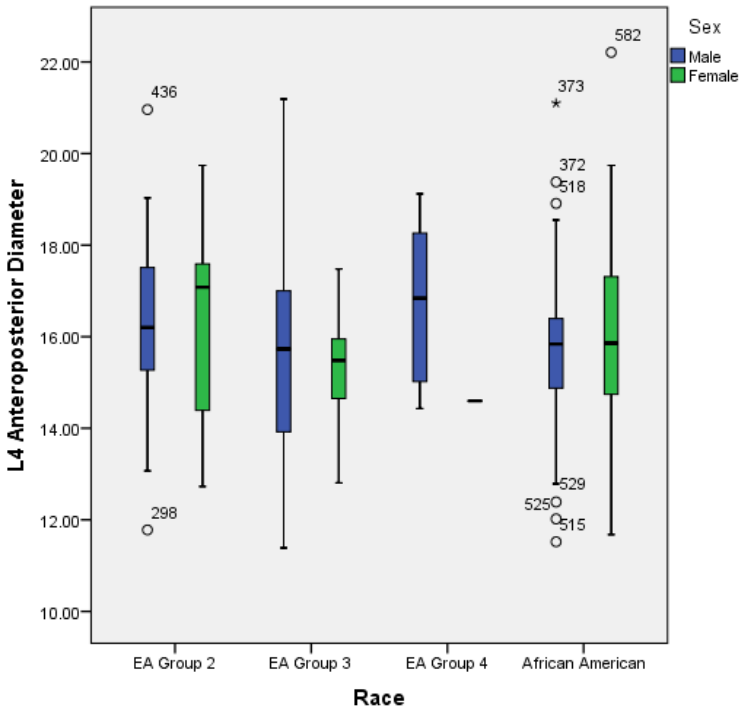

Figure A-20. Early cohort (1828-1881) L4 anteroposterior diameter among race/sex groups.

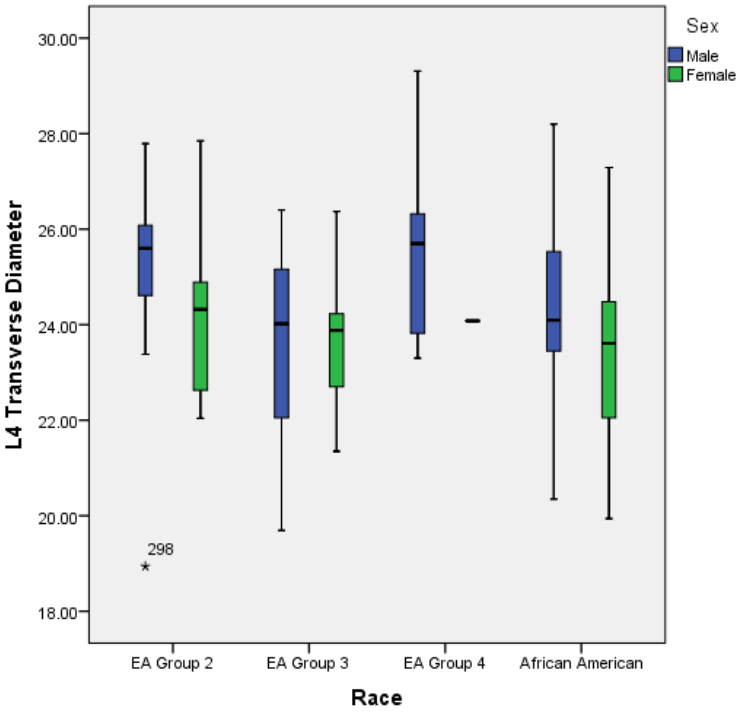

Figure A-21. Early cohort (1828-1881) L4 transverse diameter among race/sex groups.

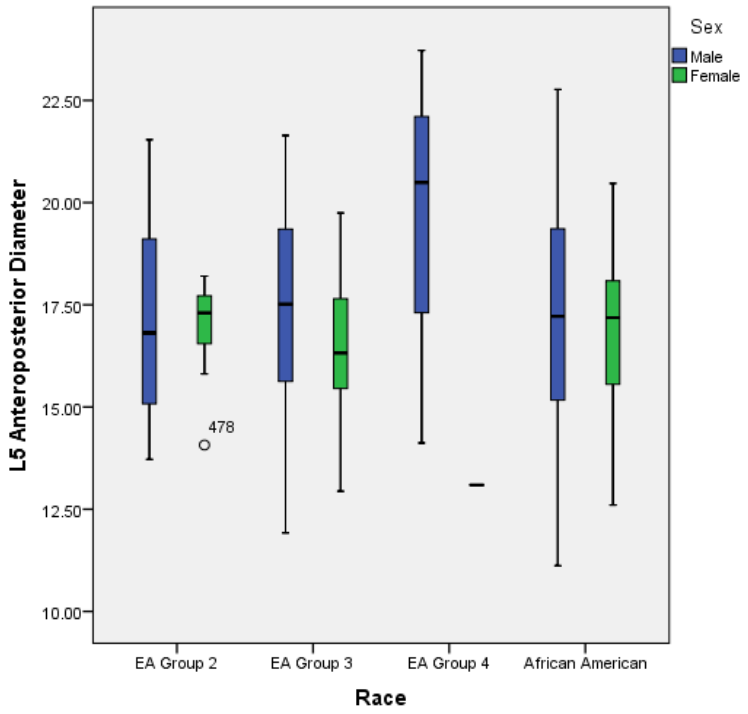

Figure A-22. Early cohort (1828-1881) L5 anteroposterior diameter among race/sex groups.

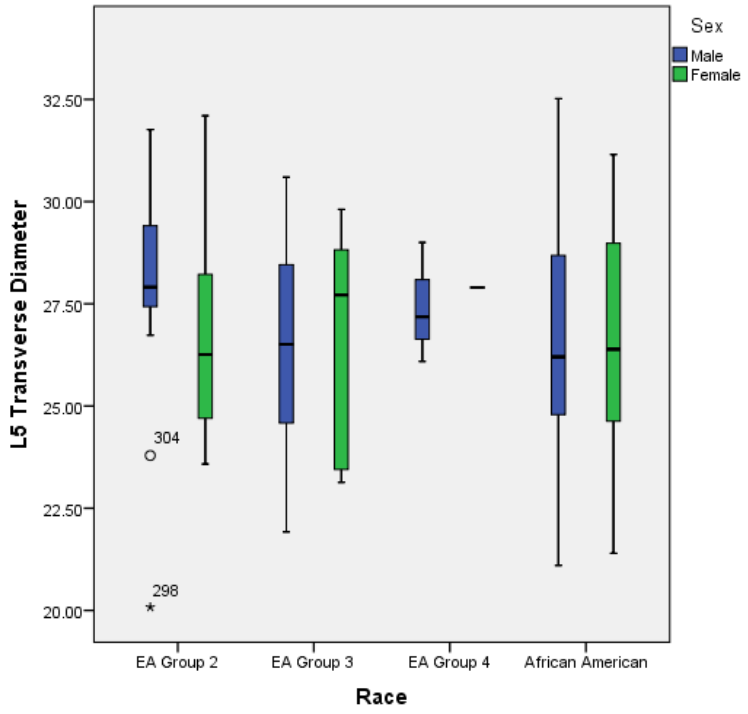

Figure A-23. Early cohort (1828-1881) L5 transverse diameter among race/sex groups.

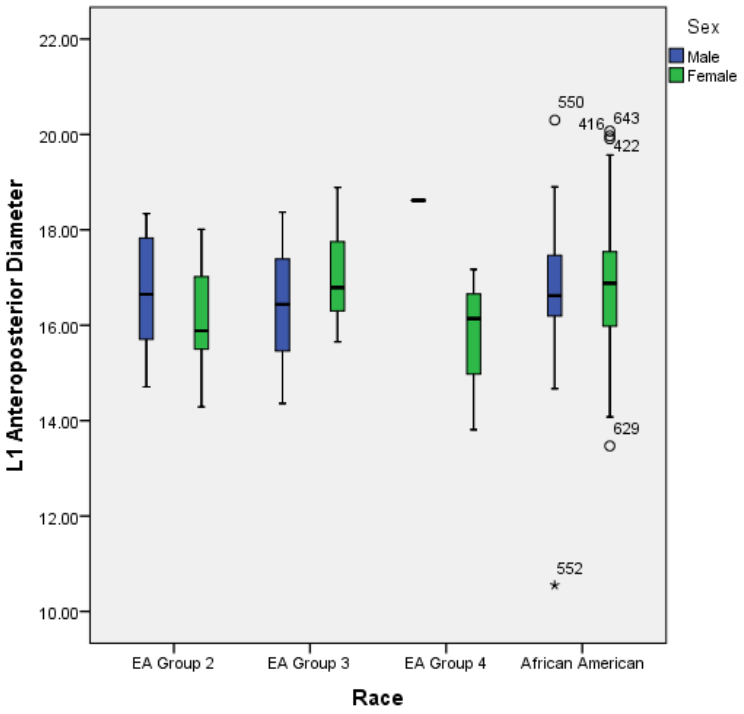

Figure A-24. Middle cohort (1882-1913) L1 anteroposterior diameter among race/sex groups.

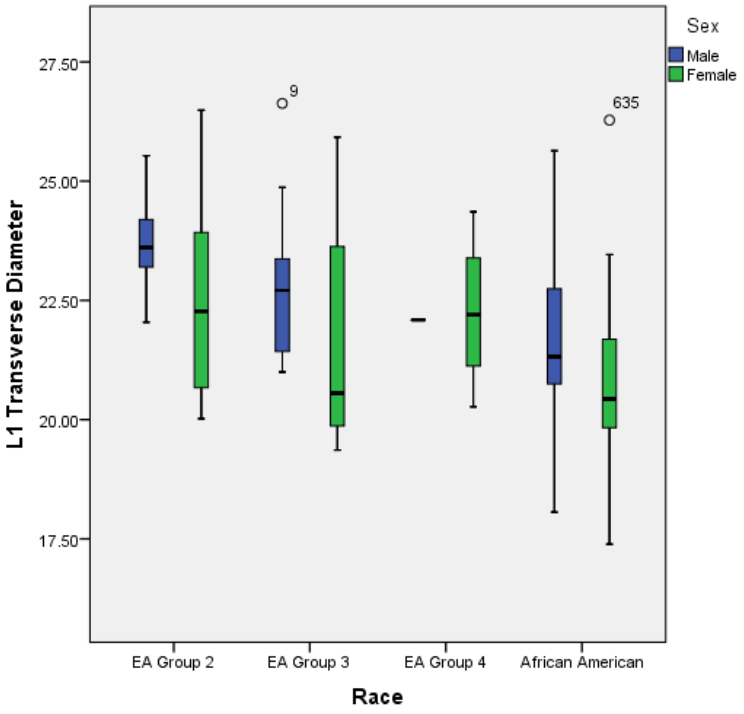

Figure A-25. Middle cohort (1882-1913) L1 transverse diameter among race/sex groups.

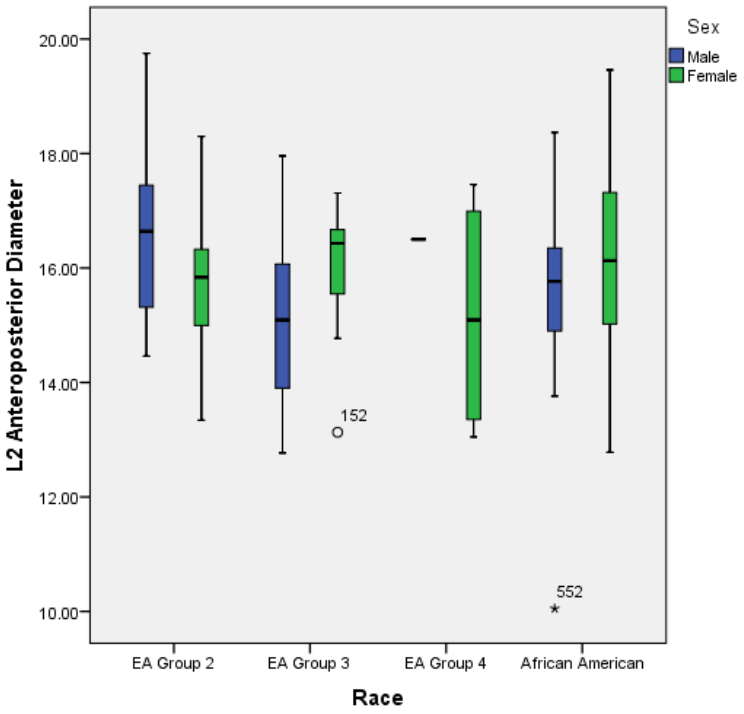

Figure A-26. Middle cohort (1882-1913) L2 anteroposterior diameter among race/sex groups.

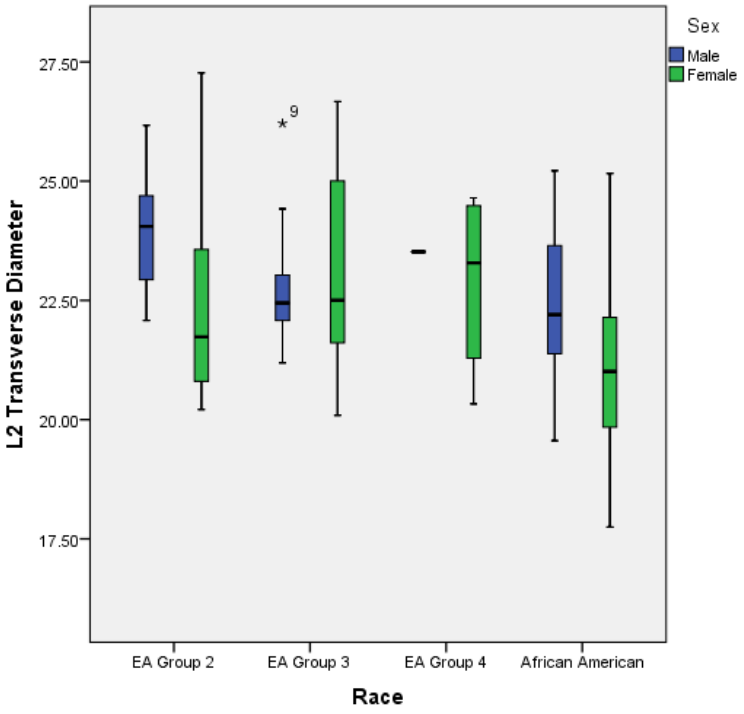

Figure A-27. Middle cohort (1882-1913) L2 transverse diameter among race/sex groups.

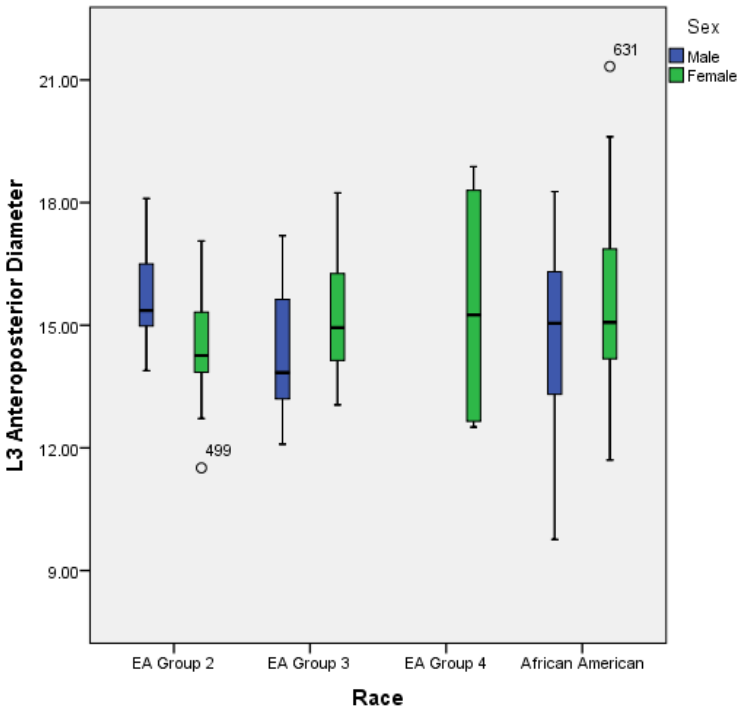

Figure A-28. Middle cohort (1882-1913) L3 anteroposterior diameter among race/sex groups.

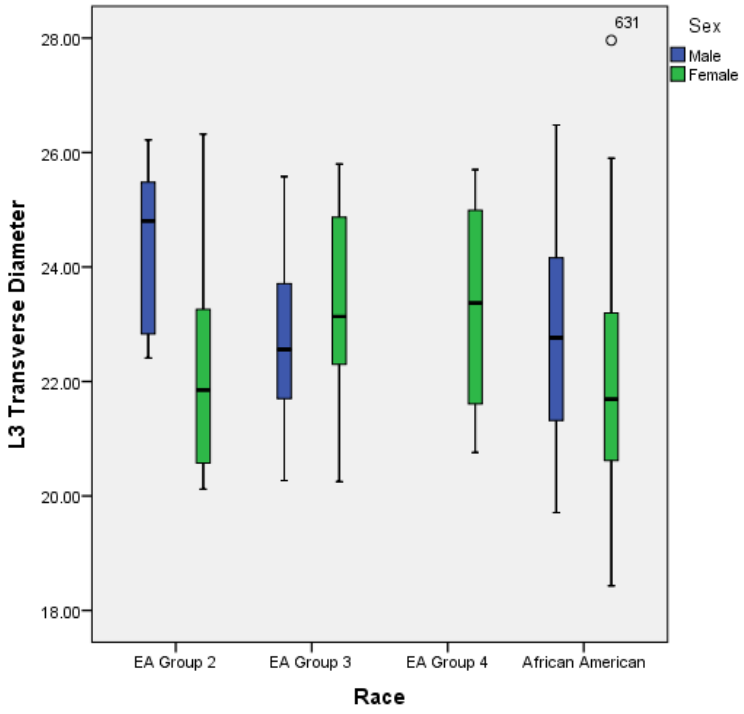

Figure A-29. Middle cohort (1882-1913) L3 transverse diameter among race/sex groups.

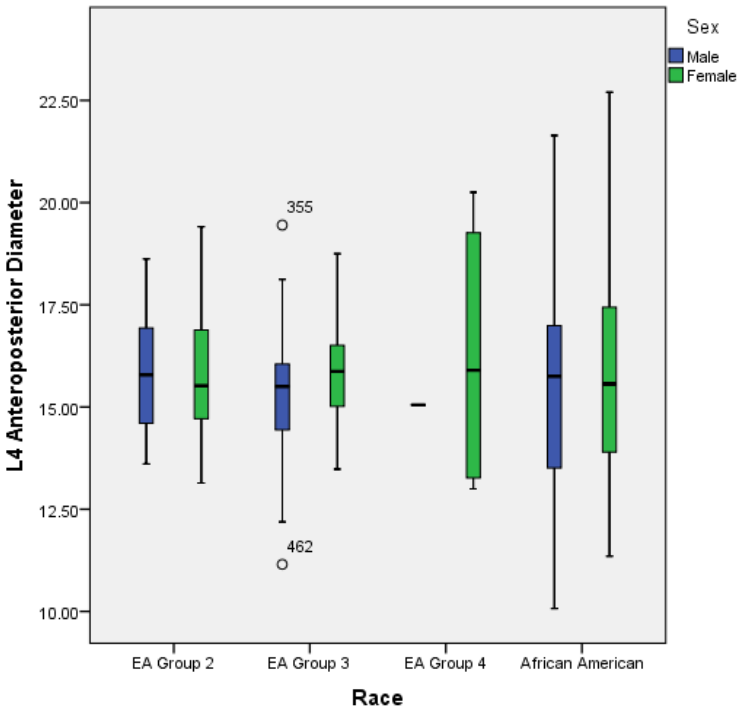

Figure A-30. Middle cohort (1882-1913) L4 anteroposterior diameter among race/sex groups.

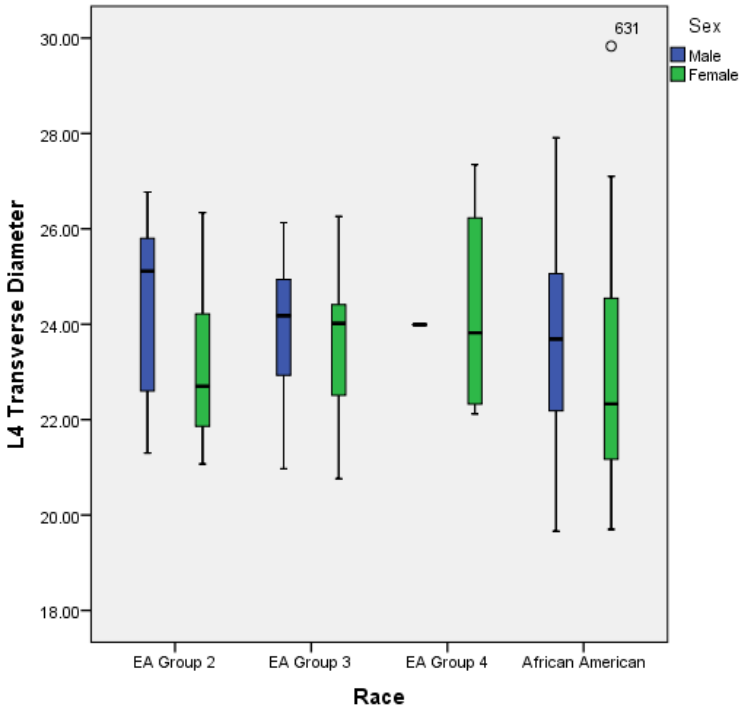

Figure A-31. Middle cohort (1882-1913) L4 transverse diameter among race/sex groups.

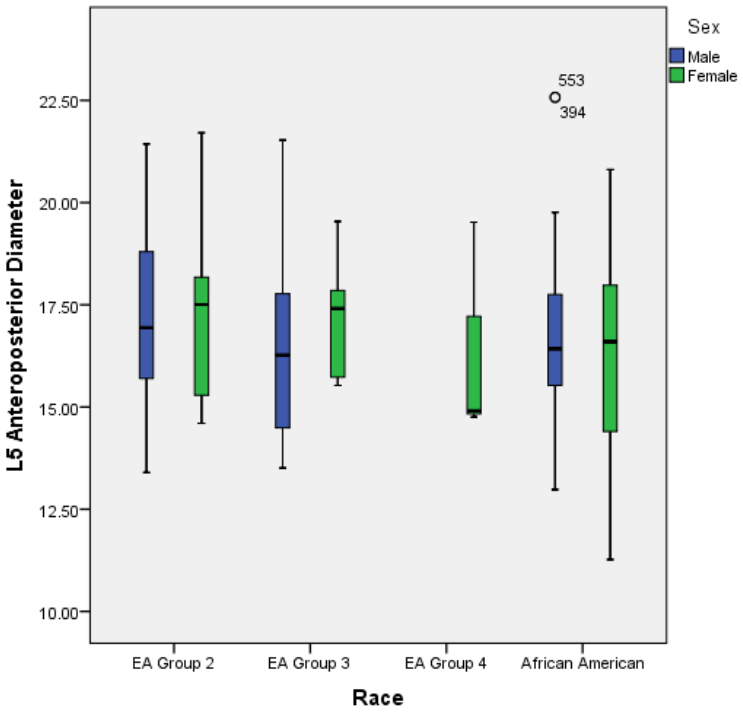

Figure A-32. Middle cohort (1882-1913) L5 anteroposterior diameter among race/sex groups.

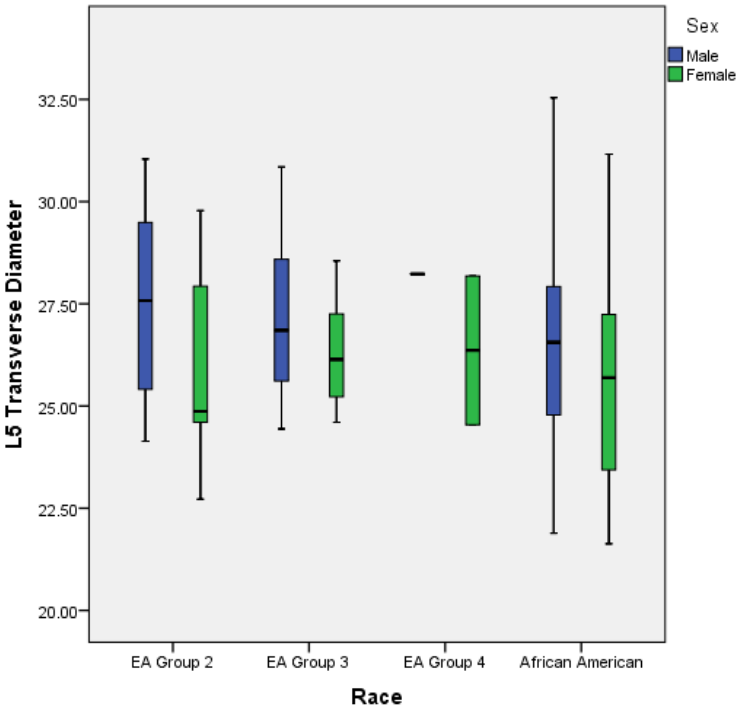

Figure A-33. Middle cohort (1882-1913) L5 transverse diameter among race/sex groups.

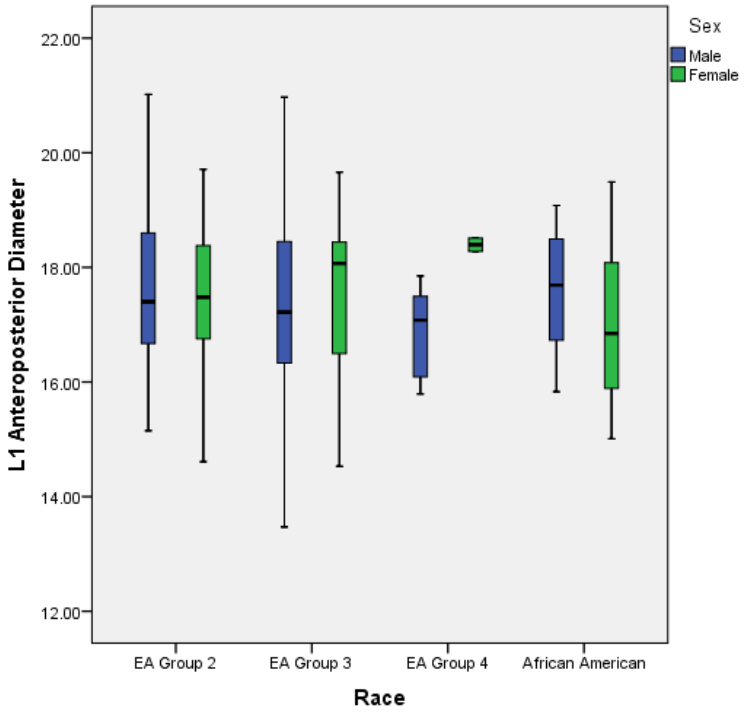

Figure A-34. Intermediate cohort (1914-1945) L1 anteroposterior diameter among race/sex groups.

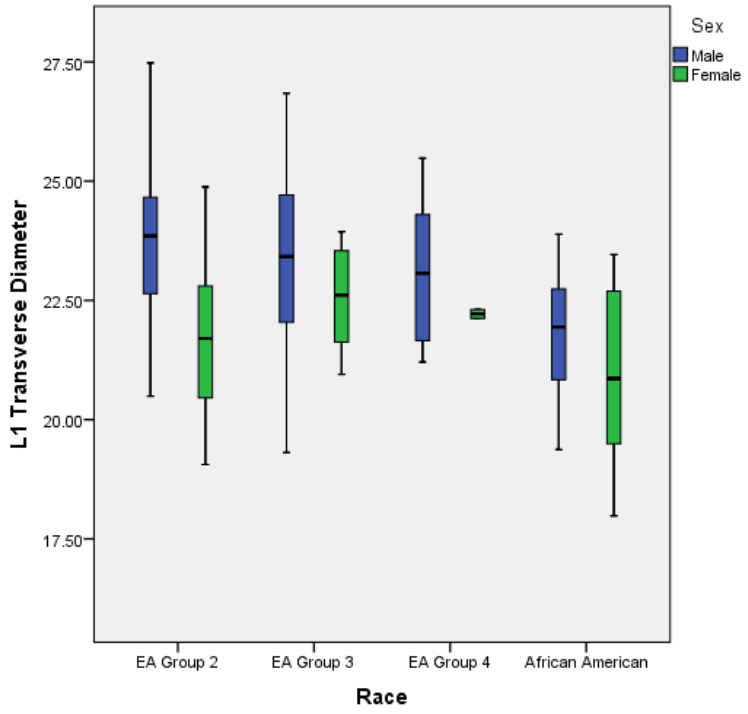

Figure A-35. Intermediate cohort (1914-1945) L1 transverse diameter among race/sex groups.

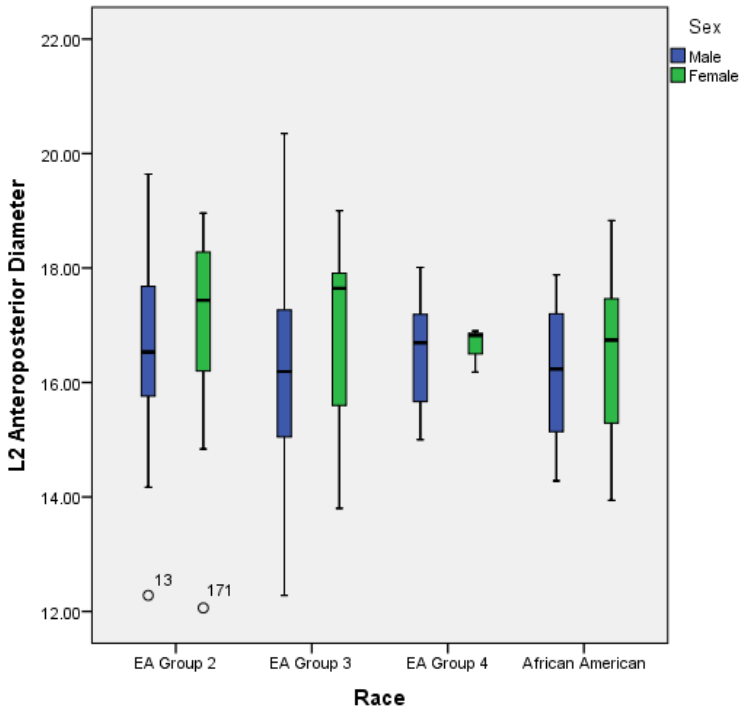

Figure A-36. Intermediate cohort (1914-1945) L2 anteroposterior diameter among race/sex groups.

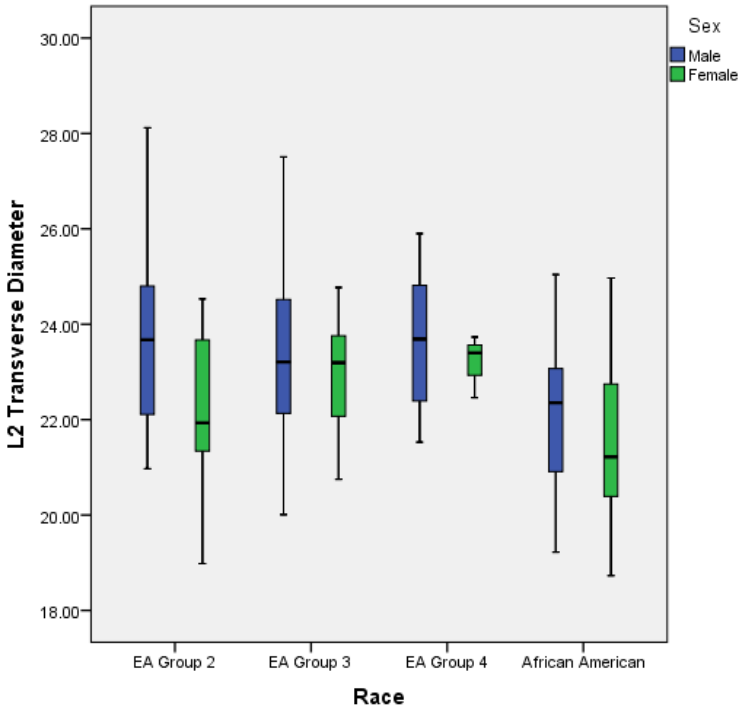

Figure A-37. Intermediate cohort (1914-1945) L2 transverse diameter among race/sex groups.

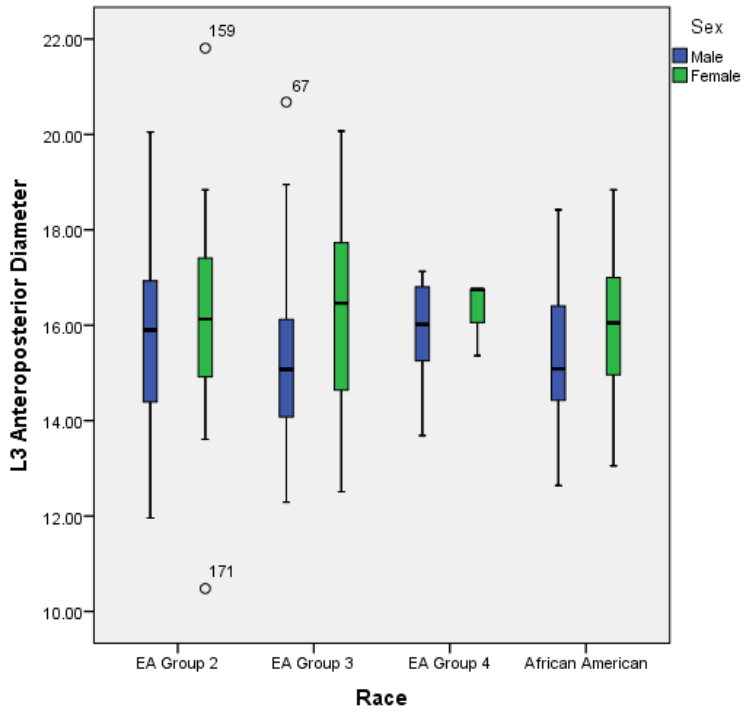

Figure A-38. Intermediate cohort (1914-1945) L3 anteroposterior diameter among race/sex groups.

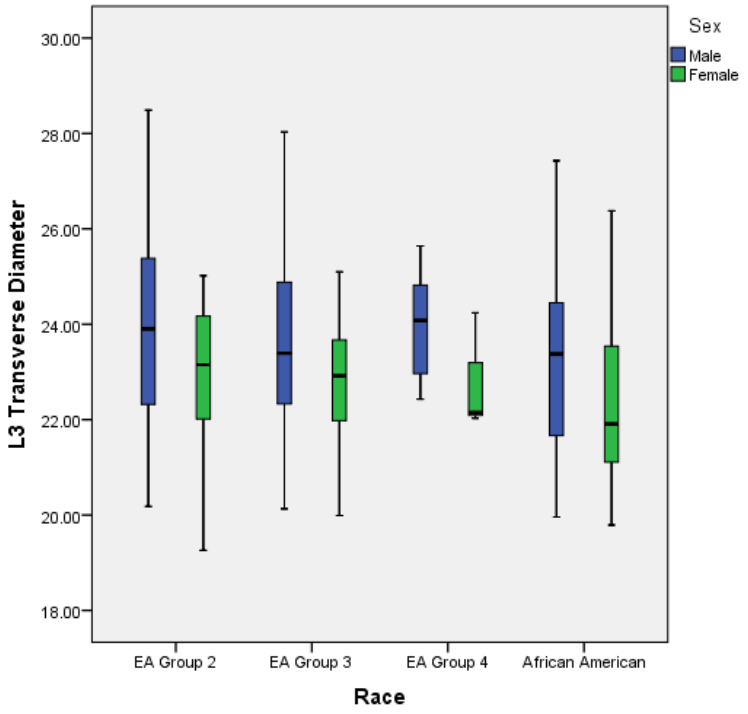

Figure A-39. Intermediate cohort (1914-1945) L3 transverse diameter among race/sex groups.

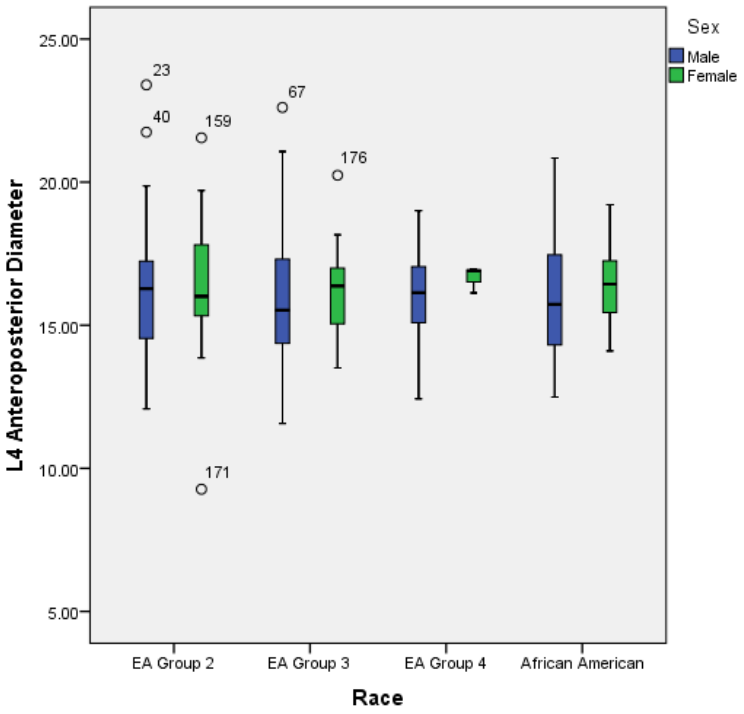

Figure A-40. Intermediate cohort (1914-1945) L4 anteroposterior diameter among race/sex groups.

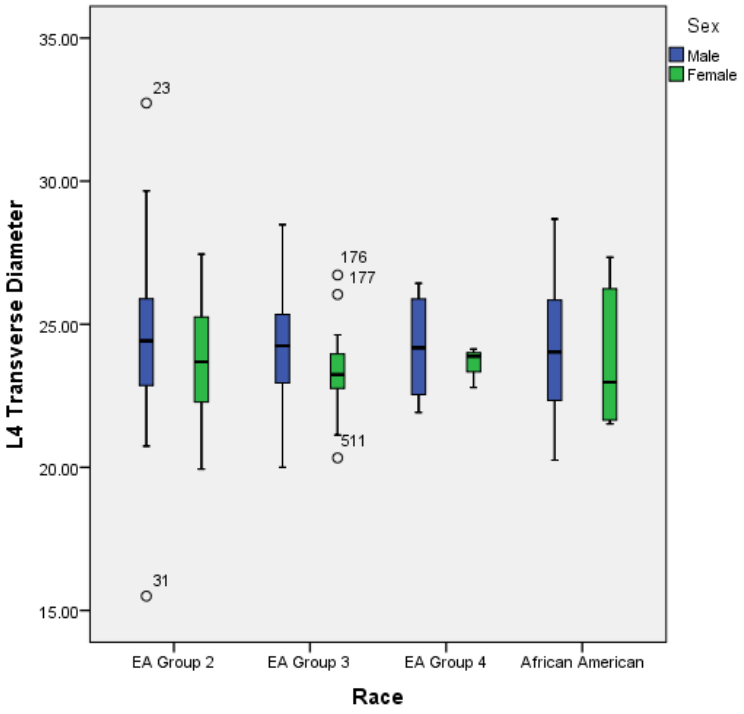

Figure A-41. Intermediate cohort (1914-1945) L4 transverse diameter among race/sex groups.

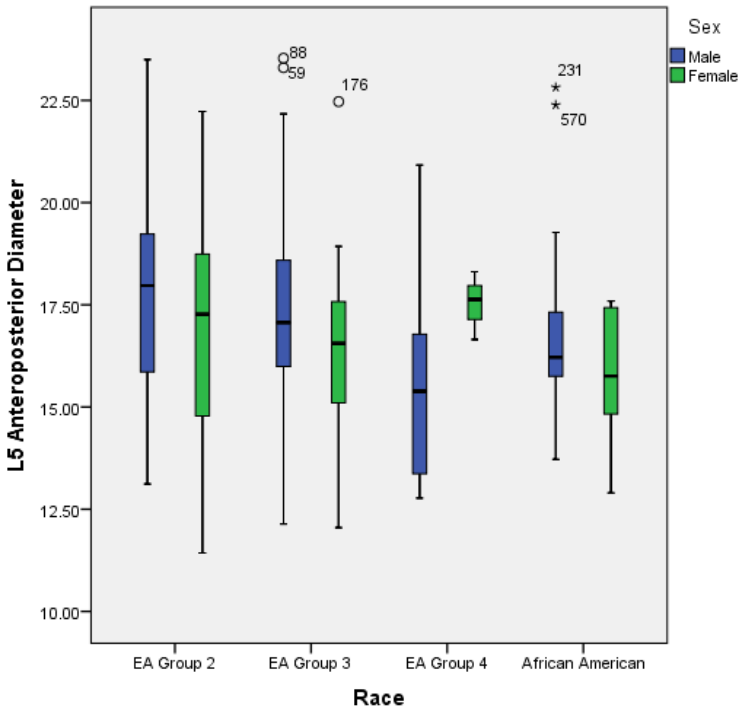

Figure A-42. Intermediate cohort (1914-1945) L5 anteroposterior diameter among race/sex groups.

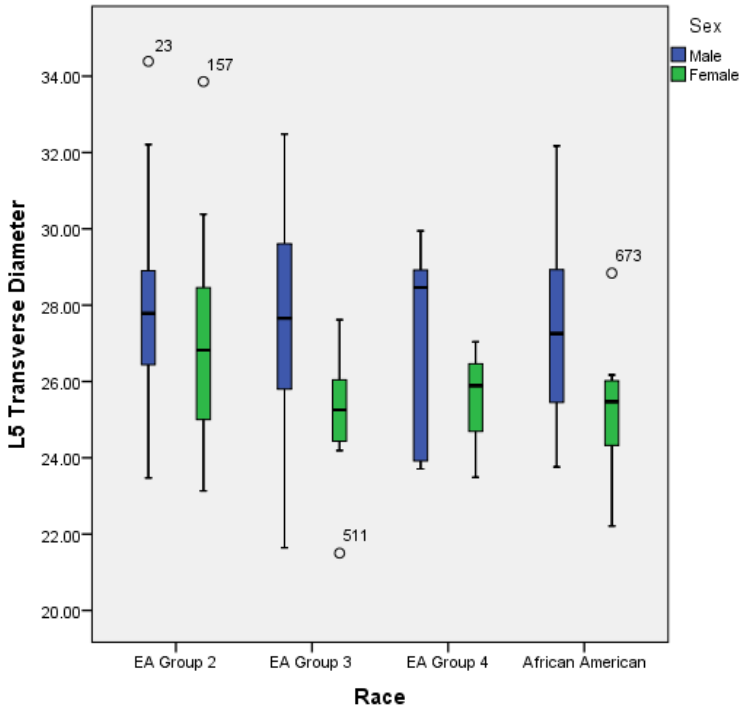

Figure A-43. Intermediate cohort (1914-1945) L5 transverse diameter among race/sex groups.

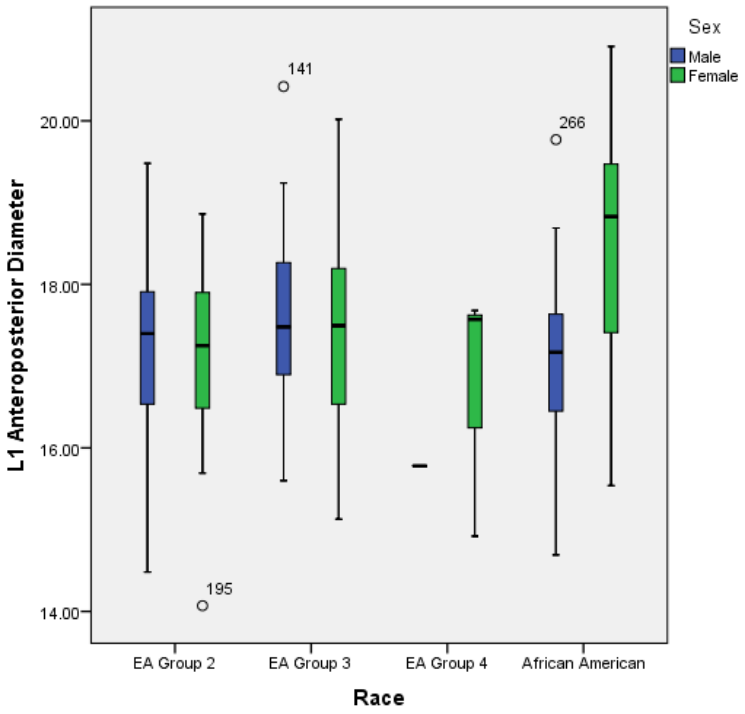

Figure A-44. Late cohort (1946-1984) L1 anteroposterior diameter among race/sex groups.

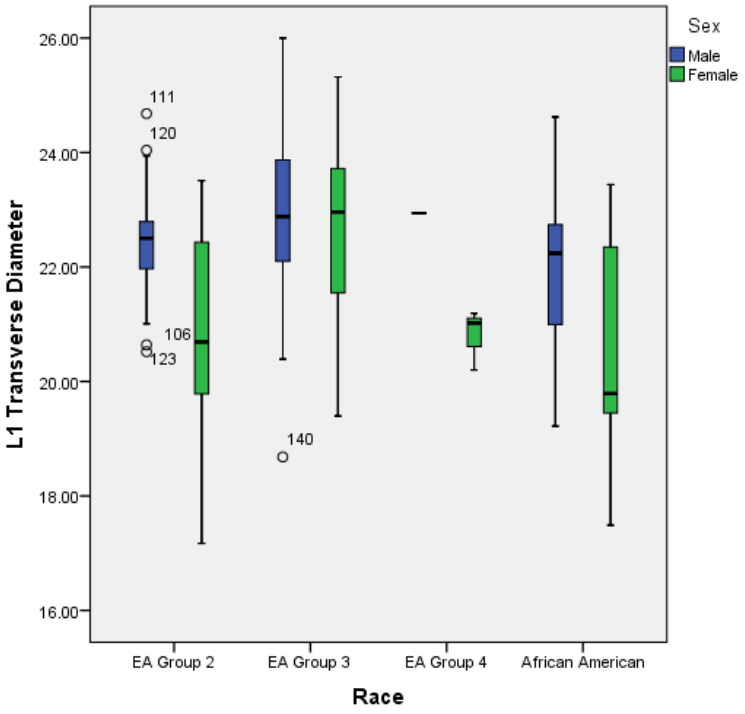

Figure A-45. Late cohort (1946-1984) L1 transverse diameter among race/sex groups.

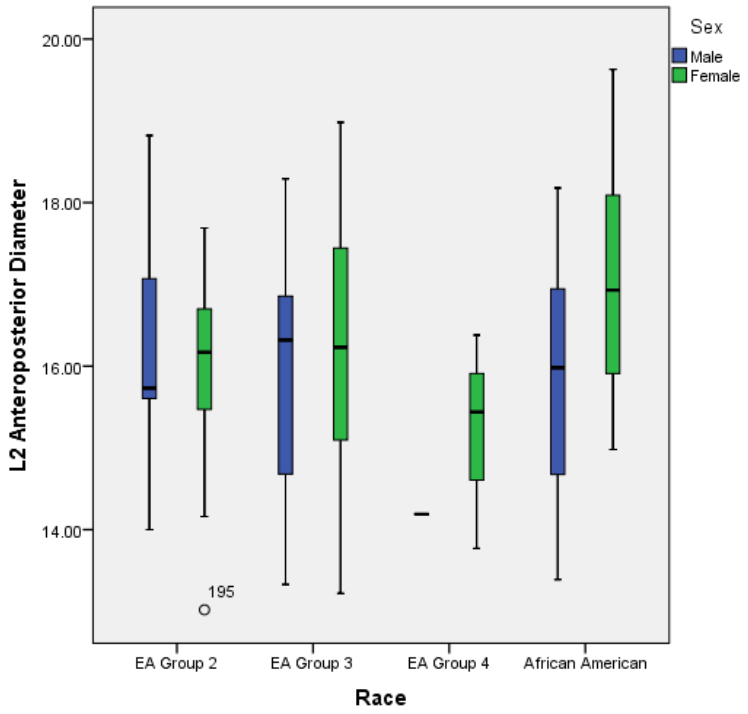

Figure A-46. Late cohort (1946-1984) L2 anteroposterior diameter among race/sex groups.

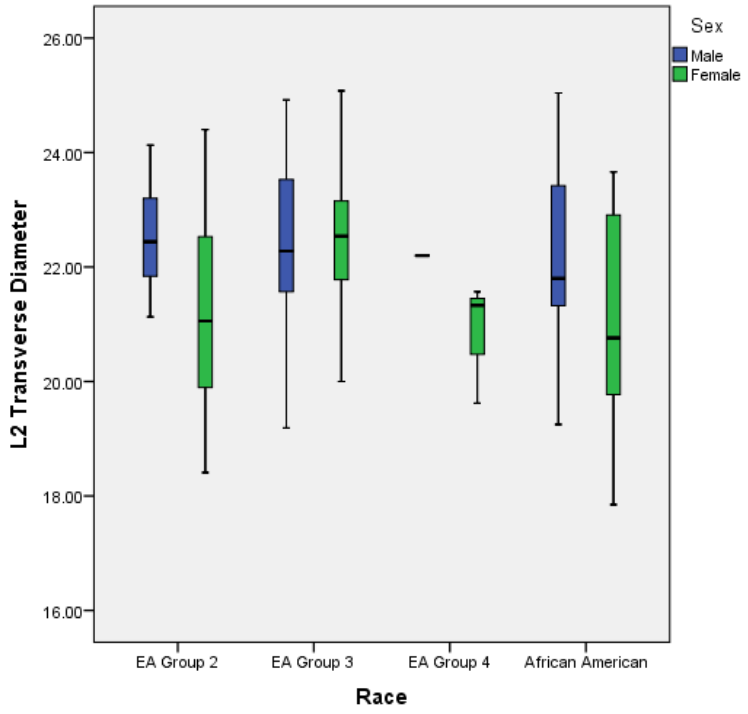

Figure A-47. Late cohort (1946-1984) L2 transverse diameter among race/sex groups.

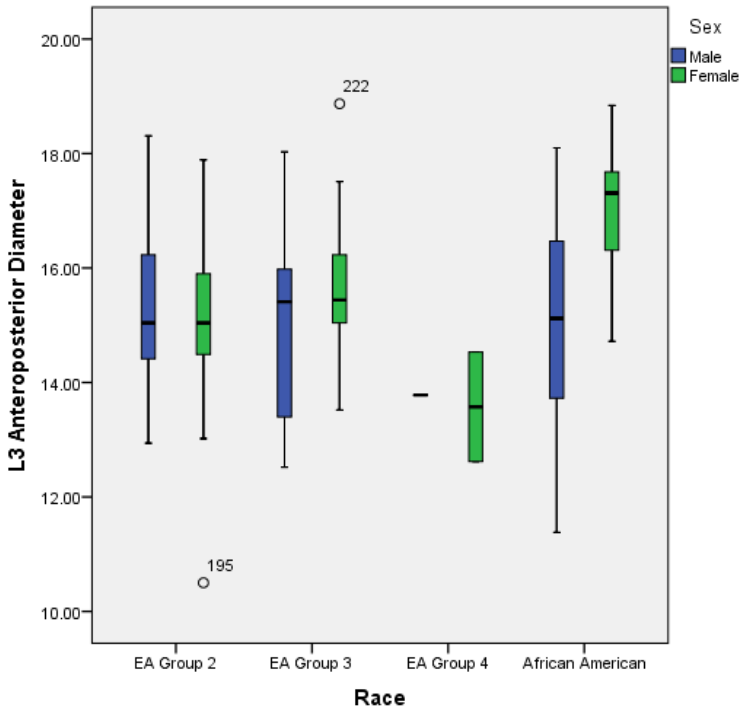

Figure A-48. Late cohort (1946-1984) L3 anteroposterior diameter among race/sex groups.

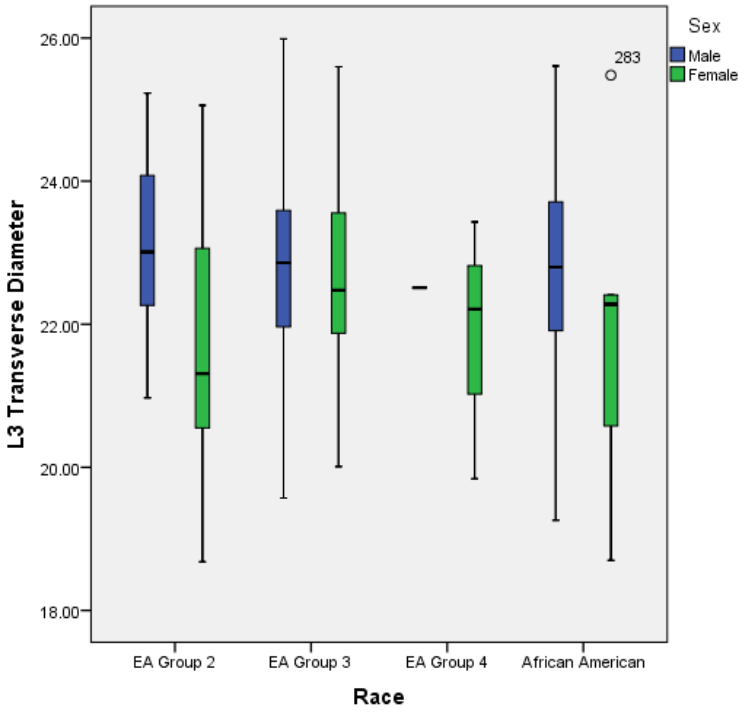

Figure A-49. Late cohort (1946-1984) L3 transverse diameter among race/sex groups.

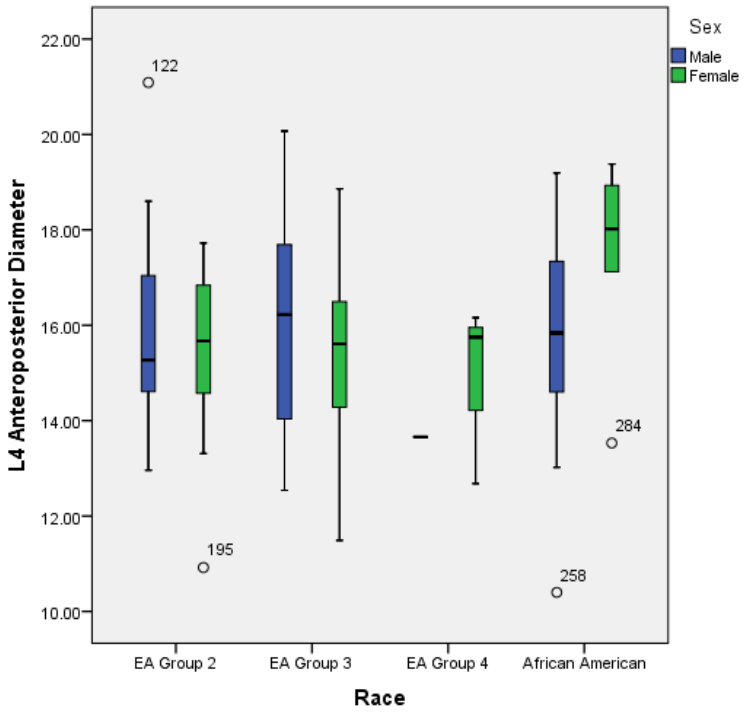

Figure A-50. Late cohort (1946-1984) L4 anteroposterior diameter among race/sex groups.

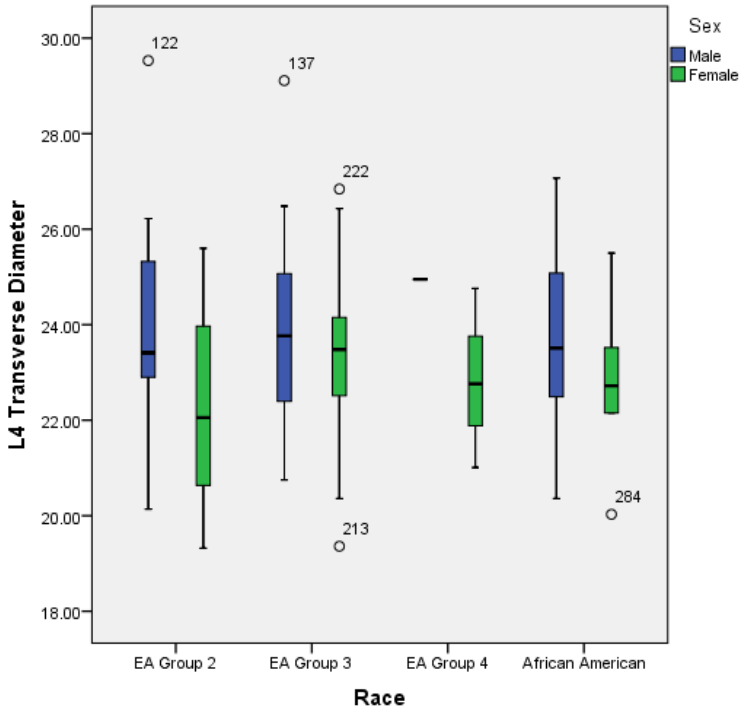

Figure A-51. Late cohort (1946-1984) L4 transverse diameter among race/sex groups.

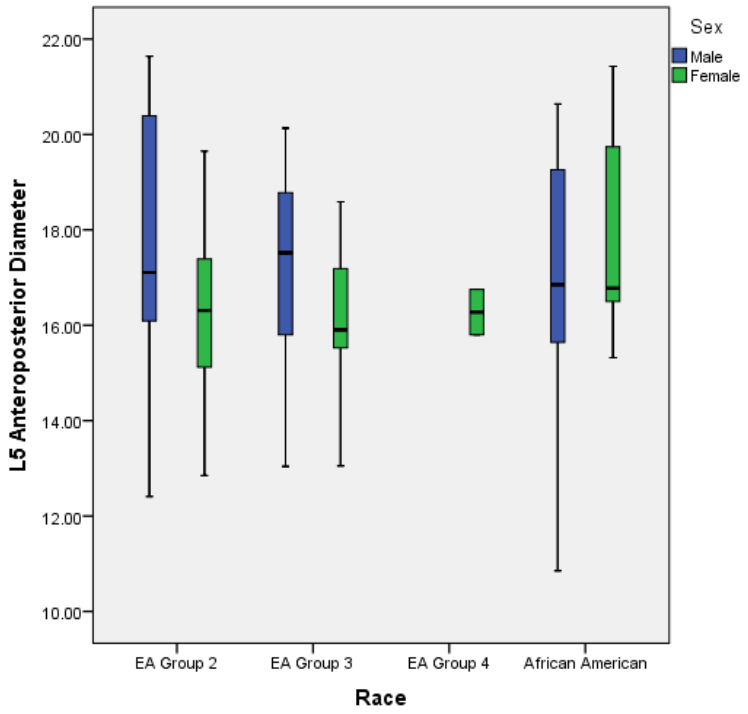

Figure A-52. Late cohort (1946-1984) L5 anteroposterior diameter among race/sex groups.

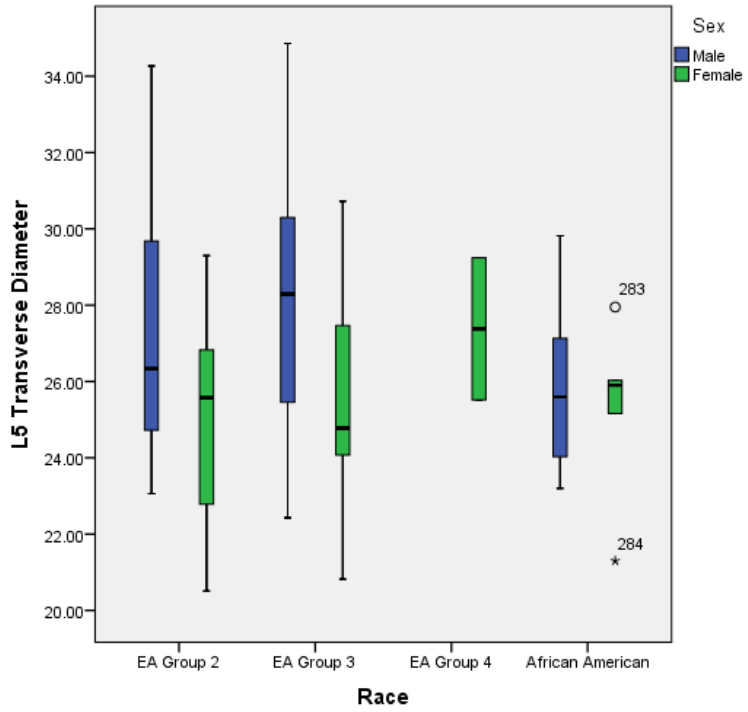

Figure A-53. Late cohort (1946-1984) L5 transverse diameter among race/sex groups.

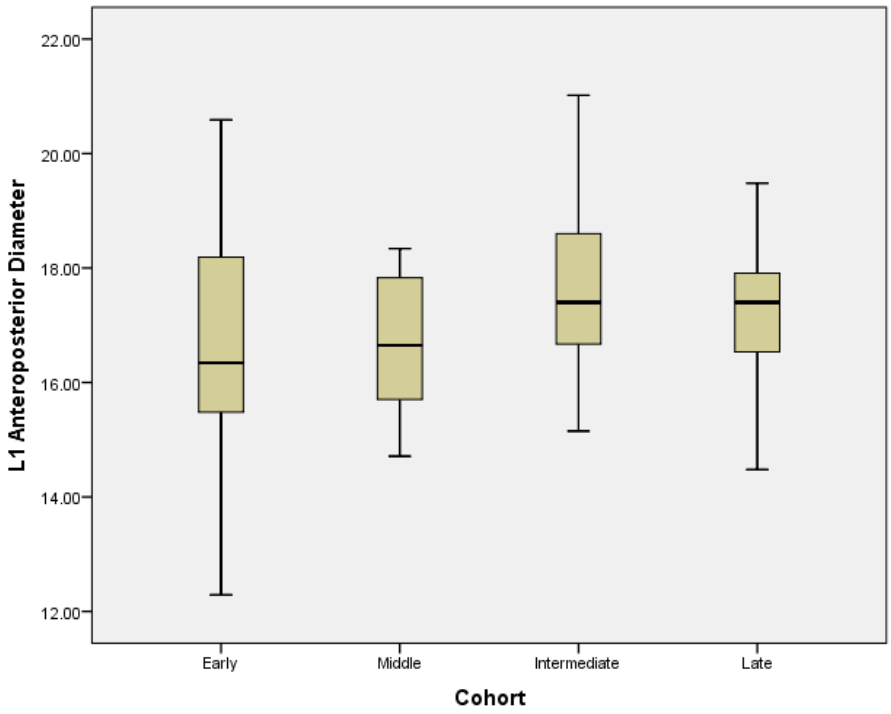

Figure A-54. Group two male L1 anteroposterior diameter across cohorts.

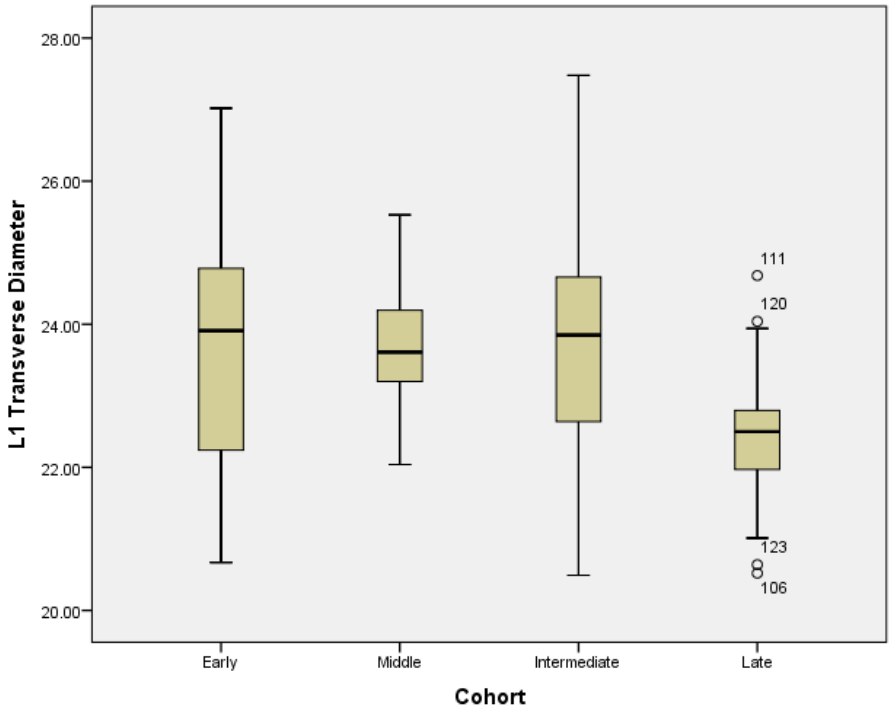

Figure A-55. Group two male L1 transverse diameter across cohorts.

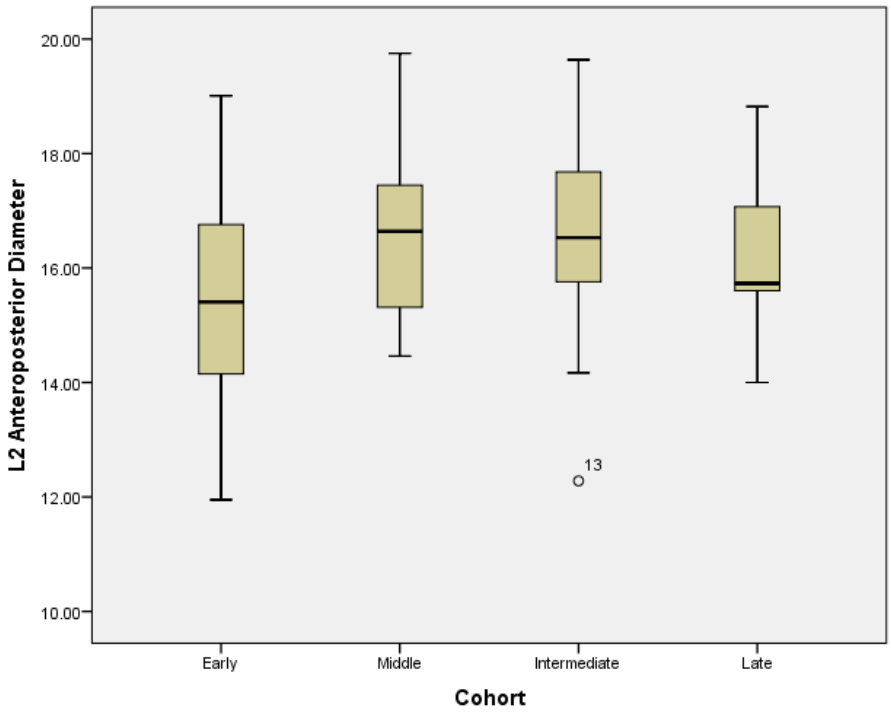

Figure A-56. Group two male L2 anteroposterior diameter across cohorts.

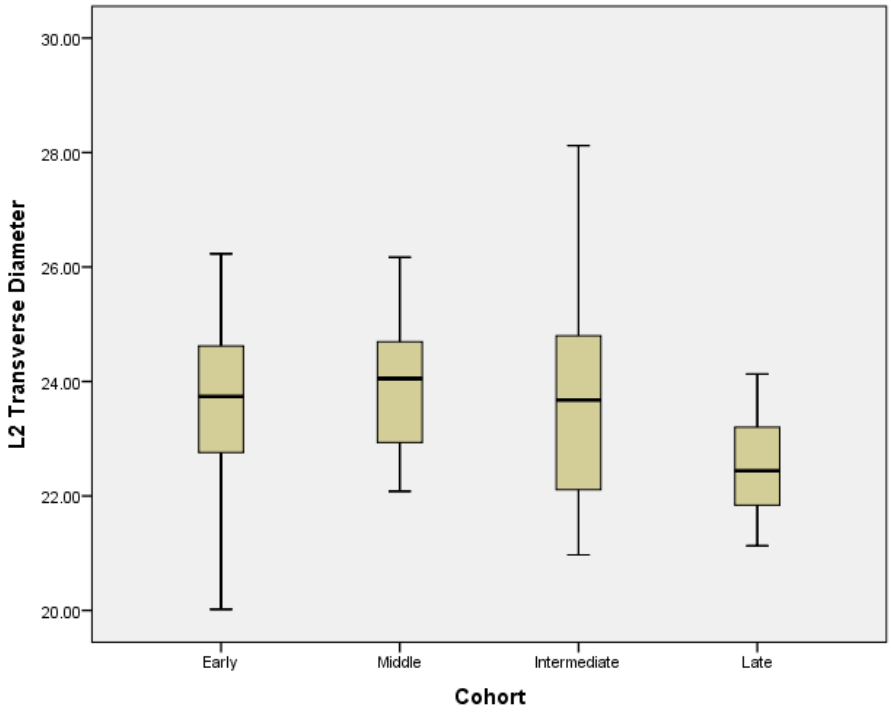

Figure A-57. Group two male L2 transverse diameter across cohorts.

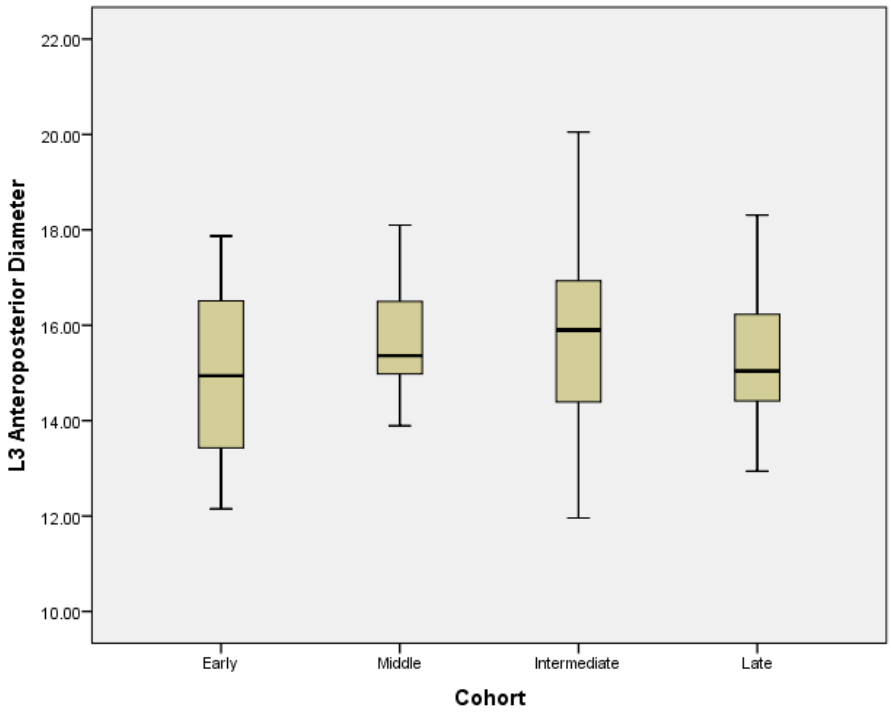

Figure A-58. Group two male L3 anteroposterior diameter across cohorts.

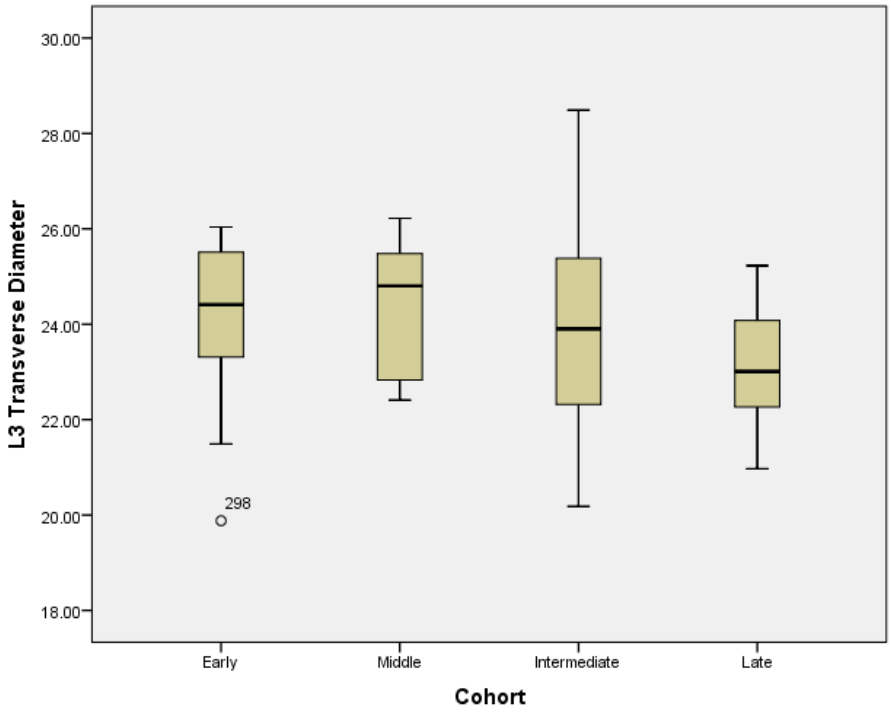

Figure A-59. Group two male L3 transverse diameter across cohorts.

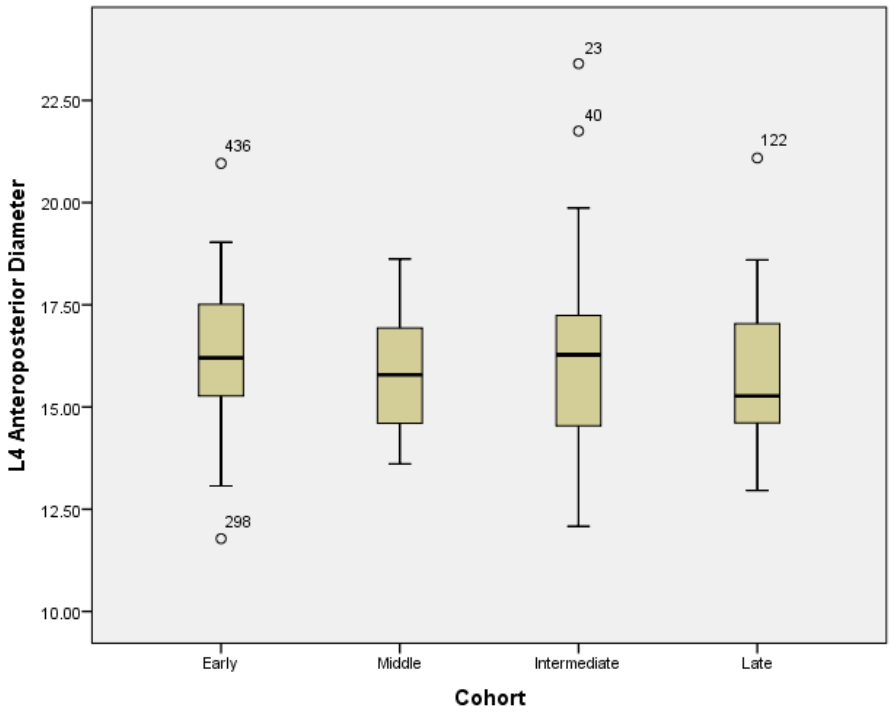

Figure A-60. Group two male L4 anteroposterior diameter across cohorts.

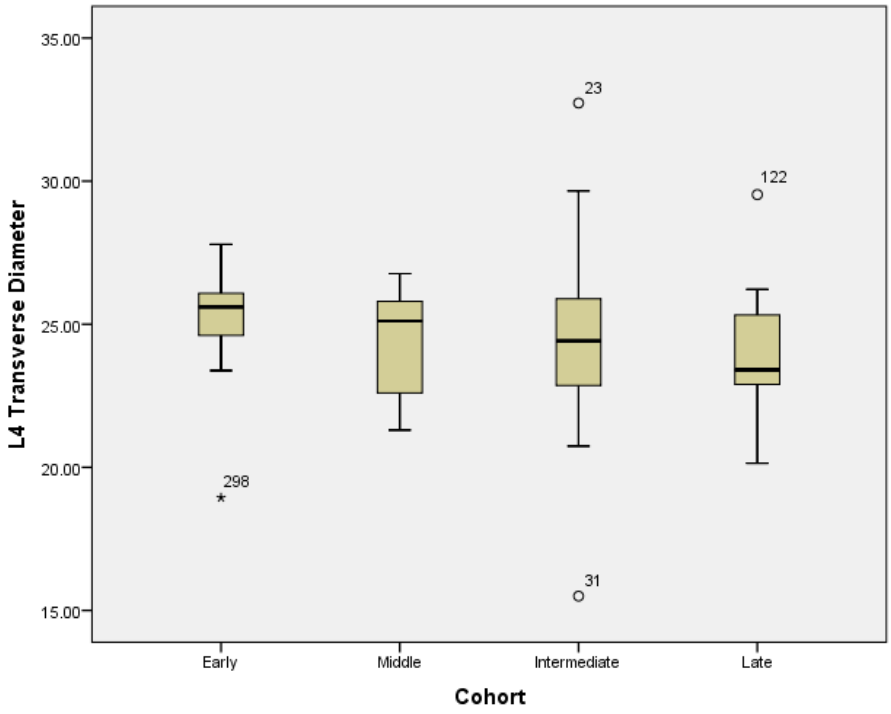

Figure A-61. Group two male L4 transverse diameter across cohorts.

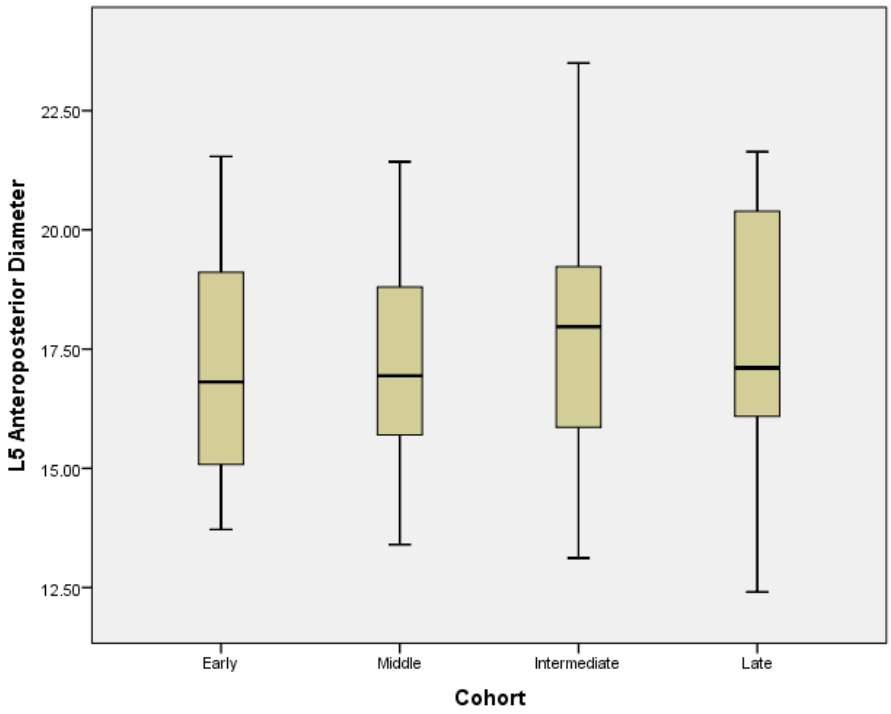

Figure A-62. Group two male L5 anteroposterior diameter across cohorts.

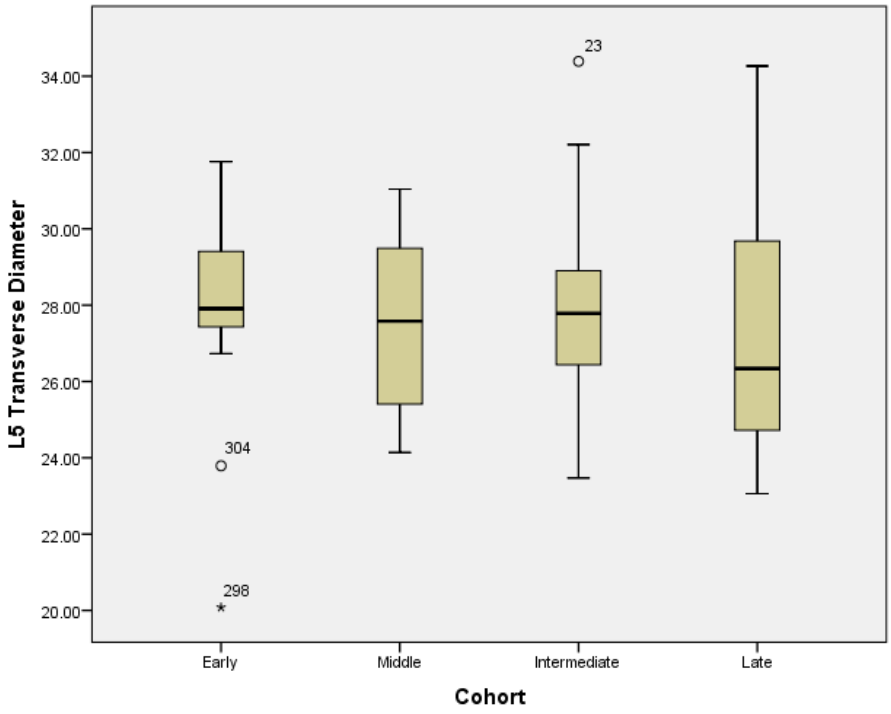

Figure A-63. Group two male L5 transverse diameter across cohorts.

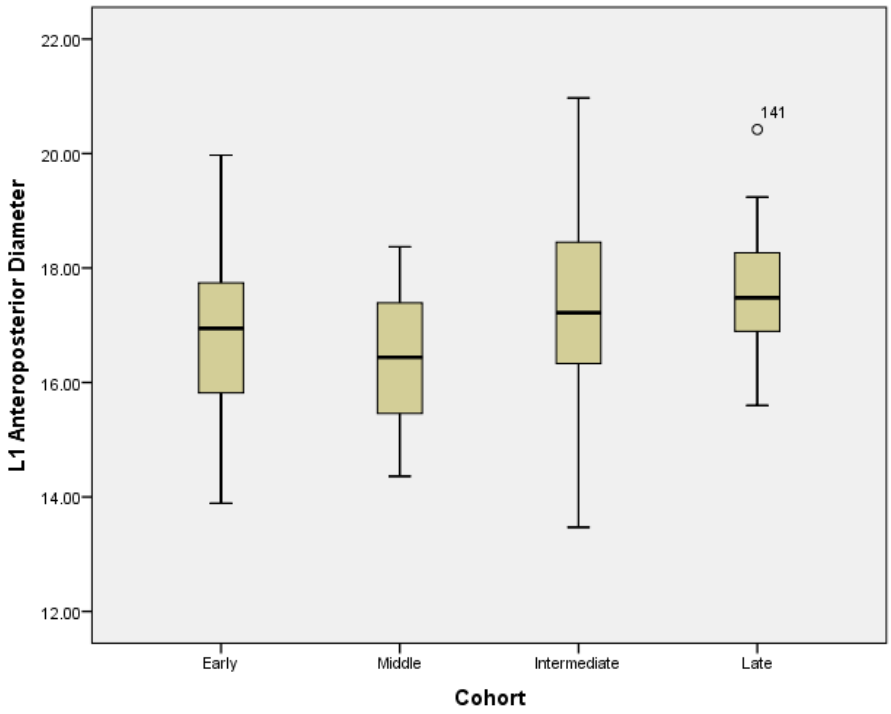

Figure A-64. Group three male L1 anteroposterior diameter across cohorts.

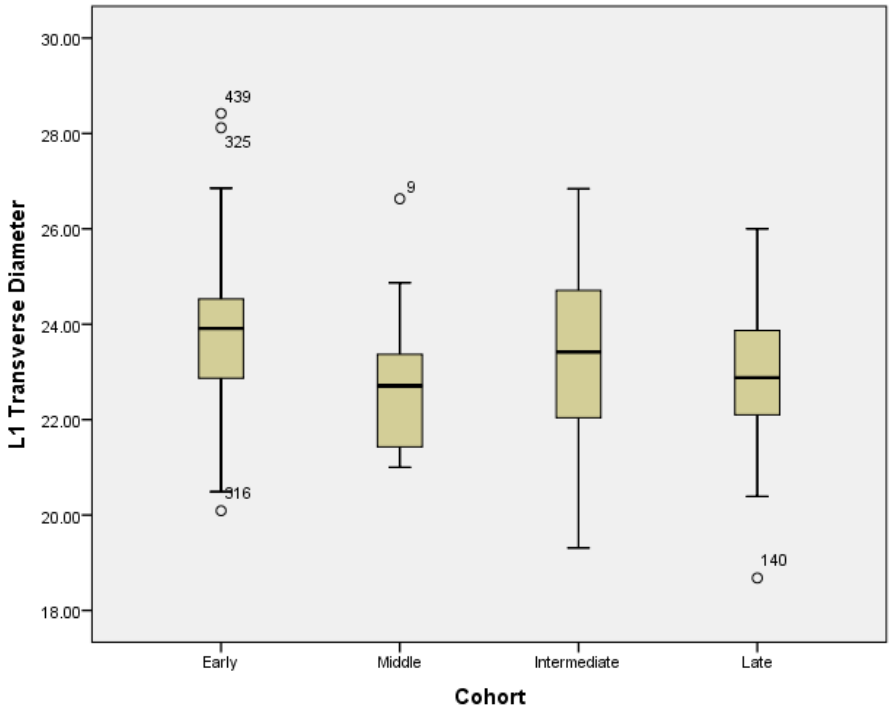

Figure A-65. Group three male L1 transverse diameter across cohorts.

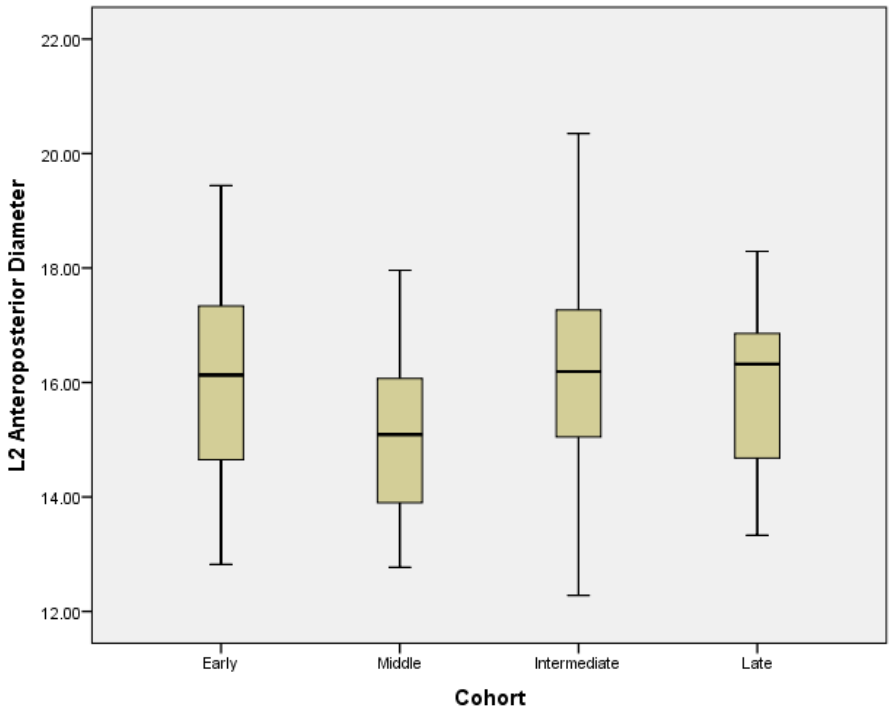

Figure A-66. Group three male L2 anteroposterior diameter across cohorts.

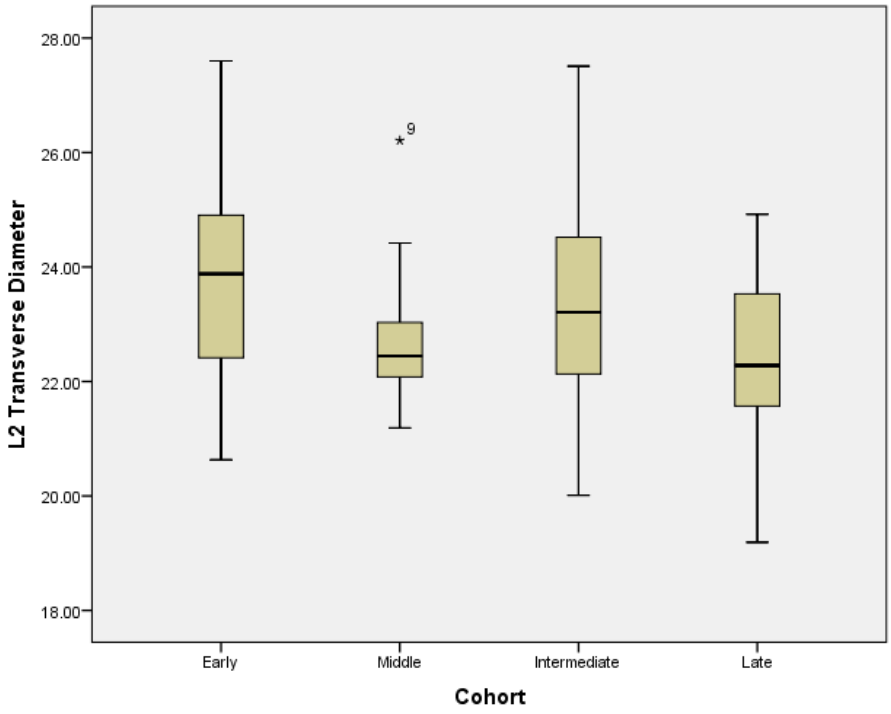

Figure A-67. Group three male L2 transverse diameter across cohorts.

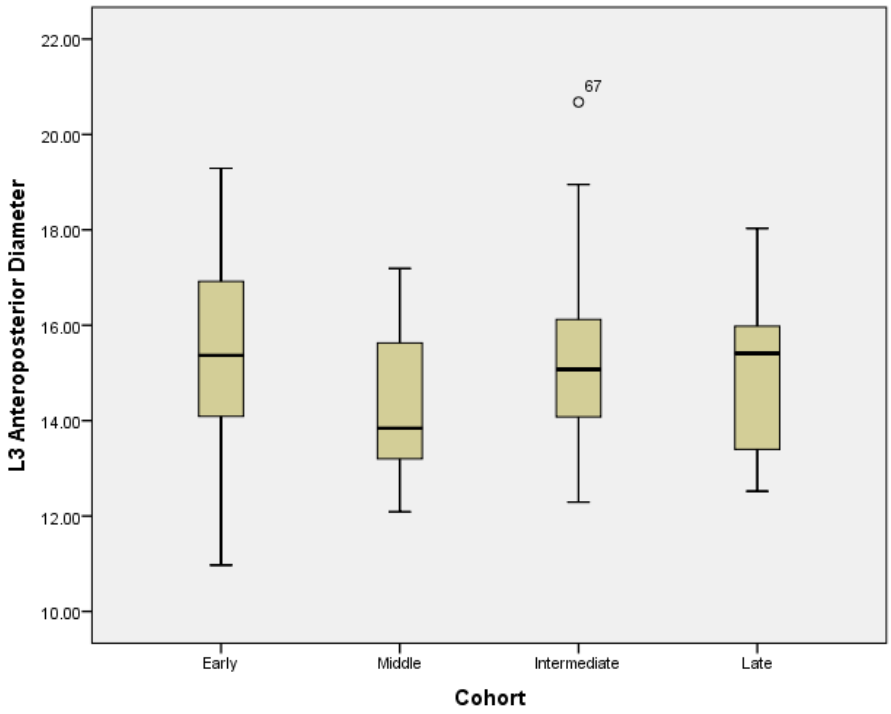

Figure A-68. Group three male L3 anteroposterior diameter across cohorts.

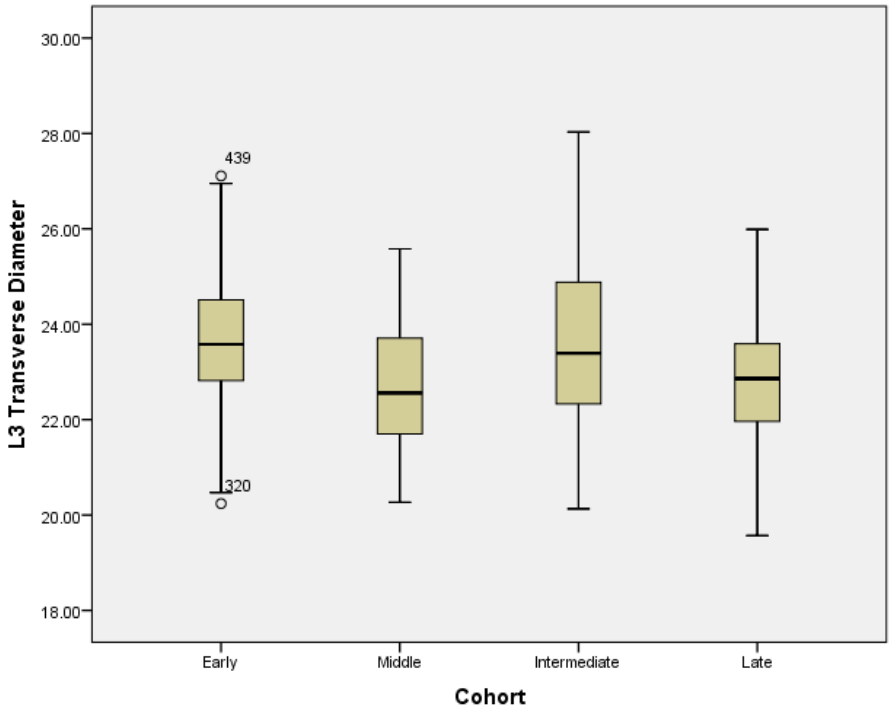

Figure A-69. Group three male L3 transverse diameter across cohorts.

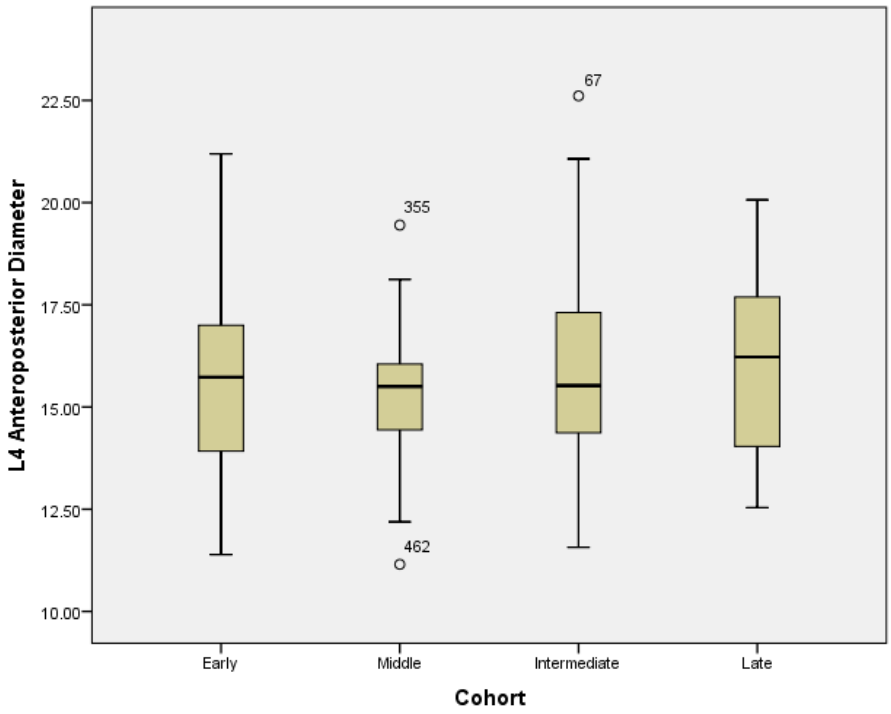

Figure A-70. Group three male L4 anteroposterior diameter across cohorts.

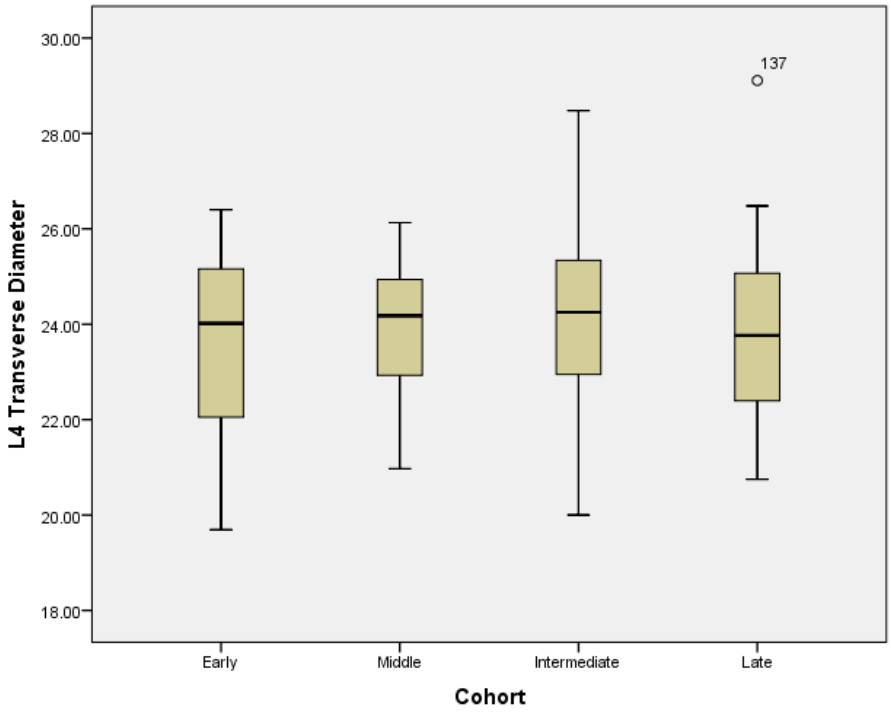

Figure A-71. Group three male L4 transverse diameter across cohorts.

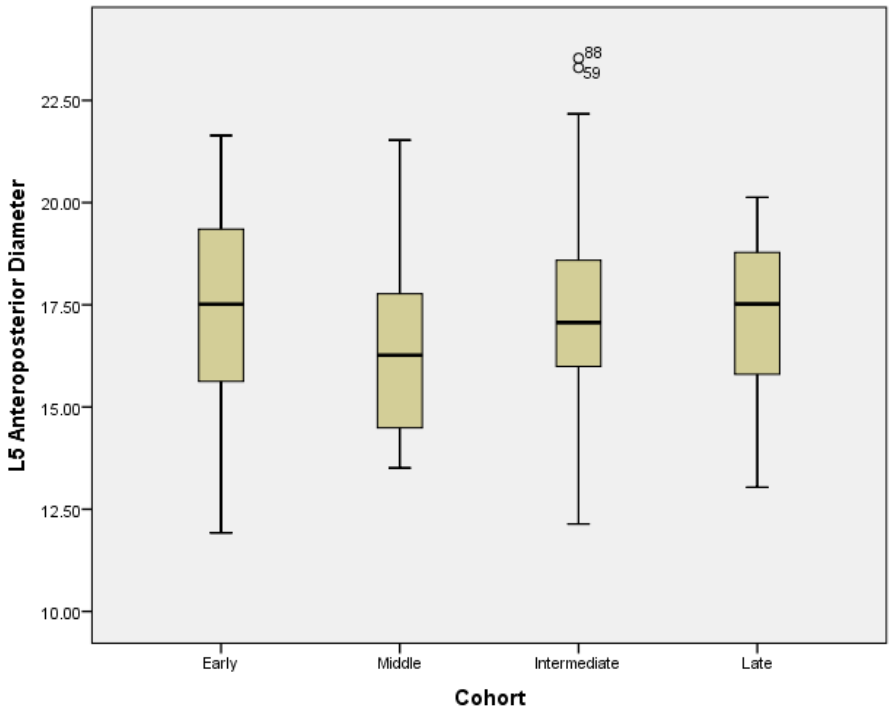

Figure A-72. Group three male L5 anteroposterior diameter across cohorts.

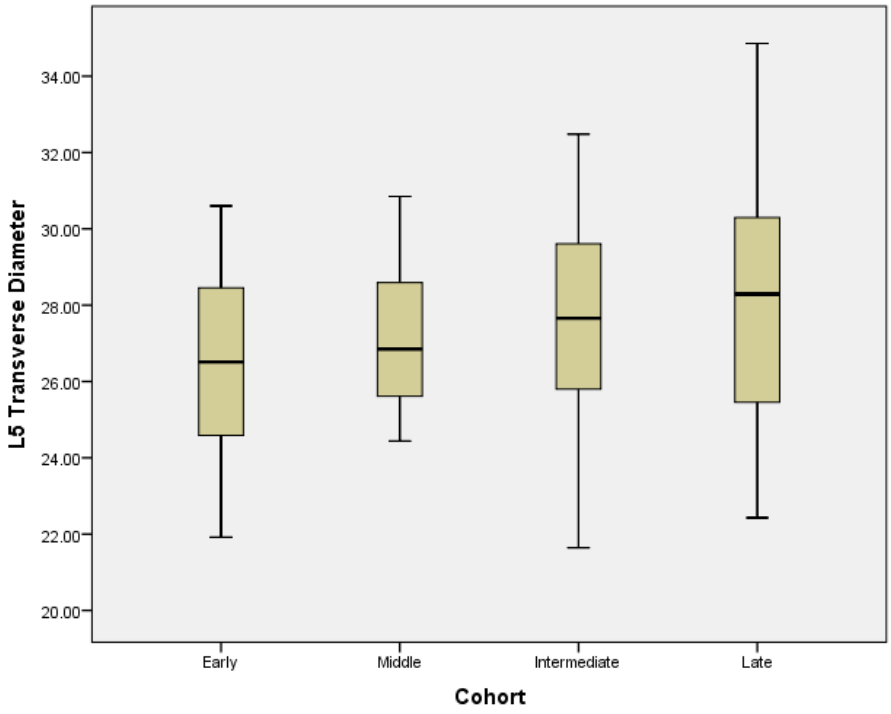

Figure A-73. Group three male L5 transverse diameter across cohorts.

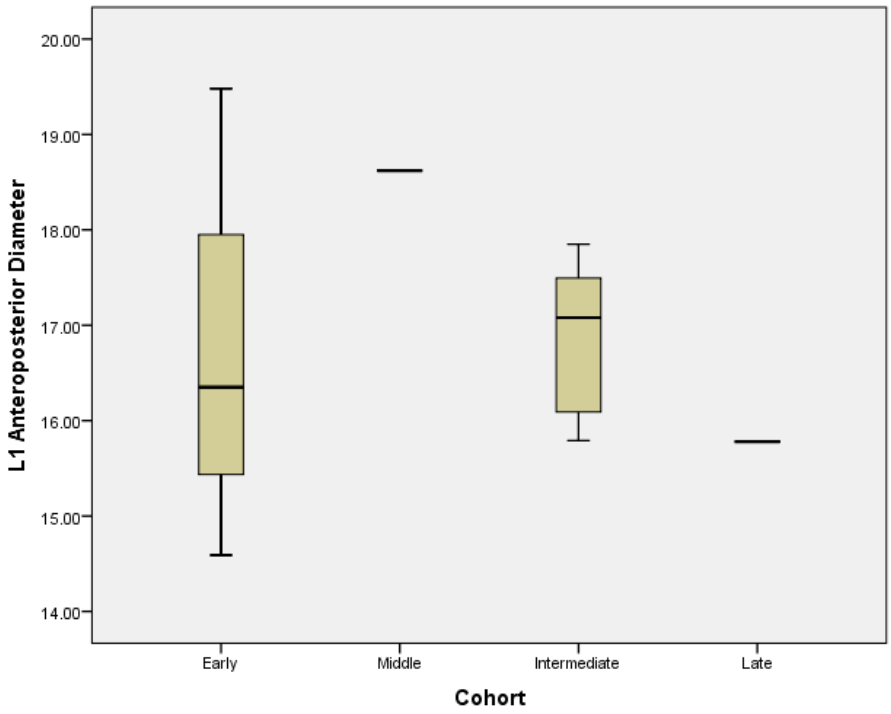

Figure A-74. Group four male L1 anteroposterior diameter across cohorts.

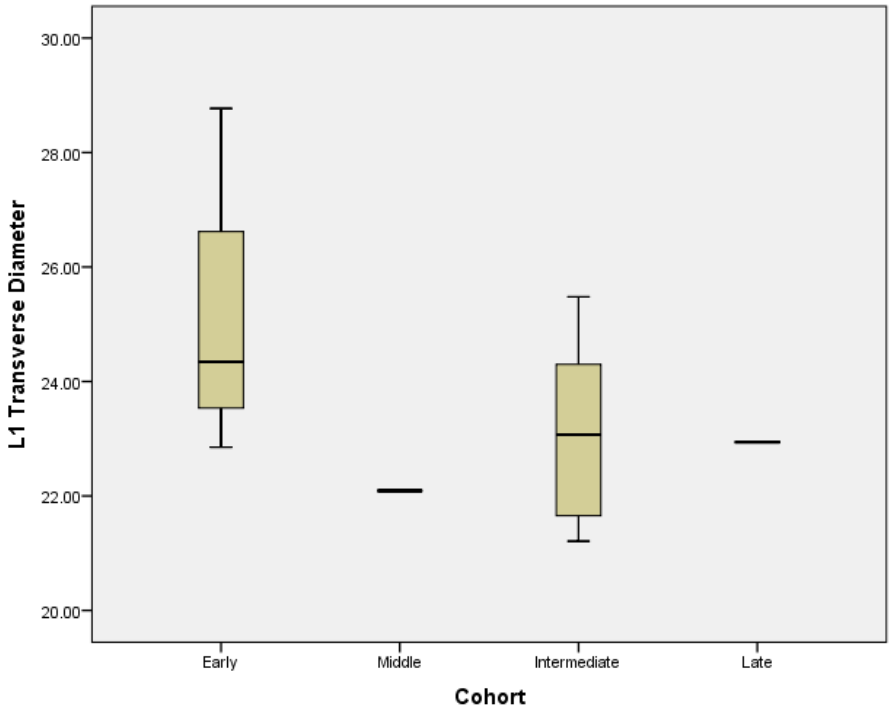

Figure A-75. Group four male L1 transverse diameter across cohorts.

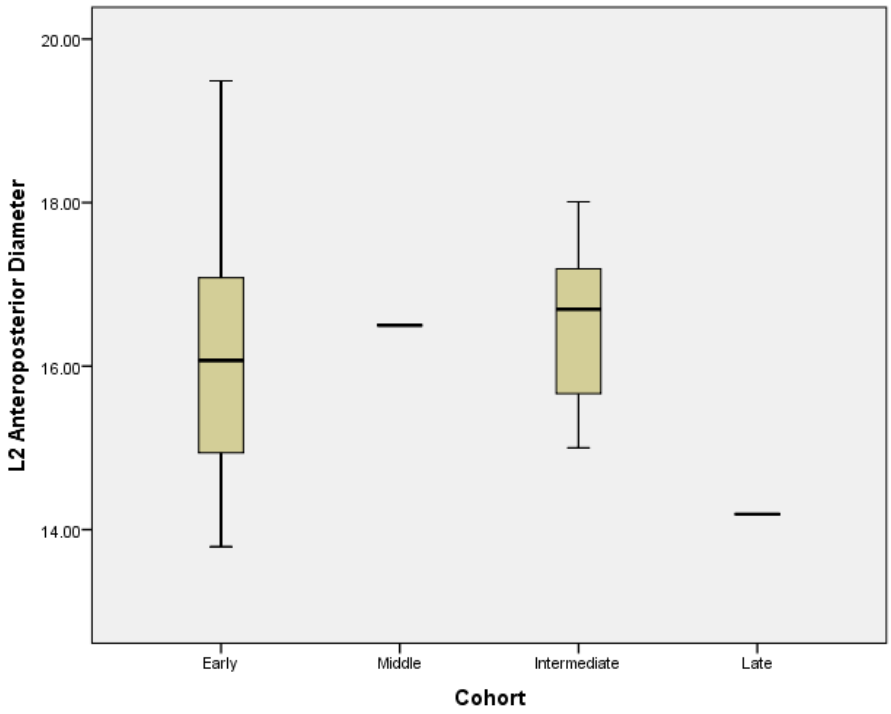

Figure A-76. Group four male L2 anteroposterior diameter across cohorts.

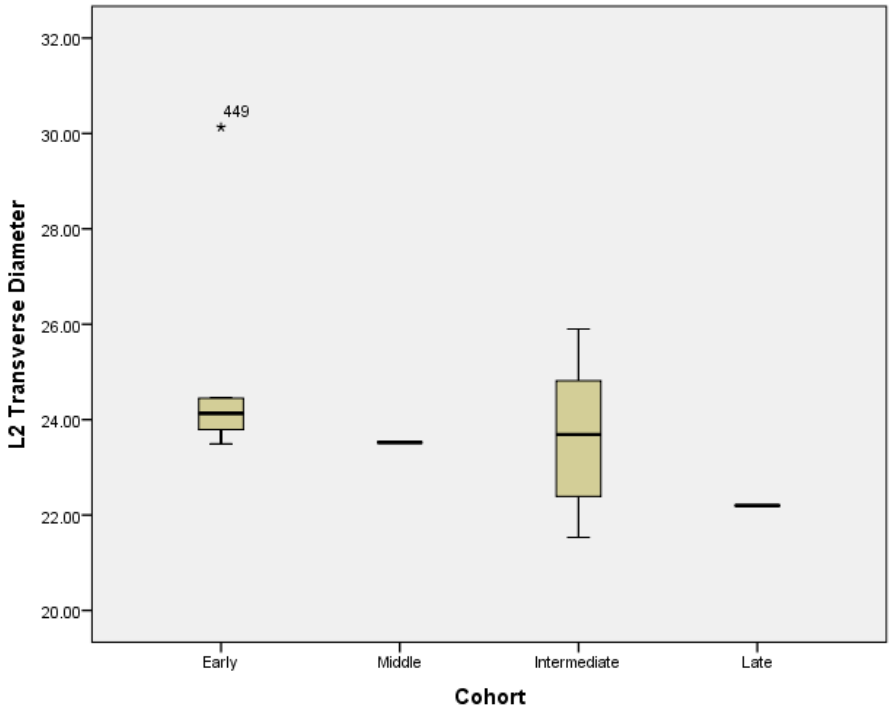

Figure A-77. Group four male L2 transverse diameter across cohorts.

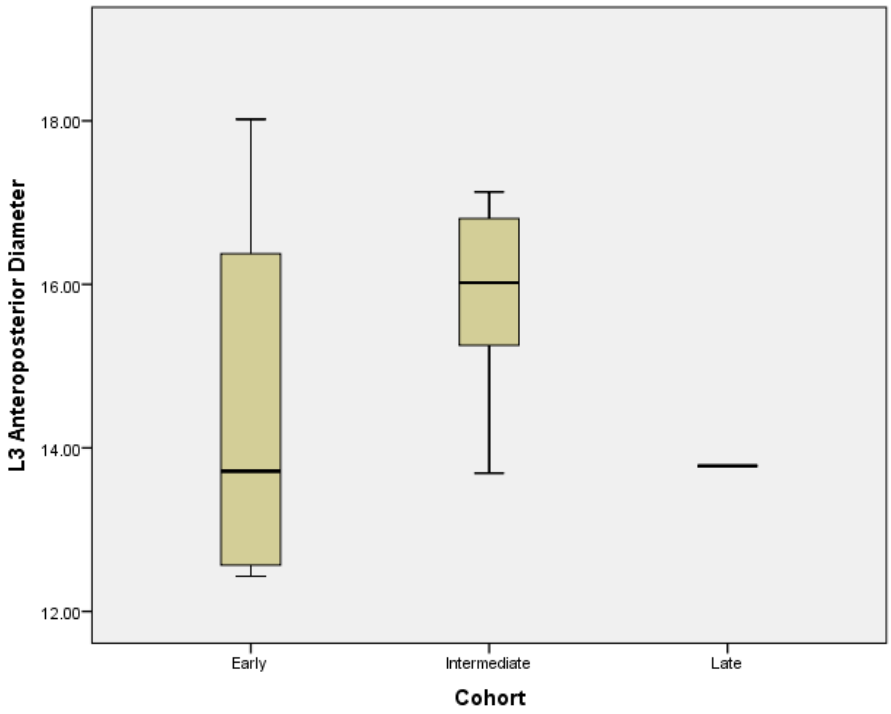

Figure A-78. Group four male L3 anteroposterior diameter across cohorts.

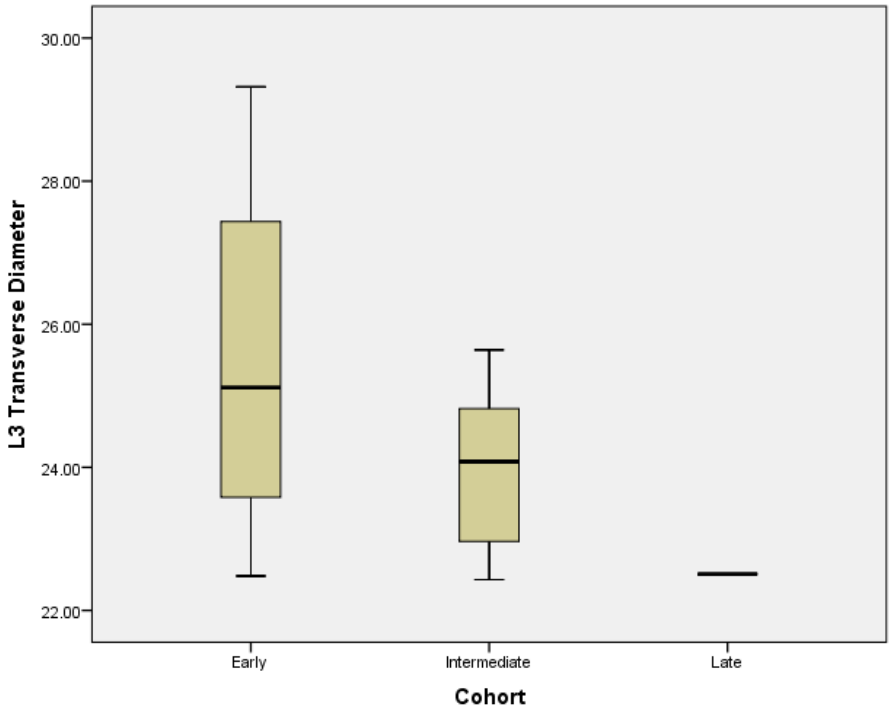

Figure A-79. Group four male L3 transverse diameter across cohorts.

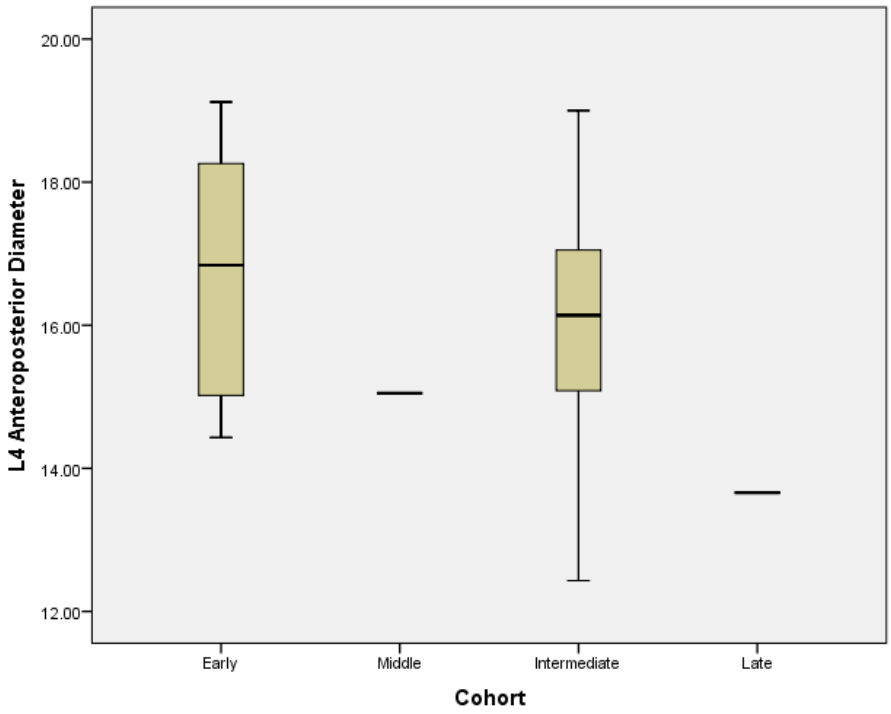

Figure A-80. Group four male L4 anteroposterior diameter across cohorts.

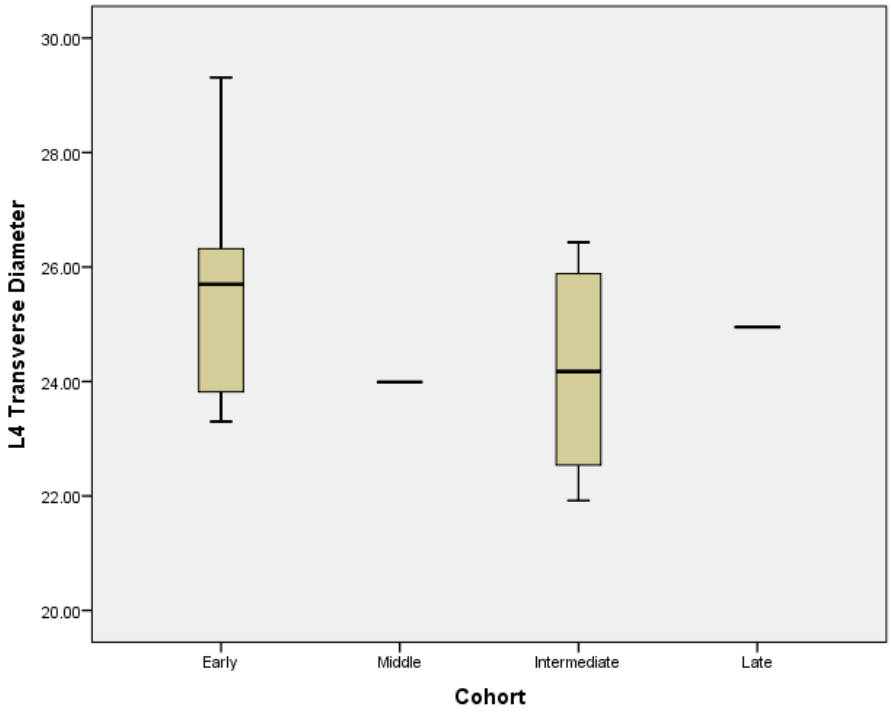

Figure A-81. Group four male L4 transverse diameter across cohorts.

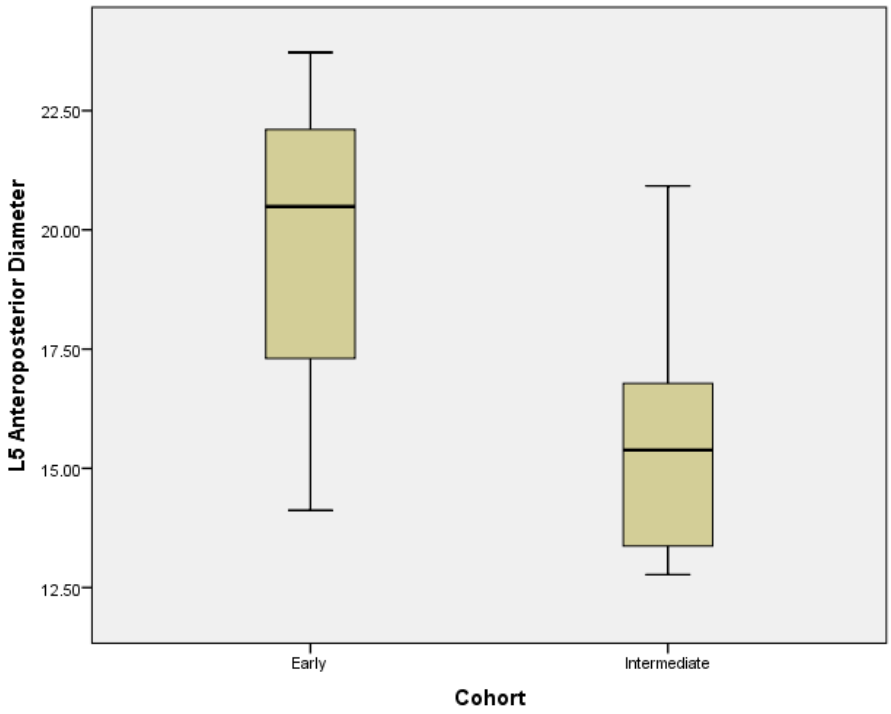

Figure A-82. Group four male L5 anteroposterior diameter across cohorts.

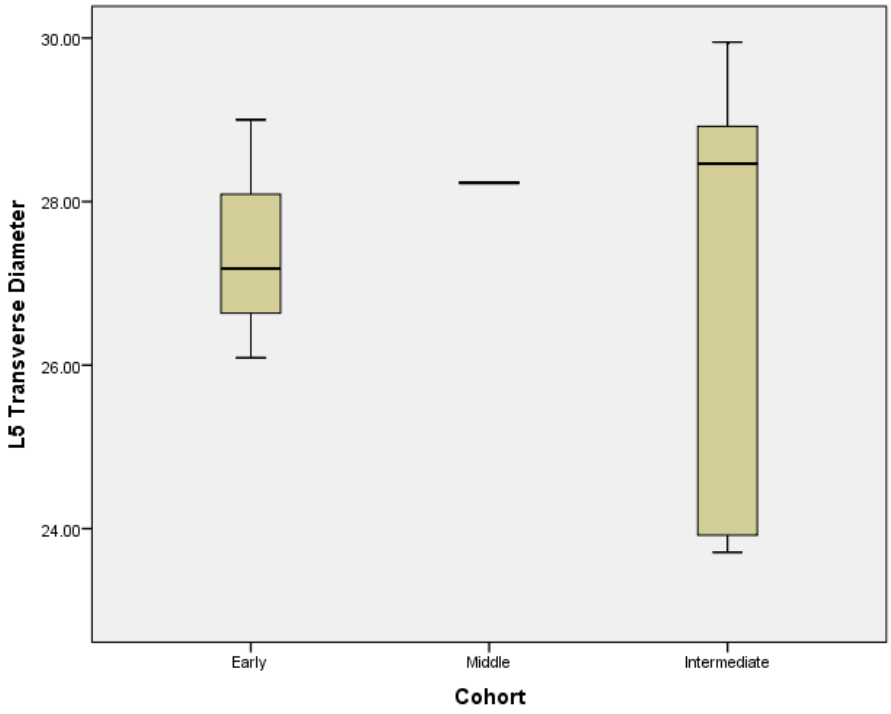

Figure A-83. Group four male L5 transverse diameter across cohorts.

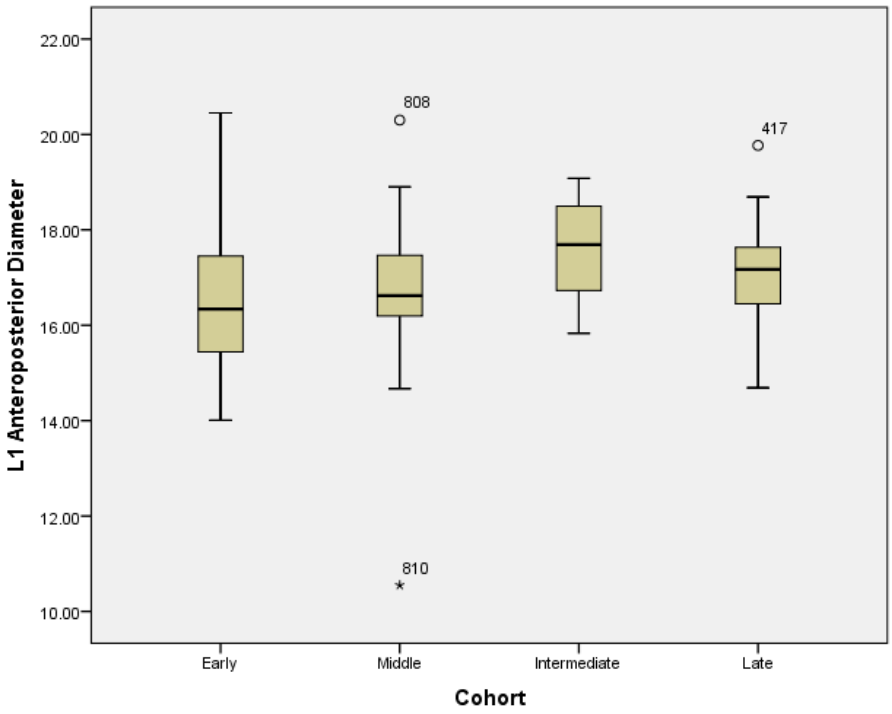

Figure A-84. African American male L1 anteroposterior diameter across cohorts.

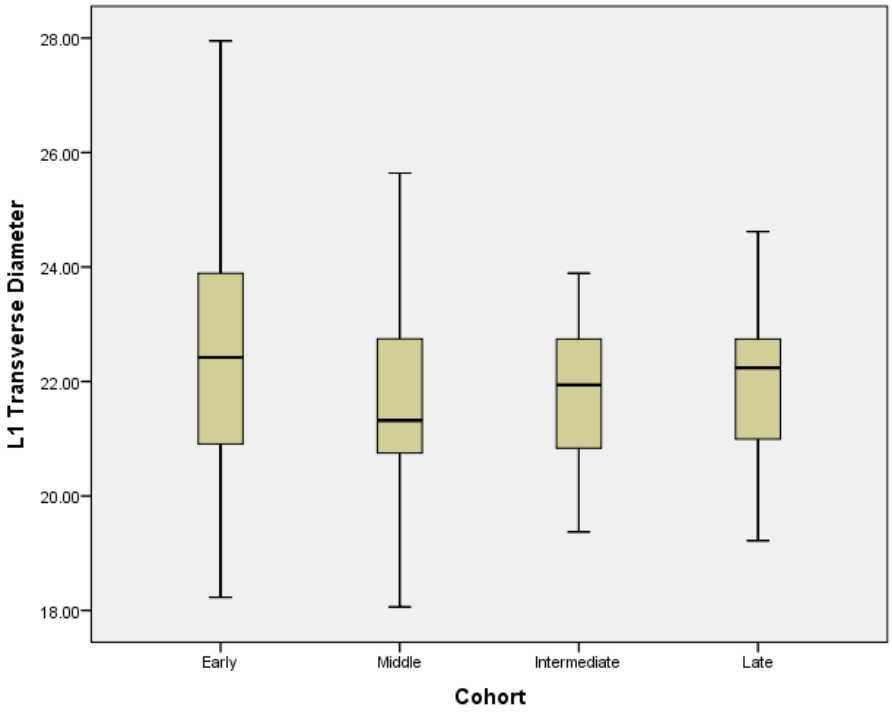

Figure A-85. African American male L1 transverse diameter across cohorts.

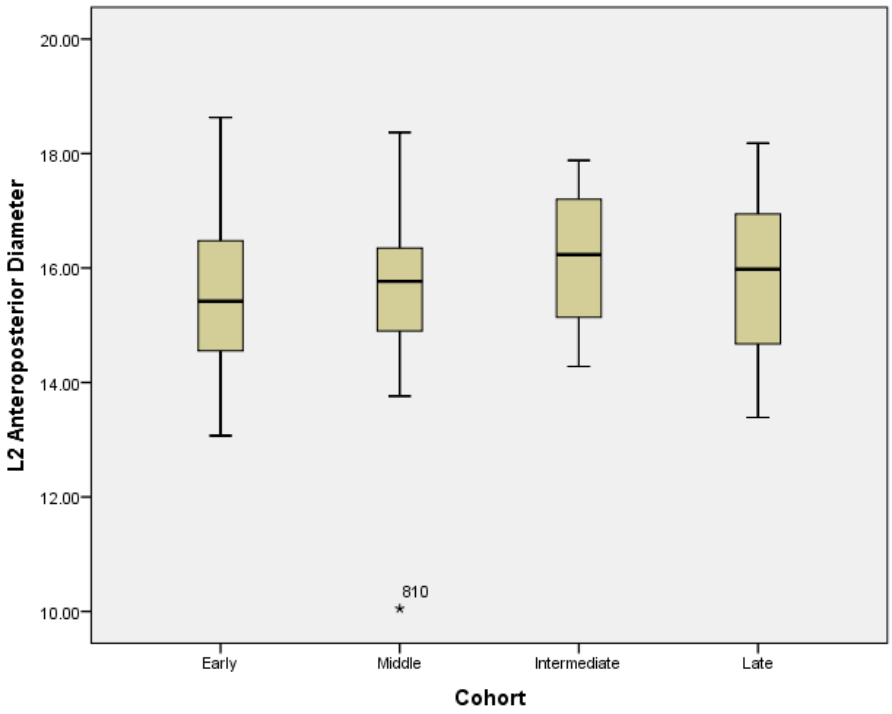

Figure A-86. African American male L2 anteroposterior diameter across cohorts.

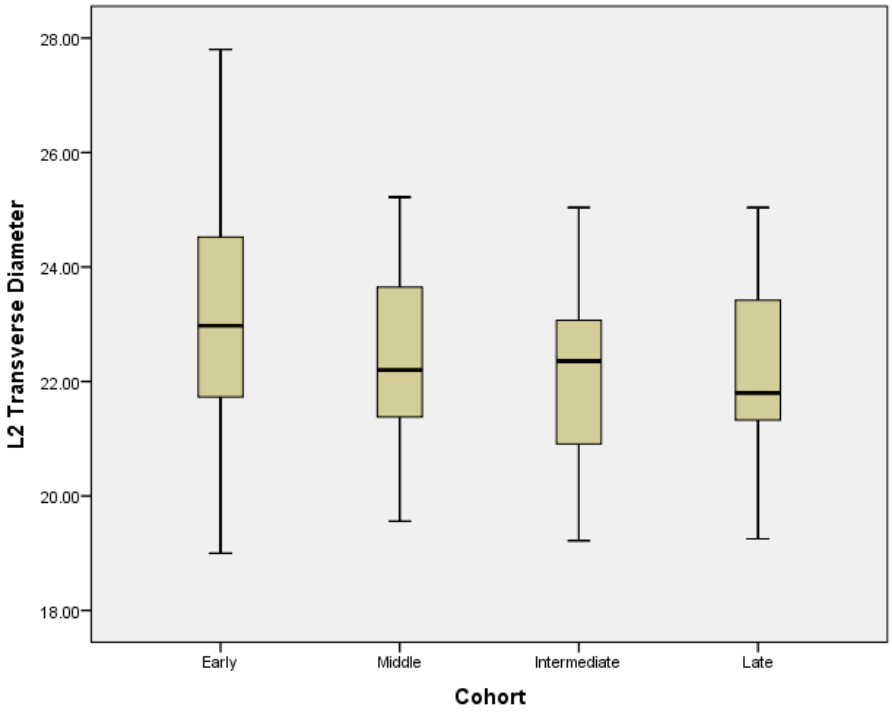

Figure A-87. African American male L2 transverse diameter across cohorts.

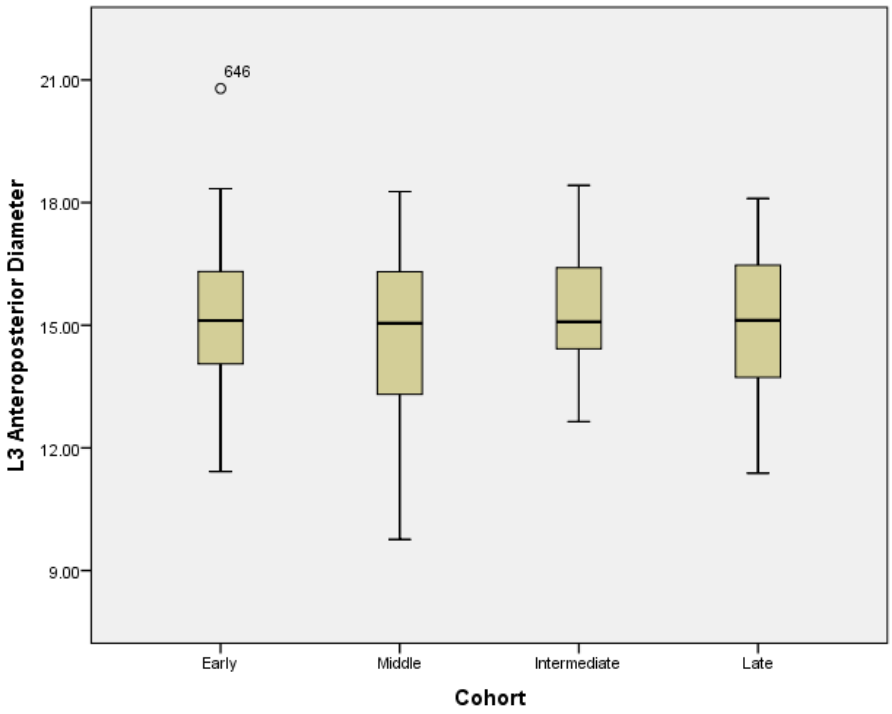

Figure A-88. African American male L3 anteroposterior diameter across cohorts.

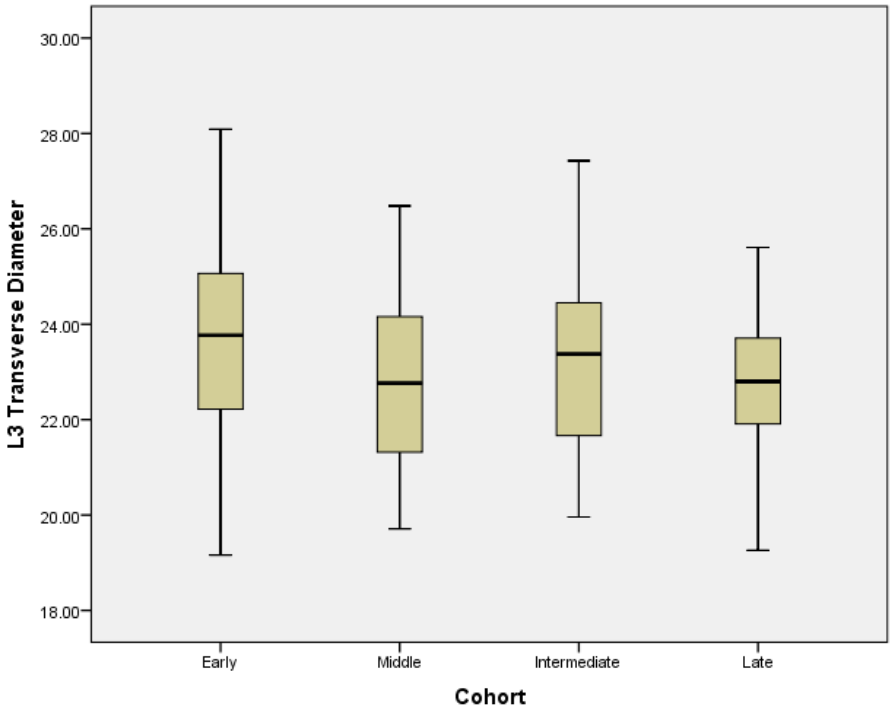

Figure A-89. African American male L3 transverse diameter across cohorts.

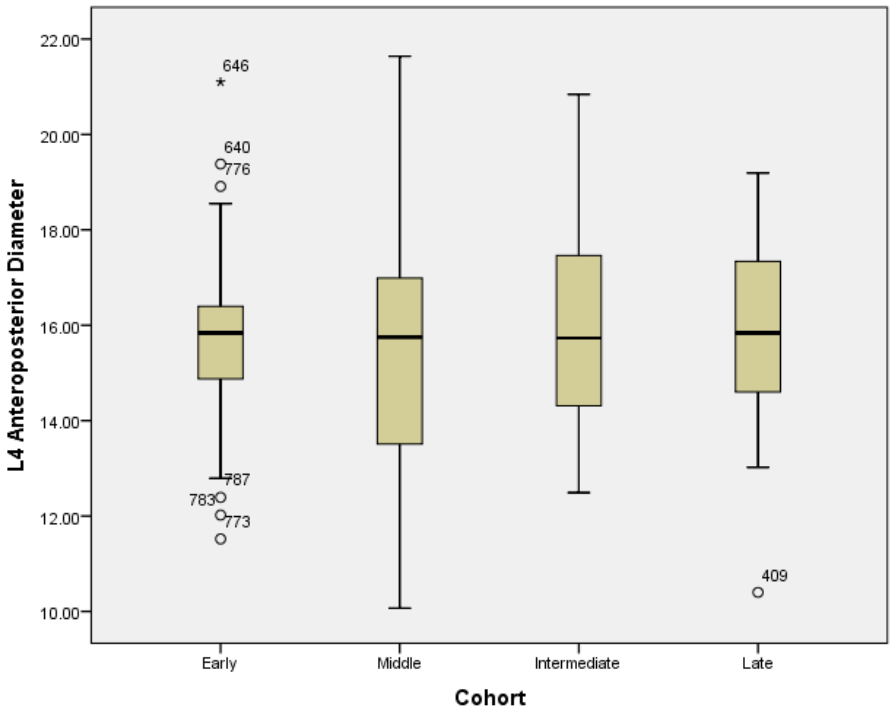

Figure A-90. African American male L4 anteroposterior diameter across cohorts.

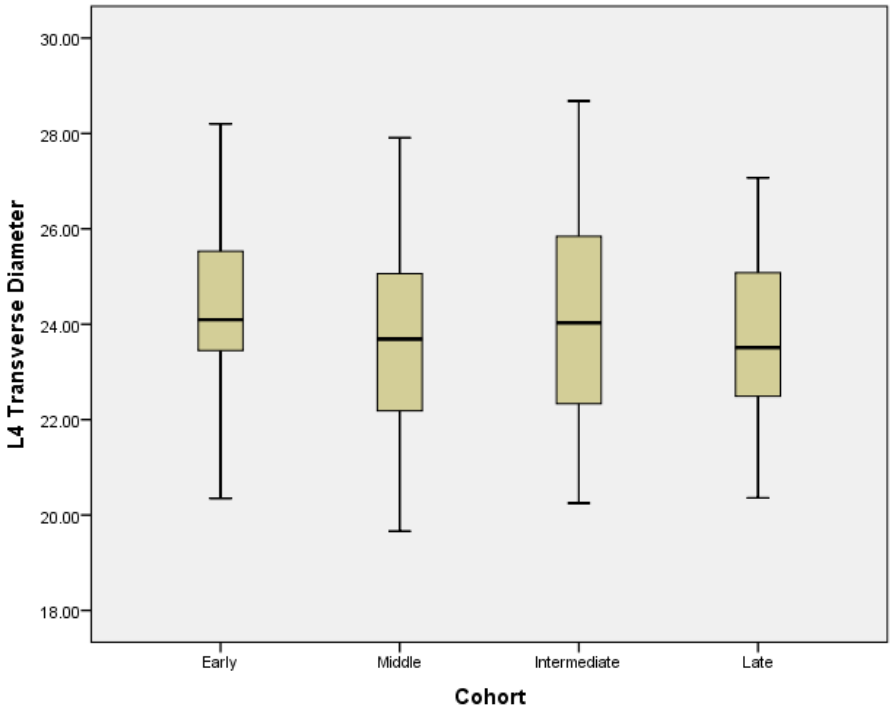

Figure A-91. African American male L4 transverse diameter across cohorts.

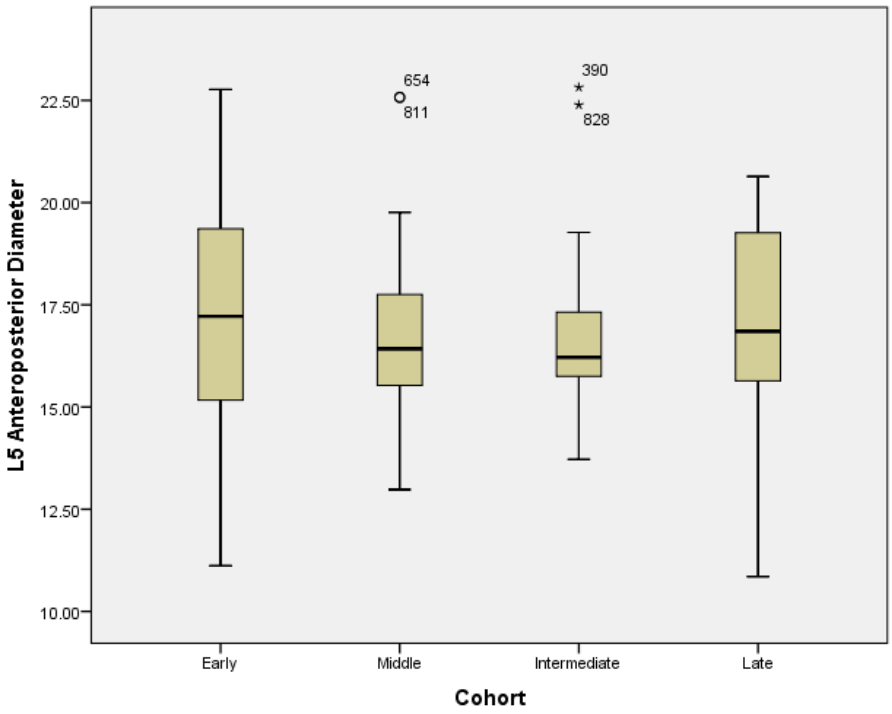

Figure A-92. African American male L5 anteroposterior diameter across cohorts.

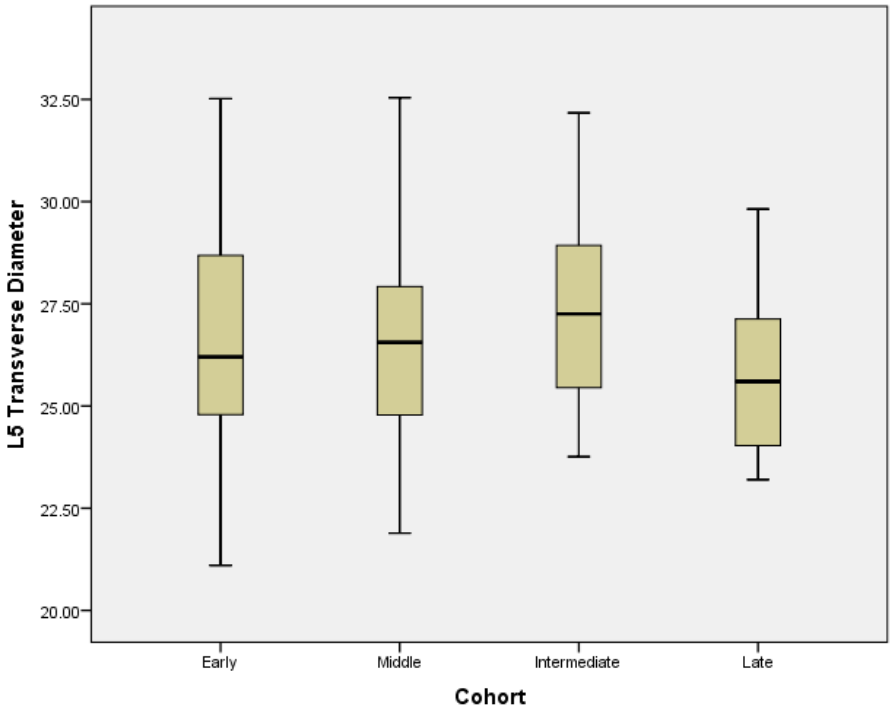

Figure A-93. African American male L5 transverse diameter across cohorts.

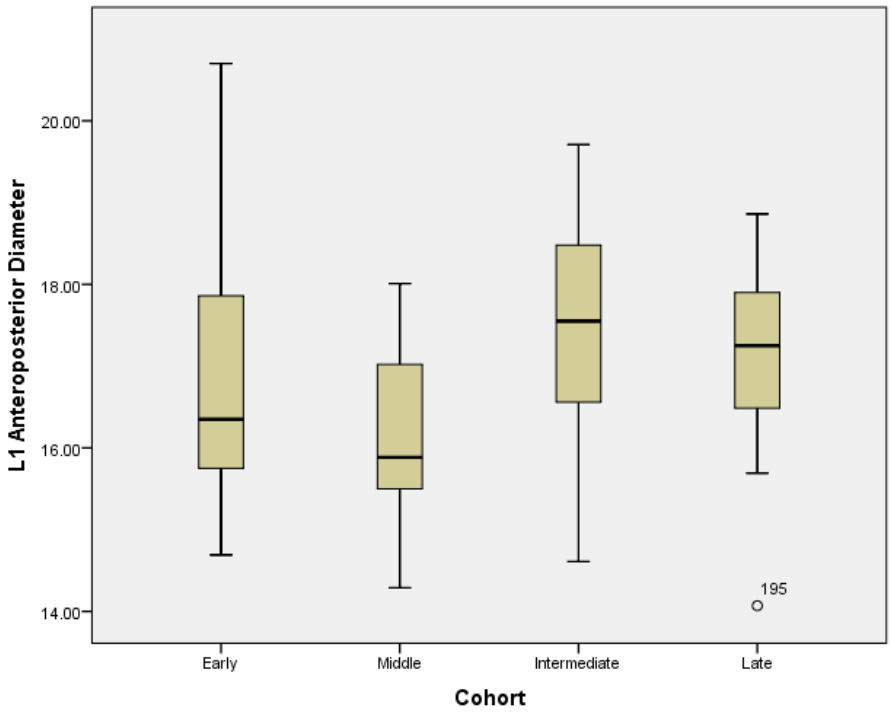

Figure A-94. Group two female L1 anteroposterior diameter across cohorts.

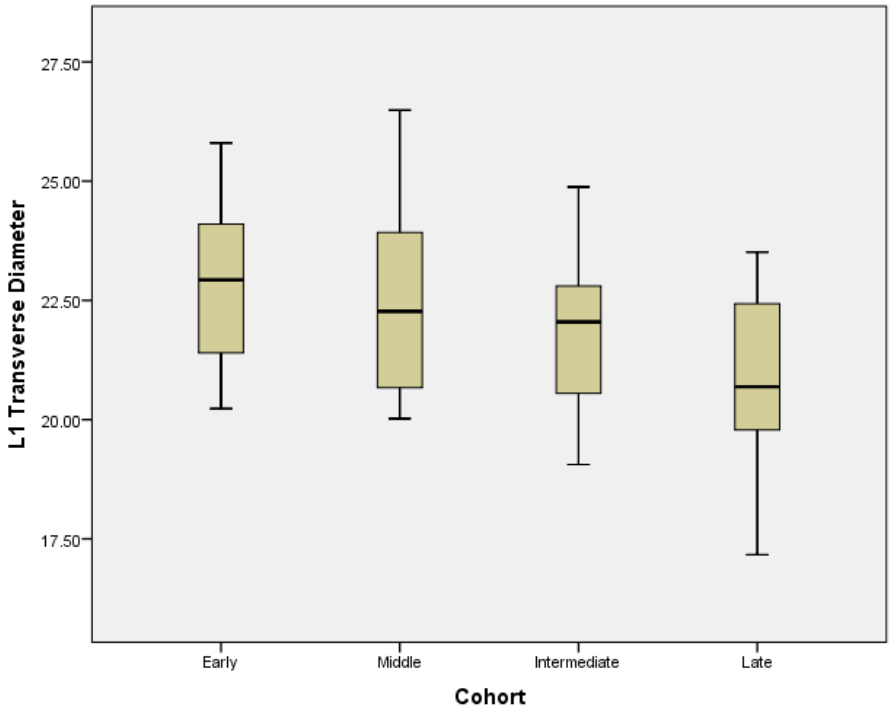

Figure A-95. Group two female L1 transverse diameter across cohorts.

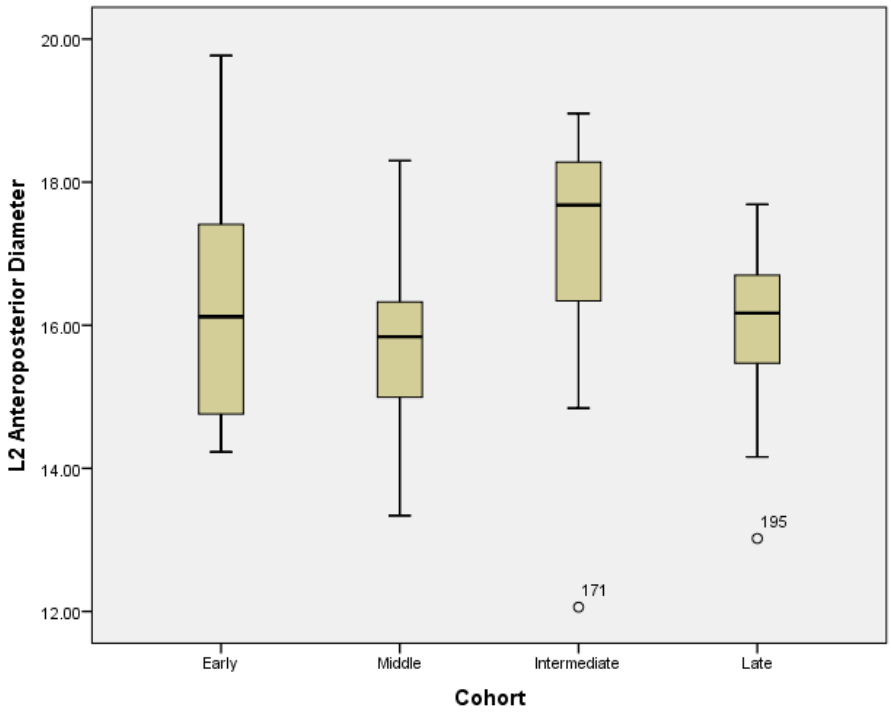

Figure A-96. Group two female L2 anteroposterior diameter across cohorts.

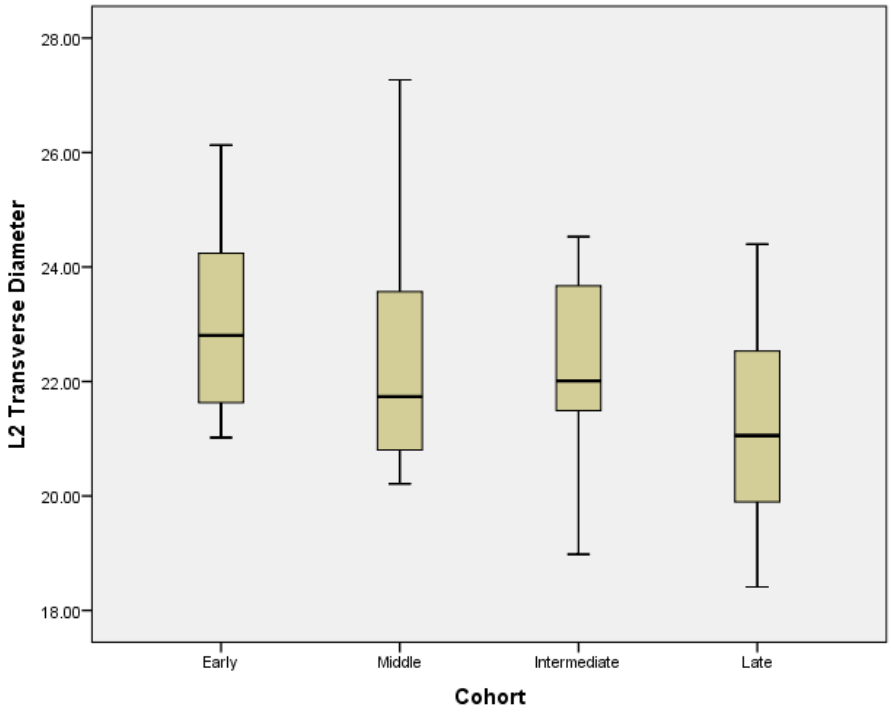

Figure A-97. Group two female L2 transverse diameter across cohorts.

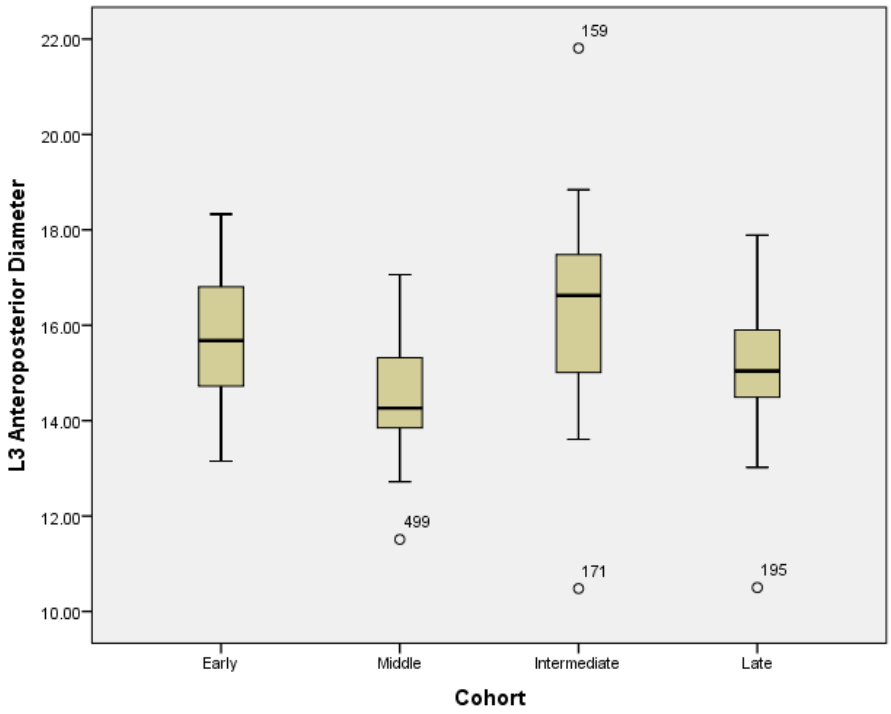

Figure A-98. Group two female L3 anteroposterior diameter across cohorts.

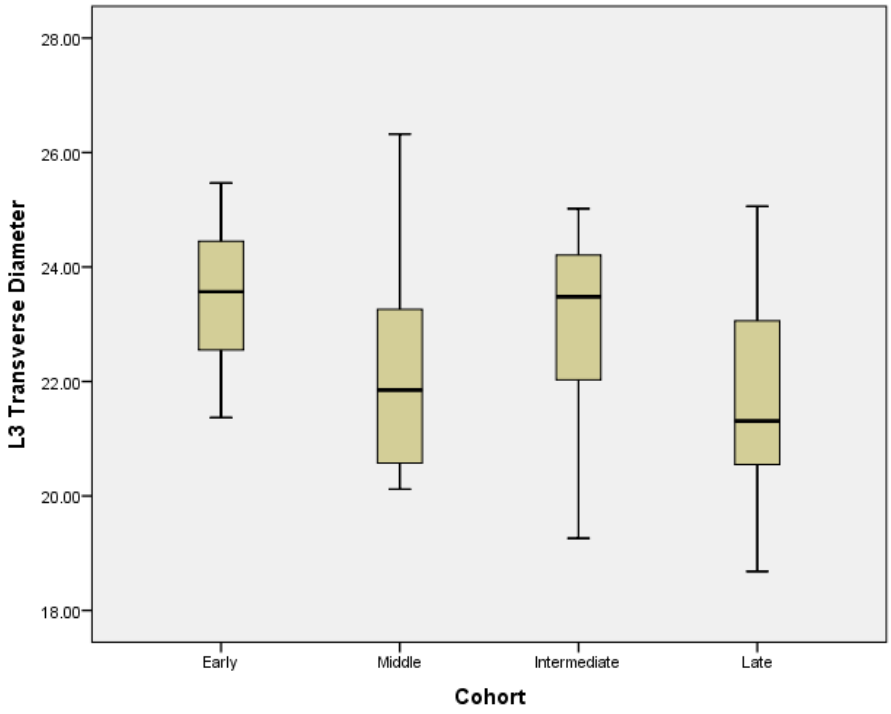

Figure A-99. Group two female L3 transverse diameter across cohorts.

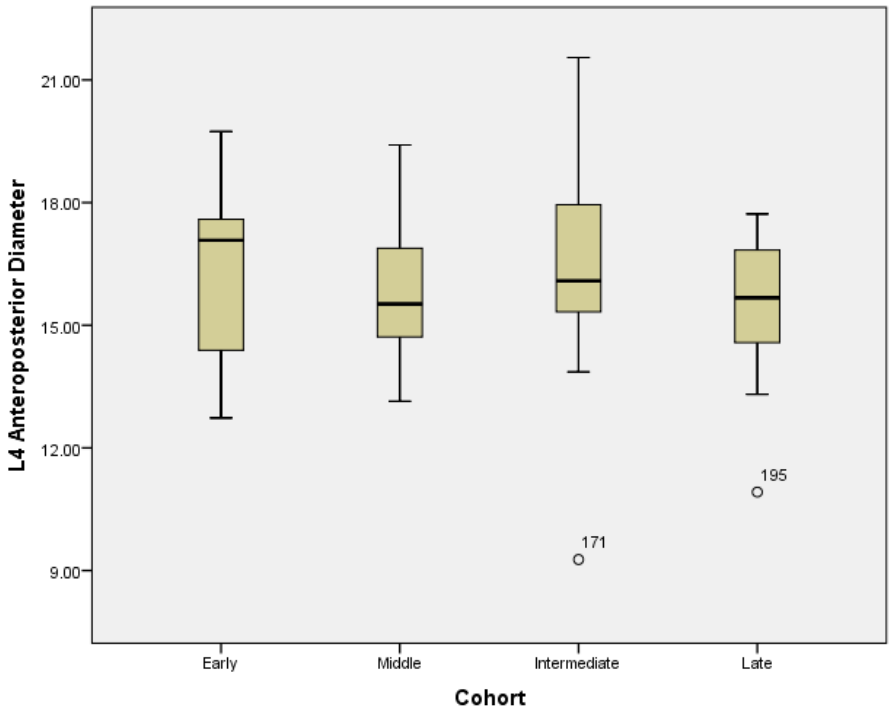

Figure A-100. Group two female L4 anteroposterior diameter across cohorts.

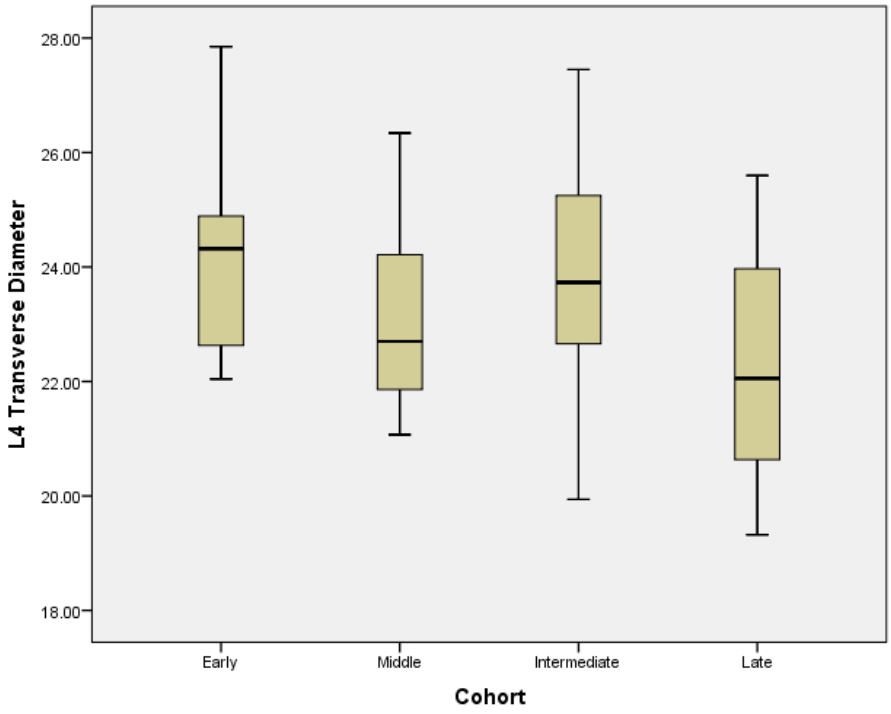

Figure A-101. Group two female L4 transverse diameter across cohorts.

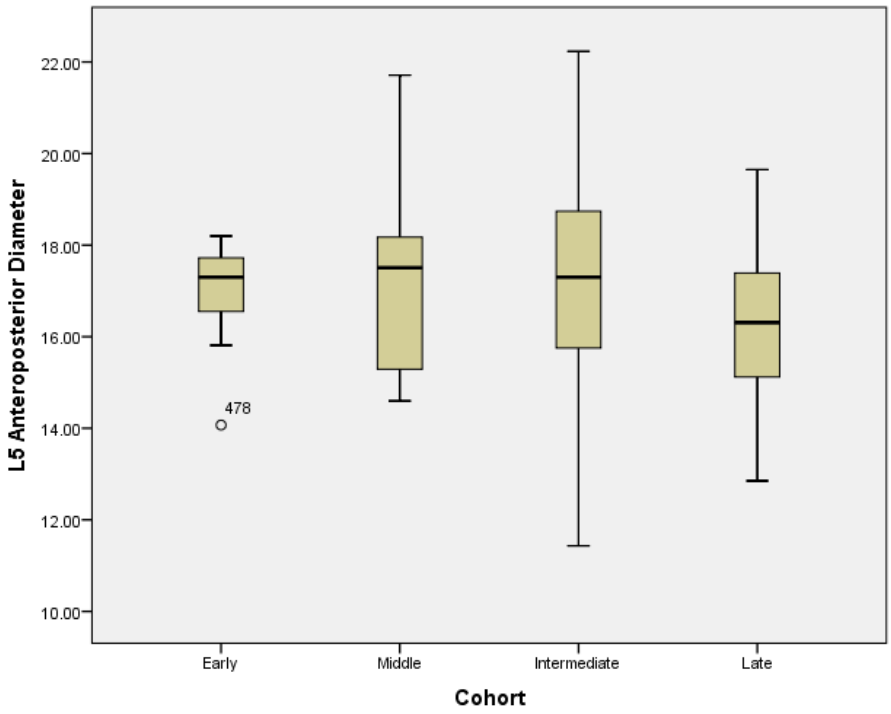

Figure A-102. Group two female L5 anteroposterior diameter across cohorts.

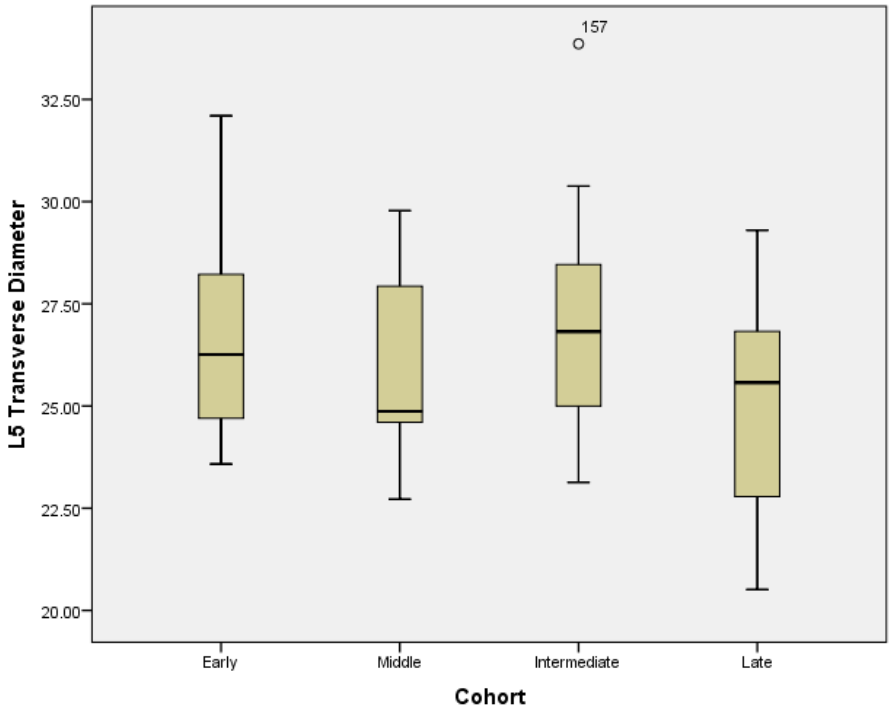

Figure A-103. Group two female L5 transverse diameter across cohorts.

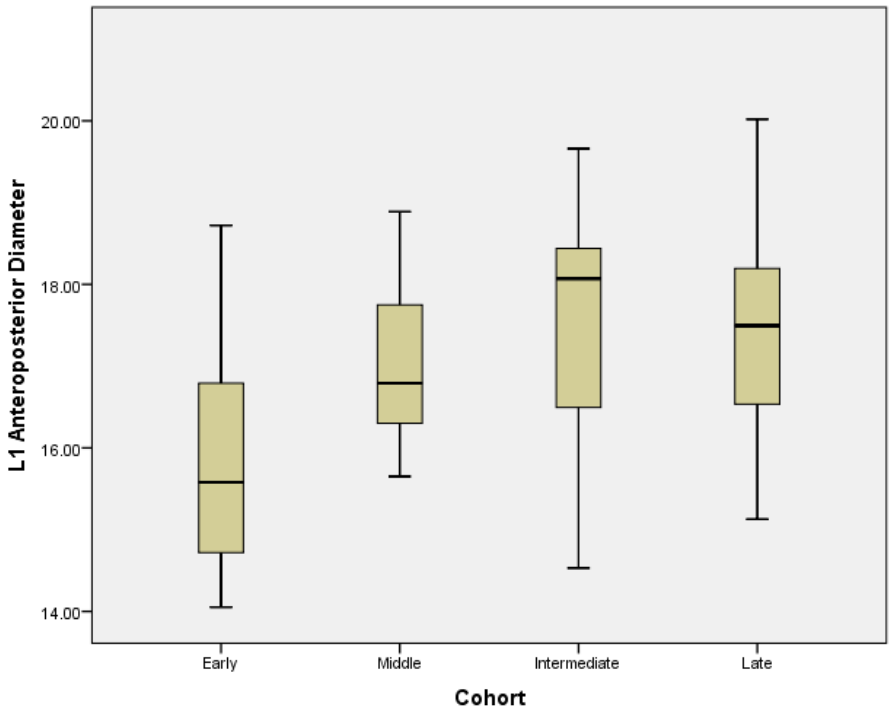

Figure A-104. Group three female L1 anteroposterior diameter across cohorts.

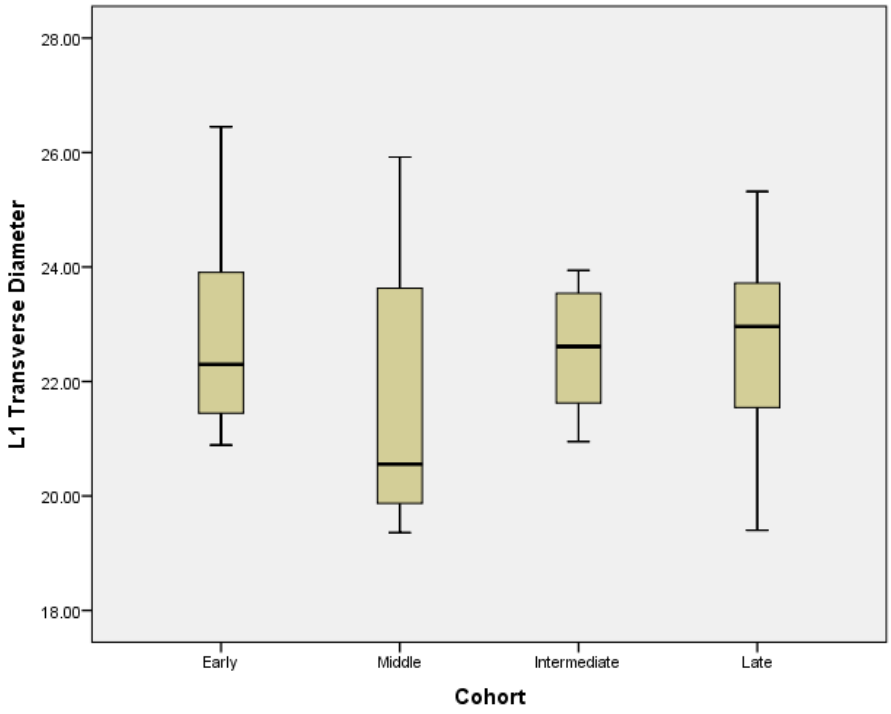

Figure A-105. Group three female L1 transverse diameter across cohorts.

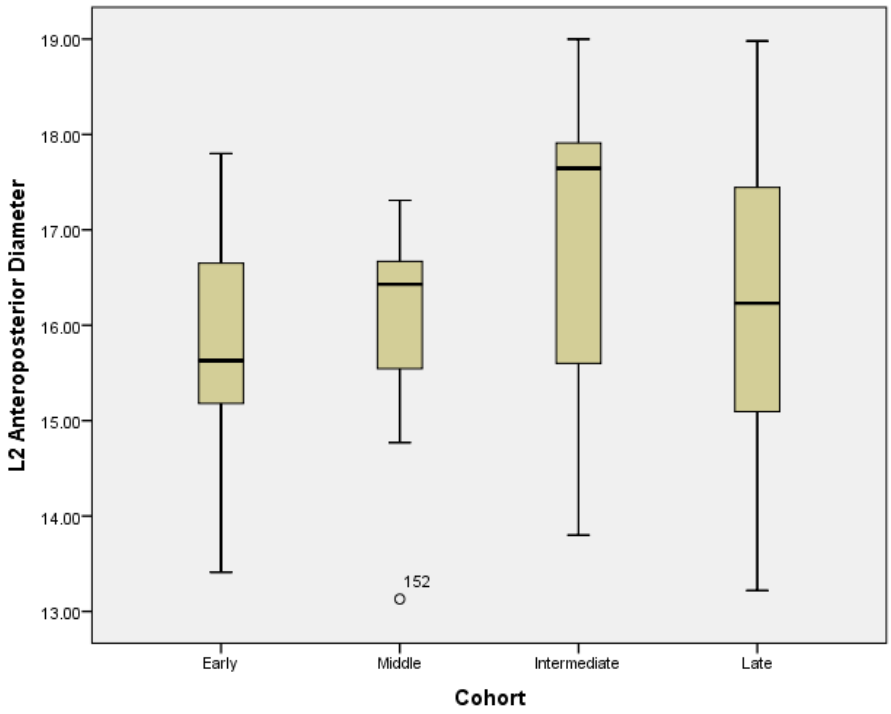

Figure A-106. Group three female L2 anteroposterior diameter across cohorts.

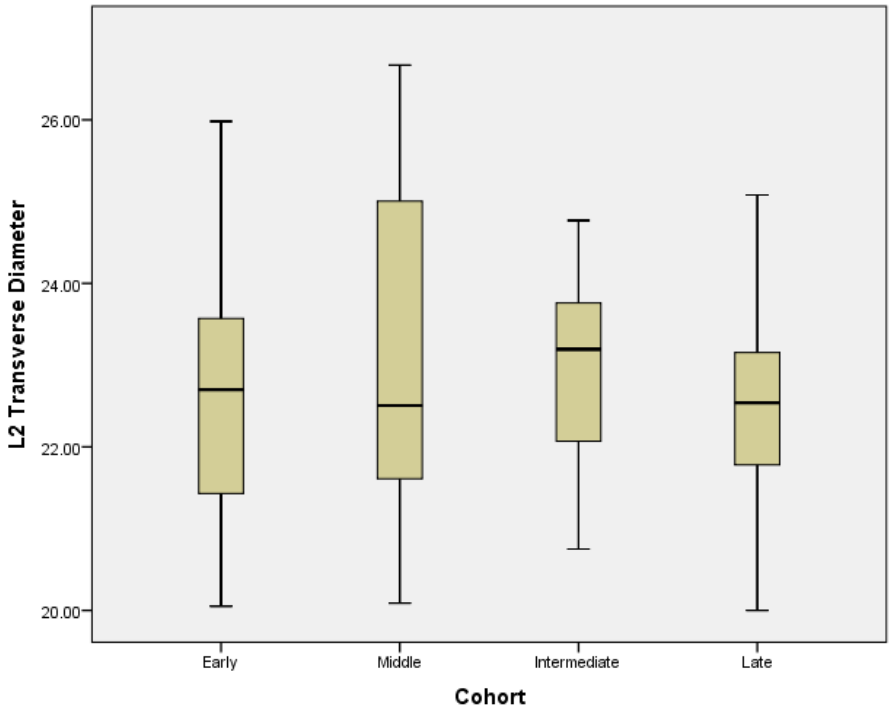

Figure A-107. Group three female L2 transverse diameter across cohorts.

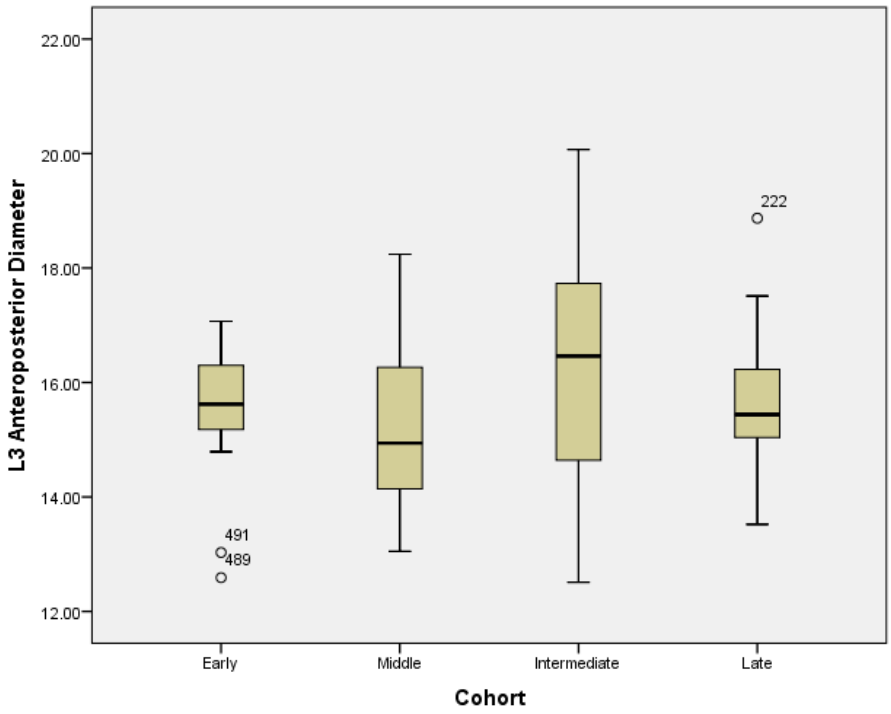

Figure A-108. Group three female L3 anteroposterior diameter across cohorts.

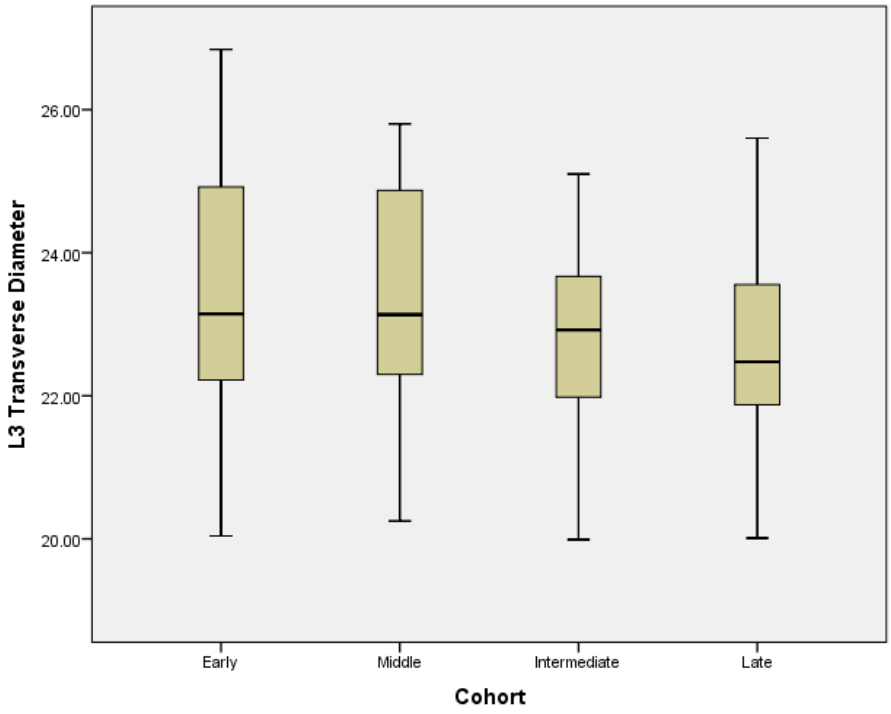

Figure A-109. Group three female L3 transverse diameter across cohorts.

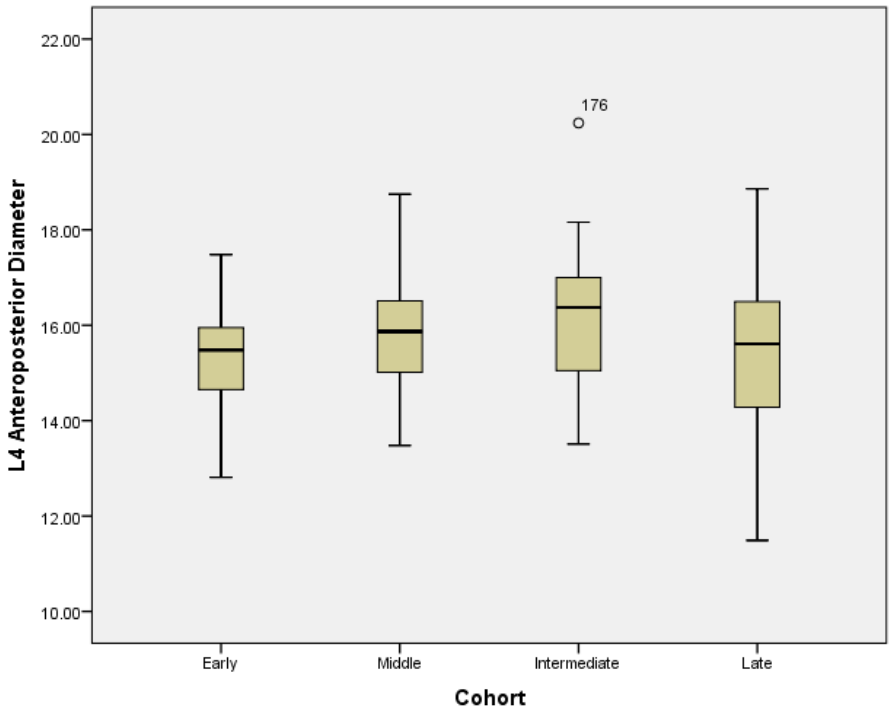

Figure A-110. Group three female L4 anteroposterior diameter across cohorts.

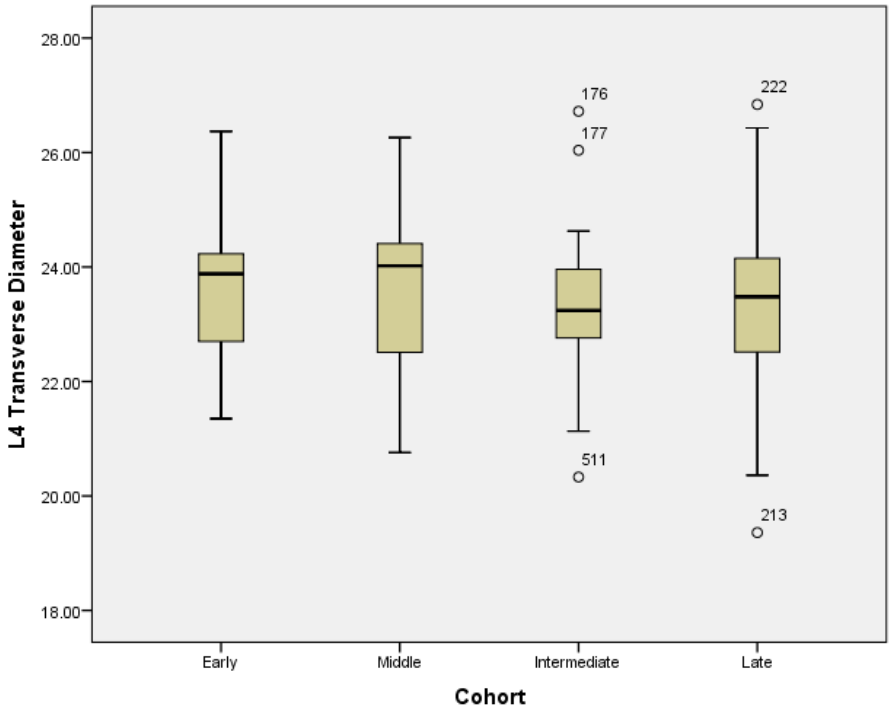

Figure A-111. Group three female L4 transverse diameter across cohorts.

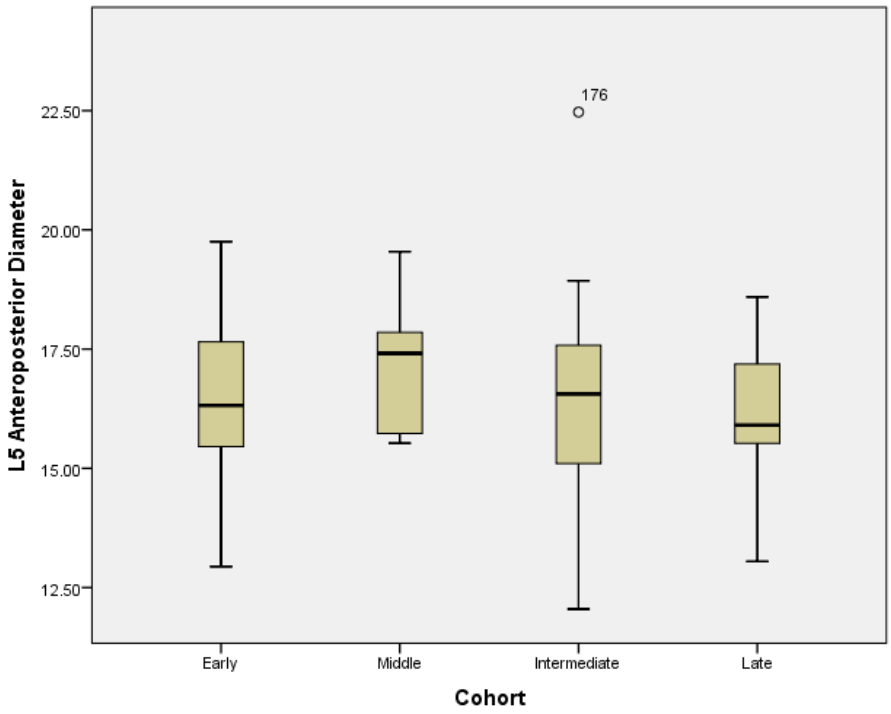

Figure A-112. Group three female L5 anteroposterior diameter across cohorts.

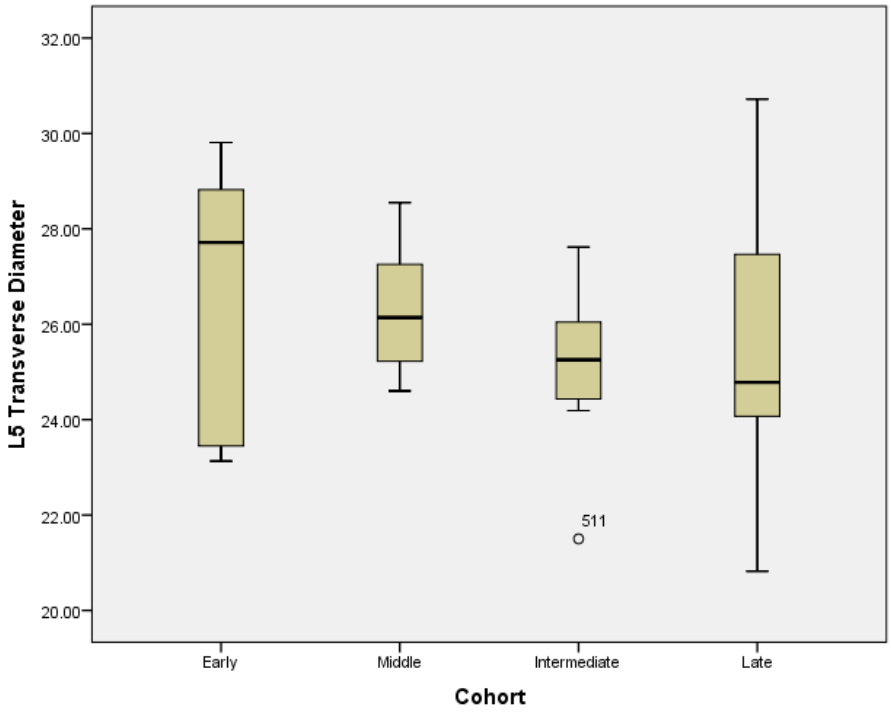

Figure A-113. Group three female L5 transverse diameter across cohorts.

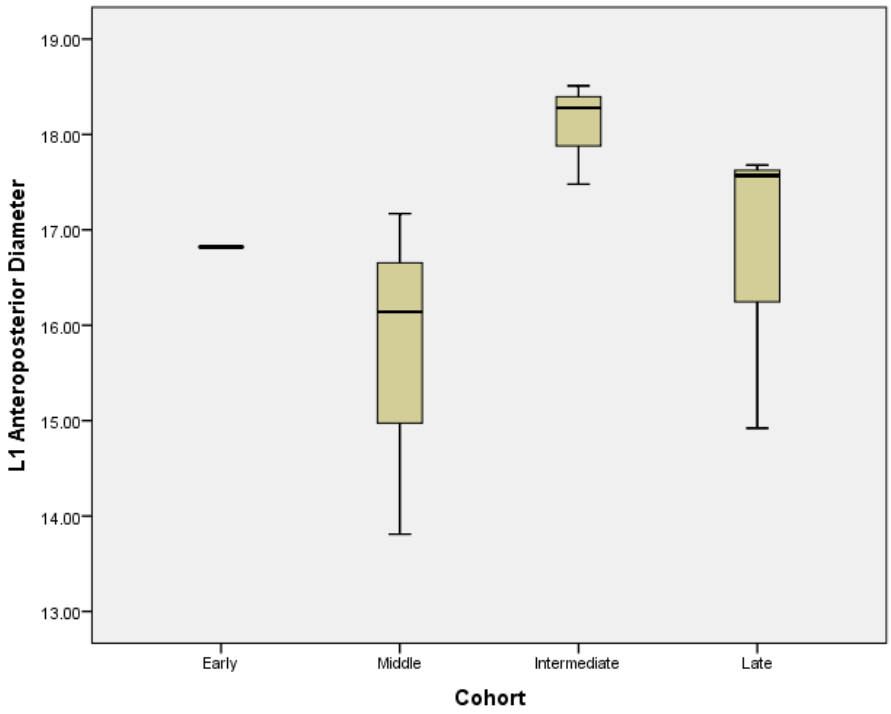

Figure A-114. Group four female L1 anteroposterior diameter across cohorts.

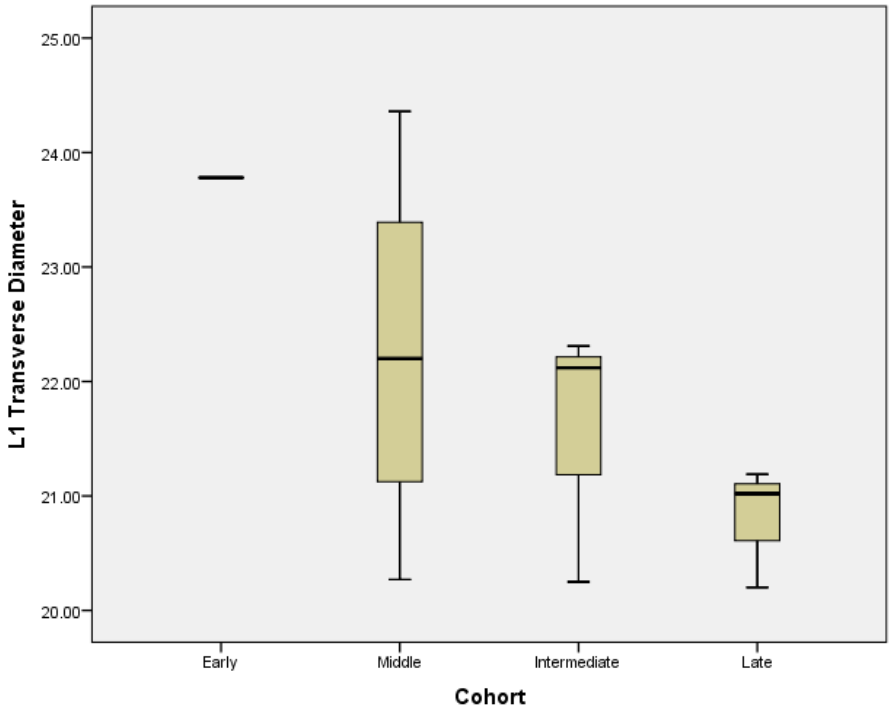

Figure A-115. Group four female L1 transverse diameter across cohorts.

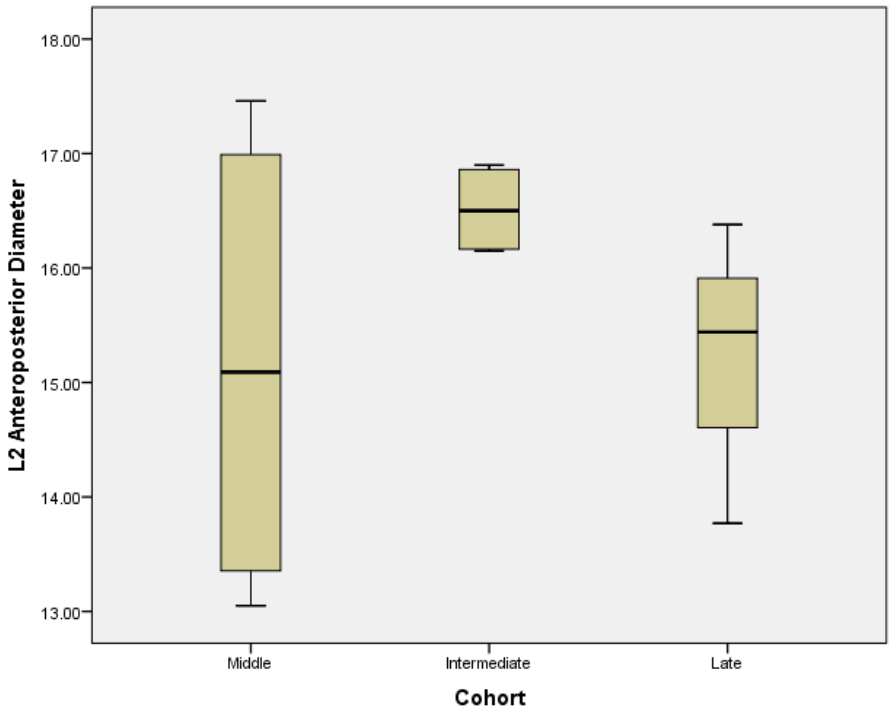

Figure A-116. Group four female L2 anteroposterior diameter across cohorts.

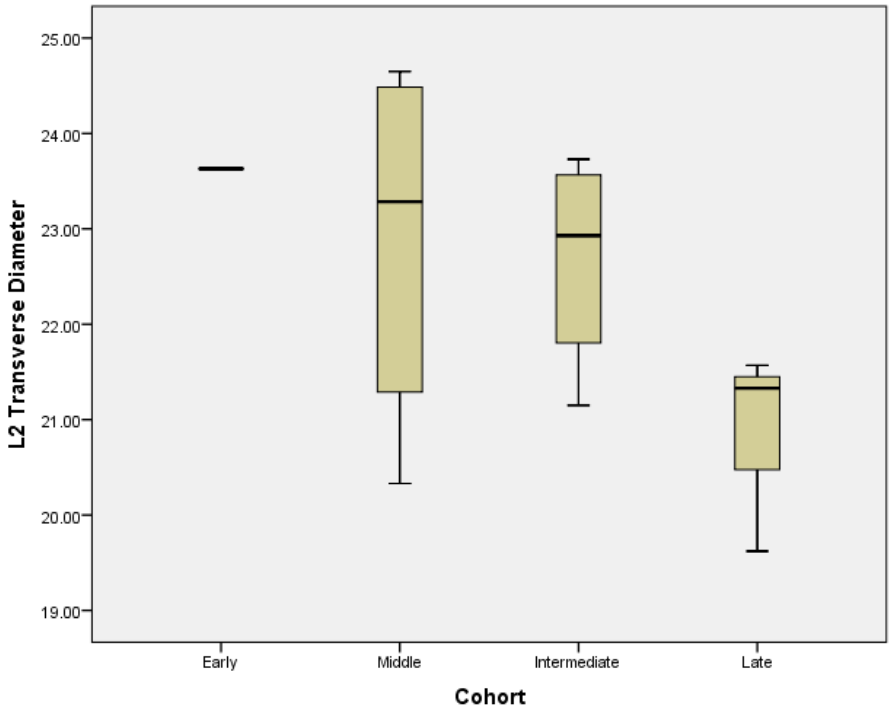

Figure A-117. Group four female L2 transverse diameter across cohorts.

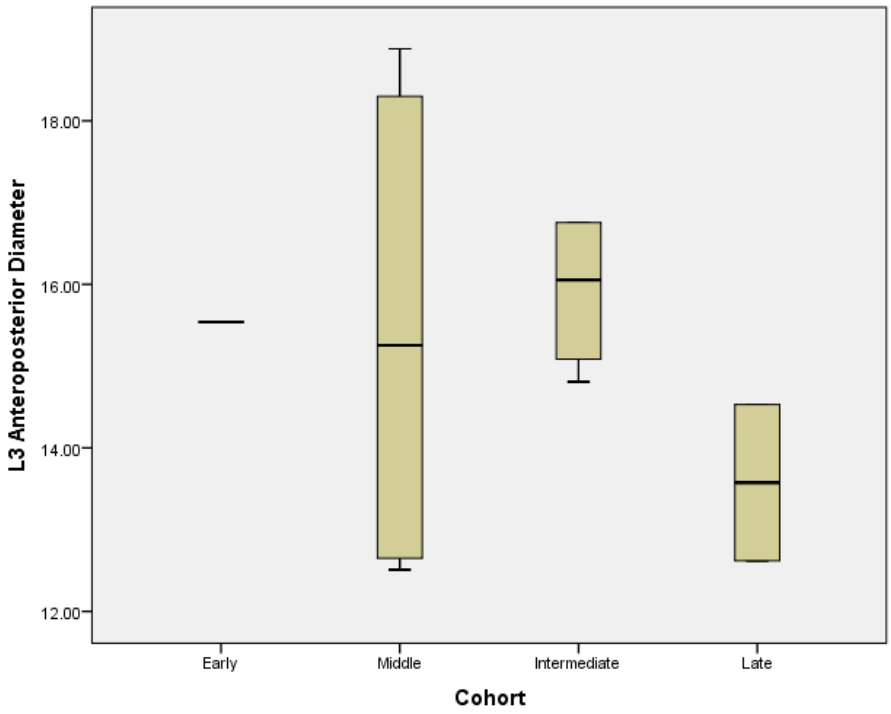

Figure A-118. Group four female L3 anteroposterior diameter across cohorts.

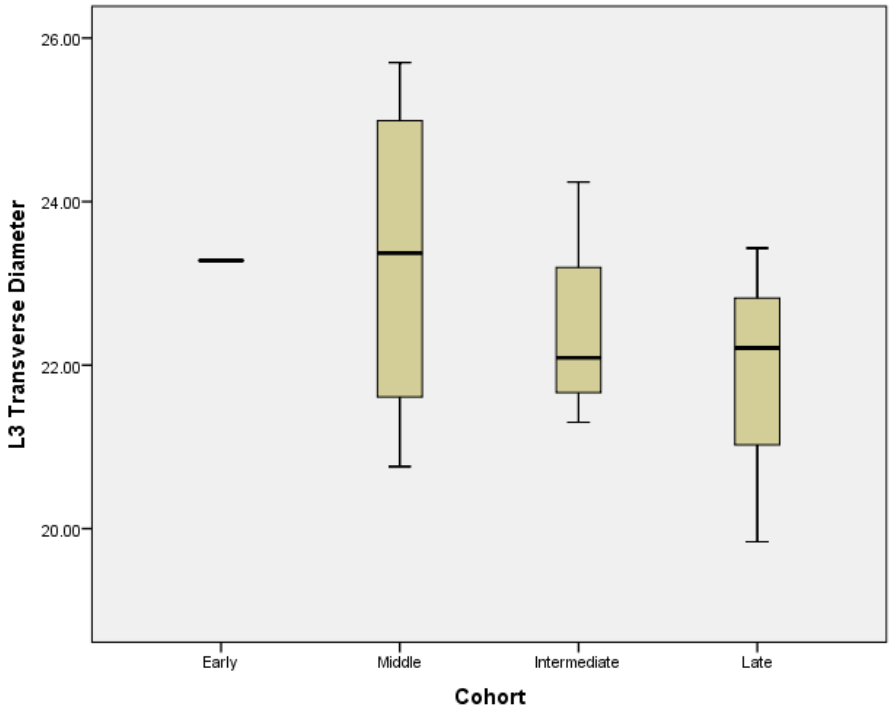

Figure A-119. Group four female L3 transverse diameter across cohorts.

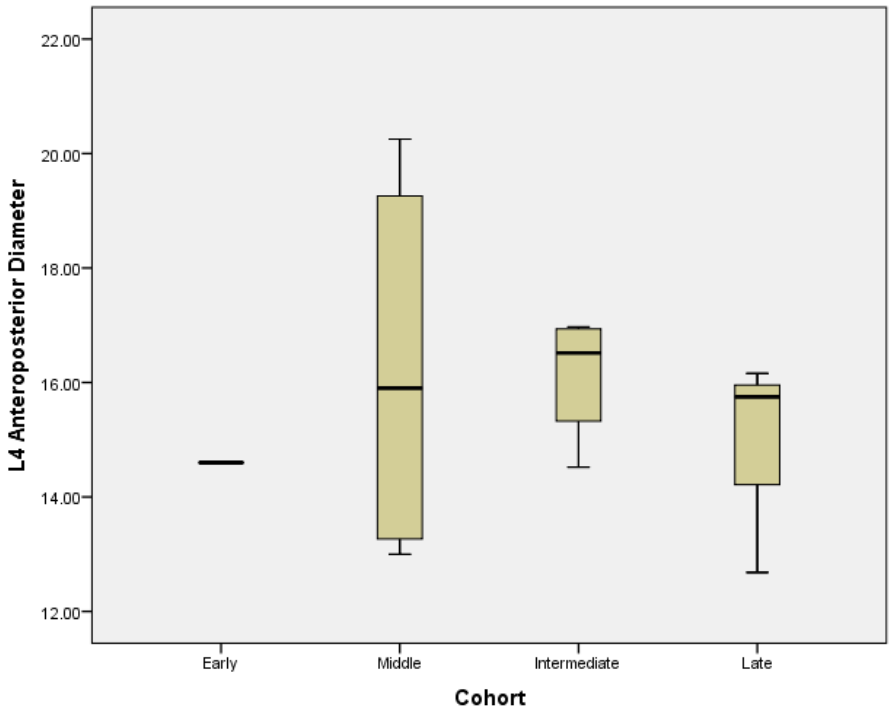

Figure A-120. Group four female L4 anteroposterior diameter across cohorts.

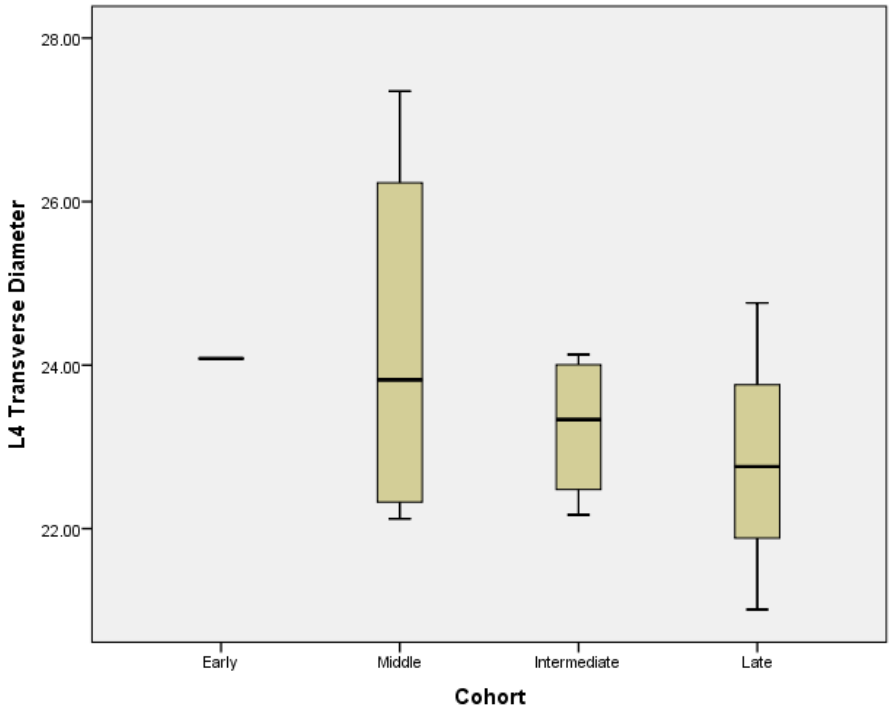

Figure A-121. Group four female L4 transverse diameter across cohorts.

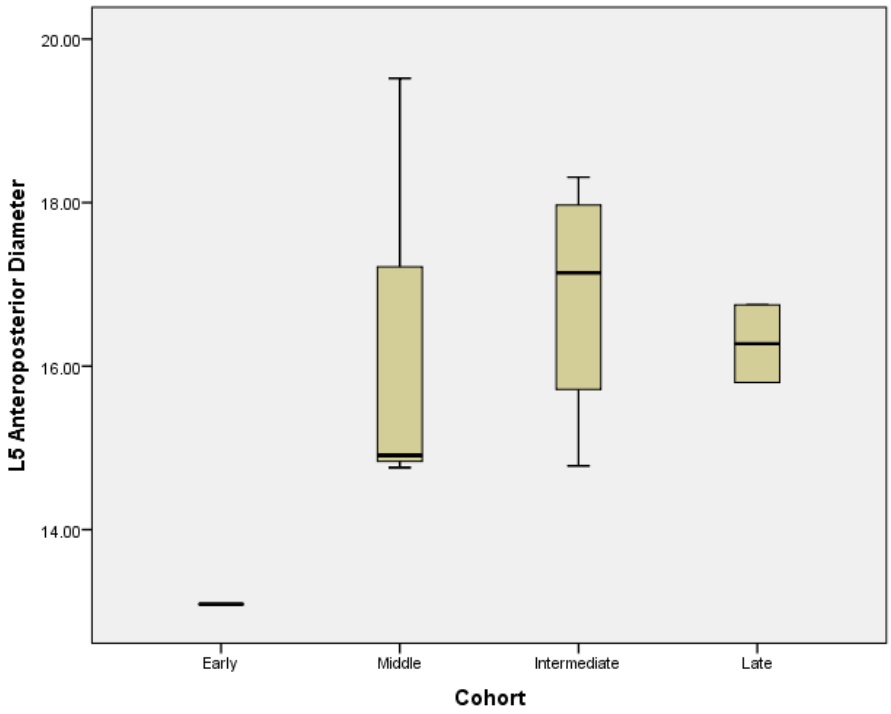

Figure A-122. Group four female L5 anteroposterior diameter across cohorts.

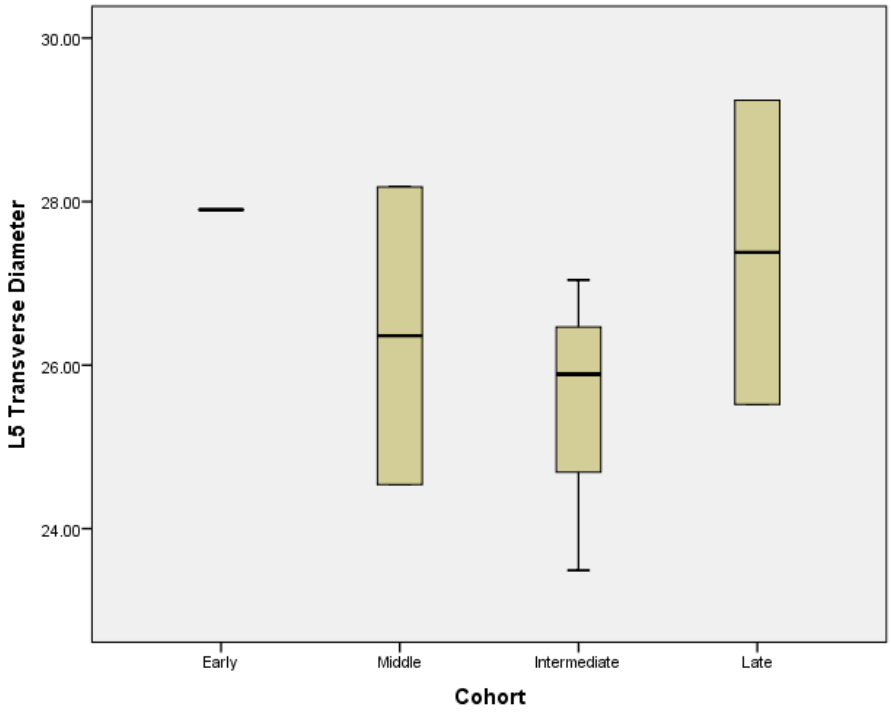

Figure A-123. Group four female L5 transverse diameter across cohorts.

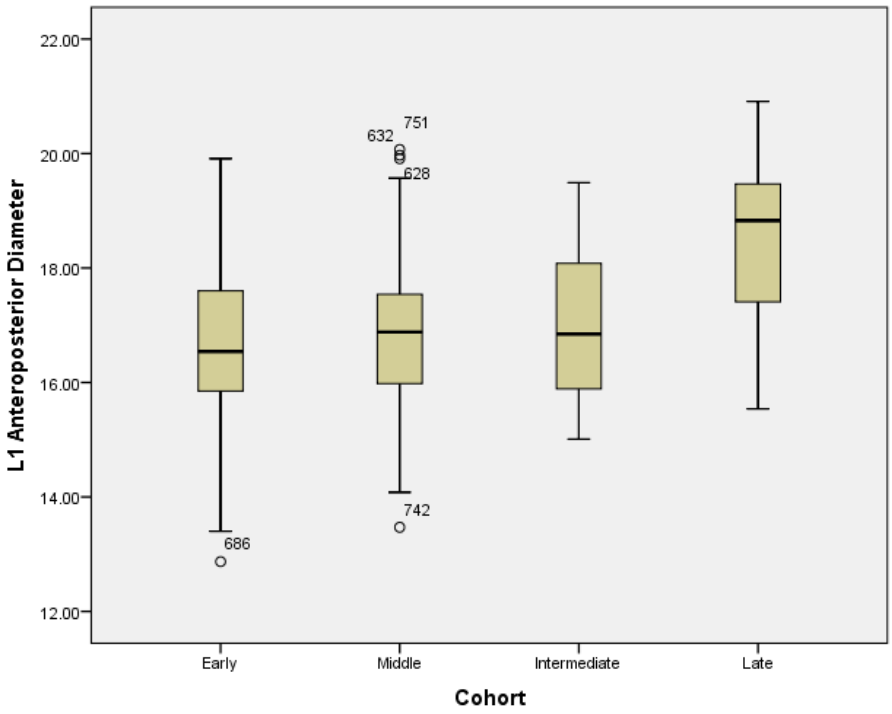

Figure A-124. African American female L1 anteroposterior diameter across cohorts.

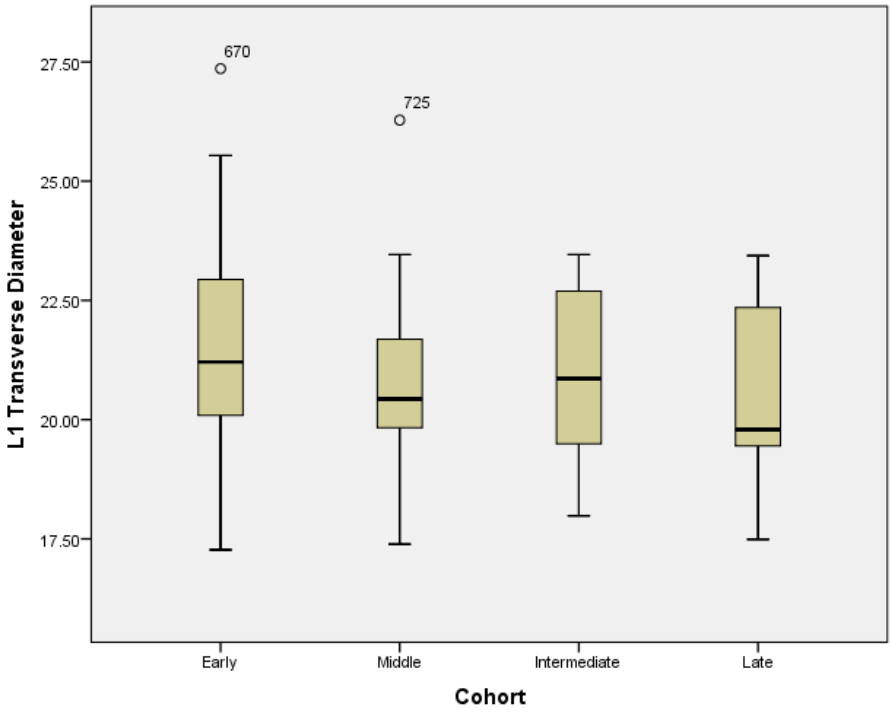

Figure A-125. African American female L1 transverse diameter across cohorts.

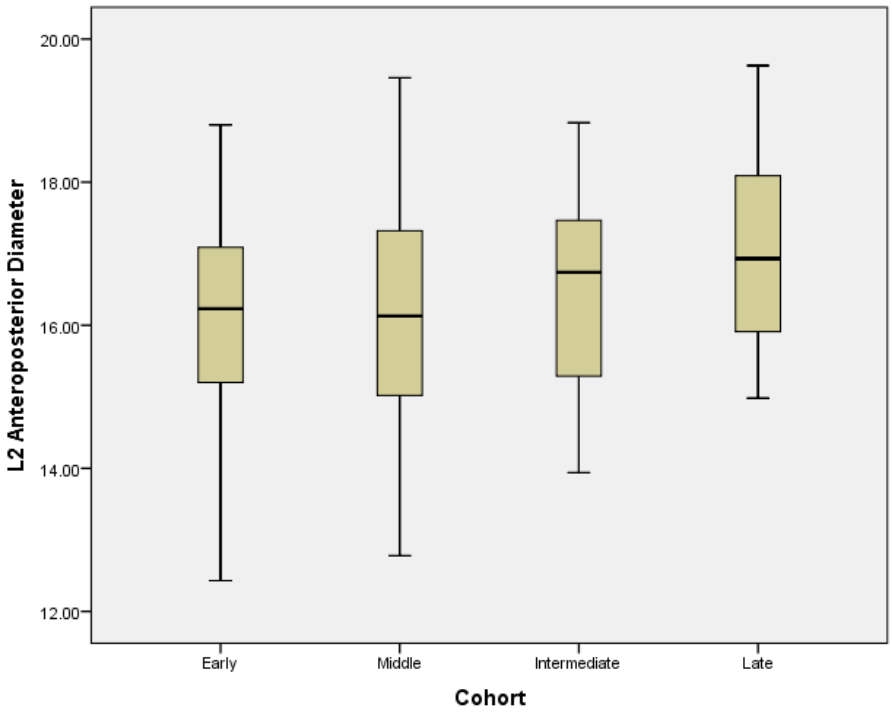

Figure A-126. African American female L2 anteroposterior diameter across cohorts.

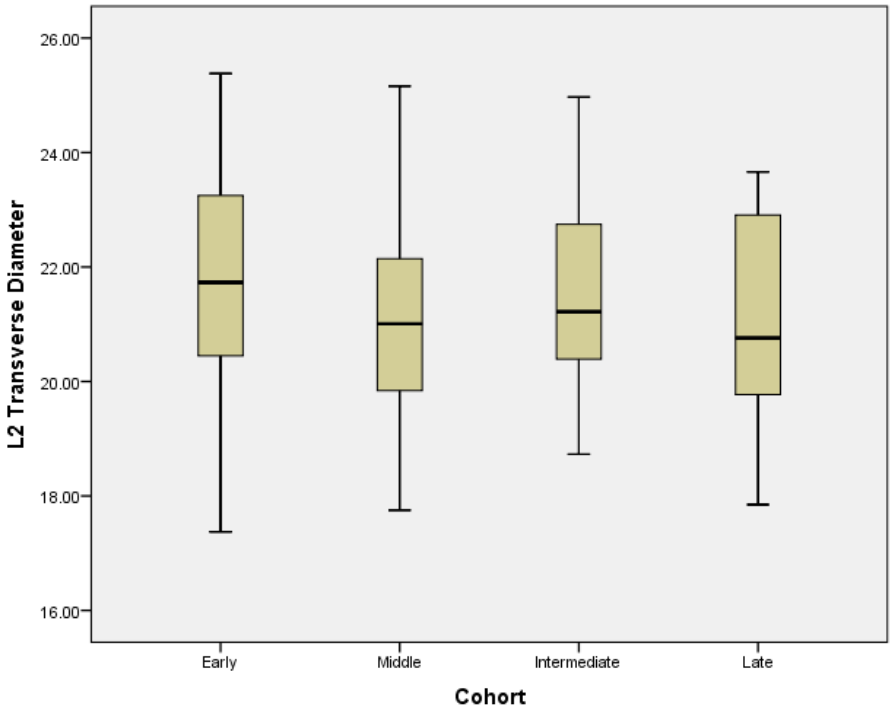

Figure A-127. African American female L2 transverse diameter across cohorts.

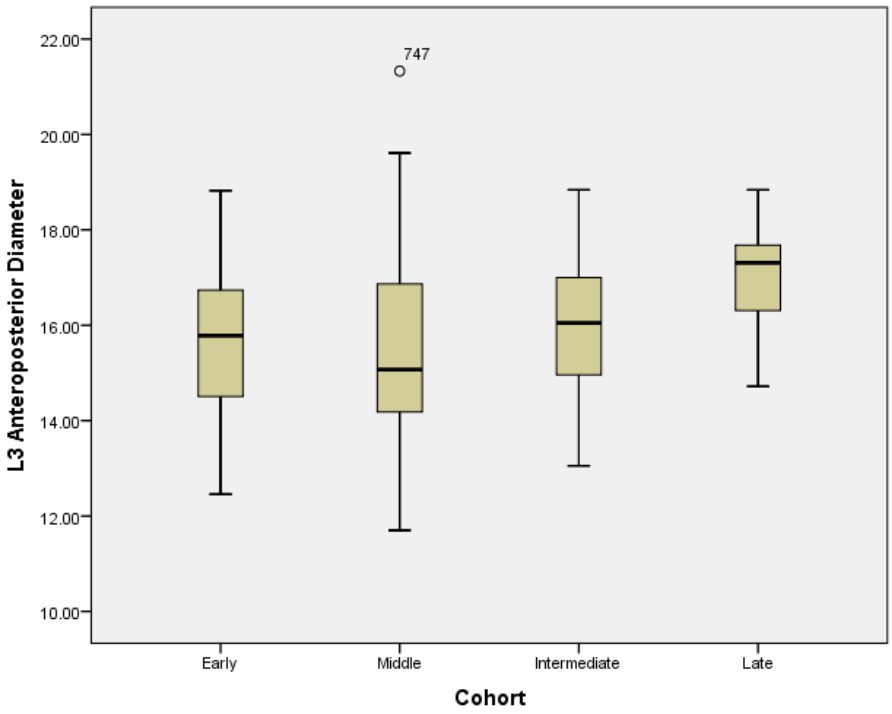

Figure A-128. African American female L3 anteroposterior diameter across cohorts.

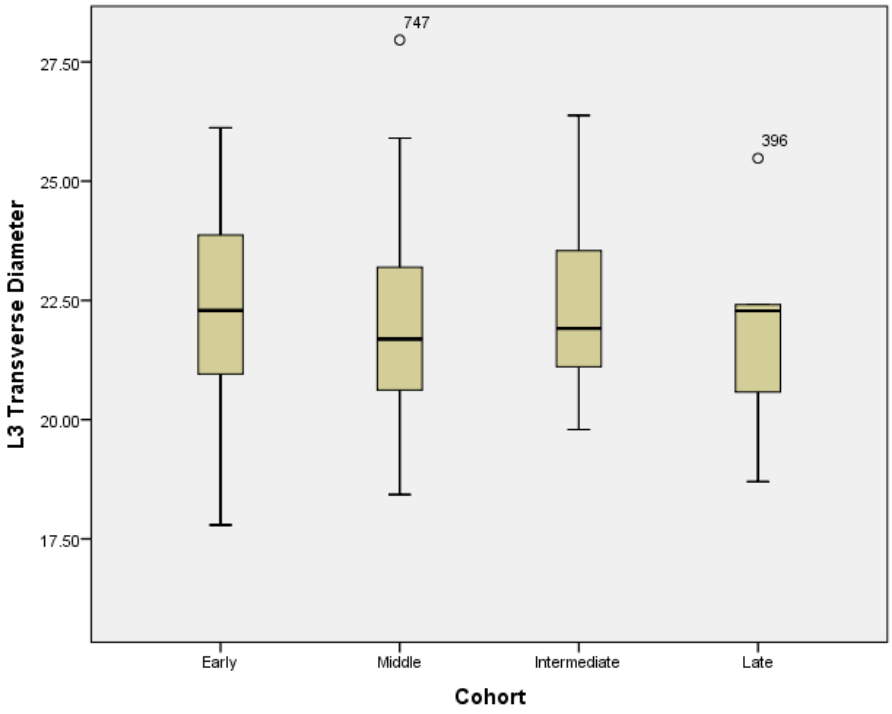

Figure A-129. African American female L3 transverse diameter across cohorts.

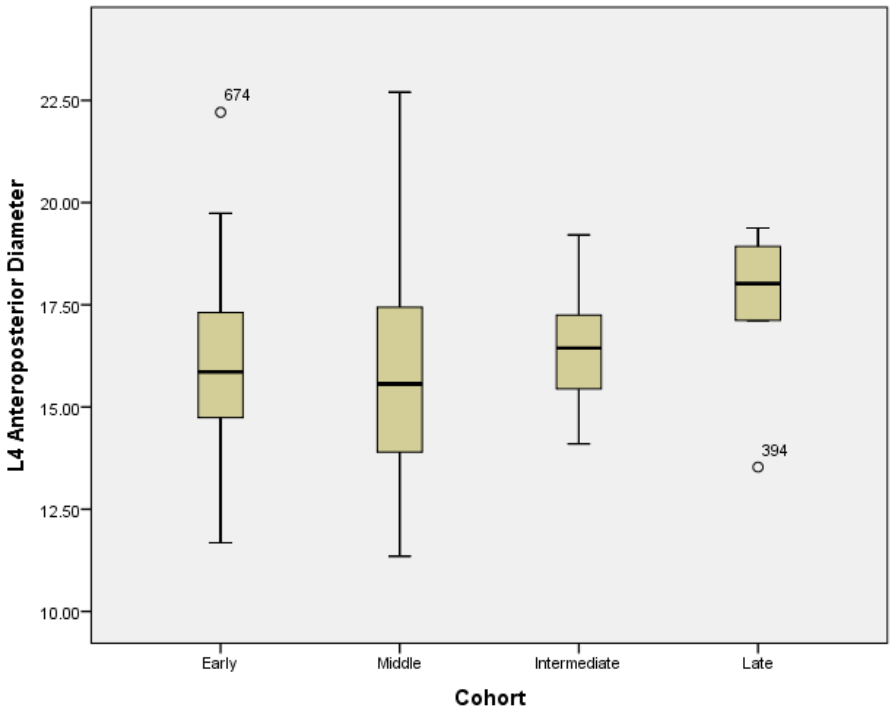

Figure A-130. African American female L4 anteroposterior diameter across cohorts.

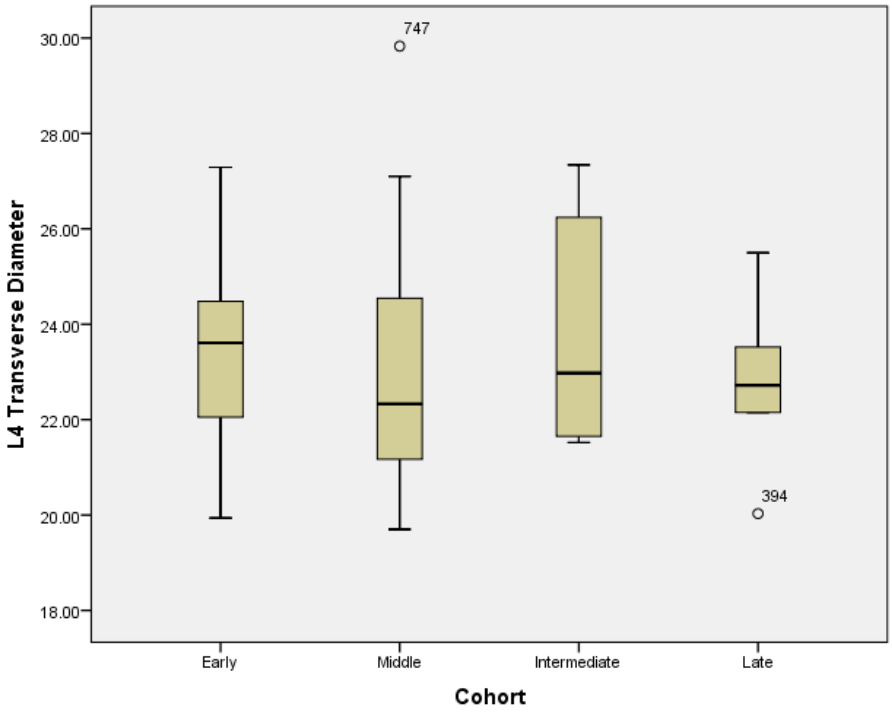

Figure A-131. African American female L4 transverse diameter across cohorts.

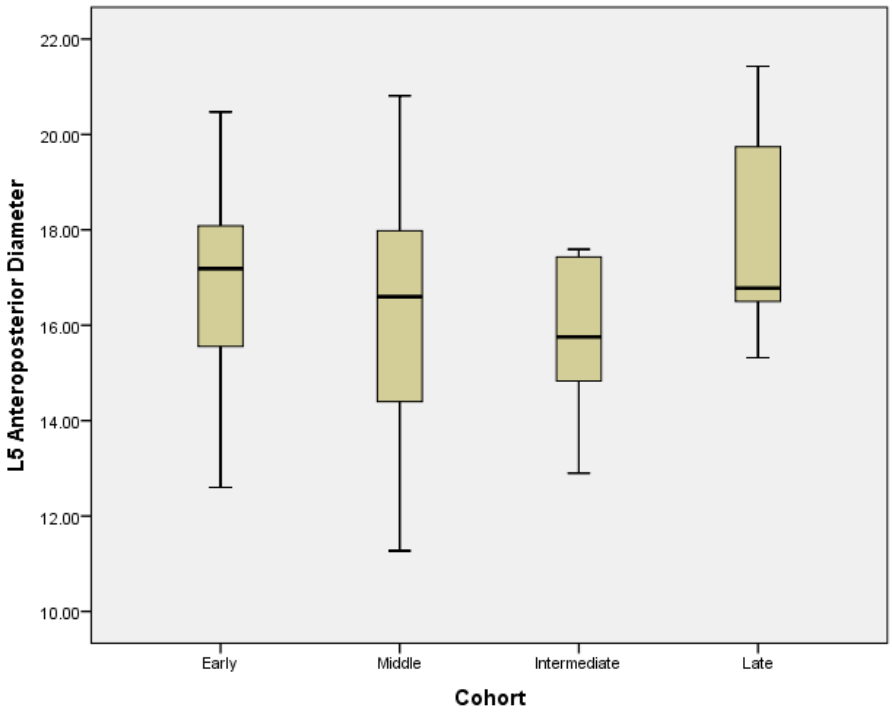

Figure A-132. African American female L5 anteroposterior diameter across cohorts.

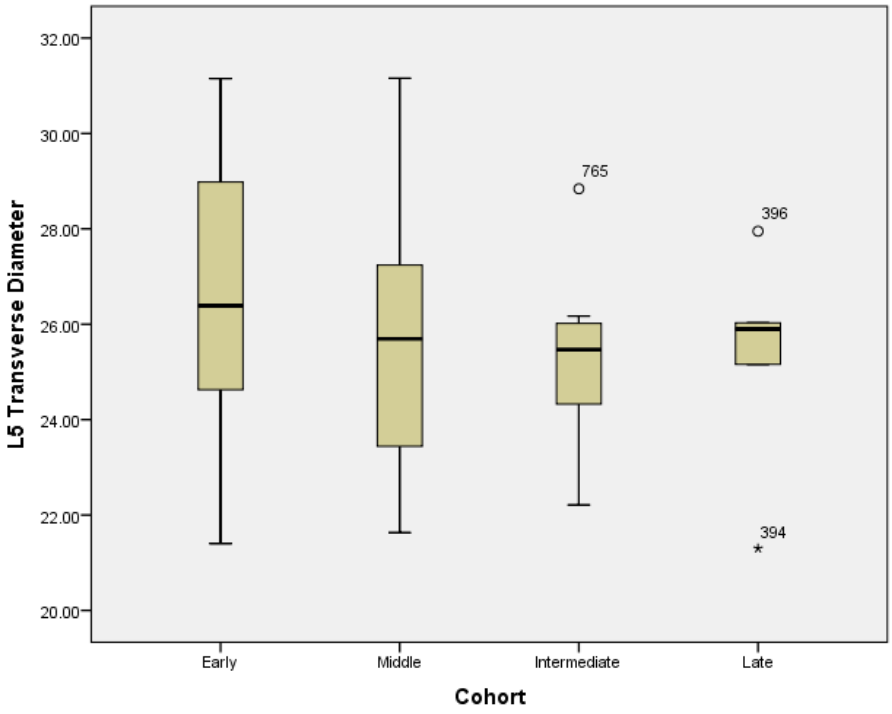

Figure A-133. African American female L5 transverse diameter across cohorts.

VITA

Kimberly Wren was born in Dallas, Texas to Therolan Wren and William Brigham, Jr. She graduated from Plano East Senior High School in 2001 and went on to complete her Associate of Arts degree at Collin County Community College in 2002. In 2004, she completed her Bachelor of Arts degree in Archaeology and Linguistics at the University of Texas, Austin. She was accepted into the Master's program in the Department of Anthropology at the University of Tennessee, Knoxville in 2004 and subsequently completed her Master of Arts degree in Biological Anthropology in 2007. She completed her Doctor of Philosophy in Biological Anthropology at the University of Tennessee, Knoxville in August of 2017. While in graduate school, Kimberly worked as a graduate research assistant to the Curator of Paleoethnobotany and an assistant to the Curator of Archaeology at the McClung Museum of Natural History and Culture. She also worked as a graduate assistant with the National Park Service through the University of Tennessee, Knoxville.