"Race Becomes Biology": Co-occurring Oral and Systemic Disease as Embodiment of Structural Violence in an American Skeletal Sample

Rieti G. Gengo

Western Michigan University, rigengo@gmail.com

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“RACE BECOMES BIOLOGY”: CO-OCCURRING ORAL AND SYSTEMIC DISEASE AS EMBODIMENT OF STRUCTURAL VIOLENCE IN AN AMERICAN SKELETAL SAMPLE

by

Rieti G. Gengo

A thesis submitted to the Graduate College in partial fulfillment of the requirements for the degree of Master of Arts Anthropology Western Michigan University December 2014

Thesis Committee:

Jacqueline Eng, Ph.D., Chair
Bilinda Straight, Ph.D.
LouAnn Wurst, Ph.D.
In recent years, a large number of biomedical studies have demonstrated that the bacteria that contribute to periodontal disease can migrate outside the oral cavity, causing a host of systemic infections. Yet, to date, only one bioarchaeological investigation has addressed this co-occurring disease process in a past population. The results of this thesis confirm the bioarchaeological visibility of the correlation between oral and systemic disease based on data derived from a sample of white and black adults from the Robert J. Terry Anatomical Skeletal Collection. Vertical recessions and porous remodeling of the alveolar crest were examined to identify periodontitis. Periosteal lesions on the femur, tibia and fibula were used as indicators of non-specific, systemic disease. As in a previous study of periodontitis and periostitis in a medieval British cemetery, a significant correlation between these disease processes in the Terry Collection sample is reported, suggesting the importance of further work on co-occurring disease processes in skeletal remains. Furthermore, not only are rates of periostitis and moderate-to-severe periodontitis significantly higher among black individuals than white, but the severity of each disease process is significantly more severe in the black sample. These results offer an example of the physical embodiment of structural inequality, highlighting the complexity of disease states as existing in a dialectical relationship with social processes.
For my grandpa, “Rock” Gengo, whose light still brightens the darkest day, and whose memory remains an inspiration.
ACKNOWLEDGEMENTS

I would like to begin by thanking the members of my thesis committee—Dr. Jacqueline Eng, Dr. Bilinda Straight, and Dr. LouAnn Wurst—for their support and guidance throughout this project. Their diverse expertise and devoted mentorship have been absolutely essential to my development as a scholar. I would also like to thank Dr. Sarah Schrader, who has offered her love, encouragement, and faith in my ability to complete this thesis; helped me celebrate each milestone, large and small; kept me sane; and provided invaluable help with statistics.

Dr. David Hunt, curator of the Robert J. Terry Anatomical Skeletal Collection, granted me access to the collection, gave me lab space to collect my data, and helped me understand the remains more deeply, for all of which I am extremely grateful. Dr. Susan Sheridan at the University of Notre Dame, was kind enough to allow me access to her skeletal collection to take high-quality photos of periosteal lesions, since I did not have the opportunity to photograph good examples while visiting the Smithsonian. Additionally, I want to thank Peggy Rafferty, who extended tremendous hospitality in opening her home to me while I visited Washington, D.C., to collect my data.

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he is remembered with immense love, and his memory is a constant source of inspiration.

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Any errors, oversights, or inadequacies that remain in this document are entirely my own.

Rieti G. Gengo
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CHAPTER I

INTRODUCTION

Race, like any other mode of social inequality, is a culturally constructed framework of structural disenfranchisement that has dire consequences for those occupying its lower strata. While many of these consequences are social—unfair housing policies, unequal access to employment opportunities, educational disparities, and many others—some outcomes of structural racism are also biological, such as decreased longevity and higher morbidity rates of many diseases. These health outcomes have proximate causes such as malnutrition, increased susceptibility to disease, and long-term elevated cortisol (a stress hormone), but ultimately they are products of structural inequality. Decades of racial science, however, attempted to explain health disparities as inherent maladaptations in non-white races, and even worse, to construe socially understood differences between arbitrary “races” as natural, biological realities (Baker, 1998). The entrenched biological model of race drew (and still draws) its strength from the sober neutrality that positivist science claims to possess. Yet scientists, as products of and interlocutors in a cultural milieu, can never divorce themselves completely from biases in developing their research questions and conclusions; many, however, remain unconscious of these cultural influences on their work.

Anthropologists and other social scientists have recognized the problems of scientific racism as being rooted in attempts to naturalize a culturally produced concept and have reacted with a pendulum shift that places the race concept in the realm of
cultural construction (Sanjek, 1994; American Anthropological Association, 1998; Urciuoli, 2011). They have rejected formerly deep-seated notions that racial groups are unchanging, quantifiable products of human biology, instead developing a theory of race that highlights the social and historical processes that constructed the biological race concept (Smedley and Smedley, 2011). This way of understanding race reveals its culturally constructed core and exposes the racial worldview as the ultimate culprit responsible for inequality along racial lines. Current biomedical literature, on the other hand, attempts to integrate some of these ideas about race as a cultural construct, but it still tends to reify the outmoded view of race as biology through research designs that attempt to isolate biological differences between racial groups without adequately accounting for social factors.

Recent changes in anthropologists’ understanding of race have been largely positive. Positioning race as a culturally constructed identity that has arisen from centuries of systematic oppression has rightly assigned culpability for race-based inequalities to the socially more powerful (white) members of society. Furthermore, it has allowed for a deeper understanding of how extensive the structural violence enacted against non-white racial groups really is. Yet, paradoxically, by disregarding biology in discussions of race, scholars have overlooked a critical element of its effects (Hartigan, 2013). A growing body of literature has recognized the error inherent in the extreme pendulum shift toward viewing race as a social construction and has attempted to address the ways in which race and other modes of social inequality become embodied through their biological effects. Clarence Gravlee’s (2009:47) idea that “race becomes biology” is an elegant statement of this notion. In addition to race, scholars have also turned a critical
eye toward gender (e.g. Fausto-Sterling, 2005; Connell, 2012), class (e.g., Najman and Smith, 2007), indigeneity (e.g. Adelson, 2005), sexuality (e.g. Meyer, 1995; Fausto-Sterling, 2007), and others as biocultural processes—that is, social identities that exist in a dialectical relationship with biological outcomes (Goodman and Leatherman, 1998).

My study adds to this body of literature by using skeletal evidence of periodontitis (a kind of oral disease) and periostitis (infection of the bone) in a mixed race sample as a case study for thinking about the health disparities that characterize embodied inequality. I present a way for bioarchaeologists to enter into this discourse through the methods of skeletal analysis they already use. Bioarchaeology, which examines skeletal remains in their archaeological context, has a tremendous capacity to engage in critical dialogue with biomedical science with regard to the dialectical relationship between social inequality and disease manifestation. Skeletal tissue’s combination of both plasticity and durability provides a window through which to interpret embodied experiences of disease, stress, and trauma that occurred over many years of a person’s life. Though for much of its history bioarchaeology has been a largely descriptive science, increasingly practitioners have become more theoretically driven and concerned with social processes (Armelagos and Van Gerven, 2003). Given these theoretical developments, bioarchaeology is poised to contribute significant time depth to our understandings of how inequality becomes expressed through differential health outcomes.

Study Objectives

Several biomedical studies have demonstrated a link between oral disease and extra-oral infections, but many have also failed to adequately account for race or other
forms of structural inequality as factors in disease presentation and outcome. This study asks whether the connection between these pathological conditions is bioarchaeologically visible by examining markers of periodontal disease and non-specific, systemic infections in skeletal remains. DeWitte and Bekvalac (2011) employed a similar methodology in their recent study of a medieval British cemetery sample, in which they found a correlation between periodontal disease and the presence of periosteal lesions. Interestingly, they did not find that social status had any bearing on either pathological condition. However, their measure of social status was based on individuals’ spatial proximity to the abbey church beside the cemetery: those individuals buried closest to the church were considered high status, and those farther away were deemed low status.

While that study measured social status, the question remains whether the separate factor of race might play a role in how oral and extra-oral infections are correlated. Given the vast, multifaceted literature showing differential health outcomes in the United States based on racial identity, I found it inconceivable that the effects of race would be bioarchaeologically invisible with respect to this co-occurring disease process. As such, I analyzed data collected from a mixed-race, mixed-sex sample of the Robert J. Terry Anatomical Skeletal Collection—a 20th-century American skeletal collection that includes both white and black individuals. The Terry Collection was curated originally at Washington University of St. Louis, drawing specimens from the local population, but it is currently held by the Smithsonian Institution in the Physical Anthropology Division of the National Museum of Natural History in Washington, D.C.

I commenced this study with a set of expectations (Table 1). Based on the results of relevant biomedical studies (e.g., Li et al., 2000; Holmstrup et al., 2003; Pucar et al.,
2007) and one bioarchaeological case (DeWitte and Bekvalac, 2011), I predicted that oral and systemic disease markers would be correlated. Second, I hypothesized that the black and female samples would have higher rates and severities of both oral and systemic disease than their white or male counterparts, assessed by alveolar bone recession and periosteal lesions, respectively. A demonstrated connection between these pathological conditions and race or sex independent of socioeconomic status would support the idea that the socially meaningful categories of race and gender are susceptible to embodied health consequences. My third hypothesis was that rates of both oral and systemic disease would increase with age, since older adults are typically more susceptible to disease than younger individuals, and since the effects of periodontal disease are typically compounded over time. My final hypothesis was that childhood physiological stress (in this study, represented by linear enamel hypoplasias [LEH]) would be a predictor of adult disease, as it is in living populations. Since adults can carry the marks of their childhood nutritional and physiological stress in defects of their tooth enamel, I was able to assess both adult and childhood health within each adult individual.

Table 1

<table>
<thead>
<tr>
<th>Hypotheses</th>
<th>Expectations</th>
<th>If Expectations Not Met</th>
</tr>
</thead>
<tbody>
<tr>
<td>1. Oral and systemic disease are linked in the sample.</td>
<td>A significant correlation exists between periodontitis and periostitis.</td>
<td>A link between oral and systemic disease cannot be demonstrated from these data.</td>
</tr>
<tr>
<td>2. Structural racism and sexism result in increased susceptibility to both oral and systemic disease.</td>
<td>Blacks and females have a higher rate and severity of periodontitis and periostitis than whites and males.</td>
<td>Race and sex are not significant factors in disease susceptibility in this sample.</td>
</tr>
</tbody>
</table>
Table 1—continued

3. Older individuals are more susceptible to oral and systemic disease than younger individuals.
   Rates of disease markers increase in successive age sets.
   Age is not a significant factor in disease susceptibility in this sample.

4. Adult disease is related to stress during childhood.
   A significant correlation exists between LEH presence and one or both disease markers.
   A link between childhood stress and adult health cannot be demonstrated from these data.

Study Outline

I begin this study by situating the disease processes observed in historical and geographic context, with a description of the 1917 race riot in East St. Louis—an example of the fraught racialized social environment that characterized Urban America during the Great Migration. I then offer a definition of race and explore how it has been used in the American bioanthropological and biomedical literatures, and I show how the structural violence of race and racism can become physically embodied as poor health outcomes that are visible in skeletal material. Following this is detailed information about the skeletal sample used for this study, along with an explanation of the methods employed to collect and analyze data. Notably, I offer a modification of the standard methods used to identify periodontal disease that is more conservative and may be more reliable in the context of the Terry Collection sample. Finally, I present and interpret the results of data analysis and explain this study’s implications for bioarchaeological and broader anthropological research.
CHAPTER II

REVIEW OF RELEVANT CONCEPTS AND LITERATURE

Some Historical Context

The East St. Louis Race Riot of 1917

St. Louis, Missouri, and the surrounding area represent a locus of racial tension during the late 19th and early 20th centuries, the period during which Robert Terry was building his collection. This tension exploded in violence in July of 1917, when for two days a mob of white men and women in East St. Louis began assaulting black families, looting and burning their homes and businesses (McLaughlin, 2005). Not only were dozens of African-Americans killed and hundreds injured in this horrific event that included police officers and National Guardsmen among its perpetrators, but it also displaced over 7,000 black refugees, who took flight from the city to avoid the violence, which Lumpkins (2008) has called an “American Pogrom.” Of course, this race riot did not occur in a social vacuum, nor was it a simple reaction to an isolated event. Rather, it took place in an historical context of long-standing struggle for political, economic, and social influence that increasingly threatened white domination.

While the violent race riot of 1917 occurred in the city of East St. Louis, Illinois, this city lies just across the Mississippi River from St. Louis, Missouri. This arbitrary geographic border could not have insulated the black community of St. Louis from the violence occurring on the other side of the river; indeed, many of the refugees from the
riot sought asylum on the Missouri side, bringing the reality of racial violence with them (Lumpkins, 2008). Instead, the underlying tensions were shared between these two industrial centers, and with other metropolitan areas throughout the country. Indeed, the 1917 race riot in East St. Louis was not the only one of its kind in the first two decades of the 20th century; similar conditions of political struggle precipitated other episodes of anti-black violence in urban areas across the country, in Evansville, Indiana; Springfield, Illinois; Tulsa, Oklahoma; Wilmington, North Carolina; Atlanta; Houston; New Orleans; Chicago; Washington, D.C.; and New York City (Hair, 1976; Senechal, 1990; Cecelski and Tyson, 1998; Butler, 2000; Godshalk, 2005; Lumpkins, 2008). These pogroms represent the boiling over of racially driven resentment of the black communities in these areas and the perceived threat to the hierarchical social structure. Attempts by these communities to advance their political and economic standing in a post-slavery United States were deeply disturbing to their white counterparts, who saw social privilege as a zero-sum game in which they would lose power as blacks gained it (Marable, 2000).

Contextualizing Disease Within Racialized Historical Processes

The eruption of violence against the black citizens of East St. Louis signifies a rupture in the social order that reveals the extremely tense underlying social relations between socially powerful whites and relatively less powerful blacks. This kind of social environment would undoubtedly have resulted in a great deal of psychological stress as part of everyday life. Recent studies have shown that those suffering structural oppression and racial or ethnic discrimination produce higher levels of the stress hormone cortisol than their socially dominant counterparts (Miller et al., 2007; Kaholokula et al.,
2012; Zeiders et al., 2012). Since long-term elevated cortisol levels, responding to stressors of social relations, can negatively affect immune function and contribute to increased susceptibility to disease (Sapolsky, 2004; Karb et al., 2012; Agnew and South, 2014), it is clear how relevant this fraught social context is for the present study of differential health outcomes in members of the St. Louis black community.

By examining the 1917 riot in the context of the area’s racial history and that of the United States more broadly, it is possible to better understand the racially charged social climate that impacted the daily lives of those black individuals whose bodies became part of the Terry Collection. As noted above, the East St. Louis riot and the social environment that fostered it were not unique to the St. Louis area; instead, they were all too common across the United States in the late 19th and early 20th centuries, and tensions worsened as large numbers of people moved from rural areas of the country to urban centers.

The Great Migration

As industrial capitalism expanded in the early 20th century, blue-collar jobs became abundant in America’s large cities. Hoping to take advantage of the steady, year-round employment these jobs could offer (as opposed to seasonal agricultural work), the rural poor began to pour into urban areas en masse beginning around 1915 and continuing past the middle of the twentieth century—a dramatic demographic shift known as the Great Migration (Gregory, 2005). Although these migrants represented several different ‘racial’ groups, the majority were African-American (Marable, 2000). Moreover, most migrants were moving from the rural south to cities in the Northeast or Midwest,
relocating from areas that were intensely racialized to those that were seen as more tolerant, with better opportunities (Gregory, 2005). Lemann (1991) shows that black migrants often invoked a biblical trope to describe their movement out of the South to urban destinations, calling it an “Exodus” in reference to the biblical story of the Israelites’ escape from their Egyptian captivity and return to the Promised Land.

The St. Louis area—including East St. Louis—was one of these destinations. The U.S. Census Bureau reports that only 6.4% of the population of St. Louis, Missouri was black and in 1910, but this number increased steadily over the next several decades to 40.8% in 1970 (Gibson and Jung, 2005). As a comparison, the black population of Springfield—a much smaller, less industrialized city in Missouri—remained more stable over the same time period, changing from 5.7% to 2.0% (Gibson and Jung, 2005). The same trend can be found in the other major American industrial centers, with the proportion of the black population increasing drastically as waves of rural migrants poured in well into the mid-century. Unfortunately, when black migrants arrived in urban centers, conditions were far from optimal. While there were more options for employment, they faced discrimination and segregation in the work place and were systematically excluded from membership in most unions—particularly at the beginning (Marable, 2000; Lumpkins, 2008).

The 1917 race riot is presented as an illustration—a symptom—of the severity of racial tensions in St. Louis during the period when Robert Terry was building his skeletal collection. Many of the people whose remains would become part of the collection were alive in 1917 and would have experienced the riot or its aftermath. Yet even if they were not present in St. Louis at that time, but instead were born later or arrived in the city after
1917, they almost certainly experienced the tension produced by structural racism during their lives, given its sheer ubiquity throughout the United States. Although data on place of birth are available for some individuals, records are incomplete, and these data are not included in this study. It is safe to assume that many of the individuals in my sample were not native to the St. Louis area, but rather than being problematic for my study, this fact means that observations of embodied racialization are not limited to a local area. Instead, the Terry Collection sample allows me to observe this phenomenon as a more widespread process occurring at the regional or even national level.

Race and Science

The Race Concept

Before continuing further, it is necessary to define what I mean when I use the term race. First of all, race is essentially ideological, not biological: it is a culturally mediated and systematic way of interpreting human phenotypic variation that sorts individuals into arbitrary, discrete categories (Gravlee, 2009). This worldview is hierarchical by definition and operates on a number of assumptions about the categories it creates: that they are numerable, that they are static, that they are generally homogeneous, and most importantly, that they are natural and mutually exclusive divisions of human biology. One of the particularly insidious powers of the race concept is its use of a narrow sampling of superficial phenotypic variations to derive a litany of categorical assumptions about individuals’ characters and personalities. External differences in appearance are grouped together, and racial ideology allows people to be pegged variously as criminal, unintelligent, hyper-intelligent, lazy, threatening, extremist, or even
‘illegal,’ based exclusively on their phenotypic appearance (Thiesmeyer, 1995; Mehan, 1997; Dick and Wirtz, 2011; Saghaye-Biria, 2012).

As an entrenched ideology, race is also a historically constituted process. The particular ways in which Americans conceive of race are largely products of this country’s unique experiences of population-level encounters. Clearly a major event in this history involved the forced migration of the trans-Atlantic slave trade, with its dynamics of ownership and ideology of African inferiority. A wide range of modern ideas about black Americans trace their roots to this period (Smedley and Smedley, 2011), one of which being the assumption of an inverse relationship between musculature and intelligence, since slaves—often quite strong due to the intense labor demands placed on them—were viewed as simple-minded and brutish. Another example is the abhorrent idea still spouted by a number of Americans that black Americans are inherently lazy. This idea has its roots in justifications for slave ownership based on the claim that slave owners were doing their Christian duty of engaging Africans in labor, when their natural inclination was to lie about unproductively (Smedley and Smedley, 2011). As waves of migration brought other population subsets from China, Latin America, various European countries, and even from within America’s borders (Native Americans), these groups were similarly racialized, their characters and appearances stereotyped as essentially inferior to the politically powerful “white” population.

As with any longstanding historical process, the race concept also necessitates an element of erasure when it encounters facts that disprove its central tenets, and the durability that racial ideology enjoys is due largely to this process. A fundamental erasure is the way in which clinal variation in phenotypic expression of traits such as skin color
and facial features over geographic space goes unacknowledged. Instead, the limited, yet stark, variation represented among visible non-white communities in the United States is taken as representative of all human variation. Furthermore, it is erasure that permits a racially minded white American to be personally acquainted with Latinos who entered the United States with legal documentation, while still being able to objectify Latinos in general terms as ‘illegal aliens,’ the discursive logic of which Hillary Dick (2011) elucidates. Likewise, it is erasure that justifies the belief that young black men are more likely to be poor, uneducated, and unemployed than their white counterparts because of some imagined, inherent laziness. This worldview ignores the facts of unequal access to resources, education, and jobs—both historical and contemporary—that have resulted in that state of affairs. Racial categories are imposed on individuals as essential aspects of their social identity—as predictors of class, personality, and values that are assumed to be natural and biologically predisposed. Then in the logic of the racial worldview, those who do not fit the categorical mold are explained away as outliers and exceptions that do not negate the rule. A final element of erasure assumes that racial categories are fixed, and that whiteness as it is imagined today can be mapped recursively onto the past as well. However, whiteness is an unstable category that has undergone changes over time. Immigrants from Eastern and Southern Europe who would be considered white today might not have been when they arrived in the early 20th century (Sacks, 1994; Smedley and Smedley, 2011).

Having established that race is the product of cultural and historical processes, it must be noted that simple truisms along the lines of race is cultural, not biological, while nominally correct, border on the irresponsible in their failure to account for the
overwhelming complexity of racial ideology and how deeply it pervades Western (and now globalized non-Western) culture. To say simply that race is not biological and leave it at that is to ignore the real physical effects that racial identity can have for individuals’ health and wellbeing. As will be explained below, socially constructed racial inequality has very real biological results that belie simplistic notions of race and biology as entirely separable.

History of Race in Physical/Biological Anthropology

Since its inception American physical anthropology has been occupied with the study of race, whether attempting to count the number of races that exist or measuring the differences between identified racial groups (Baker, 1998; Caspari, 2003). Early anthropologists had a great deal of influence on cementing the race concept into American culture, providing scientific legitimacy to the concept by subjecting cultural constructions of race to scientific quantification. Baker (1998) traces the historical connection between anthropology and the development of the race concept, showing how race has been justified using anthropological science. For example, Samuel Morton, a prominent American physician (and pre-professional physical anthropologist) of the 19th century, made a career out of measuring skulls, construing minute differences in brain size (from small samples analyzed with extreme bias) as definitive evidence of a racial hierarchy of intelligence (Gould, 1993). Josiah Nott and George Gliddon (1854) expanded on Morton’s work in their *Types of Mankind*, in which they argued for the theory of polygenism—the idea that distinct races were created and placed by God in different areas of the world—an antecedent to the multiregional hypothesis for human
origins. As research in skeletal biology and morphology coalesced into the discipline of physical anthropology proper, its father and greatest early champion, Ales Hrdlička, made a career out of devising methods to quantify racial differences (Blakey, 1987). The scientific studies that Morton, Hrdlička, and other early physical anthropologists produced had devastating effects not only on Americans of African descent, but also on Native Americans. Their assessment that indigenous Americans were savage and of low intellectual capacity contributed to racist attitudes toward these groups and justified the doctrine of Manifest Destiny that had come to prominence in American politics during the early nineteenth century (Horsman, 1981). The America this ideology envisioned was Anglo-Saxon and Christian, and because anyone else was considered inferior, the collateral damage to Native groups of territorial and economic expansion was seen as justified.

Early physical anthropology also lent credence to Social Darwinist ideology, championed by the likes of Herbert Spencer and Francis Galton, which arose toward the end of the 19th century and endured well into the 20th. This movement held that social inequalities such as poverty and poor health could be attributed to biological, selective maladaptations in poor or sickly individuals (Jeynes, 2011). Social Darwinists included non-white race as one such maladaptive trait and felt justified in their worldview not only by a misappropriation of Darwin’s work, but also by anthropologists who claimed that races could be measured and ranked through scientific means.

Central to the scientific study of race has been the biological race concept, which attempts to separate humans into discrete categories based on external phenotypic variations that are ostensibly tied to other biological and personality characteristics. For
decades anthropology, along with the rest of society, both utilized the biological race concept in research designs and reproduced it through findings that seemed to support the model of race as biology. Scientists like Morton and Hrdlička documented what they viewed as essential (i.e., natural, biological) differences between racial groups, but they made a number of unverifiable assumptions. For example, they held to the ancient Aristotelian model of the Great Chain of Being, maintaining that each ‘race’ had a place in the hierarchy, and they assumed that races were homogeneous (Smedley and Smedley, 2011).

Unfortunately, race science continued well into the 20th century in American physical anthropology, and still has some vestiges today, thanks in large part to the legacy of Harvard’s Earnest Hooton. In addition to his tremendously influential writings—e.g., “Methods of Racial Analysis” (1926), Up From the Ape (1931), and “Plain Statements about Race” (1936)—Hooton also trained a large proportion of the next generation of physical anthropologists, thereby ensuring his ideas would maintain prominence for decades. Carleton Coon, one of Hooton’s students, was still publishing racial typologies in the 1960s that were then reprinted in new editions into the 1970s (Coon, 1973 [1962]; Coon and Hunt, 1974 [1965]).

Today, forensic anthropology—an applied field within physical anthropology that uses osteological methods for medico-legal ends—still adheres to a degree on racial classification. Most forensic anthropologists now refer to ‘population ancestry’ or ‘geographic origin’ instead of ‘race,’ but many of the craniometric methods employed for classifying remains into these

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1 Coon’s (1973 [1962]) The Origin of Races was heavily criticized by a number of his peers at the time (Dobzhansky and Montagu’s (1963) review of the book was particularly scathing), revealing a significant early split between traditional physical anthropologists and more progressive biological anthropologists.
groups (and the names given to each group: mongoloid, negroid, and caucasoid) have remained largely unchanged since Hooton’s time (Ousley et al., 2009). In addition to forensic anthropologists, some scholars working in other areas within physical/biological anthropology continue to maintain that race is a useful concept for their work [see Edgar and Hunley (2009) for a synthesis of views on human variation in contemporary biological anthropology].

However, the majority of the subfield eventually shifted away from racial typology. Sherwood Washburn, another of Hooton’s students, departed meaningfully from his mentor’s ideology and called for a “new physical anthropology” that would embrace new theoretical paradigms, evolutionary and genetic approaches, and population-level analysis (Washburn, 1951). This paper was critical of the contemporary practice of classification as an end in itself, and it helped to usher in major changes in the field, despite some holdouts like the ones mentioned above. Many scholars have even chosen to refer to themselves recently as biological anthropologists rather than physical anthropologists, a testament to the importance of the distinction between the old classificatory approach and those that are more theoretically engaged and integrative. Hereafter, I will use this language as well: when discussing the kinds of studies that produce or tweak classification systems, I will use ‘physical anthropology’; those that fall under Washburn’s new physical anthropology, I will call ‘biological anthropology.’

Race in the Biomedical Literature

Since this study relates to the bioarchaeological visibility of a co-occurring disease process that has been studied extensively in contemporary populations, a review
of the race concept’s use in the biomedical literature is appropriate. A significant body of clinical research exists linking periodontal disease to symptoms and illnesses throughout the body (e.g., Beck et al., 1996; Page, 1998; Li et al., 2000; Holmstrup et al., 2003; Beck and Offenbacher, 2005; Amabile et al., 2008; Williams et al., 2008). However, most of these studies give little consideration to racial identity as a factor affecting disease presentation and severity. A common trend in clinical research is to instead attempt to account for racial health disparities by attributing them to differences in socioeconomic status, thereby assuming that race and status are equivalent and interchangeable. Neglecting to account for these as separate contributors to disease manifestation serves to reduce a highly complex biosocial process to a deceptively simplistic model. This long-standing practice of making disease purely clinical allows issues of racial inequality to be overlooked—a process of erasure that is mutually constitutive between the scientific community and society more broadly. The fact that scientists are themselves subjects occupying particular social contexts has been well established (Greene, 1981; Kincaid et al., 2007); culturally and politically entrenched attitudes that minimize racial inequality as an important factor affecting health outcomes can influence scientists’ research questions, methodologies, and results (Hoberman, 2012). As Gould (1978) has expressed, seemingly empirical and unbiased findings can often be the results of unconscious manipulations that fit data into scientists’ understandings of the world they inhabit, based on their social environment. Findings that fail to account for race reproduce and legitimate these attitudes. Thus, biomedical research can sometimes serve inadvertently as a vector in a circular process through which society reproduces and maintains racial hierarchies.
A similar trend in clinical research is to acknowledge race, but to treat it as a set of biological categories rather than a socially constructed system of classification based very loosely on a few external phenotypic characteristics. Parallel to and informed by societal (mis)understandings of race, much biomedical research tends to assume that observable differences in disease processes and outcomes along racial lines are genetically determined. The underlying assumption of this ideology is that such disparities are the results of essential differences in the biological makeup of discrete racial groups, which has the effect of discounting socially mediated, structural forces that help cause poorer health in racial minorities. This attitude is easily observable in current medical research studies dealing with a wide array of diseases such as type 2 diabetes, heart disease, obesity, stroke, kidney disease, liver disease, and even cancer (e.g., Ferdinand and Nasser, 2013; Harman et al., 2013; Lucas et al., 2013; Nee et al., 2013; Palmer et al., 2013; Phatak et al., 2013; Rockman et al., 2013). The papers referenced above (and many others) discuss differential outcomes based at least in part on racial group, operating as though patients can be easily placed into neat, homogeneous races, and attempting to establish important biological characteristics of each race to the exclusion of social factors. In a particularly revealing study, Shanawani and colleagues (2006) found that of 268 clinical studies published in 2003 that claimed to reach conclusions about the connection between race, genetics, and health outcomes, 72% did not explain their methods for placing participants into racial categories, yet 67% stated conclusions about race, genetics, and health. Their results quantify just how commonly the race concept is conceived as a natural system of categorizing individuals that is so obvious as to need no explanation, even within scientific research.
Even when race is ignored—or at least not mentioned as a salient factor independent of socioeconomic status—a good deal of the biomedical and public health literature does account for nutritional status, which has clear linkages to racial identity. Nutrient deficiency has adverse effects on immune response, infection severity, and overall health outcomes (Scrimshaw and SanGiovanni, 1997; Boyd and Madden, 2003). Protein deficiency is particularly problematic for disease immunity because of the immune system’s dependence on a balance of amino acids needed to facilitate cell replication and synthesize active protein compounds (Scrimshaw and SanGiovanni, 1997). Similar depressions in the function of various immune response mechanisms result from deficiencies of Vitamins A, B, and D, as well as iron and a number of other micronutrients (Hodges et al., 1962; Gross and Newberne, 1980; Bryan and Stone, 1993; Scrimshaw and SanGiovanni, 1997). Adequate levels of each of these nutrients can be maintained with a balanced diet; however, independent of class, racial and ethnic minorities in the United States have more limited access to a healthy, nutritious diet than do white Americans and are therefore at elevated risk of immune dysfunction (Bahr, 2007; Morland and Filomena, 2007; Galvez et al., 2008; Larson et al., 2009; Fahlman et al., 2010; Kant and Graubard, 2012). Consumption of high-quality, nutrient-rich foods is not simply a product of choice, but rather a matter of availability—a fact that demonstrates in part the social inequality that contributes to differential health outcomes between racial groups.
“Race Becomes Biology”

Structural Violence and Racial Embodiment

In the last two decades many biological anthropologists have come to embrace biocultural approaches to their research. The “biocultural synthesis” (Goodman and Leatherman, 1998) acknowledges that human biology is dialectically connected with social and cultural factors: since humans are animals that create meaning from their social relations, this biocultural approach to questions about humans results in richly complex answers. This new paradigm allows me to situate the quantitative osteometric data derived from the Terry Collection sample at the intersection of biological and social factors that affected the life experiences of individuals in this study.

A useful theoretical framework for analyzing racially differential health outcomes is that of structural violence. This concept, developed by Johan Galtung (1969) and elaborated upon by a number of prominent scholars, including Nancy Scheper-Hughes (1992), Philippe Bourgois (2001), and Paul Farmer (2004, 2005), views inequality—especially inequalities in health outcomes and access to healthcare—as a type of violence enacted on the bodies of the oppressed by the political and economic structures of society. Farmer (2004:307) goes so far as to say that “structural violence is violence exerted systematically—that is, indirectly—by everyone who belongs to a certain social order.” It is part of a social machinery of oppression in which all socially powerful members of a society are implicated. Structural violence involves a process of historical erasure that—much like racism—naturalizes inequality as a product of biology, or worse, blames the poor for their own poverty. These effects help to create plausible deniability,
which the privileged classes can use to displace blame from themselves and deny their
complicity in the systematic oppression that maintains their high quality of life at the
expense of the poor.

The concept of structural violence can be applied just as easily to race relations as
to the interactions of economic classes. Higher rates of poor health among racial
minorities have been commonly attributed to a number of factors. One, outlined above, is
a product of biological determinism, and blames genetic differences for these outcomes.
Another, which is parallel to the processes of historical erasure that characterize
structural violence, attempts to account for the comparatively poor health of minorities by
claiming their health is their own fault. Such arguments cite poor dietary behaviors and
ignorance of healthful nutrition as major problems, failing to acknowledge factors that
chronically plague minority groups, such as lower income, limited access to health
education, and low neighborhood availability of healthy foods, all of which can be traced
to structural inequality. While there is some degree of agency at play, and clearly poor
health and nutrition is not a universal characteristic of every individual of racial minority
status, the role of societal pressures on the poor health of racial minorities compared to
white Americans cannot be overstated.

A similar and equally useful line of inquiry into the nature of social inequality lies
in embodiment theory. In this framework, social identity and the negative effects of
structural inequality can be seen “written” on the bodies of the disenfranchised. As
mentioned briefly above, while race is socially constructed, it becomes embodied as the
health, nutrition, and mortality of individuals occupying different racial groups. Instead
of claiming a complete split between race and biology, as has been done in anthropology
for the past few decades, it can be accurately said that “race becomes biology” (Gravlee, 2009:47 [emphasis added]). The concept of embodiment accounts not only for economic inequality, but also for the physiological stress of discrimination and the accumulation of everyday insults that arise from being treated as inferior throughout the life course (Krieger, 1999). Yet the embodiment explanation for racial health inequality should not be mistaken as an argument for the biological basis of race. Instead, it is a way of demonstrating the biological effects of a cultural system. Fausto-Sterling (2008) describes embodiment as a constant feedback system between the body and social experience, calling into question the ways that medical research naturalizes and generalizes racial categories.

Embodiment could at least partially explain the ease with which the effects of inequality become naturalized ideologically. This possibility is true even within the scientific community, since embodied inequalities can be easily mistaken for natural characteristics of a racial group in the context of a social milieu that takes essential racial difference for granted. To demonstrate my point, consider a case of a disease that is commonly thought to present differently in black and white patients due to purely genetic reasons. Kuzawa and Sweet (2009) show evidence that differences in cardiovascular disease morbidity along racial lines may arise from the effects of socially-mediated physiological stress upon the epigenome during development rather than from strictly genetic sources. They suggest that in the case of cardiovascular disease, the embodiment of socially imposed racial categories is the change in epigenetic control of gene expression that was enacted through maternal or developmental stress, and which causes disease later in life as a product of early (indeed, even prenatal) environmental pressures.
Such epigenetic manipulation, though derived as a result of social pressures and structural violence, is heritable, and it thus becomes a biological, inter-generational embodiment of social and racial identity. This kind of evidence calls into question the findings of many biomedical studies, such as the ones cited as examples in the previous section, which attribute unique biological characteristics to the social categories of race, using these to reinforce the commonly held notion that races are discrete, natural, scientifically demonstrable categories.

Skeletal Markers of Race and Social Inequality

Interestingly, the evidence of social inequality and structural violence is not limited to clinical health outcomes in living populations: it can also be observed in skeletal remains. Klaus (2012) provides a model for how to apply the conceptual framework of structural violence—typically one reserved for contemporary sociocultural analysis—to the context of bioarchaeology and osteology. He recommends looking for several indicators, including markers of nutritional deficiency, physiological stress, growth velocity, female fertility, degenerative joint disease, and skeletal infections to point toward systematic structural inequality. Making skeletal remains the objects of analysis for an anthropology of structural violence actually fits quite well into Farmer’s (2004) call for a materialist approach that aims to reveal the mechanisms of structural oppression through an accounting of their physically embodied effects. However, I maintain that structural violence cannot be reduced to a checklist of skeletal markers and specific demographic indicators; rather, it can also be inferred through an examination of subsets of Klaus’s list of indicators in the context of historical patterns as well.
Biological anthropologists have documented a number of specific impacts of inequality on the skeletons of marginalized individuals. Since the discovery of the New York African Burial Ground, Michael Blakey and his colleagues have done a great deal of work to document the biological responses to the conditions of enslavement that are visible in skeletal remains. These researchers have observed a high mortality rate among children and young adults (Blakey, 2001), and the remains of nearly all the children in the burial ground show evidence of anemia (assessed by the presence of porotic hyperostosis) and infectious diseases (Blakey et al., 2000). They have also examined childhood physiological stress via evidence of growth disruption in teeth and long bones, i.e., observations of enamel hypoplasias and Harris lines (Barrett and Blakey, 2011), and efforts have been undertaken to reconstruct dietary patterns through stable isotope analysis (Blakey, 2001). Yet the biological ramifications of slavery, while important to consider here, cannot necessarily be equated with those of structural racism. In a study of a post-slavery African-American cemetery sample in Arkansas, Rose (1989) found a higher than normal occurrence of skeletal lesions, indicating high infection rates and dietary deficiencies. Watkins (2003) found particularly high rates of osteoarthritis in the right shoulders of African-Americans in the W. Montague Cobb Collection, a 20th-century skeletal collection at Howard University in Washington, D.C., which suggests highly strenuous manual labor. In the same vein, Muller (2006) attributes a great deal of the skeletal trauma seen in individuals from the same collection to adverse—indeed dangerous—working conditions, which were to a certain extent byproducts of racial inequality.
While these and a few other studies have explored the effects of race on skeletal biology, the literature is predominately focused on social inequalities more generally, including those based on gender, class, and socioeconomic status. To that end, a wide variety of skeletal markers have been analyzed, including degenerative joint disorder, periosteal lesions, dental caries, enamel hypoplasias, porotic hyperostosis, and growth rates, among many others (Goodman et al., 1980; Goodman et al., 1984; Walker and Hewlett, 1990; Goodman and Rose, 1991; Saunders, 1992; White, 1994; Webb, 1995; Larsen, 1997; Schultz et al., 1998; Tung, 2012). These bioarchaeologically visible markers can easily be deployed as indicators of racial inequality as well.

Furthermore, there is osteological evidence of the intersectionality of various forms of marginalization. Watkins (2012) demonstrated that the severity of disease manifestation and traumatic experience differed by class status within two African-American subgroups represented in the Cobb collection. Carlina de la Cova (2012) has also shown evidence of structural violence enacted in the form of skeletal trauma on bodies of women in the context of mental hospitals. In her study, she notes significant differences in the kinds of trauma suffered by white and black women. Osteological analysis can thus contribute valuably to broader understandings of how race and other forms of social inequality become embodied.

Pathological Conditions Analyzed

Periodontal Disease

Periodontitis is a disease process that refers to the results of anaerobic, gram-negative bacterial proliferation in the sulcus between the tooth surface and the
surrounding gingival tissue (Slots, 2010). Although periodontitis can arise from over 100 species of bacteria, the three that most commonly lead to a disease state in the modern American context are *Aggregatibacter actinomycetemcomitans*, *Tannerella forsythia*, and *Porphyromonas gingivalis* (Dentino et al., 2013; Laine et al., 2013). Prolonged infection with these species results in the disintegration of alveolar bone and gingival tissue at the site of infection; however, this effect is not a direct result of the bacterial activity, but rather of the host’s immune response. Host leukocytes indiscriminately destroy tissue at the site of infection, eroding the host’s own cells along with the bacteria (Liu et al., 2010). Over time compounding tissue damage exposes the tooth root as the sulcus erodes away. Macroscopic observation of this phenomenon constitutes diagnosis of periodontitis both clinically and osteologically (Strohm and Alt, 1998). Eventually, if enough skeletal and gingival tissue surrounding the tooth disintegrates—especially if the periodontal ligament is damaged—the tooth may be lost. The bacterial species responsible for periodontal disease have also been linked to diseases throughout the body, including cardiovascular disease (Hollister et al., 1993; Beck and Offenbacher, 2005; Pucar et al., 2007; Amabile et al., 2008; Reyes et al., 2013; Schenkein and Loos, 2013) and other serious systemic illnesses (Beck et al., 1996; van Winkelhoff and Slots, 1999; Li et al., 2000; Holmstrup et al., 2003; Genco et al., 2005; Williams et al., 2008; Han and Wang, 2013).

Periodontal disease often results in loss of cortical bone of the alveolus (dental sockets); this loss can occur on all surfaces—buccal/labial and lingual—of the alveolar crest (AC), revealing the cancellous bone beneath. This process produces a porous, rough, or grooved appearance of the AC (Clarke and Hirsch, 1991; Larsen, 1997; Strohm
and Alt, 1998). Following the recommendations of Hildeboldt and Molnar (1991) and Clarke and Hirsch (1991), recession of the AC away from the cementoenamel junction (CEJ) is the primary indicator of periodontitis considered in this study, but it is employed with considerable qualification and is corroborated by the presence of the rough, porous texture characteristic of cortical bone loss. A substantial distance between the CEJ and the AC alone is inadequate to diagnose periodontitis—either clinically or in skeletonized remains—as normal physiological responses to aging and occlusal attrition also frequently result in continued tooth eruption and subsequent dentin exposure above the level of the alveolar margin (Clarke and Hirsch, 1991; Varrela et al., 1995; Streckfus et al., 1999; Ogden, 2008).

Furthermore, a difference should be noted between horizontal and vertical AC recession. Recession that appears generally even across the entire alveolar margin is considered horizontal and can be attributed to the non-pathological processes mentioned above. However, recession that appears as a local extreme at only one or more teeth is considered vertical and can be reasonably assumed to have pathological etiology (Clarke and Hirsch, 1991; Hillson, 1996).

Osteoperiostitis

Osteoperiostitis (or simply, periostitis) is characterized by lesions at sites of local skeletal inflammation, caused by bacterial infection or trauma. These lesions represent various stages of new bone formation in bioarchaeological context (Larsen, 1997; Walker et al., 1997; Ortner, 2008; Weston, 2008). Remodeling of the periosteum following infectious modification appears as a raised woven or crosshatch texture of the bone
surface that can be identified visually as well as through palpation of the area (Larsen, 1997). Although periosteal lesions can sometimes be traced to a wide range of infectious diseases through careful differential diagnosis (Ortner, 2008), Weston (2008) questions the reliability of such conclusions, finding that disease progress—and not specific etiology—can be established with a higher degree of certainty from periosteal lesions. In any case, the causative pathogen is not extremely important for this study. Rather, the purpose of accounting for periosteal lesions is to establish whether periodontal disease can be associated with the occurrence of non-specific systemic disease, not to pinpoint which non-oral diseases are present. Still, several common bacteria that cause periodontal disease are also known to cause infections throughout the body, and to do so after migrating from the oral cavity through the bloodstream (van Winkelhoff and Slots, 1999; Li et al., 2000; Holmstrup et al., 2003).

Childhood Physiological Stress

Linear enamel hypoplasias (LEH)—lines of abnormal or incomplete enamel deposition visible on tooth surfaces—have been linked to physiological stress during childhood (Goodman et al. 1980; Goodman et al., 1984; Goodman and Rose, 1991; Schultz et al., 1998; Goodman and Martin, 2002). Enamel is deposited onto permanent teeth as they develop during childhood, while they are still inside the bones of the jaw. Enamel deposition is particularly sensitive to conditions such as nutrition, illness, and even psychological stress, and during times of adverse conditions, resources are diverted away from enamel deposition toward more essential functions to ensure survival. Since enamel is laid down in linear rows at a predictable rate, it is possible to estimate the
timing of childhood physiological stress with considerable precision by observing the location of hypoplasias on adult teeth—lines on the surfaces of teeth where enamel is absent.

Although a number of studies on living populations have suggested that childhood nutritional status does affect health later in life (Gluckman et al., 2008; Kuzawa and Quinn, 2009; Kuzawa and Sweet, 2009; Godfrey et al., 2010; Mensah and Hobcraft, 2008; Thayer and Kuzawa, 2011), bioarchaeologists have had mixed results in establishing a connection between LEH and adult health indicators in skeletal remains. While some have found significant evidence to link LEH with poor health or early mortality (e.g., Cook and Buikstra, 1979; Goodman and Armelagos, 1988), others have found no significant correlation in their samples (e.g., DeWitte and Bekvelac, 2011; Murphy, 2012). This seeming contradiction may be explained by the limited data source available to the osteologist: the only diseases that can be detected in osteological context are those that affect skeletal tissue, whereas the health indicators studied in the examples from living groups above were diseases of soft tissue, such as diabetes and heart disease.
CHAPTER III

MATERIALS AND METHODS

Skeletal Sample

Robert J. Terry Collection

The data presented in this study derives from a sample of 197 white and black adults (race ascribed at autopsy) in the Robert J. Terry Anatomical Skeletal Collection, housed in the Smithsonian Museum of Natural History’s Department of Physical Anthropology. This collection—one of the most heavily researched in the world—contains skeletal remains of over 1,700 individuals (almost exclusively black and white), each with records detailing age at death, sex, and ascribed race. In some cases cause of death as assessed during autopsy, major pathological conditions, and occupation are also available.

Robert Terry was a member of the anatomy faculty in the medical school at Washington University of St. Louis. In 1910 he began collecting and preserving specimens in St. Louis and the surrounding area, which he continued until his retirement in 1941. A colleague, physical anthropologist Mildred Trotter, continued collecting remains until 1967, when the collection was transferred to the Smithsonian. The vast majority of the collection consists of the remains of low socioeconomic status individuals whose families could not afford burial or whose bodies were never claimed (Hunt and Albanese, 2005). It became the charge of the state to dispose of these bodies, and some
eventually arrived in the medical school’s cadaver lab, where Terry cleaned and preserved their skeletons with wax after their autopsy and use as teaching instruments (Hunt and Albanese, 2005). However, Missouri’s Willed Body Law of 1955 required a signed release from a donor or immediate family member allowing human remains to be used for scientific purposes. Mildred Trotter (1981) has reported that in fact after 1955 the collection shifted from mostly unclaimed bodies to willed donations. However, demographic records are incomplete, and it can be difficult to determine the socioeconomic status of specimens from the last decade of collection. Information about occupation is available for some individuals, but not all; their date of death provides the best clue of their economic standing, since those collected before 1955 were all impoverished at the time of their death. Also important for this study is the fact that much of the Terry Collection consists of adults over the age of 45, although a wide spectrum of ages is represented.

Sampling Methods

I have limited my sample to individuals who died between the ages of 25 and 55. This somewhat arbitrary age range minimizes the presence of false positives for periodontal disease due to normal alveolar remodeling that accompanies aging. I have also subdivided the sample into age sets of 10 years for the purposes of assessing the effects of age on the disease processes under study. However, as will be discussed later, measuring the effects of age in this sample is problematic. Furthermore, I limit my sample to specimens collected before 1955. Although it is impossible to truly equalize a diverse group’s experience of class, this study attempts to control for socioeconomic
status in order to focus on the effects of ascribed race. By using only those specimens collected before the Willed Body Law was enacted, I can at least be reasonably certain that these individuals lived under similar conditions of poverty. This criterion should mitigate uncertainty as to the impact of socioeconomic status on disease markers in my sample. Specimens were selected at random from a list of specimens provided by the collections manager, although every effort was made to collect data from approximately equal numbers of males and females, blacks and whites, and age sets within these groups. Unfortunately, however, a relative dearth of young adults (especially white females) exists in the collection. Because dental analysis is central to this study, specimens found to be edentulous (lacking teeth) or nearly so were rejected. This final caveat resulted in a very small sample size (n=3) of black males over the age of 45. Table 2 provides basic data regarding sample size and demographic information about the sample.

Table 2

Sample size and demographic composition.

<table>
<thead>
<tr>
<th></th>
<th>Female</th>
<th>Male</th>
<th>Total</th>
</tr>
</thead>
<tbody>
<tr>
<td>Total Sample</td>
<td>82</td>
<td>115</td>
<td>197</td>
</tr>
<tr>
<td>Black</td>
<td>50</td>
<td>52</td>
<td>103</td>
</tr>
<tr>
<td>White</td>
<td>31</td>
<td>63</td>
<td>94</td>
</tr>
<tr>
<td>YA (25–35)</td>
<td>23</td>
<td>27</td>
<td>50</td>
</tr>
<tr>
<td>MA (36–45)</td>
<td>28</td>
<td>57</td>
<td>85</td>
</tr>
<tr>
<td>OA (46–55)</td>
<td>31</td>
<td>31</td>
<td>62</td>
</tr>
<tr>
<td>Black YA</td>
<td>17</td>
<td>17</td>
<td>34</td>
</tr>
<tr>
<td>Black MA</td>
<td>18</td>
<td>32</td>
<td>50</td>
</tr>
<tr>
<td>Black OA</td>
<td>16</td>
<td>3</td>
<td>19</td>
</tr>
<tr>
<td>White YA</td>
<td>6</td>
<td>10</td>
<td>16</td>
</tr>
<tr>
<td>White MA</td>
<td>10</td>
<td>25</td>
<td>35</td>
</tr>
<tr>
<td>White OA</td>
<td>15</td>
<td>28</td>
<td>43</td>
</tr>
</tbody>
</table>
Assessing Pathological Conditions

Periodontal Disease

Though several osteological methods for assessing periodontal disease exist (Lukacs, 1989; Clarke and Hirsch, 1991; Hildeboldt and Molnar, 1991; Hillson, 1996), several studies have considered a CEJ–AC distance at a tooth locus of >2mm to be positive evidence of periodontitis when accompanied by porosity or fenestration of the alveolar bone (e.g., DeWitte and Bekvalac, 2011). Because it is so common for teeth to erupt naturally in the absence of periodontitis or other pathological conditions, and due to the apparent unsuitability of the standard assessment method to my sample, I made a slight methodological adjustment. Rather than simply measuring each vertical recession, a CEJ–AC measurement was also taken at the left mandibular first molar (M1) for each specimen. Clarke and Hirsch (1991) warn that the practice of considering 2mm of recession to be periodontitis may be too simplistic. Instead, they suggest measuring the level of normal alveolar recession at the first molars (M1) of several individuals of a given age set and comparing the average to suspected sites of periodontitis. The distance at this tooth should account for any normal eruption experienced due to aging or other factors. In other words, it must be assumed that even those teeth at which alveolar bone is experiencing degradation from periodontal infection are erupting for normal reasons at the same rate as other teeth; therefore, it seems reasonable to account for this normal eruption in measuring the CEJ–AC distance of each vertical alveolar recession. However, instead of obtaining an average normal recession value for a group of individuals, I suggest obtaining a standard value for each individual. Rather than comparing an
individual to a group at the risk of overlooking factors—such as population ancestry, sex, or physiological processes—that could cause differences in normal eruption, it seems more prudent to compare an individual to him- or herself. That is, I compared loci of vertical recession to an individual’s own M1 measurement.

In the Terry Collection sample, nearly every individual displayed horizontal (normal) recession with a CEJ–AC distance of more than 2mm, highlighting the usefulness of my methodological adjustment. All measurements were taken with digital calipers calibrated to one one-hundredth of a millimeter. I accounted for recessions in all four quadrants of the mouth, but observation was limited to buccal/labial surfaces. Notably, I excluded loci of pathological vertical recession that also appeared to have sustained postmortem damage. A typical recession due to periodontal disease would be expected to resemble a somewhat sharp parabola with smooth edges; postmortem damage should have a jagged contour, making the parabolic apex unobservable.

A locus of AC recession is defined by tooth. For example, if there was more than one recession at a tooth locus (as sometimes happens at molars), the deeper recession was recorded. The M1 value was subtracted from each measurement of recession in a given individual to arrive at an adjusted CEJ–AC distance for each affected locus. If the left mandibular M1 was absent or pathological recession existed at that locus, an M1 in another quadrant was measured. Since there is negligible difference between the timing of eruption of maxillary and mandibular first molars, this substitution should not result in a miscalculation of the standard measurement. An adjusted recession value of >2mm was considered positive evidence of periodontitis. Since Clarke and Hirsch (1991) warn that
periodontal disease may sometimes be over-reported, using the adjustment factor helps to reduce the occurrence of false positives.

To illustrate this method, let us take a hypothetical case in which two loci of vertical alveolar recession are observed in one individual, and the surface of the alveolar bone exhibits a rough appearance at each locus. The first has a CEJ–AC distance of 4.78mm, and the second, 8.35mm. The CEJ–AC distance at the individual’s left mandibular M1 was measured to be 2.93mm. In this case, the CEJ–AC distances at the sites of suspected periodontitis will be adjusted to 1.85mm (4.78mm – 2.93mm) and 5.42mm (8.35mm – 2.93mm). Because only the latter adjusted value is greater than 2mm, only that locus is considered positive for periodontitis.

An individual exhibiting at least one vertical recession locus, accompanied by porosity in the alveolar crest, was scored positive for periodontitis. Periodontitis severity scores from 0 to 3 (based on the number of vertical alveolar recessions present) are utilized for simply and quickly comparing trends in periodontal disease severity (Table 3). A score of 0 indicates the absence of vertical alveolar recessions and no periodontitis; 1 indicates 1-3 vertical recessions (mild); 2 indicates 4-6 recessions (moderate); and 3 indicates more than 6 recessions (severe).

Table 3

<table>
<thead>
<tr>
<th>Severity scoring system for periodontitis</th>
</tr>
</thead>
<tbody>
<tr>
<td>0</td>
</tr>
<tr>
<td>1</td>
</tr>
<tr>
<td>2</td>
</tr>
<tr>
<td>3</td>
</tr>
</tbody>
</table>

36
Although loss of alveolar bone due to severe periodontal disease has been associated with antemortem tooth loss (AMTL), I did not take tooth loss into account for the purposes of this study because of the diversity of its etiology (Larsen, 1997). Furthermore, alveolar recession at teeth adjacent to sites of antemortem tooth loss was considered unobservable, following Hillson’s (1996) method, since the alveolar recession and remodeling associated with AMTL can extend to neighboring teeth as well. Additionally, if vertical recessions were present at teeth on which the CEJ was absent due to severe occlusal wear or postmortem damage, they were also considered unobservable and were not measured. Figure 1 illustrates the types of alveolar recession that characterize periodontitis.

Figure 1. Mandibular incisors, canines, and alveolus—labial surface. Vertical alveolar recessions associated periodontitis occur at varying levels of attrition are present on all anterior teeth. Individual also has supernumerary incisors. Terry Collection, Smithsonian NMNH. Photo by the author.
Osteoperiostitis

I scored the presence or absence of periosteal lesions—either active or healing—visually on the left and right femur, tibia, and fibula of each specimen. Although many studies of periostitis look for lesions on the anterior tibia because of the high frequency with which lesions appear there (Eisenberg, 1991; Milner, 1991; DeWitte and Bekvalac, 2011), Goodman and Martin (2002) suggest that because the anterior tibia’s anatomical superficiality leaves it particularly prone to trauma, periosteal lesions located there may not be the most reliable indicators of systemic infection. Therefore, lesions on the anterior tibia were considered, but examined very carefully to differentiate pathological versus traumatic causation. In all cases, lesions of pathological etiology were scored on a scale from 1 (mild) to 5 (severe). Following Buikstra and Ubelaker (1994), I recorded the area of a skeletal element (or anatomical aspect of the element) that was affected by periosteal lesions, in three categories: <1/3, 1/3–2/3, or >2/3. However, other effects of periostitis were also considered, based on qualitative assessment: woven/crosshatch or sclerotic appearance, abnormal diaphyseal expansion (thickening or broadening of the bone), spiculation and other abnormal bone growth, and deformation—phenomena described in Ortner and Putschar (1981), Buikstra and Ubelaker (1994), Larsen (1997) and Roberts and Manchester (2005). Periostitis severity scores were assigned based on these combined quantitative and qualitative criteria. Lesions arising from trauma were recorded but not included in this study’s periostitis analysis. Table 4 outlines the severity scoring system, and Figure 2 illustrates periosteal lesions of various severities.

Periostitis data are reported in composite form for each specimen, meaning that the lesion(s) of greatest severity from either the left or right of any given skeletal element
(femur, tibia, fibula) are reported as the score for an individual. Any periosteal lesion of infectious etiology on any skeletal element observed was scored. No comparison is made between skeletal elements, either within individuals or groups in the sample.

Table 4

Severity scoring system for periostitis

<table>
<thead>
<tr>
<th>Score</th>
<th>Description</th>
</tr>
</thead>
<tbody>
<tr>
<td>0</td>
<td>No lesions</td>
</tr>
<tr>
<td>1</td>
<td>Mild (&lt;\frac{1}{3}) element affected, superficial rxn, no other abnormalities</td>
</tr>
<tr>
<td>2</td>
<td>Mild—Moderate (\frac{1}{3} - \frac{2}{3}) element affected, mainly superficial rxn</td>
</tr>
<tr>
<td>3</td>
<td>Moderate (\frac{2}{3} - \frac{3}{3}) element affected, some abnormalities</td>
</tr>
<tr>
<td>4</td>
<td>Moderate—Severe (\frac{3}{3} ) element affected, significant abnormalities</td>
</tr>
<tr>
<td>5</td>
<td>Severe (&gt;\frac{3}{3}) element affected, extreme abnormalities</td>
</tr>
</tbody>
</table>

Figure 2. Examples of periosteal lesions. (A) Mild. Right tibial fragment, lateral aspect. Teaching collection, Univ. of Notre Dame. (B) Moderate. Left distal fibula. St. Stephens skeletal collection, Univ. of Notre Dame. (C) Severe. Fused femur and tibia. Terry Collection, Smithsonian NMNH. Photos by the author.
Childhood Physiological Stress

As DeWitte and Bekvalac (2011) did in their study, I assess whether childhood physiological stress is associated with periodontal disease or systemic illness later in life using linear enamel hypoplasias (LEH) as the primary indicator. I scored LEH macroscopically on the anterior teeth of each individual examined. Following Goodman et al. (1980), I observed only the maxillary incisors and mandibular canines for LEH, as these teeth are most sensitive to enamel hypoplasia and are therefore the most reliable indicators of stress. An individual was scored positive for LEH if it was observed on at least one of these teeth. I considered LEH unobservable only if none of these teeth were present or if the only ones present were too damaged to observe the labial surface. In total, twenty-three individuals (11.7% of the total sample) were excluded from these calculations because LEH could not be observed on the maxillary incisors or mandibular canines, either because these teeth were too damaged to assess or because they were missing (due to antemortem tooth loss or postmortem damage).

Statistical Analyses

To test for a correlation between periodontitis and periostitis, I ran a McNemar’s test (also known as McNemar’s chi-square). This test is similar to chi-squared, but instead of comparing dichotomous variables between two groups of subjects, it compares dichotomous variables within the same subject. In this case, McNemar’s test allowed me to evaluate whether individuals who have periodontitis are also likely to have periostitis. I used Spearman’s rank-order correlation to test whether a correlation exists between periodontal and periosteal disease severity, as well as between age and the severity of
each disease state. I used Pearson’s chi-squared tests when 2x2 contingency tables were appropriate to compare two groups with respect to presence/absence data (e.g., Is there a difference between the rates of periodontitis in males and females?). To test whether statistical differences exist between age sets with respect to each disease state, I employed Kruskal-Wallis tests.

Mann-Whitney U tests were used to compare each racial group and sex group with respect to each disease state and to childhood physiological stress (LEH). Mann-Whitney U also proved useful in testing whether disease severity is correlated with race, sex, or childhood physiological stress in this sample (comparing ordinal data with categorical/nominal data in each case). All statistical data reported in this study were produced using SPSS version 20.
CHAPTER IV

RESULTS AND DISCUSSION

Link between Periodontitis and Periostitis

As expected, a strong correlation exists between the presence of periodontitis and periostitis within individuals in the sample ($P<0.000$; see Table 5). Of the 77 individuals presenting with evidence of periodontitis, 54 (70%) of them also had periostal lesions. However, of the 137 individuals with periostal lesions, less than half (39%) showed signs of periodontitis. Yet these results are not contradictory, as they might seem at first glance; they confirm the results of biomedical and dental studies that show periodontal disease to be a strong predictor for more systemic disease processes, while suggesting that the relationship is unidirectional. This one-way relationship makes sense given what we know about the co-occurrence of oral and systemic disease in the clinical context. The inflammation and bleeding that characterize periodontal disease provide the conditions for bacteria that inhabit the oral cavity to enter the bloodstream and migrate throughout the body, including to skeletal tissue. However, the process does not operate in the opposite direction.

Most importantly, the strong positive correlation between periodontitis and periostitis in this sample demonstrates that the relationship between oral and systemic disease processes may be bioarchaeologically visible. Still, given that this study is only
the second to investigate this link using skeletal indicators, additional work in other bioarchaeological contexts is needed.

Table 5

Results of the McNemar test for association between periodontitis and periostitis within individuals

<table>
<thead>
<tr>
<th>Periostitis</th>
<th>Periodontitis</th>
<th>Present</th>
<th>Absent</th>
<th>Total</th>
</tr>
</thead>
<tbody>
<tr>
<td>Present</td>
<td></td>
<td>54</td>
<td>83</td>
<td>137</td>
</tr>
<tr>
<td>Absent</td>
<td></td>
<td>23</td>
<td>37</td>
<td>60</td>
</tr>
<tr>
<td>Total</td>
<td></td>
<td>77</td>
<td>120</td>
<td>197</td>
</tr>
</tbody>
</table>

\[ P = 0.0001 \]

Despite the statistically significant correlation between periodontitis and periostitis, Spearman’s test shows there is no correlation between the severities of each disease state in the overall sample. As the number of alveolar recessions increases, there is no correlated increase in periostitis lesion severity. Figure 3 plots each individual with evidence of both periodontal disease and periosteal lesions by the number of alveolar recessions and the periostitis severity score. Therefore, although I demonstrate a clear link between oral and systemic disease in the Terry Collection sample, it appears that systemic disease does not become more severe as oral disease progresses in severity.

There are at least four potential explanations for this result. (1) It is important to remember that periosteal lesions are sites of healing following injury or infection that typically become obliterated after the bone is completely healed. A periosteal infection that occurs early in life may no longer be visible a decade later, whereas alveolar
recessions associated with chronic periodontitis appear to be progressive and non-reversible without advanced stem cell or biomaterial treatments that were unavailable in the early 20th century (e.g. Chen et al., 2012; Vaquette et al., 2012). Therefore, it is possible that bacteria migrating into the bloodstream as a product of periodontal infections that occurred several years before a person’s death may have resulted in a periosteal infection that had completely healed by the time of death. While the periosteal lesions may have vanished, or may even be at a state of healing that mimics the appearance of a mild lesion, older alveolar recessions remain. (2) On the other hand, the severity classification system used for periodontitis may contribute to the absence of correlation between disease severities. Rather than relying on the vertical height of alveolar recessions, I consider the number of significant recessions to indicate severity. This is quite different from the periostitis severity scoring method, which assesses the degree of bone damage and remodeling associated with a given infection, not the number of distinct infections. It is possible that bacteria entering the bloodstream from a single periodontal infection (which would be considered mild periodontitis in my scoring system) could have resulted in a moderate or severe periosteal infection. Conversely, repeated periodontitis episodes may only result in mild periosteal infections. (3) Because periodontal disease can cause tooth loss, some individuals who scored with low periodontitis severity could have already lost some of their teeth to periodontal disease, which would cause severity underestimation. Therefore, some of those with high periostitis severity scores and low periodontitis scores may just be missing teeth at loci of periodontal disease. (4) Finally, not all periosteal lesions are the results of infection by oral bacteria, and not all periodontal infections can be said to cause periostitis.
Interestingly, Pearson’s chi-squared test showed the difference between blacks and whites in this sample based on a simple presence/absence comparison of periodontal disease is not quite significant \( (P=0.096) \); however, the black subset of the sample does have a significantly higher rate of moderate-to-severe periodontal disease than the white subset \( (P=0.001) \). It is well established that the individuals who comprise this sample occupied a low socioeconomic status (at least at the time of their death), and since even the best dental medicine of the early twentieth century was limited, it follows that the poor—regardless of race—would have poor dental health. Indeed, this can be observed in living populations today (e.g., Hudson et al., 2007). Yet the differences in severity

![Figure 3. Distribution of periodontitis and periostitis severity within individuals. Periodontitis severity is plotted as the number of alveolar recessions observed. Bubble size corresponds to the number of individuals occupying a given datum point. No correlation exists between the severities of these diseases in this sample.](image-url)
between whites and blacks in this sample demonstrate that these racial groups likely did have different experiences that led to their respective oral health outcomes.

It should be noted again that the figures I report for periodontitis might be somewhat conservative because I did not account for antemortem tooth loss (AMTL) when I collected my data. Since periodontitis is only one of many reasons for AMTL, I chose not to include AMTL as a sign of periodontal disease and thereby risk polluting my periodontal measurements with potentially false positives. Therefore, it is likely that some of the many missing teeth I observed but did not record were absent due to periodontitis, making oral disease more prevalent and/or more severe in this sample than I have reported.

With respect to systemic disease (periostitis), the black subset of the sample has a significantly higher rate of periostitis than the white subset \((P=0.011)\). Mann-Whitney U tests show that the overall severities of both periodontal disease and periosteal disease are significantly higher in the black subset of this sample than the white subset. However, there is no significant difference in the rates of LEH between the black and white subsets (Table 6).

Table 6

*Results of Pearson’s \( \chi^2 \) and Mann-Whitney U tests for correlation between race and periodontal disease, periosteal disease, and childhood physiological stress. Significant results in bold.*

<table>
<thead>
<tr>
<th>Periodontal Disease</th>
<th>Pearson’s ( \chi^2 ) results</th>
</tr>
</thead>
<tbody>
<tr>
<td></td>
<td>n Present</td>
</tr>
<tr>
<td>Black</td>
<td>77</td>
</tr>
<tr>
<td>White</td>
<td>60</td>
</tr>
</tbody>
</table>
### Table 6—continued

<table>
<thead>
<tr>
<th></th>
<th>Black</th>
<th>White</th>
</tr>
</thead>
<tbody>
<tr>
<td>Periodontal Disease</td>
<td></td>
<td></td>
</tr>
<tr>
<td>Moderate – Severe</td>
<td>36</td>
<td>14</td>
</tr>
<tr>
<td></td>
<td>67</td>
<td>80</td>
</tr>
<tr>
<td></td>
<td><strong>10.440</strong></td>
<td><strong>0.001</strong></td>
</tr>
<tr>
<td></td>
<td>49</td>
<td>28</td>
</tr>
<tr>
<td></td>
<td>54</td>
<td>66</td>
</tr>
<tr>
<td></td>
<td><strong>6.530</strong></td>
<td><strong>0.011</strong></td>
</tr>
<tr>
<td>Black</td>
<td></td>
<td></td>
</tr>
<tr>
<td>White</td>
<td></td>
<td></td>
</tr>
<tr>
<td>LEH</td>
<td>54</td>
<td>28</td>
</tr>
<tr>
<td></td>
<td>43</td>
<td>66</td>
</tr>
<tr>
<td></td>
<td>0.612</td>
<td>0.434</td>
</tr>
</tbody>
</table>

### Mann-Whitney U results

<table>
<thead>
<tr>
<th></th>
<th>Mean Rank</th>
<th>P</th>
</tr>
</thead>
<tbody>
<tr>
<td>Periodontal Disease</td>
<td></td>
<td></td>
</tr>
<tr>
<td>Black</td>
<td>103</td>
<td>109.99</td>
</tr>
<tr>
<td>White</td>
<td>94</td>
<td>86.96</td>
</tr>
<tr>
<td>Periosteal Disease</td>
<td></td>
<td></td>
</tr>
<tr>
<td>Black</td>
<td>103</td>
<td>105.83</td>
</tr>
<tr>
<td>White</td>
<td>94</td>
<td>91.52</td>
</tr>
</tbody>
</table>

### Sex

The proportion of females with periodontal disease in a simple presence/absence comparison is not quite significant (P=0.061); however, when mild cases of periodontitis are discounted, females do have a significantly higher proportion of moderate-to-severe periodontitis than males (P=0.040). Again, given a similar class status between the racial groups represented in this sample, it is to be expected that a similarly high proportion of individuals would have at least mild levels of periodontal disease. Mann-Whitney U results confirm the greater severity of periodontal disease in females compared to males. Calcium and Vitamin D deficiency have been linked to increased risk of periodontal disease in modern populations (Hildeboldt, 2005; Boggess et al., 2011), and undernourished women have been found to lack adequate levels of these nutrients during pregnancy and lactation (Mardis et al., 1999). Since the women in this sample were impoverished as well, it is unlikely that their nutritional needs were being met.
On the other hand, females in this sample do not have a significantly higher proportion of periostitis than males, nor is this disease process more severe in females. As with race and childhood physiological stress, there is no significant difference in the presence of linear enamel hypoplasias (LEH) between males and females (Table 7).

Table 7

Results of Pearson’s $\chi^2$ and Mann-Whitney U tests for correlation between sex and periodontal disease, periosteal disease, and childhood physiological stress. Significant results in bold.

<table>
<thead>
<tr>
<th></th>
<th>Pearson’s $\chi^2$ results</th>
<th>Mann-Whitney U results</th>
</tr>
</thead>
<tbody>
<tr>
<td></td>
<td>$n$ Present</td>
<td>$n$ Absent</td>
</tr>
<tr>
<td>Periodontal Disease</td>
<td></td>
<td></td>
</tr>
<tr>
<td>Female</td>
<td>63</td>
<td>19</td>
</tr>
<tr>
<td>Male</td>
<td>74</td>
<td>41</td>
</tr>
<tr>
<td>Moderate – Severe Periodontal Disease</td>
<td></td>
<td></td>
</tr>
<tr>
<td>Female</td>
<td>27</td>
<td>55</td>
</tr>
<tr>
<td>Male</td>
<td>23</td>
<td>92</td>
</tr>
<tr>
<td>Periosteal Disease</td>
<td></td>
<td></td>
</tr>
<tr>
<td>Female</td>
<td>38</td>
<td>44</td>
</tr>
<tr>
<td>Male</td>
<td>39</td>
<td>76</td>
</tr>
<tr>
<td>LEH</td>
<td></td>
<td></td>
</tr>
<tr>
<td>Female</td>
<td>44</td>
<td>29</td>
</tr>
<tr>
<td>Male</td>
<td>58</td>
<td>44</td>
</tr>
</tbody>
</table>

Childhood Physiological Stress

As predicted, McNemar tests showed a significant correlation within individuals between LEH and periostitis in this sample, as well as between LEH and periodontal disease (see Table 8). These results coincide with findings in the biomedical literature.
that demonstrate a strong connection between poor health in childhood and poor health in adulthood. Still, some bioarchaeological studies have been ineffective in confirming this link using skeletal materials (e.g., DeWitte and Bekvelac, 2011; Murphy, 2012). This strange outcome may be due to the ubiquity of enamel hypoplasias in bioarchaeological samples, which could throw off or render statistically meaningless the potential connections between LEH and disease markers.

The demonstrated correlation between childhood physiological stress and adult illness in the Terry Collection sample suggests that the stressors underlying LEH may have been maintained into adulthood and contributed to oral and systemic disease, or they may have impacted the immune system in such a way that even as adults, individuals who experienced stress as children were more susceptible to disease. However, these results are not sufficient to suggest a true causative relationship between childhood physiological stress and adult disease, nor can the link between oral and systemic illness be explained solely in terms of childhood stressors. The fact that LEH is correlated to adult disease states in the sample as a whole but not to one sex or ascribed race indicates that perhaps malnutrition and other early stressors were shared experiences among many of the St. Louis-area poor regardless of positional differences.

Table 8

*Results of McNemar tests for correlation between LEH and pathological conditions within individuals*

<table>
<thead>
<tr>
<th></th>
<th>Present</th>
<th>Absent</th>
<th>Total</th>
</tr>
</thead>
<tbody>
<tr>
<td><strong>Periostitis</strong></td>
<td></td>
<td></td>
<td></td>
</tr>
<tr>
<td>Present</td>
<td>39</td>
<td>29</td>
<td>68</td>
</tr>
<tr>
<td>Absent</td>
<td>61</td>
<td>45</td>
<td>106</td>
</tr>
<tr>
<td><strong>Total</strong></td>
<td>100</td>
<td>74</td>
<td>174</td>
</tr>
</tbody>
</table>

*P=0.001*
Unsurprisingly, Kruskal-Wallis tests show that the number of alveolar recessions increases significantly in consecutive age sets ($P=0.023$; see Table 9). Given consistently poor oral hygiene and little or no access to dental care, it is reasonable that more loci of alveolar recession would accrue over time. However, there is no significant increase in the severity of periosteal lesions in consecutive age sets ($P=0.298$). Again, this result is not entirely surprising. As noted above, periosteal lesions are sites of tissue healing that may become invisible when the process is complete. Still, older adults are typically more susceptible to disease than younger adults, making it plausible that frailty might play a role (Wood et al., 1992); on the other hand, since no individuals in the sample were older than 55 when they died, the frailty factor of age should not be particularly significant.

Unfortunately, because some demographic categories are severely underrepresented in the Terry Collection or because of very low numbers of usable samples (e.g., young adult white females and older adult black males; see Table 2), the results I report with regard to age may be unreliable. However, despite being skewed toward a younger median age at death—36 for the black sample vs. 44 for the white—the black sample shows higher rates of both periodontal and periosteal disease than the white
sample. This result indicates that the embodied effects of race can be explained outside the context of aging processes.

### Table 9

*Results of Kruskal-Wallis tests for correlation between pathological conditions and age. Both sexes and ascribed races are combined in each age set. Periodontitis severity is based on number of vertical alveolar recessions. Significant results in bold.*

<table>
<thead>
<tr>
<th>Age</th>
<th>n</th>
<th>Mean Rank</th>
<th>P</th>
</tr>
</thead>
<tbody>
<tr>
<td>Periodontitis Severity</td>
<td></td>
<td></td>
<td></td>
</tr>
<tr>
<td>25–35</td>
<td>50</td>
<td>107.93</td>
<td></td>
</tr>
<tr>
<td>36–45</td>
<td>85</td>
<td>105.42</td>
<td><strong>0.023</strong></td>
</tr>
<tr>
<td>46–55</td>
<td>62</td>
<td>83.00</td>
<td></td>
</tr>
<tr>
<td>Periostitis Severity</td>
<td></td>
<td></td>
<td></td>
</tr>
<tr>
<td>25–35</td>
<td>50</td>
<td>102.80</td>
<td></td>
</tr>
<tr>
<td>36–45</td>
<td>85</td>
<td>92.71</td>
<td>0.298</td>
</tr>
<tr>
<td>46–55</td>
<td>62</td>
<td>104.56</td>
<td></td>
</tr>
</tbody>
</table>

### Conclusions

As expected, there is a significant correlation between oral and systemic disease in the Terry Collection sample. Although not surprising, these results demonstrate an interesting parallel with current biomedical research and highlight the importance of examining co-occurring disease processes in skeletal remains. More significantly, however, this study highlights the complexity of disease states that can be studied using bioarchaeological methods, not just as potentially co-occurring alongside other diseases and compromising stressors, but also as existing in a dialectical relationship with social processes. My results show a higher rate of both oral and systemic disease in blacks relative to whites in the sample, pointing to the effects of racial inequalities on the bodies of black individuals. The intersection of structural inequalities with skeletal biology is of
growing interest to bioarchaeologists, but its potential for productive research remains largely untapped.

This study’s results indicate that the biological effects of the social categories of structural racism are readily visible in skeletal remains. The concept of structural violence, as well as insights from embodiment theory, are particularly useful for understanding the biocultural processes that take place at the intersection of racial inequality and skeletal biology. However, these frameworks are currently underutilized in skeletal analysis, and the singular, laundry list approach to structural violence that does exist in the bioarchaeological literature (Klaus, 2012) may be too narrow to capture the breadth of structural inequalities that manifest themselves in the skeleton. Such systematic methods for assessing skeletal remains in light of structural violence and embodiment are needed, but they should take a broader view. Even a decade ago, many bioarchaeologists were still engaged in anemic descriptive studies (Armelagos and Van Gerven, 2003), but more recently they have asked important questions about the intersection of bodies with social process (Larsen, 2010). Some examples include Agarwal’s (2011) important edited volume *A Social Bioarchaeology*, work on bioarchaeological approaches to identity and ethnogenesis (Knudson and Stojanowski, 2009; Stojanowski, 2010; Schrader, 2013) and several contributions to a recent special issue of the American Journal of Physical Anthropology on health and stress (Klaus, 2014; Reitsema and McIlvaine, 2014; Temple and Goodman, 2014; Vercellotti et al., 2014). My application of the social theories of structural violence and embodiment aligns well with this trend in bioanthropological research that embraces theoretically rich, biocultural analyses.
Outside the disciplinary context of anthropology, bioarchaeological research—particularly studies like this one—can help problematize trends in the biomedical sciences that examine diseases outside of their social context. Many anthropologists have critiqued this practice, but side-by-side comparisons of disease processes using discreet biomarkers could have a more significant impact than theoretical arguments alone. The necessity for anthropologists to challenge disease research that erases social environments of inequality or conflates social categories with biological ones is urgent. Bioarchaeology’s mixed qualitative and quantitative methods, along with the historically close ties to disease research that paleopathologists have cultivated, place these scholars in an ideal position to engage in this critical dialogue.

Though descriptive and methodological papers have perhaps been overrepresented in the skeletal biology and paleopathology literatures, the important issue of how to assess periodontal disease in the dry skeleton remains unresolved. The novel method I propose appears to work reasonably well for the Terry Collection sample, but more research is needed to assess the accuracy, efficacy, and applicability of this method for osteological analyses more broadly. However, since my method was more stringent than most others, any negative impact it may have had on my results favors a conservative underestimation of periodontal disease. Therefore, if anything, rates of periodontitis in my sample may be even higher than I report.

Still, viable analytical alternatives do exist. For example, I could have compared each locus of vertical alveolar recession to a sample mean or to discrete demographic cohort means using z-scores. The method I chose was designed to eliminate problematic comparisons between an individual and a sample with a very wide range of recession
severity, which may have distorted the analytical power of my data. However, the z-score method would have avoided other potential pitfalls, such as the possibility that the periodontal disease processes affecting a locus where recession was observed may also have been at play at the M1. Future research will compare these methods and assess which is most suitable for the data.

This study represents exciting possibilities for bioarchaeological inquiry into the biocultural processes of embodied racism and other forms of inequality. By adopting the theoretical tools of structural violence and embodiment as explicit foci of their research, bioarchaeologists and skeletal biologists can become more engaged with other researchers, not only in biological anthropology, but also in other subfields of anthropology and the social and natural sciences more broadly.


