Biocultural Analysis of Otitis Media and its Relationship to Traditional Skeletal Stress Markers in the Assessment of Structural Violence

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This dissertation explored the relationship between traditional skeletal stress markers and mastoid air cell system (MACS) hypopneumatization, as a measure of otitis media (OM), using the proposed *biocultural skeletal stress marker pathway* to examine structural violence. Low socioeconomic status and marginalization are known risk factors of OM resulting from immunosuppression and increased pathogen exposure. Structural violence, which is the unequal distribution of resources in a society, is expected to create stressful conditions resulting in an increase in OM prevalence among the marginalized segment of the society.

Structural violence was examined using skulls from the Hamann-Todd Human Osteological Collection (n=20) and Robert J. Terry Anatomical Skeletal Collection (n=105). The Tigara, Point Hope Skeletal Collection (n=69) was a control group that did not experience structural violence and was expected to have a low prevalence of OM. The skeletal stress markers included in this dissertation were limited to the cranium and included porotic hyperostosis, cribra orbitalia, sinusitis, antemortem tooth loss, periapical granulomatous lesions, periodontal disease and dental enamel hypoplasia. Correlations between MACS hypopneumatization, measured skeletal stress markers and skeletal collections were made using the Chi-square test of independence, Student's t-test and analysis of variance ($p<0.05$).

The results suggest that MACS hypopneumatization is a skeletal stress marker capable of delineating differences in marginalization and structural violence. MACS hypopneumatization

was lower than expected in the Terry collection and co-occurred with porotic hyperostosis, which is a measure of anemia. Clinical data suggests that anemia exacerbates OM resulting in more frequent OM episodes. The results suggest that structural violence created conditions, such as anemia, in the marginalized group which increased OM prevalence. However, it is also likely that some individuals experienced OM complications of hearing loss and language delays which were perceived as learning disabilities and were carried into adulthood resulting in lower socioeconomic status and incorporation into the skeletal collections examined. The relationship between OM and anemia should continue to be tested within other populations, using additional theoretical models and incorporating postcranial skeletal stress markers using the proposed *biocultural skeletal stress marker pathway*.

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Preface

This amazing journey would not have been possible without the support of my mentors, friends and family. First and foremost, I would like to thank Dr. Margaret Judd, my graduate advisor. She has been fundamental in helping me develop my dissertation research and has seen me through research projects, grant proposals, and numerous drafts. Her continued support and guidance have been instrumental in my progress and success. I also owe gratitude to Dr. Seth Weinberg who graciously took his time to research and publish with me; thank you for the support and advice throughout the years. I would also like to thank Dr. Tara Meyers, the Assistant Dean for the Dietrich School of Arts and Sciences Graduate Studies and my supervisor, for always lending an ear and sharing advice. I would like to thank my committee- Dr. Mike Siegal, Dr. Mark Mooney and Dr. Doug Swarts- not just for their guidance and constructive feedback on my dissertation, but for the years of instruction and advice.

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1.0 Introduction

This dissertation hypothesized that individuals in marginalized populations subjected to structural violence experienced sufficient stress to overwhelm biological and cultural buffering systems, which was embodied as an increased risk for otitis media (OM) detectable in skeletal tissue as mastoid air cell system (MACS) hypopneumatization. The effects of stress on the body are diverse but anthropological emphasis has been placed on skeletal stress markers of growth and metabolic disturbances (e.g., dental enamel hypoplasia, porotic hyperostosis, cribra orbitalia, reduced stature and mortality) (Geber 2014; Goodman et al. 1984b; Walker et al. 2009). Skeletal stress markers capture physiological disruptions that occur when sociocultural and physiological buffering systems are insufficient to maintain biological homeostasis (Goodman et al. 1988). Frequencies of these markers can delineate marginalized groups within a population (Geber 2014; Goodman et al. 1984b; Goodman et al. 1988; Offenbecker and Case 2015; Wright 1997). It was hypothesized that MACS hypopneumatization is a skeletal stress marker resulting from OM that enables inferences about pathogen exposure and immunodepression in a population, and can be used to assess structural violence, marginalization and socioeconomic status in past populations.

OM prevents the development of the MACS during infancy, childhood and adolescence resulting in MACS hypopneumatization (Cambon et al. 1965; Mey et al. 2006; Paradise et al. 1997). MACS hypopneumatization is an attractive addition to the suite of skeletal stress markers, because it can inform about the social theory of structural violence by using a biocultural framework to model events that trigger immunosuppression and pathogen exposure thus increasing OM risk. In addition the temporal bone and mastoid process typically preserve well in the archaeological record and therefore MACS hypopneumatization is a consistently measurable

pathology across geographically and temporally diverse populations (Flohr and Schultz 2009a; Flohr and Schultz 2009b). Finally MACS hypopneumatization has a single etiology and OM has a well-defined pathogenesis which allows for specific interpretations about stress and disease in past populations (Bluestone and Klein 2007).

For this dissertation the *biocultural skeletal stress marker pathway* was applied to the social theories of structural violence and evolution and adaptation to chronic cold to assess the viability of MACS hypopneumatization as a skeletal stress marker. Structural violence was tested in the Hamann-Todd Human Osteological Collection (Hamann-Todd) and the Robert J. Terry Anatomical Skeletal Collection (Terry). Evolution and adaptation to chronic cold stress was tested in the Tigara, Point Hope Skeletal Collection (Tigara) which served as a control because it was expected to have a low prevalence of OM. These theoretical models were applied to the *biocultural skeletal stress marker pathway* which combines the biocultural approach, incorporating social, cultural and physical environments, with biological responses to reconstruct marginalization and structural violence from skeletal stress markers (Armelagos and Van Gerven 2003; Buikstra and Cook 1980). Structural violence and marginalization increase the risk of pathogen exposure and psychological stress which increases the risk of OM and MACS hypopneumatization (Bentdal et al. 2010; Bruce et al. 2000; Cohen et al. 2007; Cohen et al. 1991; da Costa et al. 2004; Engel et al. 2005; Kucur et al. 2015; Paradise et al. 1997; Uhari et al. 1996). Environmental and cultural factors that regulate OM risk (e.g., breastfeeding, child daycare, secondhand smoke, and maternal marital status) may buffer against or contribute to stress (Bluestone and Klein 2007; Goodman et al. 1988; Schell 1997). Cultural stressors that reinforce health disparities are socioeconomic status, health behaviors and structural violence (Dressler et al. 2005), and psychological stress stemming from these cultural circumstances downregulates immune response resulting in increased susceptibility

to pathogens (Cohen et al. 1991). These stressors have the potential to disrupt homeostasis resulting in stress and disease, signs of which may be expressed as growth disruption, disease and death, which can be interpreted from skeletal tissue (Goodman et al. 1988). This research is an application of structural violence to the *biocultural skeletal stress marker pathway* to determine if MACS hypopneumatization is a useful skeletal stress marker.

1.1 Statement of the Research Question

Mastoid air cell system (MACS) hypopneumatization is a skeletal stress marker and its prevalence will be associated with other measured skeletal stress markers.

MACS hypopneumatization is a hypothesized skeletal stress marker resulting from OM. OM risk is increased by marginalization which results in immunodepression and exposure to pathogens (Bentdal et al. 2010; Bruce et al. 2000; Cohen et al. 2007; Cohen et al. 1991; da Costa et al. 2004; Engel et al. 2005; Kucur et al. 2015; Paradise et al. 1997; Uhari et al. 1996). OM adds valuable information to research on social theories when combined with traditional skeletal stress markers and interpreted using a biocultural framework. Furthermore, OM provides a more contextualized approach to the study of population dynamics than benign skeletal stress markers (e.g., dental enamel hypoplasia) because OM risk is augmented by specific factors and results in complications such as deafness, balance problems and language delays (Bluestone and Klein 2007).

The efficacy of MACS hypopneumatization as a skeletal stress marker was tested within the framework of structural violence which creates circumstances that increase OM risk in the

marginalized segment of the population. This hypothesis was tested in two skeletal samples that experienced structural violence, the Hamann-Todd and Terry collections, and one skeletal sample that did not experience structural violence, the Tigara collection.

1.2 Impact

This research adds to the methodological framework for interpreting social theories within the *biocultural skeletal stress marker pathway* by establishing MACS hypopneumatization as a skeletal stress marker capable of adding information about marginalization and impairment. Although MACS hypopneumatization has been examined in the archaeological record (e.g., Birkby 1975, Dalby and colleagues 1993, Flohr and colleagues 2014, Flohr and Schultz 2009a, Gregg and colleagues 1981, Homøe and colleagues 1996, Mann 1992, Qvist 2000 and van Duijvenbode and colleagues 2015), a biocultural approach that includes a broad assessment of skeletal stress markers in the context of a specific social theory has not been applied. MACS hypopneumatization, as a measure of OM, will complement the more traditional skeletal markers of stress as an additional marker of structural violence in historical populations.

For this dissertation MACS hypopneumatization was calculated from CT scans, however future research holds the possibility of identifying OM from dry bone. Histological studies by Flohr et al. (2009) and Flohr and Schultz (2009b) have revealed patterns of remodeling consistent with OM. The temporal bone is robust and frequently recovered during excavation with minimal taphonomic damage (Flohr et al. 2009). Although radiographic analysis is the current standard for diagnosing OM in skeletal remains, continued study of MACS remodeling patterns may permit the diagnosis of OM from temporal bones with taphonomic damage that has revealed the internal

MACS. This would make the use of OM as a skeletal stress marker more broadly applicable in the future because a CT scan would no longer be necessary.

1.3 Chapter Contents

This dissertation tested the applicability of MACS hypopneumatization, a measure of OM, as a skeletal stress marker capable of delineating differences in marginalization and structural violence. In Chapter 2 the etiology, pathogenesis and risk factors of OM are reviewed, and MACS hypopneumatization as a diagnostic criterion for OM is discussed in context of previous research. In Chapter 3 stress is discussed in the context of physiological responses of Selye's (1950) general adaptation syndrome and the hypothalamus-pituitary-adrenal axis. The interactions between stress and the environment, culture and biology is discussed in the context of the *biocultural skeletal stress marker pathway*. Two theoretical models, (1) structural violence and (2) evolution and adaptation to chronic cold, are used to interpret stress within the framework of the biocultural model.

In Chapter 4 the etiology and pathogenesis of the skeletal stress markers included in this dissertation are reviewed. Skeletal stress markers were grouped as nutritional stress (porotic hyperostosis and cribra orbitalia), infectious disease (sinusitis) and dental disease (dental enamel hypoplasia, granulomatous periapical lesions, periodontal disease and antemortem tooth loss). In Chapter 5 the skeletal collections, skeletal stress marker recording methods and statistical methods are described. In Chapter 6 the results of the statistical tests by collection, age group, sex and ancestry are reported.

In Chapter 7 the results are discussed in the context of the applied social theories, (1) structural violence and (2) evolution and adaptation to chronic cold. The co-occurrence of skeletal stress markers with OM and the limitations of using the biocultural model are also discussed. In Chapter 8 the role of anemia in OM risk, the potential role of OM in incorporation into the skeletal collections, the effect of sample bias on the results, and future directions are discussed.

2.0 Otitis Media

Otitis media (OM) is an inflammation of the middle ear mucosa and is often accompanied by fluid accumulation (Figure 1) (Bluestone and Klein 2007). The middle ear is an air-filled cavity within the temporal bone, which houses the ear ossicles and is responsible for sound wave transduction and transmission (Bluestone and Klein 2007). When negative pressure develops in the middle ear, in comparison to the environment, fluid from the surrounding tissues is drawn into the space; this fluid not only inhibits hearing but can serve as a breeding ground for infection (Bluestone and Klein 2007; Doyle 2000). Middle ear pressure is regulated by the Eustachian tube and buffered by the mastoid air cell system (MACS) (Bluestone and Klein 2007; Doyle 2000). The Eustachian tube is a cartilaginous tube, which opens via muscular contraction from the middle ear cavity to the nasopharynx and equalizes the pressure between the environment and the middle ear space (Bluestone 2005). The Eustachian tube is important for pressure regulation, middle ear fluid clearance and protection from nasopharyngeal reflux (Coticchia et al. 2013). Inefficient Eustachian tube opening is called Eustachian tube dysfunction and can introduce pathogens into the middle ear (Bluestone 2005). Common viral and bacterial pathogens isolated from middle ear effusions are respiratory syncytial virus, influenza, rhinovirus, *Streptococcus pneumoniae* and *Moraxella catarrhalis* (Bhutta 2014; Heikkinen and Chonmaitree 2003; Vergison 2008). The MACS buffers the middle ear system by increasing the volume of gas within the middle ear cavity (Alper et al. 2011; Doyle 2000); the larger the combined middle ear and air cell volume, the smaller the effect of pressure change will have on middle ear physiology. If this system is compromised, OM can develop. In children with OM more than 50% will have at least temporary hearing loss (Roberts et al. 2004), which has negative consequences on language, communication, cognitive

development, and academic performance (Acuin 2004; Haggard and Smith 1999; Vernon-Feagans 1999). If untreated severe complications of meningitis and brain abscess can result from OM (Acuin 2004; Berman 1995).

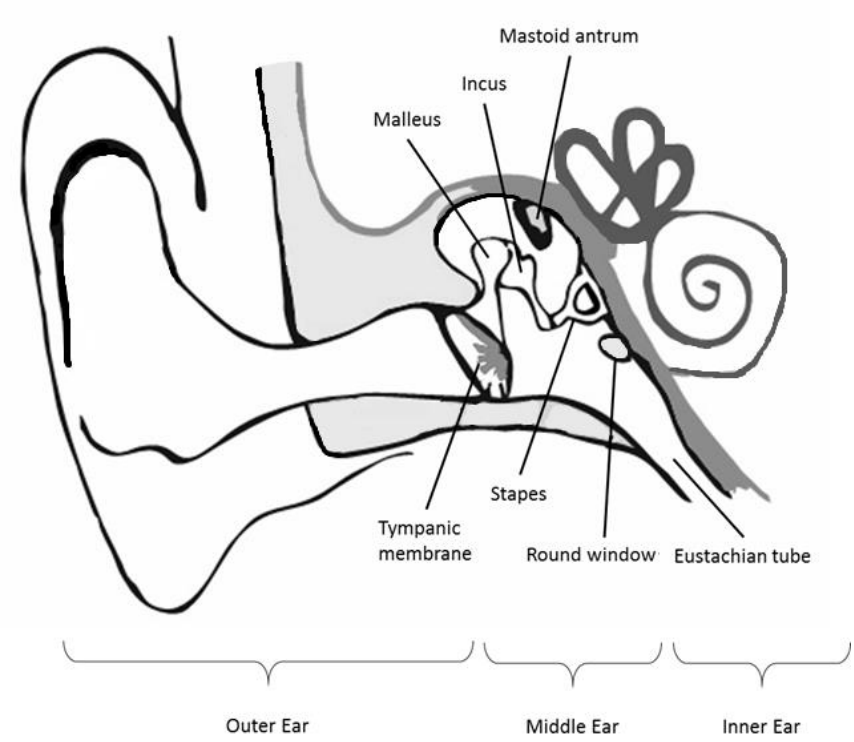

Figure 1: The middle ear anatomy

The middle ear cavity is open to the MACS via the mastoid antrum creating an interconnected air space volume and continuous mucosa. The MACS is a pneumatized network of cells within the mastoid process, which is a prominence on the temporal bone where the sternocleidomastoid muscle inserts. Pneumatization of the mastoid process lightens the skull (Krantz 1963), and increases the volume of the middle ear cavity to absorb and minimize changes in inner ear temperature and middle ear pressure (Magnuson 2003). Pneumatization of the mastoid process creates the MACS and occurs in three phases, rapid pneumatization from birth to one year of age, linear pneumatization from one to six years of age, and slow pneumatization from six years

of age until puberty (Cinamon 2009). Childhood OM damages the mucosa and inhibits pneumatization of the mastoid process resulting in MACS hypopneumatization which is visible radiographically (Andréasson 1976; Ruedi 1963).

Risk factors of OM are anatomic, immunogenic and pathogenic (Bluestone and Klein 2007). Slight anatomical variations in the orientation of the muscles that open the Eustachian tube affect their function. The Eustachian tube is controlled by four muscles, the tensor veli palatini, levator veli palatini, salpingopharyngeus and tensor tympani. The tensor veli palatini muscle dilates the Eustachian tube creating an opening between the middle ear cavity and nasopharynx and originates from the scaphoid fossa of the sphenoid bone, wraps around the pterygoid hamulus and inserts in the palatine bone (Bluestone et al. 1998). The levator veli palatini, salpingopharyngeus and tensor tympani muscles assist the tensor veli palatini in Eustachian tube dilation. The Eustachian tube closes by passive recoil (Bluestone et al. 1998). The orientation of these muscles is altered by age and craniofacial dysmorphologies. Infants and children have a shorter, more horizontal Eustachian tube which is more difficult to open (Sadé et al. 1985; Sadler-Kimes et al. 1989). At birth the Eustachian tube is at a 10° angle relative to the horizontal axis and rotates to a 45° angle during craniofacial growth and development (Proctor 1967; Sadler-Kimes et al. 1989). By seven years of age the Eustachian tube has reached its adult size and orientation (Proctor 1967; Sadler-Kimes et al. 1989). Craniofacial abnormalities, such as those arising from cleft palate (Flynn et al. 2009; Paradise et al. 1969) and Down syndrome (Austeng et al. 2013; Mitchell et al. 2003), can also affect muscular action and Eustachian tube opening. Inefficient Eustachian tube opening can result in negative middle ear pressure and pathogen exposure.

Immune response and pathogen exposure are important factors in OM risk. Immune response may be affected by age (e.g., immature immune system) (Bentdal et al. 2010; Engel et

al. 2005), behavior (e.g., immune protection from breastfeeding) (Bowatte et al. 2015; Uhari et al. 1996) and psychosocial stress (e.g., immunodepression) (Cohen et al. 2007; Cohen et al. 1991). Pathogen exposure and transmission are affected by environmental and cultural conditions including exposure to smoke and pollution (Bruce et al. 2000; Csákányi et al. 2012; da Costa et al. 2004), number of siblings (Paradise et al. 1997; Teele et al. 1989), daycare practices (Paradise et al. 1997; Uhari et al. 1996), socioeconomic deprivation (Kırıs et al. 2012; Kucur et al. 2015) and upper respiratory tract infections (Kırıs et al. 2012; Kucur et al. 2015). Heritability is also a main component of OM risk, but it is unclear which system(s) is compromised (i.e., anatomic, immunogenic, or pathogenic) (Casselbrant et al. 1999; Rovers et al. 2002). Although complex biological, cultural and environmental factors influence OM risk, age less than six years is the single best predictor of OM due to an immature immune system and infant craniofacial structure resulting in inefficient Eustachian tube function (Bhutta 2014; Bluestone 2005).

Demographic factors of ancestry and sex are not strongly associated with OM prevalence. While some studies have identified an increased prevalence in boys (Apostolopoulos et al. 1998; Paradise et al. 1997; Teele et al. 1989), others have found no relationship between biological sex and OM prevalence (Biles et al. 1980; Casselbrant et al. 1995; Gravel and Wallace 2000). Similarly, there are mixed reports on the prevalence of OM by ancestry, with some studies indicating a greater prevalence in white (Bush and Rabin 1980; Griffith 1979; Kessner 1974; Marchant et al. 1984) or black children (Paradise et al. 1997), and others reporting no racial differences (Biles et al. 1980; Casselbrant et al. 1995). Racially identified differences in OM prevalence may result from uncontrolled factors as Park and colleagues (2002) note attributing racial differences to differences in access to and the type of healthcare received.

Evidence of OM is visible within the MACS of skeletal remains. The MACS is an aerated chamber open to the middle ear via the mastoid antrum. OM during childhood inhibits the development of the MACS resulting in hypopneumatization, a condition characterized by a reduction in the size and number of air cells and an overall decrease in mastoid volume; this hypopneumatization is maintained throughout the individual's life (Bayramoğlu et al. 1997; Csakanyi et al. 2010; Swarts et al. 2012). After MACS development is complete chronic OM lasting longer than 3 months causes secondary remodeling and sclerosis of the MACS (Sadé and Berco 1974; Sade and Halevy 1974; Thomsen et al. 1974).

Diagnosis of OM from skeletal remains is performed by radiographic assessment of MACS pneumatization. Gregg and Steele (1965) were the first to apply this method to skeletal remains. They examined 417 temporal bones from Arikara, Middle Plains Woodland, historical Sioux and miscellaneous burials and found hypopneumatization in 44% of the Arikara temporal bones and 52% of the other temporal bones and attributed these changes to infectious OM. Following Gregg and Steele (1965), Titche and colleagues (1981) examined radiographs from 1,296 temporal bones from 742 prehistoric Western Pueblo Native American skulls and found 25% of the temporal bones had hypopneumatization. Flohr and Schultz (2009a) identified secondary MACS remodeling in 83% of temporal bone radiographs from medieval German populations. Rathbun and Mallin (1977) examined radiographs from 15 skulls from prehistoric Iran and diagnosed depneumatization, or a secondary filling in of the MACS, in 40% of the skulls.

Researchers have assigned physiological processes to the different types of pneumatization observed in radiographs. Gregg and Steele (1982) defined diploic pneumatization as a result of early life OM and sclerotic pneumatization as a result of chronic or severe OM in adulthood. Flohr and colleagues (2009) diagnosed primary, secondary and mixed pneumatization from

radiographic, macroscopic and microscopic examination in MACS dry bone histology sections. They characterized primary hypopneumatization as a result of poor development in the first five years of life and secondary hypopneumatization as MACS destruction in adulthood. Rathbun and Mallin (1977) described secondary pneumatization as depneumatization of the MACS and is distinguished by the visualization of former air cell walls. Homøe and colleagues (1994) measured MACS pneumatization from radiographs in living individuals with clinically diagnosed OM to create a statistical model and establish a quantitative MACS cut-off volume (400mm^2) thus improving the accuracy and consistency of OM diagnosis from skeletal remains. Homøe and colleagues (1994) measured the pneumatized area of the MACS using a planimetric analysis performed by a computer in 34 individuals, 9 with self-reported childhood OM. The 400mm^2 value is a conservative cut-off value. Homøe and colleagues (1994) relied on self-reported childhood OM. The most severe OM histories will be the most likely to be recalled and reported, and those cases will have the smallest MACS area. Also, some of adults may not have been aware of their OM history which most frequently occurs before 6 years of age. Within the self-reported OM group (n=9), Homøe and colleagues (1994) measured MACS area as large as 2,105mm^2. Only three of the individuals, all OM group, had a MACS area <400mm^2; six of the OM group had a MACS area >400mm^2. The 400mm^2 cut-off value was conservative ensuring there were no false positives included, but false negatives were present with 67% (n=9) of the OM individuals above the 400mm^2 MACS cut-off area. Swarts and colleagues (2012) and Csakanyi and colleagues (2010) measured MACS volume (mm^3 or ml) and found the majority of individuals with OM had a MACS volume <5ml. Swarts and colleagues (2012) and Csakanyi and colleagues (2010) used a slice by slice reconstruction. Csakanyi and colleagues (2010) measured MACS volume from 96 children (56 with OM with effusion, 40 without significant history of OM (control)) from ages 2.5

to 17.5 years and found the mean volume in the OM group was 2.82ml (range: 0.29 to 9.27) and the mean volume in the control group was 10.05ml (range: 3.95 to 28.34). They concluded that almost all of the control children and almost none of the OM children had a MACS >5ml. Swarts and colleagues (2012) measured MACS volume from radiographs of 37 children with a history of OM and concluded that MACS pneumatization is disrupted by OM and the extent is dependent on the duration and severity of the OM. Their results supported the findings by Csakanyi and colleagues (2010) and they concluded that MACS <5ml was an appropriate cut-off value to distinguish between individuals with childhood OM without effusion and with effusion.

Other researchers have established OM using non-radiographic methods, however the accuracy of these methods in positively diagnosing OM is questionable. Bruintjes (1990) evaluated the relationship between lepromatous leprosy and ossicle erosion. He hypothesized that the thickening of the nasal mucosa associated with leprosy may lead to Eustachian tube dysfunction, decreased middle ear drainage and OM. His results revealed that individuals with rhinomaxillary changes of leprosy had a greater prevalence of ear ossicle erosion, which he attributed to OM. He concluded that mucosal changes of leprosy led to the spread of infection from the sinus to the middle ear via the Eustachian tube, resulting in OM. Several researchers have examined stapedial footplate fixation in the archaeological record (Birkby 1975; Dalby et al. 1993; Flohr et al. 2014; Gregg et al. 1981), however stapedial footplate fixation may be congenital, secondary to infection, or taphonomic and is not diagnostic of OM (Flohr et al. 2014). Flohr and colleagues (2014) endoscopically examined 621 temporal bones from five German medieval sites for stapedial footplate fixation. A differential diagnosis was performed on four temporal bones with evidence of stapedial footplate fixation. Part of the temporal bone was removed, and histological sections were cut so microscopic analysis could be performed. Using this method, they were able to

positively diagnose stapedial footplate fixation in two (50%) of the temporal bones. Flohr and colleagues (2014) promote this method of differential diagnosis for stapedial footplate fixation, however it is destructive and requires advanced microscopic techniques. Flohr and colleagues (2009) identified morphological features of three types of MACS hypopneumatization that were visible during macroscopic examination. The first type was described as poorly defined boundaries between pneumatized and nonpneumatized bone with trabecular thickening in the nonpneumatized bone. The second type was described as clear boundaries between the pneumatized and nonpneumatized bone with normal bone in the nonpneumatized portion. Flohr and colleagues (2009) identified these two types as the result of improper pneumatization during MACS development. The third type was described as the secondary filling-in of air cells with visible walls of the former air cells and was described as the result of OM during adulthood after the MACS had developed. This macroscopic identification is a new exciting approach that would allow for OM diagnosis from skulls with exposed MACS without the use of radiography, however, to standardize this method skulls with taphonomic damage to the mastoid process and a known history of OM would need to be analyzed using the MACS hypopneumatization types described by Flohr and colleagues (2009).

Although anthropologists have recognized that differences in time periods, culture and socioeconomic status can impact OM risks many have failed to embrace a biocultural approach. Gregg and colleagues (1981) compared OM prevalence from pre-contact Arikara, post-contact Arikara, Sioux, contemporary Sioux and mixed cultures. They found the pre-contact Arikara and contemporary Sioux had the lowest prevalence rates and were comparable. Schultz (1979) examined two Frankish-Alemannic settlements (500-725AD and 500-700AD) and associated status from grave goods with the presence of OM, based on MACS pneumatization and mastoid

abscess. He found that OM was more prevalent in individuals with fewer grave goods and concluded that individuals with fewer grave goods were of lower socioeconomic status and that this environmental component increased both the risk and prevalence of OM, supporting the hypothesis that socioeconomic status is a risk factor in OM.

Structural violence is the unequal distribution of resources by a social structure which negatively affects the marginalized group and ensures the continuance of their lower position in the society (Galtung 1969). Bergmark and Sedaghat (2017) identified several factors of structural violence that affect otolaryngology patients which include access to healthcare, low socioeconomic status and discrimination, and exposure to other forms of violence; these factors impact two (immunogenic and pathogenic) of the three (anatomic) pathways of OM risk. Although OM has not been studied extensively in the context of structural violence many researchers have explored the association between socioeconomic status and marginalization on OM risk.

Many studies have associated low socioeconomic status with OM (Apostolopoulos et al. 1998; Cambon et al. 1965; Casselbrant et al. 1995; Cunningham 1979; Daly et al. 2010; Howie et al. 1990; Johonnott 1973; Kero and Piekkala 1987; Roberts et al. 1991; Saim et al. 1997; Sipilä et al. 1988; Stahlberg et al. 1986; Tong et al. 2006), but several studies have found no relationship (Aydemir and Ozkurt 2011; Gultekin et al. 2010; Kessner 1974; Martines et al. 2011; Myers et al. 1984; Niemelä et al. 1995; Robinson et al. 1967; Teele et al. 1989). The discrepancy between the association between socioeconomic status and OM is probably the result of inconsistent definitions of socioeconomic status which ranged from parental income (Aydemir and Ozkurt 2011; Saim et al. 1997; Tong et al. 2006) and education (Apostolopoulos et al. 1998; Aydemir and Ozkurt 2011; Cunningham 1979; Kucur et al. 2015) to socioeconomic index scores (Cambon et al. 1965; Robinson et al. 1967). Other factors such as bottle feeding in place of breastfeeding (Bowatte et

al. 2015; Kırıs et al. 2012; Kucur et al. 2015; Uhari et al. 1996) are known to increase OM risk, and have also been associated with socioeconomic indicators like parental education (Cunningham 1979; Howie et al. 1990). Generally, the research suggests that there is a socioeconomic component to OM risk which most likely results from immunodepression and increased pathogens exposure and transmission. In marginalized populations, many of which experience structural violence, there are high prevalence rates of OM (Table 1) (Acuin 2004; Bluestone 2005; Monasta et al. 2012). In developing countries there is a modest amount of OM while developed countries have the lowest prevalence rates (Acuin 2004; Bluestone 2005; DeAntonio et al. 2016; Monasta et al. 2012). This epidemiological perspective suggests that OM prevalence is affected by socioeconomic status and structural violence.

Table 1: OM prevalence in marginalized populations, developing countries and developed countries

Reference	Marginalized Populations/ Least Developed Countries	OM	Recently Industrialized/ Developing Countries	OM	Developed Countries	OM
Bluestone (2005)	Alaska Inuit Australian Aborigine Native North American	30-46% 12-25% 12-25%	South Pacific Islands Sierra Leone Gambia Kenya	4-6% 6% 4% 4%	United States Denmark Finland	<1% <1% <1%
DeAntonio and colleagues (2016)			Nigeria Egypt Iran China India Russia	9.2% 10% 9.1% 6.7% 9.2% 5.1-7.8%		
Acuin (2004)	Solomon Islands Australian Aborigine Greenland Inuit Eskimos American Indians	>4% >4% >4% 2-4% <1%	Tanzania India Guam Nigeria Angola Mozambique Thailand Malaysia Thailand Philippines Vietnam Micronesia China Brazil Kenya Gambia Saudi Arabia	>4% >4% >4% 2-4% 2-4% 2-4% 2-4% 2-4% 2-4% 2-4% 2-4% 2-4% 2-4% 1-2% 1-2% <1% <1%	Israel Australia United Kingdom Denmark Finland	<1% <1% <1% <1% <1%

Reference	Marginalized Populations/ Least Developed Countries & Regions	Acute/ chronic OM	Recently Industrialized/ Developing Countries & Regions	Acute/ chronic OM	Developed Countries & Regions	Acute/ chronic OM
Monasta and colleagues (2012)	Sub-Saharan Africa Central	43.37/7.56%	Asia East	3.93/3.67%	Asia Pacific- high income	3.75/3.02%
	Sub-Saharan Africa East	22.82/6.06%	Asia South	14.52/6.56%	Europe Central	3.64/3.69%
	Sub-Saharan Africa West	43.36/7.22%	Asia South East	8.15/4.69%	Europe Western	5.91/3.39%
			Caribbean	9.08/4.18%	North America- high income	5.46/3.06%
			Europe Eastern	3.96/3.75%		
			Latin America Andean	5.39/1.70%		
			Latin America Central	6.78/3.92%		
			Latin America Southern	4.25/3.60%		
			Latin American Tropical	5.90/3.74%		
			North Africa Middle East	8.67/4.41%		
			Oceania	28.56/9.37%		
			Sub-Saharan Africa Southern	14.71/4.79%		

Although OM can provide valuable insight into pathogen exposure and transmission, changing sociopolitical landscapes, and hearing loss, few bioarchaeological studies include OM. OM has an established skeletal response, MACS hypopneumatization, that has been measured in skeletal remains. This research aimed to determine if MACS hypopneumatization was triggered by factors affecting a population (i.e., social theories) and if it associated with established skeletal stress markers. To test this theory, OM prevalence, defined as MACS <5ml, was recorded in three populations, two that experienced structural violence (Hamann-Todd and Terry) and one that did not (Tigara). In the population that did not experience structural violence the social theory of evolution and adaptation to chronic cold was used to explain patterns of skeletal stress markers.

3.0 Biocultural Framework

Stress is a physiological response to a threat to homeostasis which results in stress-specific and generalized responses (Herman et al. 2016). The stress-specific response is dependent on the type of stressor (e.g., shivering thermogenesis in response to cold exposure), whereas the generalized response is a predictable and established response to any stress and was termed the 'general adaptation syndrome' by Selye (1946). The general adaptation syndrome describes the stages from the initial stress response through the transition from resistance to stress fatigue (Selye 1950). The first stage, the alarm reaction, is an exaggerated response to a stress upon first exposure (Selye 1973). Repeated stress causes the body to enter the stage of resistance where it adapts to withstand the stimuli (Selye 1973). Under prolonged stress the body reaches the stage of exhaustion where the organism can no longer adapt resulting in disease (Selye 1973). The gradual build-up of stress responses results in illness, but the interpretation of disease from the general adaptation system is complicated when a stressor triggers both a specific and general stress response (e.g., a general stress response and a specific response to a nutrient deficiency can result in stunted growth) (Selye 1950).

The stress response is regulated by the hypothalamic-pituitary-adrenal (HPA) axis. The hypothalamus, a small area of the brain responsible for control of metabolic processes and the autonomic nervous system, releases corticotropin releasing hormone (CRH) in response to a stress stimulus (Figure 2). CRH is released into the hypophyseal portal system, which provides blood to the base of the brain, and stimulates the pituitary gland. In response to CRH, the pituitary gland, which is located at the base of the skull and is attached to the hypothalamus by a small stalk, releases adrenocortocotropic hormone (ACTH). ACTH is secreted into the bloodstream and travels

to the adrenal glands, located on top of the kidneys, to stimulate the release of glucocorticoids (e.g., cortisol). Glucocorticoids ensure energy is free to meet the demands of the stress stimuli and inhibits the further secretion of CRH and ACTH in a negative feedback loop. (Myers et al. 2014)

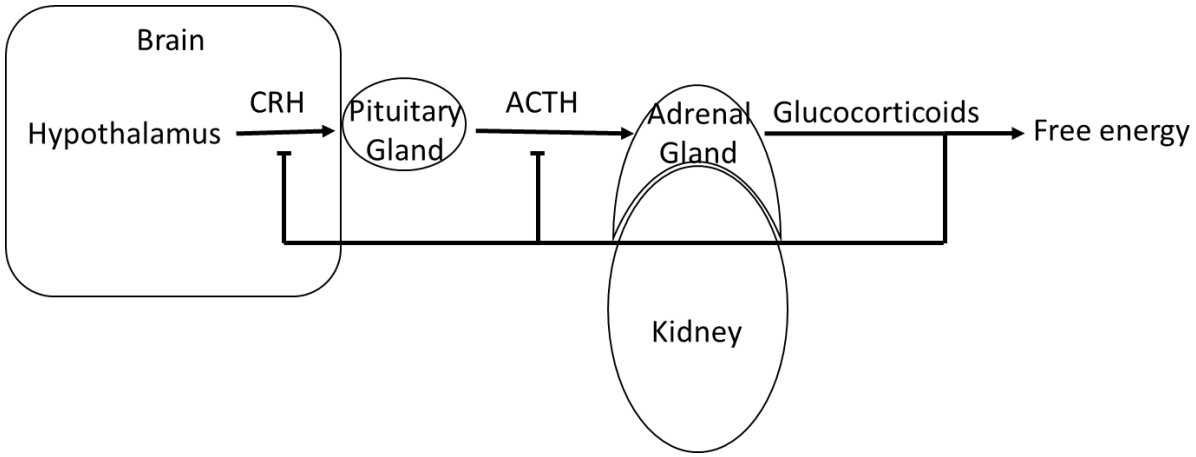

Figure 2: HPA axis and feedback loop

The HPA axis is activated when homeostasis is challenged and is dependent on the intensity, duration, frequency and type of stress (Herman et al. 2016). The type of stress can be categorized as reactive, which is a response to a homeostatic challenge, or anticipatory, which is a response to a psychological challenge. A homeostatic challenge will result in a rapid HPA axis response by acting directly on the hypothalamic neurons, whereas an anticipatory stressor is a slower response and acts on the hypothalamus by intermediary nuclei (Herman et al. 2016). Acute stress of short duration produces a pronounced but limited response during stage 1 of the general adaptation syndrome whereas chronic stress which produces a blunted but extended response in stage 2 (Dhabhar et al. 1997). A chronic stress response occurs in the presence of a prolonged or repeated stress stimulus (Herman et al. 1995; Ulrich-Lai et al. 2006). The extended (although diminished) release of glucocorticoids during chronic stress results in pathological organ changes (e.g., an enlarged adrenal gland (Ulrich-Lai et al. 2006) and a diminished thymus (Herman et al.

1995)) and symptomatic responses (e.g., adrenal fatigue (Herman et al. 2016)) during stage 3 of the general adaptation syndrome. A hyperactive HPA response produces excessive glucocorticoids which damage physiological systems resulting in pathology. However, if a response is insufficient then an individual may not be prepared to overcome the stress, and disease or death will result. For example, individuals with a diminished stress response are more likely to develop post-traumatic stress disorder (Radley et al. 2011). The difference between adaptation and pathology is not always clear and depends on the context; if the response was necessary for survival, even if it resulted in disease, then it can classified as an adaptive strategy (Herman et al. 2016).

The stress response is influenced by biological sex and age. In response to stress the HPA axis frees energy to meet increased demands during a stress stimulus and since men and women have different energy demands sexual dimorphism within the stress response is expected (Vamvakopoulos 1995). Sex hormones modulate the HPA response and within animal models testosterone has been demonstrated to reduce the stress response while estradiol increases the stress response (Viau and Meaney 1996). Within humans females have a greater variability in HPA axis response than males due to cyclical hormonal changes experienced by females (Choi et al. 2007; Figueiredo et al. 2002; Kirschbaum et al. 1999; Viau and Meaney 1991; Weiss et al. 1999). It is necessary to consider the impact of biological sex on the HPA response when interpreting stress outcomes (Herman et al. 2016).

The stress response also changes with age. After the first year of life and during early childhood the stress response is reduced, this is known as the stress hyporesponsive period (Gunnar and Quevedo 2007). During this time adrenal sensitivity to hormones is developing (Gunnar and Quevedo 2007). Maternal interaction is especially important for the development of the HPA response and a lack of stimulation can result in an abnormal stress response in adulthood (Gunnar

and Quevedo 2007; Lupien et al. 2009). For example, in mouse models maternal deprivation resulted in an exaggerated HPA response later in life (Ladd et al. 2000), whereas, mice pups that experienced more maternal grooming had a dampened stress response later in life (Liu et al. 1997). The relationship between early life stressors and health outcomes in later life is known as the *developmental origins of health and disease hypothesis* and there are two opposing arguments for the influence of early life stress on health outcomes later in life (Barker 2007). The *cumulative stress hypothesis* states that stress is cumulative over a lifetime and early life stressors increase risk for stress and disease in later life (Taylor 2010). There is a trade-off between a stress response in early life to provide short-term survival at the expense of health later in life (Temple 2019). This hypothesis is supported by research which shows that childhood stress is strongly associated with elevated adult morbidity and early mortality (Clark et al. 1986; Goodman and Armelagos 1988). Alternatively the *adaptive phenotypic plasticity hypothesis* states that early life stress better equips an individual to handle stress later in life (Bateson et al. 2004; Nederhof and Schmidt 2012). This hypothesis means an individual calibrates their stress response to their early environment and if there is a mismatch between the anticipated and actual environment later in life then an inappropriate stress response can threaten reproduction and survival (Nederhof and Schmidt 2012). Nederhof and Schmidt (2012) suggest that both hypotheses are correct and an individual's reliance on one strategy over another is dependent on their innate ability to respond to early life stressors with plasticity. To explore the developmental origins of health and disease hypothesis evidence of early life stress (e.g., DEH) should be compared to later life stress outcomes (e.g., mortality) within a biocultural framework that includes sociopolitical and historical contexts and considers hidden heterogeneity and selective mortality (Temple 2019; Wood et al. 1992).

Following childhood there is an exaggerated response to ACTH and glucocorticoid hormones which occurs during puberty (Romeo 2010). This is due to a lag in the feedback response and does not produce the expected pathological phenotypes that are observed under other circumstances. With advanced age, the stress response increases and as the body increases the amount of glucocorticoids released negative feedback is reduced (Sapolsky et al. 1986). Cortisol is the most common glucocorticoid and with age there is an increase in cortisol levels and production (Purnell et al. 2004; Seeman et al. 2001). Cortisol exposure over time diminishes the feedback response (Boscaro et al. 1998; Raff et al. 1999; Wilkinson et al. 1997; Wilkinson et al. 2001). The HPA stress response is a balanced feedback system that is modified by early life stressors and influenced by age and sex related hormones and these components should be carefully considered when measuring stress in a population.

3.1 Biocultural Stress Model

The biocultural stress model explains the cyclical relationship between stress, disease and skeletal pathology (Figure 4) (Goodman et al. 1984c). Stressors are categorized as environmental or cultural, and can be differentially allocated to members of the society depending on status (Schell 1997). Culture can also act as a buffer to minimize the stress experienced by an individual (Goodman et al. 1984c). Buffers act to minimize the effects of stress on homeostasis, which when disrupted causes disease. Stress that is not buffered by culture acts upon an individual, but the outcome is dependent on the host's resistance, which is a biological buffering system (Goodman et al. 1984c). If environmental and cultural stressors are not buffered by culture or the host then homeostasis is not maintained and an individual will develop stress and disease, signs of which

may be expressed as changes in skeletal tissue, referred to as skeletal stress markers (Goodman et al. 1984a; Huss-Ashmore et al. 1982; Lewis 2002b). The resultant stress and disease states can set up a cyclical relationship by adding new functional stressors to the system (e.g., disease results in decreased work and income), which, in turn, amplify the impact from the initial stressors and further burdens the system (Goodman et al. 1988). This feedback system was termed the biocultural stress model and first defined by Goodman and colleagues (1984c) and then revised by Goodman and Armelagos (1989).

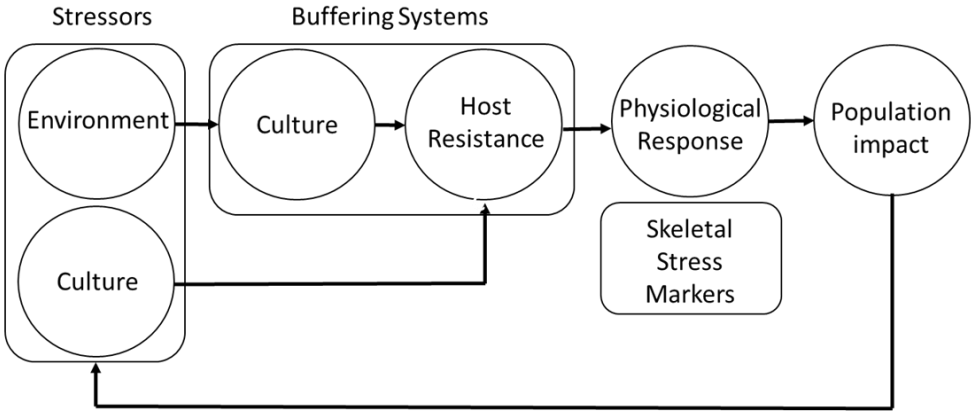

Figure 3: Goodman and Armelagos (1989) biocultural stress model

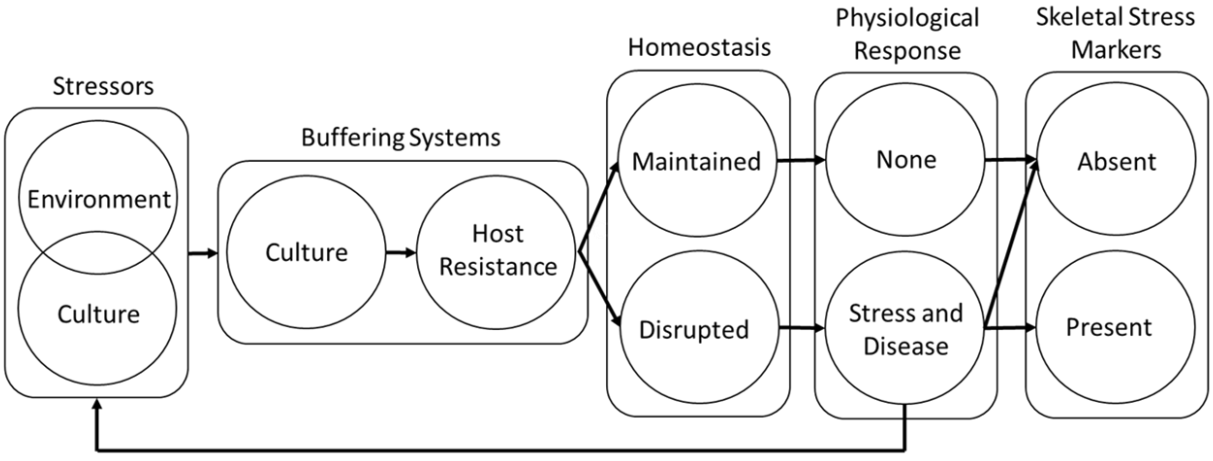

Figure 4: Biocultural skeletal stress marker pathway

3.1.1 Biocultural Skeletal Stress Marker Pathway

The *biocultural skeletal stress marker pathway*, proposed in this dissertation and depicted in Figure 4 (page 25), differs from previous models proposed by Goodman and colleagues (1984c) and Goodman and Armelagos (1989) in several important ways. First, the environmental and cultural stressors are overlapping. Many environmental stressors (e.g., urban-living stressors of noise pollution, toxin exposure and air pollution) are differentially allocated to marginalized groups making it impossible to separate the environmental and cultural components of these stressors (Schell 1997). Second, *both* environmental and cultural stressors can be buffered by cultural systems. For example, an individual can experience both the cultural stressor of food insecurity and the cultural buffer of a strong social support network. The social network can mitigate the risks of food insecurity by food sharing, charity and donation, but even without support in the form of food, the emotional and social support can dampen the psychological stress associated with food insecurity. Third, this model represents two pathways that are ultimately determined by host resistance. Two individuals can experience identical stressors and cultural buffering systems yet have different homeostatic responses (Wood et al. 1992). This is due to individual differences in host resistance (e.g., variability in responsiveness to developmental adaptive plasticity) (Goodman et al. 1984c; Wood et al. 1992). Last, the population impact included in Goodman and Armelagos (1989) was removed because an individual's health status has more of an impact on their individual exposure to stressors than population stress, since risk is differentially allocated (Schell 1997).

The *biocultural skeletal stress marker pathway* is applicable to population interpretations (if from appropriately amassed samples) because it assumes a population is composed of a normal distribution of individuals with differential stress responses, but, given this hidden frailty of risk,

conditions cannot be ascribed to an individual (Wood et al. 1992). In the model depicted in Figure 4 there are two possible outcomes when examining skeletal data, either the presence or absence of skeletal stress markers. The absence of skeletal stress markers can be the result of several pathways. First, the individual did not experience stress and therefore this pathway was never initiated. Second, the individual experienced stress, but the buffering systems were able to overcome the stress resulting in no homeostatic response. Third, the stressor disrupted homoeostasis resulting in disease and pathology, but it was not manifested in the skeletal tissue. Fourth, extreme stress resulted in death before a skeletal response could occur. When skeletal stress markers are present it indicates homeostasis was disrupted, but the etiology is often unknown. Within the skeletal record there is a lot of hidden stress and where it is visible it is not often attributable to a specific stressor. Furthermore, the presence or absence of skeletal stress markers (or other skeletal pathology) cannot be used to make interpretations about health, only disease (Reitsema and McIlvaine 2014; Wood et al. 1992). The concepts of health and quality of life are culturally constructed and incorporate biological, social and psychological spheres of well-being (Temple and Goodman 2014). Interpretations of skeletal stress markers must be limited to the biological understanding of the etiology and pathogenesis of the pathology within the historical and cultural context of the individual and population and should not be used to make assertions about health.

The *biocultural skeletal stress marker pathway* is a framework for understanding the past and can be used to interpret data in the context of different social theories (Martin et al. 2013). Martin and colleagues (2013) state that the biocultural stress model, which is the foundation of the *biocultural skeletal stress marker pathway*, is not a theoretical model, but a formula that uses known skeletal stress markers to make interpretations about life in the past. They stress that the

biocultural skeletal stress marker pathway must be used as a tool to interpret social theories and cannot provide valuable information outside of a theoretical context. In this dissertation structural violence is the social theory used to interpret patterns of stress and the applicability of OM as an additional skeletal stress marker capable of contributing to interpretation about life course in past populations. It is impossible to include all of the factors that impact stress and therefore the researcher must decide which factors to include in the model based on the social theory (e.g., structural violence) and design limitations (e.g., only skulls examined) (Martin et al. 2013).

3.2 Structural Violence

Structural violence is the normalized oppression of a group of individuals by a socioeconomic structure and is enforced through disparities in access to resources. Galtung (1969) coined the term *structural* violence which distinguishes it from other forms of violence (e.g., physical). Structural violence is often invisible, and a tool used by those in power in strongly hierarchical societies to control the powerless through the disproportionate allocation of resources despite resource abundance. There are no individual actors of violence and therefore it is often overlooked and accepted as a normal part of society. However, the damage of structural violence is real and visible within the marginalized segment of a society through poor health outcomes. (Galtung 1969)

Farmer and colleagues (2004) examined structural violence in Haiti to understand disparities in medical care and poor health outcomes from AIDS and tuberculosis. This application of structural violence to living individuals by medical anthropologists demonstrated the utility of structural violence in anthropology. Klaus (2012) demonstrated the applicability of structural

violence by physical anthropologists to interpret violence and hierarchy in deceased populations. Klaus (2012) used Goodman and Armelagos' (1989) biocultural stress model to explain the embodiment of structural violence in the Lambayeque Valley of Peru during Spanish colonialism. Using a diachronic and contextualized approach he demonstrated that structural violence is embodied as systemic biological stress. His case study modeled a successful application of the biocultural stress model to bioarchaeological analyses of structural violence. However, he cautions a careful and critical approach to interpreting structural violence in a bioarchaeological context with four main tenants. First, structural violence should only be interpreted within a rigidly hierarchal society. Second structural violence should be examined in relation to other forms of violence. Third, the recipients of structural violence should not be considered victims because they are active participants in the society that passively distributes structural violence. Fourth, structural violence should be considered within the archaeological and historical contexts. This was echoed by Farmer and colleagues (2004) who stated that the embodiment of inequality must be interpreted within the historical and cultural contexts that created the hierarchical society and the resulting institutionalized violence. Klaus (2012) argues that structural violence is valuable to bioarchaeology because it combines science and culture, contributes to political economy theory, and contributes to understanding other forms of violence.

Within the context of the *biocultural skeletal stress marker pathway* structural violence can be explained as a system of oppression that creates stress within the marginalized faction of the system through decreased socioeconomic status and increased psychosocial stress, which becomes embodied as health disparities (Dressler et al. 2005; Klaus 2012). In this way culture acts as a stressor. While culture can act as both a stressor and a buffer, the benefits of cultural buffers are often disproportionately dispersed within a society based on social stratification (Schell 1997).

The powerful benefit of cultural buffers are in the form of health behaviors, and social and cultural contexts (Schell 1997). Whereas the marginalized experience a constraint in choices on where they live, where they work, and access to resources (e.g., food, and healthcare) (Schell 1997). The marginalized are often economically constrained to the most undesirable living areas, which expose them to additional risk factors (Schell 1997). This increased exposure to risk sets up a cyclical relationship where the marginalized and their descendants become more marginalized (Schell 1997). Risk factors make them more likely to experience stress, illness and injury, which further limit nutritional, housing and occupation choices, which increases the likelihood of low socioeconomic status and obstructs upward mobility (Schell 1997). While structural violence is the systemic oppression of a group of living individuals by a society, the incorporation of unclaimed individuals into the Hamann-Todd and Tigara collections is an extension of structural violence in death (Nystrom 2014).

3.3 Application of Structural Violence to the Biocultural Skeletal Stress Marker Pathway

The social theory of structural violence was applied to the *biocultural skeletal stress marker pathway* to test the hypothesis that MACS hypopneumatization, as a measure of OM, is a skeletal stress marker in the Hamann-Todd and Terry collections (Table 2). The Hamann-Todd and Terry collections are composed of marginalized and indigent individuals that experienced structural violence at the time of death which is reflected in their skeletal biology as skeletal stress markers (Muller et al. 2017; Nystrom 2017). These collections are composed of individuals from lower socioeconomic statuses who were not claimed by relatives at the time of their death either due to inadequate social ties or financial inability, and thus were immortalized in these skeletal

collections (Albanese 2003). This disproportionate access to a proper burial and the incorporation into a skeletal collection without the ability to consent is a form of structural violence (Nystrom 2017).

Table 2: Application of structural violence to the biocultural skeletal stress marker pathway

Environmental Constraints	Cultural Stressors	Cultural Buffer	Host Resistance	Skeletal Stress Marker
Climate Crowded living conditions Pollution Poor sanitation Food instability Lack of medical care	Discrimination Socioeconomic status Working conditions Segregation	Social networks Government aid	Age Hidden heterogeneity Nutrition Disease Trauma	Porotic hyperostosis (PH) Cribra orbitalia (CO) Sinusitis Antemortem tooth loss (AMTL) Granulomatous periapical lesion Periodontal disease Dental enamel hypoplasia (DEH)

The use of marginalized individuals in anatomical collections was facilitated by the United States legislation and these laws represent tangible evidence of the structural violence which appropriated the physical bodies of marginalized individuals for the benefit of the educated which are still used to this day. In the US, the legalization of teaching cadavers in the 1800s legitimized the use of the bodies of the indigent and poor and offered the cheapest option for body disposal (Muller et al. 2017). The first American law legalizing the use of unclaimed bodies by medical schools for dissection was passed in Massachusetts in 1831 (Sappol 2002). The Act to Promote Medical Science and Protect Burial Grounds was passed to allow the dissection of bodies not claimed within 24 hours in New York in 1854 (Muller et al. 2017). Laws like these existed all over the United States and the enactment of these laws did not change the social origins of the bodies, but now sanctioned the use of unclaimed bodies (Humphrey 1973; Muller et al. 2017). In 1968, the Uniform Anatomical Gift Act changed donation practices putting a stop to the use of unclaimed bodies in teaching laboratories (Muller et al. 2017). However, this act was not passed until after the completion of the Hamann-Todd and Terry collections in 1938 and 1967, respectively (Hunt and Albanese 2005; Muller et al. 2017).

Within the Hamann-Todd and Terry collections unclaimed human remains became the property of these collections, in most cases without the consent of the individual or due to the limited resources of the surviving family and friends (de la Cova 2019; Nystrom 2014). This practice, which was legitimized by legislation, targeted the most vulnerable segments of society and was a form of structural violence (Muller et al. 2017). Individuals who lacked the resources for a proper burial became the property of these collections (Muller et al. 2017). The Hamann-Todd and Terry collections represent a segment of the population that experienced structural violence either due to the lack of a social network or financial means at least at the time of their

death, but these individuals also likely experienced low socioeconomic status and structural violence prior to death.

Individuals from the Hamann-Todd and Terry collections died in Cleveland, Ohio and St. Louis, Missouri during the 19th and 20th centuries (Figure 5). In both St. Louis and Cleveland social stratification left the lower socioeconomic strata vulnerable to unequal access to healthcare and unsanitary and crowded living conditions which were compounded by systemic issues of pollution, resulting from rapid technological advancements and population growth (Cleveland Hospital Council 2017; Primm 1998). Open sewage gutters and untreated water in the 19th century contributed to the spread of infectious diseases and in particular the 1849 cholera epidemic in St. Louis which forced the city to build sanitation systems (Arenson 2011; Primm 1998). The Civil War introduced hardships and soldiers suffered from poor nutrition and sanitation, and disease and exposure. African American soldiers experienced additional discrimination in the form of decreased rations and access to medical care (Primm 1998). Discrimination and segregation continued well past the end of the Civil War. In 1911 the United Welfare Association in St. Louis campaigned for segregation and a new law was established where no person could move to a block where 75% of the residents were of another race (Primm 1998). Similar ordinances in 1916 restricted where an individual could reside and required segregated churches and schools (Vexler 1974). In 1917 race riots broke out in St. Louis and hundreds were wounded, 47 died and 300 houses were burned down (Primm 1998). Similar tensions were growing in Cleveland as African Americans moved north and the population expanded at a rate the current infrastructure was unable to sustain (Miller and Wheeler 1997). In the 20th century some parts of Cleveland were still segregated and black neighborhoods began to form (Miller and Wheeler 1997).

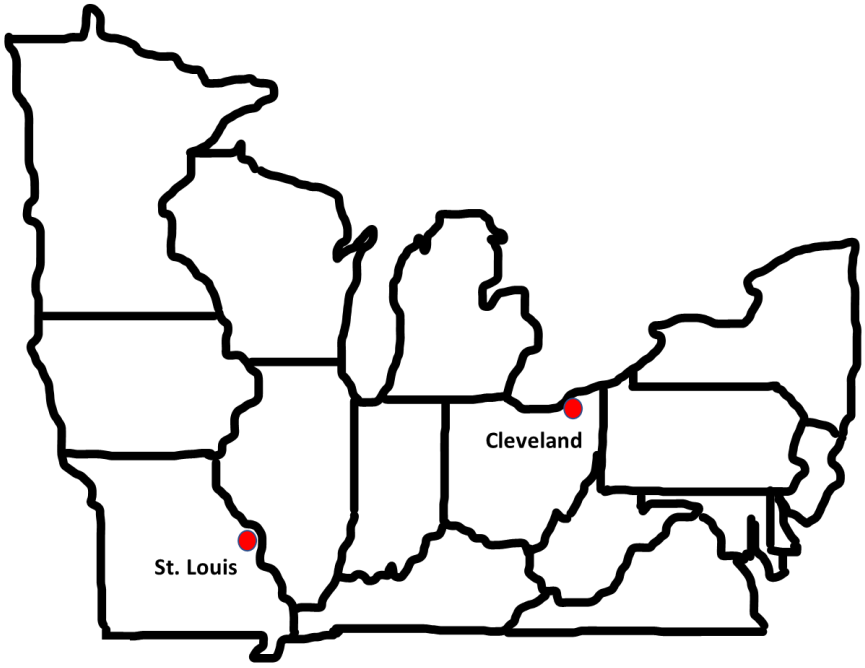

Figure 5: Map of midwest United States with St. Louis, Missouri and Cleveland, Ohio marked with a red circle

Living conditions were equally dim and in 1907 the Civic League Housing Commission did an inspection of the poorest areas in St. Louis and found dangerous unfit living conditions with more than 90% of individuals sharing an outdoor bathroom with multiple families (Rumbold 1908). A survey in 1937 found a 15 block area of St. Louis contained 65% of the tuberculosis deaths, 50% of the infant deaths, 75% of the illegitimate children and 65% of the delinquents (Primm 1998). Similar crowded and unsanitary living conditions were found in Cleveland with human feces outside of some residences (Cleveland Hospital Council 2017). Cleveland slums were hot beds for crime and delinquency, but also infectious disease outbreaks, most notably, tuberculosis (Miller and Wheeler 1997; Navin and Green 1939). Despite living in the worst conditions African Americans were often charged higher rent as a form of price discrimination (Miller and Wheeler 1997; Navin and Green 1939; Rumbold 1908).

In the 20th century, St. Louis and Cleveland flourished as industrial centers for a short time before their devastating fall (Berger 2015; Lewis 2004; Schroeder 1997). With industry, technology and advancements in transportation came pollution of the ground water and air which threatened the livelihood of plants and people (Cleveland Hospital Council 2017; Schroeder 1997; Tarr and Zimring 1997). Healthcare systems were limited, overextended and short staffed (Cleveland Hospital Council 2017; Miller and Wheeler 1997). The City Hospital in St. Louis was segregated and there was racial and economic discrimination (Berg 2003). When a second City Hospital opened specifically for African Americans less than half was spent on the patients in City Hospital II ($1.55/patient/day) compared to City Hospital I ($3.22/patient/day) which highlights the racial discrimination and differential access to care in St. Louis (Berg 2003). Inextricably linked environmental and cultural stressors, like living conditions, pollution and medical care, burdened the lower class and economic and racial discrimination added more stress. There were few social and cultural systems established (e.g., social networks and government aid) to buffer this stress. As a result, marginalized individuals in Cleveland and St. Louis lost their autonomy in death and were incorporated into the Hamann-Todd and Terry skeletal collections. The Hamann-Todd and Terry collections are composed of individuals of low socioeconomic status who experienced structural violence at least at the time of death and due to the prevalent environmental and cultural stressors of the time period high prevalence of skeletal stress markers are expected. The crowded unsanitary living conditions, lack of medical care and pervasive discrimination experienced by the individuals in the Hamann-Todd and Terry collections would have contributed to increased pathogen exposure and transmission and a depressed immune response increasing risk of OM. An elevated prevalence of MACS hypopneumatization, as a marker of OM, is hypothesized in the Hamann-Todd and Terry collections.

The Hamann-Todd and Terry collections have a known age, sex and ancestry, and have been extensively used to establish and validate methods of skeletal analysis. Until recently little research has focused on the life history of these individuals. De la Cova (2008; 2010; 2011; 2012; 2014), Coolidge (2015), Gengo (2014) and Atwell (2017) contextualized the lives of those in the Hamann-Todd and Terry collections. De la Cova (2008; 2010; 2011; 2012; 2014) examined stress, disease and trauma in individuals born between 1800 and 1877 from the Hamann-Todd, Terry and Cobb skeletal collections. Coolidge (2015) used the developmental origins of health and disease hypothesis as a model within the Hamann-Todd and Terry collections to determine if early life conditions affected adult longevity and health. Gengo (2014) studied co-occurring disease processes in the context of racial health inequality. Atwell (2017) examined the lives of institutionalized women from the Terry collection. Their graduate theses included some of the skeletal stress markers included in this analysis and their results are reviewed.

In their analyses of the Hamann-Todd and Terry collections de la Cova (2008) included PH/CO, DEH, dental abscess and AMTL and Coolidge (2015) included in DEH. Gengo (2014) examined DEH and periodontal disease in his analysis of the Terry collection, and Atwell (2017) examined PH/CO and dental abscess in her analysis of the Terry women. De la Cova (2008) found that 84.6% (n=357) of the Terry sample and 87.6% (n=178) of the Hamann-Todd sample had PH and/or CO (PH/CO) (Table 3). This was higher than comparative research studies, which included marginalized US samples from a similar time period, and she attributed this to greater illness and biological stress in her sample than the other populations. Atwell (2017) found 50.94% (n=53) of the women she studied from the Terry collection had PH/CO. De la Cova (2008) found at least one DEH on the anterior teeth of 54% (n=202) of individuals in the Terry sample and 63.2% (n=117) of individuals in the Hamann-Todd sample. Coolidge (2015) found higher prevalence

rates of DEH with 80.0% (n=475) of individuals with at least one canine DEH in the Terry sample and 69.8% (n=298) in the Hamann-Todd sample, which was higher than similar research studies and she concluded a DEH formation-time method would be more valuable to compare the results across different studies due to inconsistent DEH tooth inclusion. Gengo (2014) identified DEH from maxillary incisors and mandibular canines in 58% (n=175) of the Terry collection. De la Cova (2008) found at least one dental abscess in 51.9% (n=366) of the Terry and 63.2% (n=182) of the Hamann-Todd sample, which is higher than Atwell (2017) reported for the institutionalized women in the Terry collection (18.87% with at least one abscess, n=28). De la Cova (2008) found evidence for AMTL in 99.2% (n=367) of the Terry and 98.4% (n=182) of the Hamann-Todd sample. Gengo (2014) identified periodontal disease in 70% (n=197) of the Terry sample.

Table 3: Prevalence of skeletal stress markers from previous studies of the Hamann-Todd and Terry collections

Skeletal Stress Marker	Reference	Hamann-Todd Prevalence (n)	Terry Prevalence (n)
PH/CO	De la Cova (2008)	87.6% (178)	84.6% (357)
	Atwell (2017)	--	50.94% (53)
DEH	De la Cova (2008)	63.2% (117)	54% (202)
	Coolidge (2015)	69.8% (298)	80% (475)
	Gengo (2014)	--	58% (175)
Dental abscess	De la Cova (2008)	63.2% (182)	51.9% (366)
	Atwell (2017)	--	18.98% (28)
AMTL	De la Cova (2008)	98.4% (182)	99.2% (367)
Periodontal disease	Gengo (2014)	--	70% (197)

3.4 Evolution and Adaptation to Chronic Cold

The third collection examined in this dissertation was the Tigara Inuit sample from Point Hope, Alaska. According to Klaus (2012) the social theory of structural violence should only be applied to strongly hierarchical Western populations with clear inequalities in resource distribution. The Tigara were an egalitarian pre-European contact marine-hunting society which differed vastly from the stratified population in the United States in the 19th and 20th centuries from which the Hamann-Todd and Terry collections were derived. Thus, the Tigara fall outside the parameters of a hierarchical Western population and not capable of experiencing structural violence. Although the Tigara did not experience structural violence this does not mean that they did not experience stress, but that other social theories (e.g., evolution and adaptation to extreme cold) must be applied to interpret skeletal stress marker patterns (Martin et al. 2013).

In response to chronic cold stress the body may undergo functional changes or a population may genetically adapt (Leonard 2015). Acute cold stress causes the body to use shivering thermogenesis to produce heat, however this mechanism expends a lot of energy and is metabolically inefficient. Therefore, in individuals exposed to chronic cold the body relies on nonshivering and delayed shivering thermogenesis to produce heat at the cellular level and maintain body temperature (Leonard 2015). Chronic cold stimulates the hypothalamus which activates the sympathetic nervous system to release norepinephrine and the pituitary gland to release thyroid stimulating hormone. This stimulates the thyroid gland to produce thyroid hormones. Norepinephrine and thyroid hormones uncouple oxidative phosphorylation releasing energy in the form of heat (Leonard 2015). Within central Asian and Siberian lineages, a mitochondrial DNA mutation increases the metabolic rate of uncoupled oxidative phosphorylation making the thyroid hormone more effective at triggering nonshivering thermogenesis, however it

is unknown if this mutation is found in North American Inuits (Mishmar et al. 2003; Ruiz-Pesini et al. 2004). Arctic and Antarctic conditions also produce seasonal changes in body mass ratio and increased thyroid hormone uptake in winter which increases uncoupled oxidative phosphorylation; this is known as polar T3 syndrome (Leonard 2015). If nonshivering thermogenesis is inadequate Inuit populations rely on delayed shivering thermogenesis which is an earlier onset of vasodilation and higher peripheral body temperatures (Adams and Covino 1958; Brown and Page 1952; Leonard 2015). The 'hunting response', which is a cold induced cyclical pattern of vasodilation and vasoconstriction, prevents frostbite and excessive heat loss and is used by cold-adapted individuals (So 1980).

Evolutionary and adaptive changes to morphology in response to chronic cold prevent heat loss. A short and stocky build decreases the body's surface area to volume ratio and by decreasing the surface area the amount of heat lost to the environment is diminished (Beals et al. 1983). Other morphological changes have been identified in the nasal region in cold-adapted populations. Shea (1977) identified a smaller maxillary sinus in cold-adapted populations and suggested it was a reflection of changes to the nasal region. Hubbe and colleagues (2009) found a large mean nasal height which resulted in a low nasal index in northern North American groups. They also identified a rounder skull in these populations and attributed it to heat conservation. Morphological and physiological adaptations will buffer against chronic cold stress, but when buffering systems are overwhelmed the general adaptation syndrome will be triggered resulting in skeletal stress markers.

3.5 Application of Evolution and Adaptation to Chronic Cold to the Biocultural Skeletal Stress Marker Pathway

Within the Tigara population chronic cold stress is buffered by cultural adaptations and host resistance and if these buffering systems are overrun then a physiological disruption will occur (Table 4). The Tigara occupied Point Hope in northwest Alaska from AD900-1700 (Figure 6) (Giddings 1967). Point Hope is 125 miles north of the Arctic Circle on the Lisburne Peninsula on the Chukchi Sea. It is located on the most western point of North America north of the Bering Strait where two narrow strips of land extend westward enclosing the Marryat Inlet to the northeast and the Aiautak Lagoon to the southeast.

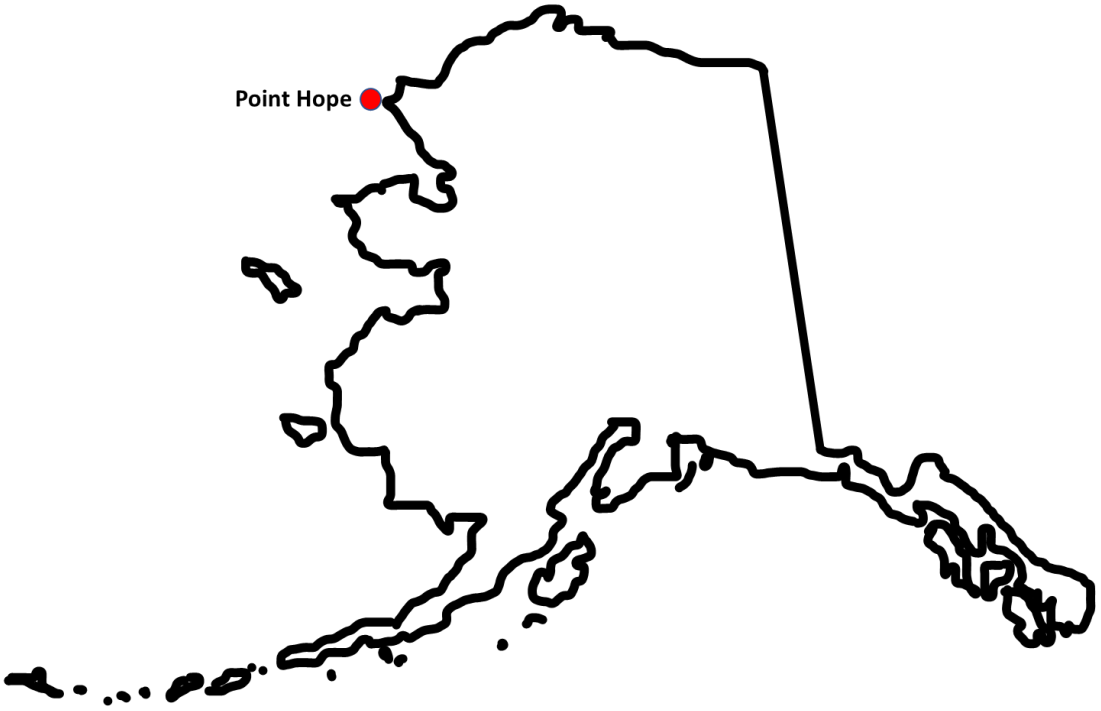

Figure 6: Map of Point Hope, Alaska

Table 4: Application of evolution and adaptation to extreme cold to the biocultural skeletal stress marker pathway

Environmental Constraints	Cultural Stressors	Cultural Buffer	Host Resistance	Skeletal Stress Marker
Climate Diet	Whale hunting Long exposure to cold Food instability	Clothing Shelter Fire Nutrition Food storage	Age Hidden heterogeneity Nutrition Disease Trauma Nonshivering thermogenesis Delayed shivering thermogenesis	Porotic hyperostosis (PH) Cribra orbitalia (CO) Sinusitis Antemortem tooth loss (AMTL) Granulomatous periapical lesion Periodontal disease Dental enamel hypoplasia (DEH)

The Tigara lived in the coastal arctic and faced harsh winters in 30-40°F below zero and high winds (Rainey 1947). They employed several cultural adaptations to survive the cold arctic winters. Their clothing was made from caribou and reindeer hide obtained via trade with inland Inuit for seal oil and seal skin and was so effective that core body temperature was not affected by the cold (Rainey 1947; So 1980). They lived in sod igloos heated with seal oil (Larsen and Rainey 1948). The high protein high fat Tigara diet buffered against the cold through maintenance of adipose tissue and a high metabolic response (Haas and Harrison 1977; Hart et al. 1962; Rennie et al. 1962).

Whale hunting was the most important activity in the Tigara society. It was dependent upon an ice-free corridor for the whales to migrate south in the spring (Hilton et al. 2014a). Preparation was extensive, time-consuming and required the cooperation of the entire population, while the hunting itself was dangerous and required a large group (Anderson 1984; Hilton et al. 2014a). The spring was dedicated to hunting migratory sea mammals (e.g., baleen whale, bearded seals, beluga and walrus) that followed the edge of the ice packs (Anderson 1984). The fall and winter were dedicated to religious ceremonies and preparing for the spring whaling season. Seal hunting occurred in November and December once the ice was packed up against the peninsula and daylight trips continued through April. Besides whale and seal hunting the Tigara also relied on gathering and trade with terrestrial populations (Michael and Rudenko 1961).

The Tigara relied primarily on a maritime hunting subsistence with minimal gathering. Access to year-round food was limited and food stores were essential for survival through the winter. This unstable food supply put the Tigara at risk for caloric and nutritional deficiencies. The Tigara diet composition was unbalanced with a heavy reliance on animal protein and fat and had high total cholesterol, low triglycerides and very high free fatty acids (Bang et al. 1971). Studies

of contemporary Inuit identified low calcium, folic acid, magnesium and vitamin D in Wainwright Eskimos (Mazess 1970) and hypervitaminosis A in Inuit (Landy 1985). However, Draper (1977) determined that the Wainwright Eskimos consumed adequate essential nutrients despite their low carbohydrate diet. So (1980) estimated that the Tigara diet was only 2% carbohydrates, which would be classified as a low carbohydrate ketogenic diet. Caloric restriction was most frequently experienced by the Tigara during the winter. This seasonal nutritional and caloric insecurity placed stress on the population that may have been embodied as stunted growth and nutritional diseases, indoor fires would have increased sinusitis risk, and a high grit diet would have caused granulomatous periapical lesions and AMTL Chronic cold exposure is not expected to affect anatomic or pathogenic risk factors of OM, but can increase OM risk by immunogenic factors of immune suppression (Gein and Sharav'eva 2017a; Gein and Sharav'eva 2017b; Sesti-Costa et al. 2012; Zhao et al. 2014).

There are few bioarcheological research studies on the Tigara that include the skeletal stress markers examined in this analysis. PH and CO were measured in the Tigara by Dabbs (2011) and Giardini and Eggers (2005). PH was found in 1.8% (n=277) of Tigara by Dabbs (2011) and 3.22% (n=31) by Giardini and Eggers (2005), whereas CO was found in 30.5% (n=269) by Dabbs (2011) and 13.04% (n=46) by Giardini and Eggers (2005) (Table 5). PH and CO were attributed to parasitic-induced anemia possibly acquired through close contact with marine animals which was compounded by lower host resistance due to chronic cold stress (Giardini and Eggers 2005).

Table 5: Prevalence of skeletal stress markers from previous studies of the Tigara population

Skeletal Stress Marker	Reference	Tigara Prevalence (n)
PH	Dabbs (2011)	1.8% (277)
	Giardini and Eggers (2005)	3.22% (31)
CO	Dabbs (2011)	30.5% (269)
	Giardini and Eggers (2005)	13.04% (46)
Periodontal disease	Schwartz and colleagues (1995)	69% (48)
AMTL	Schwartz and colleagues (1995)	52% (48)
	Giardini and Eggers (2005)	57.14% (63)
Dental abscess	Giardini and Eggers (2005)	61.90% (63)
DEH- maxillary incisor	Dabbs (2011)	6.9% (116)
DEH- canine	Dabbs (2011)	31.1% (116)

Costa (1982) identified severe localized periodontal disease in the Tigara between 35 and 40 years of age which was attributed to the high protein and high fat marine-based diet. Schwartz and colleagues (1995) also found periodontal disease in 69% of the Tigara (n=48) and 52% (n=48) had tooth loss. Costa (1980) found the Tigara only had moderate amount of AMTL. Within females there was an increase in incisor tooth loss in the 26 to 30 year age group and an increase in first and second molar tooth loss in the 36 to 40 year age group. This was most likely due to heavy occlusal wear and not likely the result of periodontal disease or caries (Costa 1980). Within the males there was an absence of 15 to 20% of their molars from 16 to 35 years of age which was mostly likely due to third molar agenesis. Tooth loss was progressive after the third decade and most likely due to heavy occlusal surface wear which exposes the pulp chambers (Costa 1980). Costa (1980) attributed anterior tooth loss to accident or heavy wear and posterior tooth absence to heavy wear, periodontal disease and agenesis. Similar to the findings by Schwartz and colleagues (1995), Giardini and Eggers (2005) found AMTL in 57.14% (n=63) of the Tigara studied while 61.90% (n=63) of those examined had at least one dental abscess. They attributed

the AMTL and dental abscesses to grit dental wear. The Tigara consumed large amounts of frozen meat that was dried on racks and stored underground which introduced sand particles during food processing and preparation resulting in grit in the diet (Costa 1982; El-Zaatari 2008).

DEH was recorded by Dabbs (2011) on the permanent maxillary incisors and permanent canines. She found that 6.9% (n=116) of the individuals had DEH on a permanent maxillary incisor and 31.1% (n=116) of the individuals had DEH on a permanent canine. She attributed this to acute dietary stress in response to the unpredictable whale-hunting culture and increased metabolic susceptibility to nutrition in children. Although the study of skeletal stress markers in the Tigara is limited, chronic cold, diet and the whaling culture play a major role in the stress of the Tigara.

4.0 Skeletal Stress Markers

Skeletal stress markers represent a disruption to physiological homeostasis at a specific point in time (Goodman et al. 1984c; Wood et al. 1992). Although skeletal stress markers are not indicative of cumulative health a biocultural approach which incorporates the environment, culture and biology can provide a framework for reconstructing the past (Goodman and Leatherman 1998; Reitsema and McIlvaine 2014). Skeletal stress markers are classified as general (i.e., resulting from any factor that stimulates the general stress response), or specific (i.e., resulting from a known etiology), and can disrupt growth and development or remodel skeletal tissue (Goodman et al. 1984c). The location and appearance of the skeletal stress marker is dependent on the type and duration of the stress, as well as the individual's response (Goodman et al. 1984c). Careful interpretation of the pathology can provide a biocultural perspective of the population. This chapter reviews the skeletal stress markers used in the following analysis.

4.1 Porotic Hyperostosis

Porotic hyperostosis (PH) is often combined with cribra orbitalia (CO) to describe cranial vault pathology resulting from anemia, but more recent research suggests these are two separate pathologies except in cases of severe anemia (Cole and Waldron 2019; Klaus 2017; McIlvaine 2015; Walker et al. 2009; Wapler et al. 2004). However due to this historical narrative PH and CO will be jointly discussed in the following paragraphs.

Increased red blood cell production (i.e., erythropoiesis) during childhood and adolescence causes hypertrophy of the parietal bone and orbital roof diploë known as PH and CO, respectively (Ponec and Resnick 1983). During the human life course the location of erythropoiesis shifts (Kent 1986; Stuart-Macadam 1985; Stuart-Macadam 1992). Erythropoiesis occurs in the yolk sac, spleen and liver, then bone marrow during fetal development, the cranial vault and long bones during childhood and adolescence, and the vertebrae, sternum and ribs during adulthood (Brickley 2018; Halvorsen and Bechensteen 2002; Hoffbrand et al. 2016). During periods of increased erythropoiesis the marrow within the diploë of the cranial vault expands which creates pressure on the tables causing the outer table to be resorbed resulting in a porous thinned outer table, thickened diploë and unaffected inner table characteristic of PH and CO (Ponec and Resnick 1983).

Angel (1966) was the first to describe PH and CO and hypothesized that the hypertrophied diploë was in response to iron-deficient anemia where red blood cells could not effectively deliver oxygen to the tissues triggering erythropoiesis. He attributed anemia to parasites, bacterial infections, environmental conditions and hereditary anemias. While chronic iron deficiency was the accepted etiology of PH and CO (Carlson et al. 1974; Stuart-Macadam 1987), recent researchers argue that iron deficiency actually inhibits PH and CO (Gowland and Western 2012; Lewis 2012; Rothschild 2002; Walker et al. 2009; Wapler et al. 2004). Walker and colleagues (2009) argue that iron is necessary for erythropoiesis and therefore a deficiency would prevent synthesis resulting in decreased red blood cell production inhibiting marrow hypertrophy and thus PH and CO. In cases of anemia where iron is adequate (e.g., red blood cell loss or destruction) the bone marrow will expand to support increased erythropoiesis (Hoffbrand et al. 2016; Metcalf 1969). Rather than iron deficient anemia, Walker and colleagues (2009) argue hemolytic or megaloblastic anemias result in PH and CO (Walker et al. 2009). Hemolytic anemia is the

premature destruction of red blood cells and may be genetic (e.g., thalassemia and sicklemia), or a result of extrinsic factors (e.g., toxins, cancer, cyanotic heart disease), and megaloblastic anemias are the result of chronic deficiencies (e.g., B_{12}, folic acid) or malabsorption (Antony 2011; Schrier and Price 2008)

While Walker and colleagues (2009) argue that iron inhibits erythropoiesis, Oxenham and Cavill (2010) suggest that in the case of ineffective erythropoiesis, the process continues and PH and CO can develop. In this scenario iron deficiency may lead to ineffective erythropoiesis, but not necessarily halt the process (Cavill 2002; Han et al. 2001; Oxenham and Cavill 2010). Ineffective erythropoiesis would result in red blood cells that are not viable and destroyed before they are released into circulation, which would increase marrow activity despite inadequate iron stores resulting in a vicious cycle and eventual marrow hypertrophy (Cavill 2002). PH and CO caused by ineffective erythropoiesis may result from iron-deficient anemia, genetic disorders, chronic blood loss or diet (Angel 1964; Holland and O'Brien 1997; Oxenham and Cavill 2010; Stuart-Macadam 1989). PH and CO appear among nursing infants but can have different etiologies. A B_{12} deficiency resulting from the combination of a maternal plant-based diet and weaning foods can result in both PH and CO (McIlvaine 2015). Whereas vitamin C deficiency resulting from diarrheal disease and intestinal parasites can produce CO via orbital roof hematoma, but will not produce PH (McIlvaine 2015). An individual may have multiple deficiencies, but if iron deficiency suppresses erythropoiesis then secondary deficiencies (e.g., vitamin B_{12} deficiency) may be masked (McIlvaine 2015). Therefore individuals who have multiple deficiencies and are the most stressed may not have PH or CO (McIlvaine 2015). Although PH and CO are used to assess nutritional stress a cautious approach to interpretation should be applied.

4.2 Cribra Orbitalia

Recent research suggests that CO is often misclassified as an expansion of the diploë resulting from anemia (Cole and Waldron 2019). Wapler and colleagues (2004) performed histological analysis on orbital roof lesions from 333 individuals from the Sudan and found that CO was the result of anemia in only 40% of the cases; inflammation (30%), taphonomy (20%) and other and undetermined causes (10%) were responsible for the remainder of the supraorbital lesions diagnosed as CO. This suggests that CO is multifactorial and the result of several different pathological processes. Cole and Waldron (2019) created a classification system that described the morphological characteristics of the bone dependent on the pathogenesis of the supraorbital lesion. The first classification was porosity with no change to the concavity of the orbit and was a normal variant that resulted from vascularization during growth and development. The second and third classifications were a convex orbit with either periosteal new bone formation or cortical bone expansion. They attributed these abnormal supraorbital lesions to trauma, hemorrhage and severe anemia. While PH is an accepted pathology of anemia, recent research questions whether CO should be included in this classification (Cole and Waldron 2019; Klaus 2017; McIlvaine 2015; Walker et al. 2009; Wapler et al. 2004). For this dissertation PH and CO were both considered a nutritional skeletal stress marker but were analyzed separately.

4.3 Sinusitis

Sinusitis is an inflammation of the mucous membrane of the paranasal sinuses (Brook 2009; Roberts 2007). The sinuses are bilateral air-filled passages lined with ciliated epithelium

and connected to the nasal cavity (Brook 2009). There are four pairs of sinuses, the maxillary, frontal, ethmoidal and sphenoidal (Brook 2009). The maxillary sinuses are the largest and drain into the middle meatus of the ethmoid via the sinus ostium (Roberts 2007). The sinuses function as a physical and chemical defense system against inhaled particulates by producing a particulate-trapping mucous which is carried out of the sinus and into the nasal cavity by ciliary action (Roberts 2007; Slavin et al. 2005); Maynard (1998) found that 90% of inhaled particulates are removed within 6 hours.

Disruption to this defense system can occur through mechanical or inflammatory obstruction of the ostium, which results in sinusitis (Brook 2009; Roberts 2007). When an obstruction occurs mucous cannot drain from the sinus causing a buildup of fluid within the sinus cavity. This results in a decreased gas exchange and decreased oxygen in the sinus cavity creating a bacteria friendly environment (Slavin et al. 2005). The introduction of pathogens or inability to fight infection can result in an inflammatory blockage and sinus infection (Brook 2009; Handelsman et al. 1984; Merrett and Pfeiffer 2000; Reid and Shearer 1994; Roberts 2007; Slavin et al. 2005; Umetsu et al. 1985).

There are five categories of a sinus infection, acute (infection lasting ≤4 weeks), recurrent acute (>4 acute infections within 1 year), subacute (infection lasting 4-12 weeks), chronic (infection lasting >12 weeks) and acute exacerbation of chronic (Brook 2009). Tovi and colleagues (1992) were the first to recognize that chronic sinusitis can result in osteitis. Chronic sinusitis is defined as two or more of the following symptoms experienced for ≥12 weeks: nasal blockage or obstruction, facial pain or pressure and reduction or loss of smell (Leung et al. 2016). Chronic sinusitis results in inflammation and a mucosal response that affects the underlying bone (Sethi 2015; Snidvongs et al. 2014). The pathological features of chronic sinusitis in skeletal tissue are

periosteal thickening, new woven bone formation and fibrosis (in only the most severe cases) (Lee et al. 2006; Snidvongs et al. 2014; Tovi et al. 1992). Aggravating factors include exposure to living organisms and man-made materials (Table 6).

Table 6: Aggravating factors of sinusitis

Organisms	Man-made
Mold growth (Bush et al. 2006) Dust mites (Jones 1999) Cockroaches (Jones 1999) Animals (Jones 1999) Dental infection (Panhuysen et al. 1997) Tuberculosis (Roberts and Buikstra 2003)	Building materials (Jones 1999) Furnishings (Jones 1999) Air conditioning (Jones 1999) Poor ventilation (Roberts 2007) Air pollution (DiGangi and Sirianni 2017; Roberts 2007) Overcrowding (Roberts 2007)

4.4 Dental Enamel Hypoplasia

Dental enamel hypoplasia (DEH) is a deficiency in enamel thickness and can range from a single pit to a completely absent enamel (Goodman and Rose 1991). DEH is the result of prolonged nonspecific stress and the location of the DEH is dependent on completeness of the crown at the time of the stress (Goodman and Armelagos 1985; Hammerl 2012; Kreshover 1960; Lukacs 1989; Sarnat and Schour 1941). Therefore DEH provide a kymographic record of stress that allows for estimation of age at the time of the stress (Armelagos et al. 2009; Hammerl 2012). DEH forms in deciduous teeth from the 5^{th} fetal month to the 12^{th} postnatal month and in permanent teeth from birth to adolescence with peak frequency occurring between 2 and 5 years of age (Shaw and Sweeney 1973; Swärdstedt 1966). The maxillary central incisors (amelogenesis: birth to 3 years) and mandibular canines (amelogenesis: 3 to 6.5 years) contain 95% of the DEH information (Goodman et al. 1980). Enamel does not remodel so it provides a permanent record of

developmental defects (Goodman and Armelagos 1988; Hillson 1996; Trammel and Kroman 2012).

Tooth development is strongly genetically controlled and begins in utero during the 6th intrauterine week as the epithelial cells swell to form the beginning of tooth germs (Hammerl 2012; Hillson 2005). By the 10th intrauterine week there are 10 swellings in each arcade which will become the enamel organs of the deciduous teeth (Hillson 1996). The enamel organs of the permanent teeth form between the 16 intrauterine week and shortly after birth (Hillson 1996). Tooth development begins with the thickening of the oral epithelium at the dental lamina and follows through three stages: bud, cap and bell stage (Hillson 2005). During the bell stage the epithelial cells lining the tooth germ differentiate into ameloblasts and deposit enamel matrix (Hillson 2005). Enamel deposition begins at the apex of the cusps and works down to form the crowns (Trammel and Kroman 2012). There are two stages of enamel deposition. During the first stage the ameloblasts secrete a matrix of organic and inorganic components from the Tome's process. Disruption of this process (e.g., mechanical or metabolic) can lead to constriction of the Tome's process (Hillson 1996). This results in a permanent line in the enamel called a striae of Retzius and is visible histologically (Trammel and Kroman 2012). If the stress is chronic the line will become visible macroscopically and is known as a dental enamel hypoplasia (DEH) (Trammel and Kroman 2012). Transverse lines or grooves are the most common form of DEH (Hammerl 2012). During the second stage the ameloblasts break down the organic component resulting in a crystal formation in the inorganic component (Trammel and Kroman 2012). However, any incongruities (e.g., DEH) will remain (Trammel and Kroman 2012).

There are three conditions that can disrupt amelogenesis resulting in enamel defects. The first is a hereditary anomaly, however these are rare and often found in conjunction with other

congenital problems (Goodman and Rose 1991; Sarnat and Schour 1941). Second is localized trauma; this is also rare and tends to be severe and only affect one tooth (Goodman and Rose 1991; Pindborg 1970; Suckling and Thurley 1984). Third is systemic metabolic stress, which is the most common cause of DEH and affects many teeth (Goodman and Rose 1991). Many stressors have been linked with DEH including nutritional deficiencies or excesses (e.g., fluoride supplementation) (Goodman et al. 1987; Infante 1974; Suckling and Purdell-Lewis 1982; Sweeney et al. 1971), diarrhea (Pindborg 1982), bacteria (Kreshover 1944), viral infection (Kreshover et al. 1954; Kreshover and Hancock JR 1956), fever (Kreshover et al. 1953), diabetes (Kreshover et al. 1953), parasitism (Suckling et al. 1983; Suckling et al. 1986), decreased socioeconomic status (Ford et al. 2009; Goodman and Armelagos 1988; Infante 1974; Infante and Gillespie 1974; Sweeney and Guzman 1966; Sweeney et al. 1969; Sweeney et al. 1971), respiratory infections (Ford et al. 2009), exposure to cigarette smoke (Ford et al. 2009), asthma (Ford et al. 2009; Jälevik et al. 2001), otitis media (Arnadottir et al. 2005; Ford et al. 2009), urinary tract infection (Ford et al. 2009; Tapias-Ledesma et al. 2003), chickenpox (Ford et al. 2009) and weaning (Corruccini et al. 1985; Goodman et al. 1987). This demonstrates that DEH is a general, or non-specific, stress marker and has many etiologies.

DEH has been associated with an increase in mortality (Amoroso et al. 2014; Cook and Buikstra 1979; Duray 1996; Goodman and Armelagos 1989; Steckel 2005). However, some researchers have found an increase in life expectancy with DEH (Bennike et al. 2005; Lewis 2002a; Saunders and Keenleyside 1999). Armelagos and colleagues (2009) describe three mechanisms for explaining the relationship between DEH and mortality. First an individual has lifelong frailty making them more susceptible to both DEH and early mortality. Second, the individual has experienced multiple (possibly unrelated) stressors over their lifetime resulting in

both DEH and early mortality. Third, the early life stress that resulted in DEH biologically damaged the individual decreasing their ability to further mitigate stressors (i.e., cumulative stress hypothesis).

4.5 Periodontal Disease

Periodontal disease is an inflammation of the periodontal tissues, which include the mandible, maxilla, alveolar process, periodontal ligaments, cementum, gingivae and mucosa (Hillson 1996). There are four stages of periodontal inflammation (Hillson 1996). Stage 1 begins with the swelling of the gingivae (i.e., oral mucosa) and an immune response (raised T- and B-lymphocytes) following exposure to plaque bacteria and lasts around one week (Hillson 1996). In stage 2 the gingiva continues to swell and there is an increase in macrophages and lymphocytes and lasts for a few weeks (Hillson 1996). The inflammatory response degrades the gingival collagen (Ejeil et al. 2003); it is not the bacteria, but the body's own immune response that damages the periodontal tissues. In stage 3 the inflammation often stabilizes and may remain stable for months or even reverse (Hillson 1996). There is a further increase in antibodies and lymphocytes and collagen disruption continues resulting in the loss of attachment of periodontal ligament. This creates a periodontal pocket which exposes cementum on the root surface and can result in the accumulation of sub-gingival plaque (Hillson 1996). Stages 1 to 3 are all classified as gingivitis and are an inflammation of the gingiva only. Stage 4 affects all periodontal tissues and is classified as periodontal disease (Hillson 1996). Stage 4 is a further disruption of the collagen and periodontal ligaments and absorption of the alveolar bone (Hillson 1996). This stage can last for years and cycle through resting and active phases (Hillson 1996). Risk factors for periodontal disease are

decreased immunocompetence, increased pathogen susceptibility, increased exposure to environmental factors and an increased inflammatory response (DeWitte and Bekvalac 2011). Periodontal disease rarely occurs before puberty and there is an increase in prevalence and severity with age (Hillson 1996).

Periodontal disease is associated with a wide range of adverse health outcomes and increased mortality (DeStefano et al. 1993; DeWitte and Bekvalac 2010; Garcia et al. 1998). Periodontal disease pathogens can travel from the periodontal pockets to the heart and lungs through the bloodstream (Amabile et al. 2008; Loos 2005). In the heart these pathogens trigger the release of proinflammatory immune cytokines (Loos 2005; Spahr et al. 2006) and cause increased platelet aggregation and trigger atherogenesis (Amabile et al. 2008; Demmer and Desvarieux 2006; Dorn et al. 2000; Kamer et al. 2008; Katz et al. 2002; Mattila et al. 2005; Pucar et al. 2007; Pussinen et al. 2004; Watts et al. 2008). Cardiovascular disease events linked with periodontal disease include stroke (Grau et al. 2004; Joshipura et al. 2003; Pussinen et al. 2004), atherosclerosis (Desvarieux et al. 2003) and coronary artery disease (Spahr et al. 2006).

Bacteria from dental plaque can travel to the lungs via the bloodstream or can be shed into the saliva and then aspirated into the lungs where an infection can occur (Li et al. 2000; Paju and Scannapieco 2007; Pan et al. 2009). Periodontal disease is a risk factor for pulmonary diseases including pneumonia, chronic bronchitis and emphysema (Hayes et al. 1998; Scannapieco and Rethman 2003; Terpenning et al. 2001). An improvement in oral health has been linked with decreased mortality from pneumonia (Paju and Scannapieco 2007; Pan et al. 2009). Additional comorbidities include cancer, systemic diseases and pregnancy complications (Table 7).

Table 7: Periodontal disease comorbidities

Cancer	Systemic Diseases	Pregnancy Complications
Oral (Zheng et al. 1990) Pancreatic (Michaud et al. 2008; Stolzenberg-Solomon et al. 2003) Upper gastrointestinal (Abnet et al. 2005) Lung (Michaud et al. 2008) Kidney (Michaud et al. 2008) Hematological (Michaud et al. 2008)	Obesity (Khader et al. 2009) Diabetes (Khader et al. 2006; Saremi et al. 2005; Taylor and Borgnakke 2008) Kidney disease (Shultis et al. 2007)	Preeclampsia (Ruma et al. 2008) Spontaneous preterm birth (Goepfert et al. 2004; Polyzos et al. 2009) Low birth weight (Boggess et al. 2006) Stillbirth (Mobeen et al. 2008)

4.6 Antemortem Tooth Loss

Antemortem tooth loss (AMTL) is the loss of a tooth prior to death and is evident by resorption of the alveolus. AMTL results from of a variety of causes including periodontal disease, trauma, carious lesions, attrition, extraction and nutritional deficiency, however the underlying etiology of AMTL is often not discernable (Lukacs 2007; Waldron 2008). The majority of AMTL results from periodontal disease and causation can be determined when AMTL occurs in the same location as periodontal disease (Al-Shammari et al. 2005; Hillson 1996; Waldron 2008). In the case of trauma, AMTL will likely affect adjacent teeth and the surrounding bone structure (Lukacs 2007; Lukacs and Hemphill 1990). Carious lesions and severe dental wear can result in AMTL if the tooth pulp becomes exposed and an abscess forms (Beckett and Lovell 1994; Featherstone 1987; Lukacs 1992; Lukacs and Pal 1993; Nelson et al. 1999). Tooth extraction may be the result of ritual ablation or for medical reasons (e.g., disease or malposition) (Lukacs 2007; Pietrusewsky and Douglas 1993; Tayles 1996). AMTL can result from nutritional deficiency, but it is also a risk factor for malnutrition due to impaired mastication (Musacchio et al. 2007; Stuart-Macadam 1989). Although AMTL is multifactorial it is used in conjunction with other dental pathologies to assess stress in skeletal remains.

4.7 Granulomatous Periapical Lesion

A granulomatous periapical lesion is alveolar bone destruction at the tip of the tooth root in response to tooth pulp infection. Pulp infection occurs when the pulp is exposed to oral bacteria via carious lesions, attrition, or trauma (Dias and Tayles 1997; Dias et al. 2007; Leigh 1925; Scott

and Turner 1988). The infection travels down through the pulp and out through the apical foramen infecting the periapical region (Dias et al. 2007; Scott and Turner 1988). Granulation tissue and inflammatory cells accumulate in the periapical region (Dias et al. 2007; Scott and Turner 1988).

Granulomatous periapical lesions are a group of lesions that begin as an infection of the dental pulp but manifest differently. A periapical granuloma forms when the periapical region fills with inflammatory cells resulting in osteoclastic remodeling forming a cavity less than 3mm in diameter with smooth walls. If the periapical granuloma is untreated then an apical periodontal cyst can develop and the cavity will enlarge. If pyogenic bacteria infect the cavity it will become an abscess and fill with pus, becomes painful and causes fever and malaise. In the most severe cases, the infection can spread to the bone marrow cavity resulting in osteomyelitis causing systemic responses of cellulitis, septicemia and even death. With osteomyelitis bone necrosis, sequestrum, involucrum and cloaca would be present. Often granulomatous periapical lesions are diagnosed as dental abscess. An abscess contains pyrogenic bacteria and indicates the individual was ill with symptoms of pain, fever and malaise. However, not all dental lesions are abscesses which implies pus and physical symptoms. Therefore for this analysis granulomatous periapical lesion was used to describe dental lesions (Figure 7 and Table 8). (Dias and Tayles 1997)

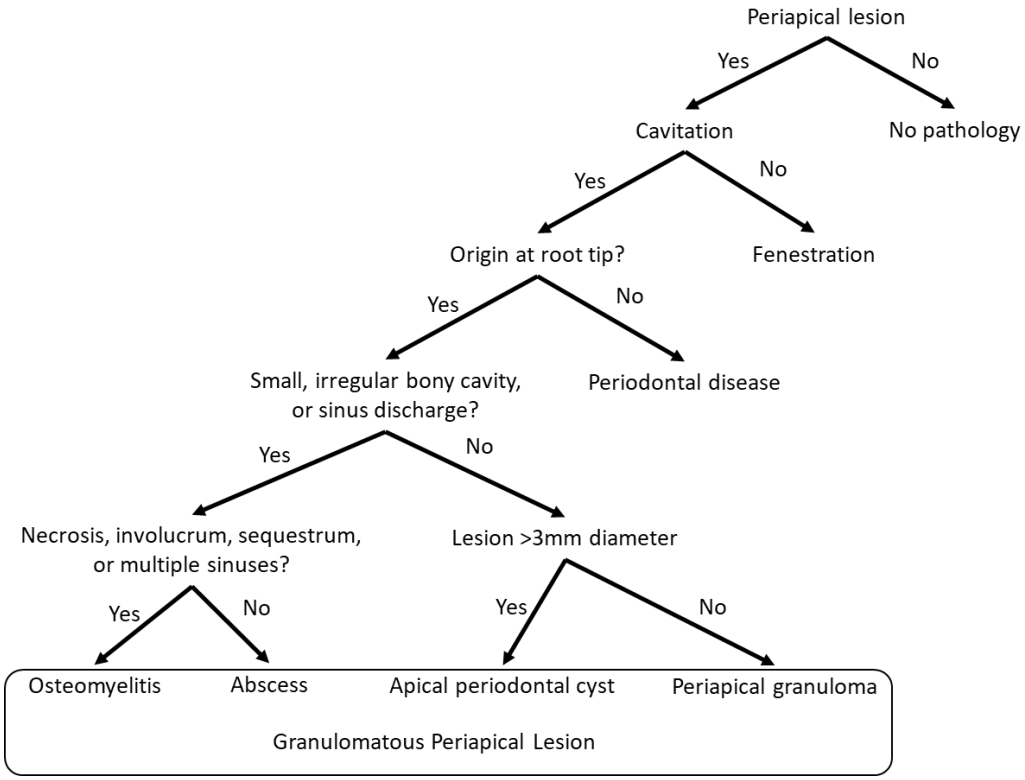

Figure 7: Periapical lesion differential diagnosis decision tree

Table 8: Periapical lesion differential diagnosis

Pathology	Description
No pathology	Alveolar bone intact
Fenestration	Bone defect exposing root tip
Periodontal disease	Periodontal inflammation
Periapical granuloma	Smooth round lesion <3mm diameter
Apical periodontal cyst	Smooth round lesion >3mm diameter
Abscess	Irregular, rough lesion
Osteomyelitis	Bone infection

5.0 Materials and Methods

5.1 Anatomical Collections

Skulls and CT scans from the Hamann-Todd Human Osteological Collection, Robert J. Terry Anatomical Skeletal Collection and Tigara, Point Hope Skeletal Collection were used for this analysis.

5.1.1 Hamann-Todd Human Osteological Collection

Carl A. Hamann initiated the Hamann-Todd Human Osteological Collection (Hamann-Todd) in 1893 at Western Reserve University (now Case Western University). In 1912 he was replaced by T. Wingate Todd who continued the collection until 1938 (Muller et al. 2017). The collection is now curated at the Cleveland Museum of Natural History, Cleveland, OH (Muller et al. 2017). It is composed of more than 3,100 individuals, mostly unclaimed bodies, born between 1825 and 1910 (Muller et al. 2017). Demographic information, cause of death and pathologies were recorded (Muller et al. 2017).

Twenty skulls from the Hamann-Todd collection with a CT scan or mastoidectomy were included in this analysis. Of the 13 skulls with a CT scan there were no records of who, why or how the CT scans were obtained and there were no obvious sample selection criteria. Age,

ancestry[1] and biological sex was determined by morgue records. The ages were grouped into young (16-35 years), middle (36-55 years) and old (56+ years) adult (Petry 2002).

5.1.2 Robert J. Terry Anatomical Skeletal Collection

The Robert J. Terry Anatomical Skeletal Collection (Terry) was composed at Washington University by Robert J. Terry from 1920 to 1941 and following Terry's retirement Mildred Trotter continued the collection until 1967 (Hunt and Albanese 2005). It is composed mainly of unclaimed bodies from hospitals and institutional morgues in Missouri (Hunt and Albanese 2005). The Terry collection is stored at the National Museum of Natural History in Washington, D.C. (Muller et al. 2017). It is composed of 1,728 individuals ranging from 14 to 102 years of age (Hunt and Albanese 2005). Demographic data (age, sex and ancestry), cause of death and pathologies were recorded (Albanese 2003).

Copes (2012) CT scanned 105 (69 male and 36 female) skulls from the Terry collection and made the scans available online at https://www.lynncopes.com/human-ct-scans.html. She randomly selected individuals with no evidence of scurvy, rickets, treponemal disease, Paget's disease, osteomyelitis, or severe osteoarthritis (Copes 2012). All 105 skulls that were CT scanned by Copes (2012) were included in this dissertation analysis. Age, ancestry and biological sex were determined by morgue records. The ages were grouped into young (16-35 years), middle (36-55 years) and old (56+ years) adult (Petry 2002).

[1] In this dissertation ancestry is used as a proxy for race (African American in place of black and European American in place of white) in the Hamann-Todd and Terry collections to keep in line with current anthropological terminology. Within the Hamann-Todd collection race was recorded as black or white and within the Terry collection 'color or race' was recorded as negro or white. Although researchers should cautiously interchange ancestry with race, within these collections there is likely substantial overlap between African American and black race and European American and white race.

5.1.3 Tigara, Point Hope Skeletal Collection

Froelich Rainey and Helge Larsen excavated 253 Tigara individuals from Point Hope, Alaska in 1941 (Costa 1977; Larsen and Rainey 1948). The Tigara were precontact Inuits (AD 900-1700) that resided at the westernmost tip of North America in the Arctic Circle (Giddings 1967). The collection is currently stored at the American Museum of Natural History in New York, New York (Copes 2012).

Copes (2012) CT scanned 204 (95 male and 109 female) skulls from the Tigara collection and made the scans available online at https://www.lynncopes.com/human-ct-scans.html. The individuals CT scanned by Copes (2012) were randomly sorted and 35 male and 34 female[2] skulls with complete MACS were included in this dissertation analysis. The biological sex recorded in the museum documents was used. Age estimates reported by Costa (1977) were used to assign ages to 61 out of 69 individuals in the Tigara sample[3]; age was assigned in 5-year intervals using Todd's (1920) stages of pubic symphysis development.

5.2 Pathologies

Three types of pathologies were examined: dietary deficiencies, infectious diseases and dental pathologies (Table 9).

[2] Only 34 female skulls had complete mastoids for analysis.
[3] The age ranges reported by Costa (1977) were used because the Tigara postcrania were stored in a different area of the museum and were not accessible at the time of cranial examination, and aging methods were not used for the other skeletal collections. Of the eight individuals not aged by Costa (1977), three individuals were aged by the Goldman Data Set (https://web.utk.edu/~auerbach/GOLD.htm) and Hilton (2014), but those age ranges were not included because different aging techniques were used and it only provided age ranges for an additional three individuals.

Table 9: Pathologies examined

Dietary Deficiencies	Infectious Diseases	Dental Pathologies
Porotic hyperostosis (PH) Cribra orbitalia (CO)	Sinusitis Otitis media (OM)	Antemortem tooth loss (AMTL) Granulomatous periapical lesion Periodontal disease Dental enamel hypoplasia (DEH)

5.2.1 Dietary Deficiencies

5.2.1.1 Porotic Hyperostosis and Cribra Orbitalia

PH and CO were recorded separately and scored for degree of expression following Larsen and Crosby (2002) (Table 10) (Figures 8 and 9). For PH the ectocranial vault including the frontal, parietal, occipital and temporal bones were examined for lesions. For CO the orbital roof was examined for lesions. The lesions were recorded as absent, present, gross lesion with cranial expansion and exposed diploë, and bone not present (see Larsen and Crosby (2002), 424; Table 11). All lesions were recorded and photographed. A differential diagnosis of the ectocranial vault pathologies was performed. The differential diagnosis included natural variation, taphonomy, treponemal disease, vitamin D deficiency and rickets, vitamin C deficiency and scurvy, and tuberculosis. Only present/absent data was used in the statistical analysis.

Table 10: Dietary deficiency data collected

PH	CO
Present/absent[a] Severity	Present/absent[a] Severity

[a]Data used in statistical analysis

Figure 8: PH with severity score 1 (Hamann-Todd#744)
Photograph courtesy of Lyman Jellema, Cleveland Museum of Natural History. Photograph by Allison P. Gremba.

Figure 9: CO with severity score 1 (Terry#880)
Photograph courtesy of Department of Anthropology, Smithsonian Institution. Photograph by Allison P. Gremba.

Table 11: PH and CO severity scoring protocol

Severity	Code	Description
Absent	0	Absent
Mild	1	Lesion present on at least one side
Severe	2	Gross lesion with cranial expansion and exposed diploë
Not observable	9	Not observable

5.2.2 Infectious Diseases

5.2.2.1 Sinusitis

The maxillary sinus was examined for osseous remodeling using a 30° and 70° endoscope with a LED light attachment through careful insertion of the endoscope anteriorly into the nasal aperture and posteriorly into the ethmoid between the medial pterygoid plate and vomer. Evidence of sinusitis and remodeling type (i.e., pitting, spicule-type bone formation, remodeled spicules, white pitted bone, thickened walls, porous walls and lobules of white bone) (Figure 10) was recorded as present, absent or not observable following the classification system from Boocock and colleagues (1995). Sinusitis was photographed if possible. Only present/absent data was used in the statistical analysis (Table 12).

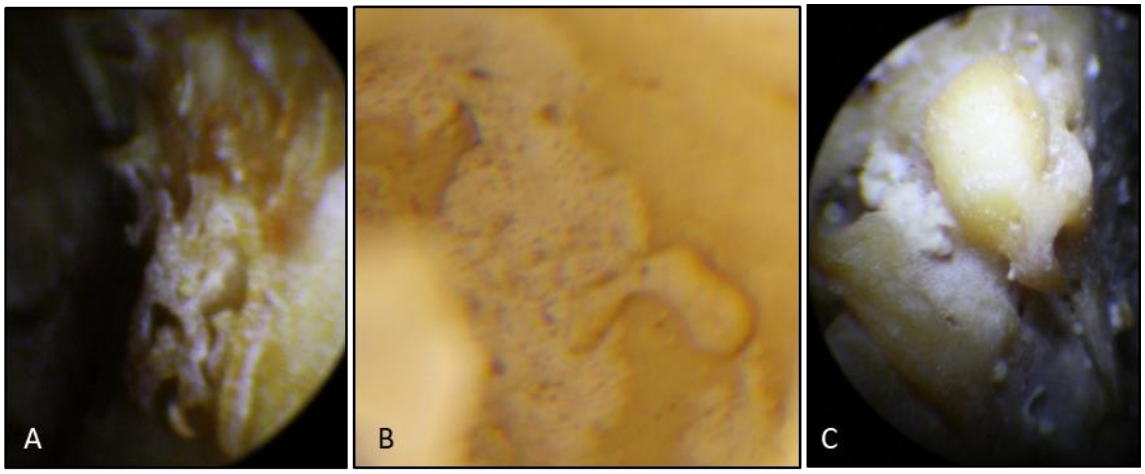

Figure 10: Sinusitis with spicule type bone formation (Terry#906) (A), white pitted bone (Terry#236) (B) and lobules of white bone (Terry#1281) (C)
Photographs courtesy of Department of Anthropology, Smithsonian Institution. Photographs by Allison P. Gremba.

Table 12: Infectious disease data collected

Sinusitis	OM
Present/absent[a]	MACS V <5ml[a]
Remodeling types	Diseased ossicles (includes stapedial footplate fixation)
	Mastoid abscess
	Mastoidectomy
	Recorded cause of death

[a]Data used in statistical analysis

5.2.2.2 Otitis Media

OM was diagnosed as present/absent by MACS <5ml (Table 12). Other measures of OM were recorded including diseased ossicles, mastoid abscess, mastoidectomy and recorded cause of death but not included in the analysis (Figure 11). Present/absent for MACS volume <5ml was used in the statistical analysis.

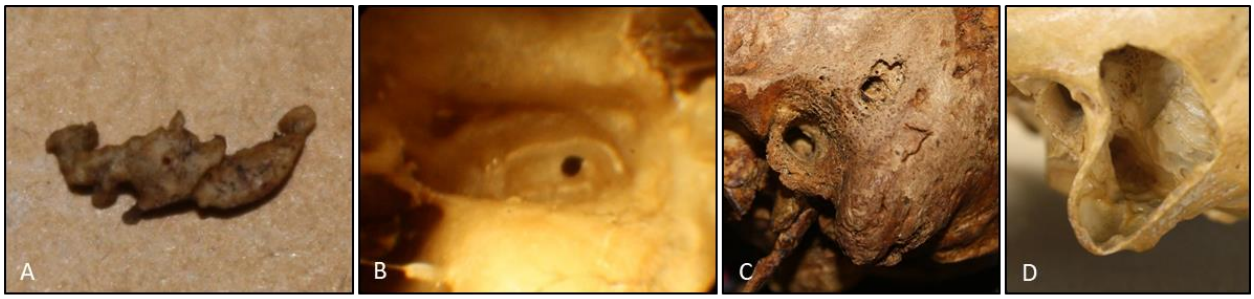

Figure 11: Alternative evidence of OM of ankylosed incus and malleus (Tigara#228) (A), stapedial footplate fixation (Terry#1154) (B), mastoid abscess (Tigara#235) (C) and mastoidectomy (Hamann-Todd#620) (D)
Photographs courtesy of Division of Anthropology, American Museum of Natural History (A), Department of Anthropology, Smithsonian Institution (B and C), and Lyman Jellema, Cleveland Museum of Natural History (D). Photographs by Allison P. Gremba.

MACS volume (ml) and surface area (cm^2) was measured from high resolution CT scans that were previously imaged by Copes (2012) from the Terry and Tigara collections (Siemens Somatom Spiral Scanner, 70µA, 110kV, table height 215, slice width 1.0mm, reconstruction 0.5mm). The specifications and protocol for the CT scans from the Hamann-Todd collection were not available.

CT scans were analyzed following Swarts and colleagues (2012). The CT scan image sequence was imported into ImageJ (https://imagej.nih.gov/ij/) (Abràmoff et al. 2004) starting with the first slice that the MACS was visible in the temporal bone. For the Point Hope and Terry collection every 4th image was imported (reconstruction 0.5mm x 4 = 2mm) and every 7th image was imported for the Hamann-Todd collection (reconstruction 0.377mm x 7 = 2.6mm). The sequence was converted to binary with a black background automatically calculated by ImageJ. Slices after the last visible MACS of the mastoid were deleted. The image with the largest area of MACS was then identified (Figure 12: A) and the MACS was "cut out" using the box tool for the entire image sequence and the surrounding area deleted using the clear tool (Figure 12: B). Slices were then individually viewed and the paintbrush tool in black was used to delete any air space that was not MACS and individual air cells were "closed off" using the pencil tool in white (Figure 12: C). The area of the air cells from the image sequence was measured in ImageJ using the threshold function to exclude the bone and area greater than 2000mm^2 to exclude the background. The overlay function was used and each image in the sequence was checked to ensure only the air cells were included in the calculation (Figure 12: D). ImageJ recorded the area (mm^2) and perimeter (mm) of each air cell by CT slice. The air cell areas and perimeters for each slice were summed and then the total area and perimeter of all the slices were summed. Area (mm^2) was used to calculate volume (ml) and perimeter (mm) was used to calculate surface area (cm^2). First millimeters were converted to centimeters by multiplying by 0.01 for the area and 0.1 for the surface area. Next the section interval (Point Hope and Terry: 2mm, Hamann-Todd: 2.6mm) was converted to centimeters by multiplying by 0.1. The section interval in centimeters was multiplied by the area (cm^2) and perimeter (cm) to calculate MACS volume (ml) and surface area (cm^2), respectively. The calculations are reported in Table 13. A MACS volume <5ml was scored as

positive for OM (Csakanyi et al. 2010; Swarts et al. 2012). MACS volume was also calculated for each side (left and right). Only present/absent data was used in the statistical analysis.

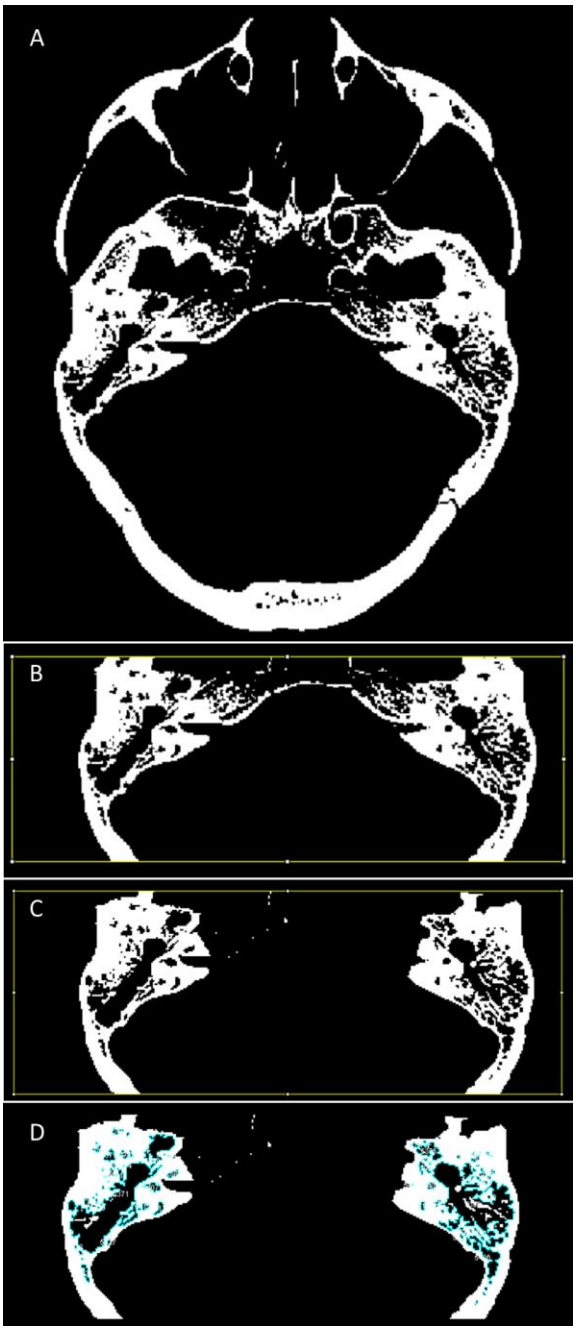

Figure 12: Inferior view of a CT scan slice (A) with the MACS sectioned out (B and C) and an overlay showing the area included in the MACS total volume (D)

Table 13: MACS volume and surface area calculations

	Volume (ml)	Surface Area (cm^2)
Terry and Tigara	V= area x 0.0020	SA= perimeter x 0.020
Hamann-Todd	V= area x 0.0026	SA= perimeter x 0.026

Other evidence of OM was examined, but not included in the statistical analysis because the purpose of this analysis was to determine if MACS hypopneumatization was a skeletal stress marker of OM. Ex situ ossicles were examined using a 70° endoscope with a LED light attachment for erosive changes indicative of OM described by Bruintjes (1990). The middle ear cavity was also examined using a 30° and 70° endoscope with a LED light attachment to view the oval window and diagnose stapedial footplate fixation described by Dalby and colleagues (1993). The temporal bone and mastoid process were examined visually for mastoid abscess and mastoidectomy. In individuals with a mastoidectomy the mastoid process lateral wall and mastoid air cells were surgically removed. The cause of death from the morgue record for the Hamann-Todd and Terry collections was reviewed for complications from OM.

5.2.3 Dental Pathology

5.2.3.1 Antemortem Tooth Loss

The mandibular and maxillary alveoli were examined for evidence of tooth loss and resorption of the tooth socket. Each tooth was assessed and the socket had to be completely resorbed to be classified as AMTL (Figure 13). AMTL was scored as present/absent following Cucina and Tiesler (2003) (Table 14). For unerupted teeth and congenital missing teeth the code '9' was used. If the third molar was not present the code '9' was used unless alveolar remodeling

was present. Present/absent data for number of individuals with AMTL and mean number of AMTL per individual was used in the statistical analysis (Table 15). Frequency graphs of the mean number of AMTL by individual for each cohort and number of individuals with edentulism (Figure 14) by cohort were created.

Figure 13: AMTL of the right and left mandibular second molars (Hamann-Todd#1112)
Photograph courtesy of Lyman Jellema, Cleveland Museum of Natural History. Photograph by Allison P. Gremba.

Figure 14: Edentulous maxilla (A) and mandible (B) (Terry#1154)
Photographs courtesy of Department of Anthropology, Smithsonian Institution. Photographs by Allison P. Gremba.

Table 14: AMTL scoring protocol for each tooth

Code	Description
0	No AMTL
1	AMTL
9	AMTL not observable

Table 15: Dental data collected

AMTL	Granulomatous periapical lesion	Periodontal disease	DEH
Teeth affected Present/absent[a] Number per mouth[a] Edentulism	Present/absent[a] Number per mouth[a] Total lingual number Total buccal number	Present/absent[a] Severity per tooth Highest severity per individual	Severity per tooth Present/absent from canines and maxillary incisors[a] Maximum severity from canines and maxillary incisors[a]

[a]Data used in statistical analysis.

5.2.3.2 Granulomatous Periapical Lesion

The maxilla and mandible were visually examined for evidence of granulomatous periapical lesions on the lingual and buccal surfaces (Figure 15). Granulomatous periapical lesion was scored as present/absent and lingual/buccal for each tooth following Buikstra and Ubelaker (1994) (Table 16). Present/absent data for the number of individuals with at least one granulomatous periapical lesion and number of granulomatous periapical lesions per individual were used in the statistical analysis (Table 15).

Figure 15: Granulomatous periapical lesion (Terry#357)
Photograph courtesy of Department of Anthropology, Smithsonian Institution. Photograph by Allison P. Gremba.

Table 16: Granulomatous periapical lesion scoring protocol for each tooth

Code	Description
0	No lesion
1	Buccal lesion
2	Lingual lesion
9	Lesion not observable

5.2.3.3 Periodontal Disease

The maxilla and mandible were examined for evidence of inflammation and remodeling. Individual in situ teeth were scored for periodontal disease following Ogden (2008) (Figure 16) (Table 17). The tooth with the highest score for periodontal disease was assigned as the periodontal disease score for the individual. In the individuals with periodontal disease the highest score was recorded from the premolars and molars and all of the individuals without molars and second premolars exhibited no evidence of periodontal disease in the teeth present and therefore they were excluded from the analysis (n=37). Present/absent data for number of individuals with periodontal disease and maximum severity score per individual was used in the statistical analysis (Table 15).

Figure 16: Periodontal disease with severity score of 4 (Terry#969)
Photograph courtesy of Department of Anthropology, Smithsonian Institution. Photograph by Allison P. Gremba.

Table 17: Periodontal disease severity scoring protocol

Severity	Code	Description
No disease	1	Alveolar margin meets root at acute angle
Mild	2	Alveolar margin is blunt with slightly raised edge
Moderate	3	Alveolar margin is rounded with porous trough
Severe	4	Alveolar margin ragged and porous with irregular trough
Not observable	9	Not observable

5.2.3.4 Dental Enamel Hypoplasia

DEH was scored by the number of hypoplasias for each tooth following Steckel and Rose (2002) (Table 18). The highest score recorded from the canines and maxillary incisors (Figure 17) was used as the severity score for the individual. DEH was recorded as present for the individual if DEH occurred on the canines or maxillary incisors. Present/absent data for number of individuals with DEH and DEH severity by individual was used in the statistical analysis (Table 15).

Figure 17: DEH severity score 2 on a mandibular canine (Tigara#541) (A) and maxillary incisor (Hamann-Todd#859) (B)
Photographs courtesy of Division of Anthropology, American Museum of Natural History (A), Lyman Jellema, Cleveland Museum of Natural History (B). Photographs by Allison P. Gremba.

Table 18: DEH severity scoring protocol

Code	Description
0	No DEH
1	1 DEH
2	≥2 DEH
9	Tooth not present

5.3 Statistical Analyses

All statistical analyses were performed using IBM SPSS Statistics 24.0 (IBM Corp., 2016).

5.3.1 Demographic Analysis

A Chi-Square Test of Independence ($p<0.05$) between collection, sex, ancestry and age group was performed. The Fischer's Exact Test (5-minute run time) was used if greater than 20% cells had an expected count less than 5; if SPSS could not compute the Fischer's Exact Test because of "insufficient memory" then the Monte Carlo Method (99% CI, 10,000 samples) was

used. The strength of association was reported as Phi for a 2x2 table and Cramer's V for a larger table. Post-hoc analysis for statistically significant adjusted standardized residuals (Z-score > |1.96|) was performed using the Bonferroni correction.

5.3.2 Analysis by Pathology

Following the method described in Section 5.3.1, a Chi-Square Test of Independence ($p<0.05$) between pathology and cohort was performed for frequency (i.e., PH, CO, sinusitis, OM, AMTL, granulomatous periapical lesion, periodontal disease and DEH) and ranked data (i.e., periodontal disease and DEH severity). Student's t-test and analysis of variance (ANOVA) ($p<0.05$) was performed for continuous variables (i.e., mean number of AMTL per individual and mean number of granulomatous periapical lesions per individual). Tukey's HSD post-hoc analysis was performed for statistically significant groups. If equal variances were not assumed the Levene statistic ($p<.05$) was used.

5.3.3 Analysis of OM as a Skeletal Stress Marker

OM was analyzed against the traditional skeletal stress markers using a Chi-Square Test of Independence ($p<0.05$) following the method described in Section 5.3.1 for ranked and frequency pathology variables. A Student's t-test ($p<0.05$) following the method described in Section 5.3.2 was used for continuous pathology variables.

6.0 Results

6.1 Demographics

The sample demographics are reported in Tables 19 and 20. A Chi-Square Test of Independence ($p<0.05$) (Table 21) followed by post-hoc analysis using a Bonferroni correction (Table 22) revealed the Hamann-Todd had a high proportion of young adult, the Terry had a low proportion of young adult and high proportion of old adult, and the Tigara had a low proportion old adult ($n=0$) and a high proportion of middle adult ($p<0.005$) (Figure 18). For the ancestry by age group crosstabulation the European American group had a low proportion of young adult and a high proportion of old adult ($p<0.005$) (Figure 19).

Table 19: Demographics

Cohort	N	Group	Frequency	Percentage
Collection	194	Hamann-Todd	20	10%
		Terry	105	54%
		Tigara	69	36%
Sex	194	Male	120	62%
		Female	74	38%
Ancestry	194	African American	78	40%
		European American	47	24%
		Native American	69	36%
Age Group	186	Young Adult	64	34%
		Middle Adult	70	38%
		Old Adult	52	28%

Table 20: Demographics by collection

Collection	Group	Cohort	Frequency	Percentage
Hamann-Todd (n=20)	Sex	Male	16	80%
		Female	4	20%
	Ancestry	African American	16	80%
		European American	4	20%
		Native American	0	0%
	Age Group	Young Adult	15	75%
		Middle Adult	3	15%
		Old Adult	2	10%
Terry (n=105)	Sex	Male	69	66%
		Female	36	34%
	Ancestry	African American	62	59%
		European American	43	41%
		Native American	0	0%
	Age Group	Young Adult	21	20%
		Middle Adult	34	32%
		Old Adult	50	48%
Tigara (n=69)	Sex	Male	35	51%
		Female	34	49%
	Ancestry	African American	0	0%
		European American	0	0%
		Native American	69	100%
	Age Group[a]	Young Adult	28	46%
		Middle Adult	33	54%
		Old Adult	0	0%

[a]n=61

Table 21: Demographics χ^2 statistics

	Collection	Sex	Ancestry
Sex	χ^2= 7.077, **p= .029** v= .191[b]		
Ancestry	χ^2= 198.879, **p= .000**[a] v= .716[b]	χ^2= 5.78, p= .056	
Age Group	χ^2= 60.020, **p= .000** v= .402[b]	χ^2= 1.647, p= .439	χ^2= 74.813, **p= .000** v= .448[b]

Statistically significant p-values indicated in bold
[a]Not statistically significant between Hamann-Todd and Terry
[b]Cramer's V

Table 22: Demographics adjusted standardized residuals

Crosstabulation	Variable	Obs. Count	Exp. Count	Z-score	Bonferroni adjusted p-value
Collection x Sex	Tigara Male	35	42.7	-2.4	p > .008
	Tigara Female	34	26.3	2.4	p > .008
Collection x Age Group	Hamann-Todd Young Adult	15	6.9	4.0	**p < .005**
	Hamann-Todd Middle Adult	3	7.5	-2.2	p > .005
	Terry Young Adult	21	36.1	-4.7	**p < .005**
	Terry Old Adult	50	29.4	6.8	**p < .005**
	Tigara Young Adult	28	21	2.3	p > .005
	Tigara Middle Adult	33	23	3.2	**p < .005**
	Tigara Old Adult	0	17.1	-5.9	**p < .005**
Ancestry x Age Group	African American Young Adult	34	26.8	2.2	p > .005
	European American Young Adult	2	16.2	-5	**p < .005**
	European American Middle Adult	11	17.7	-2.3	p > .005
	European American Old Adult	34	13.1	7.8	**p < .005**

Statistically significant p-values indicated in bold

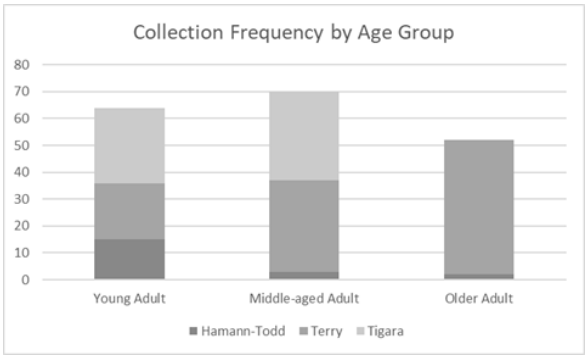

Figure 18: Bar graph illustrating the number of individuals in each collection by age group

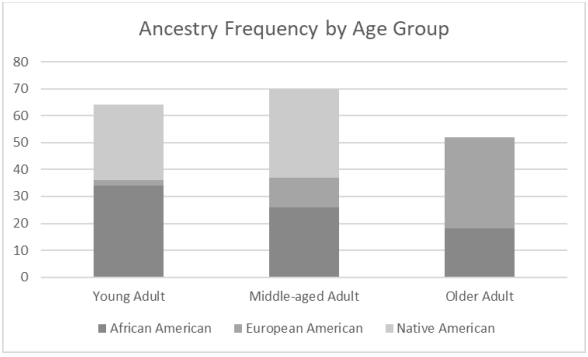

Figure 19: Bar graph illustrating the number of individuals in each ancestry cohort by age group

6.2 Dietary Deficiencies

6.2.1 Porotic Hyperostosis

There were eight skulls with ectocranial vault pathologies located on the parietal bones. A differential diagnosis of the cranial lesions in the absence of the post-crania was performed (Tables 23 and 24; Figure 20). Seven skulls were diagnosed with PH, six from the Hamann-Todd collection and one from the Tigara collection.

Table 23: Pathologies excluded from PH differential diagnosis

The following pathologies were excluded:
1. Vitamin D deficiency and rickets **Location**: Cranial vault. Rarely affects the skull in adults. **Appearance**: Irregular nodular masses and thickening of the outer table; thinner, softer vault bones; craniotabes (flattening of the parietal and occipital bones); frontal bossing resulting in a "square head". **Excluded because** none of the skulls exhibit cranial lesions or changes associated with vitamin D deficiency or rickets.
2. Vitamin C deficiency and scurvy **Location**: Parietals, greater wing of the sphenoid, lateral zygomatic arches, maxillary alveolus, lambdoidal area of occipital. **Appearance**: Cortex-penetrating pores from pinpoint to pen size; periosteal new bone formation; frontal and parietal bossing. **Excluded because** the lesions are only located on the parietal and frontal bones and there is no skull bossing present.
3. Tuberculosis **Location**: Cranial vault and may cross cranial sutures. The skull is a rare site of skeletal tuberculosis. Adults almost always a solitary tubercular focus. **Appearance**: Round lytic lesion with or without moth-eaten appearance; perforation of inner and outer tables; small areas of destruction less than 2cm. **Excluded because** the skulls exhibited porosity and remodeling of the outer table and did not exhibit round lytic lesions that are associated with tuberculosis.

(Aufderheide and Rodriguez-Martin 1998)

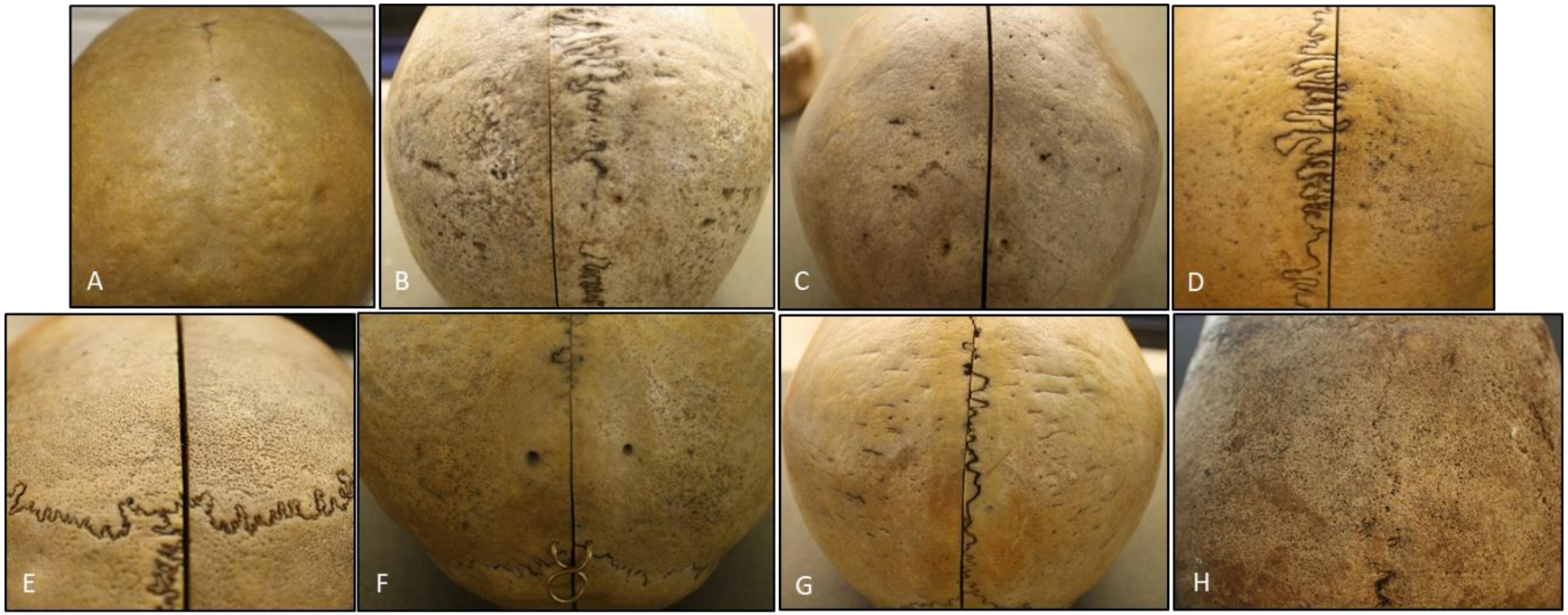

Figure 20: Ectocranial vault pathology
(A: Terry#1336, B: Hamann-Todd#638, C: Hamann-Todd#744, D: Hamann-Todd#858, E: Hamann-Todd#859, F: Hamann-Todd#1120, G: Hamann-Todd#1130, H: Tigara#390)
Photographs courtesy of Department of Anthropology, Smithsonian Institution (A), Lyman Jellema, Cleveland Museum of Natural History (B-G), and Division of Anthropology, American Museum of Natural History (H). Photographs by Allison P. Gremba.

Table 24: PH differential diagnosis

Demographic Data						PH						Treponemal disease				Differential diagnosis
Collection	ID	Sex	Ancestry	Age/ Age range	Figure	Symmetrical	Outer table	Frontal bone	Parietal bone	Multiple, discreet, pin-sized lesions	Coral or sieve-like appearance	Frontal bone	Cavitating lesions	Nasopalatal involvement	Active, remodeling and stellate lesions	
Terry	1336	M	B	68	A		X		X							Natural variation
Hamann-Todd	638	M	B	33	B	X	X	X	X	X	X	X	X	X		PH & treponemal disease
Hamann-Todd	744	M	B	22	C	X	X	X	X	X	X	X	X			PH & unknown
Hamann-Todd	858	M	B	24	D	X	X		X	X						PH
Hamann-Todd	859	M	B	26	E	X	X	X	X	X	X					PH
Hamann-Todd	1120	M	B	28	F	X	X		X	X						PH
Hamann-Todd	1130	F	B	22	G	X	X		X	X						PH & unknown
Tigara	390	M	NA	21-25	H	X	X	X	X	X		X				PH & taphonomy

The frequency data for PH are reported in Table 25. A Chi-Square Test of Independence ($p<0.05$) followed by post-hoc analysis using a Bonferroni correction (Table 26) revealed significant relationships between collection and age group ($p<0.008$). The Hamann-Todd had a high proportion and the Terry had a low proportion (n=0) of PH (Figure 21). All of the individuals with PH were in the young adult age group (Figure 21) and there were significantly more individuals in the young adult age group with PH than expected.

Table 25: PH frequency

Cohort	Group	n	PH Frequency	Percentage
Collection	Hamann-Todd	20	6	30%
	Terry	105	0	0%
	Tigara	69	1	1%
Sex	Male	120	6	5%
	Female	74	1	1%
Ancestry	African American	78	6	8%
	European American	47	0	0%
	Native American	69	1	1%
Age Group[a]	Young Adult	64	7	11%
	Middle Adult	70	0	0%
	Old Adult	52	0	0%
Total		194	7	4%

[a]n=186

Table 26: PH χ^2 test and adjusted standardized residuals

Cohort	Test Statistic	Variable	Obs. Count	Exp. Count	Z-score	Bonferroni adjusted p-value
Collection	X^2= 23.308[a], **p= .000** v= .481[b]	Hamann-Todd	6	0.7	6.7	**p < .008**
		Terry	0	3.8	-2.9	**p < .008**
Sex	X^2= 1.752[a], p= .255					
Ancestry	X^2= 5.153[a], p= .063					
Age Group	X^2= 11.215[a], **p= .001** v= .273[b]	Young Adult	7	2.4	3.7	**p < .008**
		Middle Adult	0	2.6	-2.1	p > .008

Statistically significant p-values indicated in bold
[a]Fischer's Exact Test
[b]Cramer's V

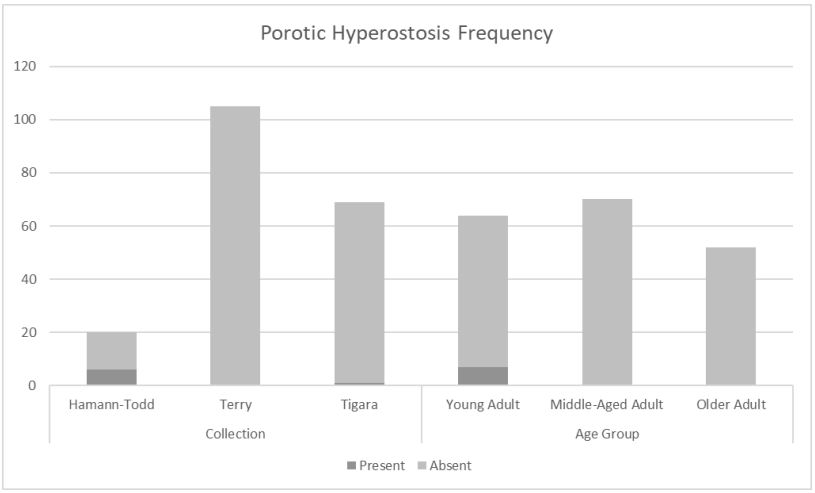

Figure 21: Bar graph illustrating number of individuals with PH by collection and age group

6.2.2 Cribra Orbitalia

The orbital roofs of nine Tigara individuals were unobservable due to taphonomic damage. CO was present in 17 out of 185 (9%) individuals. The frequency data for CO and the results of the Chi-Square Test of Independence ($p<0.05$) revealed no significant relationships between CO and collection, sex, ancestry or age group (Table 27).

Table 27: CO χ^2 test and frequency

Cohort	Test Statistic	Group	n	Frequency	Percentage
Collection	X^2= .910, p= .634	Hamann-Todd	20	1	5%
		Terry	105	9	9%
		Tigara	60	7	12%
Sex	X^2= .089, p= .766	Male	115	10	9%
		Female	70	7	10%
Ancestry	X^2= .677, p= .713	African American	78	6	8%
		European American	47	4	9%
		Native American	60	7	12%
Age Group[a]	X^2= 1.036, p= .596	Young Adult	63	7	11%
		Middle Adult	62	6	10%
		Old Adult	52	3	6%
Total			185	17	9%

[a]n=177

6.3 Infectious Diseases

6.3.1 Sinusitis

All of the sinusitis remodeling types described by Boocock and colleagues (1995) were present in the sample. Spicule-type bone formation was the most frequent (n=29) and white pitted bone was the least frequent (n=1) remodeling type (Figure 22).

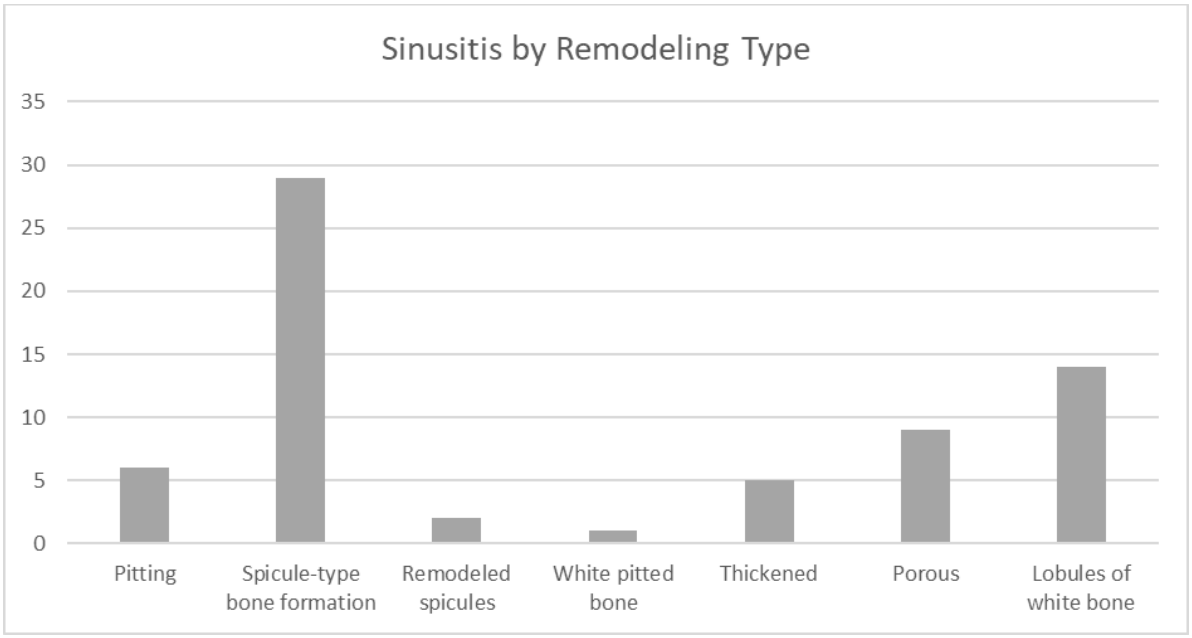

Figure 22: Bar graph illustrating number of individuals with sinusitis by remodeling type

The frequency data for sinusitis are reported in Table 28. A Chi-Square Test of Independence ($p<0.05$) followed by post-hoc analysis using a Bonferroni correction (Table 29) revealed the European American ancestry group had a significantly higher proportion of sinusitis than expected ($p<0.008$) (Figure 23).

Table 28: Sinusitis frequency

Cohort	Group	n	OM Frequency	Percentage
Collection	Hamann-Todd	20	0	0%
	Terry	105	19	18%
	Tigara	69	11	16%
Sex	Male	120	17	14%
	Female	74	13	18%
Ancestry	African American	78	6	8%
	European American	47	13	28%
	Native American	69	11	16%
Age Group[a]	Young Adult	64	8	13%
	Middle Adult	70	8	11%
	Old Adult	52	12	23%
Total		194	30	15%

[a]n=186

Table 29: Sinusitis χ^2 test and adjusted standardized residuals

Cohort	Test Statistic	Variable	Obs. Count	Exp. Count	Z-score	Bonferroni adjusted p-value
Collection	X^2= 4.227, p= .121					
Sex	X^2= .405, p= .505					
Ancestry	X^2= 8.963, **p= .011** v=. 215[a]	African American	6	12.1	-2.5	p > .008
		European American	13	7.3	2.7	**p < .008**
Age Group	X^2= 3.663, p= .160					

Statistically significant p-values indicated in bold
[a]Cramer's V

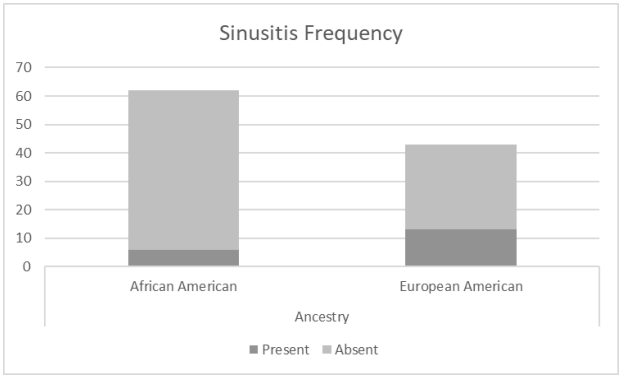

Figure 23: Bar graph illustrating number of individuals with sinusitis by ancestry cohort

6.3.2 Otitis Media

CT scans were only available for 186 individuals in the sample. The frequency data for OM are reported in Table 30. A Chi-Square Test of Independence ($p<0.05$) followed by post-hoc analysis using a Bonferroni correction revealed statistically significant ($p<0.008$) relationships between OM and collection, ancestry and age group (Table 31). The Hamann-Todd collection, African American ancestry group and young adult age group had a higher proportion of OM than expected and the Terry collection had a lower proportion than expected (Figure 24). The majority of OM was within the Hamann-Todd sample (64%), which was the smallest collection (7%), and the sample of Hamann-Todd used for the OM analysis was 100% African American and young adult which skewed the OM results within the age group and ancestry cohorts.

Table 30: OM frequency

Cohort	Group	n	Frequency	Percentage
Collection	Hamann-Todd	13	9	69%
	Terry	104	3	3%
	Tigara	69	2	3%
Sex	Male	114	8	7%
	Female	72	6	8%
Ancestry	African American	74	11	15%
	European American	43	1	2%
	Native American	69	2	3%
Age Group[a]	Young Adult	62	10	16%
	Middle Adult	66	3	5%
	Old Adult	50	1	2%
Total		186	14	8%

[a]n=178

Table 31: OM χ^2 test and adjusted standardized residuals

Cohort	Test Statistic	Variable	Obs. Count	Exp. Count	Z-score	Bonferroni adjusted p-value
Collection	X^2= 76.455, **p= .000** v= .641[a]	Hamann-Todd	9	1	8.7	**p < .008**
		Terry	3	7.8	-2.7	**p < .008**
Sex	X^2= .110, p= .740					
Ancestry	X^2= 9.520, **p= .009** v= .226[a]	African American	11	5.6	3.1	**p < .008**
Age Group	X^2= 8.096[b], **p= .014** v= 228[a]	Young Adult	10	4.9	3.0	**p < .008**

Statistically significant p-values indicated in bold
[a]Cramer's V
[b]Fischer's Exact Test

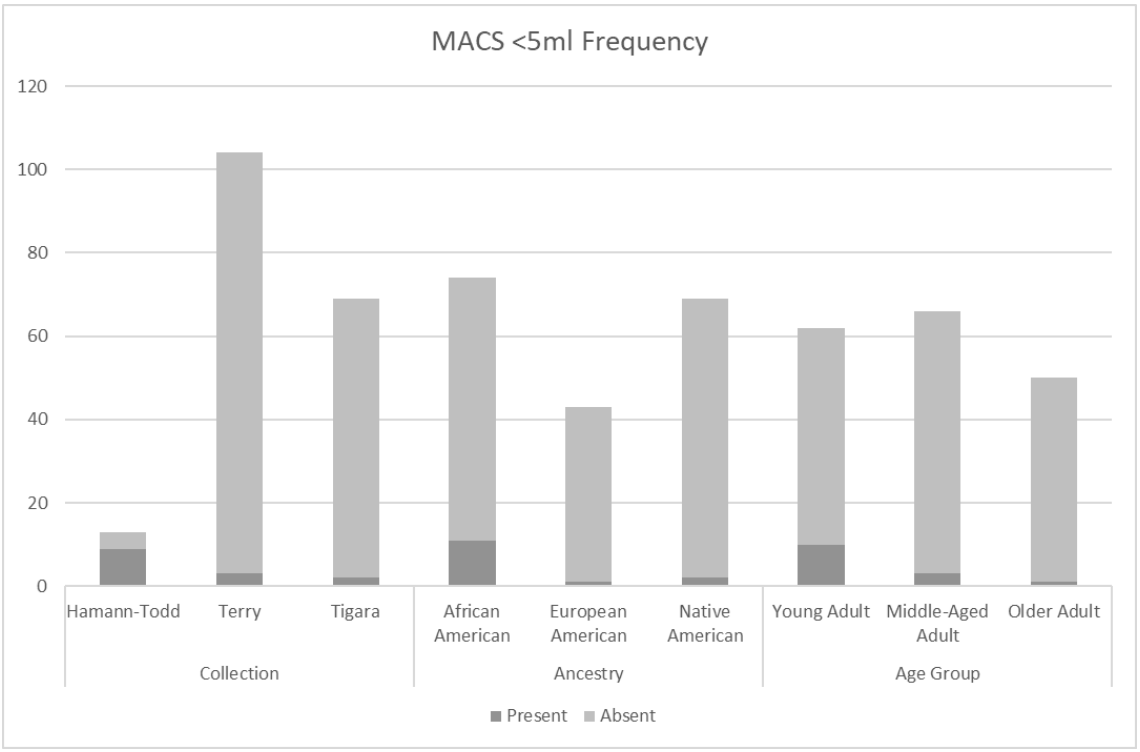

Figure 24: Bar graph illustrating number of individuals with OM by collection, ancestry and age group cohorts

6.4 Dental Pathology

The dental inventory is reported by tooth type is reported in Table 32 (Figure 25) and by individual tooth in Appendix B.1. The Hamann-Todd had the most teeth present (95%), then the Tigara (82%) and the Terry collection had the least (60%).

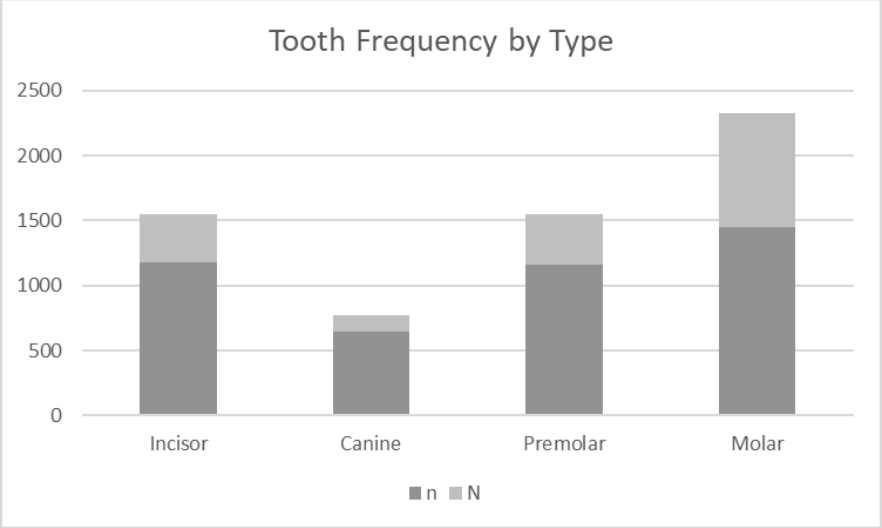

Figure 25: Tooth frequency by type

Table 32: Dental inventory by tooth type

			Incisor			Canine			Premolar			Molar			Total		
		I	N	n	%	N	n	%	N	n	%	N	n	%	N	n	%
Total		194	1552	1180	76%	776	645	83%	1552	1159	75%	2328	1451	62%	6208	4435	71%
Collection	Hamann-Todd	20	160	160	100%	80	79	99%	160	156	98%	240	216	90%	640	611	95%
	Terry	105	840	572	68%	420	314	75%	840	526	63%	1260	601	48%	3360	2013	60%
	Tigara	69	552	448	81%	276	252	91%	552	477	86%	828	634	77%	2208	1811	82%
Sex	Male	120	960	774	81%	480	420	88%	960	745	78%	1440	921	64%	3840	2860	74%
	Female	74	592	406	69%	296	225	76%	592	414	70%	888	530	60%	2368	1575	67%
Ancestry	African Am.	78	624	524	84%	312	269	86%	624	501	80%	936	634	68%	2496	1928	77%
	European Am.	47	376	208	55%	188	124	66%	376	181	48%	564	183	32%	1504	696	46%
	Native American	69	552	448	81%	276	252	91%	552	477	86%	828	634	77%	2208	1811	82%

I= total individuals, N= expected count, n= observed count, %= tooth frequency

6.4.1 Antemortem Tooth Loss

Seventy-five percent of the individuals had antemortem tooth loss (AMTL). By the 56-60 year age range all of the individuals had AMTL and by the 61-65 year age range some individuals were edentulous (Figure 26). Eleven individuals were edentulous, all from the Terry collection. Analyses for present/absent AMTL and mean number of AMTL per individual were performed.

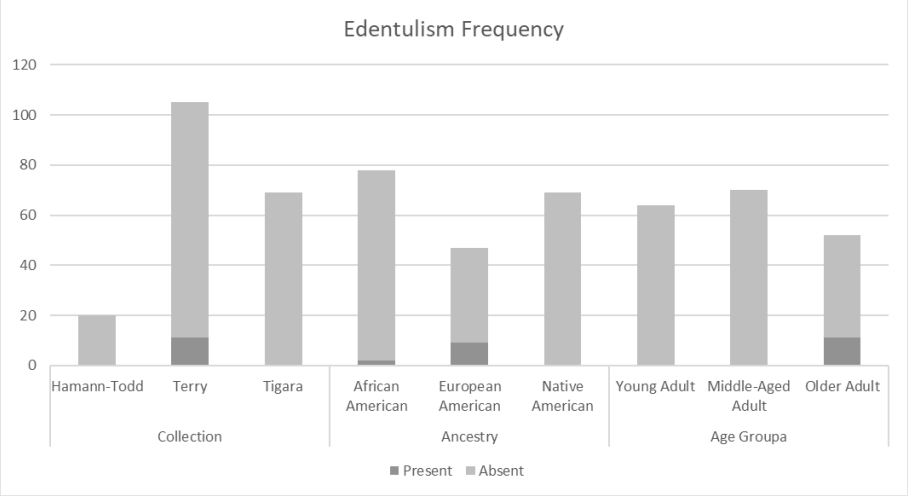

Figure 26: Bar graph illustrating number of individuals with edentulism by collection, ancestry and age group cohort

The frequency data for AMTL are reported in Table 33. A Chi-Square Test of Independence ($p<0.05$) followed by post-hoc analysis using a Bonferroni correction revealed statistically significant ($p<0.008$) relationships between AMTL and collection, ancestry and age group (Table 34, Figure 27). The Terry collection, European American ancestry group and older adult age group had a greater prevalence of AMTL than expected and the young adult age group had a lower proportion than expected.

Table 33: Individuals with AMTL frequency

Cohort	Group	n	Frequency	Percentage
Collection	Hamann-Todd	20	14	70%
	Terry	105	87	83%
	Tigara	69	45	65%
Sex	Male	120	88	73%
	Female	74	58	78%
Ancestry	African American	78	55	71%
	European American	47	46	98%
	Native American	69	45	65%
Age Group[a]	Young Adult	64	31	48%
	Middle Adult	70	57	81%
	Old Adult	52	52	100%
Total		194	146	75%

[a]n=186

Table 34: Individuals with AMTL χ^2 test and adjusted standardized residuals

Cohort	Test Statistic	Variable	Obs. Count	Exp. Count	Z-score	Bonferroni adjusted p-value
Collection	X^2= 7.289, **p= .026** v= .194[a]	Terry	87	79	2.7	**p < .008**
		Tigara	45	51.9	-2.4	p > .008
Sex	X^2= .626, p= .429					
Ancestry	X^2= 17.587, **p= .000** v= .301[a]	European American	46	35.4	4.1	**p < .008**
		Native American	45	51.9	-2.4	p > .008
Age Group	X^2= 43.264, **p= .000** v= .482[a]	Young Adult	31	48.2	-6.1	**p < .008**
		Old Adult	52	39.1	4.9	**p < .008**

Statistically significant p-values indicated in bold
[a]Cramer's V

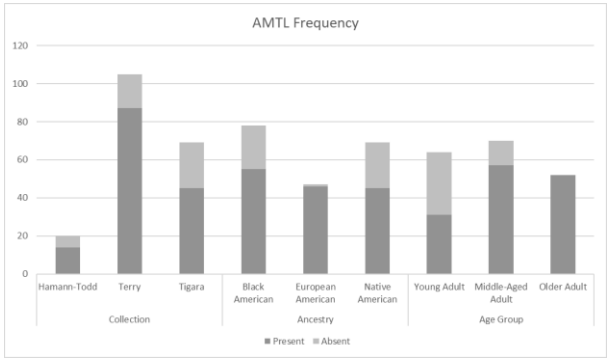

Figure 27: Bar graph illustrating number of individuals with AMTL by collection, ancestry and age group cohort

The Student's t-test and ANOVA with Tukey's HSD post-hoc analysis revealed that mean AMTL in the Terry collection (12.46 teeth) was significantly different than mean AMTL in the Hamann-Todd (1.45 teeth) and Tigara (5.62 teeth) collections (Table 35 and Figure 28). Within the ancestry cohort, European American (16.66 teeth) mean AMTL was significantly different than African American (7.10 teeth) and Native American (5.62 teeth), and within the age range cohort old adult (19.19 teeth) mean AMTL was significantly different than the young adult (2.53 teeth) and middle adult (6.94 teeth) age groups.

Table 35: Statistical analyses of cohorts and mean AMTL

Cohort	N	Group	Mean AMTL	Test statistic	p-value	Tukey's HSD
Collection	194	Hamann-Todd	1.45	F^a=55.391	**p= .000**	Hamann-Todd x Terry Terry x Tigara
		Terry	12.46			
		Tigara	5.62			
Sex	194	Male	7.92	t^b=-1.636	p= .104	
		Female	10.47			
Ancestry	194	African American	7.10	F^a=18.368	**p= .000**	African American x European American European American x Native American
		European American	16.66			
		Native American	5.62			
Age Group	186	Young Adult	2.53	F^a=54.920	**p= .000**	Young adult x old adult Middle x old adult
		Middle Adult	6.94			
		Old Adult	19.19			

Statistically significant p-values indicated in bold

[a]Homogeneity of variances violated (Levene statistic: $p<0.05$) and Welsh's Robust Test of Equality of Means reported

[b]Equal variances not assumed (Levene statistic: $p<.05$)

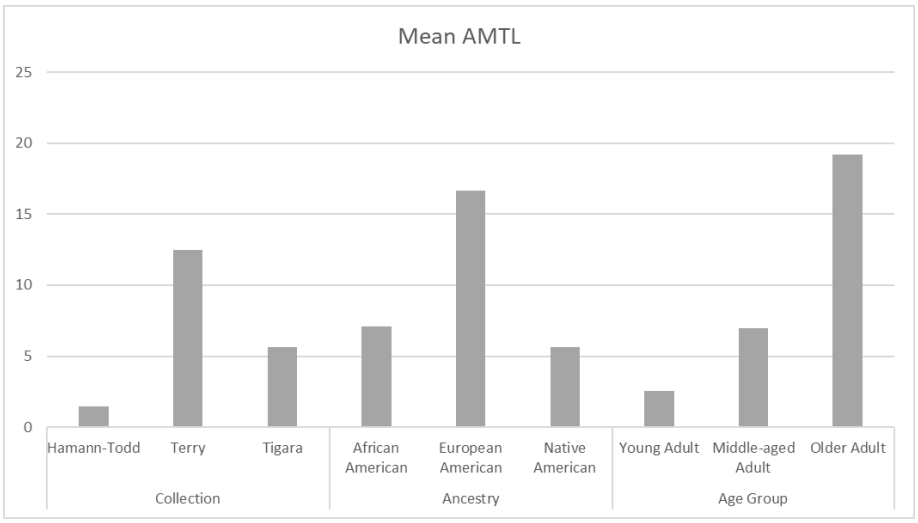

Figure 28: Bar graph illustrating mean number of teeth lost by individual by collection, ancestry and age group cohort

6.4.2 Granulomatous Periapical Lesion

Granulomatous periapical lesions were present in 31 individuals, with a maximum of five lesions in one individual (Figure 29).

Figure 29: Granulomatous periapical lesions visible on the left central incisor, right lateral incisor and right first premolar (Tigara#526)
Photograph courtesy of Division of Anthropology, American Museum of Natural History. Photograph by Allison P. Gremba.

A Chi-Square Test of Independence ($p<0.05$) revealed no significant relationships between granulomatous periapical lesions and collection, sex, ancestry or age group (Table 36).

Table 36: Granulomatous periapical lesion χ^2 test and frequency

Cohort	Test Statistic	Group	n	Frequency	Percentage
Collection	X^2= 1.517, p= .468	Hamann-Todd	20	3	15%
		Terry	105	14	13%
		Tigara	69	14	20%
Sex	X^2= .542, p= .462	Male	120	21	18%
		Female	74	10	14%
Ancestry	X^2= 1.974, p= .373	African American	78	12	15%
		European American	47	5	11%
		Native American	69	14	20%
Age Group[a]	X^2= .294, p= .863	Young Adult	64	10	16%
		Middle Adult	70	13	19%
		Old Adult	52	8	15%
Total			194	31	16%

[a]n=186

The results from the mean number of granulomatous periapical lesions using the statistical analyses, Student's t-test and ANOVA, are displayed in Table 37. There were no significant differences in mean number of lesions by collection, sex, ancestry or age group.

Table 37: Statistical analysis of mean granulomatous periapical lesion

Cohort	N	Group	Mean lesion	Test statistic	p-value	Tukey's HSD
Collection	194	Hamann-Todd	0.25	F^a=1.774	p= .181	
		Terry	0.15			
		Tigara	0.36			
Sex	194	Male	0.23	t=-.103	p= .918	
		Female	0.24			
Ancestry	194	African American	0.19	F^a=1.857	p= .161	
		European American	0.13			
		Native American	0.36			
Age Group	186	Young Adult	0.23	F=.413	p= .662	
		Middle Adult	0.30			
		Old Adult	0.25			

[a]Homogeneity of variances violated (Levene statistic: $p<0.05$) and Welsh's Robust Test of Equality of Means reported

6.4.3 Periodontal Disease

Periodontal disease was assessed in 157 individuals and present in 107 individuals. Periodontal disease was not scored on 37 individuals due to ante- and post-mortem tooth loss. Frequency data for periodontal disease and the results of the Chi-Square Test of Independence ($p<0.05$) revealed no significant relationships between periodontal disease and collection, sex, ancestry or age group (Table 38).

Table 38: Periodontal disease χ^2 test and frequency

Cohort	Test Statistic	Group	n	Frequency	Percentage
Collection	X^2= 1.251, p= .535	Hamann-Todd	20	13	65%
		Terry	73	53	73%
		Tigara	64	41	64%
Sex	X^2= .089, p= .765	Male	101	68	67%
		Female	56	39	70%
Ancestry	X^2= 4.997, p= .082	African American	66	51	77%
		European American	27	15	56%
		Native American	64	41	64%
Age Group[a]	X^2= 3.795[1], p= .150	Young Adult	61	36	59%
		Middle Adult	65	46	71%
		Old Adult	24	19	79%
Total			157	107	68%

[a]n=150

There were 50 individuals with a severity score of ‘1’, 65 individuals with a severity score of ‘2’, 21 individuals with a score of ‘3’ and 21 individuals with a score of ‘4’. The Chi-Square Test of Independence ($p<0.05$) for periodontal disease severity was not significant for the collection (X^2=6.484, p=.371), sex (X^2=1.512, p=.680), ancestry (X^2=9.617, p=.142), or age group (X^2=9.069, p=.170).

6.4.4 Dental Enamel Hypoplasia

Dental enamel hypoplasia (DEH) could not be analyzed for all teeth due to antemortem tooth loss (Figure 13), taphonomy (Figure 30) and wear (Figure 31). Canines had the highest percent of complete enamel present (51-59%) (Table 39). The European American group had the lowest percent of canines with complete enamel present (30-47% present) (Appendix B.2). Of the teeth with complete enamel, the maxillary incisors and canines had the highest percent of DEH, followed by the mandibular incisors. The canines and maxillary incisors were chosen for DEH analysis and the highest DEH score of these eight teeth was used for the individual.

Figure 30: Taphonomic tooth damage (Terry#826)
Photograph courtesy of Department of Anthropology, Smithsonian Institution. Photograph by Allison P. Gremba.

Figure 31: Tooth wear (Tigara#260)
Photograph courtesy of Division of Anthropology, American Museum of Natural History. Photograph by Allison P. Gremba.

Table 39: Percent of tooth type present and DEH

	% Teeth Present	% DEH on Teeth Present
Canine[a]	51-59%	46-63%
Maxillary Incisor[a]	36-40%	49-66%
Mandibular Incisor	41-48%	32-44%
Premolar 1	45-49%	22-27%
Premolar 2	38-44%	13-18%
Molar	34-41%	0-13%

[a]Teeth used for DEH analysis.

There were no significant relationships between DEH and collection, sex, ancestry or age group using the the Chi-Square Test of Independence ($p<0.05$) (Table 40). The Chi-Square Test of Independence ($p<0.05$) for DEH severity was not significant for the collection ($X^2=4.066$, $p=.669$), sex ($X^2=5.494$, $p=.139$), ancestry ($X^2=2.586$, $p=.878$) and age group ($X^2=2.358$, $p=.670$) cohorts.

Table 40: DEH by individual χ^2 test and frequency

Cohort	Test Statistic	Group	n	Frequency	Percentage
Collection	$X^2= .633$, $p= .729$	Hamann-Todd	20	14	70%
		Terry	81	62	77%
		Tigara	45	32	71%
Sex	$X^2= 2.510$, $p= .113$	Male	96	75	78%
		Female	50	33	66%
Ancestry	$X^2= .570$, $p= .752$	African American	73	56	77%
		European American	28	20	71%
		Native American	45	32	71%
Age Group[a]	$X^2= 1.173$, $p= .556$	Young Adult	58	45	78%
		Middle Adult	54	37	69%
		Old Adult	30	22	73%
Total			146	108	74%

[a]n=142

6.5 Otitis Media and Skeletal Stress Markers

Results from the statistical analyses for OM and traditional skeletal stress markers found less mean AMTL and greater prevalence of PH in individuals with OM (Table 41). The Student's t-test for mean AMTL was statistically significant ($p<0.05$) (no OM: 9.58, OM: 4.07). PH and OM was statistically significant using the Bonferroni correction (observed: 4, expected: 0.5, Z-score: 5.6, $p<0.013$). Six of the seven cases (86%) of PH and nine of the 14 cases (64%) of OM were present within the Hamann-Todd collection. Within the Hamann-Todd, four individuals had both PH and OM, which is 57% of the PH cases and 29% of the OM cases.

Table 41: Analysis of OM and skeletal stress markers

Pathology	Test value	p-value
Sinusitis	$X^2=0.904$[a]	p=0.474
Porotic hyperostosis	$X^2=31.54$[a]	**p=0.000**
Cribra orbitalia	$X^2=0.059$[a]	p=1.000
Sinusitis	$X^2=0.904$[a]	p=0.474
Present/absent AMTL	$X^2=0.776$[a]	p=0.358
Mean AMTL	t=2.776[b]	**p=0.012**
Present/absent granulomatous periapical lesion	$X^2=0.741$[a]	p=0.698
Mean granulomatous periapical lesion	t=0.917	p=0.361
Present/absent periodontal disease	$X^2=1.136$[a]	p=0.356
Periodontal disease severity	$X^2=4.576$[a]	p=0.171
Present/absent DEH	$X^2=0.358$[a]	p=0.511
DEH severity	$X^2=1.745$[a]	p=0.462

Statistically significant p-values indicated in bold
[a]Fischer's Exact Test
[b]Equal variances not assumed (Levene statistic: $p<.05$)

7.0 Discussion

Skeletal stress markers and MACS hypopneumatization were examined in the context of structural violence in the Hamann-Todd and Terry collections and chronic cold in the Tigara collection and comparative studies were used to interpret the results. The hypothesis that MACS hypopneumatization is a skeletal stress marker and its prevalence will be associated with other measured skeletal stress markers is discussed in this chapter.

7.1 Structural Violence

Structural violence is the systemic oppression of a group of individuals by a society in the form of disproportionate access to resources (Galtung 1969). The individuals within the Hamann-Todd and Terry collections were denied access to a proper burial and therefore experienced structural violence at the time of death (Muller et al. 2017; Nystrom 2014). These individuals were most likely indigent lacking the financial or social resources to prevent inclusion into these collections. They likely also faced hardship during their childhood and adolescence, but the historical circumstances of their lives before the time of their death is unknown. Traditional skeletal stress markers were expected to be elevated in the Hamman-Todd and Terry collections. MACS hypopneumatization, as a measure of OM, was also expected to be elevated and captured baseline levels of OM as well as increased prevalence in childhood and adulthood OM resulting from low socioeconomic status, marginalization and structural violence.

The results of this dissertation analysis are discussed in the context of the following comparative studies which include the same skeletal stress markers (Table 42). This summary only includes the skeletal stress markers that were used in this dissertation analysis and is not inclusive of all of the skeletal stress markers or pathologies that were examined in the comparative studies. De la Cova (2008) and Coolidge (2015) examined the Hamann-Todd and Terry collections, and Atwell (2017) and Gengo (2014) examined the Terry collection. De la Cova's (2008) dissertation contextualized the sociocultural and environmental components of the individuals in the Hamann-Todd and Terry collections using a biocultural framework and historical sources; PH/CO, AMTL, dental abscess and DEH were included in her analysis. Coolidge (2015) examined the developmental origins of health and disease hypothesis to determine if early life conditions affected health later in life and included DEH in her analysis. Atwell (2017) examined institutionalized women from the Terry collection to explore how structural violence was embodied and recorded PH/CO and dental abscess. Gengo (2014) studied the relationship between co-occurring pathologies within a framework of structural violence and recorded DEH and periodontal disease.

Table 42: Structural violence comparative studies

Author	Collection	Time Period	Region
De la Cova (2008)	Terry	19th-20th c.	St. Louis, Missouri
	Hamann-Todd	19th-20th c.	Cleveland, Ohio
Coolidge (2015)	Terry	19th-20th c.	St. Louis, Missouri
	Hamann-Todd	19th-20th c.	Cleveland, Ohio
Atwell (2017)	Terry institutionalized women	19th-20th c.	St. Louis, Missouri
Gengo (2014)	Terry	19th-20th c.	St. Louis, Missouri
DiGangi and Sirianni (2017)	Monroe County Almshouse	19th c.	Rochester, New York
Grauer and colleagues (1998)	Cook County Poor Farm (i.e., Dunning Poorhouse)	19th c.	Chicago, Illinois
Null and colleagues (2004)	African Burial Ground	18th c.	New York, New York
Rathbun (1987)	African Americans from a South Carolina Plantation	19th c.	Charleston, South Carolina

Other skeletal collections that experienced structural violence and marginalization in the 19th and 20th centuries were examined. DiGangi and Sirianni (2017) recorded maxillary sinusitis in the individuals from the Monroe County Almshouse cemetery (1826-1853) which was established to treat poverty through hard work. Grauer and colleagues (1998) explored the relationship between gender and poverty in individuals from the Cook County Poor Farm cemetery (also known as the Dunning Poorhouse) (1851-1869), and examined PH/CO and DEH. Null and colleagues (2004) examined nutrition and infection in slaves from the New York African Burial Ground (1697-1794), and recorded PH and CO. Rathbun (1987) examined gender, anemia, physical labor and trace elements in African Americans from a 19th century plantation cemetery in South Carolina (1840-1870) and recorded PH, CO and DEH. The results from these studies were used to interpret the results from this dissertation analysis.

Skeletal stress markers of nutritional stress, infectious disease and dental pathology were recorded to assess structural violence and marginalization in the Hamann-Todd and Terry collections. Nutritional stress was measured by the presence of PH and CO, and de la Cova (2008) and Atwell (2017) combined their PH and CO (PH/CO) results. De la Cova (2008) found that PH/CO was present in 87.6% (n=178) of the Hamann-Todd individuals and 84.6% (n=357) of the Terry individuals (Table 43). She attributed these high prevalence rates to diet and parasitic infections. Atwell (2017), who examined only institutionalized women in the Terry collection, found PH/CO in 50.94% (n=53) of the individuals. Grauer and colleagues (1998) reported PH in 35% (n=49) of the Cook County Poor Farm individuals. Null and colleagues (2004) and Rathbun (1987) reported PH in African Americans and found PH in 50.5% (n=184) and 30% (n=23) of the individuals, respectively. Rathbun (1987) attributed this to both iron deficiency and sickle cell anemia, which has a higher prevalence in African Americans compared to European Americans.

Table 43: Summary of skeletal stress marker prevalence (n) from this dissertation and structural violence comparative studies

Author	Collection	PH	CO	Sinusitis	OM	AMTL	Periodontal Disease	Granulomatous Periapical Lesions	DEH
This dissertation	**Terry**	**0% (105)**	**9% (105)**	**18% (105)**	**3% (104)**	**83% (105)**	**73% (73)**	**13% (105)**	**77% (81)**
	Hamann-Todd	**30% (20)**	**5% (20)**	**0% (20)**	**69% (13)**	**70% (20)**	**65% (20)**	**15% (20)**	**70% (20)**
De la Cova (2008)	Terry	84.6% (357)		--	--	99.2% (367)	--	51.9% (366)	54% (202)
	Hamann-Todd	87.6% (178)		--	--	98.4% (182)	--	63.2% (182)	63.2% (117)
Coolidge (2015)	Terry	--	--	--	--	--	--	--	80% (475)
	Hamann-Todd	--	--	--	--	--	--	--	69.8% (298)
Atwell (2017)	Terry women	50.94% (53)		--	--	--	--	18.87% (28)	--
Gengo (2014)	Terry	--	--	--	--	--	70% (197)	--	58% (175)
DiGangi and Sirianni (2017)	Almshouse	--	--	60% (90)	--	--	--	--	--
Grauer and colleagues (1998)	Poor Farm	35% (49)	--	--	--	--	75% (51)	--	37% (49)
Null and colleagues (2004)	African Burial Ground	50.5% (184)	22% (164)	--	--	--	--	--	--
Rathbun (1987)	South Carolina Plantation	30% (23)	35% (28)	--	--	--	--	--	85% (27)

The unsanitary living conditions and inadequate medical care experienced by the individuals in the Hamann-Todd and Terry collections were expected to increase the risk of anemia from diet, diarrheal disease and parasites resulting in an increased prevalence of PH and CO (Cleveland Hospital Council 2017; Primm 1998). This study found 30% (n=20) of the Hamann-Todd individuals had PH while de la Cova (2008) reported 87.6% of the individuals in her sample had PH/CO; an over-reporting of PH by de la Cova (2008) may have resulted from combining PH and CO, and an absence of a differential diagnosis for ectocranial lesions. Within the comparative studies the range of individuals that had combined PH/CO was 50.4% to 87.6% (Atwell 2017; de la Cova 2008), and the range of individuals with PH, when PH and CO were recorded separately, was 30% to 50.5% (Grauer et al. 1998; Null et al. 2004; Rathbun 1987). The results of the Hamann-Todd collection (30%) from this dissertation analysis fall within the range reported by other researchers.

Within the Terry collection the prevalence of PH was 0% (n=105) and was significantly less than the Hamann-Todd collection (30%, n=20). This was also much lower than reported in similar studies; de la Cova (2008) reported 87.6% and Atwell (2017) reported 50.4% of the Terry individuals had PH/CO. The Terry and Tigara samples for this analysis were selected based on available CT scans from Copes' (2012) dissertation on cranial robusticity which excluded specimens with scurvy, rickets, treponemal disease, Paget's disease, osteomyelitis and severe osteoarthritis. Although PH was not specifically mentioned as an exclusionary criteria it is likely Copes (2012) did not perform an in depth differential diagnosis on cranial vault lesions and eliminated all skulls with cranial vault pathology which would explain the absence of PH in the Terry collection.

The prevalence of CO in the Hamann-Todd collection was 5% (n=20) and the Terry collection was 9% (n=105) and was much lower than other marginalized samples. Null and colleagues (2004) found CO in 22% (n=164) of the New York African Burial Ground collection, and Rathbun (1987) found CO in 35% (n=28) of the 19th century African Americans from a South Carolina plantation. The PH and CO results suggest there was less nutritional stress during childhood in the Hamann-Todd and Terry collection individuals than in the comparative studies, which is a reasonable expectation because the childhood status of the Hamman-Todd and Terry collection individuals is unknown and therefore their risk of anemia may have been mitigated by cultural and other unknown factors.

Sinusitis and OM were measured as markers of infectious disease. The air pollution from bitumous coal, the steam engine and industry, as well as poorly ventilated living spaces was expected to increase sinusitis risk in the Hamann-Todd and Terry collections (Cleveland Hospital Council 2017; Primm 1998; Schroeder 1997; Tarr and Zimring 1997). The prevalence of sinusitis was 0% (n=20) in the Hamann-Todd and 18% (n=105) in the Terry collections. This is lower than expected based on the results from DiGangi and Sirianni (2017) on the Monroe County Almshouse where they found sinusitis in 60% (n=90) of the sample. They attributed this to poor sanitation, hygiene and pollution associated with communal living. The difference between DiGangi and Sirianni (2017) and this analysis may be the result of different methodological approaches for assessing sinusitis. In this analysis access was limited to the mastoid antrum and limited access made visualization of the sinus difficult and incomplete. DiGangi and Sirianni (2017) drilled a 1cm hole into the maxilla to access the mastoid sinus which allowed for consistent visualization and an unimpeded view. DiGangi and Sirianni (2017) likely reported the true prevalence of sinusitis in the Monroe County Almshouse, while sinusitis was likely underreported in this analysis

Crowded unsanitary living conditions and inadequate access to medical care were expected to increase pathogen exposure and transmission increasing OM risk and MACS hypopneumatization prevalence in the Hamann-Todd and Terry collections (Cleveland Hospital Council 2017; Primm 1998). The prevalence rate of OM, defined as MACS <5ml, was 69% (n=13) in the Hamann-Todd collection and statistically higher than expected, and was 3% (n=104) in the Terry collection and lower than expected. OM has not been measured in the Hamann-Todd, Terry or other marginalized collections from a similar time period so there is no comparative data. The significant difference in OM prevalence between the Hamann-Todd and Terry collections is discussed in Section 7.3.

Dental pathology was assessed by AMTL, periapical granulomatous lesions, periodontal disease and DEH. AMTL was expected to be elevated in the Hamann-Todd and Terry collections due to inadequate medical care and late-life stress as evidenced by inclusion in the collections (Cleveland Hospital Council 2017; Muller et al. 2017; Nystrom 2014; Primm 1998). AMTL was present in 70% (n=20) of the Hamann-Todd individuals and 83% (n=105) of the Terry individuals. This was lower than de la Cova (2008) reported; 98.4% (n=182) of the Hamann-Todd individuals and 99.2% (n=367) of the Terry individuals had AMTL. Similarly, Atwell (2017) examined institutionalized women from the Terry collection and found a higher prevalence rate for edentulism (47.17%, n=28) than this study (10%, n=105). In this analysis AMTL was diagnosed only if the socket was completely resorbed and only if there was evidence of alveolar remodeling in the third molar to eliminate third molar agenesis. De la Cova (2008) and Atwell (2017) do not describe their methods for AMTL diagnosis and therefore may have been less stringent in their diagnoses resulting in a higher reported prevalence of AMTL.

Periapical granulomatous lesions and periodontal disease were expected to be elevated in the Hamann-Todd and Terry collections due to inadequate medical care and unsanitary living conditions (Cleveland Hospital Council 2017; Primm 1998). Presence of at least one periapical granulomatous lesion was in 15% (n=20) of the Hamman-Todd individuals and 13% (n=105) of the Terry individuals. This was lower than De la Cova (2008) reported; 63.2% (n=182) of the Hamann-Todd individuals and 51.9% (n=366) of the Terry individuals had periapical granulomatous lesions. Atwell (2017) found prevalence rates more similar to this study in the Terry collection with 18.87% (n=28) of institutionalized women having at least one dental abscess. The prevalence of periodontal disease was 65% (n=20) of the Hamann-Todd individuals and 73% (n=73) of the Terry individuals. This was within the ranges found in other studies. Gengo (2014) found periodontal disease in 70% (n=197) of the Terry individuals and Grauer and colleagues (1998) found periodontal disease in 75% (n=51) in the Cook County Poor Farm individuals.

DEH on the canines or maxillary incisors was found in 70% (n=20) of the Hamann-Todd individuals and 77% (n=81) of the Terry individuals. The Hamann-Todd and Terry results are within the range found by other studies. De la Cova (2008) recorded at least one DEH on the anterior teeth of 63.2% (n=117) of the Hamann-Todd individuals and 54% (n=202) of the Terry individuals. Coolidge (2015) found 69.8% (n=298) of the Hamann-Todd individuals and 80.0% (n=475) of the Terry individuals had DEH on at least one canine. Gengo (2014) recorded DEH in 58% (n=175) of the Terry individuals. Within other marginalized collection Rathbun (1987) found 85% (n=27) of 19th c. African American individuals from a South Carolina Plantation had DEH on at least one tooth, and Grauer and colleagues (1998) found 37% (n=49) of the Cook County Poor Farm individuals had DEH on a canine. The DEH results found in this study were within the

ranges reported by the comparative studies which was surprising because of the lower prevalence of other childhood skeletal stress markers (PH and CO) and unknown childhood status.

The prevalence of skeletal stress markers in the Hamann-Todd and Terry collections are within expected ranges based on comparative studies of structural violence and marginalization. There was evidence of all of the measured skeletal stress makers except for PH and OM in the Terry collection, and this was most likely the result of sample bias and will be discussed in Section 7.3. In the instances where the prevalence rates were lower than what was reported in the comparative studies it was most often due to methodological differences (e.g., AMTL and sinusitis). Within the Hamman-Todd and Terry collections there were high prevalence rates of age-related skeletal stress markers (sinusitis (Verbrugge 1984), periodontal disease (Hillson 1996), periapical granulomatous lesion (Stockdale and Chandler 1988) and AMTL (Al-Shammari et al. 2005))[4] supporting the notion that these individuals experienced stress prior to their death. However, they also had childhood skeletal stress markers (PH (Ponec and Resnick 1983) and DEH (Trammel and Kroman 2012)) indicating their status as an adult may have been an extension of their childhood status (e.g., low socioeconomic stress and marginalization).

Chronic OM during childhood affects hearing and can cause language delays resulting in impairment and disability that could place an individual at a disadvantage resulting in low socioeconomic status and eventual inclusion in the Hamann-Todd and Terry collections. Long term consequences of OM (bilateral OM with effusion at five years of age) were examined in a 26 year longitudinal study (n=1019) of individuals born between 1972 and 1973 in Dunedin New Zealand (Bennett et al. 2001; Silva et al. 1986; Welch and Dawes 2007). Results were reported at

[4] The prevalence of sinusitis and AMTL were statistically higher in the older age group.

11, 18 and 26 years of age. Silva and colleagues (1986) reported on academic and behavioral differences between OM and control children from ages 5 to 11. They found that hearing loss, language development, speech, reading, and behavioral problems (teacher and parent reported) were significantly worse in the OM group but the difference between groups decreased over time. Bennett and colleagues (2001) reported on the same group from ages 11 to 18. The parent reported behavioral problems were only available up until age 15 and inattentive and antisocial/aggressive behavior increased until 15 years of age. The teacher reported behavioral problems were only available until 13 years of age and the same variables were significant with the addition of neurotic behavior. All of the academic measures (verbal IQ, nonverbal IQ, full IQ, reading and spelling) worsened until 13 years of age. The only outcome measured until 18 years of age, reading, was significant. These poor academic and behavioral outcomes have also been reported in other studies (e.g., Bowd 2005, Gottlieb 1988, Holm and Kunze 1969, Klein 2001, Luotonen et al. 1998, Luotonen et al. 1996, Middle Ear Assessment 1990, Mortensen 2013, Teele 1984, Williams and Jacobs 2009 and Zinkus et al. 1978).

Welch and Dawes (2007) studied the same group at 26 years of age and found no association between OM severity in the psychological (personality, mental health, antisocial and criminal behavior and personal health) and socioeconomic measures (socioeconomic status, employment status and educational outcomes). They attributed the apparent disappearance of differences between the OM and control groups to a difference in the sensitivity of the variables measured. The real-world variables are able to be buffered by an individual's behavior whereas the previous measures used finely tuned scales that were calibrated for picking up minute differences in the outcome variables. Welch and Dawes (2007) suspected that differences still existed but in the real-life measures there was more flexibility to adapt and succeed. They

concluded that there are no negative adult outcomes from treated childhood OM. No other studies have reported long term consequences of childhood OM on adult psychological and socioeconomic status.

However, the absence of negative outcomes in adulthood may not be applicable to the Hamman-Todd and Terry individuals because treatment options and health outcomes in the late 19th and early 20th centuries were very different than the Welch and Dawes (2007) treatment outcomes of the 1970s. In the 19th century public opinion was ear drainage was beneficial and prevented brain infection therefore treatment was often not pursued (Mudry 2013). While OM was a recognized disease by clinicians, treatment was often not sought or unsuccessful. Researchers in the 18th and 19th centuries observed resolution of OM symptoms in patients with tympanic membrane destruction (Cooper 1800). This observation led Cooper (1801) to start performing myringotomies by creating a small incision in the tympanic membrane and he found an improvement in OM symptoms in patients. By the 1860s myringotomy was a popular treatment for OM, but due to quick healing times of the tympanic membrane and the high infection rates, by the early 20th century surgical measures (e.g., adenoidectomy and mastoidectomy) replaced myringotomy (Alberti 1974; Bento and de Oliveira Fonseca 2013). Myringotomy and tympanostomy tubes, which were first invented in the mid-19th century, were not widely used again for treatment of OM until the mid-20th century when antibiotics and the surgical microscope became available (Alberti 1974). Although they remain the standard treatment today early applications of these techniques were unsuccessful. Therefore the absence of the "real-life" long term outcomes measured by Welch and Dawes (2007) in individuals *treated* for OM may not be synonymous with the outcomes of the possibly untreated or unsuccessfully treated Hamann-Todd and Terry collection individuals.

Poor health outcomes and measurable impairment in hearing, language and learning resulting from childhood OM may result in disability. Disability occurs when an impairment prevents an individual from fulfilling expected roles and can result in marginalization (Matczak and Kozłowski 2017). Chronic OM that would result in MACS hypopneumatization is more likely to result in impairments in hearing, language and learning, which in turn may be perceived as a disability in the past. Although the perception of impairment as a disability is unknowable and caution should be used, it is reasonable to assume that at least some individuals in the past may have experienced significant impairment from OM which would have been considered a disability and resulted in an increased risk for lower socioeconomic status, marginalization and incorporation into the Hamann-Todd or Terry collections (Dettwyler 1991).

7.2 Chronic Cold

The Tigara population was expected to have minimal expression of OM resulting from immunosuppression following cold stress. The traditional skeletal stress markers were expected to be present in the Tigara, except PH and CO, because the Tigara had an iron rich diet. Costa (1980), Costa (1982), Dabbs (2011), and Giardini and Eggers (2005) compared the marine-hunting Tigara (900-1700AD) to the terrestrial-hunting Ipiutak (100BC-500AD) from Point Hope, Alaska. Costa (1980, 1982) recorded AMTL and periodontal disease; Dabbs (2011) recorded PH, CO and DEH; and Giardini and Eggers (2005) recorded PH, CO, AMTL and dental abscess. Schwartz and colleagues (1995) examined tooth drilling in the Tigara collection and recorded dental abscess. The results of the Tigara analysis are discussed in the context of these comparative studies (Table 44).

Table 44: Cold adaptation comparative studies

Author	Population	Time Period	Region
Costa (1980)	Tigara	AD 900-1700	Point Hope, Alaska
	Ipiutak	100 BC-AD 500	Point Hope, Alaska
Costa (1982)	Tigara	AD 900-1700	Point Hope, Alaska
	Ipiutak	100 BC-AD 500	Point Hope, Alaska
Dabbs (2011)	Tigara	AD 900-1700	Point Hope, Alaska
	Ipiutak	100 BC-AD 500	Point Hope, Alaska
Giardini and Eggers (2005)	Tigara	AD 900-1700	Point Hope, Alaska
	Ipiutak	100 BC-AD 500	Point Hope, Alaska
Schwartz and colleagues (1995)	Tigara	AD 900-1700	Point Hope, Alaska
Guatelli-Steinberg and colleagues (2004)	Point Hope Inuits	500 BC-present	Point Hope, Alaska
Homøe and colleagues (1996)	Precontact Inuit	Before AD 1884	Greenland
Keenleyside (1998)	Eskimo (Kugusugaruk, Kugok, Piginik and Tigara)	AD 500-1850	Point Barrow and Point Hope, Alaska
	Aleut	1000 BC-AD 1500	Umnak Island, Alaska

Guatelli-Steinberg and colleagues (2004), Homøe and colleagues (1996) and Keenleyside (1998) measured skeletal stress markers in other cold adapted populations. Guatelli-Steinberg and colleagues (2004) recorded DEH in Inuit populations from Point Hope, Alaska (500BC-present). Homøe and colleagues (1996) recorded OM in precontact Greenland Inuit. Keenleyside (1998) recorded PH, CO, AMTL and dental abscess in four precontact Eskimo populations, the Kugusugaruk, Kugok, and Piginik from Point Barrow and the Tigara from Point Hope, Alaska, and one precontact Aleut population from Umnak Island.

Nutritional stress was measured as PH and CO and was not expected to be elevated in the Tigara population due to their iron-rich marine diet. The prevalence of PH in the Tigara population was 1% (n=69), which was similar to what was recorded by Dabbs (2011) and Giardini and Eggers (2005), 1.8% (n=277) and 3.2% (n=31), respectively (Table 45). In the terrestrial-hunting Ipiutak the prevalence of PH recorded by Dabbs (2011) was 3% (n=67) and by Giardini and Eggers (2005) was 20.10% (n=10). Keenleyside (1998) examined PH in four precontact Eskimo populations from Point Barrow and Point Hope, Alaska and found PH in 8% (n=118) of the individuals. She also examined one precontact Aleut population from Unimak Island and found PH in 4% (n=54) of the individuals. Low prevalence rates of PH were expected within precontact Eskimo and Aleut populations because their high protein diet is iron rich, and the few cases of PH were attributed to parasitic, bacterial and viral infections (Keenleyside 1998). The prevalence range of PH in the Tigara collection was 1.8% to 3.2% but increases to 20.01% in other cold adapted populations. Although the Tigara had a similar prevalence rate of PH compared to the comparative studies, this is likely an underrepresentation of PH prevalence due to the selection bias using the Copes (2012) sample that was discussed in Section 7.1.

Table 45: Summary of skeletal stress marker prevalence (n) from this dissertation and chronic cold comparative studies

Author	Population	PH	CO	Sinusitis	OM	AMTL	Periodontal disease	Granulomatous Periapical Lesions	DEH
This dissertation	**Tigara**	**1% (69)**	**12% (60)**	**16% (69)**	**3% (69)**	**65% (69)**	**64% (64)**	**20% (69)**	**71% (45)**
Dabbs (2011)	Tigara	1.8% (277)	30.5% (269)	--	--	--	--	--	31.1% (151)
	Ipiutak	3% (67)	40% (60)	--	--	--	--	--	12.1% (66)
Giardini and Eggers (2005)	Tigara	3.22% (31)	13.04% (46)	--	--	57.14% (63)	--	61.90% (63)	--
	Ipiutak	20.10% (10)	22.22% (27)	--	--	67.56% (37)	--	62.60% (37)	--
Schwartz and colleagues (1995)	Tigara	--	--	--	--	52% (48)	69% (48)	--	--
Guatelli-Steinberg and colleagues (2004)	Point Hope Inuits	--	--	--	--	--	--	--	38.1% (21)
Homøe and colleagues (1996)	Precontact Inuit	--	--	--	4.7% (127)	--	--	--	--
Keenleyside (1998)	Eskimo	8% (118)	4% (103)	--	--	65% (118)	43% (114)	57% (118)	29% (69)
	Aleut	4% (54)	0% (52)	--	--	74% (57)	47% (57)	67% (57)	3% (38)

CO was also expected to be present in low percentages and was found to be present in 12% (n=60) of the Tigara. Dabbs (2011) reported CO in 30.5% (n=269) of the Tigara individuals, and Giardini and Eggers (2005) reported CO in 13.04% (n=46). Giardini and Eggers (2005) attributed CO to parasitic infections contracted from marine animals and a lower host resistance due to chronic cold stress. In the Ipiutak Dabbs (2011) reported CO in 40% (n=60) and Giardini and Eggers (2005) reported CO in 22.22% (27). Keenleyside (1998) found CO in 4% (n=103) of four Eskimo populations from Point Barrow and Point Hope, Alaska, and in 0% (n=52) of Aleut from Unimak Island. The Tigara prevalence was within the range found by other researchers.

If CO was the result of iron deficiency in the Tigara collection, then PH would also be present. The prevalence rate of CO may be the result of vitamin C deficiency. The Tigara consume a low carbohydrate ketogenic diet which results in the use ketone bodies, instead of carbohydrates, for energy metabolism (Westman et al. 2003). There are several adverse effects of a ketogenic diet (e.g., kidney stones, amenorrhea and hypercholesterolemia) including water-soluble vitamin deficiencies (e.g., vitamin C) (Ballaban-Gil et al. 1998; Edelstein and Chisholm 1996). Vitamin C deficiency is associated with periodontal disease and orbital roof hematoma, which can mimic cribra orbitalia, and both periodontal disease and CO were present in the Tigara (Brickley and Ives 2006; Pussinen et al. 2003). Vitamin C rich food sources available to the Tigara were raw ringed seal liver (35mg/100g), cooked ring seal liver (13.6mg/100g), raw beluga whale epidermis (38mg/100g), raw licorice root (21mg/100g) and raw mountain sorrel (36mg/100g) (Geraci and Smith 1979). However, these foods, with the exception of the ringed seal, were limited and available only during certain seasons (Geraci and Smith 1979). Geraci and Smith (1979) measured vitamin C intake in three Inuit families from a seal hunting camp in the Northwest Territories of Canada for six weeks during the summer and concluded that they received on average 30mg of

vitamin C per day which was sufficient to ward off scurvy. They calculated presumed vitamin C consumption for the winter months and estimated it would have been about the same as the summer and that vitamin C deficiency was not a major health concern. However seasonal and yearly food instability could have created limited periods of vitamin C deficiency which would be visible in a segment of the population. Although sexual division of labor was present in the Tigara there was no evidence of sex-related differences in diet and access to vitamin C[5].

In this analysis PH prevalence was highest in the Hamann-Todd collection and nearly non-existent in the Terry and Tigara collections, whereas CO prevalence was lowest in the Hamann-Todd collection and highest in the Tigara collection (Table 46). While anemia is the established cause of PH, CO has multiple etiologies. The most likely cause of the significantly greater prevalence Tigara CO prevalence compared to the Hamann-Todd and Terry collections is vitamin C deficiency which causes orbital roof hematoma in the absence of PH (McIlvaine 2015). The inverse prevalence rates of PH and CO in this analysis support the conclusion by Cole and Waldron (2019) that PH and CO result from different etiologies and pathological processes. PH and CO are not synonymous and should be recorded and analyzed separately (Cole and Waldron 2019). Although severe anemia can result in CO, CO is more likely the result of trauma, childhood disease, vitamin C deficiency or natural variation (Cole and Waldron 2019).

Table 46: Prevalence of PH and CO by collection

Collection	PH prevalence	CO prevalence
Hamann-Todd	30%	5%
Terry	0%	9%
Tigara	1%	12%

[5] CO prevalence in Tigara males was 9% (n=35) and in Tigara females was 12% (n=34).

Sinusitis and OM were recorded as measures of infectious disease. Sinusitis was expected to be present in the Tigara due to the use of indoor fires. Sinusitis was present in 16% (n=69) of the Tigara. There were no comparative studies for sinusitis.

OM was expected to be present in the Tigara due to immunosuppression resulting from cold stress. The prevalence rate of OM, defined as MACS <5ml, in the Tigara was 3% (n=69) (Table 47). Homøe and colleagues (1996) compared MACS hypopneumatization in pre- and post-contact Greenland Inuit archaeological remains to living Greenland Inuit with known OM and found an increase in OM prevalence over time. The precontact Inuit had no European genetic or cultural contribution and the OM prevalence was 4.7% (n=127). The post-contact Inuit were from the 18th and 19th centuries and had minor European genetic contributions, but major cultural contributions, including pathogen exposure, and the OM prevalence was 17.9% (n=56). The prevalence of OM in the living Inuit was 23.5% (n=34). The living group had a known history of OM and MACS pneumatization from X-rays had a positive OM predictive value of 75% (sensitivity: 67%, specificity: 92%). Although the Greenland Inuit experienced a restrained form of European colonialization and did not experience loss of land or culture, Bjerregaard and Larsen (2016) demonstrated that even subtle less obvious forms of marginalization following European colonization affected the mental health of the post-contact Greenland Inuit (e.g., alcoholism and suicide).

Table 47: OM prevalence (n) in pre- and post- European contact cold adapted poplations

Population	OM Prevalence
This dissertation precontact Tigara	3% (69)
Homøe and colleagues (1996) precontact Greenland Inuit	4.7% (127)
Homøe and colleagues (1996) post-contact Greenland Inuit	17.9% (56)
Homøe and colleagues (1996) living Greenland Inuit	23.5% (34)
Vasilyev and Borutskaya (2017) marginalized Sami	24.3% (125)

Vasilyev and Borutskaya (2017) examined OM in Sami individuals from 19th and 20th century Russia. During this time the Sami were undergoing major changes and hardship as their way of life as reindeer herders was being threatened by forced relocation (Lantto 2014). OM prevalence in the Sami was 24.3% (n=125). The high prevalence of OM observed in the Sami population can be attributed to marginalization and structural violence they experienced at the time and exposure to novel pathogens. However, Vasilyev and Borutskaya (2017) did not describe their methods for recording OM so it is unclear what these results are reporting. These results demonstrate that precontact cold adapted populations had a low prevalence of OM that increased following European contact, novel pathogen exposure and marginalization. The low prevalence of OM found in the Tigara collection was expected.

Dental pathology was assessed by AMTL, periapical granulomatous lesions, periodontal disease and DEH. The high grit Tigara diet was expected to expose dentine leading to a high prevalence of AMTL and periapical granulomatous lesions. The prevalence of AMTL in the Tigara collection was 65% (n=69). Schwartz and colleagues (1995) found AMTL in 52% (n=48) of the Tigara individuals, while Giardini and Eggers (2005) found AMTL in 57% (n=63) of the Tigara. Giardini and Eggers (2005) recorded AMTL in 67% (n=37) of the Ipiutak from Point Hope. Keenleyside (1998) observed AMTL in 65% (n=118) of four Eskimo populations from Alaska, and in 74% (n=57) of Aleut. AMTL was attributed to attrition resulting from a high grit diet (Costa 1980; Giardini and Eggers 2005). Costa (1980) identified sex-related differences in Tigara AMTL. Females had incisor tooth loss in the 26 to 30 year age group and molar tooth loss in the 36 to 40 year age group, whereas males had increasing AMTL after 30 years of age. Tooth absence was attributed to wear from a high grit diet and third molar agenesis (Costa 1980). The prevalence of periapical granulomatous lesions in the Tigara collection was 20% (n=69). This was less than

reported by Giardini and Eggers (2005) for the Tigara collection (62%, n=63) and by Keenleyside (1998) for four Eskimo (57%, n=118) and one Aleut (67%, n=57) populations. Dental abscesses in these populations was attributed to attrition resulting from grit in the diet and the lower prevalence of granulomatous periapical lesions found in this analysis suggests the Tigara sample had less severe dental attrition and fewer associated complications (Giardini and Eggers 2005; Keenleyside 1998).

The prevalence of periodontal disease in the Tigara collection was 64% (n=64). Schwartz and colleagues (1995) identified periodontal disease in 69% (n=48) of the Tigara sample. Costa (1982) reported severe localized periodontal disease in Tigara between 35 and 40 years of age and attributed this to a high protein and high fat marine-based diet. Keenleyside (1998) reported periodontal disease in 43% (n=114) of four Eskimo populations and in 47% (n=57) of one Aleut population. The Tigara were expected to have periodontal disease due to the high protein diet and the results were within the range found in other studies.

The Tigara were expected to have DEH resulting from chronic cold stress. The prevalence of individuals in the Terry collection that had DEH on the canines or maxillary incisors was 71% (n=45), which was higher than reported by other researchers. Dabbs (2011) found 6.9% (n=116) of the Tigara individuals had DEH on a permanent maxillary incisor and 31.1% (n=151) of the Tigara individuals had DEH on a permanent canine. Guatelli-Steinberg and colleagues (2004) found DEH on the permanent anterior teeth of 38.1% (n=21) Point Hope Inuits which they attributed cold stress. Keenleyside (1998) identified DEH on the incisors and canines in 29% (n=69) of four Eskimo populations and 3% (n=38) of one Aleut population. This difference may have been due to higher stress in the Tigara collection and differences in methods and tooth selection.

The stark contrast between lived experiences in the Hamann-Todd and Terry collections, which are composed of marginalized individuals from an industrial westernized society, and the Tigara population, which were egalitarian pre-European contact indigenous hunter gatherers from the Arctic, provided a basis for interpreting the role of marginalization and structural violence on OM and MACS hypopneumatization. Compared to the Hamann-Todd and Terry collections the Tigara collection was expected to have a lower prevalence of PH and CO due to an iron rich diet and a lower prevalence of OM because pathogen exposure and transmission was minimal. The results support this hypothesis, but sample bias in the Terry and Tigara collections complicated the interpretation of results. The co-occurrence of OM and traditional skeletal stress markers in the context of the sample bias is discussed in Section 7.3.

7.3 Co-occurrence of OM and Skeletal Stress Markers

This analysis tested the hypothesis that MACS hypopneumatization, as a measure of OM, is a skeletal stress marker and its prevalence will be associated with other measured skeletal stress markers. OM was significantly associated with PH ($p<0.013$) (Table 48) and mean AMTL ($p<0.05$). Four individuals had both PH and OM however statistically less than one individual should have had both pathologies (observed: 4, expected: 0.5). Fifty-seven percent of the individuals with PH (n=7) and 29% of the individuals with MACS <5ml (n=14) had both OM and PH, and all of these individuals were from the Hamann-Todd collection. The Terry and Tigara collections had a low prevalence of PH likely resulting from sample bias which excluded individuals with cranial vault pathologies. These collections had significantly less prevalence of

OM ($p<0.008$). Mean AMTL was 4.07 teeth in the individuals with OM and 9.58 teeth in the individuals without OM.

Table 48: Prevalence of PH and OM by collection

	PH	OM
Hamann-Todd	30% (n=20)	69% (n=13)
Terry	0% (n=105)	3% (n=104)
Tigara	1% (n=69)	3% (n=69)

The Hamann-Todd collection had expected levels of PH, which is a marker of childhood and adolescent anemia. The Terry collection had no evidence of PH and therefore the individuals included in the sample did not have anemia or it was not sufficient to cause diploic expansion. De la Cova (2008) and Atwell (2017) reported a combined prevalence of PH and CO greater than 50% in the Terry collection and the absence of PH in this analysis is most likely the result of sample selection. Similarly, OM was significantly less prevalent in the Terry collection compared to the Hamann-Todd collection. The individuals from the Hamann-Todd and Terry collections lived during the same time period and experienced similar stressors so similar values of skeletal stress markers were expected. However, major differences in demographics and sample selection existed between these samples. The Hamann-Todd sample was primarily young adult, African American and male, whereas the Terry sample had a more even distribution of individuals by age, sex and ancestry. The Terry sample was composed of individuals without PH and it appears that when PH was excluded from the sample that OM was concurrently removed. This suggests that there is an association between anemia and OM.

Several researchers have identified a link between OM and anemia. Akcan and colleagues (2019) compared iron levels in children with OM to children without OM (control) and they found significantly more ($p<0.001$) individuals with anemia in the OM group (15.9%, n=113) than the control group (3.4%, n=117). They concluded anemia was a risk factor for OM. Levy and

colleagues (2005) compared OM prevalence in Bedouin infants with and without anemia. At six months of age they found significantly more ($p<0.01$) chronic OM (≥5 episodes of OM within one year) in anemic children than non-anemic children. In children between 7 and 18 months of age they found significantly more ($p<0.01$) episodes of OM in anemic children (3.4+/-2.9 episodes) than nonanemic children (2.4+/-2.2 episodes). They concluded that anemia increases infectious disease prevalence and treatment should be considered to reduce disease burden. However, in contrast to the conclusions by Akcan and colleagues (2019) and Levy and colleagues (2005) other researchers have noted anemia is a natural defense to a bacterial infection by depriving the bacteria of iron necessary for reproduction, and iron supplementation in individuals with malaria and tuberculosis have demonstrated worse outcomes (Murray et al. 1978; Sazawal et al. 2006). Therefore, it is possible that anemia was not an exacerbating factor in the OM infection, but the body's natural response to a bacterial infection that may have also been the cause of the OM.

Golz and colleagues (2001) compared 680 children with recurrent OM (>4 episodes of OM within one year) to 200 children with no history of OM (control). The OM children had an average of 8.3+/-2.7 episodes of OM per year and a mean hemoglobin level of 11.4+/-2.7 g/dl. The control children had a mean hemoglobin level of 13.1+/-2.5 g/dl. Anemia was defined as a hemoglobin level below 9.5g/dl. In the OM children 20% (n=136) were anemic, whereas only 1.5% (n=3) of the control children had were anemic. Within the anemic OM children 83.8% (n=114) had more than 12 episodes of OM per year (compared to the average of 8.3+/-2.7 episodes). Iron supplementation was administered until iron levels reached 11g/dl. Of the anemic OM children 96% (130/136) of them had a decrease in the number of OM episodes per year following iron supplementation, and only 4% (6/136) of children had no change. However, iron supplementation only decreased the number of episodes per year and did not completely eliminate OM in any child.

Iron deficiency likely exacerbates an underlying issue (e.g., decreased resistance to infection) and results in an increased OM risk. Although the authors claim the results were not due to an age-related decrease in number of OM episodes because all of the children remained within the high-risk age range, other factors could affect the number of OM episodes (e.g., influenza season severity) and they should have controlled for age and external factors by reporting the number of OM episodes per year in the non-anemic OM group. Although these studies demonstrate anemia as a risk factor for OM few researchers have measured iron levels when assessing OM risk factors.

OM was demonstrated to be a skeletal stress marker of structural violence that co-occurs with PH. A higher than expected prevalence of OM was identified in the young adult age group (observed: 10, expected: 4.9) and the African American ancestry cohort (observed: 11, expected: 5.6). This is likely a reflection of the high proportion of young adult (75%) and African American (80%) individuals in the Hamann-Todd collection, which had a higher prevalence of OM than the Terry and Tigara collections. Individuals with OM also had significantly less mean AMTL (OM: 4.07 teeth, no OM 9.58 teeth), due to the higher prevalence of the young adult age group with OM. The high prevalence of OM (and PH) in the young adult age group suggests early life stress embodied as childhood skeletal stress markers burdened the individual and the stress accumulated during life and placed these individuals at a higher risk for early mortality (Taylor 2010). There was also a higher prevalence of OM in the African American ancestry cohort. Within the Hamann-Todd and Terry collections the assertion of structural violence can only be made at the time of death, however in the late 19th and early 20th centuries African American individuals experienced structural violence throughout their lives. Therefore the significantly greater prevalence of OM, a childhood disease, in the African American cohort may be a reflection of the lifelong stress of structural violence unique to the African American cohort. Although the low prevalence of OM in

the Terry collection and high prevalence of OM in the Hamann-Todd collection, young adult age group and African American ancestry cohort was likely the result of sample bias, environmental, cultural and biological factors cannot be ruled out.

7.4 Limitations

There are several limitations to testing a skeletal stress marker within the biocultural model. First, the biocultural model cannot be tested on its own, but must be interpreted within a theoretical framework (Martin et al. 2013). The number of variables that can be placed into the model is unlimited and a theoretical framework allows those variables to be narrowed down to focus on one aspect of a society. The risk when selecting a theoretical model is choosing one that does not capture the skeletal stress marker of interest. For example, evolution and adaptation to extreme cold is not expected to affect PH prevalence. OM is strongly associated with low socioeconomic status which almost certainly was the condition of the Hamman-Todd and Terry individuals, therefore structural violence can confidently be used as a model to test OM as a skeletal stress marker. Second, the sample selected must experience the social theory that is being measured. Within the Hamann-Todd and Terry collections structural violence has been established (Muller et al. 2017; Nystrom 2014), but these individuals experienced structural violence at the time of death and their status during childhood, when OM would have been most likely to develop, is unknown. Therefore, these collections may not be the most appropriate choice when testing OM as a skeletal stress marker within the theoretical framework of structural violence. Third, once an appropriate collection is determined sample selection is important. In this analysis sample selection was restricted to individuals with radiographed skulls and it was not until after the statistical

analysis was performed that it became clear that the skulls scanned by Copes (2012) did not have PH or OM. Fourth, the selection of skeletal stress markers is important[6] and should be tailored to the social theory.

Finally, stress is not always embodied as a skeletal stress marker. Cultural and biological (i.e., host resistance) buffers can protect an individual from disruption to homeostasis. Hidden heterogeneity of risk is an individual's unique and differential response to a stress (DeWitte and Stojanowski 2015; Wood et al. 1992). Under identical circumstances two individuals may have different physiological responses to stress and expression of skeletal stress markers and therefore interpretations of skeletal stress markers should only be ascribed to a population and not an individual. Under extreme stress an individual may die before the stress can be embodied as a skeletal stress marker; this individual would appear to be stress-free but would have experienced extreme stress at or near the time of death. The social status of the Hamann-Todd and Terry individuals prior to death was unknown and it is possible some individuals lived a high-status life and experienced hardship just prior to death resulting in their incorporation into the Hamann-Todd and Terry collections.

[6] The skeletal stress markers were limited to the skull because originally the Samuel George Morton Cranial Collection was included in this analysis.

8.0 Conclusion

This dissertation affirmed the hypothesis that MACS hypopneumatization, as a measure of OM, was a skeletal stress marker of structural violence capable of delineating differences of socioeconomic status and marginalization. OM risk is augmented by anatomic, immunogenic and pathogenic processes, and risk to the immunogenic and pathogenic pathways was increased in the groups that experienced structural violence. For the purpose of this study OM was categorized as a skeletal stress marker of infectious disease. The pathogenic components of structural violence and marginalization (e.g., pollution and crowded living conditions) (Bruce et al. 2000; Csákányi et al. 2012; da Costa et al. 2004; Klein 2001) were expected to increase the risk of pathogen exposure and transmission and the immunogenic components (e.g., psychological stress and depression) (Cohen et al. 2007; Leonard 2000) were expected to limit the host's immune response and ability to fight infection. The results revealed that OM infection and anemia co-occurred either due to an underlying bacterial infection which produced the dual diagnosis or OM was secondary to anemia. Although OM is a common childhood infection, frequent or chronic episodes are necessary to produce skeletal changes (i.e., MACS hypopneumatization). Under anemic conditions OM may be exacerbated and become chronic and recurrent, and therefore more likely to result in MACS hypopneumatization (Golz et al. 2001). In this scenario the contributing factor to OM prevalence is anemia and OM acted as both a nutritional and infectious disease skeletal stress marker. However, it is also possible that an underlying bacterial infection resulted in the presence of both anemia and OM in an individual.

These results lend some support to the limited clinical literature that reports anemia as a risk factor of OM (Akcan et al. 2019; Golz et al. 2001; Levy et al. 2005). Despite the Golz and

colleagues (2001) study that showed a reduction in the number of OM episodes in anemic patients following iron supplementation few researchers have explored the relationship between anemia and OM. The relationship between anemia and OM is not discernable through skeletal remains, but more robust clinical studies can aid in determining the association.

Measures of OM (e.g., MACS hypopneumatization, ossicle remodeling and stapedial footplate fixation) are often studied in isolation and in the absence of other disease processes (e.g., Birkby 1975, Flohr and colleagues 2014, Gregg and Steele 1965, Rathbun and Mallin 1977, and Titche and colleagues 1981). Although these studies include the historical and archaeological context the biological circumstances are often limited to demographic information. The findings from this analysis highlight the importance of always recording PH in conjunction with OM to either exclude or include it as a mitigating factor in OM risk. Inclusion of biological information should not be limited to PH, but expanded to include measures of stress, disease and activity to create a complete understanding of OM in the past.

High prevalence rates of OM were expected in both the Hamann-Todd and Terry collections, but the Terry collection had significantly less OM. The Terry collection was selected from skulls previously radiographed by Copes (2012) and she excluded skulls with certain cranial vault pathologies likely excluding individuals with PH. By excluding PH it seems that OM was incidentally excluded from the Terry sample. The Tigara collection was also scanned by Copes (2012) and although the Tigara were expected to have low prevalence of PH, the sample bias prevents confirming this assumption and as a result the anemic status of the Tigara and subsequent OM infection is unknown. OM may have been inadvertently removed from the Tigara sample and therefore MACS hypopneumatization, as a marker of OM, was not tested as a skeletal stress marker using the evolution and adaptation to chronic cold theoretical model. The efficacy of

MACS hypopneumatization as a skeletal stress marker should be tested using other theoretical models and in conjunction with established cranial and postcranial skeletal stress markers.

It is also possible that OM was not a result of structural violence and marginalization, but rather the cause. Anemia resulting from childhood malnutrition or disease could increase OM risk resulting in more frequent OM episodes in susceptible individuals. Chronic OM can result in hearing loss and language delays. These impairments can be perceived as disabilities (e.g., learning disability) which could carry over into adulthood and result in decreased socioeconomic status which can serve as a precursor to incorporation into the Hamann-Todd and Terry collections. It is likely that OM is both the result and cause of marginalization depending on the individual circumstances.

This dissertation serves as a blueprint for applying a social theory to the biocultural model to test OM as a skeletal stress marker using the proposed *biocultural skeletal stress marker pathway*. It demonstrated that OM prevalence was increased in the structural violence group with anemia (Hamann-Todd) compared to the structural violence group without anemia (Terry) and the cold stress group (Tigara). The relationship between OM and anemia (PH) should continue to be tested within other populations, using additional theoretical models and incorporating postcranial skeletal stress markers. However, radiography is often cost-prohibitive and inaccessible. Therefore, secondary measures of OM (e.g., mastoid abscess, ossicle remodeling and stapedial footplate fixation) should be explored, and Flohr and colleagues' (2009) macroscopic classification system of MACS pneumatization should be tested in archaeological material.

Appendix A : MACS and Skeletal Stress Marker Data

					MACS													
					#	Volume (cm3)		Surface Area (cm2)								Granulomatous	Periodontal	
Collection	ID#	Biological Sex	Race	Age	Slides	Left	Right	Left	Right	OM	PH	CO	Sinusitis	AMTL	# AMTL	Periapical Lesion	Disease	DEH
Hamann	529	Female	Black	Young adult						9	0	0	0	1	2	0	1	1
Hamann	620	Male	Black	Young adult	28	1.89	11.59	13.03	41.26	1	0	0	0	0	0	0	0	1
Hamann	627	Male	White	Middle adult						9	0	0	0	1	3	0	1	0
Hamann	638	Male	Black	Young adult						9	1	0	0	1	1	1	1	1
Hamann	684	Male	White	Old adult						9	0	0	0	1	4	1	1	1
Hamann	744	Male	Black	Young adult	23	1.79	1.66	17.49	16.05	1	1	0	0	1	3	0	1	1
Hamann	749	Female	White	Old adult						9	0	0	0	1	2	0	1	1
Hamann	799	Male	White	Middle adult						9	0	0	0	1	4	0	0	0
Hamann	858	Male	Black	Young adult	35	2.96	3.52	32.15	33.96	1	1	0	0	0	0	0	0	1
Hamann	859	Male	Black	Young adult	31	3.52	4.05	29.19	32.93	1	1	0	0	1	2	0	1	1
Hamann	862	Male	Black	Young adult	19	6.00	6.12	47.50	54.15	0	0	0	0	0	0	0	1	1
Hamann	913	Male	Black	Young adult	22	4.97	4.34	56.47	55.00	1	0	0	0	0	0	0	0	0
Hamann	955	Male	Black	Young adult	30	5.17	4.52	44.27	34.20	1	0	0	0	0	0	0	0	0
Hamann	1009	Male	Black	Young adult	36	6.03	9.63	35.59	51.09	0	0	0	0	1	1	0	0	1
Hamann	1109	Male	Black	Young adult	27	6.64	8.02	46.14	49.10	0	0	0	0	0	0	0	1	1
Hamann	1112	Male	Black	Young adult	15	3.25	3.48	27.45	32.72	1	0	0	0	1	2	0	1	1
Hamann	1120	Male	Black	Young adult	36	6.80	6.06	51.33	41.74	0	1	0	0	1	1	0	1	0
Hamann	1130	Female	Black	Young adult	21	3.08	1.86	28.29	14.96	1	1	0	0	1	2	0	1	0
Hamann	1277	Female	Black	Young adult	29	3.06	2.88	30.88	26.30	1	0	1	0	1	1	0	0	1
Hamann	1468	Male	Black	Middle adult						9	0	0	0	1	1	1	1	1
Terry	236	Female	White	Old adult	32	16.39	16.22	215.40	196.55	0	0	0	1	1	32	0	9	9
Terry	289	Female	White	Middle adult	41	26.86	31.21	166.48	187.49	0	0	0	0	1	14	0	0	1
Terry	634	Male	White	Old adult	22	9.44	13.38	120.72	150.79	0	0	0	0	1	25	0	9	1
Terry	635	Male	Black	Middle adult	23	13.64	13.73	114.97	131.08	0	0	0	0	1	13	0	1	1
Terry	638	Male	Black	Middle adult						9	0	0	0	1	4	0	1	1
Terry	648	Male	Black	Old adult	27	18.06	12.24	121.15	100.72	0	0	0	0	1	14	1	1	0
Terry	651	Male	White	Old adult	23	8.85	13.64	123.46	129.07	0	0	0	1	1	14	0	1	1

					MACS													
					#	Volume (cm3)		Surface Area (cm2)								Granulomatous	Periodontal	
Collection	ID#	Biological Sex	Race	Age	Slides	Left	Right	Left	Right	OM	PH	CO	Sinusitis	AMTL	# AMTL	Periapical Lesion	Disease	DEH
Terry	685	Female	Black	Old adult	25	20.99	17.76	177.41	175.11	0	0	0	1	1	32	0	9	9
Terry	687	Male	White	Old adult	24	17.89	16.42	183.17	154.10	0	0	0	0	1	7	0	1	1
Terry	688	Male	White	Old adult	23	19.64	18.55	111.51	104.03	0	0	0	0	1	12	1	1	0
Terry	697	Male	White	Old adult	24	13.63	12.62	138.56	119.00	0	0	0	1	1	17	0	9	9
Terry	698	Male	Black	Middle adult	25	12.17	13.48	122.02	122.02	0	0	0	0	1	7	0	1	1
Terry	701	Male	White	Middle adult	20	5.63	7.35	66.48	81.44	0	0	0	0	1	15	0	0	1
Terry	703	Male	Black	Old adult	23	6.56	9.61	100.43	146.77	0	0	0	0	1	22	0	9	1
Terry	711	Male	Black	Old adult	28	10.69	13.18	149.50	182.16	0	0	0	0	1	6	0	1	1
Terry	712	Male	Black	Middle adult	24	14.85	12.16	136.26	119.00	0	0	0	0	0	0	0	1	0
Terry	713	Male	White	Old adult	25	13.01	24.99	152.38	181.44	0	0	0	0	1	5	1	0	1
Terry	714	Male	White	Old adult	21	10.22	11.73	116.69	109.07	0	0	0	1	1	19	0	9	9
Terry	723	Female	Black	Young adult	24	11.54	14.23	180.29	177.27	0	0	0	0	0	0	0	1	1
Terry	724	Male	Black	Middle adult	22	12.07	12.45	126.05	122.45	0	0	0	0	1	4	0	1	1
Terry	725	Male	Black	Old adult	26	11.86	17.15	128.64	169.93	0	0	0	0	1	9	0	1	1
Terry	736	Female	White	Old adult	20	5.38	9.34	64.17	92.81	0	0	0	1	1	17	0	0	0
Terry	738	Female	Black	Middle adult	26	19.40	17.44	134.54	115.25	0	0	0	0	1	3	0	1	0
Terry	813	Male	White	Old adult	24	15.41	13.71	150.51	130.51	0	0	0	0	1	15	0	1	9
Terry	814	Male	White	Middle adult	23	16.89	16.81	151.23	162.88	0	0	0	0	0	0	0	0	1
Terry	815	Female	Black	Young adult	24	7.40	5.40	92.23	71.80	0	0	0	0	0	0	0	1	1
Terry	816	Male	Black	Middle adult	28	24.68	26.94	196.84	191.80	0	0	0	0	1	1	0	1	1
Terry	817	Male	Black	Middle adult	21	16.73	19.67	96.26	102.59	0	0	0	0	1	13	0	1	9
Terry	825	Male	Black	Middle adult	26	14.76	13.37	149.21	133.10	0	0	0	0	1	13	0	0	1
Terry	826	Male	Black	Middle adult	25	8.45	9.02	117.56	108.20	0	0	0	0	1	6	1	1	1
Terry	830	Male	Black	Young adult	30	18.39	17.53	235.54	224.75	0	0	1	0	0	0	0	0	1
Terry	840	Female	Black	Young adult	21	14.46	13.01	147.92	118.56	0	0	0	0	1	6	1	0	1
Terry	842	Male	White	Old adult	30	29.11	27.53	145.47	158.42	0	0	0	0	1	22	0	9	1
Terry	843	Male	White	Old adult	26	21.32	18.29	153.53	144.61	0	0	0	0	1	2	0	1	1
Terry	850	Male	Black	Young adult	25	13.83	14.55	135.40	142.45	0	0	0	0	1	1	0	1	1
Terry	853	Male	White	Old adult	21	14.09	12.99	115.97	105.18	0	0	0	0	1	8	0	0	9
Terry	855	Male	White	Old adult	20	7.27	6.01	94.25	84.32	0	0	0	0	1	29	0	9	9
Terry	864	Male	Black	Middle adult	20	8.76	8.63	105.76	94.25	0	0	0	1	1	4	0	1	0
Terry	865	Male	White	Old adult	22	12.10	13.60	129.07	122.45	0	0	1	0	1	32	0	9	9
Terry	867	Male	White	Middle adult	22	8.79	10.01	119.57	128.35	0	0	0	0	1	10	0	0	1
Terry	868	Male	White	Old adult	26	11.11	11.76	130.65	138.71	0	0	0	0	1	13	0	1	1
Terry	869	Female	White	Old adult	21	16.00	14.99	125.90	117.12	0	0	0	1	1	27	0	9	0
Terry	878	Male	White	Old adult	30	14.10	15.53	157.41	187.49	0	0	0	0	1	19	0	1	1
Terry	879	Male	White	Middle adult	27	10.85	14.92	156.69	184.90	0	0	0	1	1	9	0	0	1
Terry	880	Female	White	Young adult	21	8.37	9.30	88.06	107.92	0	0	1	0	1	4	0	0	1

					MACS													
					#	Volume (cm3)		Surface Area (cm2)								Granulomatous	Periodontal	
Collection	ID#	Biological Sex	Race	Age	Slides	Left	Right	Left	Right	OM	PH	CO	Sinusitis	AMTL	# AMTL	Periapical Lesion	Disease	DEH
Terry	881	Male	Black	Young adult	28	16.94	16.63	169.50	170.08	0	0	0	0	0	0	0	0	0
Terry	882	Male	Black	Young adult	26	11.02	11.37	136.41	153.38	0	0	0	0	1	13	0	0	0
Terry	890	Male	Black	Old adult	30	30.03	24.88	168.78	180.44	0	0	0	0	1	14	0	1	0
Terry	893	Female	Black	Old adult	30	11.55	23.77	166.62	246.62	0	0	0	0	1	32	0	9	9
Terry	904	Female	White	Middle adult	25	14.01	13.04	167.63	155.26	0	0	0	0	1	23	0	0	0
Terry	905	Male	Black	Young adult	32	27.70	23.89	253.39	239.72	0	0	0	0	0	0	0	1	1
Terry	906	Female	Black	Young adult	19	5.53	7.22	65.61	71.66	0	0	0	1	1	2	0	1	1
Terry	908	Male	White	Middle adult	24	11.47	11.35	130.36	124.03	0	0	0	1	1	5	1	9	9
Terry	917	Female	Black	Old adult	25	15.73	18.72	149.64	149.21	0	0	0	1	1	27	0	9	0
Terry	918	Male	White	Middle adult	22	6.07	6.99	77.56	96.84	0	0	1	1	1	6	1	1	1
Terry	920	Female	Black	Middle adult	18	5.94	5.09	66.04	60.00	0	0	0	0	1	4	0	1	1
Terry	921	Female	Black	Middle adult	22	7.70	11.54	87.77	106.48	0	0	0	0	0	0	0	1	1
Terry	929	Female	Black	Young adult	27	26.06	25.14	140.43	153.53	0	0	0	0	1	2	1	1	1
Terry	942	Male	Black	Middle adult	24	10.35	14.01	153.10	174.97	0	0	0	0	1	6	0	1	1
Terry	956	Male	White	Old adult	25	12.22	11.50	137.70	119.71	0	0	0	0	1	4	0	0	1
Terry	969	Male	Black	Middle adult	27	20.04	13.50	216.41	140.58	0	0	1	0	0	0	0	1	1
Terry	970	Female	Black	Young adult	25	8.30	10.79	106.48	114.53	0	0	0	0	0	0	0	1	1
Terry	979	Male	White	Old adult	25	8.83	10.48	145.90	161.01	0	0	0	1	1	32	0	9	9
Terry	980	Male	Black	Old adult	29	21.74	30.48	235.83	266.19	0	0	0	0	1	6	0	1	1
Terry	981	Male	Black	Young adult	25	17.35	19.11	164.18	168.35	0	0	0	1	0	0	0	1	1
Terry	982	Male	White	Old adult	23	10.91	13.12	126.33	138.28	0	0	0	1	1	22	0	1	1
Terry	990	Male	Black	Young adult	27	27.68	20.50	155.97	140.00	0	0	1	0	0	0	0	1	1
Terry	1015	Female	Black	Middle adult	19	4.62	11.30	71.94	104.32	1	0	0	0	0	0	0	1	1
Terry	1028	Male	White	Old adult	25	14.60	13.84	189.07	170.51	0	0	0	0	1	10	0	1	9
Terry	1040	Male	White	Old adult	20	9.61	9.22	92.38	103.02	0	0	0	0	1	21	0	9	9
Terry	1052	Female	Black	Middle adult	24	11.12	10.65	155.11	140.72	0	0	0	0	1	17	0	1	1
Terry	1064	Female	Black	Young adult	22	13.65	19.02	133.38	141.30	0	0	0	0	1	18	1	1	1
Terry	1087	Male	White	Old adult	30	23.17	24.84	205.90	204.90	0	0	0	0	1	32	0	9	9
Terry	1089	Male	White	Middle adult	25	14.55	16.82	144.90	136.55	0	0	0	0	1	9	0	1	1
Terry	1108	Male	Black	Old adult	24	12.86	11.53	183.46	170.51	0	0	0	0	1	30	1	9	1
Terry	1110	Male	White	Old adult	24	9.94	11.77	127.34	153.96	0	0	0	0	1	32	0	9	9
Terry	1120	Female	White	Young adult	23	10.85	9.77	129.93	126.33	0	0	0	0	1	16	0	9	0
Terry	1131	Male	Black	Young adult	22	9.60	10.36	112.09	99.71	0	0	0	0	0	0	0	1	0
Terry	1135	Female	Black	Old adult	24	6.85	5.97	95.40	90.07	0	0	0	0	1	5	1	1	1
Terry	1154	Female	White	Old adult	23	16.23	12.06	216.84	177.41	0	0	0	0	1	32	0	9	9
Terry	1155	Female	White	Old adult	24	17.24	14.07	122.16	109.35	0	0	0	0	1	32	0	9	9
Terry	1168	Male	Black	Young adult	24	9.65	9.48	89.79	104.32	0	0	1	0	1	3	0	1	1
Terry	1206	Male	Black	Young adult	24	11.93	12.13	141.30	137.99	0	0	0	0	0	0	0	1	1

					MACS													
					#	Volume (cm3)		Surface Area (cm2)								Granulomatous	Periodontal	
Collection	ID#	Biological Sex	Race	Age	Slides	Left	Right	Left	Right	OM	PH	CO	Sinusitis	AMTL	# AMTL	Periapical Lesion	Disease	DEH
Terry	1207	Male	Black	Old adult	28	10.91	14.04	147.20	166.48	0	0	0	0	1	13	1	0	0
Terry	1233	Female	White	Old adult	17	3.37	6.78	49.93	89.35	1	0	0	1	1	2	0	1	0
Terry	1257	Male	Black	Middle adult	28	22.40	20.95	230.80	247.78	0	0	0	0	1	7	0	1	1
Terry	1280	Male	Black	Old adult	27	19.22	18.62	194.54	174.25	0	0	0	0	1	4	1	1	1
Terry	1281	Male	Black	Middle adult	25	10.79	12.94	121.59	158.85	0	0	0	1	1	7	1	0	1
Terry	1292	Female	Black	Old adult	20	8.27	6.13	108.64	84.17	0	0	0	0	1	27	0	9	1
Terry	1293	Male	Black	Middle adult	25	12.29	10.33	140.58	100.15	0	0	0	0	1	9	0	0	1
Terry	1306	Female	Black	Middle adult	21	13.48	12.63	123.74	126.48	0	0	0	0	1	2	0	0	1
Terry	1321	Male	White	Old adult	21	7.90	9.64	121.87	130.51	0	0	0	0	1	30	0	9	9
Terry	1323	Male	Black	Middle adult	22	11.44	12.10	131.51	141.01	0	0	0	0	1	10	0	9	0
Terry	1332	Female	Black	Middle adult	23	12.56	12.53	216.26	187.49	0	0	0	0	0	0	0	1	1
Terry	1333	Female	Black	Young adult	20	10.07	9.37	114.39	108.06	0	0	0	0	0	0	0	1	0
Terry	1334	Male	Black	Old adult	22	13.24	6.20	144.90	86.76	0	0	0	0	1	27	0	9	1
Terry	1336	Male	Black	Old adult	25	8.89	10.96	122.45	132.66	0	0	1	0	1	24	0	9	1
Terry	1345	Female	Black	Middle adult	19	4.92	4.39	50.07	53.38	1	0	0	0	1	24	0	9	1
Terry	1349	Male	Black	Old adult	24	10.40	9.32	162.45	146.62	0	0	0	0	1	17	0	9	9
Terry	1356	Female	Black	Young adult	26	18.33	24.20	112.95	138.71	0	0	0	0	0	0	0	1	0
Terry	1361	Male	Black	Middle adult	24	11.83	13.31	156.55	161.73	0	0	0	0	1	6	0	1	1
Terry	1370	Female	White	Old adult	19	9.25	7.76	90.07	97.12	0	0	0	0	1	32	0	9	9
Terry	1496	Female	White	Old adult	39	6.79	5.55	78.42	68.63	0	0	1	1	1	32	0	9	9
Terry	1501	Female	Black	Old adult	48	31.93	28.72	199.14	190.36	0	0	0	0	1	24	0	9	9
Terry	1502	Female	White	Old adult	45	25.86	15.83	157.27	119.00	0	0	0	0	1	31	0	9	9
Tigara	226	Female	Native American	Middle adult	25	20.35	16.20	152.38	164.46	0	0	0	0	1	9	1	1	0
Tigara	228	Male	Native American	Young adult	35	28.52	26.75	283.17	290.08	0	0	0	1	1	1	0	0	1
Tigara	232	Female	Native American	Unknown	23	16.79	16.19	180.72	172.38	0	0	0	0	1	24	0	1	9
Tigara	233	Male	Native American	Middle adult	24	18.24	14.79	143.02	153.10	0	0	0	0	1	4	0	1	1
Tigara	234	Female	Native American	Unknown	25	22.20	20.68	152.09	149.64	0	0	0	0	1	9	0	1	1
Tigara	235	Male	Native American	Unknown	29	26.22	25.97	227.77	246.48	0	0	0	1	1	2	0	1	9
Tigara	237	Male	Native American	Unknown	27	24.04	20.91	260.15	240.72	0	0	0	0	0	0	0	1	1
Tigara	240	Female	Native American	Unknown	21	12.95	14.27	104.46	112.95	0	0	0	0	1	3	0	1	1
Tigara	250	Male	Native American	Middle adult	24	14.99	16.82	111.37	116.55	0	0	0	0	1	10	0	0	0
Tigara	260	Female	Native American	Middle adult	24	14.85	17.81	135.69	148.64	0	0	9	0	1	8	1	1	0
Tigara	276	Female	Native American	Young adult	19	12.33	10.01	110.22	87.63	0	0	0	0	0	0	0	0	1
Tigara	292	Male	Native American	Middle adult	13	10.23	9.38	86.48	81.44	0	0	0	0	1	16	1	0	0
Tigara	312	Male	Native American	Young adult	11	8.29	8.75	70.22	73.53	0	0	0	0	0	0	0	0	1
Tigara	336	Male	Native American	Middle adult	23	15.02	8.65	147.49	123.17	0	0	0	1	0	0	0	1	1
Tigara	357	Male	Native American	Middle adult	14	11.32	10.40	83.02	71.08	0	0	1	0	0	0	1	1	0
Tigara	362	Female	Native American	Young adult	11	4.79	5.97	60.58	72.52	1	0	0	0	1	13	0	0	9

					MACS													
					#	Volume (cm3)		Surface Area (cm2)								Granulomatous	Periodontal	
Collection	ID#	Biological Sex	Race	Age	Slides	Left	Right	Left	Right	OM	PH	CO	Sinusitis	AMTL	# AMTL	Periapical Lesion	Disease	DEH
Tigara	364	Male	Native American	Middle adult	15	16.39	16.68	95.54	111.66	0	0	0	0	0	0	0	1	0
Tigara	366	Female	Native American	Unknown	11	6.60	10.20	59.14	61.44	0	0	0	1	1	19	0	0	9
Tigara	368	Female	Native American	Young adult	13	11.58	11.05	84.46	76.55	0	0	9	0	1	28	0	9	9
Tigara	377	Male	Native American	Young adult	12	9.93	8.78	77.84	62.45	0	0	0	1	0	0	0	0	1
Tigara	381	Male	Native American	Middle adult	11	4.92	4.37	42.02	34.96	1	0	9	0	1	8	1	1	9
Tigara	390	Male	Native American	Young adult	15	16.07	15.34	99.43	95.40	0	1	1	0	0	0	0	1	1
Tigara	394	Female	Native American	Middle adult	24	22.03	23.73	166.62	149.36	0	0	0	1	1	2	0	1	9
Tigara	395	Male	Native American	Middle adult	27	30.33	23.41	164.46	140.72	0	0	0	0	1	20	0	1	9
Tigara	396	Male	Native American	Middle adult	24	19.80	22.78	171.80	166.48	0	0	0	0	1	1	0	1	9
Tigara	397	Male	Native American	Young adult	27	17.27	20.39	168.78	187.34	0	0	0	1	1	1	1	1	1
Tigara	402	Male	Native American	Middle adult	26	17.99	14.36	164.90	150.22	0	0	9	0	1	20	1	0	1
Tigara	407	Male	Native American	Young adult	28	29.78	27.08	175.26	166.48	0	0	0	0	0	0	0	0	1
Tigara	421	Female	Native American	Middle adult	22	13.37	20.19	148.06	177.99	0	0	1	0	0	0	0	1	9
Tigara	422	Female	Native American	Young adult	19	12.46	15.91	123.46	125.47	0	0	0	0	0	0	1	1	0
Tigara	428	Male	Native American	Middle adult	30	24.88	22.96	211.52	205.76	0	0	0	0	1	4	0	1	9
Tigara	438	Female	Native American	Young adult	24	16.37	10.82	150.79	84.17	0	0	0	0	1	2	0	0	1
Tigara	441	Male	Native American	Middle adult	27	24.36	26.98	156.26	160.29	0	0	0	0	1	1	0	1	1
Tigara	445	Male	Native American	Middle adult	27	38.89	31.15	213.82	207.63	0	0	9	0	0	0	0	1	1
Tigara	447	Male	Native American	Young adult	26	23.40	23.27	214.25	210.80	0	0	0	0	0	0	0	0	1
Tigara	451	Female	Native American	Middle adult	21	15.70	19.24	156.55	167.77	0	0	1	0	1	21	0	9	9
Tigara	455	Female	Native American	Young adult	19	13.65	15.27	127.34	116.98	0	0	0	1	1	16	0	9	9
Tigara	458	Female	Native American	Unknown	26	21.30	23.08	145.76	154.68	0	0	1	0	1	22	0	9	9
Tigara	463	Female	Native American	Young adult	24	18.30	19.73	125.90	141.44	0	0	0	0	1	1	0	0	9
Tigara	464	Female	Native American	Middle adult	22	13.65	16.96	133.24	158.42	0	0	0	0	1	1	0	0	1
Tigara	465	Female	Native American	Middle adult	18	13.08	11.27	118.42	117.99	0	0	0	0	1	7	0	1	9
Tigara	468	Male	Native American	Young adult	27	18.33	17.31	130.51	135.11	0	0	0	0	1	9	0	1	0
Tigara	472	Male	Native American	Young adult	23	11.14	11.12	96.69	127.20	0	0	1	1	0	0	0	1	1
Tigara	474	Male	Native American	Young adult	25	8.65	19.89	101.73	162.02	0	0	0	0	0	0	0	0	1
Tigara	476	Male	Native American	Middle adult	28	22.62	27.19	215.26	214.97	0	0	9	0	1	4	0	9	9
Tigara	488	Male	Native American	Young adult	27	20.89	26.52	156.12	156.69	0	0	0	0	0	0	1	1	1
Tigara	492	Male	Native American	Middle adult	12	9.67	10.91	72.81	78.56	0	0	0	0	0	0	0	0	9
Tigara	495	Male	Native American	Middle adult	14	10.10	12.03	69.64	68.49	0	0	0	0	1	18	0	1	1
Tigara	500	Female	Native American	Young adult	24	17.07	22.73	91.08	114.53	0	0	0	0	0	0	0	1	1
Tigara	507	Female	Native American	Young adult	23	15.37	16.33	105.76	107.77	0	0	0	0	0	0	0	0	1
Tigara	509	Male	Native American	Young adult	25	13.50	14.06	118.28	145.47	0	0	0	0	0	0	0	0	0
Tigara	512	Female	Native American	Middle adult	20	13.77	21.24	103.89	113.24	0	0	0	0	1	10	0	0	0
Tigara	513	Female	Native American	Young adult	23	16.42	13.54	137.41	132.23	0	0	0	0	0	0	1	1	9
Tigara	514	Female	Native American	Young adult	22	12.63	11.18	110.51	109.79	0	0	0	0	1	1	1	1	1

					MACS													
					#	Volume (cm3)		Surface Area (cm2)								Granulomatous	Periodontal	
Collection	ID#	Biological Sex	Race	Age	Slides	Left	Right	Left	Right	OM	PH	CO	Sinusitis	AMTL	# AMTL	Periapical Lesion	Disease	DEH
Tigara	515	Female	Native American	Middle adult	21	18.24	10.62	117.70	107.05	0	0	9	0	0	0	0	1	1
Tigara	522	Female	Native American	Middle adult	24	10.53	11.51	94.53	150.65	0	0	0	0	1	7	0	1	9
Tigara	523	Female	Native American	Middle adult	23	18.32	17.61	137.41	145.04	0	0	0	0	1	20	0	0	9
Tigara	524	Female	Native American	Middle adult	24	21.40	19.76	133.53	146.62	0	0	9	0	1	13	0	1	0
Tigara	525	Male	Native American	Young adult	27	23.51	25.70	147.20	162.02	0	0	0	0	1	3	1	1	9
Tigara	526	Female	Native American	Middle adult	22	20.13	11.78	176.98	165.04	0	0	1	0	1	6	1	1	0
Tigara	527	Male	Native American	Middle adult	16	7.73	9.25	68.35	81.87	0	0	0	0	1	14	0	0	9
Tigara	535	Male	Native American	Young adult	14	15.18	14.20	86.62	91.51	0	0	0	0	1	2	0	1	1
Tigara	537	Female	Native American	Middle adult	11	10.56	8.60	69.21	62.02	0	0	0	1	1	1	0	1	0
Tigara	541	Male	Native American	Middle adult	15	14.86	13.51	88.78	81.01	0	0	9	0	1	1	1	1	1
Tigara	542	Male	Native American	Young adult	16	12.30	14.17	88.49	89.64	0	0	0	0	0	0	0	0	1
Tigara	544	Female	Native American	Young adult	13	11.54	12.10	78.28	94.68	0	0	0	1	1	3	0	1	1
Tigara	551	Female	Native American	Middle adult	23	25.34	25.45	166.05	169.07	0	0	0	0	1	1	0	1	9
Tigara	667	Female	Native American	Young adult	23	22.01	23.54	121.15	130.79	0	0	0	0	1	2	0	0	1
Tigara	675	Female	Native American	Unknown	23	17.74	13.01	122.45	112.23	0	0	0	0	0	0	0	1	1

Appendix B : Dental Data

Appendix B.1 Dental Inventory by Individual Tooth

		Collection						Sex				Race					
		Hamann		Terry		Tigara		Male		Female		Black		White		Native American	
Tooth #	Tooth type	n	%	n	%	n	%	n	%	n	%	n	%	n	%	n	%
1	M3	19	95%	48	46%	54	78%	69	58%	43	58%	52	67%	15	32%	54	78%
2	M2	19	95%	61	58%	55	80%	74	62%	49	66%	61	78%	19	40%	55	80%
3	M1	17	85%	56	53%	53	77%	68	57%	46	62%	56	72%	17	36%	53	77%
4	P2	19	95%	66	63%	54	78%	77	64%	47	64%	63	81%	22	47%	54	78%
5	P1	19	95%	71	68%	56	81%	78	65%	52	70%	66	85%	24	51%	56	81%
6	C	20	100%	75	71%	63	91%	85	71%	53	72%	66	85%	29	62%	63	91%
7	I2	20	100%	64	61%	55	80%	77	65%	48	65%	63	81%	21	45%	55	80%
8	I1	20	100%	67	64%	56	81%	78	65%	48	65%	62	79%	25	53%	56	81%
9	I1	20	100%	68	65%	57	83%	78	65%	49	66%	62	79%	26	55%	57	83%
10	I2	20	100%	69	66%	57	83%	78	65%	51	69%	63	81%	26	55%	57	83%
11	C	20	100%	74	70%	63	91%	83	69%	55	74%	65	83%	29	62%	63	91%
12	P1	19	95%	65	62%	57	83%	79	65%	48	65%	63	81%	21	45%	57	83%
13	P2	19	95%	58	55%	55	80%	72	60%	46	62%	58	74%	19	40%	55	80%
14	M1	17	85%	50	48%	53	77%	67	56%	44	59%	54	69%	13	28%	53	77%
15	M2	19	95%	51	49%	51	74%	67	56%	43	58%	55	71%	15	32%	51	74%
16	M3	19	95%	42	40%	51	74%	61	51%	44	59%	50	64%	11	23%	51	74%
17	M3	19	95%	54	51%	53	77%	69	58%	44	59%	54	69%	19	40%	53	77%
18	M2	17	85%	54	51%	49	71%	68	57%	41	55%	55	71%	16	34%	49	71%
19	M1	17	85%	40	38%	49	71%	62	52%	38	51%	46	59%	11	23%	49	71%
20	P2	20	100%	57	54%	62	90%	75	63%	54	73%	59	76%	18	38%	62	90%
21	P1	20	100%	72	69%	64	93%	81	67%	59	80%	66	85%	26	55%	64	93%
22	C	19	95%	84	80%	62	90%	83	69%	59	80%	69	88%	34	72%	62	90%
23	I2	20	100%	77	73%	56	81%	81	67%	54	73%	69	88%	28	60%	56	81%
24	I1	20	100%	74	70%	53	77%	82	68%	50	68%	68	87%	26	55%	53	77%
25	I1	20	100%	74	70%	53	77%	83	69%	49	66%	68	87%	26	55%	53	77%
26	I2	20	100%	79	75%	61	88%	84	70%	57	77%	69	88%	30	64%	61	88%
27	C	20	100%	81	77%	64	93%	85	71%	58	78%	69	88%	32	68%	64	93%
28	P1	20	100%	72	69%	64	93%	80	66%	56	76%	64	82%	28	60%	64	93%
29	P2	20	100%	65	62%	65	94%	82	68%	52	70%	62	79%	23	49%	65	94%
30	M1	15	75%	40	38%	57	83%	61	51%	42	57%	42	54%	13	28%	57	83%
31	M2	18	90%	50	48%	56	81%	66	55%	48	65%	53	68%	15	32%	56	81%
32	M3	20	100%	55	52%	53	77%	69	58%	48	65%	56	72%	19	40%	53	77%

Appendix B.2 Percentage by Tooth with DEH

Tooth				All		Collection						Sex				Race					
						Terry		Hamann-Todd		Tigara		Male		Female		Black		White		Native American	
				n=194		n=105		n=20		n=69		n=120		n=74		n=78		n=47		n=69	
Tooth #	Tooth type	Side	Bone	%present	%DEH	%present	%DEH	%present	%DEH	%present	%DEH	%present	%DEH	%present	%DEH	%present	%DEH	%present	%DEH	%present	%DEH
1	M3	Right	Maxillary	34%	9%	30%	10%	65%	15%	30%	5%	38%	4%	27%	20%	51%	10%	9%	25%	30%	5%
2	M2	Right	Maxillary	41%	5%	41%	7%	85%	0%	28%	5%	44%	0%	35%	15%	67%	6%	17%	0%	28%	5%
3	M1	Right	Maxillary	40%	12%	42%	11%	75%	20%	26%	6%	41%	10%	38%	14%	64%	16%	19%	0%	26%	6%
4	P2	Right	Maxillary	38%	15%	40%	19%	80%	6%	22%	13%	41%	16%	32%	13%	63%	10%	19%	44%	22%	13%
5	P1	Right	Maxillary	48%	23%	47%	31%	80%	6%	41%	18%	51%	21%	43%	25%	65%	20%	30%	43%	41%	18%
6	C	Right	Maxillary	54%	46%	49%	43%	90%	56%	51%	46%	57%	50%	49%	39%	68%	45%	34%	50%	51%	46%
7	I2	Right	Maxillary	36%	49%	26%	56%	85%	47%	38%	42%	35%	50%	38%	46%	50%	54%	11%	40%	38%	42%
8	I1	Right	Maxillary	40%	62%	34%	75%	75%	40%	38%	58%	43%	58%	34%	72%	53%	66%	21%	60%	38%	58%
9	I1	Left	Maxillary	39%	66%	33%	74%	75%	33%	38%	73%	43%	62%	32%	75%	50%	67%	23%	45%	38%	73%
10	I2	Left	Maxillary	39%	60%	32%	62%	85%	47%	35%	67%	42%	66%	34%	48%	56%	55%	15%	71%	35%	67%
11	C	Left	Maxillary	51%	51%	43%	58%	95%	53%	49%	41%	52%	60%	49%	36%	64%	62%	30%	36%	49%	41%
12	P1	Left	Maxillary	45%	23%	42%	32%	80%	19%	41%	11%	46%	25%	45%	18%	65%	24%	19%	56%	41%	11%
13	P2	Left	Maxillary	41%	18%	38%	28%	90%	6%	32%	9%	41%	18%	42%	16%	60%	13%	23%	55%	32%	9%
14	M1	Left	Maxillary	36%	13%	36%	13%	70%	21%	26%	6%	38%	9%	32%	21%	55%	19%	19%	0%	26%	6%
15	M2	Left	Maxillary	39%	7%	34%	6%	90%	11%	30%	5%	43%	6%	32%	8%	59%	9%	17%	0%	30%	5%
16	M3	Left	Maxillary	35%	6%	27%	7%	75%	0%	36%	8%	38%	2%	31%	13%	47%	3%	13%	17%	36%	8%
17	M3	Left	Mandibular	37%	6%	36%	5%	55%	18%	32%	0%	37%	7%	36%	4%	50%	8%	21%	10%	32%	0%
18	M2	Left	Mandibular	38%	9%	36%	11%	85%	12%	28%	5%	40%	13%	35%	4%	59%	11%	19%	11%	28%	5%
19	M1	Left	Mandibular	35%	13%	32%	18%	80%	6%	26%	11%	38%	13%	30%	14%	54%	12%	17%	25%	26%	11%
20	P2	Left	Mandibular	43%	14%	42%	20%	80%	6%	33%	9%	44%	13%	41%	17%	65%	14%	19%	33%	33%	9%
21	P1	Left	Mandibular	49%	22%	54%	23%	80%	19%	32%	23%	50%	18%	47%	29%	73%	21%	34%	25%	32%	23%
22	C	Left	Mandibular	59%	62%	61%	64%	90%	44%	48%	67%	63%	65%	54%	55%	77%	60%	47%	59%	48%	67%
23	I2	Left	Mandibular	45%	43%	47%	55%	75%	20%	33%	30%	46%	40%	43%	47%	63%	51%	32%	33%	33%	30%
24	I1	Left	Mandibular	41%	40%	46%	52%	65%	31%	28%	16%	44%	36%	36%	48%	58%	47%	34%	50%	28%	16%
25	I1	Right	Mandibular	42%	32%	44%	37%	80%	31%	29%	20%	46%	29%	36%	37%	63%	35%	28%	38%	29%	20%
26	I2	Right	Mandibular	48%	44%	50%	50%	85%	18%	35%	50%	53%	39%	39%	55%	71%	42%	30%	43%	35%	50%
27	C	Right	Mandibular	51%	63%	52%	64%	75%	53%	41%	68%	54%	66%	45%	58%	69%	61%	34%	63%	41%	68%
28	P1	Right	Mandibular	49%	27%	50%	31%	95%	11%	35%	33%	54%	26%	41%	30%	71%	22%	34%	38%	35%	33%
29	P2	Right	Mandibular	44%	13%	44%	22%	90%	6%	30%	0%	50%	12%	34%	16%	64%	18%	30%	14%	30%	0%
30	M1	Right	Mandibular	35%	6%	30%	10%	80%	6%	30%	0%	42%	6%	24%	6%	51%	10%	15%	0%	30%	0%
31	M2	Right	Mandibular	39%	9%	35%	14%	85%	12%	30%	0%	39%	13%	38%	4%	56%	11%	21%	20%	30%	0%
32	M3	Right	Mandibular	37%	0%	36%	0%	75%	0%	26%	0%	38%	0%	35%	0%	58%	0%	17%	0%	26%	0%

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