VERTEBRAL NEURAL CANAL GROWTH AND DEVELOPMENTAL STRESS: A CASE STUDY FROM THE AMERICAN SOUTHWEST

by

Samuel M. LoPresto

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Committee:

_____ Director

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_____

_____

_____ Department Chairperson

_____ Dean, College of Humanities and Social Sciences

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by

Samuel M. LoPresto
Bachelor of Science
Grand Valley State University 2018

Director: Daniel Temple, Professor
Department of Sociology and Anthropology

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Fairfax, VA

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DEDICATION

This thesis is dedicated to the Zuni and Ancestral Pueblo people of the American Southwest.

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LIST OF ABBREVIATIONS AND SYMBOLS

Alpha ..... α
Analysis of Variance ..... ANOVA
Chi-squared ..... χ^2
Developmental origins of health and disease ..... DOHaD
Hypothalamic-Pituitary-Adrenal ..... HPA
Linear Enamel Hypoplasia ..... LEH
National Museum of Natural History ..... NMNH
Vertebral Neural Canal ..... VNC

ABSTRACT

VERTEBRAL NEURAL CANAL GROWTH AND DEVELOPMENTAL STRESS: A CASE STUDY FROM THE AMERICAN SOUTHWEST

Samuel M. LoPresto

George Mason University, 2020

Thesis Director: Dr. Daniel Temple

This study aims to understand the risk of mortality in relation to vertebral canal growth disruption at the Pueblo Bonito (AD 800-1200, n=47) and Hawikku (AD 1400-1680, n=102) sites in the pre- and protohistoric American Southwest. The vertebral neural canal encodes information from the intrauterine period until the age of 5 years for midsagittal diameter and adolescence for interpedicular diameter. Maximum midsagittal and interpedicular measurements of the neural canal were collected for all vertebrae. Age was estimated using transition analysis. An ANOVA with a Games-Howell post-hoc test found that reduced neural canal diameter was significantly associated with early adult mortality at Hawikku for several types of vertebrae ($P \leq 0.05$). Significant associations with early adult mortality were also found at Pueblo Bonito ($P \leq 0.05$). Additionally, survival analysis revealed decreased survivorship for Pueblo Bonito and Hawikku individuals who had smaller vertebral neural canals ($P \leq 0.05$). Results from this study support an accumulative stress model in these samples, where disrupted growth in

skeletal structures that form over extended periods of ontogeny have the highest mortality signal. In addition, trends in mortality appear contextual. Greater mortality risk in association with VNC growth disruptions is found at Pueblo Bonito compared to Hawikku. This may reflect drought related stressors at Pueblo Bonito and epidemic mortality at Hawikku. These results are consistent with the Developmental Origins of Health and Disease framework which argues that early life stress has a contextual impact on mortality at later stages of the life cycle.

CHAPTER ONE: THE THEORETICAL AND OSTEOLOGICAL STUDY OF STRESS

This thesis identifies evidence for growth disruption in the early life environment using vertebral neural canal dimensions. These dimensions are then explored within the context of mortality to understand if surviving these disruptions elicit an increased risk of mortality at later stages of the life cycle. These questions are explored in a contextually specific framework, comparing results from two sites, Pueblo Bonito and Hawikku. Pueblo Bonito was a prehistoric great house in the American Southwest, while Hawikku was a protohistoric site that existed across the time of Spanish colonialism. This chapter provides an outline for the study of stress and the ways in which these studies have been integrated into bioarchaeological research. The goal here is to provide a critical history for documenting relationships between lesion prevalence, stress experience, and eventually, using these data to explore questions related to mortality risk and life histories.

Stress Model

Stress is defined as a disruption to physiological homeostasis due to external perturbation (Goodman et al., 1988). This concept was first introduced by Walter Cannon (1932) in the study of "fight or flight" mechanisms in the physiological systems of vertebrates. The theory state animals react to threats through the engagement of the sympathetic nervous system, priming the animal for fighting or fleeing. The work

established a metabolic, cardiovascular, and adrenal response to stressors (Cannon, 1932). In addition, the concept of homeostasis was introduced as physiological conditions that exist within a relative state of constancy that vary between systems (Cannon, 1932). These important concepts established the initial physiological basis for understanding homeostasis and deviations from this relative constancy within physiological systems through parasympathetic pathways.

The Seylean model states how hormonal reactions occur as a result of a wide variety of noxious stimuli. The introduction of these noxious stimuli induce hormonal changes in associated with general adaptation syndrome. This syndrome has three stages: initial alarm, resistance, and adaptation or exhaustion (Selye, 1936). The initial alarm stage of general adaptation syndrome is related to the endocrine system. In a situation in which stress is experienced, the body activates the hypothalamic pituitary adrenal (HPA) axis that regulates the release of cortisol and adrenaline (Seyle, 1936). In the short-term cortisol, is an extremely useful hormone that accelerates the metabolism of fat, protein, and carbohydrates through glycogenesis in the vascular system to meet increased metabolic demand while producing short-term muscular boosts as part of the fight or flight response through the excretion of epinephrine and norepinephrine. (Cannon, 1932; Goodman et al., 1988; Jiménez, Aguilar, & Alvero-Cruz, 2012).

The second stage of the Selyean model is resistance. In this stage the body achieves homeostasis by returning to hormonal levels that existed before the stress event. This return to homeostasis includes a reduction in the production of cortisol, epinephrine, and norepinephrine (Selye, 1936). Though reduced, cortisol is still continuously released,

which may suppress immune system functionality and cellular growth. Continuous release of cortisol can lead to the depression of osteoblastic activity. The long term effect of cortisol can also lead to a variety of functional disorders, including cardiovascular disease, ulcers, and hypertension (Goodman et al., 1988; Selye, 1950). These functional disorders contribute to exhaustion, the final stage of the Selyean model.

The Selyean stress model eloquently described the physiological response to stressors. However, clinical and epidemiological approaches to stress still relied on unempirical etiological models, rather than understanding these experiences in social and environmental context. One initial attempt to incorporate an environmental model compared blood pressure in Kenyan and British populations (Donnison, 1929). Lower blood pressure was found in the Kenyan samples and attributed to the long-term persistence of pastoral economies versus the constantly transforming nature of Western industrial capitalism. Later work built on this model by comparing health across samples from three environments: agricultural workers, first generation factory employees, and second and third generation factor employees (Cassel et al., 1960). First-generation workers that experienced the most cultural change and had less family solidarity had poorer health then the other more social connected and culturally familiar groups. This work was one of the first to demonstrate the importance of considering cultural and environmental factors into long-term analysis of stress and disease.

Years later biological anthropological research began to integrate society and culture as influential factors that effected the manifestation of biological stress. This new system considered the environmental and cultural constraints that affected the activation

of the stress response system (Goodman & Martin, 1984) Examples of environmental constraints include lack of pathogen-free drinking water, lack of arable land, unequal access to food, water, shelter, climatic variables, parasites, and predators (Klaus et al., 2017). The cultural inducers of stress involve differential access to resources, social hierarchies, inequalities, faulty cultural buffering mechanisms, and sociopolitical structures. These stressors can be offset by cultural systems. However as indicated by Goodman (1984), cultural buffering systems can also exist simultaneously as a cause and deterrent against stress. One instance of the negative impacts of culture in terms of stress manifestation is in hierarchal societies where symptoms of biological stress are often magnified because of social stratification that resulted in an unequal allocation of resources and structural inequalities. This is the phenomenon known as structural violence (Farmer, 2005; Klaus, 2012; Watkins, 2007).

Once the external stressor has been introduced, from any number of ecological or cultural origins, there are still a variety of factors that affect the manifestation of stress in human populations. These factors include population genetics, individual genetics, and behavioral alteration (Goodman & Martin, 1984). All of these factors affect host resistance to manifesting evidence of stress to any particular degree. Stress can appear in the form of growth disruptions, increased susceptibility to disease, or death (Goodman & Martin, 1984). Stress acts on the individual, but that has consequences on a populational scale. These population outcomes often appear as increased mortality, reduced labor capacity, suppressed reproduction, and sociocultural disruptions that further magnify stress (Klaus, 2012). The result of this process is often biological or behavioral

adjustments to mediate the challenges caused by these stressors. The introduction and eventual manifestation of stress involves an interactive web spanning a multitude of different factors that all combine to affect the individual, which has eventual consequences for the population (Goodman & Martin, 1984)

At the same time, clinical and experimental research integrated a lifespan perspective to understanding the impacts of stress and inequality. Results from the Dutch famine found that individuals who survived at relatively young ages were at a greater risk of cardiovascular disease later in life (Roseboom et al., 2000). This work built on previous studies that hinted at the possibility that early life adversity may result in future mortality risk (Boas, 1930; Diggs, 1986). This work was followed by observations recorded from more than 13,000 death records from impoverished sections of the United Kingdom suggesting a link between cardiovascular disease and socioeconomic status (Barker & Osmond, 1986). Using notes collected from midwife Ethel Margaret Burnside, direct associations between birth weight, cardiovascular disease, and respiratory ailments were found (Barker and Osmond, 1986). The totality of these findings contributed to the Developmental Origins of Health and Disease paradigm (DOHaD), a quasi-academic discipline focused on clinical, experimental, and ethnohistoric approaches to understanding how health at later ages is rooted in developmental experience.

The DOHaD hypothesis is most clearly defined as: “associations between aggressions suffered during the initial phases of somatic development and amplified risk of chronic diseases throughout life, such as obesity, diabetes and cardiovascular diseases” (Silveira et al., 2007, p. 494). The main point of the theory looks at how issues in

development effect the individual throughout life in the form of adaptive plasticity and subsequent physiological constraints. Adaptive plasticity is defined as “reaction norm that results in the production of a phenotype that is in the same direction as the optimal value favored by selection in the new environment” (Ghalambor et al., 2007, p. 395) Early maturation in amphibians from desiccating environments provides an example of adaptive plasticity as faster maturation allows organisms to reproduce following survival of early life adversity (Crespi & Denver, 2005).There are limits placed on the capacity for survival at later stages of life of in the form of physiological constraints. These physiological constraints represent the alteration of energy to interacting systems (Partridge et al,, 1991). For example, frogs who experience early life adversity and reproduce at earlier ages have an increased risk of delayed stunted growth and early mortality, suggesting a trade-off between energy invested in reproduction, growth, and survival (Crespi & Denver, 2005). Adaptive plasticity that gave rise early maturation of the amphibians is limited by subsequent physiological constraints on growth and survival. The tradeoffs between adaptive plasticity and physiological constraints can explain the relationship between growth stunting and mortality: individuals survive adversity through reallocation of energetic reserves to essential tissue growth and function but are at an increased risk of mortality later in life. Additionally, organisms that do not reallocate resources may not survive due to an inability to adequately buffer against environmental pressures.

The Osteological Paradox

Bioarchaeology is the integrative and interdisciplinary study of human remains from archaeological contexts (Buikstra, 1977; Larsen, 2015). The history of bioarchaeology can be traced to the integration of methods associated with skeletal biology to questions and theoretical constructs developed by processual archaeologists and cultural ecologists (Armelagos, 2003). Interaction between these fields empowered an approach that privileged questions centered around adaptive transitions such as the adoption of agriculture and European colonization (Boyd & Boyd, 1989; Cohen et al., 1984; Goodman, Armelagos, & Rose, 1980; Larsen, 1995; Rathbun, 1987; Ruff et al., 1984; Wright, 1990). Using this metric, the prevalence of skeletal indicators of stress was used to compare lived experiences between populations. Examples of this are numerous in 20th century bioarcheological literature (Temple and Goodman, 2014). Studies would compare populations using statistical analysis of the prevalence of one stress indicator across both groups. Interpretations of results implied a direct relationship between the prevalence of skeletal indicators of stress in a sample and population health. However, the use of lesion prevalence in direct relationship to heath is problematic.

The authors of the Osteological Paradox (Wood et al., 1992) illuminate fallacy by introducing the concepts of hidden heterogeneity, selective mortality, and demographic nonstationary. Hidden heterogeneity refers to the fact that a population is made of an unknown mixture of individuals who varied in susceptibility to disease and death, and this may be independent of prevalence of skeletal lesions. This variation is similar to the one observed in the biological stress model in the sense that one's genetics,

socioeconomic class, and individual frailty all contribute risk of mortality (Goodman et al., 1988; Goodman & Martin, 1984; Klaus et al., 2017; Wood et al., 1992). Two individuals can live in the exact same environment and have drastically different responses to the introduction of a pathogen or instances of famine. Selective mortality references the underlying conditions that increase mortality risk among individuals. Early life stress is an example of a contributor to selective mortality, as these events weaken future responses to disease and stress. Demographic non-stationarity references fluctuations in population size due to fertility that alter the risk of death (Wood et al., 1992). Bioarchaeological research has methods to estimate fertility (Sattenspiel and Harpending, 1983; Buikstra et al., 1986), but these patterns have not yet been incorporated into questions of demographic non-stationarity.

The publication of the paradox elicited a variety of responses that ranged from praise to intense critique (DeWitte and Stojanowski, 2015). Some authors suggested that the Osteological Paradox reinterpreted lesion as meaning good health (Cohen, 1994; Goodman 1993). Other authors stated that the Osteological Paradox was rendered invalid because of contextual evidence about declining health in in association with the transition to agriculture (Cohen, 1994; Goodman 1992). One of the main critiques of the Osteological Paradox was the assumption that populations are homogenous in risk of death These new interpretations were based on a fundamentally misunderstanding of the key assertion made by the Osteological Paradox. The appearance of lesions does not exist in a binary: good health or bad health is not the focus of the Osteological Paradox. Instead, the presence of skeletal lesions represents a multitude of different responses to a

variety of factors influencing health, all of which are heavily based within the environmental and biological context of the population (Wood & Milner, 1994).

The Osteological Paradox points out systemic problems in bioarcheological investigations. First, it is unwise to compare stress experiences from lesion prevalence alone. Second, the prevalence of lesions may represent different information then orginally perceived. The authors of the Osteological Paradox suggest new methods that compare population health using survivor modeling. This still fails to transcend the comparative approach of bioarchaeological research that seeks to categorize populations according to levels of health (Temple & Goodman, 2014; Temple, 2019). Despite the continuing presence of the comparative approach the Osteological Paradox still brings attention to various potential shortcomings in linking complexities between health, survival, and lesion formation (DeWitte & Stojanowski, 2015).

When considering the stressors that effect a population, it is important to keep in mind that skeletal lesions are not analogous to health. Previously the field of bioarchaeology relied on the assumption that populations could be compared for health based on the appearance and frequency of skeletal lesions (Cohen, 1984; Larsen, 1995). That is a rather reductionist view that ignores the factors that make up health. Instead, it is more accurate to fathom health as a multifaceted system that is made up of information about well-being, mortality, basic functioning. and community interactions which all interrelate with the evolutionary and genetic make-up of individuals (Reitsema & McIlvaine, 2014; Temple & Goodman, 2014). Instead, biological anthropologists study

biological stress, allostatic oad, and the disruption of physiology of the skeletal system (Klaus et al., 2017).

Life History in Bioarcheology

The authors of the Osteological Paradox and the subsequent literature surrounding the topic have made it unequivocally obvious that looking at stress indicators for prevalence to make statements about health is problematic (DeWitte & Stojanowski, 2015). The Osteological Paradox provided the methodological background to study relationships between lesions that form in the early life environment and subsequent mortality risk . These new methods include morality risk that is associated with early life stress as elucidated by dental lesions, chronic infections, diminished body size, and the comparisons of survivors and non-survivors (Temple, 2019). Through the application of life history theory these methods can be used to elucidate the relationship between skeletal lesions and mortality. Life history theory, within biological anthropology, studies the interaction between growth, reproduction, and survival across an organism's life cycle. The DOHaD hypothesis can be applied to bioarcheology by studying skeletal indicators of early life adversity that results in reduced growth and survivorship at later stages of the life courses (Temple, 2019).

The concept of adaptive plasticity in reference to bioarcheology emphasizes short-term investments in survival, while physiological constraint reflects long-term trade-offs for this investment (Temple, 2019). The application of the DOHaD hypothesis in bioarcheology is restricted, because of the lack of expansive longitudinal data that can be elucidated from skeletal remains. However, careful scrutiny of specific skeletal and

dental stress indicators can preserve evidence for early life adversity where individuals survive (Temple, 2019). Dental and skeletal indicators of stress such as liner enamel hypoplasia, periosteal lesions, cribra orbitalia, and porotic hyperostosis preserve evidence of stress events experienced early in the life of an individual that continued to live. In addition, morbidity and mortality at later stages of the life cycle provide a window into physiological constraints associated with surviving early life stress. Therefore, in order to evaluate physiological constraints, bioarcheologist test the relationship between indicators of stress and mortality.

Previous research on the relationship between stress and mortality have been applied to a variety of different archeological samples. A study involving individuals from Medieval London found that those with active periosteal lesions had the lowest estimated survival (DeWitte, 2014). Another study found that individuals with cribra orbitalia had a higher mortality then those without evidence of this lesion (Benus et al., 2010). Individuals with evidence of LEH during a period of famine exhibited higher frailty which correlated with an increased risk of death (Yaussy et al., 2016). Other studies have found similar results when evaluating LEH and mortality (Boldsen, 2007; Cook & Buikstra, 1979; Miliauskienė, 2001). All of these investigations demonstrate how bioarchaeological research has the capacity to identify early life adversity and explore relationships with physiological constraints that may increase risk of morbidity and mortality at later ages.

As stated previously, bioarcheology is the contextual study of humans remains. As such, the relationship between early life adversity and later outcomes is not always

clear because of the mitigating effects of social and ecological contexts. An analysis of a Greek colony in modern day Sicily found LEH to be inconsistent in its relationship to mortality. However, through the contextual analysis of burial data it became clear that there was differential treatment of the solider class in society that buffered them against skeletal indicators of stress (Kyle et al., 2018). An additional study on LEH found that when contextual data such as cause of death, year of birth, and socioeconomic status was included in the analysis the significant relationship between mortality was reduced (Amoroso et al., 2014). Research on crypt fenestration enamel defects in colonial Peru found no differences in mortality between individuals with and without defects. The lack of differences is likely a result of colonial epidemics that killed individuals regardless of survival to early life stress (Thomas et al., 2019). Analysis of a 20th century Portuguese sample in Lisbon revealed no increase in mortality even though there was an upsurge in prevalence and severity of porotic hyperostosis and cribra orbitalia. The authors attributed these skeletal indicators of stress to socioeconomic processes and increased urbanization resulting in water-borne parasites that contributed to iron deficient anemia, but did not increase mortality (Hens et al., 2019). Finally, bioarchaeological studies reveal that stress experienced during critical phases of the early life cycle increase risk of mortality and growth disruption at future ages (Temple, 2014; Lorentz et al., 2019; Garland, 2020). All of these studies reveal that there is a relationship between early life stress that is contingent on environmental and cultural contextual data (Temple, 2019).

Conclusion

The study of stress has a long history in clinical, experimental, and anthropological research. Early experimental work emphasized a phased response to noxious stimuli that increased risk of morbidity and mortality. Epidemiological and anthropological work found links between these experiences with environmental and social context. Lifespan approaches demonstrated a strong relationship between early life adversity with morbidity and mortality, while endocrinological work has argued for an evolutionary perspective that unites these findings under a life history perspective.

Bioarchaeological research explored stress in the past by comparing lesion prevalence between populations. This approach was challenged by arguments stating that lesion prevalence did not represent population health and that mortality risk was crucial to better understanding stress experience in the past and is now referenced under the broader title, The Osteological Paradox. This approach introduced new methods to evaluate stress in the past but did not move bioarchaeology beyond a binary approach to stress as healthy/unhealthy. The application of life history theory provides bioarcheologists with the ability to explore questions surrounding stress under a unified evolutionary framework while leveraging archaeological context.

CHAPTER TWO: THE STUDY OF VERTEBRAL NEURAL CANALS IN BIOARCHAEOLOGY: EMBRYONIC ORIGIN, PATHOLOGICAL CONDITIONS, AND INDICATORS OF STRESS

This study uses size of the vertebral neural canal (VNC) as an indicator of early life adversity. This chapter outlines the embryological origins of the VNC and provides a clinical perspective on tracking of this hard tissue indicator of growth disruption in concert with crucial organs. In addition, this chapter describes the results of bioarchaeological research that explores the relationship between VNC growth disruptions and mortality, while discussing VNC measurements that have the strongest relationship with life span. Finally, recent studies that advocate for a contextual understanding of VNC growth disruptions and mortality are introduced. The goal of the chapter is to develop a set of expectations for VNC growth disruptions, prior to building a set of contextual hypotheses in the subsequent chapter.

Vertebral Canal Development

The embryonic origin of the paired vertebral column occurs in mesenchymal tissue columns that extend on either side of the notochord (Aoyama & Aamito, 1988; Barnes, 2012; Scaal, 2016). The columns then segment into paired blocks of tissues known as somites, which differentiate into tissues that surround the notochord. The

progenitor of the vertebral body, the sclerotome, then forms from these masses of somatic tissues. The blocks of sclerotome somatic tissue surround the notochord and the neural tube that lies within the cord. The condensed blocks of tissues then develop into the caudal and cranial portions to form the final tissue blocks. The ventral medial portions form the centra of the vertebral bodies and the dorsal lateral portions are then directed to form the transverse process and the neural arches of the still developing spinal cord. Absent of any stressful events, there are still a multitude of errors that occur in the embryonic development around the vertebral canal. Spinia bifida, which is a birth defect that occurs when the spinal cord and sacrum don't form properly. is a common disease that occurs from a lack of folate when the fetus is developing (Barnes, 1994; Mitchell et al., 2004). While spina bifida only effect the sacrum, it is possible that this could produce some sort of effect on the neural canals of the other vertebrae as well.

The last step of the development into mature vertebrae involves the developing of the intervertebral disks from previous notochord tissues, followed by retreat of the notochord tissue. Genetic signals then direct differentiation of the segments and borders of the vertebral column. The cervical vertebrae derive from eight sclerotomes, with the first sclerotome being partially incorporated with occipital sclerotome to form the first cervical vertebrae. The eighth cervical sclerotome contributes to both the seventh cervical vertebra and the first thoracic vertebra by segmenting into two different portions. The formation of the sclerotomes and the resegmentation proceeds inferiorly through the developing vertebral column and terminates with the formation of the coccyx. The formation of the vertebra occurs at different stages *in utero* depending on the element.

However, in general the growth of the vertebral spine is most rapid from 18-36 weeks across all levels of the spine, the most sensitive period of VNC growth during fetal development. (Porter, 1998).

At birth all primary centers of ossification are present, except the distal coccygeal segments (Porter, 1998). Postnatally the vertebra exists as three separate entities, one paired set of neural arches and a still developing body segment. Around the age of 3-4 years the neurocentral fusion has occurred in C3-C7 and all of the thoracic and lumbar vertebrae. C1 and C2 finish fusing around the age of 5-6 years (Schaefer et al., 2009). Once the vertebrae have fused, the neural canal is relatively fixed in size, and often does not significantly remodel for the remainder of life. This stagnation of remodeling makes the VNC useful for elucidating growth disruption in development. The one clear exception to that statement is the C1, or the atlas, that seems to remodel and grow more sporadically opposed to the other vertebra (Tulsi, 1971). Since the cessation of VNC growth occurs at a young age, it is easier to compare the vertebrae among a sample because of its relatively unchanging nature. In that sense, a smaller vertebral canal in adulthood may correspond to a stress experienced in the early life environment as the VNC does not remodel at later ages.

When it comes to the neural canal of the lumbar vertebrae. it is generally accepted that 70% of growth is completed *in utero* (Clark et al., 1986; Watts, 2011, 2013). Clark et al. (1986) found that neural canal growth generally ceases by four years of age. Other research on both historic and modern samples have agreed by finding that "vertebral foramen dimensions, do not alter significantly with individual age" (Rühli et al., 2005, p.

463). Other portions of the vertebrae are sensitive to age related change such as the diameter of the body and the height of the pedicle. In addition, some studies have noted variability in the growth of the VNC. For example, midsagittal diameter the size of the cervical canals for individuals between 2-4 years of age were 95% of the adult size, and the thoracic canals were 90% of the adult size (Tulsi, 1971). The lumbar vertebrae were found not to be different for the midsagittal diameter between the ages of 2-4 years and adult. This aligns with other work that has been done on midsagittal diameter that states that the VNC does not continue to grow after 3-5.9 years (Watts, 2013a). By contrast, the interpedicular diameter may experiences a pattern of more continuous growth. Early studies found that the transverse or interpedicular diameter does not attain full size until adolescence. The transverse diameter of the neural canal follows the craniocaudal sequence of maturation, with the cervical, thoracic, and lumbar vertebrae being at 90%, 88%, and 83% adult size by the age of 2-4 years respectively (Tulsi, 1971). This once again supports more recent studies that show interpedicular diameter continues growth through approximately 17 years of age. Other analysis suggested that 85% to 91% of adult interpedicular diameter was attained by three years of age (Newman and Gowland, 2015). The authors suggest that there are no changes in VNC diameter at later ages. Instead differences after three years of age are associated with greater frailty among individuals who experienced growth disruptions of the VNC. The authors go on to state that remodeling is substantially reduced after the first three years of life and changes in interpedicular diameter are insubstantial past this age. Regardless, comparisons of the transverse diameter are still possible, but these measurements need to be considered

within the conceptual model of accumulative stress burdens rather than as evidence for early life adversity. The cessation of growth at a later age, if true, could be of benefit for studies because it captures later periods of development up until adolescence. These findings provide evidence into the window of development for VNC dimensions and suggest that the measurements track growth in the early life environment as well as cumulative hard tissue growth through adolescence.

In consideration of biological sex, it is necessary to note that the majority of research on the VNC has been focused on changes prior to sexual maturity (Newman & Gowland, 2015; Wizemann & Pardue, 2001). *In utero*, there is no known sexual dimorphism present in the vertebrae, even after the activation of sex hormones (Szpinda et al., 2015; Szpinda et al., 2013; Wizemann & Pardue, 2001). The only possible exception to that comes from the transverse lumbar vertebral canal which continues to expand until the beginning of adolescence. Therefore, it is technically possible that the adolescent growth spurt would affect the terminal size of the transverse diameter. That said, there are no consistent signficant differences in the size of the canal of the vertebral canal between male and female (Amoroso & Garcia, 2018; Clark et al., 1986; Watts, 2011). It is not impossible that future research will reveal sexual dimorphic morphology of the vertebral neural canal. While sex could be included as a variable for differential development, there is currently nothing to suggest that it is a factor that effects the growth of the vertebral neural canal.

Inferring Stress from Vertebral Neural Canal Growth

There are numerous pathological conditions that involve vertebrae, all of which have multiple etiological pathways. One of the most common pathological conditions that affect the vertebrae through the creation of osteophytes that can result in the ankylosis of the vertebrae is osteoarthritis (Buzon, 2012; Larsen, 2015). The vertebrae are also sensitive to trauma such as compression fractures that cause herniations of the disk and depressions on the body of the vertebrae (Larsen, 2015). Infectious diseases such as tuberculosis can produce necrosis of the cartilaginous vertebral disk that can later cause vertebral compression fractures (Pott's disease), resulting in the complete collapse of one or multiple segments of the vertebral column (Spekker et al., 2018). All of these conditions have pathological origins that act as additional factors when looking at morbidity associated with vertebrae. None of these conditions are directly caused by smaller VNCs or growth disruptions, but if an individual is also afflicted by one of these then it would be fair to consider these conditions as contributing to comorbidity. However, due to the nature of nonspecific indicators this relationship would be difficult to determine.

In regard to morbidity, research has demonstrated that individuals with smaller vertebral neural canals have an increased risk of mortality (Amoroso & Garcia, 2018; Clark et al., 1986; Newman & Gowland, 2015; Watts, 2011, 2013). Early research into this subject explored question surrounding VNC growth using meta-data derived from the clinical literature (Porter et al., 1980) One study that reviewed ultrasound images of 700 patients found that there was a correlation between smaller neural canals and the presence

of chronic pain (Porter et al., 1980). During the study of chronic pain, the authors also found that occupation seems to have no effect on the size and or shape of the vertebrae, reconfirming that the vertebral canal is unaffected by activity markers.

One clinically defined condition that is related to VNC size is spinal stenosis. This is a congenital disease that results in the narrowing of the spinal cord (Waldman, 2019). Clinically spinal stenosis manifests as pain and weakness in the legs when walking. Additional symptoms may also appear as numbness and loss of reflexes in the legs (Waldman, 2019). This disease can additionally be impacted by smaller neural canals. A correlation was found between living individuals with smaller midsagittal neural canals and chronic lower back pain (Visuri et al., 2005). Besides just general localized pain there has been research that also connects directly with information about the general size of the neural canal. One study has found that smaller VNCs correlate with a variety of other conditions. Individuals with smaller VNCs were at greater risk of having gastrointestinal and cardiovascular diseases (Porter,1998). There is even evidence to suggest that there is a connection between the canal size and neurocognitive function (Porter, 1998). While this is possibly be an oversimplification of a complex multifaceted system, these results support relationships between VNC size, health across the lifespan, and possible impairment of cognitive function.

Health consequences that effect soft tissue development can be viewed via proxy through the study of the vertebral neural canal as a nonspecific indicator of stress. This relationship existed because of the alignment of VNC growth and the development of the thymolymphatic tissues. Both of these systems have a similar growth curve that may be

inhibited by cortisol that is relased as a result of the HPA-axis (Clark et al., 1986). The suppression of thymus growth has considerable effects for the rest of the body due to the underlying relationships between hormones and the lymphatic system in general (Pierpaoli & Sorkin, 1972). This is highly consequential for the development of T-cells, also known as T- lymphocytes. These cells are one of the two lymphocytes that make up the immune system, the other being B cells (Janeway et al., 2001). T-cells are formed in the medullary cavity of bones from stem cells, and then migrate to the thymus for maturation (Janeway, Travers, Walport, & Shlomchik, 2001). Additionally, as one matures formation of new T-cells slows down and occurs from the division of mature cells, therefore maturation in the thymus occurs at a critical moment and can have lifelong consequence if improper development occurs (Janeway et al., 2001).In other words, VNC growth disruptions may provide a useful window into disruptions of physiological systems that have innate relationships with maintenance and survival. Thus, it is clear that VNC size is an avatar for other systems in the body.

Vertebral Neural Canal Growth in Bioarchaeological Research

The first step in the study of VNC diameter in bioarchaeological research is to define growth a growth disruption. The term "growth disruption" is used in VNC literature to refer to any alteration of the size of the canal (Clark et al., 1986, Newman and Gowland 2015; Watts 2011; 2013a; 2013b). Some authors suggest that VNC diameters below the sample mean are used as an indication of a growth disruption (Clark et al., 1986; Watts 2011; 2013a; 2013b), while others compare the subadult to the adult VNC diameter (Newman & Gowland, 2015). Some authors have discovered a range of

measurements for certain populations. According to (Einstein, 1983) the normal variation of lumbar AP diameters is between 12 mm and 22 mm, and 19 mm and 32 mm for TR diameters in European populations. Other studies have modeled clinical literature by sorting vertebral canal measurements by standard deviation and judging categories on a spectrum of narrow to large (Beauchesne et al., 2019). Each method has advantages, and there exists no singular criterion to identify growth disruptions in VNC diameter. It is necessary to note that the exact mechanism behind the growth of the VNC is still being studied. Defining exactly what is considered a disruption is difficult because of a lack of literature on the development of the VNC.

Studies of VNC diameter in bioarchaeology date to research emphasizing health during the transition to agriculture (Clark et al., 1986). This work begins with a physiological justification that tethers VNC growth disruption to the HPA-axis. Individuals with elevated HPA-axis induction experience growth stunting and comparisons of VNC dimensions were used to explain stress during the transition to agriculture in North America. The individuals that were divided in this study (n=90) came from Dickinson Mound (AD 950-1300). VNC were measured for all thoracic and lumbar vertebrae. The individuals were split up into age cohorts of 10-year intervals starting at the age of 15 years and ending at 55 years. The authors of this study used a two-tailed T test to compare groups above and below the mean to assess growth disruptions for VNC diameters between age cohorts. Individuals in the 15-25 years age groups had significantly smaller VNC for the interpedicular measurement of the lumbar vertebrae ($P < 0.05$). While, individuals in the 15-25 years and 25-35 years age group had

significant smaller midsagittal diameters. The authors of this study conclude that "Individuals with small VNC TR and AP diameters also have a significantly reduced life-span" (Clark et al., 1986, p. 158). While this work provided groundbreaking evidence that VNC size could be used as a proxy for stress and lifespan, these measurements were not incorporated by future studies.

VNC research and stress experience in bioarchaeology was resurrected by Watts (2011). This work evaluates the possible link between neural canals, growth disruptions, stature, and age at death. The sample was excavated from the Fisher Gate House cemetery in Northern England. The main period of cemetery dated between the from the 10th to 15th century. There were 104 individuals in the sample, but for various exclusionary reasons only 47 were included. The individuals were grouped into age cohorts of 17-25 years, 26-35 years, 36-45 years, and 45+ years. Small transverse neural canal diameters of the thoracic and lumbar vertebrae were significantly associated with a younger age at death when compared to diameters above the mean. The association was greater for females than males even though it was found that there was not a statistical difference between male and female VNC. These findings added support to the idea that smaller neural canal may track internal physiological disruptions that reduce survival probability at later stages of the life course.

Previous literature has found that the VNC reaches maximum size around the age of five years. However, this possibility has been challenged by recent studies (Watts, 2013a). Measurements of the VNC were taken from 65 subadults that were 3–17 years and compared to 120 adults. The individuals of study originated from the East Smithfield

Black Death cemetery, in London, England (AD 1348–1350). Children were grouped into age cohorts of 3–5 years; 6–10 years; 11–14 years; 15–17 years and compared with adults. The midsagittal diameters of the subadult were not significantly different from adults in any age. Interpedicular diameters increased with age until 15–17 years when adult size was attained. The results of this study called into question previous research that operated under the conclusion that interpedicular VNC diameter reached full size by five years. It is necessary to note that this study was based on only one sample that originated from a Black Death cemetery in England, so it is that epidemic mortality influenced size differences between adults and subadults that may have contributed to frailty. This would suggest that this did not reflect the extended growth processes observed in interpedicular diameter.

Another article by Watts (2013b) studied stress that was elucidated by LEH and VNC size. The individuals for this study came from two North England cemeteries dating between AD 1150-1700 and AD 1700-1855. Smaller interpedicular diameter of the VNC was associated with an early age at death. Smaller midsagittal diameter of the VNC were also associated with age at death. Notably, the interpedicular diameter had twice as many signficant cases then the midsagittal diameter. Linear enamel hypoplasia was also analyzed in association with age at death; however, no significant results were found. This suggests that VNC and LEH are formed through different developmental pathways, and therefore, express different relationships with physiological constraints at later stages of the life course.

An additional study on the VNC by Newman and Gowland (2015) found similar results to previous studies. VNC diameters were compared in a study of individuals from two post-medieval sites in England (AD 1816-1856). This work focused on interpedicular diameter. The sample size was 40 adult skeletons and 96 subadults. In contrast to previous studies, the authors analyzed the results by creating vertebral growth profiles based on the ages. The results of these studies found evidence of growth disruption between the sites. Additionally, interpedicular VNC size was associated with an early age at death.

Finally, VNC diameter and mortality was explored between individuals with documented occupation, year of birth, and cause of death in a sample from Lisbon Portugal (Amoroso & Garcia, 2018). These remains originated from a cemetery dated from AD 1805 to 1970 (Amoroso & Garcia, 2018). The authors proposed two different hypotheses that were both related to the outcome of the relationship between neural canal and body size. The first hypothesis was that neural canal should have a negative relationship with mortality. The second hypothesis was that there was no connection found between the neural canal and age at death. Hazards modeling was used to assess the effect of VNC size on age-at-death. Significant results were found only in six thoracic vertebrae and one cervical vertebra. However, when including the covariates such as birth date and the cause of death, the significant mortality hazards related to VNC stunting disappears. These results suggest that factors other than growth disruption were associated with mortality in these samples. In this sense, mortality risk appears to be contextually dependent among individuals who experience growth stunting of the VNC.

Hypothesis

The relevant research on the VNC has indicated the connection between growth disruptions and mortality. Instances of smaller VNC are correlated with decreased survival and increased mortality. The goal of this study is to evaluate the association between VNC growth disruptions and mortality risk in two samples from the prehistoric and protohistoric American Southwest. This study predicts a general pattern of elevated mortality risk among individuals with VNC growth disruptions. However, it is important to note that these relationships may be context dependent as the two sites reflect varied socioecological and economic contexts (see Chapter 3).

Conclusion

The literature surrounding the VNC and the use of it as an indicator of stress is still developing within bioarchaeology. The clinical literature is clear that there is a correlation between the thymus gland development and early life stress. If there are instances of increased cortisol, then the thymus does not develop properly causing decreased functions of the immune system. Growth curves of the thymus and relationships with t-cell development suggest that VNC growth may be a useful indicator of stress experienced during the formation of these crucial systems. Bioarchaeological research is beginning to demonstrate relationships between VNC growth disruptions and mortality. These relationships must be carefully interpreted within the archaeological context of each sample, though general trends point towards this measurement as a useful proxy for early life environment. Such findings allow for the formulation of hypotheses predicting a general pattern of increased mortality related to smaller VNC in the samples

chosen by this thesis. Archaeological context will, however, be important to carefully interpreting these trends.

CHAPTER THREE: BIOCULTURUAL CONTEXT AND MATERIALS: THE PUEBLO BONTIO AND HAWIKKU

Introduction

Indigenous occupation of the American Southwest represents some of the most culturally diverse and simultaneously ecologically challenging settings of human occupation in North America. The "Chaco Phenomenon" was one of the most elaborate cultural systems in this region. The cultural occupation of this region is associated with the Ancestral Pueblo and these population thrived over a 40,000 square mile region in the northwestern quarter of New Mexico as well as parts of modern-day Arizona, Colorado, and Utah (Figure 1) (Pepper, 1996). Another expansive group that lived in the southwest after the decline of the Ancestral Pueblo was the Zuni people. Zuni people who have lived in this area for more than 1,500 years, are descendant from the Puebloan people that lived in a similar area.

The individuals included in this study originated from the Pueblo Bonito (AD 800-1200) and Hawikku (AD 1400-1680) and are currently curated by the National Museum of Natural History of the Smithsonian Institution (NMNH) (Hrdlička, 1931). The Pueblo Bonito and Hawikku were excavated by Neil Judd and Fredrick Webb Hodge respectively in the early 20th century (Judd & Allen, 1954; Smith et al., 1966). The

Ancestral Pueblo of the Pueblo Bonito and the Zuni of the Hawikku were of interest to this study because of contextual orientation. The two samples have similarities in the form of settlements, subsistence, and location, but differ dramatically when historical context. The Pueblo Bonito represents a prehistoric population that once existed as a center for the Ancestral Pueblo people (Kennett et al., 2017). Although the Pueblo Bonito existed at a time prior to contact, archaeological mortuary evidence recovered from the site suggests a strong delineation of identities which may have resulted in structural violence (see below) (Kennett et al., 2017a). The Zuni Hawikku are affiliated with the protohistoric period, and thus, diseases and epidemics introduced by European colonialists could have affected longevity (Stodder, 1990). The relationship between VNC growth disruptions and mortality will be studied against this contextual backdrop to understand 1) the interaction of early life adversity and mortality in the pre-contact world of the American Southwest and 2) if European colonialism exacerbated mortality risk following survival of these growth disruptions.

The Ancestral Pueblo civilization is split into four different time periods, Pueblo I (AD 750-900), Pueblo II (AD 900-1150), Pueblo III (AD 1150-1350) Pueblo IV (AD 1500-1600). These periods are each categorized by different socioecological systems. The first period is classified by residential pueblo structures organized in a crescent or straight row known as the jacal style. Pueblo II is classified by buildings made of stone and mortar with kivas, tower building, water conserving dams, milling bins, and maize processing. Pueblo III is classified by cliff dwelling, multi-storied, talus house communities. Finally, Pueblo IV is defined by migration away from the Pueblo Bonito

into a different area near the Pecos river in the Rio Grande Valleys (Sutton, 2011). During the most prosperous times the people of the Chaco Canyon built a series of great houses. The site of Pueblo Bonito, perhaps the largest of the great houses, stood at around four to five stories and was surrounded by several other houses such as Pueblo Alto, Chetro Ketl, Pueblo del Arroyo, and Kin Kletso (Lekson, 2006) According to dendrochronological data the Pueblo Bonito site was occupied between the first and second periods (AD 850 to 1140) (Crown & Wills, 2018; Windes & Ford, 1996). However, these time periods can be split further into the Early Bonito (AD 840 to 1040), Classic Bonito (AD 1040 to 1110) and finally the Late Bonito Phase (AD 1090 to 1140). The main period for the construction of the kiva occurred between AD 1040-1110 (Windes & Ford, 1996).

History of Excavations at The Pueblo Bonito

The site of Pueblo Bonito is located near the lower portion of the Chaco Canyon in a 15-mile section of the Chaco River. In modern terms the site is located in San Juan County in northwestern New Mexico (Figure 1) (Crown, 2016; Judd & Allen, 1954; Pepper, 1996). Over the years there have been several excavations of the site. The largest and most robust of the excavations occurred in the 1890s and the 1920s by George H. Pepper and Neil Judd respectively (Judd & Allen, 1954; Pepper, 1996). The excavations by Pepper and Judd identified a total of 131 individuals of various ages, biological sexes, and affiliations (Akins, 2003; Judd & Allen, 1954; Pepper, 1996). The excavation by Pepper was part of the Hyde expedition to the American Southwest.

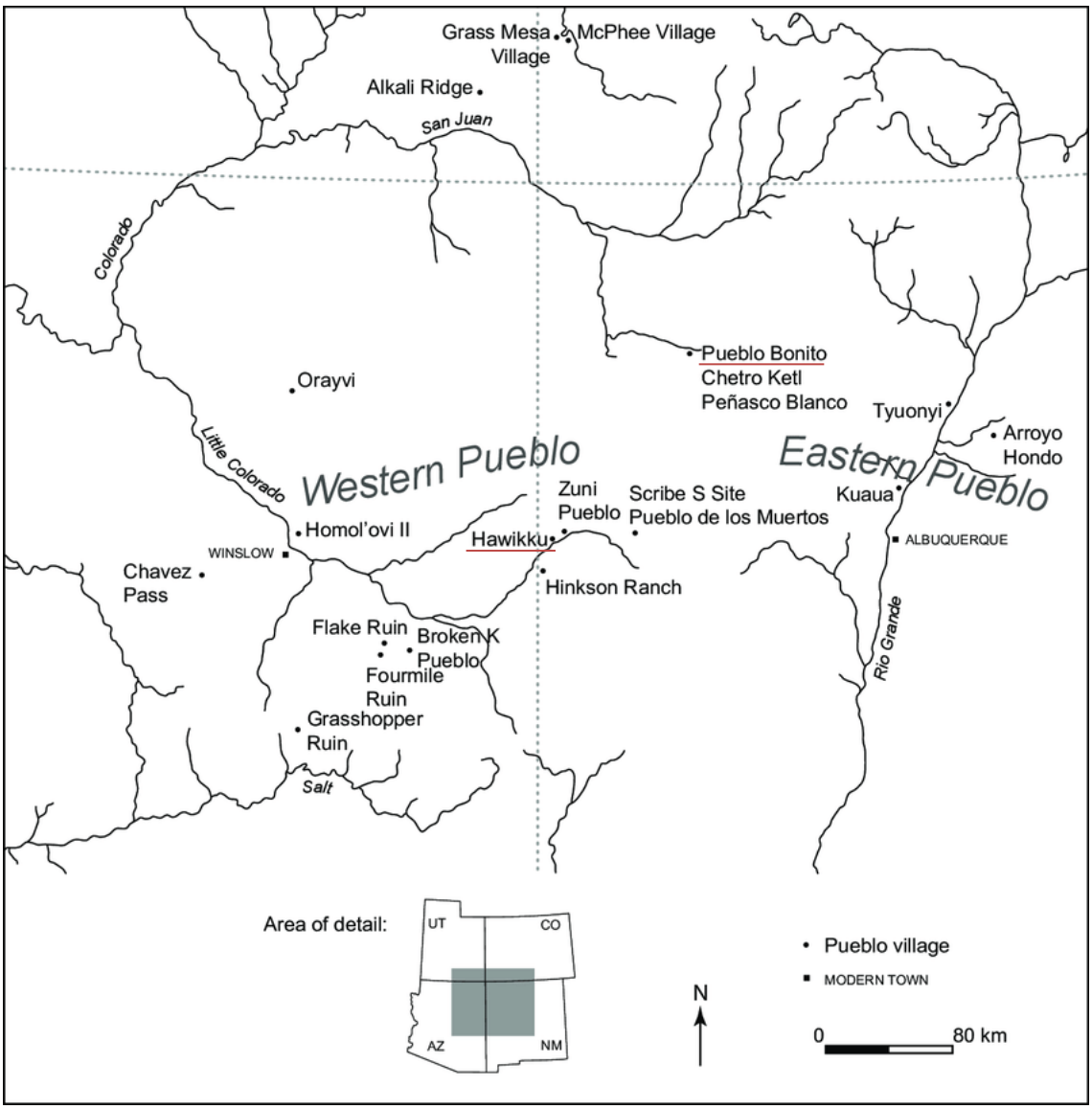

Figure 1: Map of American Southwest (Graves & Van Keuren, 2011)

During the Pepper excavation 30 burials were excavated from rooms 28, 32, 33, and 58, and these burials included the remains of approximately 24-28 individuals. The burial rooms, known as the northern burial rooms, were in converted living quarters that

were sealed off when converted to burial rooms. Once sealed, the burial rooms had no external access, suggesting a thoughtful burial and design for individuals of a more elite status. Unfortunately, the remains are not housed in the NMNH and were therefore not part of this study. Additionally, a full record of the process, stratigraphy, and distribution of the remains does not exist.

The skeletal remains that were used in this study were excavated from the western burial section that was uncovered during the Judd excavation from AD 1920-1926 (Judd & Allen, 1954). This section consisted of four rooms, 320, 326, 329, and 330. A total of 95 burials were part of the western burial section. Due to excavation issues the exact provenance of these individuals has been lost along with accurate timetables and stratigraphic evidence. It is possible that these individuals represent evidence of continuous reburial; however, there is not enough evidence to support this assertion. Additionally, around 70% of the burials have been disturbed by pre-Hispanic looters, which caused issues with creating a clear record of the burials (Akins, 2003).. These rooms were all adjacent, and opposed to the burial rooms excavated by Pepper, were not architecturally sealed off (Akins, 2003; Judd & Allen, 1954; Pepper, 1996). Room 320 had a total of 21 individuals with at least 10 identified as female. Room 326 had a total of 18 individuals with 14 adults and 4 subadults. Room 329 had 19 adults with one individual being identified as a male. Finally, room 330 had 23-32 individuals present (Akins, 2003; Judd & Allen, 1954; Palkovich, 1984; Pepper, 1996. While there is no way to establish a chronology of burial practices because of post-burial disruptions and lack of adequate records, there is some evidence to suggest, in keeping with the Chaco tradition,

individuals are buried to face the west in death. Depending on the researchers the total number of individuals excavated by Judd changes, however at the very least the NMNH houses at least 99 individuals in relation to the Judd excavation.

Paleoenvironmental Reconstruction at The Pueblo Bonito

The site of Pueblo Bonito, and the Chacoan society in general lies in the San Juan Basin in the Northwest corner on New Mexico. The basin itself covers roughly 12,000-sq-km area ranging in elevation from 2,500 meters to 1,500 meters. The average temperature in the San Juan Basin is -24°F to 106°F. Generally, there was a 150-day growing period. However, instances of frost have been known to decrease this by around 30 to 35 days. Annual precipitation is around 20 cm for the interior basin, 43 cm for the southern mountains and 50 cm for the northern mountains (Simmons et al., 1989). Major episodes drought occurred in the San Juan basin from AD 200-1300 (Grissino-Mayer, 1996). In order to buffer inconsistent precipitation, the Ancestral Pueblo of the Pueblo Bonito used a variety of food storage and irrigation techniques to combat seasonal stress (Pepper, 1996).

Subsistence Economy

Excavations of the households in Pueblo Bonito has yielded maize, pumpkins, beeplant, walnuts, grape, beans, squash, wild potato, and pinyon nuts (Judd & Allen, 1954; O'Donnell, 2019). Ethnographic evidence also suggests a subsistence that is based on Indian rice grass, fropseed, goosefoot, and pigweed (Crown, 2016). These foods could have been used by the individuals at Pueblo Bonito. Pollen evidence shows that before AD 900 there was a larger reliance on maize agriculture. However, after this point there

was a rise in the use of wild plant taxa such as prickly pear, and hedgehog cactus. The majority of farming associated with the Chaco canyon occurred in a drainage basin to the west of the settlement.

Reexamination of the trenches that were orginally excavated by Neil Judd in the early 20th century yielded a plethora of faunal remains (Crown, 2016). Through the course of reexamination 7,000 specimens were found, approximately 95% of this sample were mammals. The most common species found were two types of Lagomorpha, *Lepus* and *Sylvilagus,* and medium sized Artiodactyl (Kennett et al., 2017). Based on evidence of burning and cut marks *Lepus, Sylvilagus*, and Artiodactyl or hares, rabbits, and deer sized hoofed mammals were consistently exploited by this population. These species are also present in coprolite data (Clary, 1986).

Craft Production at Pueblo Bonito

The Ancestral pueblo people that live in Pueblo Bonito were responsible for the production of a variety of different materials. Archaeological evidence has shown an abundance of Gray Ware such as Chuska, Tusayan, Mesa Vere, and Cibola were all made by different temporal and physical production units (Crown, 2016). An abundance of white ware ceramics was also found that signifies a shift that occurred during the Pueblo II period. This shift was also accompanied by a change in vessel shape from bowls to jars. The white ware of the Pueblo III period is known for its increased complexity and more artistic decoration on ceramic artifacts. Throughout the occupation of Pueblo Bonito there are a variety of different lithics made into several types of both formal and expedient tools. There is also evidence for various types of food production and

processing at Pueblo Bonito. Lastly the Ancestral Pueblo produced several types of ornaments in the form of beads and turquoise decorated adornments. In addition to local pottery production, excavations have revealed a large number of ceramic artifacts that were brought into Pueblo Bonito as part of the trade network. At some points in the history of Pueblo Bonito, up to 50% of all ceramic object were imported from neighboring settlements (Wills et al., 2014). This coincides with various theories that state Pueblo Bonito was a regional hub for both economic and political activity.

Social Structure at Pueblo Bonito

Based on mortuary and genetic evidence it is likely that the Ancestral Pueblo of Pueblo Bonito lived in a hierarchal society with a singular kin group as the ruling class. One individual from room 33 located in Burial 14 in the northern section was interred with thousands of pieces of turquoise, shells, beads, and pendants (Judd & Allen, 1954). In addition, there was also several culturally signficant offerings such as conch shells, macaw feathers, and ceramic vessels (Kennett et al., 2017). Burial 14 was a male around the age of 40 years (Kennett et al., 2017). Another individual was interred adjacent to burial 14 and 12 other individuals were placed in the same room above the primary burial, separated by a wooden floor. Analysis of the individuals DNA has revealed that this burial is a multigenerational site with the largest age gap being a grandmother-grandson relationship (Kennett et al., 2017). Genetic evidence of kin burials along with the fact that room 33 has the most elaborate burial in all of Chaco society strongly suggests an elite class.

When comparing the style of the northern burials, especially room 33, to the western burials as a whole it is clear that one group was of a comparatively higher status. Some of the western burials, such as room 320, had grave goods such as earthenware pots and baskets; however, the elaborations found in room 320 were not recorded for any of the northern burials (Judd, 1964; Lekson, 2006b; Pepper, 1996). These results suggest strong delineations in social prestige and reflect a rigid, hierarchical social organization (Palkovich, 1984).

Skeletal indicators of violence and stress a The Pueblo Bonito

Traumatic injuries were found at lower frequencies among Early and Classic Bonito burials (AD 850 to 1140) as compared to other contemporaneous sites from the American Southwest (Harrod, 2012; Harrod, Martin, & Fields, 2017a). There has been some discussion on what caused this reduction in violence. The prevailing hypothesis is that during the Bonito phases the ruling elites enforced a decrease in violence. Additionally, skeletal evidence of stress was reduced in ruling elites from these sites. Femoral length was greater in elite compared to non-elite burials indicating that growth disruption may have been socioeconomically patterned (Stoddard, 1989). Paleodemographic data suggests that that life expectancy was greater in the elite versus non-elite section of the cemetery. (Harrod et al., 2017a; Stodder, 1989). Recent research on the Pueblo Bonito has revealed an elevated prevalence of stress indictors compared to the rest of the American Southwest. Linear enamel hypoplasia, occurred in 46% of the sample (Ham, 2018). Osteoperiostitis was observed in 51% of the Pueblo Bonito sample, and those individuals with healed versus active lesions had a greater probability of

survival (Ham, 2018). Skeletal evidence for iron deficiency was present in 20-25% of the sample and associated with a higher mortality risk (Akins 1986; O'Donnell, 2019; Palkovich, 1984; Stodder, 1989). Additionally, approximately 54% of individuals at the site expressed carious lesions on teeth, which is consistent with dependence on starchy ot high sugar foods such as maize and potatoes (Mobley et al., 2009) . Taken together, these results indicate substantial interactions between local environment, status, stress, and mortality in the occupants of Pueblo Bonito (Stodder, 1989).

Abandonment of Pueblo Bonito

Pueblo Bonito was the central hub of the Chaco society from around AD 1040 until societal decline in the 13th century. Social decline in this region is measured by a reduction in kiva use (Crown & Wills, 2018). The reason for the decrease in kiva uses and decline in the population is debated. One of the most common theories was deforestation due to an overreliance on wood for building material and fuel (Wills et al., 2014). While there is evidence of decline in plant life and available resources, research has shown that there is not enough evidence to demonstrate that, deforestation occurred at a rate that would have caused the depletion of local resources. Kiva construction was demanding, but a mixture of local and long-distance trade likely buffered local resources from severe depletion (Wills et al., 2014). Another suggestion for the decline of the Pueblo Bonito as a cultural center was drought. Rainfall was scarce over the first three hundred years of occupation (Lekson, 2006a). Near the end of the end of the occupation of Pueblo Bonito there is evidence to suggest relatively harsh summers with little rainfall that could have damaged crop production. These ecological constraints may have

exceeded the capacity of local socioecological systems to buffer against drought (Palkovich, 1984).

Hawikku

The Hawikku site, sometimes called the Hawikuh, Hawikku, Háwik'uh, in the American Southwest is associated with the Zuni culture. According to ethnographic and ceramic evidence the Zuni people occupied Hawikku during the pre-protohistoric period AD 1325-1680 (Howell, 1996; Kintigh, 1985). Settlement of Hawikku is divided into two periods. The Prehistoric period (AD 1300-1375) included the formation of large, permanent villages in the Zuni area (Eckert, 2005). The Protohistoric (AD 1375-1630) covers period covers 255 years (Eckert, 2005). The Historic (AD 1630-1680) period dates to the beginning of the Catholic mission at Hawikku and the end of settlement during the Pueblo Revolt (Eckert, 2005). The Zuni were primarily agriculturalists, despite the desert of New Mexico, which was a difficult place for agriculture-based subsistence. The Zuni people had a very intricate type of pottery that varied in its functional use. One of the most well know ceramic productions were the Kechipawan and Matsaki Polychrome jars and bowls that commonly held the motif of feathers and or shields.

History of Excavations at Hawikku

The primary excavation of skeletal material at the Zuni Hawikku site occurred during the Hendricks-Hodge expedition that occurred from AD 1917-1923 (Smith, et al., 1966). This expedition was commissioned by the Museum of the American Indian and the Bureau of American Ethnology. A full site report on this excavation was not written by Hodge, instead the work was published 43 years after the initial excavations. To this

day the Hendricks-Hodge expedition is one of the most extensive bioarchaeological excavations in the American Southwest. The excavation included 996, individuals in mostly single burials. Of the 996 individuals 679 were inhumations, and 317 were cremations. The most common grave goods were decorated bowls or jars (for a complete list see Howell, 1996, p. 64; Smith et al., 1966). The site dates to the pre-protohistoric period, but it is possible that there were several archaeological layers that spanned time periods that predated the occupation of the Hawikku by the Zuni people. Hodge states that "there is every reason to believe that, as intimated, the site was inhabited by Indians differing more or less in culture from that of the Zunis of the Hawikuh period." (Smith et al., 1966, p. 182). However, due to lack of records there is no way to know which, if any of the individuals in the modern collection are of an earlier time period. Of the 679 individuals that were excavated, only 226 individuals were sent to the United States National Museum for analysis under the supervision of Ales Hrdlička. However, 136 of these individuals do not have the field numbers that were assigned by Hodge (Smith et al., 1966). With the passing of the Native American Grave Protection and Reparation Act in 1990 the NMNH assessed and cataloged the remains in preparation for repatriation. The current Zuni people, descendants of those who lived in Zuni Hawikku, allowed the remains to stay in the NMNH for future study.

Environment at Hawikku

The reservation for the Zuni people is an area around 650 miles and is located in the southeastern edge of the Colorado Plateau, which in modern terms is the southwestern corner of McKinley county in western New Mexico (Figure 1) (Leighton, 1966). The site

of Hawikku is located on the southern end of a low ridge in the Zuni River Valley near the junction of the Zuni and Plumasano Wash river (Ferguson, 1996). Temperatures range from highs of 102 °F to 106 °F to lows of -38 °F to -48 °F in the winter (Dean, 2007;Vivian 1990, p. 21). While it does vary dramatically by elevation and location, the average rainfall in the Hawikku area is around 20.3 cm. In general, the environment does not deviate in annual temperature and rainfall, however paleoenvironmental has shown occasional variation in precipitation causing drought that affected the Zuni Hawikku (Vivian, 1990).

The Hawikku pueblo is comprised of 20 pueblo room blocks in an irregular arrangement. This large pueblo built upon or by slow acceleration and gradual additions made over several centuries (Ferguson, 1996). Therefore, the exact date of original formation and habitation is not known. Estimates place the number of ground rooms at 470 with a possibility of second and third stories extending that estimate to 800-1,060 rooms. Within the borders of the site there is also a Catholic church that was built in AD 1629 (Ferguson, 1996).

Hawikku Subsistence Economy and Socioecological Systems

The Zuni people were primarily agriculturist. Archaeological excavations have yielded evidence of subsistence on maize, squash, beans, and pumpkins (Martin & Stodder, 1992: Riley, 1975). While the diet was primarily plant based there was also some reliance on meat. Zooarchaeological analysis suggests that the meat came from American bison and or deer species (Riley, 1975). There exists ethnographic evidence for domesticated species such as turkeys. Increasing contact with the Spaniards introduced

non-native domesticates such as wheat, peaches, sheep, horse, burros, and cattle (Ferguson & Mills, 1982).

Social Structure

Based on mortuary analysis and burial data it is possible that there were four leaders that held gendered positions (Howell, 1996). Three of these individuals were biological females and one was a biological male. The three biological females share a burial orientation facing the southeast, which is an uncommon orientation for Hawikku burials (Howell, 1996). One of these burials, 915A, has the most elaborate grave preparations for any individual associated with the site. Various grave goods and wrapping were found, along with a possible shrine that was adorned with feathers from four species of bird (Howell, 1996). The biological male, from burial 113, was found in an extended and supine condition with an eastern orientation. This burial was associated with drastically different grave goods then the biological female burials. Burial 113 contained remains of human scalps, a ceramic pipe, a bow, and various pigments. These burials suggest that individuals of elevated status were present in the Zuni Hawikku community, and biodistance analysis suggests that each represent distinct kin groups (Howell 1996; Howell & Kintigh, 1996). Evidence for the rigid social hierarchies observed at Pueblo Bonito are largely absent at Hawikku.

Colonial encounters

Ethnohistoric evidence suggests that the first encounter between the Zuni and Spanish colonialists occurred around AD 1539, with Marcos De Niza's incursion into the region (Crampton, 1977). At approximately the same time, ethnohistoric sources recount

that the Zuni Hawikku executed a European emissary named Estevan (Crampton, 1977). Francisco Vasquez de Coronado arrived at the Hawikku site in AD 1540 expecting riches, only to then realize this was an agricultural settlement that did not have any abundant resources to plunder, except corn (Crampton, 1977). The Zuni people organized a strategic resistance that involved an armed force of 250, spies, smoke signals, and a robust stockpile of weapons.

After this initial interaction more violent escalations occurred in the 16th century, all of which were short lived and did not consist of extended conflict between the Zuni and the conquistadores (Crampton, 1977; Ferguson, 1996) These military expeditions eventually led to Catholic missionaries in the 17th century, which in turn resulted in the death of several Spanish priests and Hawikku leaders during a particularly tense time. During this Historic period (A.D, 1630-1690) life expectancy decreases as a result of contact with European diseases (Eckert, 2005). The most signficant escalation occurred in AD 1680 when there was a large conflict against the colonists called the Pueblo Revolt, in which several Pueblo communities worked together to temporarily expel the Spaniards. In preparation for the return of the Spaniards, the Zuni people abandoned the site of Hawikku for a more defendable position. Despite the temporary victory, the area was eventually recolonized in the AD 1700. Epidemic resurgence occurred as continuous interaction with Spanish colonialists continued to happen. In AD 1640 a lethal epidemic resulted in a mortality rate involving 10 % of the Pueblo population (Eckert, 2005, Palkovich, 1985). The population eventually rebounded to precontact levels around 600 years later (Ferguson, 1996).

Skeletal indicators of violence and stress

Approximately 53% of individuals have carious lesions on teeth, and 58% have periapical abscesses, as well as premortem tooth loss that was associated with increasing age (Stodder, 1990; Stodder & Martin 1992). These findings are similar to Pueblo Bonito and are associated with reliance on high sugar and starchy foods. Linear enamel hypoplasia was present in 30% of individuals that did not survive to adulthood (Stodder, 1994). Skeletal indicators of iron deficiency were present in approximately 87% of the subadults and adults (Schillaci et al., 2011; Stodder, 1990). These results suggest low iron bioavailability in the region, either due directly to diet or parasitic infection (Walker et al., 2009; Stodder, 1990a). Approximately 36% of the sample express skeletal indicators of chronic infection, and diseases such as treponematosis and tuberculosis have been identified in these remains (Stodder, 1990). It is highly likely that these instances represent a minimum number of infectious conditions at the site as most individuals likely died prior to forming lesions (Stodder and Martin, 1992). Skeletal indicators of violent injury are distributed in about 12% of the sample, which is comparatively low for the region (Stodder and Martin, 1992). Recent studies suggest that individuals with linear enamel hypoplasia survived for a substantially reduced period of time than those without the condition, indicating that early life adversity may have increased mortality risk at this site (Ham, 2018). In addition, infectious diseases may have differentially affected those that already had an increased mortality as a result of early life stress. These results suggest that the individuals who occupied the Hawikku site experienced high levels of stress in association with local environmental conditions as well as diseases

introduced through Spanish colonialism. In addition, growth disruptions found in enamel illustrate that early life stress elicited increases in mortality risk during this time.

Conclusion

The site of Pueblo Bonito was excavated by George Pepper in the 1890s and by Neil Judd in the 1920s. The Pepper excavation yielded 24-28 skeletonized individuals. The Judd excavation yielded 99 individuals. The Zuni Hawikku site was excavated during the Hendricks-Hodge expedition and yielded 226 individuals that are housed within the NMNH. Pueblo Bonito is a prehistoric site that occupied from AD 850-1140. The Zuni people occupied Hawikku during the pre-protohistoric period AD 1325-1680. The Ancestral Pueblo of Pueblo Bonito existed in a rigid hierarchical social system. The Zuni Hawikku had evidence of individuals with elevated status that governed the site. The individuals at both sites were agriculturists. Environmental constraints such as drought and short growing periods made agriculture difficult, but both groups subsisted on maize and other domesticates. Animal species were also exploited by individuals at both sites. As a result of maize agriculture both sites had high frequencies of dental pathological conditions. Iron anemia was also common at both sites. The Ancestral Pueblo of Pueblo Bonito were under intense stress as a result of the delineation of social prestige. The Zuni Hawikku were experienced additional pressure from infectious diseases as a result of pathogens from European colonists.

CHAPTER FOUR: METHODS

Osteometric Measurements

Midsagittal and interpedicular dimensions of the VNC were measured using digital sliding calipers that record to the nearest tenth of a millimeter. The midsagittal diameter is defined as maximum distance between posterior portion of the body and most anterior portion of the neural canal. The interpedicular diameter is defined as the maximum distances between the vertebral pedicles (Amoroso & Garcia, 2018; Beauchesne et al., 2019; Clark et al., 1986; Martin, 1928; Newman & Gowland, 2015; Watts, 2011, 2013a). Individuals with pathological conditions that effected the VNC were excluded from this study. In addition, vertebral canals must have been morphologically intact, not broken or fragmented due to postmortem alteration. In this study 47 individuals from Pueblo Bonito and 102 individuals from Hawikku had vertebrae that fit the criteria of the study. In total 2,658 vertebrae were measured for a total of 5,316 measurements. The term "growth disruption" is used in VNC literature to refer to any alteration of the size of the canal (Clark et al., 1986, Newman and Gowland 2015; Watts 2011; 2013a; 2013b). For the purpose of this study smaller VNCs are classified as below mean diameters for each sample.

Estimation of Age and Determination of Sex

Subadult age-at-death was estimated recording mandibular tooth formation and emergence and sublimated with long bone measurements. Age was then estimated using data derived from standard phases of maturity (AlQhatani et al., 2010; Buikstra and Ubelaker, 1994). Individuals below the age of five years were excluded because of incomplete vertebral fusion. Previous studies have estimated age at death in Pueblo Bonito and Hawikku using standard morphological appearance of the pubic symphysis and auricular surface as well as transition analysis (Ham, 2018; Harrod, 2012; Hrdlička, 1931; Palkovich, 1984; Schillaci & Stojanowski, 2003; Stodder, 1990). Traditional methods for the estimation of age at death rely on methods that mimic the age distribution of reference samples (Boucquet-Appel and Masset, 1982; Konigsberg and Frankenberg, 1992; Konigsberg and Herrmann, 2006) and fail to identify individuals over 55 years of age (Boldsen et al., 2002; Konigsberg and Herrmann, 2006). The use of transition analysis provides point estimates of most likely age at death, and because the probability function for this method is the inverse of that associated with traditional age estimation methods (see: Konigsberg et al., 2007), avoids mimicry of reference samples (Boldsen et al., 2002). While transition analysis still produces high levels of error at the individual level (Bethard, 2005; Milner and Boldsen, 2012), resultant demographic distributions still follow samples of known age (Milner and Boldsen, 2012). This suggests that comparisons of age distributions between individuals are still accurate when transition analysis is employed as the method of age estimation.

Age at death was estimated by recording degenerative phases of the pubic symphysis and auricular surface using methods described by Boldsen et al. (2002). These phases were entered into the ABDOU 2.0 software program, and point estimates for age at death combined with 95 percent confidence intervals were provided. Point estimates were used as the most likely age at death in each individual.

Sex was determined using morphological features of the pubis and ischium. Morphological variation in the pubis and iscium were recorded using standard protocols (Buikstra & Ubelaker, 1994). Previous studies compare VNC size by sex but have all found little to no differences between males and females (Clark et al., 1986; Newman and Gowland, 2015; Watts, 2011). Accordingly, sex specific differences in VNC dimensions are not compared by this study.

Statistical analysis of data

VNC dimensions were compared between age cohorts using one-way ANOVA with a Games-Howell post hoc test. The goal of the comparison was to test the hypothesis that VNC is smaller in the non-survivors in younger age groups than individuals who survived to later ages. Subadults in this study were defined as individuals from 5-16 years. Age groups were defined as: subadult (N Pueblo Bonito = 16; N Hawikku = 16), 17-25 years (N Pueblo Bonito = 8; N Hawikku = 17), 26-35 years (N Pueblo Bonito = 9; N Hawikku = 33), 36-45 years (N Pueblo Bonito = 4; N Hawikku = 20), 46-55 years (N Pueblo Bonito = 3; N Hawikku = 7), and 55+ years (N Pueblo Bonito = 7; N Hawikku = 9). Previous studies used 45 years as a maximum reference point (Benus et al., 2010: Goodman et al., 1980; Watts 2011.). This study uses the 56+ year cohort given the utility

of transition analysis in identifying older individuals, that being said this is a relatively large age range that was unavoidable due to life expectancy in these samples. The one-way ANOVA with Games-Howell post hoc test is used due to uneven sample sizes between age cohorts and the need to reduce the probability of a Type-II error stemming from multiple comparisons.

Kaplan-Meier Survival Analysis

Mortality risk associated with growth disruption in the VNC was evaluated at each site using Kaplan-Meier survival analysis and a log-rank test. Kaplan-Meier survival analysis calculates the probability of survival at each age based on the proportion of individuals still living, while the log-rank test estimates differences in this curve between samples. The goal is to compare survival of individuals with smaller versus larger VNC as a result of growth disruptions. Here, smaller VNC versus larger was identified as the covariate, while age at death was the time series variable. In concert of previous studies some vertebrae are analyzed independently, and others were clustered with vertebrae of similar structure and measurement (Newman & Gowland, 2015). The vertebra were divided in to the following groups for comparative purposes: C1, C2, C3-4, C5-6, C7, T1, T2, T3-5, T6-8, T9-11, T12, L1, L2-4, and L5. The clustering of the vertebra resulted in 14 tests per measurement per sample, for a total of 56 comparisons.

CHAPTER FIVE: RESULTS

The results of this studied are presented in the following chapter. The first analysis that will be discussed was a comparison of age cohorts within the Pueblo Bonito and Hawikku population using an ANOVA with a Games-Howell post-hoc test. The second analysis was the Mantel-Cox log rank with a Kaplan-Meier survivorship curves that show the differences in survivorship for individuals possessing a smaller VNC.

ANOVA Game-Howell

The individuals were compared using SPSS Version 26. The following tables show the result of the analysis. Significant results ($P \leq 0.05$) are displayed below (Table 1, Table 2, Table 3) with the rest of the results available in the appendix. Detailed breakdown of the samples are also available in the appendix (Table 10). Graphic explanations of interquartile ranges and median VNC diameters are presented in boxplots.

Interpedicular Diameter: Pueblo Bonito

Table 1: Significant ANOVA Results for the Interpedicular Measurement of the Pueblo Bonito

Sample	Measurement	Vertebra	Age Cohort	P-Value	Figure
Pueblo Bonito	interpedicular	C2	17-25	0.014	2

Pueblo Bonito	interpedicular	C4	36-45	0.027	3
Pueblo Bonito	interpedicular	T1	36-45	0.027	4
Pueblo Bonito	interpedicular	T3	Sub-Adult	0.024	5
Pueblo Bonito	interpedicular	T4	Sub-Adult	0.009	6
Pueblo Bonito	interpedicular	T4	17-25	0.043	6
Pueblo Bonito	interpedicular	T5	Sub-Adult	0.007	7
Pueblo Bonito	interpedicular	T6	Sub-Adult	0.003	8
Pueblo Bonito	interpedicular	T7	Sub-Adult	0.005	9
Pueblo Bonito	interpedicular	T8	Sub-Adult	0.030	10
Pueblo Bonito	interpedicular	T9	Sub-Adult	0.044	11
Pueblo Bonito	interpedicular	T10	Sub-Adult	0.039	12
Pueblo Bonito	interpedicular	T10	17-25	0.047	12
Pueblo Bonito	interpedicular	L1	Sub-Adult	0.013	13
Pueblo Bonito	interpedicular	L2	Sub-Adult	0.002	14
Pueblo Bonito	interpedicular	L2	26-35	0.027	14
Pueblo Bonito	interpedicular	L3	Sub-Adult	0.001	15
Pueblo Bonito	interpedicular	L3	17-25	0.046	15
Pueblo Bonito	interpedicular	L4	Sub-Adult	0.010	16
Pueblo Bonito	interpedicular	L5	Sub-Adult	0.017	17
Pueblo Bonito	interpedicular	L5	46-55+	0.024	17

Figures 2-17 are box plots that depict the interquartile range of interpedicular diameter across the lifespan for each vertebral element where significant differences between age groups were found at Pueblo Bonito. Table 1 list the instances where significant differences in interpedicular diameter were found as well as the age cohorts where these differences appear and are maintained across all subsequent years of life at the Pueblo Bonito site. VNC measurement for significant results are displayed in the appendix (Table 11). Significant differences between age-at-death cohorts were found for 16 interpedicular diameters across 21 age cohorts at the Pueblo Bonito site. These

instances reflect smaller interpedicular diameter in younger age cohorts and larger interpedicular diameter in older age cohorts.

In the majority of instances (13/21) individuals in the subadult age category had significantly smaller interpedicular VNC diameters than any other age group. These results are shown in the interpedicular diameter of T3-T10, L1-L5 (Figures 4-16). However, several instances (3/21) were also found where individuals in older age cohorts of C4, L2, L5 also had significantly smaller interpedicular diameter than those in even later age groups (Figures 3,14,17), while four instances, including C2,T4, T10, L3, where the subadult plus 17-25 years age category had significantly smaller interpedicular diameters than all subsequent age cohorts (Figures 2, 5, 12, 15). It is important to note that significant differences in interpedicular diameters are not found between earlier and later age cohorts, suggesting that smaller interpedicular diameter resulted in selective mortality even under circumstances where individuals survived beyond the subadult ages.

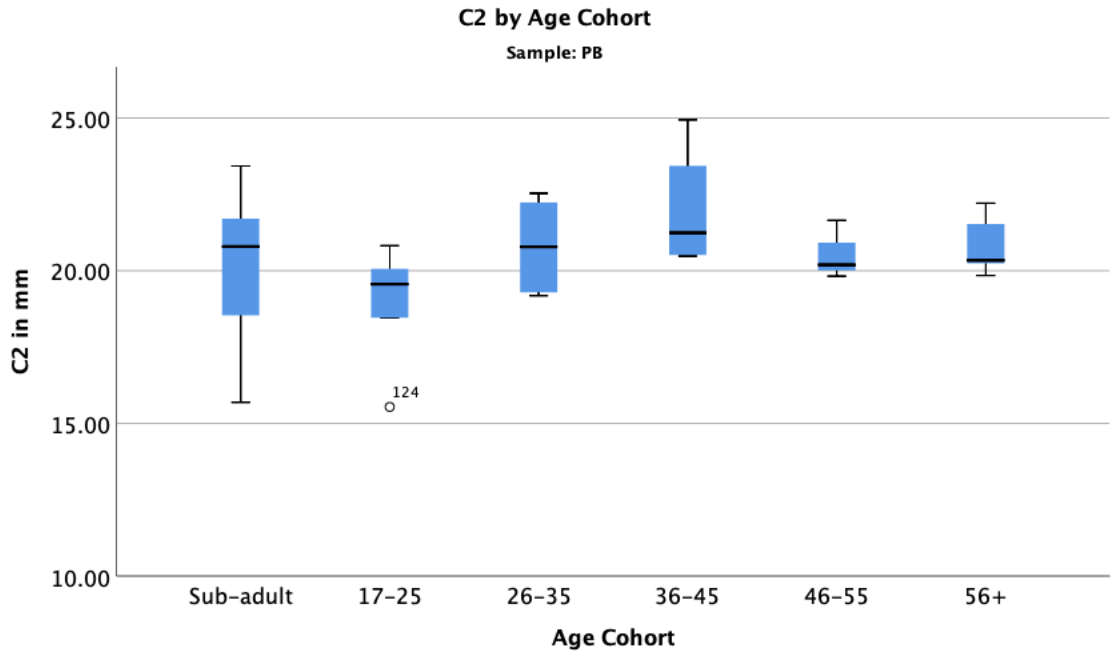

Figure 2:Boxplots of Pueblo Bonito C2 Interpedicular Diameter

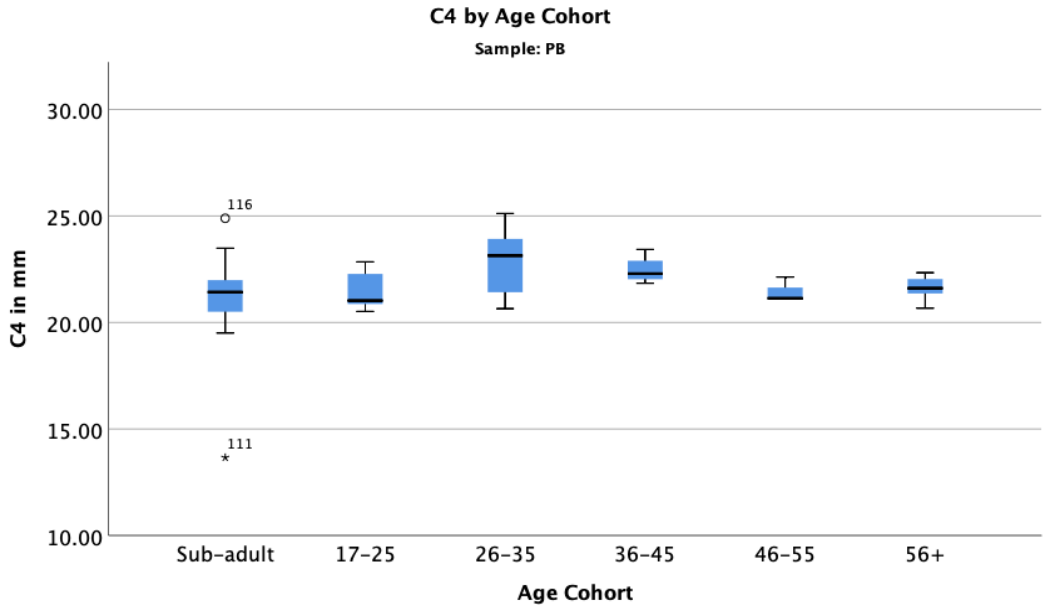

Figure 3: Boxplots of Pueblo Bonito C4 Interpedicular Diameter

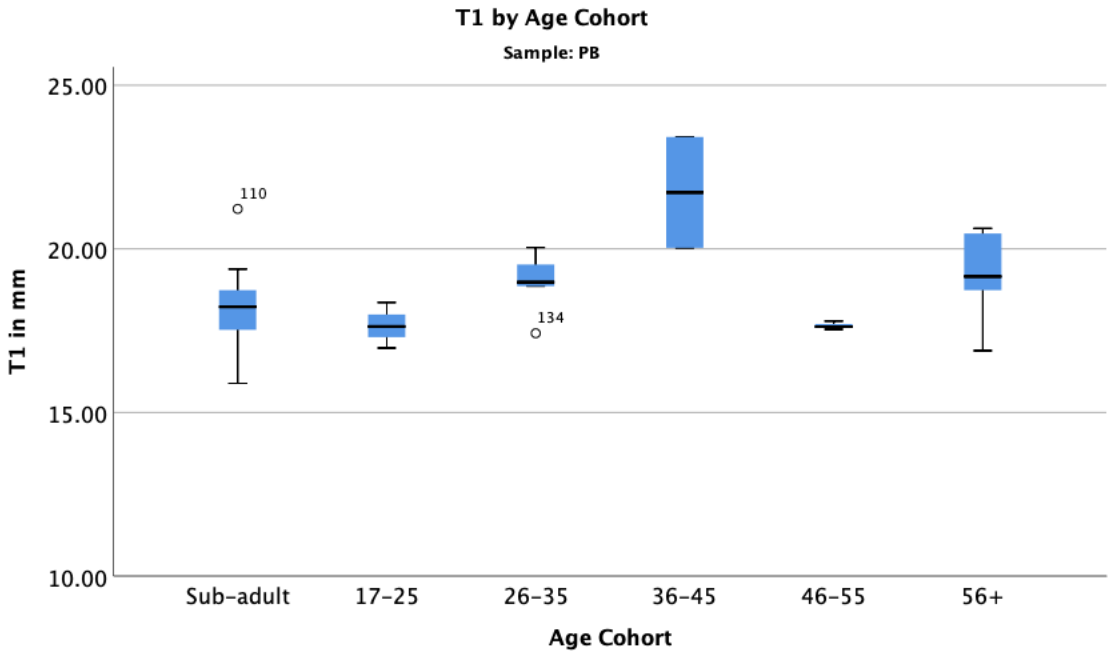

Figure 4: Boxplots of Pueblo Bonito T1 Interpedicular Diameter

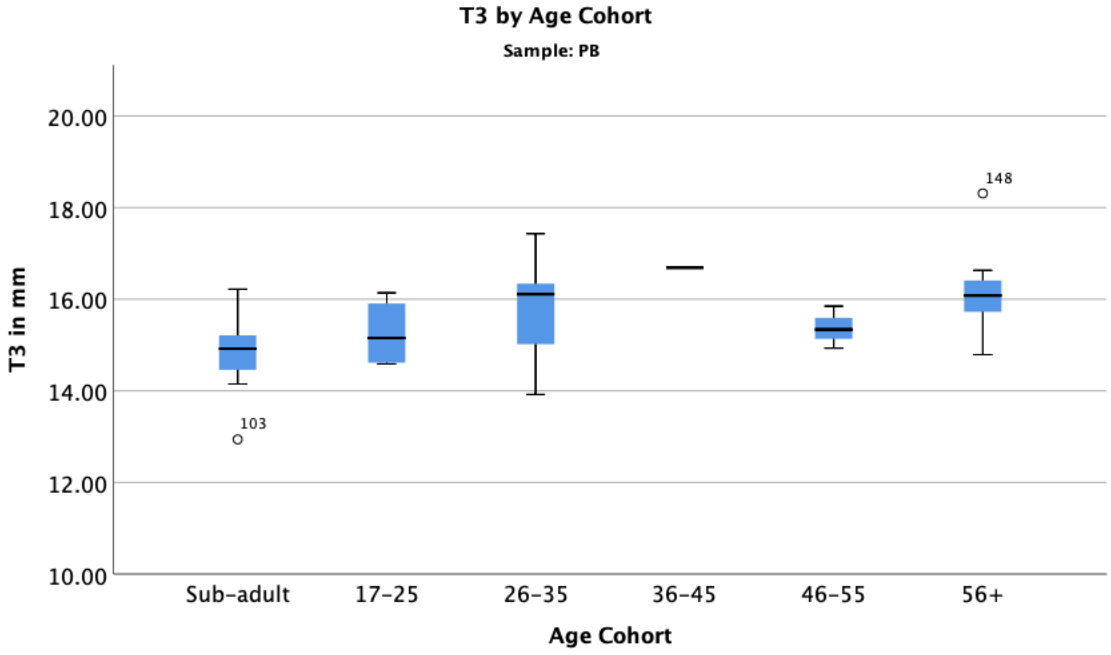

Figure 5:Boxplots of Pueblo Bonito T3 Interpedicular Diameter

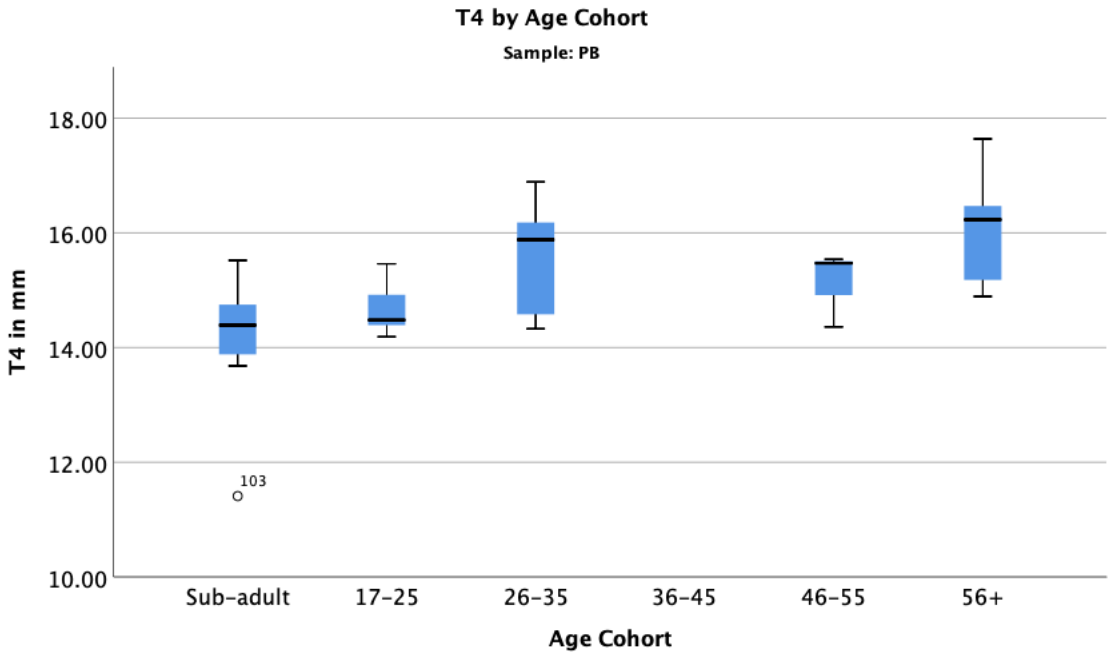

Figure 6: Boxplots of Pueblo Bonito T4 Interpedicular Diameter

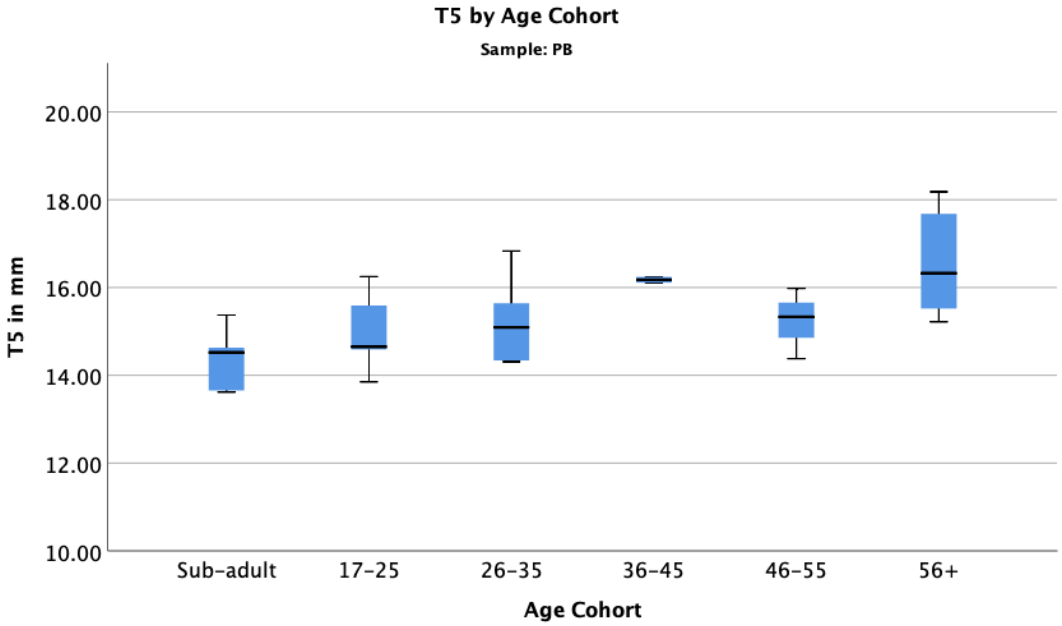

Figure 7: Boxplots of Pueblo Bonito T5 Interpedicular Diameter

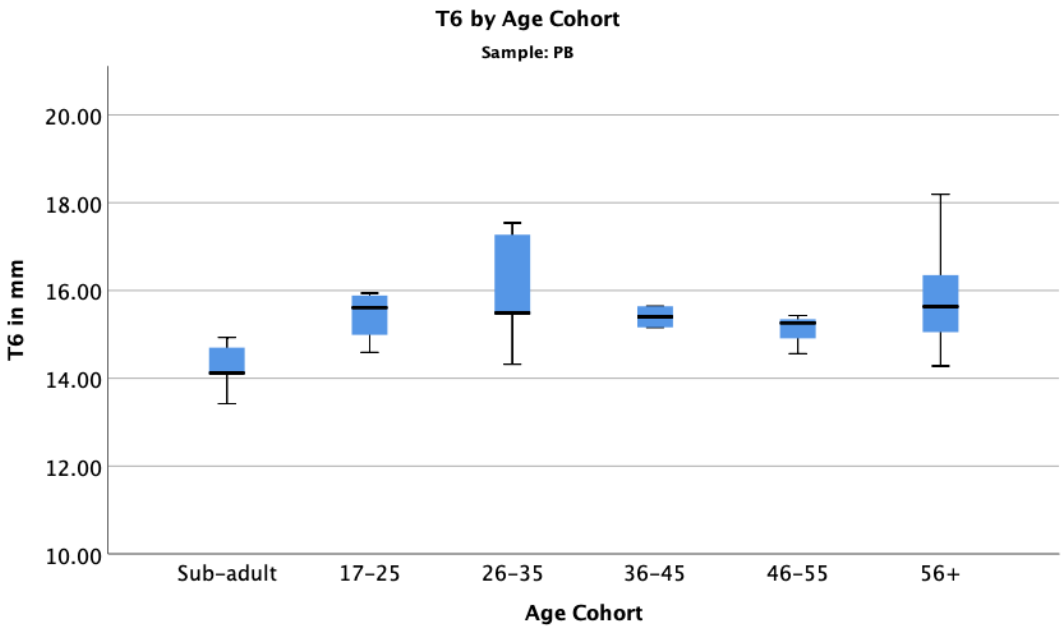

Figure 8: Boxplots of Pueblo Bonito T6 Interpedicular Diameter

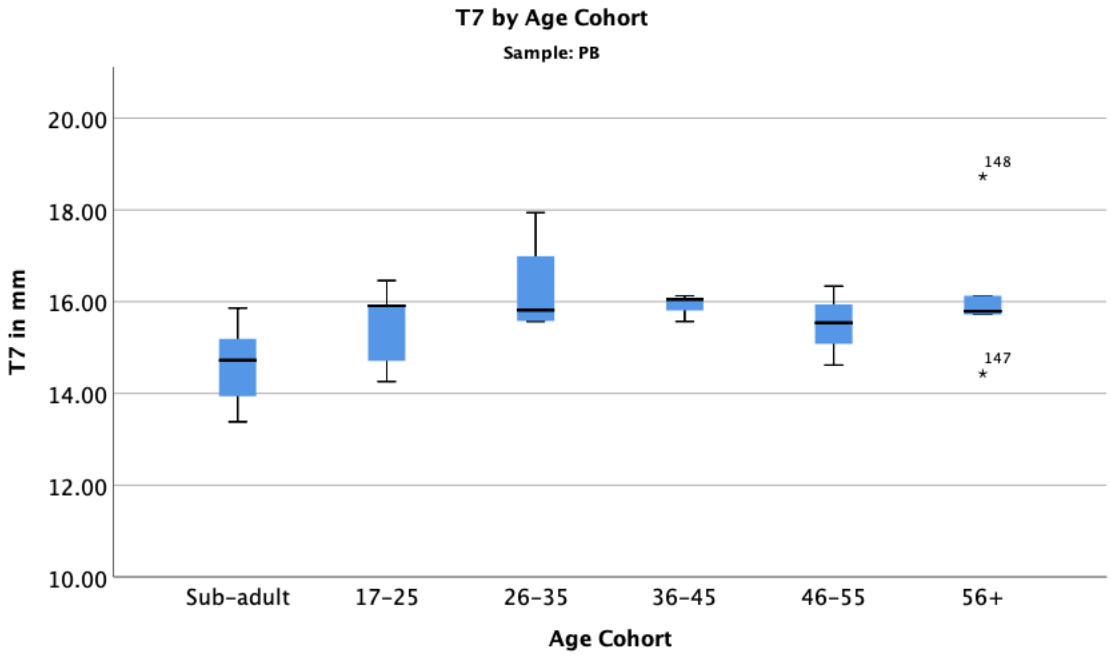

Figure 9: Boxplots of Pueblo Bonito T7 Interpedicular Diameter

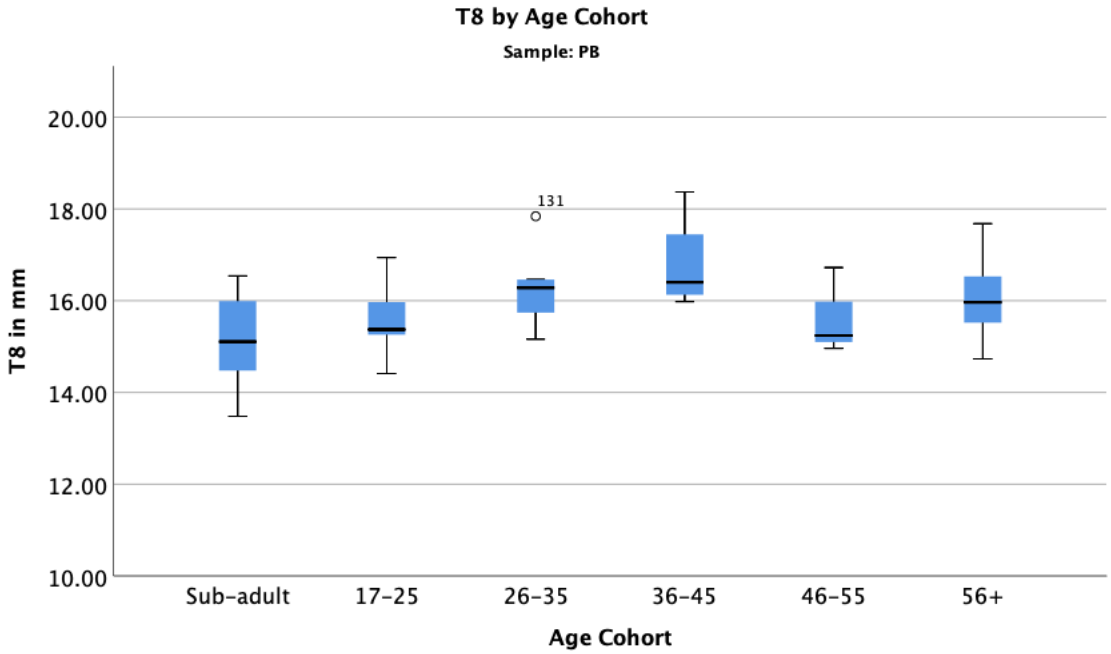

Figure 10: Boxplots of Pueblo Bonito T8 Interpedicular Diameter

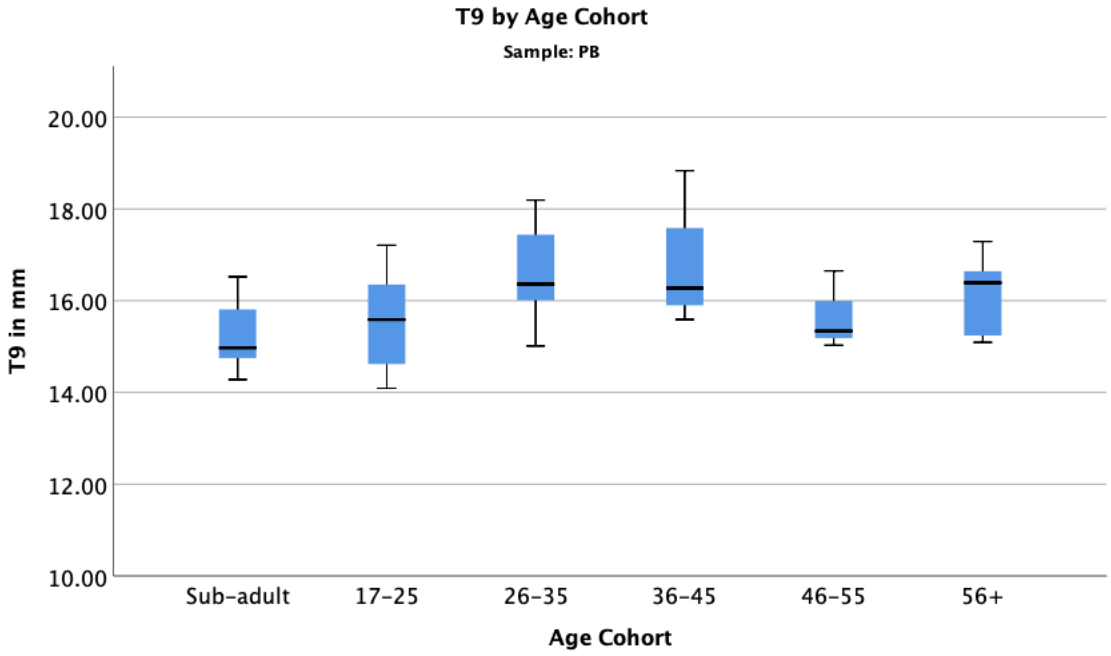

Figure 11: Boxplots of Pueblo Bonito T9 Interpedicular Diameter

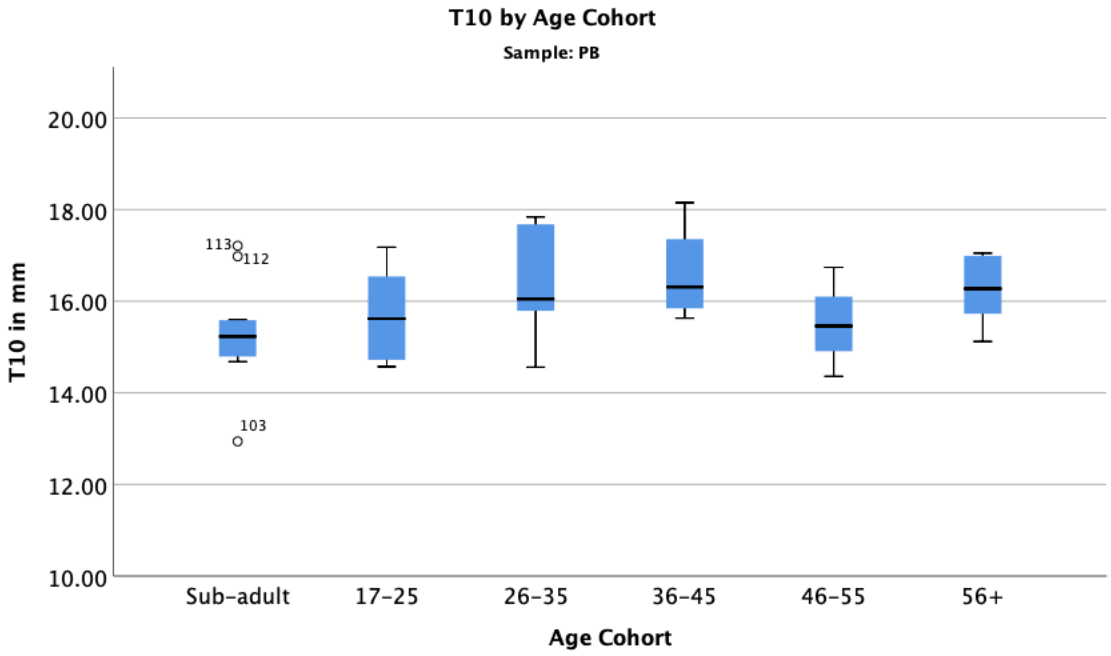

Figure 12: Boxplots of Pueblo Bonito T10 Interpedicular Diameter

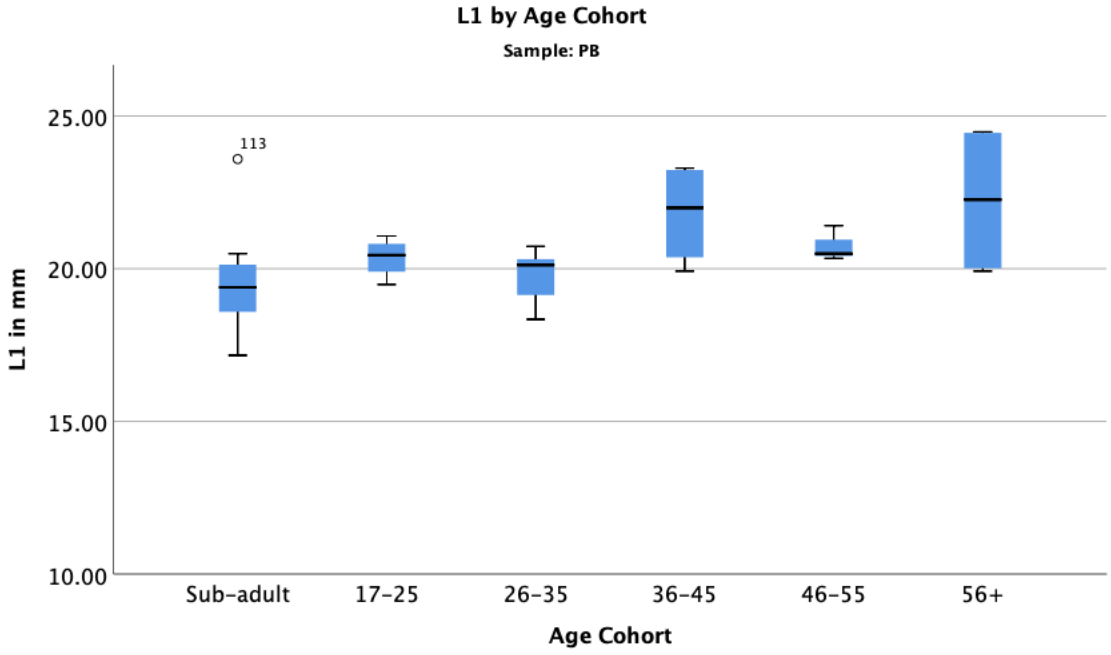

Figure 13: Boxplots of Pueblo Bonito LI Interpedicular Diameter

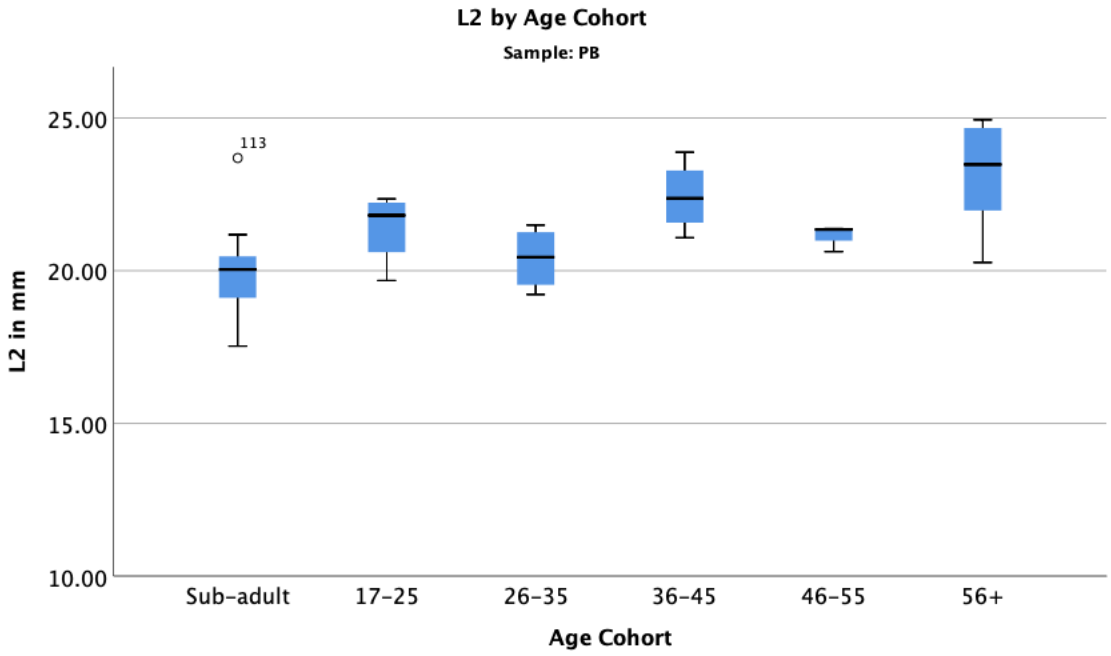

Figure 14: Boxplots of Pueblo Bonito L2 Interpedicular Diameter

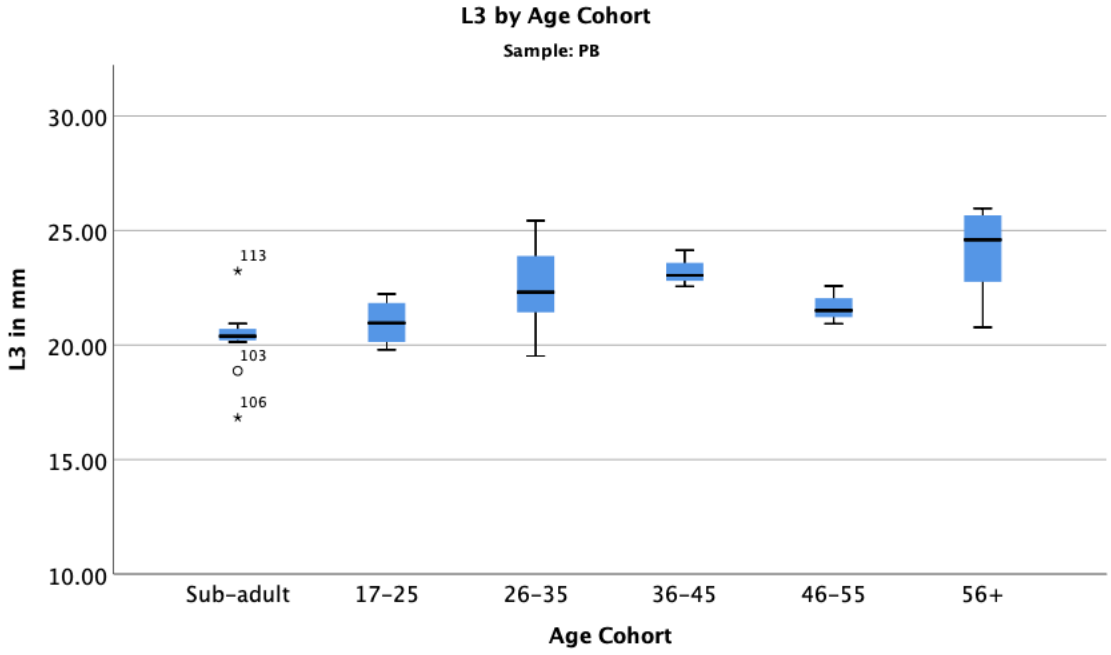

Figure 15: Boxplots of Pueblo Bonito L3 Interpedicular Diameter

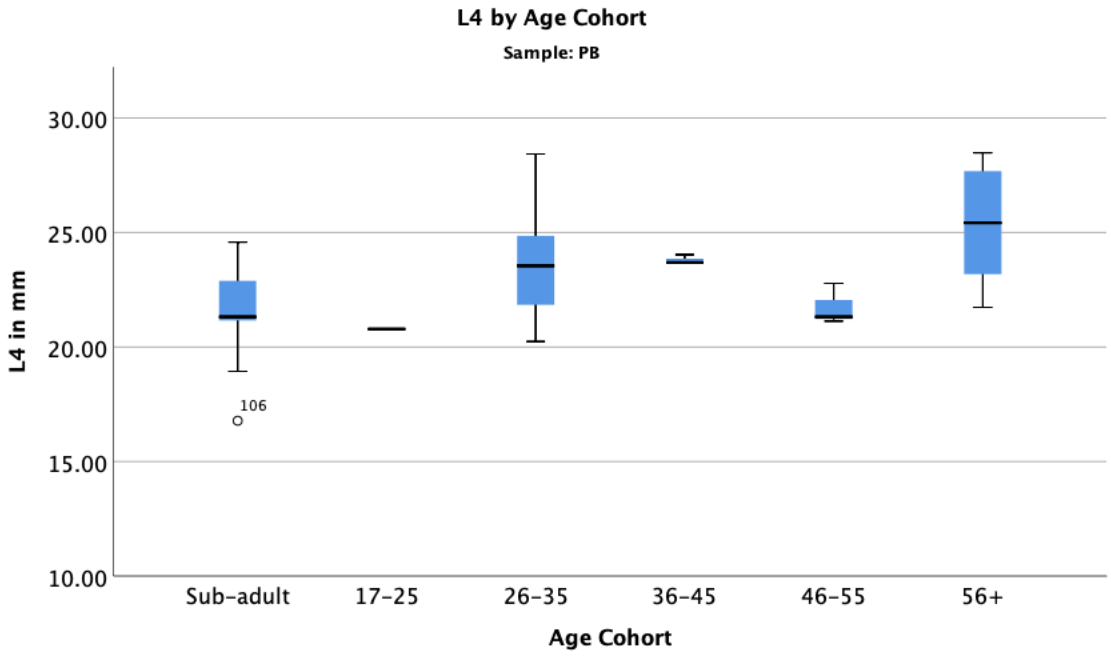

Figure 16: Boxplots of Pueblo Bonito L4 Interpedicular Diameter

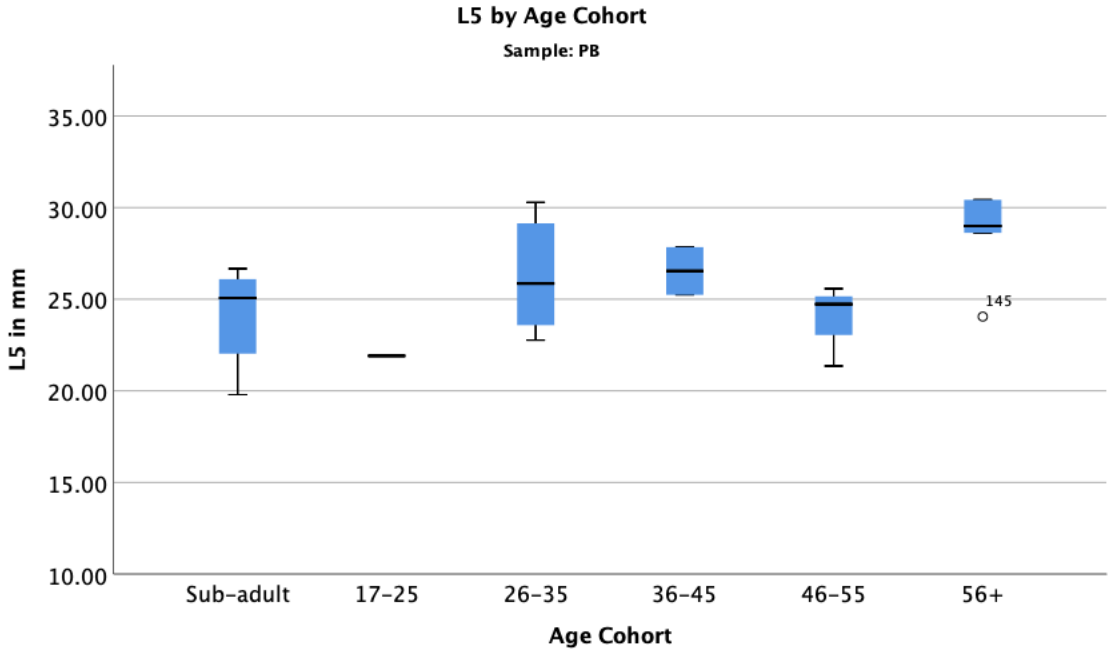

Figure 17: Boxplots of Pueblo Bonito L5 Interpedicular Diameter

Interpedicular Diameter: Hawikku

Table 2: Significant ANOVA Results for the Interpedicular Measurement of the Hawikku

Sample	Measurement	Vertebra	Age Cohort	P-Value	Figure
Hawikku	Interpedicular	C1	46-55+	0.044	18
Hawikku	Interpedicular	C2	Sub-Adult	0.013	19
Hawikku	Interpedicular	C2	46-55+	0.042	19
Hawikku	Interpedicular	C3	Sub-Adult	0.005	20

Figures 18-20 are box plots that depict the interquartile range of interpedicular diameter across the lifespan for each vertebral element where significant differences between age groups were found at the Hawikku site. Table 2 list the instances where significant differences in interpedicular diameter were found as well as the age cohorts where these differences appear and are maintained across all subsequent years of life at the Hawikku site. VNC measurement for significant results are displayed in the appendix (Table 12). Significant differences between age-at-death cohorts were found for three interpedicular diameters in four age cohorts at Hawikku. These instances reflect smaller interpedicular diameter in younger age cohorts and larger interpedicular diameter in older age cohorts.

Interpedicular diameter for C1 is smaller in the 46-55-year age cohort compared to the 56+ age cohort at Pueblo Bonito. However, Figure 18 of C1 shows no evidence for smaller interpedicular diameter at younger ages. Significantly smaller interpedicular

diameter was found in subadult compared to all later cohorts for C2 (Figure 19). The same trend is found for individuals in the 46-55 years age cohort compared to the 56+ years group for C2 (Figure 19). Finally, subadults have significantly smaller interpedicular diameter compared to all other cohorts for C3 (Figure 20).

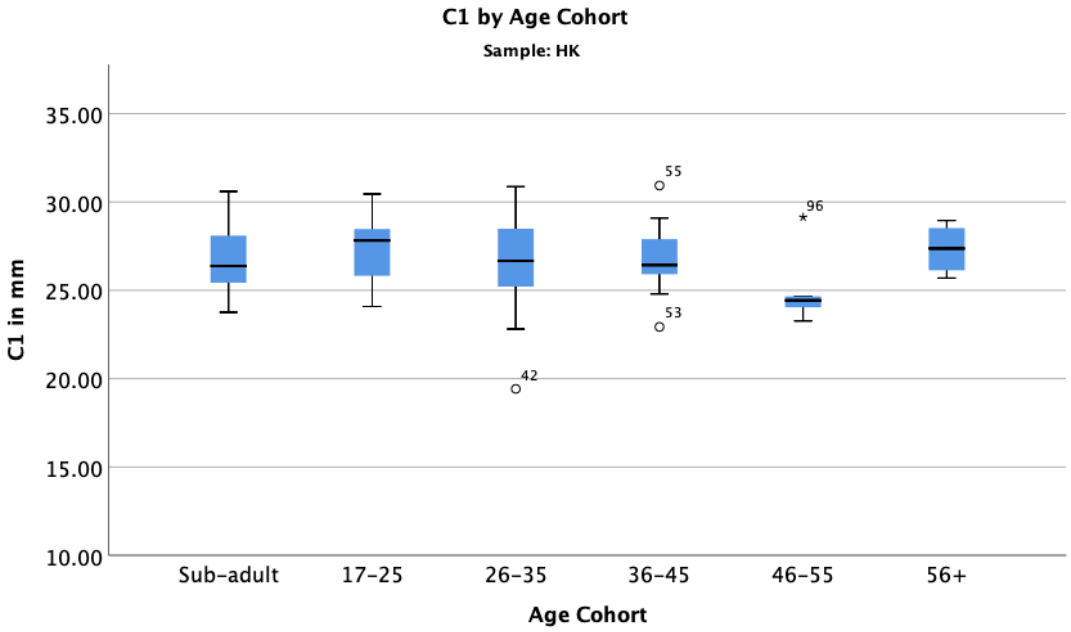

Figure 18: Boxplots of Hawikku C1 Interpedicular Diameter

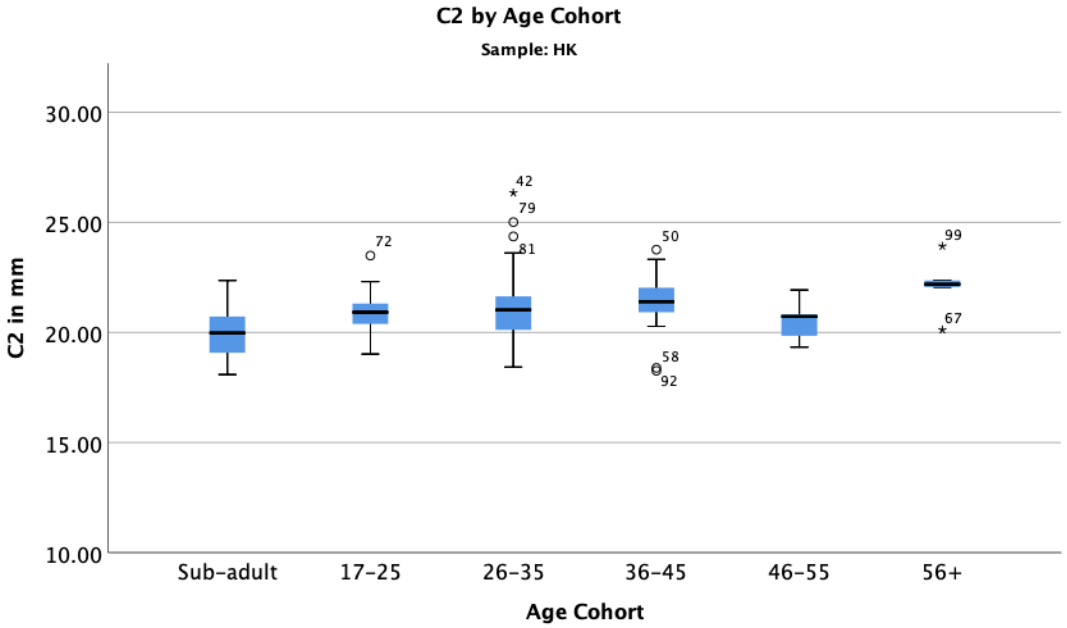

Figure 19: Boxplots of Hawikku C2 Interpedicular Diameter

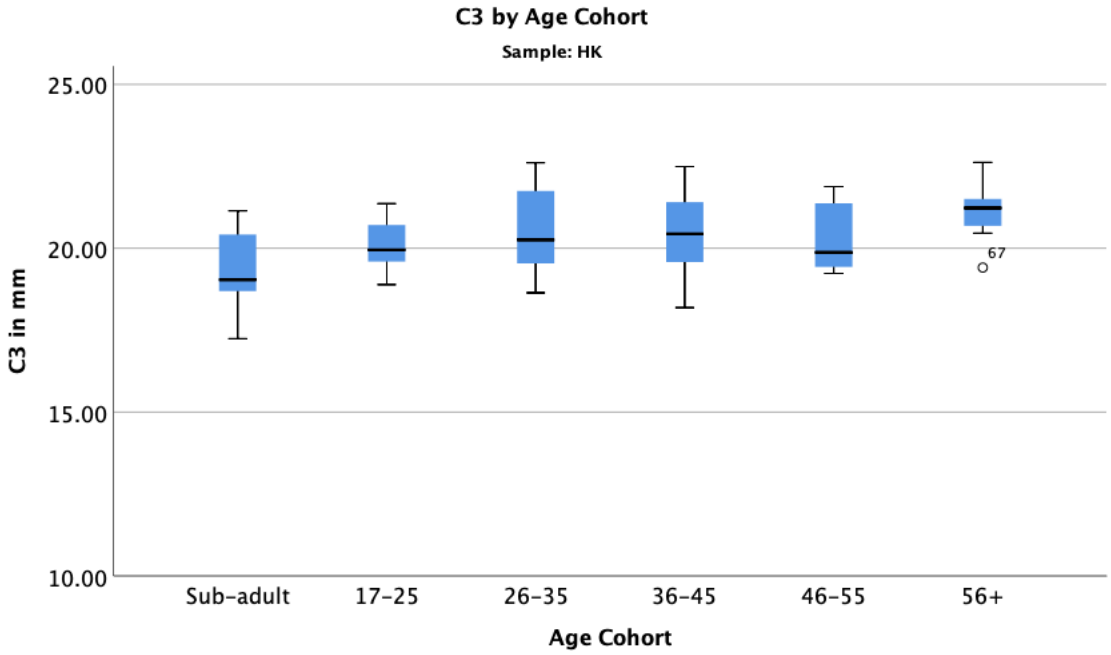

Figure 20: Boxplots of Hawikku C3 Interpedicular Diameter

Mid-Sagittal Diameter: Pueblo Bonito

Table 3: Significant ANOVA Results for the Midsagittal Measurement of the Pueblo Bonito

Sample	Measurement	Vertebra	Age at Death	P-Value	Figure
Pueblo Bonito	Midsagittal	C3	17-25	0.002	21
Pueblo Bonito	Midsagittal	C4	17-25	0.049	22
Pueblo Bonito	Midsagittal	T1	17-25	0.044	23
Pueblo Bonito	Midsagittal	T6	17-25	0.012	24
Pueblo Bonito	Midsagittal	T7	Sub-Adult	0.031	25
Pueblo Bonito	Midsagittal	T8	Sub-Adult	0.002	26
Pueblo Bonito	Midsagittal	T9	Sub-Adult	0.040	27
Pueblo Bonito	Midsagittal	T9	17-25	0.004	27
Pueblo Bonito	Midsagittal	T10	17-25	0.021	28
Pueblo Bonito	Midsagittal	L3	Sub-Adult	0.025	29
Pueblo Bonito	Midsagittal	L3	17-25	0.039	29
Pueblo Bonito	Midsagittal	L5	26-35	0.012	30

Box plots for comparisons where mid-sagittal diameters significantly differed between age cohorts are shown in Figures 21-30. In addition, Table 3 lists instances where mid-sagittal diameter differed between age cohorts by vertebrae and lists the age cohort where significant differences were found with all subsequent age categories. VNC measurement for significant results are displayed in the appendix (Table 13). Significant differences in mid-sagittal diameter were found in 12 comparisons between age groups for the Pueblo Bonito site. In two instances, smaller mid-sagittal diameters were associated with survival into older age cohorts, and in 10 instances smaller mid-sagittal diameters were observed in younger age cohorts.

Individuals in the 17-25 years age cohort do not differ from subadults and have significantly greater mid-sagittal diameter than those in older age groups for C3 and C4

(Figures 21-23). In three cases (T1, T6, T10,), individuals in the post subadult age cohort had significantly smaller mid-sagittal diameters from individuals in all additional age groups, but did not differ from subadults (Figures 23-24, 28). In one instance, individuals in the 26-35 years age cohort had significantly smaller mid-sagittal diameters than all older age classes but did not differ from younger age groups (Figure 30). In four instances subadults had significantly smaller mid-sagittal diameters than all older age groups, and in two of these instances, a smaller mid-sagittal diameter was also found in the 17-25 years age cohort compared to all future stages of the life course.

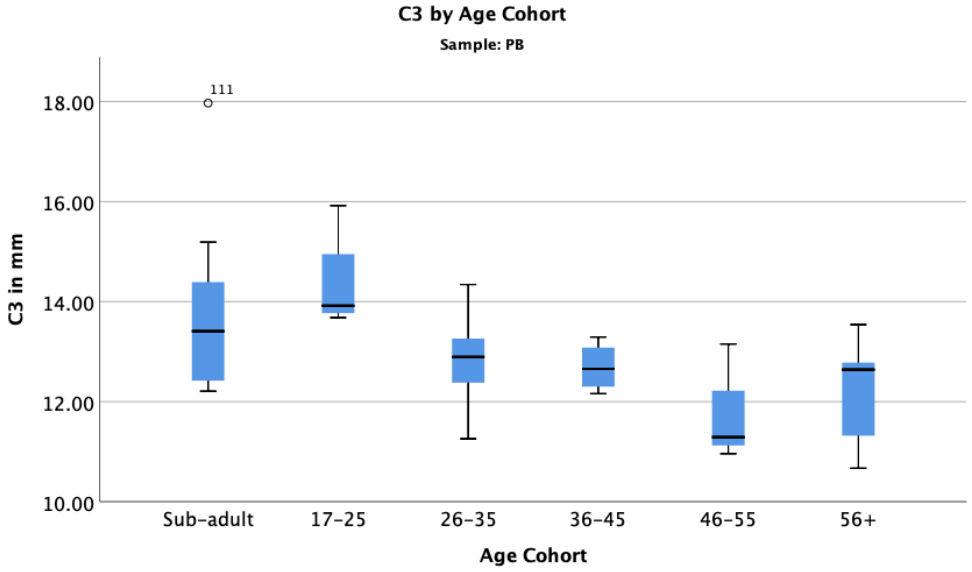

Figure 21: Boxplots of Pueblo Bonito C3 Midsagittal Diameter

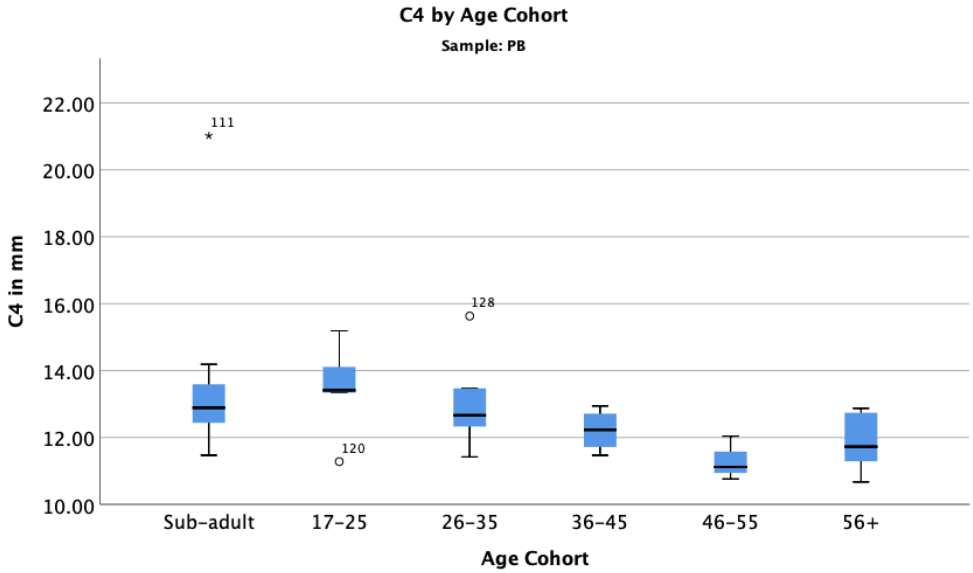

Figure 22: Boxplots of Pueblo Bonito C4 Midsagittal Diameter

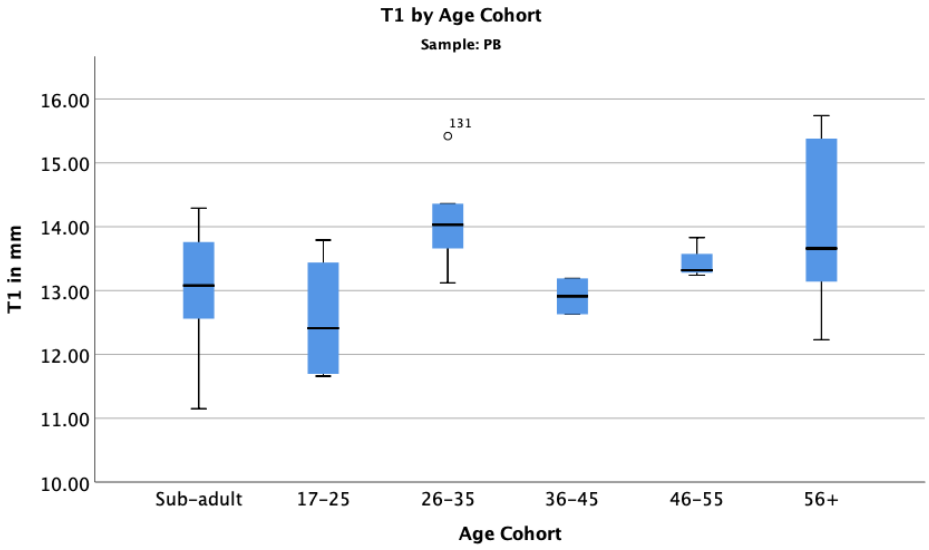

Figure 23: Boxplots of Pueblo Bonito T1 Midsagittal Diameter

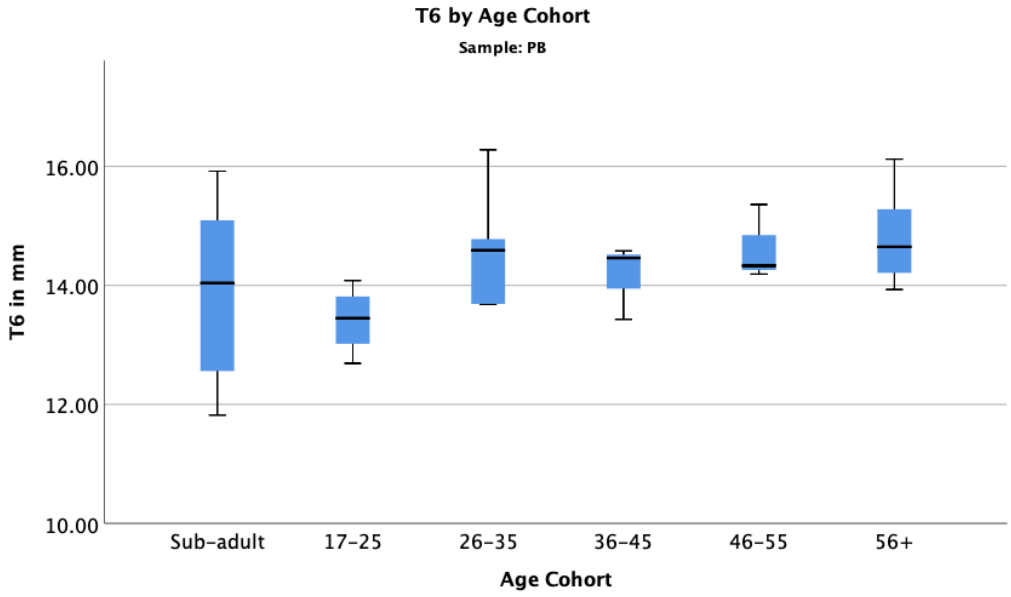

Figure 24: Boxplots of Pueblo Bonito T6 Midsagittal Diameter

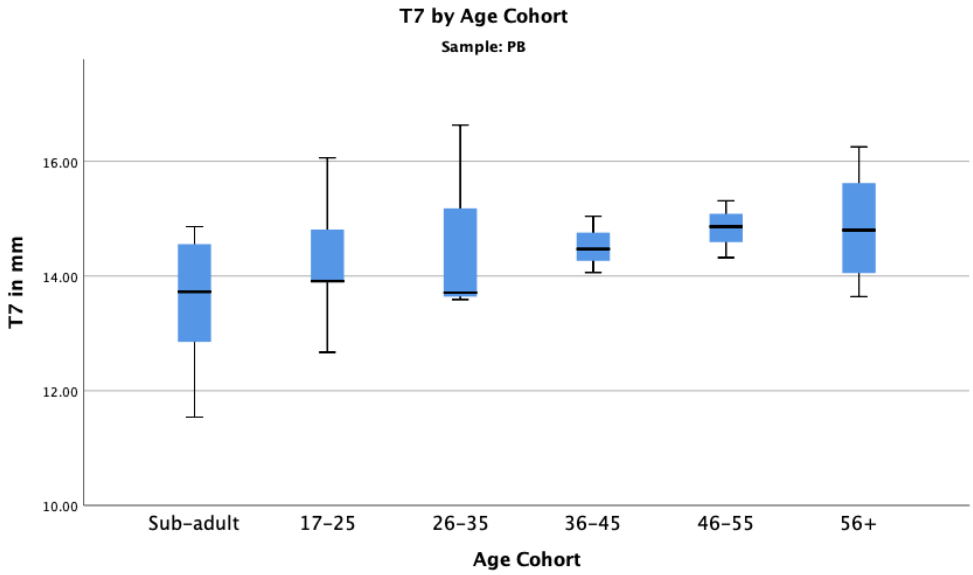

Figure 25: Boxplots of Pueblo Bonito T7 Midsagittal Diameter

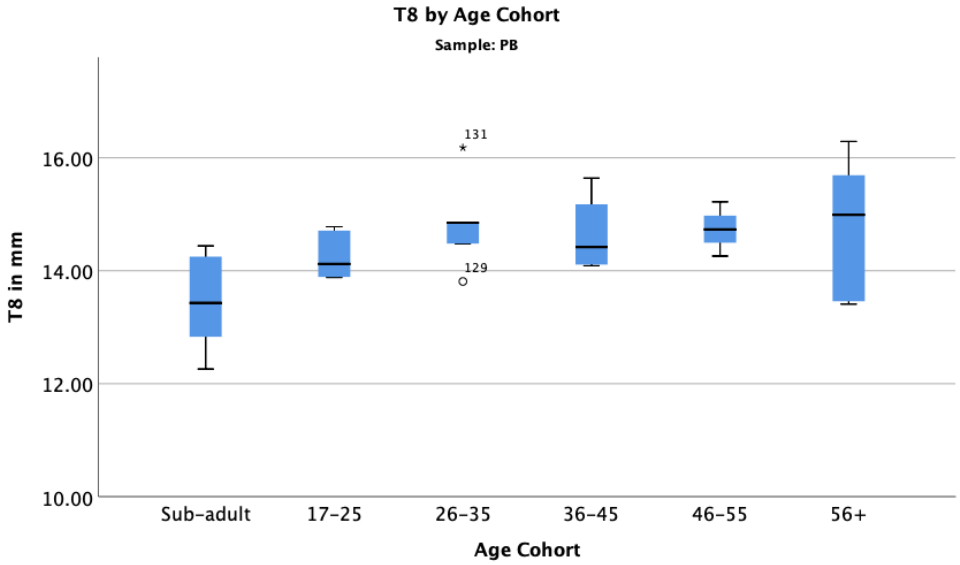

Figure 26: Boxplots of Pueblo Bonito T8 Midsagittal Diameter

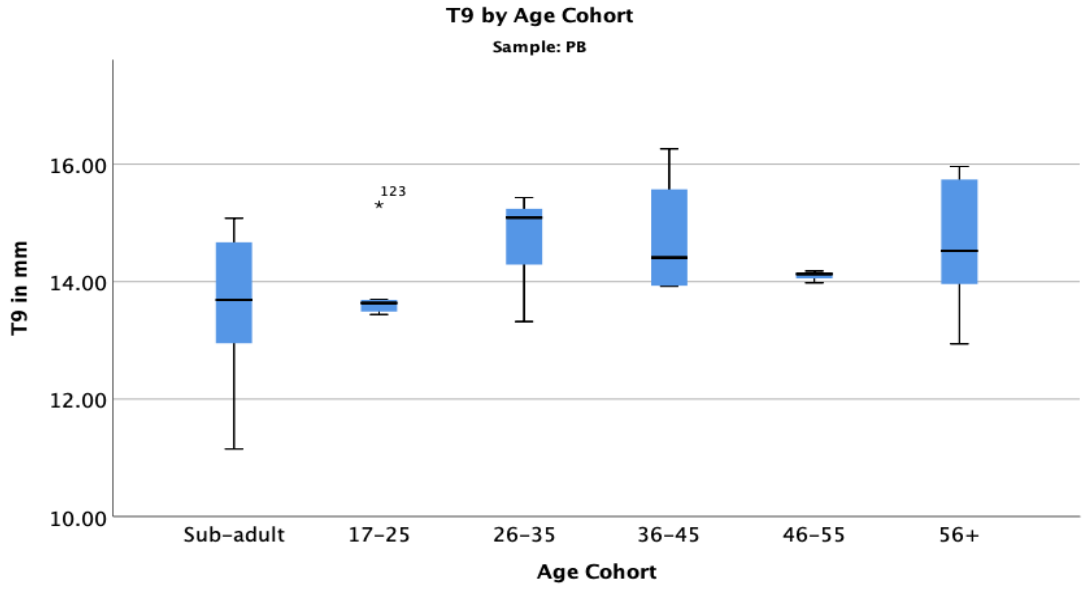

Figure 27: Boxplots of Pueblo Bonito T9 Midsagittal Diameter

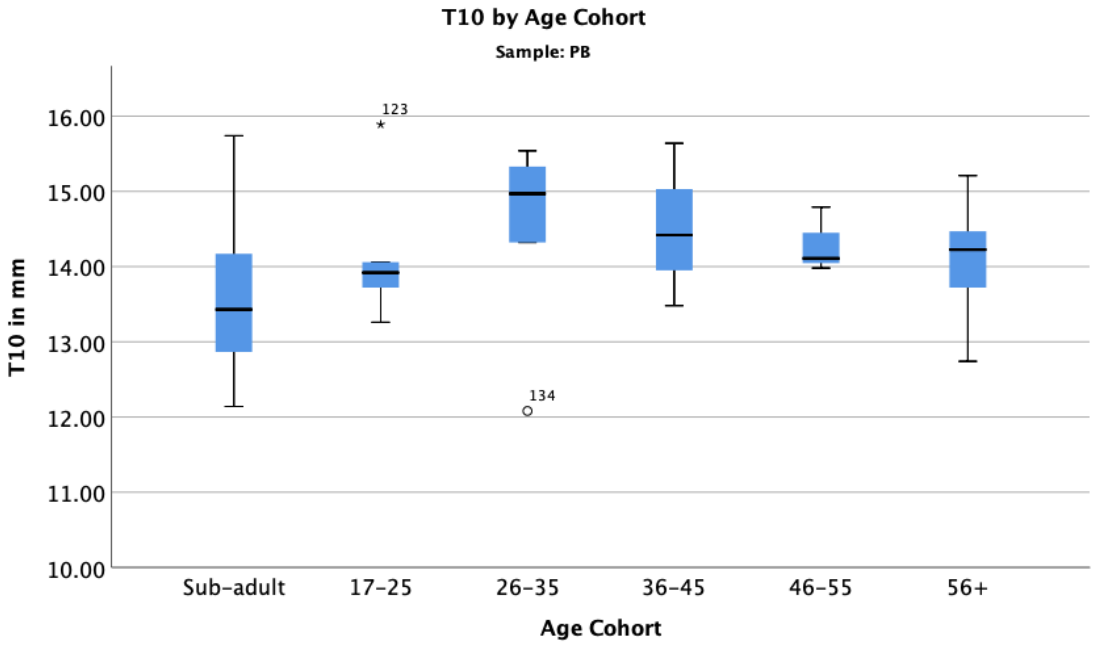

Figure 28: Boxplots of Pueblo Bonito T10 Midsagittal Diameter

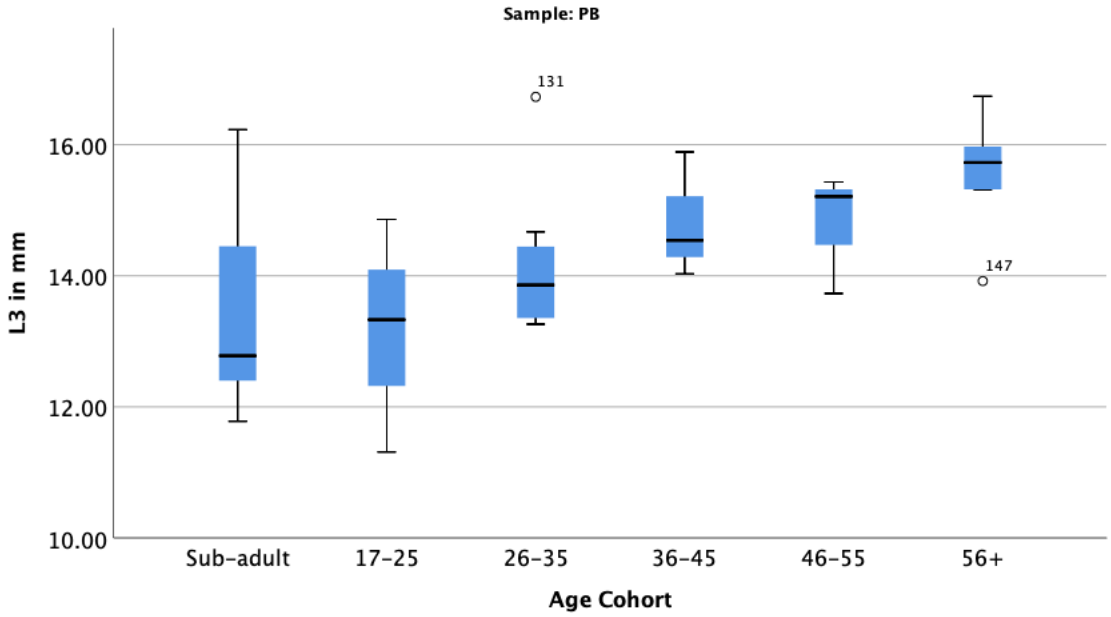

Figure 29: Boxplots of Pueblo Bonito L3 Midsagittal Diameter

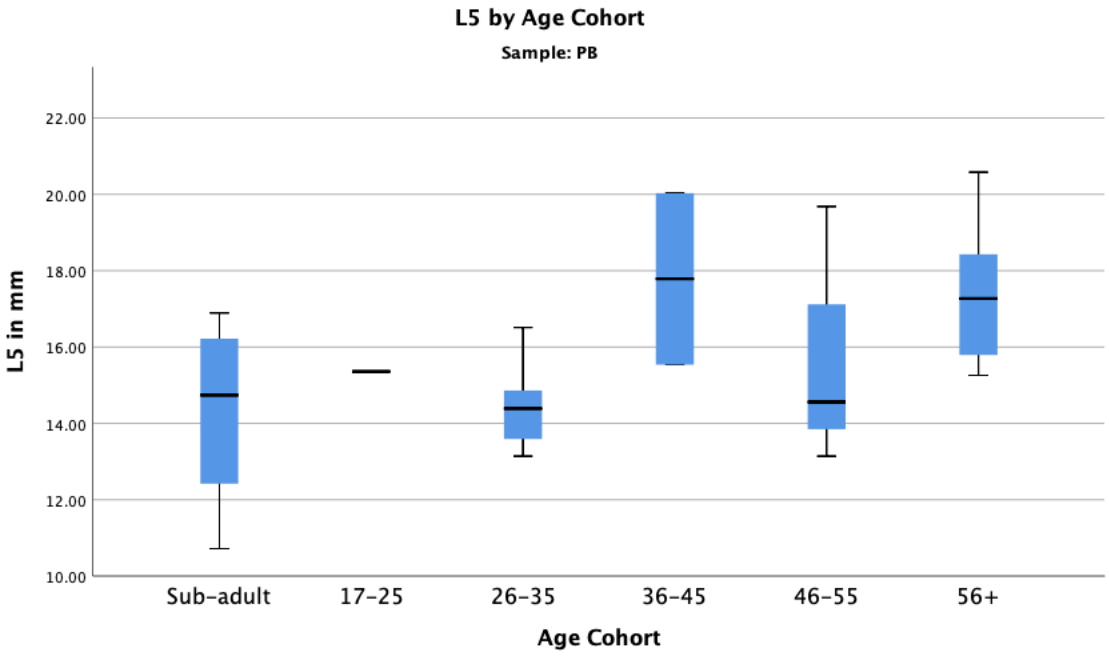

Figure 30: Boxplots of Pueblo Bonito L5 Midsagittal Diameter

Mid-Sagittal Diameter: Hawikku

Box plots depicting the interquartile ranges and 90th percentile distributions of mid-sagittal diameter for the Hawikku site may be found in Appendices as Figures 45-68 No significant differences between age cohorts were found in mid-sagittal diameter for any vertebrae at this locale.

Kaplan-Meier survival Analysis

The results of the Kaplan-Meier survival analysis for grouped and non-grouped VNCS for the Hawikku (n=60) and Pueblo Bonito (n=110) are shown below in Table 4-9. Significant results ($P \leq 0.05$) are bolded. Significance was found for the Kaplan-Meier survival analysis of the interpedicular measurements of the Pueblo Bonito sample for T3-

T5, T6-T8, T9-11, T12, L1, L2-L4 (Table 4). Individuals from Pueblo Bonito with a smaller interpedicular diameter of the VNC had a decreased life span in comparison with those who had a larger VNC. Significance was found for the Kaplan-Meier survival analysis of the midsagittal measurements of the Pueblo Bonito sample for C3-C4, T2, T3-5, T6-T8, and L2-L4 (Table 5). Individuals from Pueblo Bonito with a smaller midsagittal diameter of the VNC had a decreased life span in comparison with those who had a larger VNC. Significance was found for the Kaplan-Meier survival analysis of the interpedicular measurements of the Hawikku sample for C3-C4, C5-C6, and T9-11 (Table 6). Individuals from the Hawikku with a smaller interpedicular diameter of the VNC had a decreased life span in comparison with those who had a larger VNC There was no significance found for the Kaplan-Meier survival analysis of the midsagittal measurements of vertebra from the Hawikku sample (Table 7). Results of the Kaplan-Meier survival analysis including mean survival time are shown below in Table 8 and Table 9 for all statistically signficant results. Except for two cases all individuals that had smaller VNC diameters lived a shorter amount of time then individuals with a larger VNC. Additionally, all of the significant results of the survival analysis are graphically displayed in Figures 31-44.

Table 4: Kaplan-Meier survival Analysis results for the Interpedicular measurements of the Pueblo Bonito sample.

Population	Measurement	Vertebrae	n	Chi-Squared	P value
Pueblo Bonito	Interpedicular	C1	21	χ^2=0.497	0.481
Pueblo Bonito	Interpedicular	C2	34	χ^2=0.008	0.930
Pueblo Bonito	Interpedicular	C3-4	65	χ^2=0.865	0.352
Pueblo Bonito	Interpedicular	C5-6	61	χ^2=1.218	0.270

Pueblo Bonito	Interpedicular	C-7	30	χ^2=0.945	0.331
Pueblo Bonito	Interpedicular	T1	31	χ^2=3.238	0.072
Pueblo Bonito	Interpedicular	T2	29	χ^2=2.087	0.149
Pueblo Bonito	Interpedicular	T3-5	82	χ^2=14.324	**0.0001**
Pueblo Bonito	Interpedicular	T6-8	87	χ^2=4.444	**0.035**
Pueblo Bonito	Interpedicular	T9-11	101	$\chi^2$4=0.124	**0.042**
Pueblo Bonito	Interpedicular	T-12	35	χ^2=5.991	**0.014**
Pueblo Bonito	Interpedicular	L1	34	χ^2=4.441	**0.035**
Pueblo Bonito	Interpedicular	L2-4	103	χ^2=16.351	**0.0001**
Pueblo Bonito	Interpedicular	L5	24	χ^2=1.702	0.192

Table 5: Kaplan-Meier survival Analysis results for the Midsagittal measurements of the Pueblo Bonito sample.

Population	Measurement	Vertebrae	n	Chi-Squared	P value
Pueblo Bonito	Midsagittal	C1	20	χ^2=0.998	0.318
Pueblo Bonito	Midsagittal	C2	33	χ^2=0.642	0.423
Pueblo Bonito	Midsagittal	C3-4	71	χ^2=18.163	**0.0001**
Pueblo Bonito	Midsagittal	C5-6	59	χ^2=1.505	0.220
Pueblo Bonito	Midsagittal	C-7	30	χ^2=0.945	0.331
Pueblo Bonito	Midsagittal	T1	31	χ^2=0.929	0.335
Pueblo Bonito	Midsagittal	T2	28	χ^2=6.801	**0.009**
Pueblo Bonito	Midsagittal	T3-5	82	χ^2=5.115	**0.024**
Pueblo Bonito	Midsagittal	T6-8	87	χ^2=8.112	**0.004**
Pueblo Bonito	Midsagittal	T9-11	101	χ^2=2.658	0.103
Pueblo Bonito	Midsagittal	T-12	35	χ^2=1.065	0.302
Pueblo Bonito	Midsagittal	L1	32	χ^2=3.776	0.052
Pueblo Bonito	Midsagittal	L2-4	99	χ^2=13.773	**0.001**
Pueblo Bonito	Midsagittal	L5	33	χ^2=2.284	0.131

Table 6: Kaplan-Meier survival Analysis results for the Interpedicular measurements of the Hawikku sample.

Sample	Measurement	Vertebrae	n	Chi-Squared	P value
Hawikku	Interpedicular	C1	75	χ^2= 0.022	0.881
Hawikku	Interpedicular	C2	75	χ^2=2.644	0.104
Hawikku	Interpedicular	C3-4	150	χ^2=4.455	**0.035**

Hawikku	Interpedicular	C5-6	155	χ^2=8.763	**0.003**
Hawikku	Interpedicular	C-7	71	χ^2=0.016	0.899
Hawikku	Interpedicular	T1	75	χ^2=0.014	0.906
Hawikku	Interpedicular	T2	76	χ^2=1.380	0.240
Hawikku	Interpedicular	T3-5	196	χ^2=0.947	0.331
Hawikku	Interpedicular	T6-8	187	χ^2=0.381	0.537
Hawikku	Interpedicular	T9-11	228	χ^2=4.065	**0.044**
Hawikku	Interpedicular	T-12	82	χ^2=0.059	0.809
Hawikku	Interpedicular	L1	84	χ^2=0.008	0.930
Hawikku	Interpedicular	L2-4	264	χ^2=0.628	0.428
Hawikku	Interpedicular	L5	78	χ^2=0.450	0.502

Table 7: Kaplan-Meier survival Analysis results for the Midsagittal measurements of the Hawikku sample.

Population	Measurement	Vertebrae	n	Chi-Squared	P value
Hawikku	Midsagittal	C1	70	χ^2=0.787	0.375
Hawikku	Midsagittal	C2	72	χ^2=3.160	0.075
Hawikku	Midsagittal	C3-4	146	χ^2=3.267	0.071
Hawikku	Midsagittal	C5-6	150	χ^2=3.371	0.066
Hawikku	Midsagittal	C-7	71	χ^2=0.016	0.899
Hawikku	Midsagittal	T1	76	χ^2=0.225	0.635
Hawikku	Midsagittal	T2	76	χ^2=0.101	0.751
Hawikku	Midsagittal	T3-5	189	χ^2=0.249	0.618
Hawikku	Midsagittal	T6-8	183	χ^2=0.521	0.470
Hawikku	Midsagittal	T9-11	217	χ^2=0.152	0.697
Hawikku	Midsagittal	T-12	73	χ^2=0.047	0.828
Hawikku	Midsagittal	L1	81	χ^2=0.780	0.377
Hawikku	Midsagittal	L2-4	244	χ^2=1.232	0.267
Hawikku	Midsagittal	L5	77	χ^2=0.206	0.650

Table 8: Interpedicular Measurements Mean survival time for both samples significant results

Sample	Vertebra	VNC size	Mean Survival	95% confidence interval	P-Value
Hawikku	C3-C4	Small	30.381	27.225-33.537	0.035

		Large	35.604	31.915-39.293	
Hawikku	C5-C6	Small	29.191	20.318-38.063	0.003
		Large	41.366	34.380-48.531	
Hawikku	T9-T11	Small	35.061	32.462-37.660	0.044
		Large	31.658	29.251-34.066	
Pueblo Bonito	T3-T5	Small	25.698	19.45-31.945	0.0001
		Large	45.024	38.734-51.315	
Pueblo Bonito	T6-T8	Small	30.080	23.641-36.518	0.035
		Large	40.353	34.950-45.757	
Pueblo Bonito	T9-T11	Small	29.406	24.003-34.809	0.042
		Large	37.870	32.421-43.319	
Pueblo Bonito	T12	Small	24.989	16.786-33.193	0.014
		Large	42.325	32.723-51.927	
Pueblo Bonito	LI	Small	25.445	16.932-33.959	0.035
		Large	43.667	32.972-54.361	
Pueblo Bonito	L2-L4	Small	23.292	18.187-28.399	0.0001
		Large	41.563	36.024-47.102	

Table 9: Midsagittal Measurements Mean survival time for both samples significant results

Sample	**Vertebra**	**VNC size**	**Mean Survival**	**95% confidence interval**	**P-Value**
Pueblo Bonito	C3-C4	Small	40.091	33.255-46.927	0.0001
		Large	21.586	16.788-26.383	
Pueblo Bonito	T2	Small	21.758	11.239-32.278	0.009
		Large	42.300	31.944-52.606	
Pueblo Bonito	T3-T5	Small	28.346	21.481-35.211	0.024
		Large	41.723	35.304-48.143	
Pueblo Bonito	T6-T8	Small	28.671	22.819-34.524	0.004
		Large	41.713	35.973-47.453	
Pueblo Bonito	L2-L4	Small	23.510	18.488-28.531	0.001
		Large	40.975	34.919-47.031	

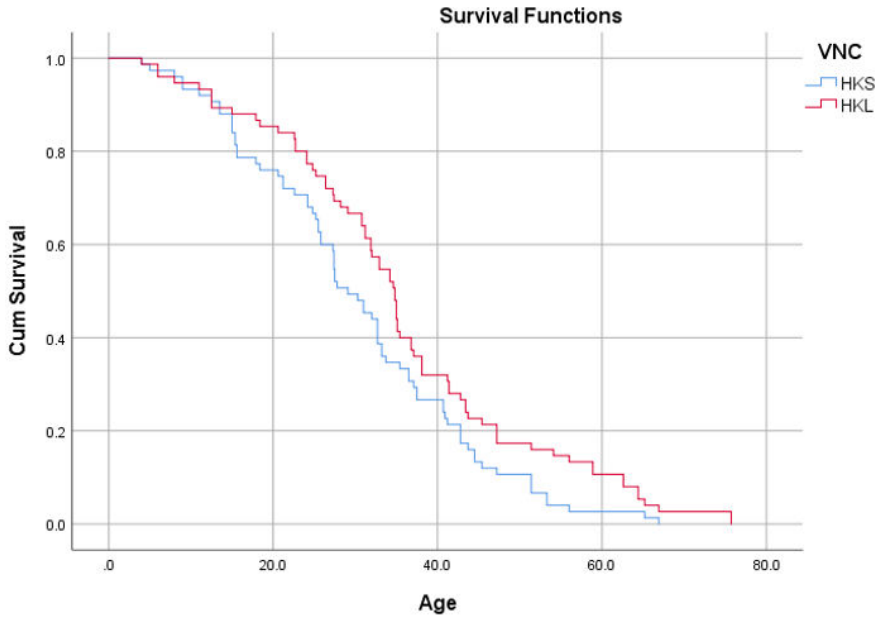

Figure 31: Hawikku interpedicular length of C3-4 (n=150)

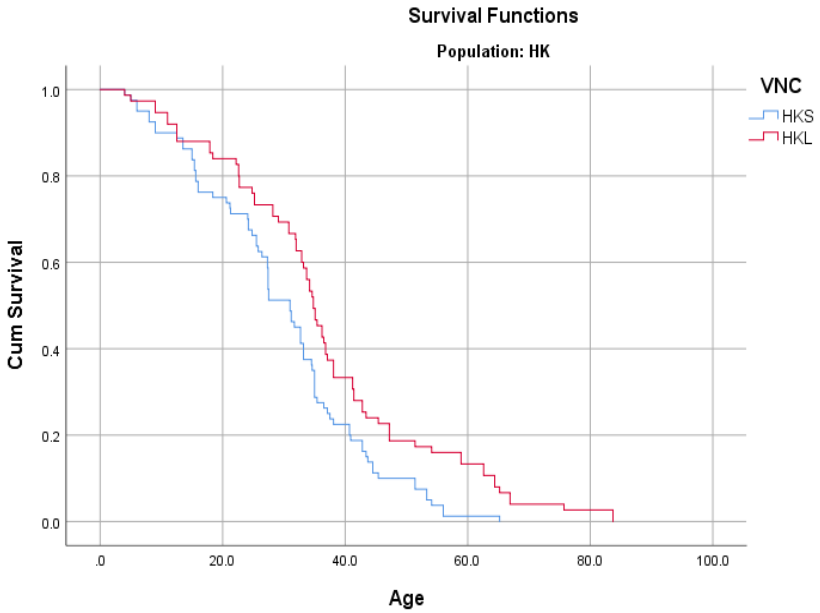

Figure 32: Hawikku interpedicular length of C5-6 (n=155)

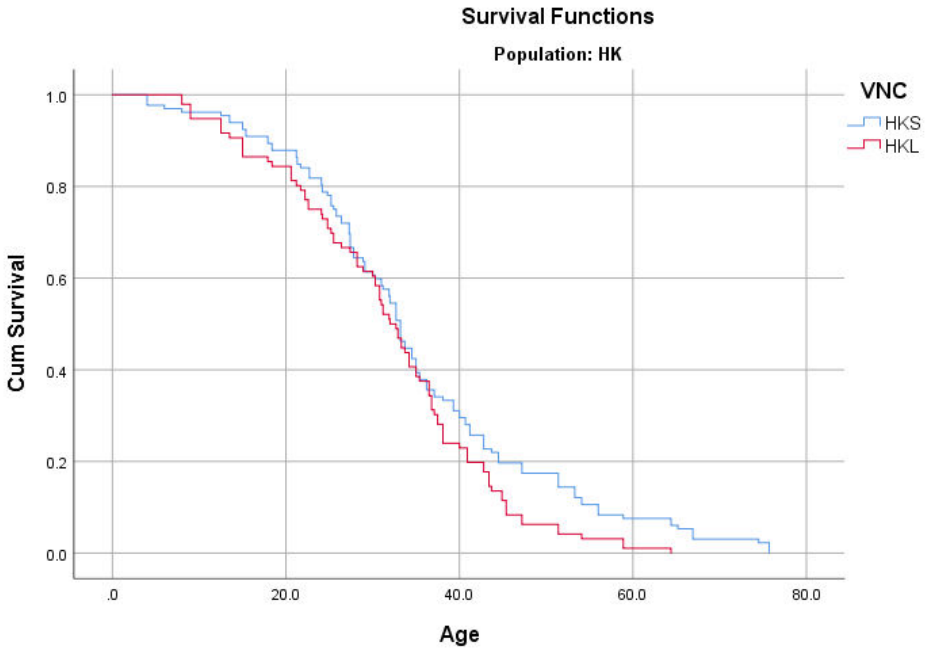

Figure 33: Hawikku interpedicular length of T9-T11 (n=228)

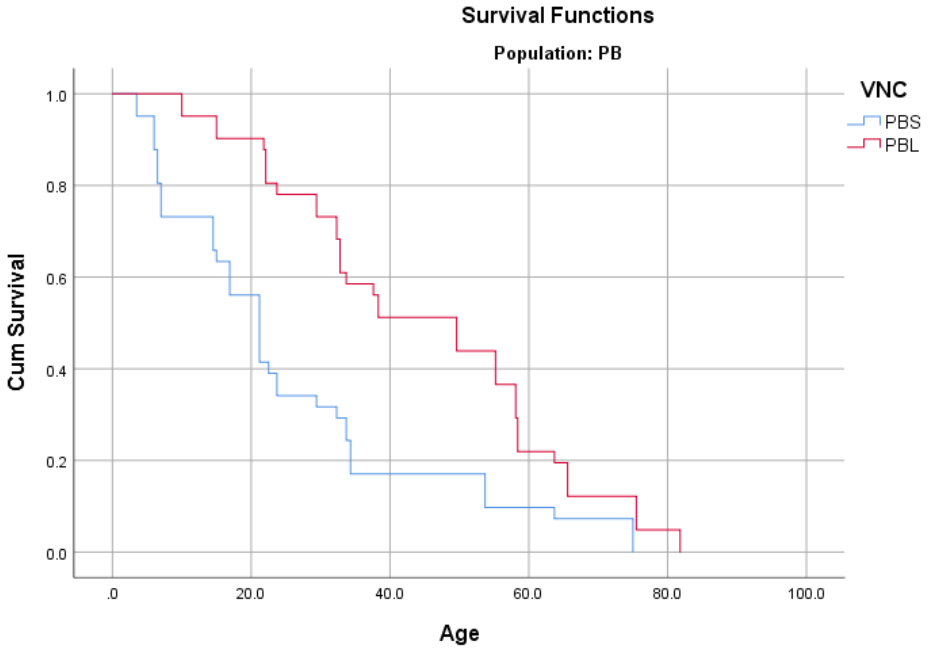

Figure 34: Pueblo Bonito interpedicular length of T3-T5 (n=82)

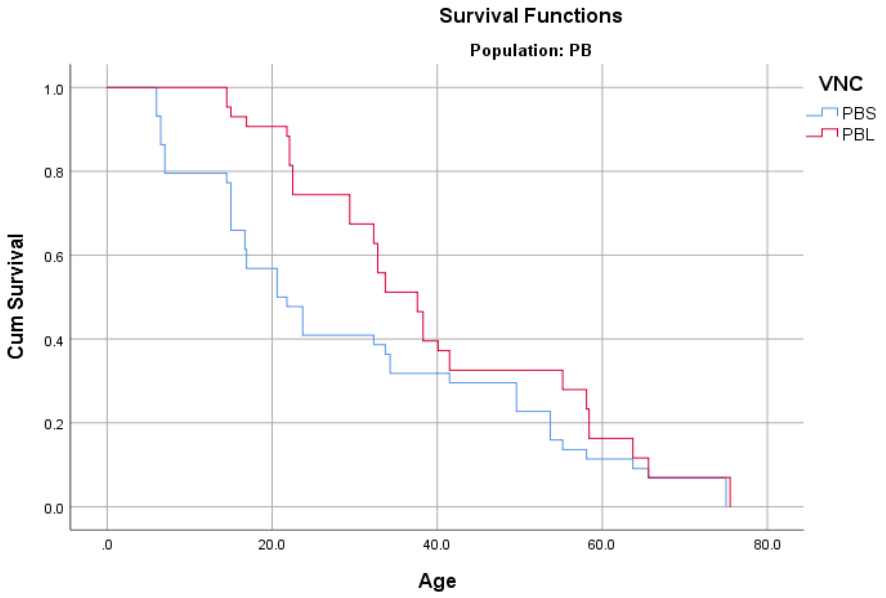

Figure 35: Pueblo Bonito interpedicular length of T6-T8 (n=87)

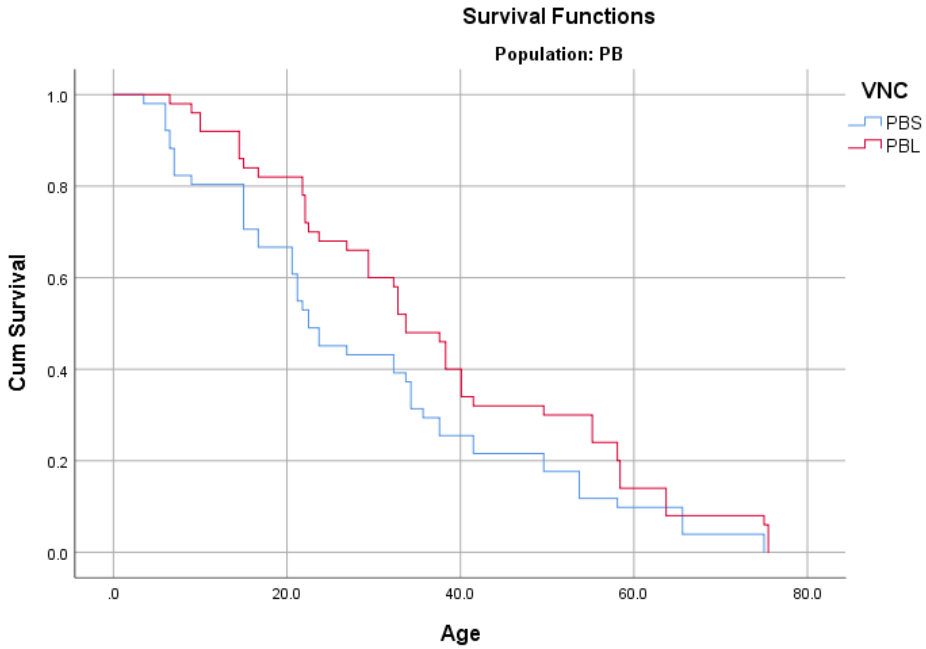

Figure 36: Pueblo Bonito interpedicular length of T9-T11 (n=101)

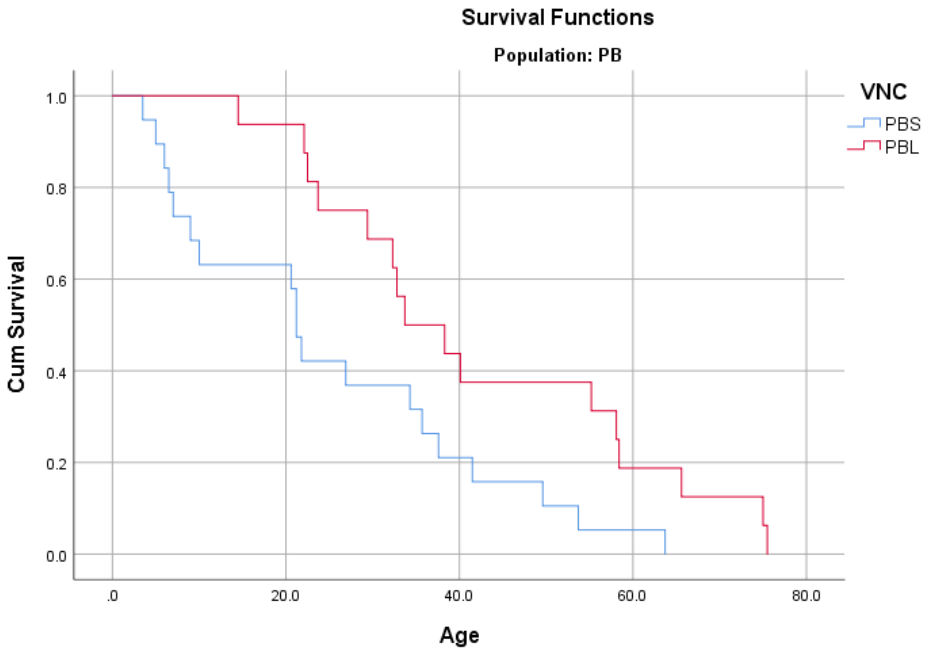

Figure 37: Pueblo Bonito interpedicular length of T12 (n=35)

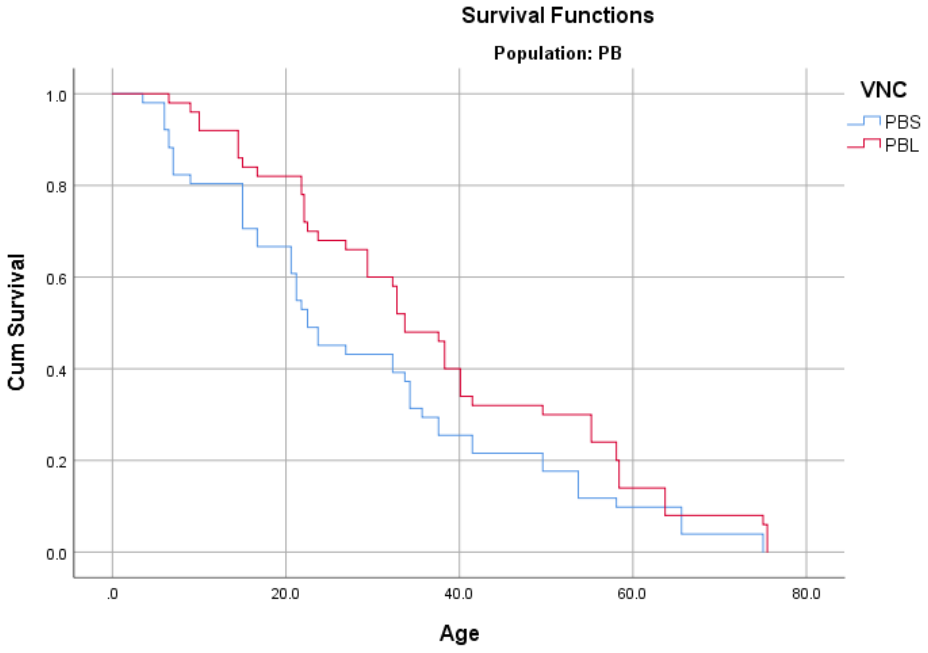

Figure 38: Pueblo Bonito interpedicular length of L1 (n=34)

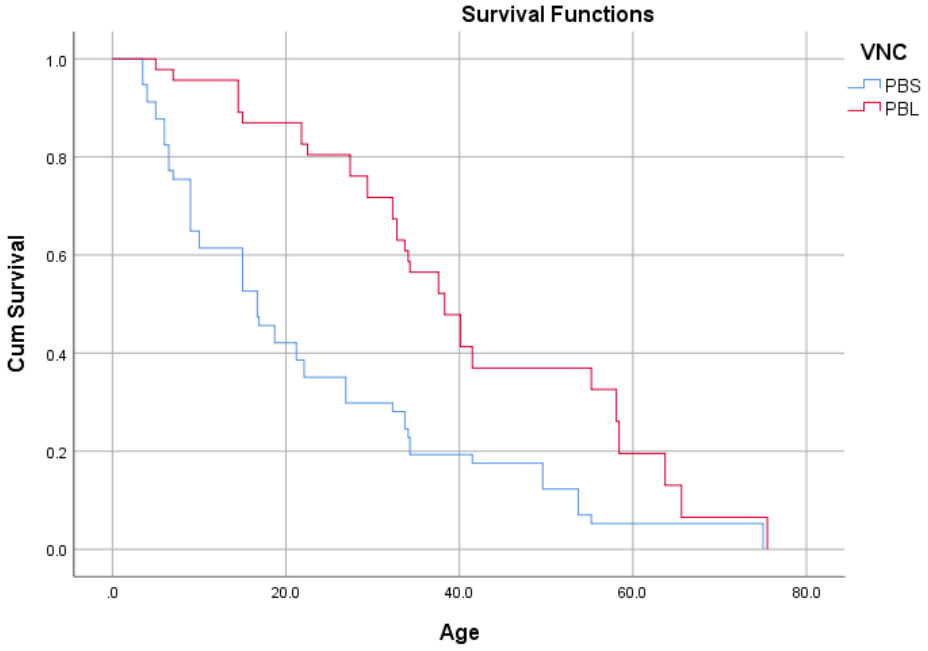

Figure 39: Pueblo Bonito interpedicular length of L2-4 (n=103)

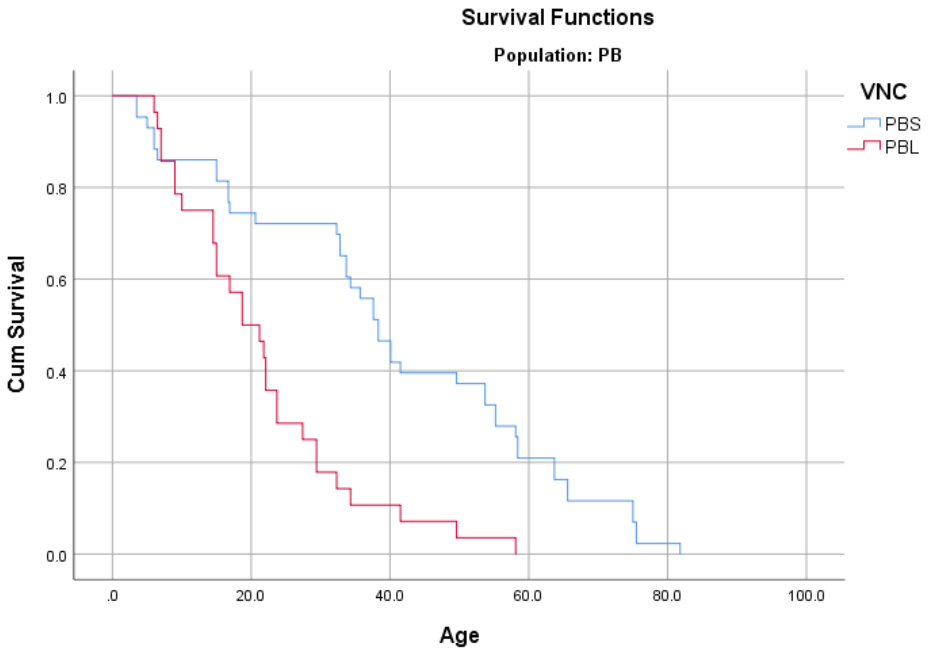

Figure 40: Pueblo Bonito midsagittal length C3-4 (n=71)

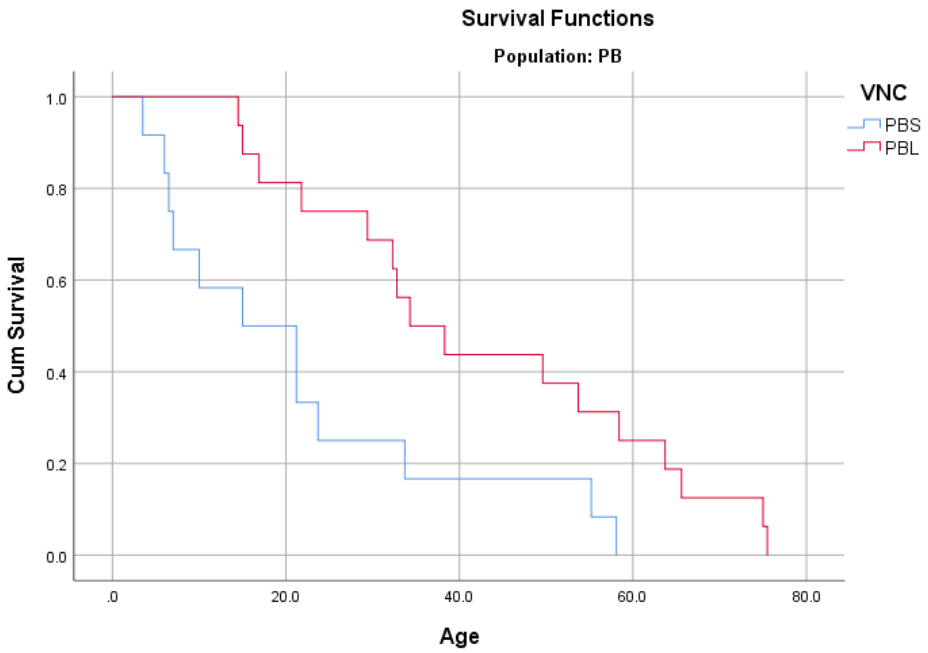

Figure 41: Pueblo Bonito midsagittal length T2 (n=28)

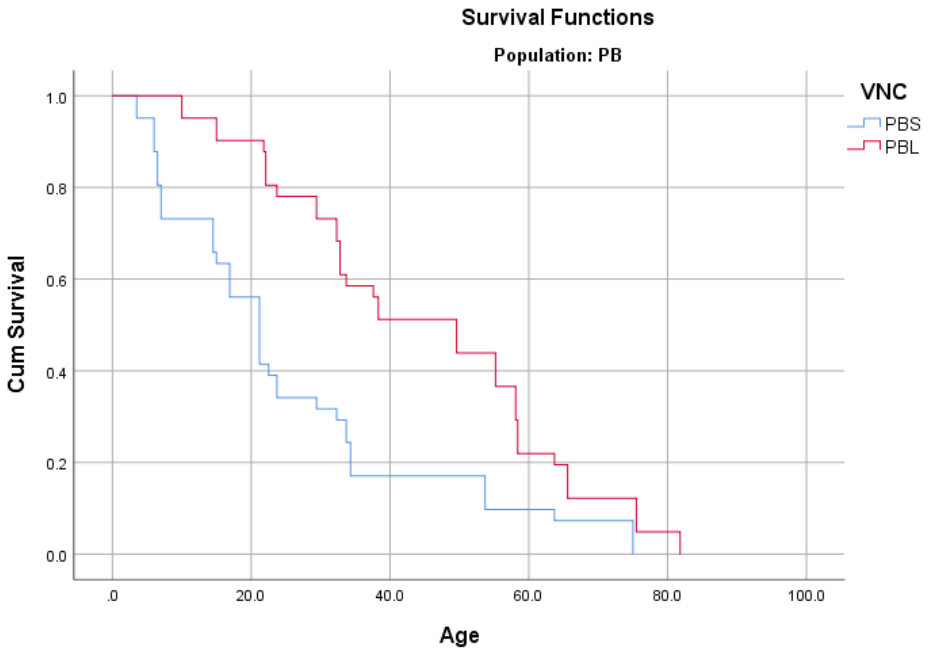

Figure 42: Pueblo Bonito midsagittal length T3-5 (n=82)

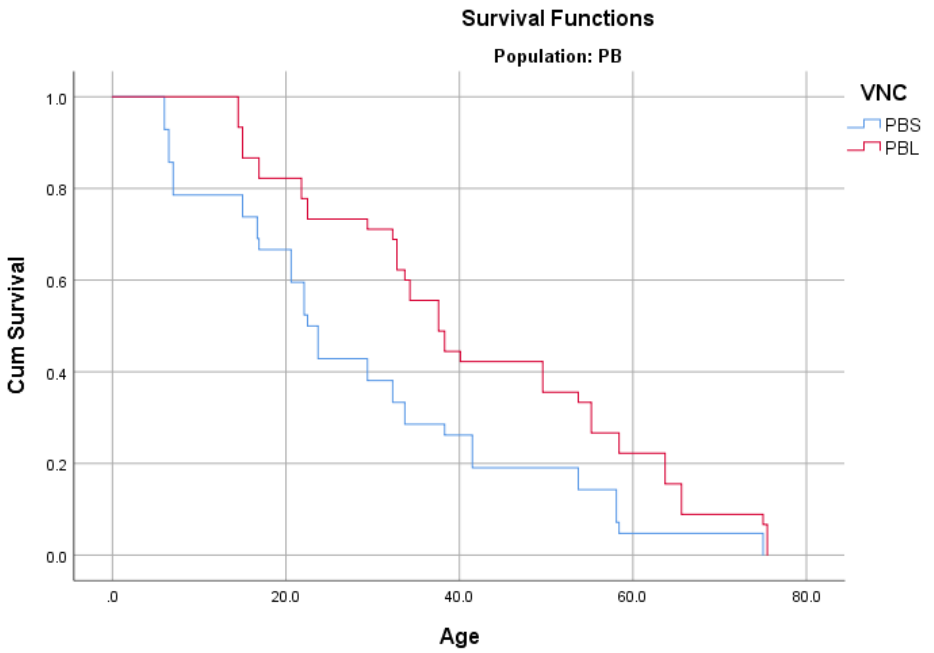

Figure 43: Pueblo Bonito midsagittal length T6-8 (n=87)

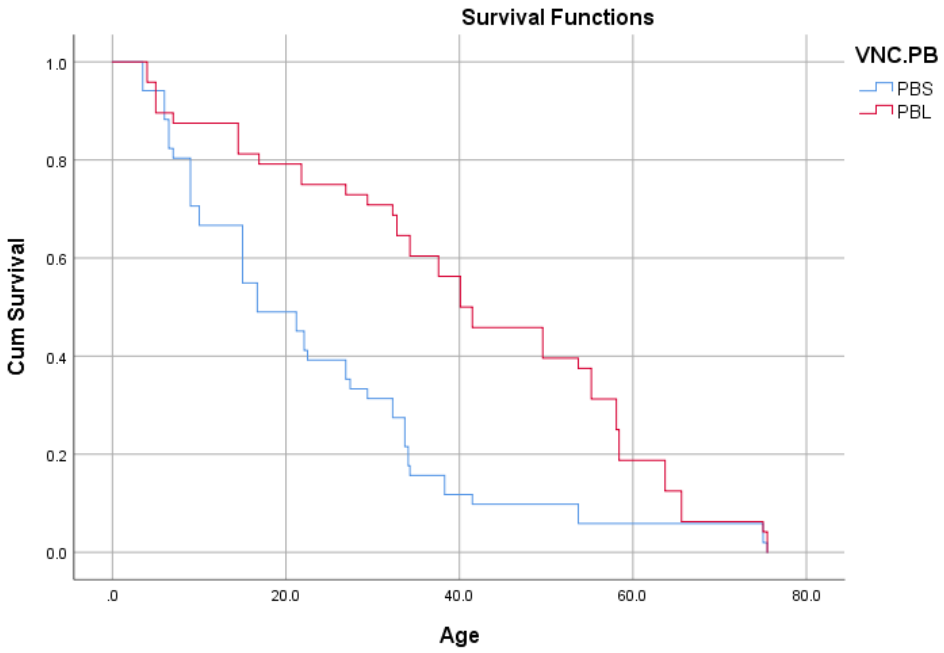

Figure 44: Pueblo Bonito midsagittal length L2-4 (n=99)

CHAPTER SIX: DISCUSSION

Vertebral Neural Canal Growth Disruptions and Mortality

Results from this thesis are consistent with previous studies in demonstrating that VNC growth disruptions may be associated with mortality risk. Precious research has shown that individuals in the 15-25 year age cohort had significantly smaller vertebral neural canals than those surviving into older age groups at the Dickson Mounds (AD 950-1300) site (Clark et al., 1986). Samples from Fisher Gate cemetery (10th-15th centuries), London demonstrate that significantly smaller VNC diameter is found in individuals 17-25 years compared with later age cohorts (Watts, 2011). Individuals buried at St. Peter's Church (1705-1855) in Barton-upon-Humber, United Kingdom expressed growth disruptions of the VNC in the 17-25 year compared to 36+ year age cohorts (Watts, 2013a).

In the current study, the vast majority of growth disruptions in VNC diameter were found between subadult or 17-25 year age cohorts compared to older age groups. In fact, 93% of all significant ANOVA tests identified the youngest age group as significantly smaller than all older samples. In this sense, where VNC growth disruptions are found,

there is gathering evidence for increasing mortality risk in association with surviving this event.

Previous studies have also explored mortality risk and VNC growth disruptions using hazards analysis (Watts, 2013b). The work found evidence for increased risk of death among individuals with reduced VNC size. Similar results were found by this study where survivor analysis identified 15 cases where suppressed VNC growth was associated with elevated mortality risk. One-way ANOVA tests found that 7/15 of these instances were attributable to smaller VNC diameter in individuals who did not survive beyond 16 years of age (Table 1-3). Examination of survivor curves further illustrate that an additional 7/14 individuals with reduced VNCs had exacerbated morality beginning no later than 17 years of age. These results provide further support for a relationship between VNC growth disruptions and mortality risk (Table 8-9).

A peculiar result that occurred in this data is the significance that is found in the analysis of the C3-C4 (Figure 40). In this analysis individuals with a smaller VNC had a mean survival of 40 years and those with a larger canal size had a mean survival of 21 years. This does not fit the trends found in previous research about the relationship between VNC and survival time. There are some explanations for this anomaly that may help expand on the issue. The cervical vertebra reach the maximum size around the age of 4, therefore it is possible that the early life stress that was captured, or not captured, in these vertebrae could have been overshadowed by catastrophic demographic events such as colonially introduced epidemics. Additionally, a more comprehensive study of each of the individuals may be needed to further elucidate this anomaly. It is also completely

possible that those with a smaller VNC had a lower risk of mortality in this instance because of some sort of energy sparring event. However any conclusions about the result of this analysis requires additionally research into the skeletal development of the VNC.

Measurement and Mortality Risk: Interpedicular and Mid-Sagittal Diameters

A substantially greater number of interpedicular diameters were observed in association with mortality risk when compared to midsagittal diameters. Significance was found in 25 cases for the interpedicular in comparison to 12 cases for the midsagittal diameter. The Kaplan-Meier survival analysis showed evidence of mortality risk in association with the VNC in 9 cases for the interpedicular diameter and 5 cases for the midsagittal diameter. In this study, interpedicular diameters were twice as likely to be growth suppressed in younger age groups and express reduced survivorship. These results both mirror and contrast with earlier studies. For example, one study at the Fisher Gate Cemetery (10th-15th centuries) found no evidence of mid-sagittal VNC growth disruption between age groups, while interpedicular vertebral growth disruption were found in the earliest age groups (Watts, 2011). Similarly, the number of instances where interpedicular diameter is associated with increased mortality risk in twice the number of instances than mid-sagittal diameter. VNC was significantly smaller in younger age cohorts for 24 interpedicular dimensions and zero midsagittal dimensions (Clark et al., 1986). However, a sign test demonstrated that the youngest age group still consistently had the smallest VNC diameters in the sample. By contrast two studies found equivalent numbers of mortality risk in association with interpedicular compared to mid-sagittal diameters (Amoroso & Garcia, 2018).

Different results between the interpedicular and midsagittal diameter makes sense when considering growth patterns these dimensions. There is evidence to suggest that Interpedicular diameter may continue to remodel until the onset of adolescence (Watts, 2013a). However, there is evidence that suggest that the interpedicular canal achieves fusion during the first three years of life (Newman & Gowland, 20015). The midsagittal diameter ceases growth roughly around the age of five years. In contrast instances of stress that are found in the interpedicular diameter could have occurred later then the first five years of life, and possibly up until adolescence. The longer period of vertebral growth may allow for a greater window to capture stressful events. The results of this study show suggest that the interpedicular diameter may have a longer time span for growth that allows it to capture more stressful events that contribute to mortality. This is exemplified by interpedicular diameter showing considerably more evidence of growth disruptions then the midsagittal diameter. These results may provide support for the accumulative stress model. Individuals that have experienced repeated stress events, especially earlier in the lifecycle, have an increased risk of mortality (Brown, 2016; Guerrant et al., 2002; Guerrant et al, 2008; Painter et al., 2005). Additionally, repeated deviation from homeostasis in the form of repeated allostatic response erode resistance to future stressors (Edes, Wolfe, & Crews, 2016). Since the interpedicular canal may encapsulates a longer period of development, an accumulation of stress events occur that cause an increased mortality for individuals with smaller neural canal. This cumulative stress may not have immediately caused death, but across the lifespan, individuals with a smaller interpedicular diameter still have an increased risk of mortality. The application

of the accumulative stress model does not invalidate the relationship between midsagittal diameter and mortality. These instances still serve as a stark reminder that surviving growth disruptions in the early life environment have a substantial impact on morbidity and mortality across the life course when afflicted by environmental or social stressors.

The credence of the accumulative stress model is increasingly likely when considering the social environment in prehistoric Pueblo communities. The ontology of personhood in Pueblo communities occurs over the first ten years of the life span (Ortiz, 1969). Rites of passage associated with personhood occur on the fourth day of life, during the first year of life, between the ages of 6 to 10 years, and then after 10 years of age (Ortiz, 1969; Parsons 1974). Up until the final rite of passage the individuals are not considered fully human and still exist in a liminal state, between the physical and spiritual worlds (Ortiz, 1969).Skeletal indicators of stress and disease have been explored among individuals prior to and following the ontology of these identities (Schillaci et al., 2011). Porotic lesions in association with cribra orbitalia was found in 84.6% of the individuals below the age of seven years. Additionally, porotic hyperostosis was found in 24.07% of the individuals below the age of seven years. The prevalence varied by age, but the absolute lowest for any presence of stress indicator was 48%. Additionally, femoral growth stunting was identified beginning around two years and continuing until seven years of age (Schillaci et al., 2011). These impoverished conditions in early life are likely reflected in the Pueblo perceptions of childhood. These results may help explain why an accumulative stress model may have such a strong association with mortality in these samples: individuals experienced stress over an extended period of

development in association with the ontological principles associated with personhood. Additionally, this period of stress during Puebloan childhood is represented in the interpedicular canal, which may be used to illustrate the later fusion of the VNC.

One final point to consider is that interpedicular diameter may actually represent stress at an even earlier stage of development than those associated with midsagittal diameters. Recent studies tracked interpedicular diameters in subadults from Bow Baptist and Coronation Street cemeteries in England (Newman and Gowland, 2015). Results suggest that 85% to 91% of adult interpedicular diameter is attained by three years of age. The authors argue that even where increases in interpedicular diameters are found the following points should be considered: 1) these increases may be associated differences in size between survivors and non-survivors and 2) remodeling is substantially reduced after the first three years of life and changes in interpedicular diameter are insubstantial. If true, these findings have important implications for interpreting the greater frequency of elevated mortality risk in association with growth disruptions of interpedicular versus midsagittal diameter. Specifically, the idea that the repeated interaction between interpedicular diameter and mortality may support a model of life history that focuses on windows of developmental sensitivity in the early life environment.

ANOVA tests identified 33 instances where smaller VNC were found in younger age groups compared to four examples at Hawikku. In addition, results from survival analysis identified 11 instances of increased mortality risk in association with smaller VNC at Pueblo Bonito compared to just three at Hawikku. This growth disruption

represents a tradeoff where the individuals allocated resources to survive the nutritional defects. In this situation the application of the accumulative stress model supports the evidence best (Brown, 2016; Guerrant et al., 2002; Guerrant et al., 2008; Painter et al., 2005). Within the first 10 years of life significant growth disruption occurs that increases an individual's mortality throughout the life span. These results are the same as the ones found when analyzing the interpedicular diameter. The significantly smaller VNC that occurred as a response to stress in the first 10 years of life resulted in accumulated growth disruption in the interpedicular canal causing increased mortality throughout the life course. The results of the midsagittal dimeter may actually support the application of the accumulative stress model. The midsagittal diameter achieves fusion around the age of five years, which is sooner than the interpedicular diameter and as a result does not allow as much time for growth disruptions to occur. This results in less signficant accumulated growth disruptions, because skeletal evidence of growth disruption may not have occurred yet.

Pueblo Bonito and Hawikku: A Contextual Portrait of Stress and Mortality

The result of both the ANOVA test and survival analysis have shown evidence of increased mortality risk in association with VNC growth disruptions among the Pueblo Bonito compared to Hawikku sample. ANOVA tests identified 33 instances where smaller VNC was found in younger age groups compared to four examples at Hawikku. In addition, results from survival analysis identified 11 instances of increased mortality risk in association with smaller VNC at Pueblo Bonito compared to just three at Hawikku. These results suggest differences in the relationship between surviving early

life adversity between the Pueblo Bonito and Hawikku samples. Stress physiology and contextual orientations of each cemetery provide important clues explaining these divergent experiences.

First, it is important to point out that these results differ from studies of linear enamel hypoplasia (LEH) and mortality at these sites (Ham, 2018), but are consistent with earlier studies evaluating relationships between LEH, VNC stunting, and survivorship (Watts, 2013b). Individuals with LEH were at a significantly greater mortality risk than individuals without this condition at Hawikku, but not Pueblo Bonito (Ham, 2018). At Hawikku, the average survival time for individuals without LEH was eight years greater than those with LEH. By contrast, a study of vertebral neural canal diameter and LEH in relation to mortality in Late and Post-Medieval London found significant associations between mortality and VNC stunting, but no evidence for increased mortality risk and LEH presence (Watts, 2013b). No relationships were found between VNC stunting and LEH presence (Watts, 2013b). One reason for this may be associated with tissue sensitivity to stress. Teeth develop through pathways associated with growth in body mass: incremental microstructures of enamel form in concert with organismal body mass and isotopes associated with weight loss have been recovered from disrupted segments of enamel (Austin et al., 2015; Bromage et al., 2009). By contrast, Clark et al. (1986) argue that the vertebral neural canal falls into the category of a neurosseuos tissue, and that these tissues may be more sensitive to developmental perturbation. There exist deep relationships between VNC growth with thymus, lymphatic, and neural growth suggests substantial consequence if these injuries are

survived (Clark et al., 1986). Recent research has even suggested that epigenetic factors may have an effect on the growth of vertebrae (Burwell et al., 2011). As such, relationships between LEH, VNC stunting, and mortality may provide different information about the life history of organisms.

In addition to differences in the underlying biology of enamel production and VNC growth, contextual aspects of these sites likely contributed to variation in mortality patterns in relation to VNC growth disruptions at Pueblo Bonito and Hawikku. The population of Pueblo Bonito may have experienced repeated drought during the terminal phase of occupation at the site. Bioarcheologists hypothesize that these droughts resulted in wide-spread starvation, particularly if crop yield was harmed (Palkovich, 1984). An additional systemic issue that could have been the cause of physiological stress or occurred concurrently as the drought was structural violence. Mortuary evidence suggests the existence of an elite ruling class at Pueblo Bonito and that this segment of the population controlled access to resources (Kennett et al., 2017). When an elite class exists, it is possible that the distribution of resources created a hierarchical infrastructure that entraps members of the population. Culture then becomes a catalyst that magnifies biological stress, which could have been introduced by drought, and exaggerated because social stratification that resulted in an unequal allocation of resources and structural inequalities (Klaus, 2012; Palkovich, 1984). Individuals from Pueblo Bonito had set socioeconomic inequality, but this inequality did not push individuals across mortality threshold. These early life events increased risk of mortality through the introduction of additional periods of stress possibly associated with drought. It is also important to note

that the religious and cultural proscriptions of these cultures were associated with ontological experiences that may have accentuated stress during the first 7-10 years of life. Individuals at younger ages were members of alternate identities from adults, and after approximately 10 years of age, became official members of the community (Ortiz, 1969; Parsons 1974). Elevated levels of skeletal growth disruption and disease are found among individuals prior to seven years of age in these communities (Schillaci et al., 2011). This social organization may have played an important role in introducing stressors at early stages of the life cycle that resulted in elevated mortality.

Reduced evidence for mortality risk in association with smaller VNC was found at the Hawikku site. These results were surprising as this site that experiences stressors introduced through colonial practices that may have increased mortality risk following the survival or early life stress events. However, colonial populations frequently encounter epidemic disease as a result of population interaction. There is no evidence for reduced VNC size in younger age groups among individuals recovered from Black Death cemeteries in London, excepting pre-adolescents where the VNC was still growing (Watts, 2013a). In addition, pre-adults with crypt fenestration enamel defects did not experience elevated mortality within sites during the Colonial period in Peru (Thomas et al., 2019). One reason that cemeteries associated with epidemics may be distinct is associated mortality patterns. Older children and younger adults are at a greater risk for mortality during epidemics, and under circumstances where few years are recorded between epidemic disease cycles, individuals of increasingly younger ages become susceptible to mortality (Paine and Boldsen, 2006). As a result, it is possible that

individuals with skeletal indicators of stress and disease do not experience differences in life expectancy compared to those without these lesions in circumstances of epidemic disease.

Evidence for colonial disease epidemics are found in Hawikku, both bioarchaeologically and ethnohistorically. Approximately 36% of all individuals at the site express skeletal evidence of infection, including cases of tuberculosis and treponemal infection, substantially higher frequencies than those observed at prehistoric sites from the same region (Stodder, 1990; Martin & Stodder, 1994). Skeletal evidence for chronic infection was observed on approximately 75% of all infants (Stodder, 1992). Ethnohistoric accounts state that the 15th-16th century witnessed at least 19 instances of epidemic disease (Dobyns & Swagerty, 1983; Reff, 1987). Ethnohistorical data has attributed epidemic illness in the 15th-16th century to cases of smallpox, measles, influenza, scarlet fever, typhus, and malaria (Dobyns & Swagerty, 1983; Reff, 1987). These results indicate epidemic mortality was experienced at the Hawikku site, and these mortality patterns may have produced convergence in frailty between individuals with smaller VNC and those who do not.

One interesting and important point to consider regarding epidemic disease and pathways for physiological constraint following survival of early life adversity may be found in evaluating survivors and non-survivors in cases of the Black Death versus European colonialism in the New World. Individuals with LEH that died were at an increased risk of mortality in examples drawn from Black Death cemeteries and individuals from sites with documented epidemics in the colonial New World (DeWitte

and Wood, 2004; Ham, 2018). In addition, individuals with shorter stature were at an increased mortality risk in Black Death cemeteries, but not attritional cemeteries (DeWitte and Morey-Hughes, 2012; Usher, 2000; Watts, 2013a), while males with smaller body mass were indeed at an increased mortality risk at Hawikku (Ham, 2018). By contrast, individuals from Black Death cemeteries who experienced VNC stunting were not at an increased risk of mortality (Watts, 2013a), similar to results reported for Hawikku where individuals with smaller VNC did not experience exacerbated morality schedules. These results provide an interesting portrait where body size and enamel disruptions appear to place individuals at greater risk of death during disease epidemics, but those associated with disturbances to neuro-osseous tissue are not at an increased mortality risk. Such results speak to differences in developmental pathways governing these hard-tissue structures as a response to multiple etiological factors across comparable contexts. Additional research is needed to explore the relationship between enamel and neuro-osseous tissues disruption.

The results of this thesis support the relationships between trade-offs. In order to survive early life events, the body allocates resources towards short terms survival (Worthman & Kuzara, 2005). The capacity for survival following early life stress does, however, appear to be contextually and systemically specific. This study found that smaller VNC is associated with earlier mortality at Pueblo Bonito, but infrequently at Hawikku. Pueblo Bonito was a prehistoric site, with a rigid hierarchical social organization, while Hawikku was a protohistoric site that experienced disease epidemics associated with European colonialism. Epidemic disease waves may shift mortality

patterns and mask mortality risk following early life stress by imposing mortality on larger segments of the population. In addition, while reduced body size and LEH are associated with mortality during epidemic disease outbreaks in Europe and the colonial New World, VNC growth occurs through different developmental pathways. As such, differences in the expression of physiological constraints following the survival or early life adversity differs between the two sites.

Research Limitations

Once common limitation of this method is subadult growth. The exact mechanisms behind the growth of the VNC are not completely understood. However, there is evidence to suggest that VNC growth ceases during childhood and growth disruptions found in this measurement may be associated with stress events. For example, Newman and Gowland (2015) suggest that after the age of three years changes in the VNC do not exists because of increased growth. Instead the authors state that differences after the age of three years are associated with survivors and non-survivors. The authors go on to state that remodeling is substantially reduced after the first three years of life and changes in interpedicular diameter are insubstantial post this age.

If subadults had smaller VNC because of lack of growth it would be illustrated in the cohort analysis. The result of the cohort analysis showed signficant difference among subadult compared to the results of the rest of the ages cohorts 19/96 times. If subadult VNC were smaller because of lack of growth this significance would be more widespread. Additionally, there were several cases were VNC was significantly smaller outside of the subadult period. This information suggests that the significance among the

subadults occurred as a result of a non-uniform process like growth disruptions opposed to standard growth. Finally, VNC diameters were significantly smaller in the Pueblo Bonito versus Hawikku in more than 2x the total number of instances where significant differences were observed. This result portends that selective mortality rather than continued growth is responsible for variation in survival and VNC growth disruption.

Nonspecific Limitations

It is necessary to note that all the results of these studies are limited by the nonspecific nature of the VNC. The results cannot be used as an explanatory factor for a single constraint that resulted in a growth disruption. The cause of specific instances of nutritional or dietary stress cannot be identified through the use of this method. Instead the VNC can only be used as an evidence of general stress from a variety of nonspecific factors. While the non-specificity limits the application of this method there is alternate methods to elucidate this information, especially through the inclusion of other contextual information or additional research on other stress indicators.

Statistical Limitations

Additional limitations include the nature of the ANOVA test, and statistical analysis in general. Results can only be interpreted in the scope of the analysis that can be performed on them. Statistically signficant results in cohort analysis only show that there is a difference in the mean of the measurements that incapsulate the unit of analysis. The results do not indicate which cohort had a larger vertebral canal, nor do they indicate information about the specific ages within the cohort. That information can only be elucidated through the use of boxplots that show the difference in the means. The use of

the ANOVA test was only done in order to be comparable to previous studies on the VNC that used cohort analysis (Clark et al., 1986; Newman & Gowland, 2015; Watts, 2011, 2013). These studies employed repetitive T-test, which is known to compound type 2 error in each analysis and be problematic. Additionally, when divided into age cohorts the sample size decreases and comparisons may begin to become compromised. In contrast to cohort analysis, survival analysis tests the probability of survival at a certain time (Singh & Mukhopadhyay, 2011). Sample size is especially problematic in older section of the population because of decreased representation of individuals. The application of survival analysis allows for an improved method to evaluate the application of hidden heterogeneity and selective mortality on a skeletal sample by more directly modeling the effects of VNC size on age at death be separating the sample based on small and large VNC (DeWitte & Stojanowski, 2015). Survival analysis is less inhibited by sample size because the analysis focuses on the longitudinal aspect of samples age range. Additionally, survivor analysis lines are not based on early life trends but capture the entire lifespan, which allows for a more holistic understanding of the growth disruption across the life span.

CHAPTER SEVEN: CONCLUSION

The results from this thesis demonstrate that VNC growth disruptions may be correlated with mortality risk. The samples analyzed in this study are the Pueblo Bonito and Hawikku from the American Southwest. In the current study, the vast majority of a smaller VNC diameter was found between subadults or 17-25-year age cohorts. Results also indicated that individuals with a smaller VNC diameter were at a higher risk of mortality throughout the life course. The results of this study align with previous research that has also explored mortality risk and VNC growth disruption.

In this study a greater number of interpedicular diameters were observed in association with mortality risk when compared to midsagittal diameters. Significance was found in twice as many age cohorts' analyses for the interpedicular diameter in comparisons to the midsagittal diameter. These results are attributed to the accumulative stress model. The interpedicular VNC fuses at a later age then the midsagittal diameters. The longer period of vertebral growth allows for a greater window to capture stressful events through growth disruptions. Individuals that have experience repeated stress events, especially earlier in the lifecycle, have an increased risk of mortality. Thus, the credence of the accumulative stress mode in this context it is increasingly likely,

especially when considering the social environment in prehistoric Pueblo communities and their complicated ontogeny associated with personhood in early life. That said, greater understanding of interpedicular diameter is needed as recent studies suggest that this measurement is set early in development. If true, results from this thesis would support a developmental sensitivity versus accumulative stress model of mortality.

The result of both the ANOVA test and survival analysis have shown evidence of additional increased mortality risk in association with smaller VNC among the Pueblo Bonito compared to Hawikku sample. The results of this study differ from analysis of linear enamel hypoplasia (LEH) and mortality at these sites. However, relationships between LEH and VNC may provide different information about the life history of organisms as result of different developmental pathways contributing to mortality. The population of Pueblo Bonito may have experienced repeated drought during the terminal phase of occupation at the site. Bioarcheologists hypothesize that these droughts resulted in wide-spread starvation, that was magnified by stressors from the delineation of socials prestige. The Hawikku were afflicted by epidemic diseases from colonial forces. Previous studies have shown that epidemics disproportionally effect the younger and older portions of a population. As a result, it is possible that individuals with smaller VNC did not experience differences in life expectancy compared to those without these lesions.

Future Research

The next step in research on the Pueblo Bonito and Hawikku would be a more holistic study of stress and lifeways. This thesis focused on VNC, other projects have looked at paleoepidemiology, while some have studied cross sectional geometry.

Combining all this research would yield a more complete view of these societies. Additionally, more research about the developmental pathways associated with the VNC would allow for a better understanding of the processes that causes growth disruptions. Further research on the VNC would be especially fruitful when combined with information elucidated through other skeletal indicators of stress. Additionally, analyzing individuals who had small versus large VNC may provide more concrete evidence of the effect of growth disruptions. The results of that analysis would provide more clear proof of whether growth disruptions that occurred systemically across the spinal cord consistently increased an individuals mortality.

Conclusion

The results of this study elucidate the stress that can occur as a result of hierarchical societies and colonialism. The stress placed on the Pueblo Bonito occurred as a result of draught that was magnified by the delineation of social prestige. The stress placed on the Hawikku occurred as a result of pathogenic conditions that were introduced by the non-native colonialists that contributed to early morbidity. The analysis of the VNC elucidated information about growth disruptions in association with mortality. The VNC is a sparsely published upon method that with the application of survival analysis will increase its validity as a nonspecific stress indicator for describing skeletal samples.

APPENDIX

Table 10: All ANOVA Comparison of Age Cohort

Sample	Measurement	Vertebra	Age at Death	Sample Size	Significance
Hawikku	interpedicular	C1	Sub-Adult	12	0.858
	interpedicular		17-25	11	0.405
	interpedicular		26-35	23	0.988
	interpedicular		36-45	16	0.641
	interpedicular		46-55	**5**	**0.044**
	interpedicular		56+	**8**	
Hawikku	interpedicular	C2	Sub-Adult	**13**	**0.013**
	interpedicular		17-25	10	0.497
	interpedicular		26-35	25	0.721
	interpedicular		36-45	16	0.891
	interpedicular		46-55+	**5**	**0.042**
	interpedicular		56+	**6**	
Hawikku	interpedicular	C3	Sub-Adult	**13**	**0.005**
	interpedicular		17-25	12	0.240
	interpedicular		26-35	22	0.919
	interpedicular		36-45	15	0.539
	interpedicular		46-55+	6	0.193
	interpedicular		56+	7	
Hawikku	interpedicular	C4	Sub-Adult	12	0.494
	interpedicular		17-25	12	0.782
	interpedicular		26-35	24	0.971
	interpedicular		36-45	15	0.801
	interpedicular		46-55+	5	0.476
	interpedicular		56+	7	
Hawikku	interpedicular	C5	Sub-Adult	14	0.142
	interpedicular		17-25	11	0.955

	interpedicular		26-35	22	0.810
	interpedicular		36-45	15	0.952
	interpedicular		46-55+	5	0.274
	interpedicular		56+	7	
Hawikku	interpedicular	C6	Sub-Adult	14	0.121
	interpedicular		17-25	11	0.851
	interpedicular		26-35	27	0.389
	interpedicular		36-45	16	0.794
	interpedicular		46-55+	5	0.208
	interpedicular		56+	8	
Hawikku	interpedicular	C7	Sub-Adult	11	0.587
	interpedicular		17-25	11	0.975
	interpedicular		26-35	22	0.746
	interpedicular		36-45	16	0.729
	interpedicular		46-55+	5	0.437
	interpedicular		56+	6	
Hawikku	interpedicular	T1	Sub-Adult	12	0.888
	interpedicular		17-25	11	0.810
	interpedicular		26-35	23	0.690
	interpedicular		36-45	16	0.286
	interpedicular		46-55+	6	0.572
	interpedicular		56+	7	
Hawikku	interpedicular	T2	Sub-Adult	12	0.666
	interpedicular		17-25	10	0.607
	interpedicular		26-35	26	0.809
	interpedicular		36-45	15	0.505
	interpedicular		46-55+	6	0.928
	interpedicular		56+	7	
Hawikku	interpedicular	T3	Sub-Adult	9	0.959
	interpedicular		17-25	10	0.110
	interpedicular		26-35	35	0.739
	interpedicular		36-45	16	0.471
	interpedicular		46-55+	5	0.736
	interpedicular		56+	6	
Hawikku	interpedicular	T4	Sub-Adult	11	0.456
	interpedicular		17-25	9	0.129
	interpedicular		26-35	22	0.492
	interpedicular		36-45	12	0.904
	interpedicular		46-55+	4	0.347

	interpedicular		56+	6	
Hawikku	interpedicular	T5	Sub-Adult	11	0.274
	interpedicular		17-25	11	0.222
	interpedicular		26-35	20	0.852
	interpedicular		36-45	10	0.428
	interpedicular		46-55+	4	0.356
	interpedicular		56+	5	
Hawikku	interpedicular	T6	Sub-Adult	9	0.215
	interpedicular		17-25	9	0.571
	interpedicular		26-35	21	0.266
	interpedicular		36-45	10	0.204
	interpedicular		46-55+	3	0.170
	interpedicular		56+	6	
Hawikku	interpedicular	T7	Sub-Adult	8	0.184
	interpedicular		17-25	8	0.085
	interpedicular		26-35	21	0.536
	interpedicular		36-45	13	0.095
	interpedicular		46-55+	5	0.561
	interpedicular		56+	6	
Hawikku	interpedicular	T8	Sub-Adult	8	0.193
	interpedicular		17-25	10	0.526
	interpedicular		26-35	24	0.354
	interpedicular		36-45	15	0.120
	interpedicular		46-55+	5	0.937
	interpedicular		56+	6	
Hawikku	interpedicular	T9	Sub-Adult	8	0.473
	interpedicular		17-25	12	0.193
	interpedicular		26-35	25	0.456
	interpedicular		36-45	17	0.650
	interpedicular		46-55+	5	0.582
	interpedicular		56+	6	
Hawikku	interpedicular	T10	Sub-Adult	9	0.893
	interpedicular		17-25	14	0.572
	interpedicular		26-35	25	0.965
	interpedicular		36-45	19	0.780
	interpedicular		46-55+	6	0.581
	interpedicular		56+	5	
Hawikku	interpedicular	T11	Sub-Adult	8	0.848
	interpedicular		17-25	15	0.628

	interpedicular		26-35	26	0.880
	interpedicular		36-45	16	0.292
	interpedicular		46-55+	6	0.341
	interpedicular		56+	6	
Hawikku	interpedicular	T12	Sub-Adult	9	0.699
	interpedicular		17-25	16	0.361
	interpedicular		26-35	35	0.718
	interpedicular		36-45	18	0.450
	interpedicular		46-55+	6	0.150
	interpedicular		56+	8	
Hawikku	interpedicular	L1	Sub-Adult	11	0.389
	interpedicular		17-25	16	0.287
	interpedicular		26-35	26	0.914
	interpedicular		36-45	18	0.449
	interpedicular		46-55+	6	0.151
	interpedicular		56+	7	
Hawikku	interpedicular	L2	Sub-Adult	12	0.342
	interpedicular		17-25	15	0.115
	interpedicular		26-35	28	0.913
	interpedicular		36-45	18	0.236
	interpedicular		46-55+	7	0.670
	interpedicular		56+	7	
Hawikku	interpedicular	L3	Sub-Adult	13	0.365
	interpedicular		17-25	14	0.172
	interpedicular		26-35	39	0.778
	interpedicular		36-45	17	0.387
	interpedicular		46-55+	7	0.128
	interpedicular		56+	7	
Hawikku	interpedicular	L4	Sub-Adult	12	0.696
	interpedicular		17-25	15	0.499
	interpedicular		26-35	31	0.708
	interpedicular		36-45	18	0.228
	interpedicular		46-55+	7	0.973
	interpedicular		56+	6	
Hawikku	interpedicular	L5	Sub-Adult	13	0.479
	interpedicular		17-25	15	0.733
	interpedicular		26-35	28	0.445
	interpedicular		36-45	19	0.457
	interpedicular		46-55+	6	0.777

	interpedicular		56+	7	
Hawikku	Midsagittal	C1	Sub-Adult	11	0.551
	Midsagittal		17-25	10	0.807
	Midsagittal		26-35	24	0.955
	Midsagittal		36-45	15	0.332
	Midsagittal		46-55+	5	0.630
	Midsagittal		56+	6	
Hawikku	Midsagittal	C2	Sub-Adult	12	0.774
	Midsagittal		17-25	8	0.455
	Midsagittal		26-35	25	0.815
	Midsagittal		36-45	15	0.492
	Midsagittal		46-55+	5	0.190
	Midsagittal		56+	7	
Hawikku	Midsagittal	C3	Sub-Adult	12	0.318
	Midsagittal		17-25	12	0.809
	Midsagittal		26-35	22	0.516
	Midsagittal		36-45	14	0.570
	Midsagittal		46-55+	5	0.415
	Midsagittal		56+	7	
Hawikku	Midsagittal	C4	Sub-Adult	12	0.362
	Midsagittal		17-25	12	0.375
	Midsagittal		26-35	23	0.662
	Midsagittal		36-45	15	0.205
	Midsagittal		46-55+	5	0.622
	Midsagittal		56+	6	
Hawikku	Midsagittal	C5	Sub-Adult	14	0.115
	Midsagittal		17-25	11	0.585
	Midsagittal		26-35	24	0.583
	Midsagittal		36-45	15	0.118
	Midsagittal		46-55+	5	0.523
	Midsagittal		56+	7	
Hawikku	Midsagittal	C6	Sub-Adult	13	0.407
	Midsagittal		17-25	11	0.407
	Midsagittal		26-35	23	0.800
	Midsagittal		36-45	15	0.176
	Midsagittal		46-55+	5	0.793
	Midsagittal		56+	7	
Hawikku	Midsagittal	C7	Sub-Adult	11	0.207
	Midsagittal		17-25	11	0.677

	Midsagittal		26-35	23	0.484
	Midsagittal		36-45	16	0.084
	Midsagittal		46-55+	6	0.411
	Midsagittal		56+	7	
Hawikku	Midsagittal	T1	Sub-Adult	12	0.404
	Midsagittal		17-25	11	0.698
	Midsagittal		26-35	24	0.710
	Midsagittal		36-45	16	0.274
	Midsagittal		46-55+	6	0.228
	Midsagittal		56+	7	
Hawikku	Midsagittal	T2	Sub-Adult	12	0.450
	Midsagittal		17-25	10	0.345
	Midsagittal		26-35	26	0.421
	Midsagittal		36-45	15	0.133
	Midsagittal		46-55+	6	0.419
	Midsagittal		56+	7	
Hawikku	Midsagittal	T3	Sub-Adult	9	0.783
	Midsagittal		17-25	10	0.226
	Midsagittal		26-35	24	0.545
	Midsagittal		36-45	15	0.161
	Midsagittal		46-55+	3	0.757
	Midsagittal		56+	6	
Hawikku	Midsagittal	T4	Sub-Adult	10	0.964
	Midsagittal		17-25	9	0.813
	Midsagittal		26-35	19	0.959
	Midsagittal		36-45	12	0.580
	Midsagittal		46-55+	4	0.440
	Midsagittal		56+	5	
Hawikku	Midsagittal	T5	Sub-Adult	11	0.703
	Midsagittal		17-25	11	0.798
	Midsagittal		26-35	19	0.339
	Midsagittal		36-45	11	0.102
	Midsagittal		46-55+	4	0.255
	Midsagittal		56+	5	
Hawikku	Midsagittal	T6	Sub-Adult	9	0.560
	Midsagittal		17-25	9	0.296
	Midsagittal		26-35	20	0.540
	Midsagittal		36-45	10	0.114
	Midsagittal		46-55+	5	0.105

	Midsagittal		56+	6	
Hawikku	Midsagittal	T7	Sub-Adult	8	0.862
	Midsagittal		17-25	8	0.847
	Midsagittal		26-35	21	0.318
	Midsagittal		36-45	12	0.149
	Midsagittal		46-55+	5	0.790
	Midsagittal		56+	6	
Hawikku	Midsagittal	T8	Sub-Adult	8	0.320
	Midsagittal		17-25	10	0.691
	Midsagittal		26-35	23	0.820
	Midsagittal		36-45	14	0.077
	Midsagittal		46-55+	5	0.090
	Midsagittal		56+	6	
Hawikku	Midsagittal	T9	Sub-Adult	8	0.670
	Midsagittal		17-25	11	0.219
	Midsagittal		26-35	23	0.776
	Midsagittal		36-45	16	0.231
	Midsagittal		46-55+	5	0.660
	Midsagittal		56+	6	
Hawikku	Midsagittal	T10	Sub-Adult	8	0.343
	Midsagittal		17-25	14	0.387
	Midsagittal		26-35	24	1.000
	Midsagittal		36-45	19	0.335
	Midsagittal		46-55+	6	0.161
	Midsagittal		56+	5	
Hawikku	Midsagittal	T11	Sub-Adult	8	0.936
	Midsagittal		17-25	11	0.156
	Midsagittal		26-35	24	0.836
	Midsagittal		36-45	16	0.408
	Midsagittal		46-55+	5	0.739
	Midsagittal		56+	6	
Hawikku	Midsagittal	T12	Sub-Adult	7	0.800
	Midsagittal		17-25	16	0.756
	Midsagittal		26-35	21	0.640
	Midsagittal		36-45	18	0.855
	Midsagittal		46-55+	5	0.440
	Midsagittal		56+	6	
Hawikku	Midsagittal	L1	Sub-Adult	11	0.330
	Midsagittal		17-25	16	0.809

	Midsagittal		26-35	24	0.771
	Midsagittal		36-45	18	0.410
	Midsagittal		46-55+	5	0.281
	Midsagittal		56+	7	
Hawikku	Midsagittal	L2	Sub-Adult	12	0.703
	Midsagittal		17-25	15	0.959
	Midsagittal		26-35	24	0.725
	Midsagittal		36-45	18	0.261
	Midsagittal		46-55+	6	0.678
	Midsagittal		56+	6	
Hawikku	Midsagittal	L3	Sub-Adult	12	0.788
	Midsagittal		17-25	14	0.530
	Midsagittal		26-35	25	0.646
	Midsagittal		36-45	14	0.310
	Midsagittal		46-55+	7	0.490
	Midsagittal		56+	6	
Hawikku	Midsagittal	L4	Sub-Adult	12	0.598
	Midsagittal		17-25	15	0.765
	Midsagittal		26-35	29	0.477
	Midsagittal		36-45	16	0.670
	Midsagittal		46-55+	7	0.985
	Midsagittal		56+	6	
Hawikku	Midsagittal	L5	Sub-Adult	12	0.703
	Midsagittal		17-25	14	0.797
	Midsagittal		26-35	24	0.883
	Midsagittal		36-45	16	0.232
	Midsagittal		46-55+	6	0.611
	Midsagittal		56+	5	
Pueblo Bonito	interpedicular	C1	Sub-Adult	11	0.309
	interpedicular		17-25	2	0.285
	interpedicular		26-35	4	0.878
	interpedicular		36-45	4	0.903
	interpedicular		46-55+	3	0.090
	interpedicular		56+	4	
Pueblo Bonito	interpedicular	C2	Sub-Adult	11	0.622
	interpedicular		17-25	**5**	**0.014**
	interpedicular		26-35	5	0.719
	interpedicular		36-45	4	0.132
	interpedicular		46-55+	3	0.773

	interpedicular		56+	6	
Pueblo Bonito	interpedicular	C3	Sub-Adult	10	0.166
	interpedicular		17-25	3	0.270
	interpedicular		26-35	6	0.225
	interpedicular		36-45	4	0.643
	interpedicular		46-55+	33	0.553
	interpedicular		56+	6	
Pueblo Bonito	interpedicular	C4	Sub-Adult	11	0.132
	interpedicular		17-25	5	0.258
	interpedicular		26-35	6	0.060
	interpedicular		36-45	**4**	**0.027**
	interpedicular		46-55+	3	0.728
	interpedicular		56+	6	
Pueblo Bonito	interpedicular	C5	Sub-Adult	9	0.112
	interpedicular		17-25	4	0.739
	interpedicular		26-35	5	0.146
	interpedicular		36-45	3	0.130
	interpedicular		46-55+	3	0.437
	interpedicular		56+	7	
Pueblo Bonito	interpedicular	C6	Sub-Adult	8	0.005
	interpedicular		17-25	4	0.840
	interpedicular		26-35	5	0.532
	interpedicular		36-45	3	0.522
	interpedicular		46-55+	3	0.127
	interpedicular		56+	7	
Pueblo Bonito	interpedicular	C7	Sub-Adult	9	0.440
	interpedicular		17-25	6	0.737
	interpedicular		26-35	5	0.895
	interpedicular		36-45	2	0.822
	interpedicular		46-55+	3	0.078
	interpedicular			6	
Pueblo Bonito	interpedicular	T1	Sub-Adult	12	0.222
	interpedicular		17-25	3	0.142
	interpedicular		26-35	5	0.779
	interpedicular		36-45	**2**	**0.027**
	interpedicular		46-55+	3	0.105
	interpedicular		56+	6	
Pueblo Bonito	interpedicular	T2	Sub-Adult	10	0.054
	interpedicular		17-25	4	0.385

	interpedicular		26-35	5	0.679
	interpedicular		36-45	1	0.769
	interpedicular		46-55+	3	0.088
	interpedicular		56+	**6**	
Pueblo Bonito	interpedicular	T3	Sub-Adult	**8**	**0.024**
	interpedicular		17-25	4	0.247
	interpedicular		26-35	5	0.666
	interpedicular		36-45	1	0.503
	interpedicular		46-55+	3	0.250
	interpedicular		56+	**7**	
Pueblo Bonito	interpedicular	T4	Sub-Adult	**7**	**0.009**
	interpedicular		17-25	**5**	**0.043**
	interpedicular		26-35	5	0.722
	interpedicular		36-45	0	
	interpedicular		46-55+	3	0.169
	interpedicular		56+	**6**	
Pueblo Bonito	interpedicular	T5	Sub-Adult	**7**	**0.007**
	interpedicular		17-25	5	0.143
	interpedicular		26-35	5	0.159
	interpedicular		36-45	2	0.940
	interpedicular		46-55+	3	0.142
	interpedicular		56+	**6**	
Pueblo Bonito	interpedicular	T6	Sub-Adult	**7**	**0.003**
	interpedicular		17-25	4	0.655
	interpedicular		26-35	5	0.472
	interpedicular		36-45	2	0.821
	interpedicular		46-55+	3	0.376
	interpedicular		56+	**6**	
Pueblo Bonito	interpedicular	T7	Sub-Adult	**8**	**0.005**
	interpedicular		17-25	5	0.316
	interpedicular		26-35	4	0.549
	interpedicular		36-45	3	0.981
	interpedicular		46-55+	3	0.531
	interpedicular		56+	**6**	
Pueblo Bonito	interpedicular	T8	Sub-Adult	**8**	**0.030**
	interpedicular		17-25	5	0.220
	interpedicular		26-35	5	0.845
	interpedicular		36-45	4	0.173
	interpedicular		46-55+	3	0.563

	interpedicular		56+	**6**	
Pueblo Bonito	interpedicular	T9	Sub-Adult	**8**	**0.044**
	interpedicular		17-25	6	0.156
	interpedicular		26-35	5	0.533
	interpedicular		36-45	4	0.261
	interpedicular		46-55+	3	0.435
	interpedicular		56+	**6**	
Pueblo Bonito	interpedicular	T10	Sub-Adult	**11**	**0.039**
	interpedicular		17-25	6	0.290
	interpedicular		26-35	6	0.786
	interpedicular		36-45	4	0.323
	interpedicular		46-55+	3	0.292
	interpedicular		56+	6	
Pueblo Bonito	interpedicular	T11	Sub-Adult	8	0.132
	interpedicular		17-25	6	0.064
	interpedicular		26-35	7	0.859
	interpedicular		36-45	4	0.867
	interpedicular		46-55+	3	0.195
	interpedicular		56+	6	
Pueblo Bonito	interpedicular	T12	Sub-Adult	8	0.133
	interpedicular		17-25	**7**	**0.047**
	interpedicular		26-35	7	0.651
	interpedicular		36-45	4	0.938
	interpedicular		46-55+	3	0.230
	interpedicular		56+	**6**	
Pueblo Bonito	interpedicular	L1	Sub-Adult	**12**	**0.013**
	interpedicular		17-25	4	0.369
	interpedicular		26-35	5	0.024
	interpedicular		36-45	4	0.953
	interpedicular		46-55+	3	0.267
	interpedicular		56+	**6**	
Pueblo Bonito	interpedicular	L2	Sub-Adult	**13**	**0.002**
	interpedicular		17-25	4	0.545
	interpedicular		26-35	**4**	**0.027**
	interpedicular		36-45	4	0.971
	interpedicular		46-55+	3	0.106
	interpedicular		56+	**6**	
Pueblo Bonito	interpedicular	L3	Sub-Adult	**11**	**0.001**
	interpedicular		17-25	**4**	**0.046**

	interpedicular		26-35	8	0.382
	interpedicular		36-45	3	0.991
	interpedicular		46-55+	3	0.093
	interpedicular		56+	**6**	
Pueblo Bonito	interpedicular	L4	Sub-Adult	**13**	**0.010**
	interpedicular		17-25	1	0.219
	interpedicular		26-35	8	0.722
	interpedicular		36-45	3	0.847
	interpedicular		46-55+	3	0.061
	interpedicular		56+	**6**	
Pueblo Bonito	interpedicular	L5	Sub-Adult	**14**	**0.017**
	interpedicular		17-25	1	0.125
	interpedicular		26-35	8	0.639
	interpedicular		36-45	2	0.846
	interpedicular		46-55+	**3**	**0.024**
	interpedicular		56+	6	
Pueblo Bonito	Midsagittal	C1	Sub-Adult	2	0.712
	Midsagittal		17-25	2	0.466
	Midsagittal		26-35	4	0.664
	Midsagittal		36-45	4	0.095
	Midsagittal		46-55+	3	0.124
	Midsagittal		56+	4	
Pueblo Bonito	Midsagittal	C2	Sub-Adult	11	0.814
	Midsagittal		17-25	4	0.610
	Midsagittal		26-35	5	0.708
	Midsagittal		36-45	4	0.185
	Midsagittal		46-55+	3	0.269
	Midsagittal		56+	6	
Pueblo Bonito	Midsagittal	C3	Sub-Adult	10	0.056
	Midsagittal		17-25	**4**	**0.002**
	Midsagittal		26-35	6	0.211
	Midsagittal		36-45	4	0.263
	Midsagittal		46-55+	3	0.651
	Midsagittal		56+	6	
Pueblo Bonito	Midsagittal	C4	Sub-Adult	13	0.077
	Midsagittal		17-25	**5**	**0.049**
	Midsagittal		26-35	**6**	**0.028**
	Midsagittal		36-45	4	0.246
	Midsagittal		46-55+	3	0.382

	Midsagittal		56+	6	
Pueblo Bonito	Midsagittal	C5	Sub-Adult	9	0.269
	Midsagittal		17-25	5	0.072
	Midsagittal		26-35	6	0.071
	Midsagittal		36-45	3	0.790
	Midsagittal		46-55+	3	0.662
	Midsagittal		56+	7	
Pueblo Bonito	Midsagittal	C6	Sub-Adult	8	0.890
	Midsagittal		17-25	4	0.104
	Midsagittal		26-35	5	0.533
	Midsagittal		36-45	3	0.991
	Midsagittal		46-55+	3	0.860
	Midsagittal		56+	7	
Pueblo Bonito	Midsagittal	C7	Sub-Adult	9	0.561
	Midsagittal		17-25	5	0.338
	Midsagittal		26-35	5	0.465
	Midsagittal		36-45	2	0.207
	Midsagittal		46-55+	3	0.594
	Midsagittal		56+	6	
Pueblo Bonito	Midsagittal	T1	Sub-Adult	11	0.236
	Midsagittal		17-25	**4**	**0.044**
	Midsagittal		26-35	5	0.393
	Midsagittal		36-45	2	0.308
	Midsagittal		46-55+	3	0.555
	Midsagittal		56+	6	
Pueblo Bonito	Midsagittal	T2	Sub-Adult	9	0.067
	Midsagittal		17-25	4	0.371
	Midsagittal		26-35	5	0.970
	Midsagittal		36-45	1	0.880
	Midsagittal		46-55+	3	0.457
	Midsagittal		56+	6	
Pueblo Bonito	Midsagittal	T3	Sub-Adult	8	0.327
	Midsagittal		17-25	4	0.270
	Midsagittal		26-35	5	0.891
	Midsagittal		36-45	1	0.925
	Midsagittal		46-55+	3	0.873
	Midsagittal		56+	7	
Pueblo Bonito	Midsagittal	T4	Sub-Adult	7	0.322
	Midsagittal		17-25	5	0.198

	Midsagittal		26-35	5	0.550
	Midsagittal		36-45	0	
	Midsagittal		46-55+	3	0.281
	Midsagittal		56+	6	
Pueblo Bonito	Midsagittal	T5	Sub-Adult	7	0.068
	Midsagittal		17-25	5	0.226
	Midsagittal		26-35	5	0.131
	Midsagittal		36-45	2	0.490
	Midsagittal		46-55+	3	0.603
	Midsagittal		56+	6	
Pueblo Bonito	Midsagittal	T6	Sub-Adult	7	0.298
	Midsagittal		17-25	**4**	**0.012**
	Midsagittal		26-35	5	0.991
	Midsagittal		36-45	2	0.237
	Midsagittal		46-55+	3	0.749
	Midsagittal		56+	**6**	
Pueblo Bonito	Midsagittal	T7	Sub-Adult	**8**	**0.031**
	Midsagittal		17-25	5	0.439
	Midsagittal		26-35	4	0.528
	Midsagittal		36-45	3	0.542
	Midsagittal		46-55+	3	0.963
	Midsagittal		56+	**6**	
Pueblo Bonito	Midsagittal	T8	Sub-Adult	**7**	**0.002**
	Midsagittal		17-25	5	0.228
	Midsagittal		26-35	5	0.834
	Midsagittal		36-45	4	0.801
	Midsagittal		46-55+	3	0.928
	Midsagittal		56+	**6**	
Pueblo Bonito	Midsagittal	T9	Sub-Adult	**9**	**0.040**
	Midsagittal		17-25	6	0.099
	Midsagittal		26-35	5	0.780
	Midsagittal		36-45	4	0.609
	Midsagittal		46-55+	3	0.479
	Midsagittal		56+	6	
Pueblo Bonito	Midsagittal	T10	Sub-Adult	11	0.080
	Midsagittal		17-25	6	0.625
	Midsagittal		26-35	6	0.560
	Midsagittal		36-45	4	0.521
	Midsagittal		46-55+	3	0.719

	Midsagittal		56+	6	
Pueblo Bonito	Midsagittal	T11	Sub-Adult	7	0.239
	Midsagittal		17-25	7	0.161
	Midsagittal		26-35	7	0.936
	Midsagittal		36-45	4	0.218
	Midsagittal		46-55+	3	0.765
	Midsagittal		56+	6	
Pueblo Bonito	Midsagittal	T12	Sub-Adult	8	0.727
	Midsagittal		17-25	7	0.100
	Midsagittal		26-35	7	0.552
	Midsagittal		36-45	4	0.789
	Midsagittal		46-55+	3	0.783
	Midsagittal		56+	6	
Pueblo Bonito	Midsagittal	L1	Sub-Adult	12	0.288
	Midsagittal		17-25	**2**	**0.004**
	Midsagittal		26-35	5	0.192
	Midsagittal		36-45	4	0.189
	Midsagittal		46-55+	3	0.466
	Midsagittal		56+	6	
Pueblo Bonito	Midsagittal	L2	Sub-Adult	12	0.056
	Midsagittal		17-25	**4**	**0.021**
	Midsagittal		26-35	4	0.076
	Midsagittal		36-45	4	0.522
	Midsagittal		46-55+	3	0.483
	Midsagittal		56+	**6**	
Pueblo Bonito	Midsagittal	L3	Sub-Adult	**11**	**0.025**
	Midsagittal		17-25	**5**	**0.039**
	Midsagittal		26-35	7	0.067
	Midsagittal		36-45	3	0.462
	Midsagittal		46-55+	3	0.278
	Midsagittal		56+	6	
Pueblo Bonito	Midsagittal	L4	Sub-Adult	12	0.151
	Midsagittal		17-25	0	
	Midsagittal		26-35	8	0.199
	Midsagittal		36-45	3	0.881
	Midsagittal		46-55+	3	0.130
	Midsagittal		56+	6	
Pueblo Bonito	Midsagittal	L5	Sub-Adult	13	0.051
	Midsagittal		17-25	1	0.814

	Midsagittal		26-35	**8**	**0.012**
	Midsagittal		36-45	2	0.666
	Midsagittal		46-55+	3	0.386

Table 11: Pueblo Bonito Interpedicular VNC Measurements for Significant Results

	Sub-adult	17-25	26-35	36-45	46-55	56+
C2	20.19	**18.89**	20.80	21.98	20.55	22.14
C4	21.0009	21.5100	22.8983	**22.4650**	21.4533	21.4600
T1	18.2150	17.6533	18.9640	**21.7250**	17.6500	18.3029
T3	**14.7925**	15.2600	15.7640	16.6900	15.3733	14.7733
T4	**14,0829**	**14.6880**	15.5720	N/A	15.1233	14.8317
T5	**14.2971**	14.9860	15.2420	16.1750	15.2300	14.3640
T6	**14.2957**	15.4375	16.0160	15.400	15.0833	14.4583
T7	**14.6188**	15..4560	16.2850	15.9167	15.5000	14.2917
T8	**15.1450**	15.5900	16.2960	16.7875	15.6400	14.3767
T9	**15.2325**	15.5733	16.6020	16.7425	15.6733	14.6233
T10	**15.2745**	15.7083	16.3283	16.6000	15.5200	14.6480
L1	**20.4975**	20.3575	19.7280	21.8000	20.7467	20.6143
L2	**19.9631**	21.4150	20.3975	22.4250	21.1167	20.9800
L3	**21.2627**	20.9850	22.5238	23.2467	21.6733	21.3214
L4	**21.5169**	20.7900	23.6438	23.8000	21.7433	21.3183
L5	**24.0107**	21.9100	26.2800	26.5400	23.8900	23.5871

Table 12: Hawikku Interpedicular VNC Measurements for Significant Results

	Sub-adult	17-25	26-35	36-45	46-55	56+
C1	26.6533	27.5070	27.3058	27.5693	**25.5540**	28.2500
C2	**19.98**	14.4438	15.1016	14.9487	**13.9080**	20.75
C3	**19..4554**	13.0092	13.0441	13.2829	12.0600	20.2883

Table 13: Pueblo Bonito Midsagittal VNC Measurements for Significant Results

	Sub-adult	17-25	26-35	36-45	46-55	56+
C3	13.7780	**14.3600**	12.8383	12.6900	11.8000	12.6086
C4	13.5646	**13.4600**	13.0300	12.2175	11.3067	12.4050
T1	13.0700	**12.5675**	14.1180	12.9100	13.4633	13.7271

T6	13.8700	**13.4175**	14.6040	14.1567	14.6267	14.4033
T7	**13.5825**	14.2680	14.4100	14.5233	14.8300	14.8283
T8	**13.4700**	14.2760	14.8360	14.6425	14.7367	14.2267
T9	**13.5356**	**13.8667**	14.6740	14.7500	14.0967	14.1233
T10	13.6591	**14.1283**	14.5350	14.5133	14.2933	14.3340
L3	13.4155	**13.1667**	14.2071	14.8200	14.7900	13.3250
L5	14.2685	15.3600	**14.4163**	17.7850	15.7933	142600

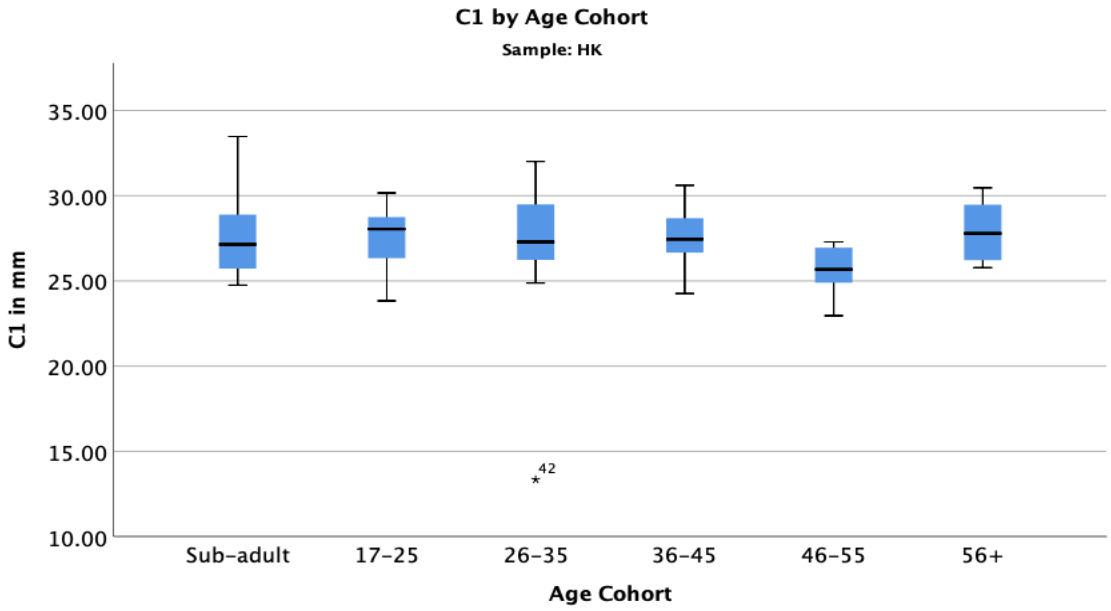

Figure 45: Boxplots of Hawikku C1 Midsagittal Diameter

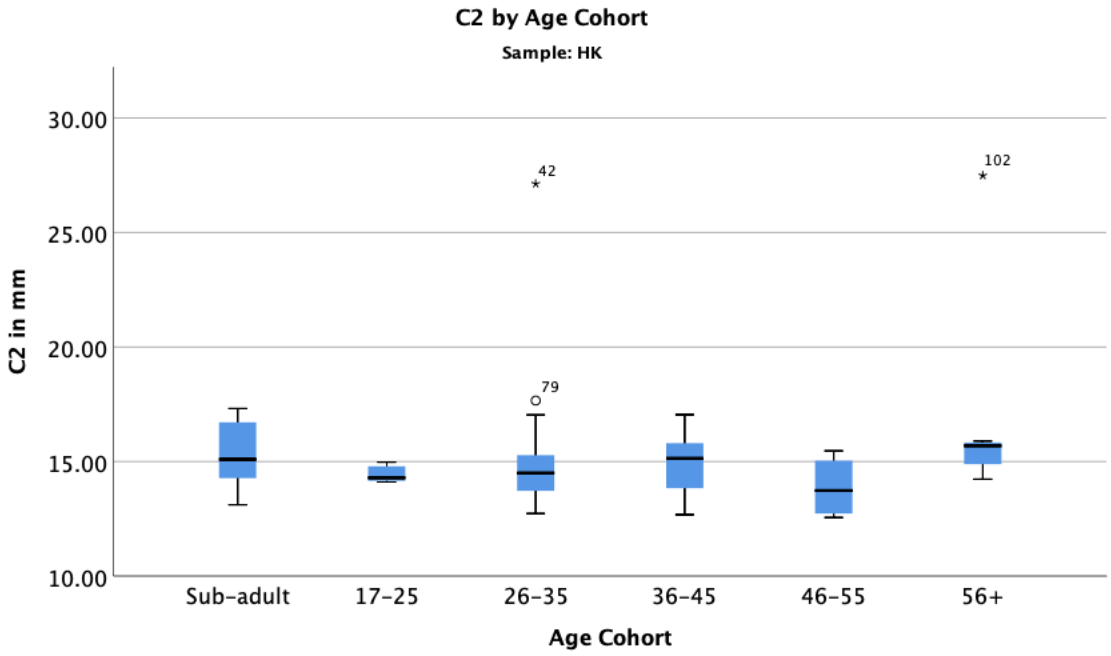

Figure 46: Boxplots of Hawikku C2 Midsagittal Diameter

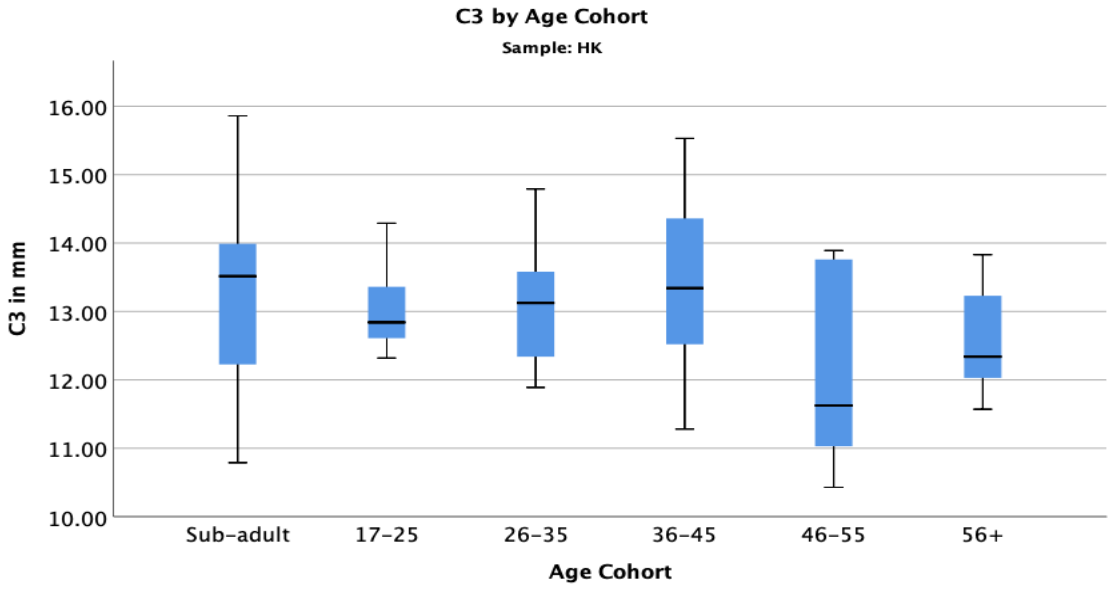

Figure 47: Boxplots of Hawikku C3 Midsagittal Diameter

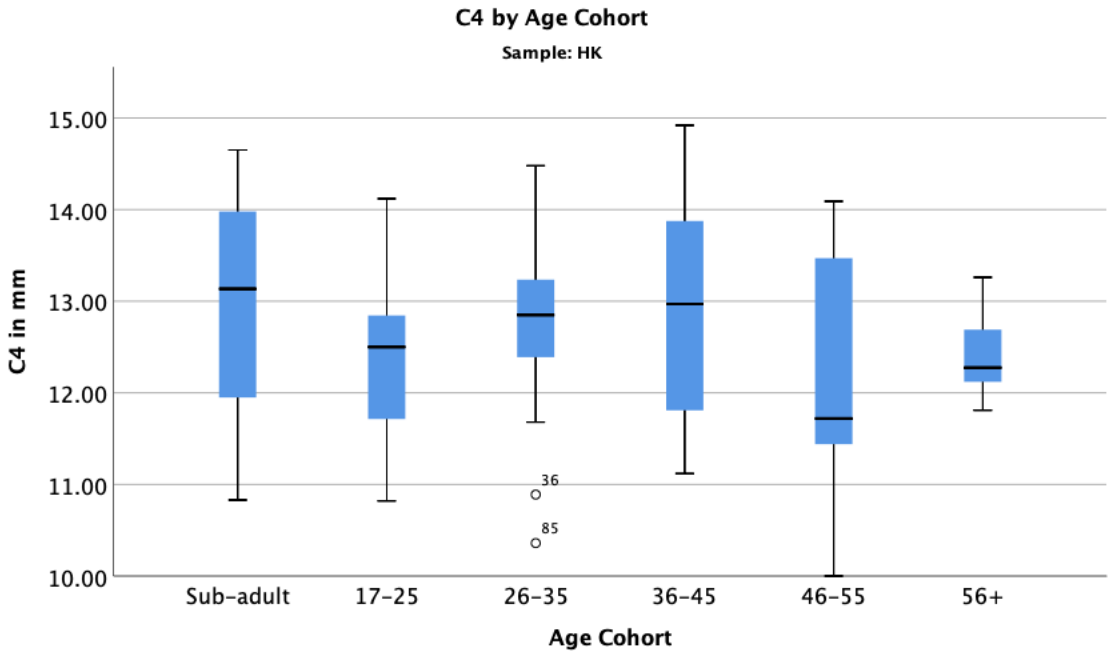

Figure 48: Boxplots of Hawikku C4 Midsagittal Diameter

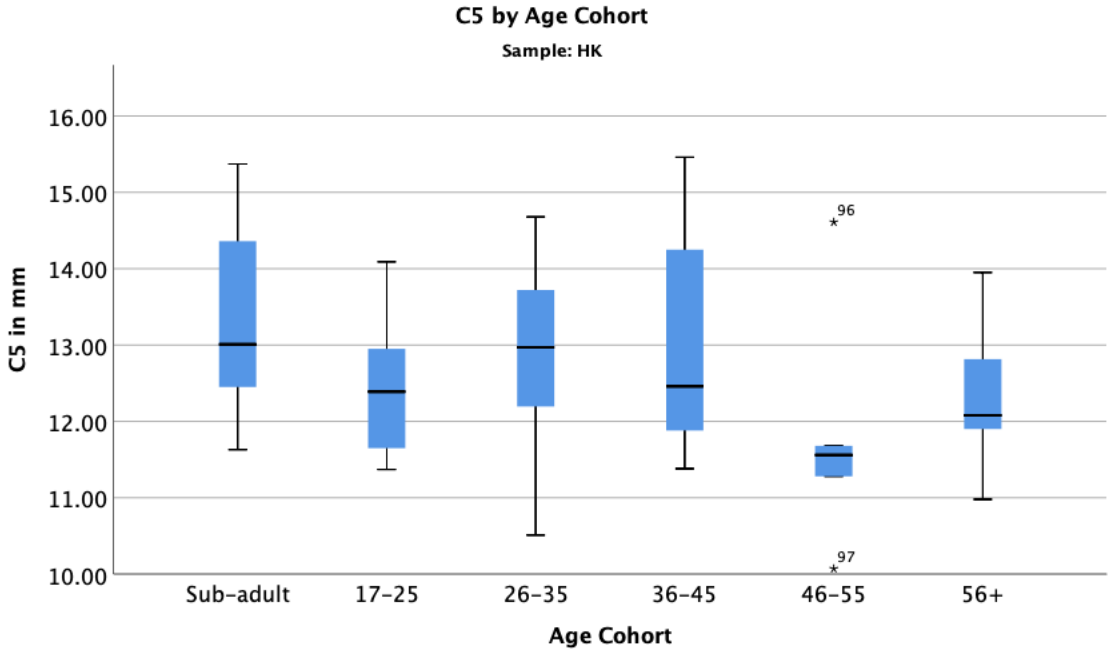

Figure 49: Boxplots of Hawikku C5 Midsagittal Diameter

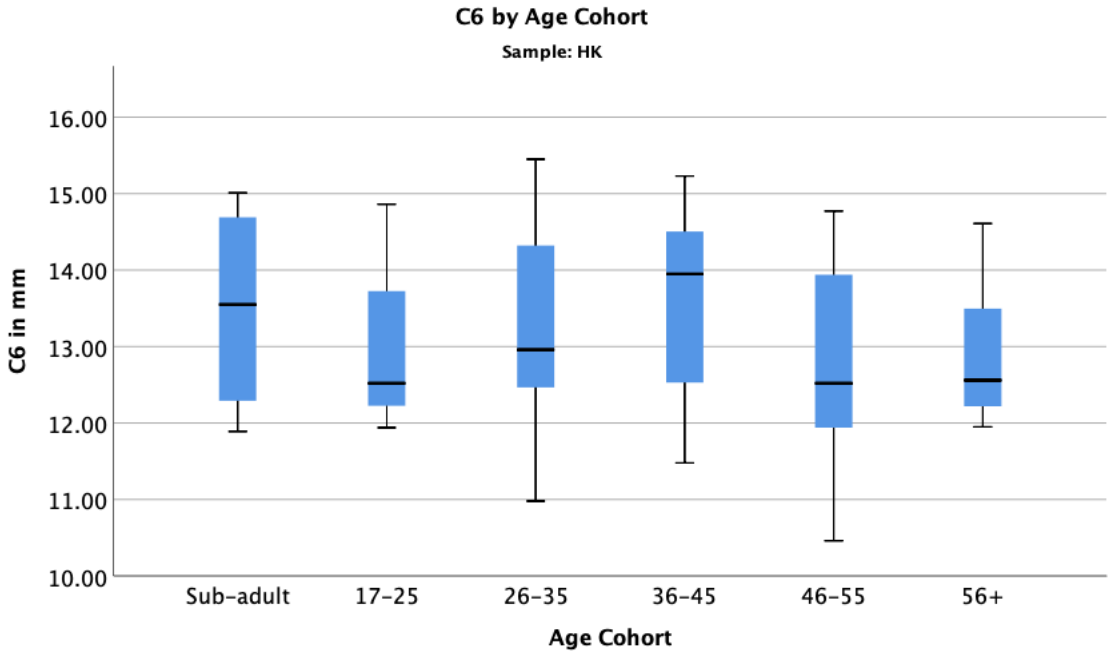

Figure 50: Boxplots of Hawikku C6 Midsagittal Diameter

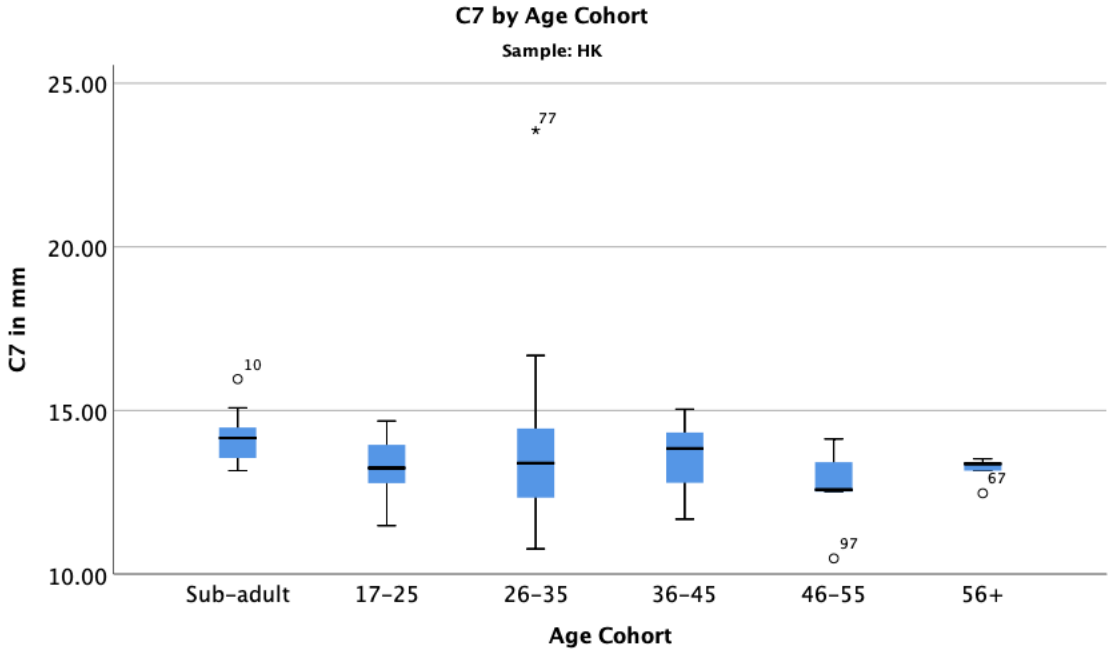

Figure 51: Boxplots of Hawikku C7 Midsagittal Diameter

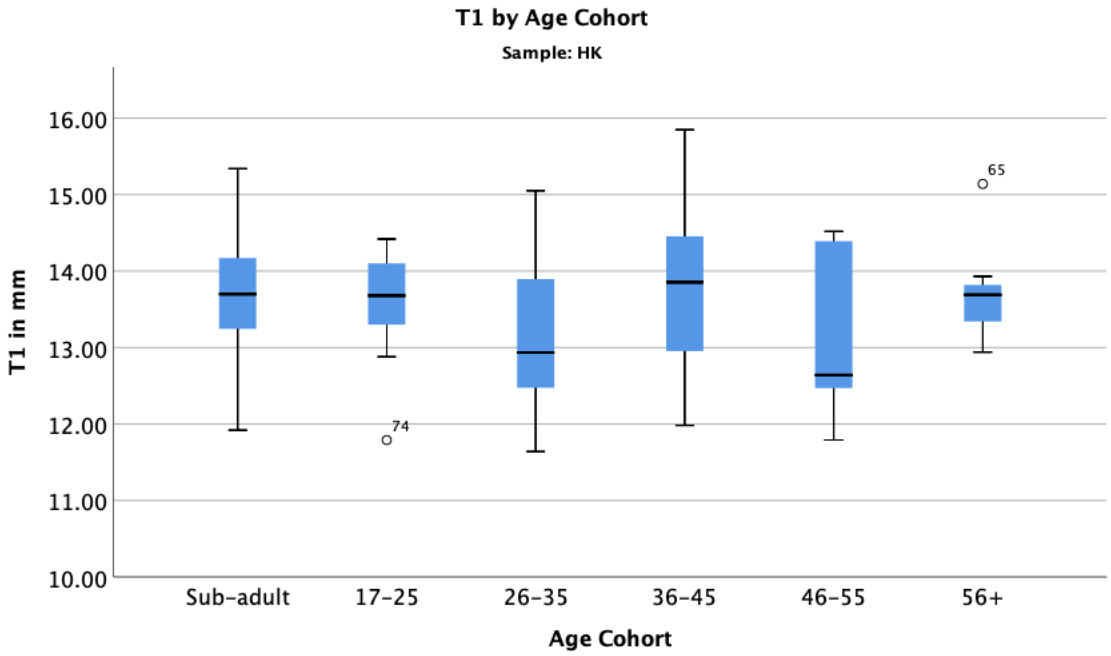

Figure 52: Boxplots of Hawikku T1 Midsagittal Diameter

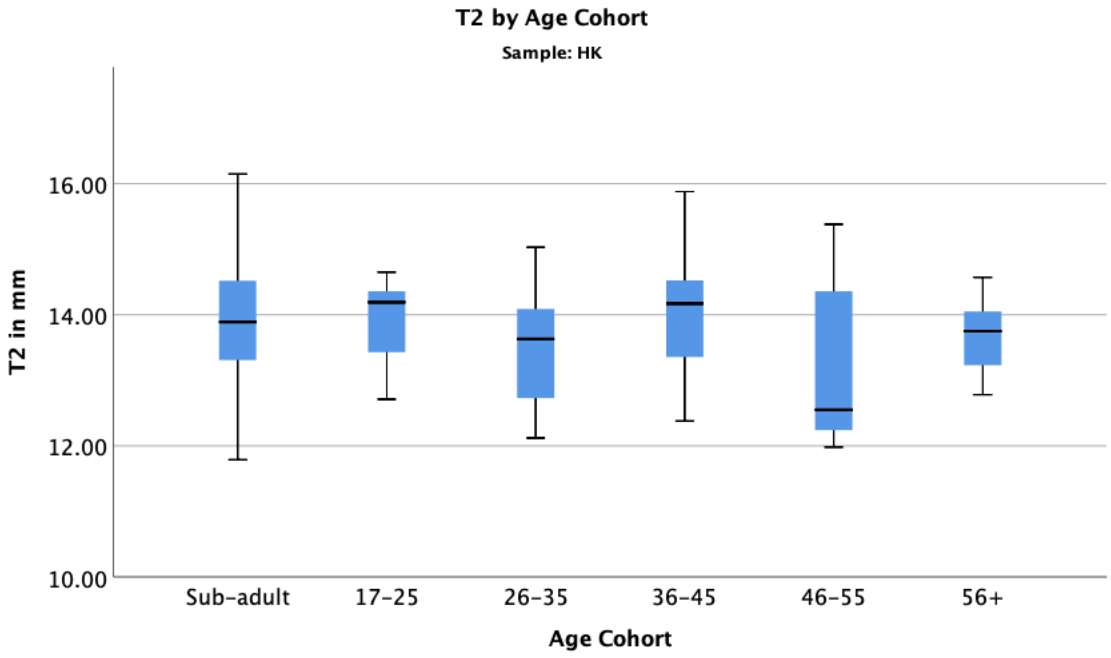

Figure 53: Boxplots of Hawikku T2 Midsagittal Diameter

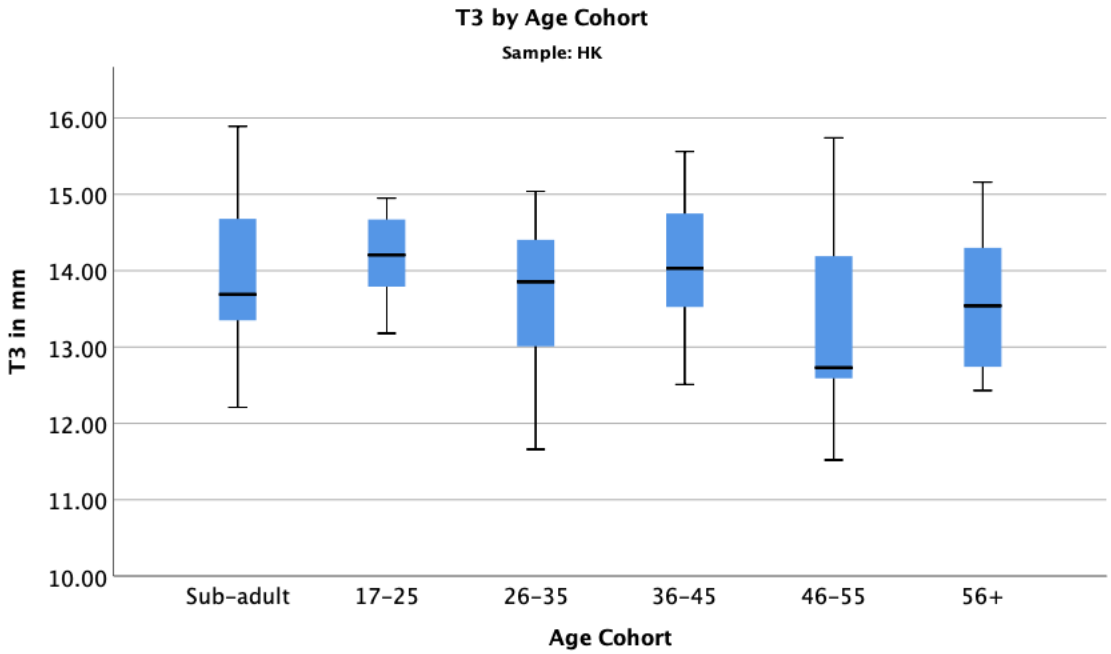

Figure 54: Boxplots of Hawikku T3 Midsagittal Diameter

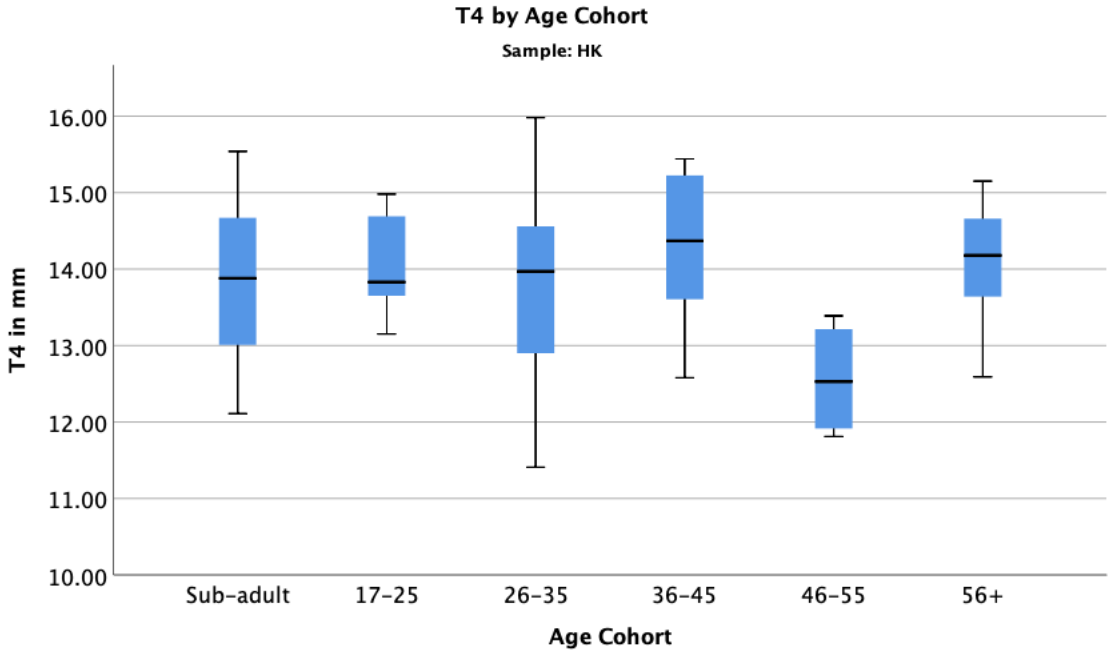

Figure 55: Boxplots of Hawikku T4 Midsagittal Diameter

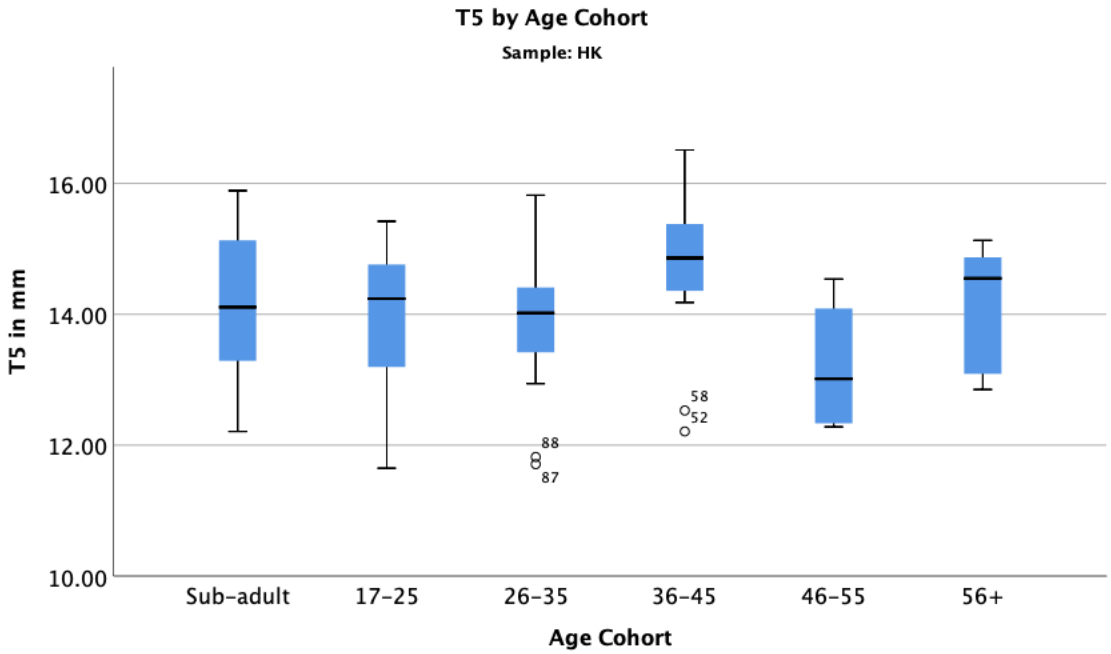

Figure 56: Boxplots of Hawikku T5 Midsagittal Diameter

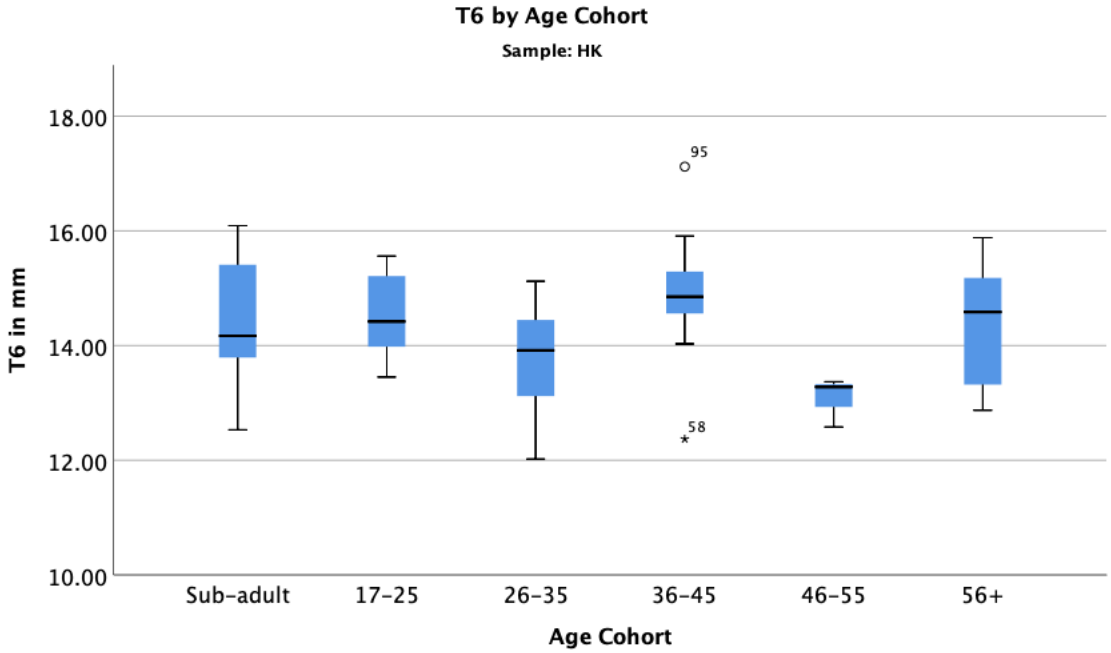

Figure 57: Boxplots of Hawikku T6 Midsagittal Diameter

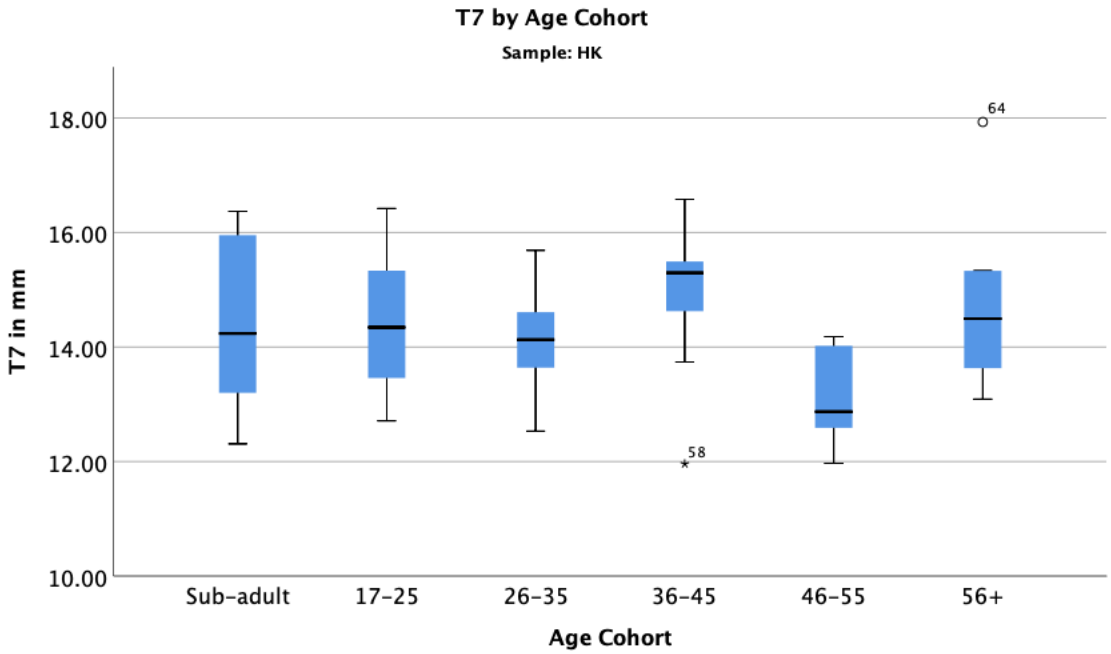

Figure 58: Boxplots of Hawikku T7 Midsagittal Diameter

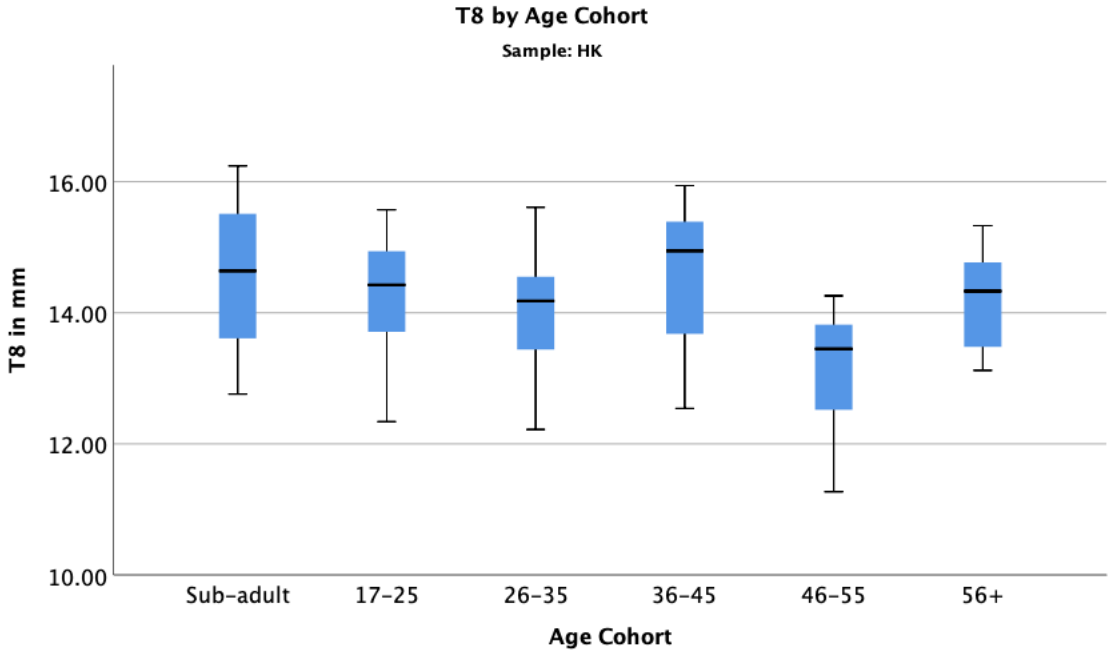

Figure 59: Boxplots of Hawikku T8 Midsagittal Diameter

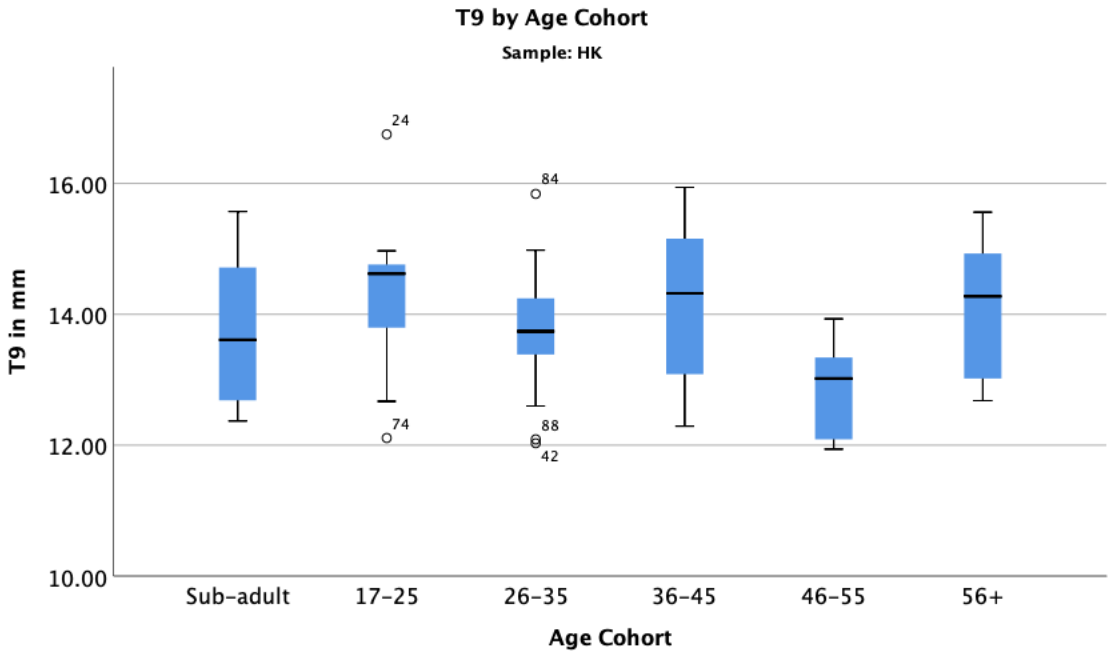

Figure 60: Boxplots of Hawikku T9 Midsagittal Diameter

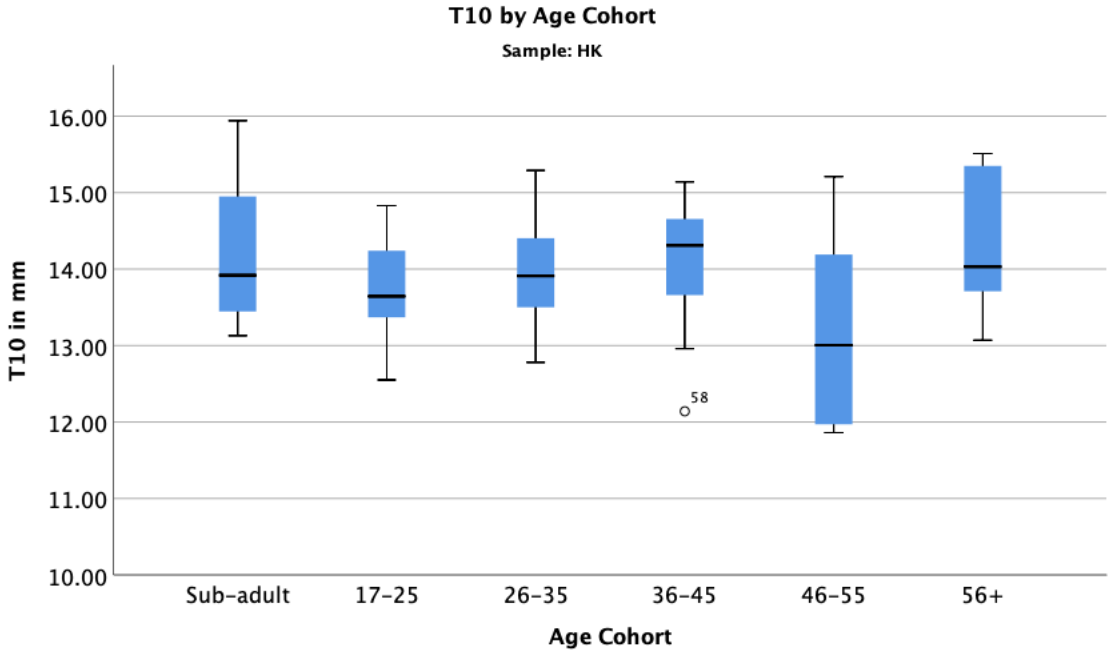

Figure 61: Boxplots of Hawikku T10 Midsagittal Diameter

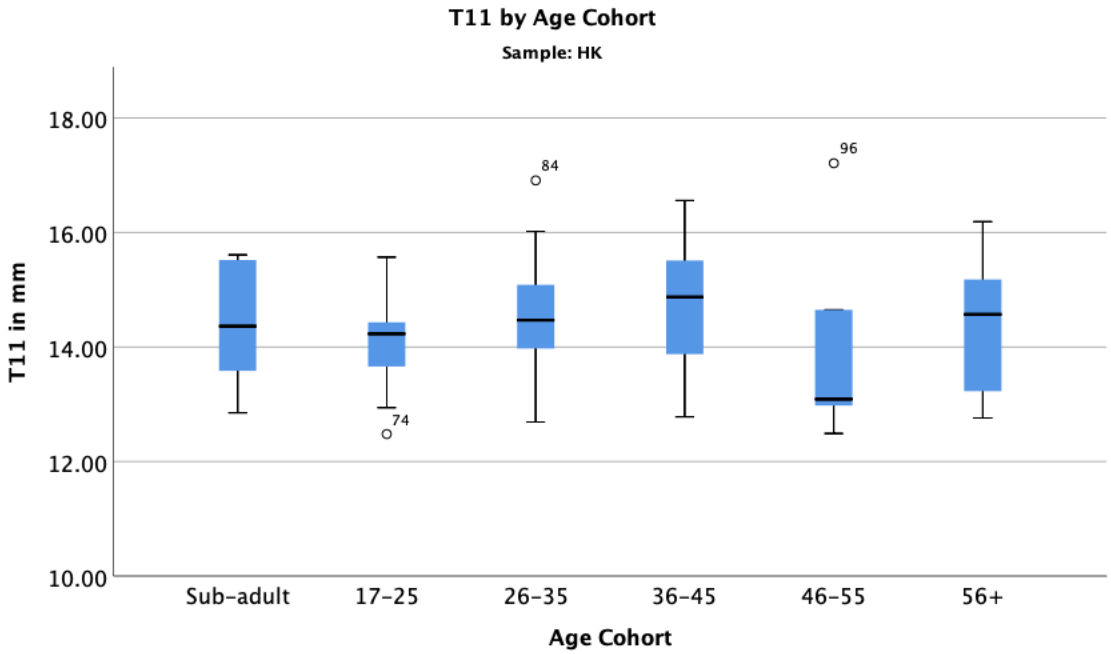

Figure 62: Boxplots of Hawikku T11 Midsagittal Diameter

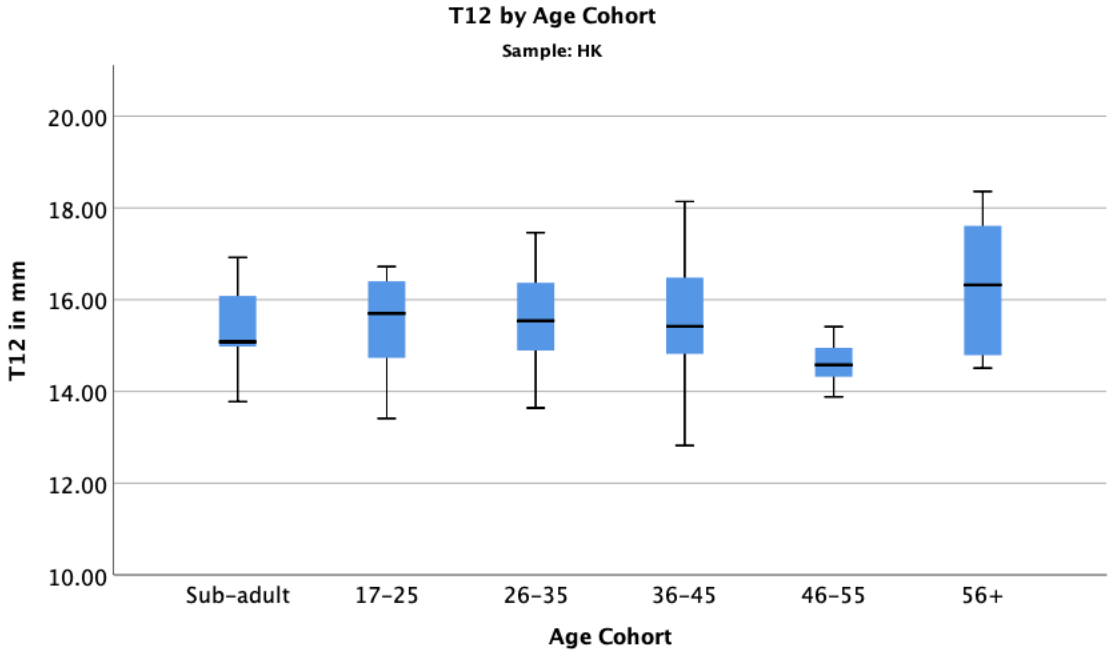

Figure 63: Boxplots of Hawikku T12 Midsagittal Diameter

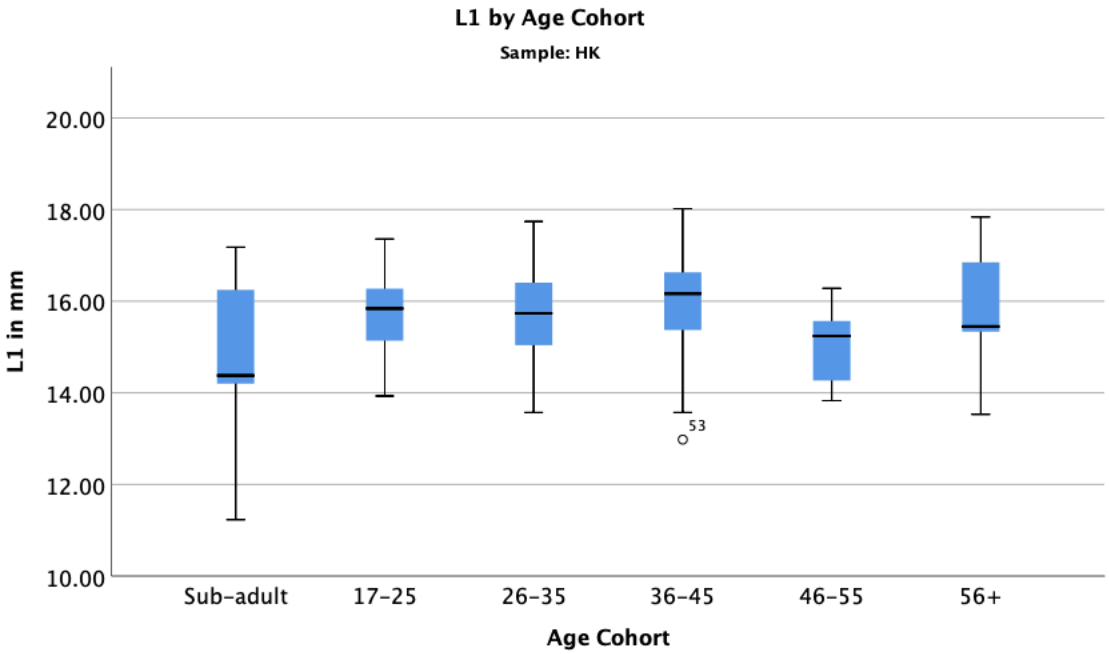

Figure 64: Boxplots of Hawikku L1 Midsagittal Diameter

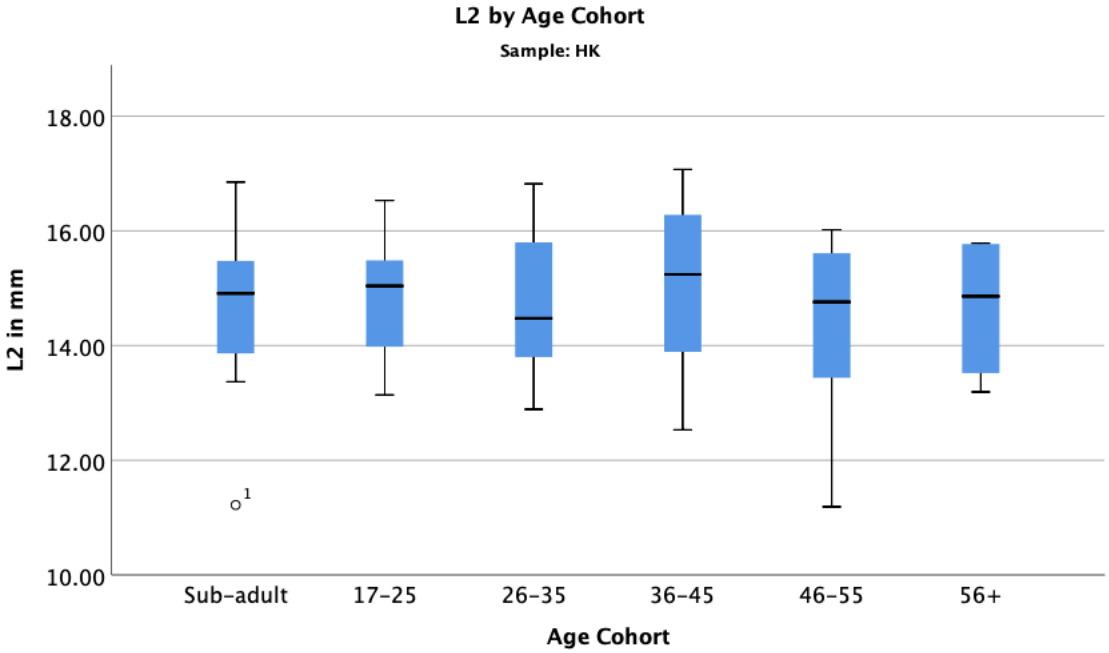

Figure 65: Boxplots of Hawikku L2 Midsagittal Diameter

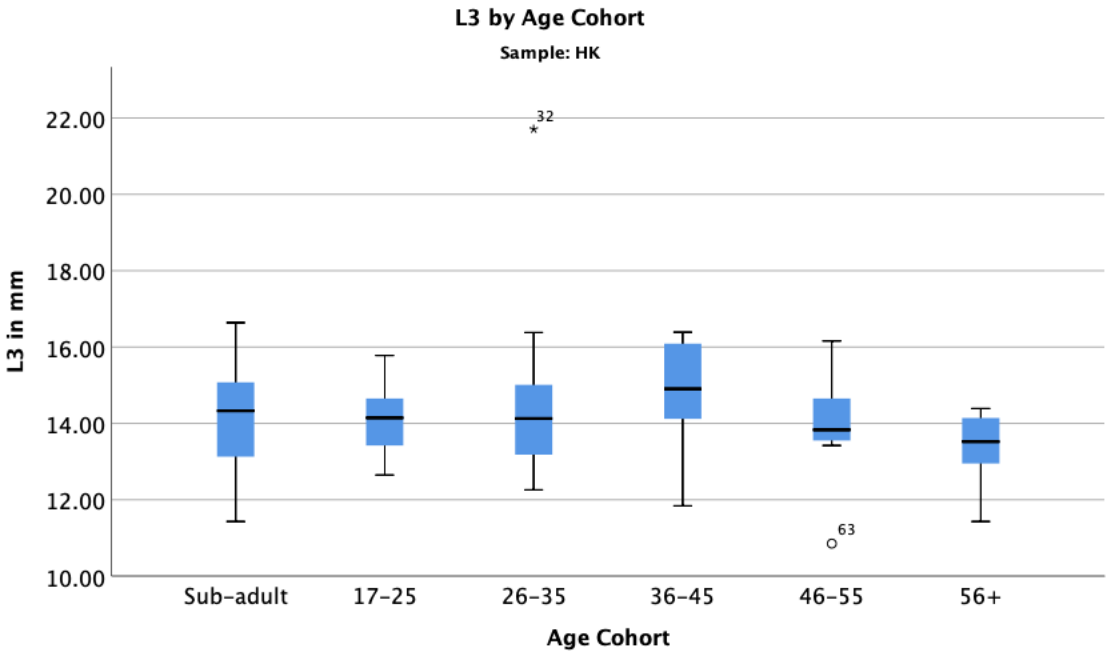

Figure 66: Boxplots of Hawikku L3 Midsagittal Diameter

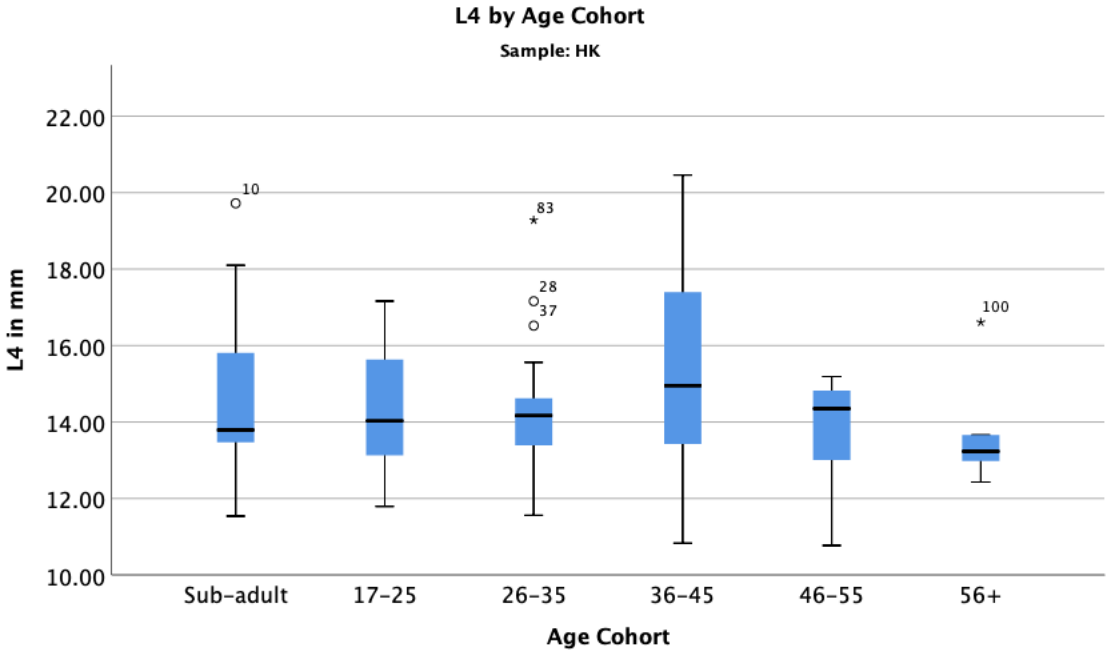

Figure 67: Boxplots of Hawikku L4 Midsagittal Diameter

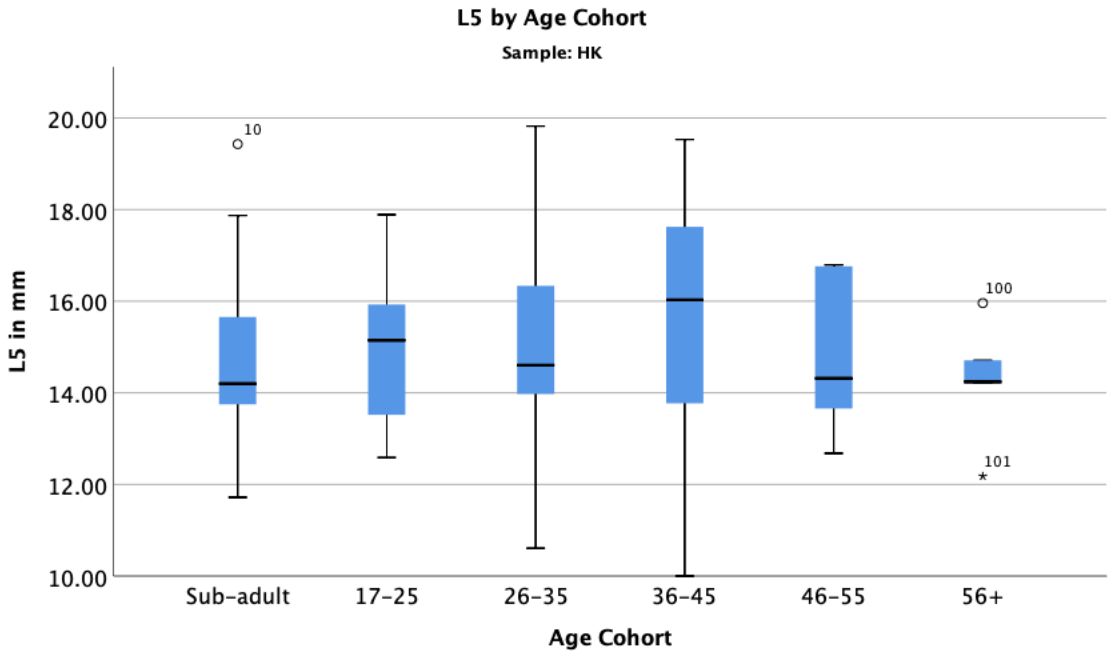

Figure 68: Boxplots of Hawikku L5 Midsagittal Diameter

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BIOGRAPHY

Samuel M. LoPresto was born and raised in Saline Michigan. He graduated from Saline High School in 2014. He received his Bachelor of Science in anthropology with a minor in biology from Grand Valley State University in 2018. He later moved to Virginia to attend George Mason University and receive his Masters in anthropology.