THE EMBODIMENT OF VERTEBRAL STRESS AMONG DOCUMENTED AMERICANS AND UNIDENTIFIED MIGRANTS FOUND ALONG THE UNITED STATES – MEXICO BORDER: A BIOCULTURAL APPROACH.

by

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DEDICATION

This research is dedicated to the underdogs, the wallflowers, the disadvantaged, the vulnerable, and the marginalized.

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LIST OF ABBREVIATIONS

Abbreviation	Description
Anteroposterior	AP
DOHaD	Developmental Origins of Health and Disease
FACTS	Forensic Anthropology Center at Texas State
LEH	Linear enamel hypoplasia
PCOME	Pima County Office of the Medical Examiner
OpID	Operation Identification
SNs	Schmorl's nodes
SES	Socioeconomic status
TXST	Texas State University
TXSTDSC	Texas State Donated Skeletal Collection
TR	Transverse
VNC	Vertebral neural canal

I. INTRODUCTION

Stressors experienced during an individual's lifetime can modify the skeleton, and one region that retains signatures of these lifetime stressors is the spinal column. Vertebrae reflect differential forms of stress and can assist with interpreting a decedent's lived experience. Through this thesis, an approach will be employed that emphasizes the potential for differential stress experiences, and their interconnectedness, through the simultaneous study of multiple skeletal indicators of stress and will address how developmental plasticity can affect adult susceptibility to pathologies. Specifically, vertebral neural canal (VNC) asymmetry, the degree of fluctuation in the VNC anteroposterior (AP) and transverse (TR) diameters from the expected norm, and Schmorl's nodes (SNs), a vertebral pathology, will allow for a longitudinal perspective to better understand how stress is experienced and skeletally embodied at different points in the life cycle. It is important to consider that human bodies are not isolated systems but interact with both tangible and non-tangible systems around us. Consequently, a biocultural approach which considers the wider context in which an individual lived can further elucidate what shaped their skeletal remains.

This research will consider correlations between pathological signatures of spinal health to investigate the embodiment of differential forms of stress such as socioeconomic status (SES) and occupation type at both the individual and population levels. The assessment of SNs will allow for a deepened understanding on whether patterns in appearance and severity are dictated by SES and occupation type, while the analysis of VNC asymmetry will provide insight on how stress is potentially embodied in

the spine in early childhood development and how it impacts an individual's predisposition for SNs later in life.

Three skeletal groups were assessed including documented individuals from the Texas State Donated Skeletal Collection (TXSTDSC) at Texas State University, and unidentified migrants curated by the Operation Identification project at the Forensic Anthropology Center at Texas State and at the Pima County Office of the Medical Examiner in Tucson, Arizona. By comparing individuals from these groups, whose different biosocial contexts will be described in more depth later in the chapter, this research can provide further insight into how different biosocial contexts are skeletally embodied.

Research Statements

More specifically, the following research questions and expectations will be tested: 1) Does VNC asymmetry significantly correlate with sex? If the VNC anteroposterior (AP) diameters reflects stress experiences during early childhood (> 5 years), and the VNC transverse (TR) diameter reflects stress experienced through adolescence (5 – 14 years), then females and males that experienced similar stress episodes in these periods will similar VNC asymmetry patterns.

2) Does VNC asymmetry significantly correlate with self-reported childhood SES in the TXSTDSC? If lower SES levels are a proxy for poor health outcomes (e.g., malnutrition, lack of medical care, etc.) that contribute to stress episodes and decreased energy allotment for VNC growth, then individuals from lower childhood SES levels will exhibit smaller VNC AP and TR diameters than those of higher SES levels.

3) Do SNs significantly correlate with sex? If the etiology of SNs is biomechanical in origin, reflecting increasing occurrences among males who are on average larger than females, then males will exhibit a higher prevalence of SNs than females.

4) Do SNs significantly correlate with self-reported occupation type in the TXSTDSC? If SNs are reflective of traumatic or work-related injury, then individuals with self-reported manual occupations will exhibit a higher prevalence of SNs than individuals with self-reported non-manual occupations.

5) Are VNC asymmetry and SNs significantly associated? If the etiology of SNs is developmental in origin, then the presence of SNs will be more strongly correlated with decreased VNC dimensions in comparison to vertebrae unaffected by SNs.

6) How do patterns in VNC and SNs compare between the three samples? If the unidentified migrants recovered along the Arizona - Mexico border reflect Mexican nationals of lower SES levels that are escaping impoverished areas, then they will exhibit increased VNC asymmetry in early childhood and adolescence and SNs presence. If the unidentified migrants recovered along the Texas - Mexico border reflect South American nationals of mixed SES levels that are escaping sociopolitical violence, then they will exhibit increasing VNC asymmetry in early childhood and adolescence and SNs presence. If the documented Americans in the TXSTDSC reflect different SES levels, then those that self-reported a lower childhood SES will exhibit increased VNC asymmetry in early childhood and adolescence and SNs presence than those that self-reported a higher childhood SES.

Overall, it is expected that the results from this research will allow for further insight into how differences in the stress experience, and resulting developing plasticity of vertebral elements, increase the susceptibility of other vertebral pathologies in adulthood. Application of these research questions in comparing unidentified migrants and documented Americans is particularly important because it can highlight differences in the life experience in these regions of the world and provide skeletal evidence of the disparities migrants face in their communities and seek to flee when crossing into the United States.

The remainder of Chapter I (Introduction) will further explore necessary background information on relevant biological and cultural influences on the spinal column, Chapter II (Theory) will present the theoretical frameworks from which interpretations will be made in this study and explore how they have been employed in previous studies, Chapter III (Materials and Methods) will present selection parameters and techniques used to collect and statistically test the data, Chapter IV (Results) will present the findings of this analyses, and Chapter V (Discussion) will provide an interpretation of the results and their implications on future research directions.

Stress in Osteology

The broad use of the term "stress" throughout various scientific fields as well as within the English language generally has contributed to its vague usage and yielded different implications for different research questions. According to Huss-Ashmore and colleagues' (1982) definition, stress in the clinical sense is the general disruption of an individual's physiological balance. For osteologists, studying stress indicates a focus on disruptions to the human body and the indirect consequences of biological strain of soft

tissues on hard tissues including bone. Factors that cause these disruptions could be from the physical environment including biological pathogens and illness, malnutrition, or physical labor. They could also be sociocultural in nature, including structural violence and epigenetic accumulations of trauma (Klaus 2014). Identification of different skeletal markers of stress and understanding their etiology can provide insight as to when the stress event was experienced and subsequently embodied by the skeletal system.

For this research, "stress" will be used to encapsulate individual and population-level experiences in the biocultural environment that indirectly lead to skeletal markers as a consequence of a disruption to physiological homeostasis and normal cellular function (Hillson 2014; Temple and Goodman 2014). When influences cause a disruption to growth resulting in developmental plasticity or cause the formation of a skeletal pathology later in life, the resulting markers are known as skeletal indicators of stress. This chapter will provide an overview of the intricacies of these interactions.

Developmental Plasticity

While development of an organism normally follows a trajectory towards maturation, the limits of which are predetermined by genes, this trajectory can be altered as an adaptive process during phases of the life course (Low et al. 2012; Halfon et al., 2014; Agarwal, 2016). In early fetal or childhood periods, stressors such as malnutrition or illness can result in the reallocation of energy towards development of the most essential structures, i.e., the brain. This reallocation results in the withholding of energy from other developing parts of the body and has been found to be associated with poorer health trajectories into adulthood (Agarwal 2016; Kuzawa 2005; Worthman and Kuzara, 2005; Leonard and Robertson, 1992; Bogin, 1995). This ability to respond to the

environment, including disruptions to growth, developmental adaptations, and acclimatization is referred to as plasticity and accounts for much of the phenotypic variation in humans (Roberts 1995). Additionally, these environmental influences can affect the epigenome, in which the patterns of gene expression, rather than the nucleotide sequences, are altered (Gowland 2015).

A particular time frame of interest for this research is plasticity in response to the environment experienced *in-utero* and through early childhood and adolescence. Baker's Hypothesis suggests that insults to the *in-utero* environment could result in "fetal programming" and alter the growth trajectories of developing structures for life as the fetus's adaptive response attempts to better restrict energy typically allocated for growth and maintenance (Agarwal 2016; Barker et al. 1989; Kuh and Smith 2004). If these environmental signals continue through infancy and early childhood, they can further result in postnatal plasticity and contribute to increased phenotypic variation (Temple 2018). The Developmental Origins of Health and Disease (DOHaD) Hypothesis proposes that this plasticity experienced *in-utero* and throughout early childhood affects the health trajectories of individuals and predisposes them to increased susceptibility to disease as adults (Agarwal 2016; Newnham 2007). This hypothesis, and its interpretative power over data, will be discussed more in Chapter II. Awareness of skeletal growth patterns, and the ages at which specific parts of the skeleton reach maturation, allows for further insight into age related disruptions to developmental growth.

Development of the Spinal Column

Different components of the vertebrae serve different primary purposes in the body. While the vertebral bodies provide support to the upper body and bear its weight,

the transverse processes anchor adjacent ribs and soft tissues, and the vertebral neural canal (VNC) protects the spinal cord within the bony structure (White and Folkens 2005). Development of the entire spinal column continues from *in-utero* throughout adolescence making its structures susceptible to developmental plasticity. This has resulted in stages of vertebral growth and development being correlated with specific ages. Understanding the typical developmental patterns of the spinal column allows for a better identification and understanding of developmental anomalies.

During development, each vertebra has three primary ossification centers including the anterior centrum, and two posteriorly at either side of the VNC (Figure 1). At birth the VNC consists of two dorsal neural arch segments and the anterior body which are connected by cartilage. In the first postnatal year, the posterior portion of the VNC in the lower thoracic and upper lumbar vertebrae fuse to the spinous process (Scheuer and Black 1993). This fusion commences in the cervical and lower lumber vertebrae in the fifth postnatal year, so that by five to size years of age, the VNC antero-posterior (AP) diameters have reached maturation. The VNC transverse (TR) diameters, however, continue to grow through late childhood so that they reach maturation around 14 years of age (Bryant 2003; Watts 2012; Benson et al. 2010). While individuals may experience catch-up growth in adulthood stature in instances of improved environmental situations, the antero-posterior (AP) and transverse (TR) widths of VNCs will remain stable and thus can reflect early developmental stress (Hinck et al. 1966; Clark et al. 1985; Watts 2011).

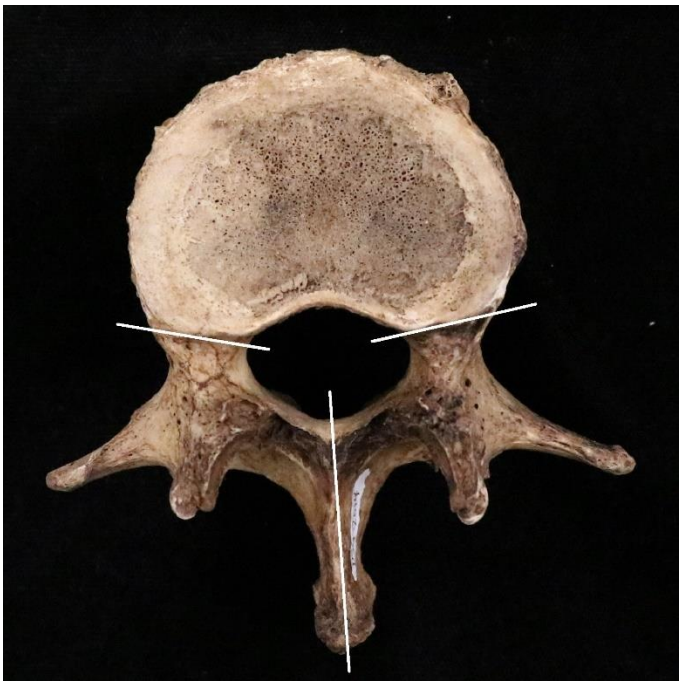

Figure 1. L1 vertebra of D52-2014 in the TXSTDSC. White lines indicate where the vertebral neural canal and vertebral body fused during development.

The mature vertebral neural canal is systematically asymmetrical along the spinal column (Masharawi and Salame 2011). Meta-analysis on mature VNC dimensions have found typical growth patterns in the VNC AP and TR diameters through the thoracic and lumbar vertebrae which can used as expected patterns of "normal" development. Masharawi and Salame (2011) found that the VNC AP width typically follows a sinusoidal pattern, increasing in size through the upper thoracic (T1-T5), decreasing through the middle thoracic (T5-T10), sharply increasing in the lower thoracic (T10-12), decreasing in the upper lumbar (L1-3), and increasing in the lower lumbar (L4-L5). VNC TR width typically decreased in size through the upper thoracic (T1-T6) followed by an increase in size through the lower lumbar (L5) (Figure 2). The authors also found that typical VNC shapes resulted from these expected dimensions and their relative dimensions (Table 1). Studies found no significant difference between the VNC sizes and

shapes in males and females despite sexual dimorphism (Watts 2011; Masharawi and Salame 2011).

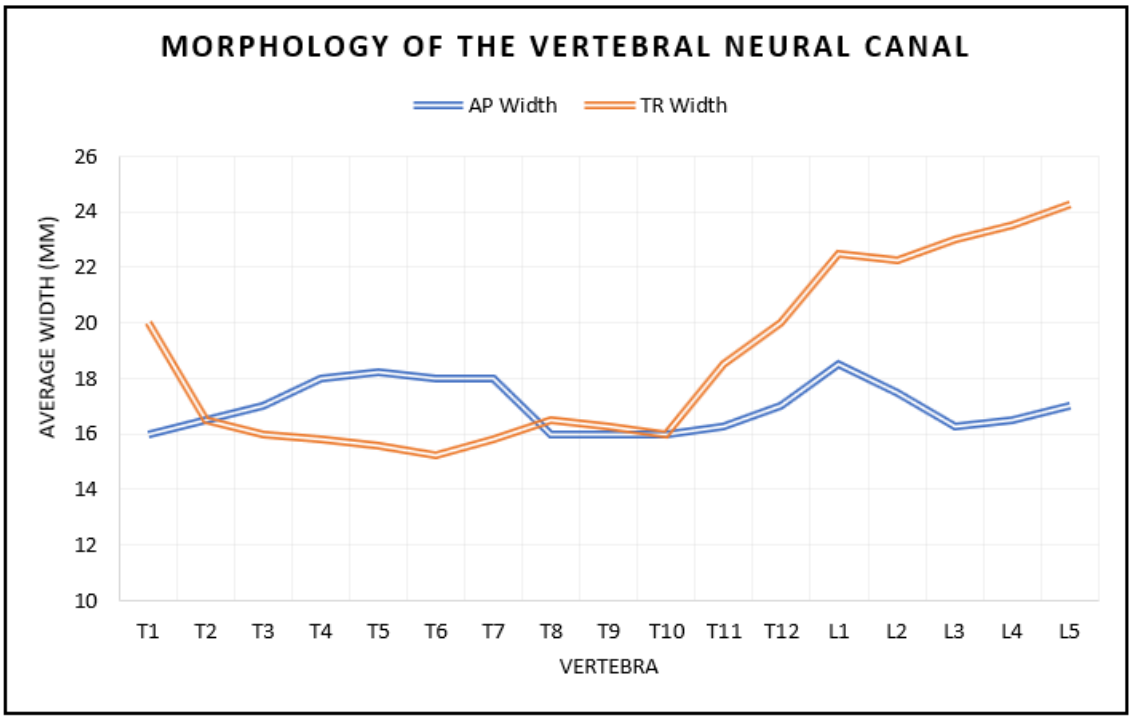

Figure 2. Expected VNC AP and TR dimensions throughout the spinal column as adapted from Masharawi and Salame's (2011) expected vertebral canal superior width (VCSW) and vertebral canal superior length (VCSL).

Table 1. Expected VNC AP/TR width ratios and shapes throughout the spinal column. Data from Masharawi and Salme (2011).

Vertebral Segment	**Expected VNC Dimensions and Shape**
T1	Anteroposterior (AP) width< Transverse (TR) width (oval shape)
T2	AP width = TR width (round shape)
T3-T9	AP width > TR width (inverted oval shape)
T10	AP width = TR width (round shape)
T11-L5	AP width < TR width (oval shape)

In a study on White European populations, Eisenstein (1983) found that the VNC dimensions exhibited normal skeletal variation that ranged from 12-22 mm in the AP widths and 19-32 mm in the TR widths in the lumbar vertebrae. Smaller VNC dimensions in mature individuals are considered pathologically small. While this proxy

for “normal” growth has limitations across different populations, applications of clinical perspectives as such can help to distinguish between differences due to genetically shorter individuals versus growth impairment (Watts 2015).

The remainder of the spinal column, including the annular rings, continue ossification and fuse to the vertebral body through adolescence, reaching maturation by sixteen to eighteen years of age (Benson et al. 2010). While the bony aspects of the spinal column follow the patterns of growth described above, it is important to consider other components of the spinal column that could influence these structures, including the spinal cord and nerves, the spinal muscles and connective tissues, and the intervertebral discs. The spinal cord and nerves primarily develop during fetal life and slow growth after birth, at which point the vertebrae experience an increased rate of development relative to the neural elements (Benson et al. 2010). The spinal muscles and connective tissues can continue to experience growth throughout adolescence and adulthood relative to locomotive patterns and biomechanical pressures. The annular rings and intervertebral discs reach maturation typically by eighteen years of age and remain in this state until around 40 years of age, at which point around 50% of the population experiences intervertebral discs degeneration and become more susceptible to other degenerative diseases (Kyere et al. 2012).

Vertebral Neural Canal (VNC) Asymmetry

Stages of vertebral growth and development have been found to correlate with specific ages. While individuals can experience catch-up growth in stature through adulthood, anteroposterior (AP) and transverse (TR) diameters of the vertebral neural canal (VNC) (Figure 3) will remain stable after maturation, by five and fourteen years of

age respectively, making them ideal indicators of early developmental stress (Hinck et al. 1966; Clark et al. 1985; Watts 2011). Due to differences in developmental patterns along the spinal column, and the ages at which each of the vertebrae reach maturation, the different vertebrae have different potentials in embodying growth disruptions, with the lower thoracic and lumbar vertebrae having the largest potential (Watts 2011).

The VNC is systematically asymmetrical along the spinal column as discussed in the development of the spinal column (Masharawi and Salame 2011). The degree of asymmetry, and the disruption of growth in response to negative environmental signals from the biosocial context, has been found to serve as a non-specific stress indicator. For this research, VNC asymmetry is defined as the fluctuations in VNC AP and TR sizes from the expected VNC AP/TR ratio as described by Masharawi and Salame (2011) (Table 1). In particular, the AP and TR widths of the VNC have been found to be associated with morbidity and mortality, with statistically significant correlations between smaller thoracic AP widths and increased adult mortality regardless of sex (Watts 2011; Watts 2015). Smaller TR widths have been found to be correlated with poorer health and lower educational levels, a proxy for socioeconomic status, in a clinical study (Porter et al. 1987). While the AP width of the VNC reaches its mature size by around five years of age, the TR widths continue to grow until around fourteen years of age. This differential rate in reaching maturation allows for an opportunity for these features to differentially embody stress. Restricted growth in the AP and TR widths of the VNC can be reflective of both infancy (birth to one year of age) and early juvenile (between one to ten years of age) and adolescence (defined for this research, between ten years of age until 18 years of age) (Schillaci et al. 2011).

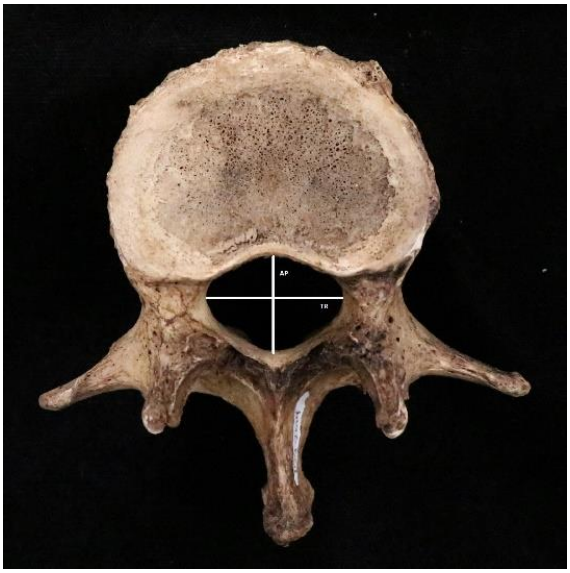

Figure 3. Superior view of L1 from D52-2014 in the Texas State Donated Skeletal Collection with the vertebral neural canal anteroposterior and transverse diameters marked.

Previous studies. Watts (2011; 2013; 2015) examined the VNC sizes of individuals in relation to childhood stress and adult longevity. When comparing VNC size to adult stature, Watts (2011) found that VNC diameters served as a better indicator of stress than stature because while individuals could experience catch up growth, their VNC AP and TR diameters reach maturation earlier and served as windows into development. Watts (2013) also compared the correlations between VNC sizes and linear enamel hypoplasia (LEH), another non-specific childhood stress indicator, incidences with adult mortality and found that VNC was a better predictor of age at death. While LEH results from stress experienced in infancy when teeth are developing, VNC asymmetry occurs over a larger window of time. Watts (2013) interpreted this to indicate the increasingly significant effect stress can have on adulthood health throughout early childhood and adolescence, rather than infancy alone. Historical documents allowed Watts (2013; 2015) to interpret a social context for the post-medieval London sample assessed in her study and identify individuals as either low or high status. Watts (2015) assessed the correlation between

VNC dimensions and age at death and found that high status females and low status males exhibited smaller TR diameters than high status males. These size differences correlated with mortality, with smaller TR diameters reflecting an increased risk of adult mortality. Watts interpreted these findings to be evidence of socioeconomic inequalities that affected individuals differently depending on social status and sex.

Significance. While VNC sizes have been studied in relation to adult morbidity and mortality in bioarchaeological contexts, this research will be the first study to use a modern, documented skeletal collection allowing for direct testing of associations between VNC and documented demographic data. Further, the analysis of VNC asymmetry among three modern skeletal samples will provide insight on how stress is embodied in the spine during early childhood development in three different contexts, and how it impacts an individual's predisposition for developing SNs later in life.

Schmorl's Nodes

Schmorl's nodes (SN) were originally described in 1927 by pathologist Christian Georg Schmorl as a lesion on the vertebral body surface that results from the herniation of the nucleus pulposus into the adjacent vertebra (Figure 4) (Schmorl 1927). A common pathology observed in both prehistoric and modern populations, SN have traditionally been utilized by bioarchaeologists to assist in understanding and reconstructing the health of past populations. Historically, the presence/absence of these lesions has been used as an indication of injury sustained during life, likely inflicted from high axial loading biomechanical forces, or as evidence for manual occupations. For example, Dar et al. (2010) interpreted higher prevalence of SN around the thoracolumbar junction, where axial loading pressures are highest, to be the result of these pressures that humans, as

bipeds, experience increasingly so towards the inferior portion of the spinal column. Additionally, studies showing associations between SNs presence and sex, with a higher prevalence among males in comparison to females, attributed this correlation to how males on average are larger than females and thus experience increasing axial loading pressures from increased body weight (Dar et al. 2009; Plomp et al. 2012; Samartiz et al. 2016).

At the population level, bioarchaeologists have traditionally interpreted increasing presence of SN among certain groups to be associated with the wear and tear stress of certain lifestyles, i.e. increasing prevalence indicating increasingly difficult manual labor. These interpretations could significantly influence other anthropological questions including social status, gender roles, et cetera. For example, Faccia and Williams (2008) used SNs to assess activity patterns and interpreted them as embodiments of gendered activities. These interpretations are based on the assumption that herniation of the intervertebral disc is the direct consequence of the exceeding limits of axial loading pressures in the spinal column.

Recent studies. More recent studies have undertaken different perspectives to better understand the etiology of SN beyond presence/absence and have generally pointed towards some sort of morphological or developmental origin, with the presence of SN being directly related to the size and shape of the vertebrae. Plomp et al. (2012) showed an association between the prevalence and severity of SN and vertebral body morphology. While the vertebral body reaches maturation around the same time as the VNC, the VNC allows for a deeper analysis of embodied stress due to differences in which the AP and TR diameters reach maturation. Epidemiological studies using

magnetic resonance imaging (MRI) have also found a strong heritability of SN indicating that they may have an (epi)genetic or embryological etiology in addition to developmental factors (Coventry et al. 1945; Pfirrmann and Resnick 2001; Moore 1998; Kyere et al. 2012). These studies provide evidence that developmental factors more likely contribute to the occurrence of SN and increasing an individual's susceptibility to SN as a result of wear-and-tear stress over the course of their lifetimes (Faccia et al. 2007; Dar et al. 2009; Plomp et al. 2012; Plomp et al. 2014; Plomp et al. 2015).

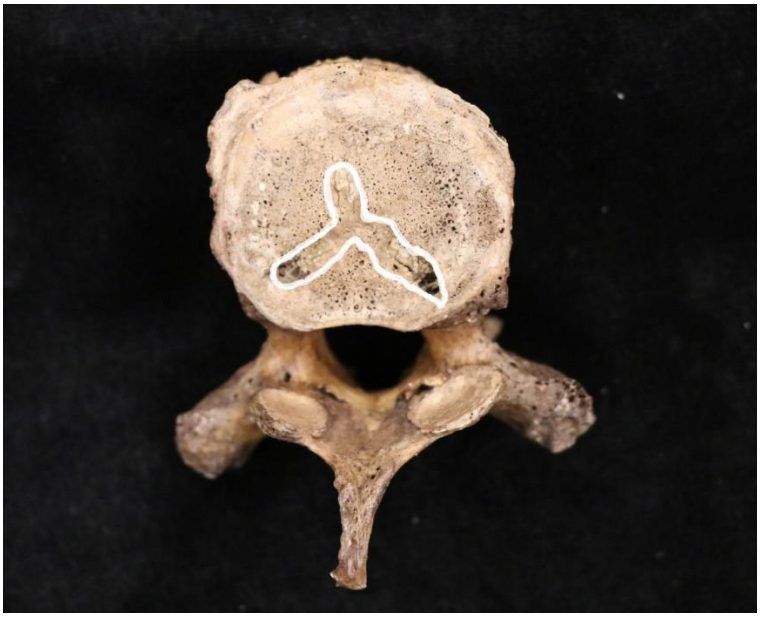

Figure 4. The presence of a Schmorl's node (outlined) on the inferior surface of T11 of D68-2015 in the Texas State Donated Skeletal Collection.

Significance. While previous research into the etiology of SN has directly investigated associations between pathology and vertebral morphology to draw interpretations on their association with developmental factors, these studies have utilized samples in which broad assumptions were made regarding demographic composition, and direct variables could not be tested. No published research has tested for correlations between SN and known socioeconomic status, known occupation type, or ancestries other than American White and American Black (Plomp et al. 2012; 2015). The analysis of SNs in this

research, particularly in the Texas State Donated Skeletal Collection (TXSTDSC) with documented demographic information, will allow for an increased awareness on whether patterns in appearance and severity are related to socioeconomic status and occupation. This analysis will allow for a deepened understanding of how this vertebral pathology results from physical wear-and-tear stress and reflects differing occupations and socioeconomic statuses. A cross-comparative analysis of SNs amongst three populations that presumably experienced different biosocial contexts will allow a deepened understanding on how SN potentially reflect different life histories.

Socioeconomic Status in the United States

Socioeconomic status (SES) serves as a classification method that divides a society into economic groups where status is assigned based on monetary worth, with the least amount of resources being assigned to a low SES and an accumulation of wealth being assigned a high SES (Baker 2014). This classification is arbitrary and relative to the context in which it is employed, as well as variable depending on the subjectivity of those determining the classification (Baker 2014). For this research, SES levels will be separated into low, low-middle, middle, upper-middle, and upper as they are in the questionnaire that every donor or next-of-kin completes before their skeleton is acquisitioned into the Texas State Donated Skeletal Collection (TXSTDSC). There is no further description of these levels on the questionnaire, so the interpretation is up to the individual, e.g., donor or next of kin, completing the form.

While previous research into VNC asymmetry has used historical context to infer the SES of mortuary sites (Watts 2011), this research will test the relationship between self-reported SES in a documented skeletal collection and VNC sizes. Every individual in

the TXSTDSC is a known individual with documented demographic information that is either self-reported or reported by the next of kin. As part of a questionnaire, individuals are asked to identify their childhood SES and adulthood SES from one of the following ranges: low, low-middle, middle, upper-middle, and upper. These categories were designed to represent the socioeconomic levels of American society which are based around household income relative to location.

Occupation Type in the United States

Bioarchaeological analyses have historically assumed simple cause-and effect relationships between presumed occupational stress markers (i.e., Schmorl's nodes) and occupation type (i.e., manual versus non-manual) in which lifestyle results in stress markers. However, published research has not clearly defined activity or occupation types nor directly tested correlations between the two. By defining occupation type, and explicitly testing the relationship between SN and occupation, this research will further elucidate this assumed relationship. Villotte et al. (2010) defined four categories of occupation to account for various types of activities, including nonmanual workers and three types of manual workers (Table 2). Classification of occupation type for individuals in the Texas State Donated Skeletal Collection (TXSTDSC) will completed according to these definitions.

Table 2. Occupation types as defined by Villotte et al. (2010).

Group	**Type of Occupation**	**Example of Occupation**
Group A	Nonmanual workers	White collar workers
Group B	Manual workers, no forceful tasks	Homemakers, teachers, tailors
Group C	Manual workers, probable heavy loads and forceful tasks	Blue collar workers, construction, carpenters
Group D	Manual workers, heavy physical labor and forceful tasks	Soldiers, day laborers

Unidentified Migrants Found Along the United States - Mexico Border

The assessment of VNC dimensions and SN in individuals from different biosocial contexts allows for a better understanding of how these processes can differ between populations. While documented individuals from the TXSTDSC sample provide a way to directly assess correlations between VNC and SN with known demographic information, application of this knowledge to analyses of unidentified migrants found along the United States - Mexico border will test its potential in be used as a proxy for different embodied biosocial contexts.

Humanitarian crisis. An amalgamation of factors including mass deportations, inadequate asylum procedures, and a revolving door of vulnerable migrants who would rather attempt to cross the border than return to various forms of violence at home, has resulted in more than 6,000 deaths along the United States – Mexico border since 2000 (Fernandez 2017). Previously, the highest concentrations of migrants, both detained or recovered, were along the Arizona portion of the U.S. – Mexico border. However, between 2011 and 2015, the number of detained migrants along the Texas portion of the border surpassed Arizona's, increasing from 4,000 to 35,000 individuals (Fernandez 2017). With the implementation of current United States Border Patrol protocols along the border, migrants are using riskier methods to cross undetected and as a result, there has been an increase in the cases of unknown migrant deaths relative to apprehensions in in both Arizona and Texas (Figure 5) (Martinez et al. 2014).

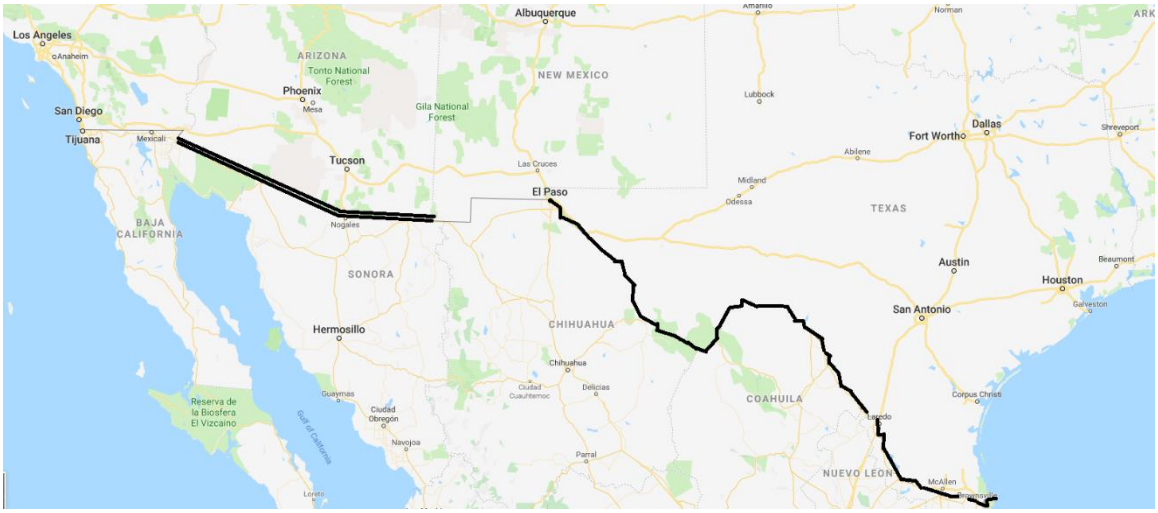

Figure 5. Map of the United State - Mexico border, with the Arizona-Mexico portion of the border bolded in double black lines and the Texas-Mexico portion of the border marked by a single bolded black line. Adapted by the author from Google Maps.

Previous studies indicate that while unidentified migrant decedents recovered in Arizona are increasingly from the “Northern Triangle Countries” of Guatemala, El Salvador, and Honduras, these migrants remain primarily from Central and South Mexico and reflect a displaced rural demographic that is seeking to cross the border for financial reasons (Figure 6) (Martinez et al. 2014). Detained migrants in Arizona have also conveyed to researchers a need to cross the border to leave a region strife to “political instability, abject poverty, gangs, and drug trafficking" (Hagan 2008; Martinez et al. 2014). In comparison, migrants along the Texas-Mexico border are increasingly from the “Northern Triangle Countries” and consist of family units; a significant number of which are primarily fleeing political violence and impoverished and marginalized communities (Keller et al. 2017). It is not unusual for migrants recovered by OpID along the Texas - Mexico border to have had dental restorations or other medical procedures completed ante mortem, indicating access to medical care. Understanding differences in life histories, and how these lives are physically embodied, could potentially assist in

individualizing unidentified migrant decedents beyond the term “Hispanic” and improve the forensic identification process.

Figure 6. Map of Central America with a focus on the Northern Triangle Countries (Guatemala, Honduras, and El Salvador). Adapted by the author from Google Maps.

Further considerations. There are several assumptions and considerations regarding deceased individuals recovered along the United States – Mexico border that are presumed to be undocumented border crossers (UBCs – a term and abbreviation used by the PCOME) or unidentified migrants that require further attention in this research. Firstly, there is not a standardized way in which individuals cross the border, since who crosses (e.g., parent, first or second son, first or second daughter, etc.) depends on familial and social circumstances. Additionally, the nature of crossing the border itself suggests that these individuals are subjected to other forms for structural violence, including violence at home that is causing them to flee and violence in the journey to cross the border.

While sociological and psychological interviews of detained migrants have suggested generally why migrants are fleeing to the United States, as mentioned previously, individual agency should not be taken from unidentified decedents and it should not be assumed that these reasons influence everyone equally. Additionally, most individuals marked as UBCs or unidentified migrants are assumed to be individuals of Hispanic ancestry who are fleeing Mexico and Latin and South America. However, this is not always true. Cases have been reported of South and Eastern Asians, and of deported DREAMers being recovered along the border as well (personal communication Dr. Jen Vollner, 2018). Only with positive identification will the origin and demographic information for these individuals be known.

This research will provide quantifiable data about how disparities associated with the migrant experience impact human skeletal biology. From an ethical and legal perspective, this research also holds potential to provide skeletal evidence to support and verify the dangerous circumstances and environments from in which people migrate and attempt to improve their lives. Chapter II (Theory) will further explore the conceptual frameworks that will be employed to interpret this data.

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II. THEORY

Biocultural Theory

Human bodies are not isolated systems, but rather, they interact with the biocultural environment around them over the course of their lifetime. While genetic material is the groundwork for biological life, various aspects of the environment contribute to its expression. Employing a biocultural approach that applies both biological and cultural theories in research on human skeletal biology allows researchers to expand interpretations beyond bone itself.

Biological anthropologists studying skeletal indicators of stress were historically more concerned with classification of the ailment itself rather than the context that contribute to its existence, and consequently, they focused on describing the prevalence of skeletal pathologies rather than holistically assessing what they indicated regarding overall health status (Larsen 2002). With S. L. Washburn's call for The New Physical Anthropology came an increased focus on the processes, e.g., evolutionary theory and genetics, that influence human variation (Fuentes 2010). Application of these biological theories, in conjunction with further cultural and medical anthropological theory, results in the biocultural approach and helps to interpret humans as both biological organisms and social beings.

Implementation of the biocultural approach in studies on stress and disease is particularly beneficial in assessing the interrelatedness of pathologies and environmental spheres of influence; e.g., sociopolitical, cultural, economic, ecological, and biological (Zuckerman 2012; Roberts and Machester 1999). Assessing interpopulational pathological patterns can thus assist in interpreting various levels of (mal)adaptiveness to

various biosocial environments (Zuckerman 2012). Additionally, this approach allows for consideration of potential influences that exacerbate skeletal pathologies. Ultimately, incorporation of biocultural theory into research on skeletal health and disease allows for a more complete interpretation of the biological data within its biosocial context holding implications for future research in both bioarchaeology, when reconstructing epidemiological models of past populations, and in biological anthropology, when predicting health trajectories of modern populations (Armelagos 2003; Buikstra and Beck 2009; Agarwal and Glencross 2011; Martin et al. 2014; Agarwal 2016).

The biocultural approach emphasizes consideration of biological data within a social context, which necessitates incorporation of relevant cultural processes of both broader structural influences (e.g., structural violence, sociopolitical violence, gendered experiences, et cetera) with documented demographic data (e.g., self-reported socioeconomic status, occupation type, sex, gender, et cetera.) into research. The goal of employing a biocultural approach in this research is to dive more deeply into the interrelatedness, and intersectionality, of the biocultural environment in influencing skeletal biology and understanding how skeletal markers of stress can be used as proxies for social context. Applying this approach to stress indicators in a modern, documented population holds implications for future bioarchaeological and forensic research.

This chapter will further discuss both the biological and cultural components of biocultural theory and the frameworks from which to consider both individual disease episodes and more anthropologically oriented, longitudinal, population-level patterns.

Embodiment Theory

As both biological organisms and social beings, the human body is contextually dependent on the "local biologies" (Lock 2015, p.151) that influence individual development over the lifetime (Ingold 1998; Lock 1993; Niewohner 2011; Palsson 2013; Agarwal 2016). The concept of embodiment encompasses the idea that our bodies at any moment reflect the sum of experiences over the lifetime (Agarwal 2016). While employed differently in various subfields of anthropology, i.e., cultural anthropology, for the purpose of this research in biological anthropology, embodiment is broadly defined as skeletal changes that occur as a result of the interactions between our bodies and the surrounding environment.

It is important to note that as skeletal changes occur, they do not do so in isolation. Surrounding biological structures, including soft and hard tissues, and future growth trajectories of the body are also altered with each change. Therefore, due to differences in cumulative life events, differences in skeletal embodiments of stress can potentially indicate inequality in embodiment that is reflective of lived disparities. To better understand inequalities in embodiment, skeletal markers of stress need to be situated into a biocultural context that uses a multifactorial approach to consider both potential biological and social influences (Figure 7).

This multilevel approach to embodiment considers life course (social influences) and developmental (biological influences) perspectives together to explore how experiences contribute to the phenotype over time and result in inter and intrapopulation level skeletal variation (Figure 4). More specifically, Life Course Theory and the Developmental Origins of Health and Disease (DOHaD) hypothesis are theoretical

frameworks situated under the concept of embodiment that will be utilized in this research. These are to be discussed below.

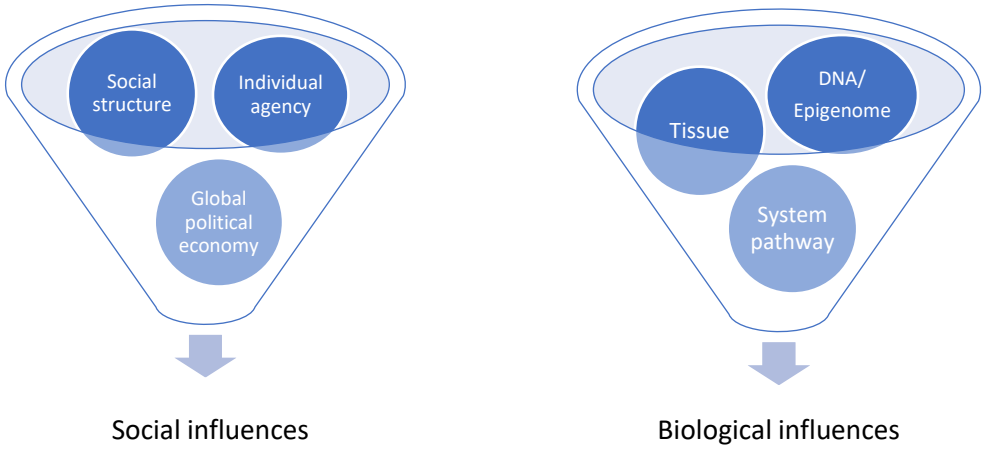

Figure 7. Multilevel social and biological influences that impact development plasticity and the subsequent phenotype.

While embodiment theory has been an underlying assumption in previous research using skeletal indicators of stress to assess health profiles, published research has not been found that explicitly states this concept in relation to VNC asymmetry and SNs. Use of the TXSTDSC sample allows this research to directly assess the connection between individual (self-reported) life histories and life courses among individuals in the TXSTDSC and their osteobiographies, and to explore population level patterns in samples of different biosocial origins.

Life Course Theory

From the biocultural perspective, the human body at any point in time is the sum of previous biosocial experiences which can be reflected in both the soft and hard tissues of the body. Life Course Theory takes this approach in assessing human skeletal biology as the product of a combination of influences on phenotypic variation (as discussed under

Embodiment Theory). Similarly, to applications of Embodiment Theory, Life Course Theory can undergo different approaches of assessment in different subfields. In terms of cultural anthropological and sociological research, an emphasis is placed on individual agency within social pathways and understanding how this exerts forces on the biological body (Agarwal 2016). In biological anthropology, an emphasis is placed on the life stages of one's life history and how they impact changes in the body.

These differences in approaches and their applications can be easily muddled and confusing. For the purpose of this research, while life course refers to the sociocultural stages of one's life, e.g., weaning age, working age, etc., life history refers to the biological stages of the life cycle, e.g., puberty, maturation, etc. In this research, the life course approach will be used to further elucidate the connection between both the individual's life history and life course as situated in document, historical contexts.

Consideration of both sociocultural and biological stages is significant, because as will be discussed under the DOHaD hypothesis, there is a connection between early life events on later life health and disease outcomes. Research supports the concept that individuals who exhibited stress-induced growth disruptions, whether biological or social in origin, then had an increased susceptibility to biological insults later in life (Mazumder et al. 2010). Therefore, adults experiencing similar events may embody it differently depending on their childhood experiences (Rothman and Greenland 1998, in Zuckerman 2012; Agarwal 2016). While individuals are the result of interrelated and cumulative events, this also affects the community level over generations and is hypothesized to be viewable in the skeletal morphology at the population level (Agarwal 2016). Application

of Life Course Theory to interpretations from this thesis will allow for a longitudinal perspective on developmental plasticity's effect on adulthood susceptibility to pathology.

Developmental Origins of Health and Disease (DOHaD) Hypothesis

The DOHaD hypothesis is a theoretical framework that focuses on the role of developmental stress in the fetal and childhood periods and its effect on subsequent developmental trajectories and adult morbidity (Gluckman et al. 2016; Gowland 2015). As defined before, stress can constitute any adverse circumstance in the biocultural environment including malnutrition, physical and psychological trauma, disease, or any combination thereof that effects the fetus or child's homeostasis. Studies into epigenetics have found relationships between stressors and future health trajectories as environmental alterations to DNA that prevent the proper replication of genetic material subsequently alter the associated phenotype (Gowland 2015). The central tenants to DOHaD, factors that need to be considered when interpreting results, include the biosocial context and timing of the stress events (Temple 2018).

DOHaD has further been linked to non-communicable diseases and disorders that affect the soft tissues without leaving traces on human skeletal remains (Gillman et al. 2007; Temple 2018). However, skeletal indicators of stress, including VNC asymmetry, that are associated with childhood stress can serve as proxies for investigating DOHaD and considering their implications on the remaining life course and susceptibility to stress indicators in adulthood.

Vertebral Neural Canal (VNC) Asymmetry and Schmorl's Nodes. When employing the DOHaD framework to interpret the significance of early and later childhood health episodes on adulthood longevity, Watts (2015) found that while stress episodes

experienced in early childhood (as seen in the AP widths) did not negatively affect morbidity or later childhood health, later episodes (as seen in the TR widths) more detrimentally affected adulthood health. As these later stress episodes were significantly correlated with increasingly early mortality, it can be interpreted that these later episodes more significantly impacted future energy usage and predisposed individuals to increased susceptibility of pathologies as adults (Watts 2015).

Later stress episodes are indicative of chronic stressors, in contrast to malnutrition or reoccurring infection in younger children, as the adolescents' immune systems have reached maturation and are more robust (Selye 1978; Sapolsky 1992; Flinn 2006; Webster Marketon and Glaser 2008; Vercellotti et al. 2011; Ulijaszek 1998; Goodman and Armelagos 1989; Watts 2015). Because chronic infection is required to alter the TR widths of older children, this longer exposure to illness increases inflammatory responses and the negative effects it has across the body, and more detrimentally affects energy expenditures for future use (Watts 2015). Additionally, Watts's (2015) study in post-medieval London found an association between smaller VNC dimensions and lower social status. These findings further support that sociocultural influences such as socioeconomic and other forms of structural violence impact the phenotype.

No published research has been found that examines the occurrence of SN beyond presence/absence and considers the theoretical frameworks discussed in this chapter.

These theories have much overlap, so it is difficult to use them in isolation. However, as each takes into account a slightly different perspective, consideration of all of them allows for a more holistic interpretation into how differences in development impact how life history manifests on the skeleton. Employing this approach in this

research to assess both VNC asymmetry and SN will allow for more strengthened interpretative power into the interconnectedness of developmental plasticity and adult susceptibility to illness.

Structural Violence

Beyond the biological frameworks of interpretations, understanding the cultural context and its role in influencing the body is necessary to employing the biocultural approach. Structural violence is a term used to describe the process of sociocultural structures causing harm to individual, group, and population-level bodies within a particular social context (Farmer 2004; Farmer et al. 2006). When viewed from an intersectional perspective, there are various levels of violence that can affect the subject (e.g., low sociocultural status and malnutrition, gendered violence, sociopolitical violence, age, migration, and cause of death). Structural violence, however, can be so embedded into a culture's societal norms that the systems which bring harm to particular identities seem invisible. While symbolic, these systems of violence have biological repercussions and can be embodied in the human body, particularly in the skeleton.

In Mexico, not only are various regions strife with poverty and malnutrition, but conceptions of gender norms have led to further levels of interpersonal violence. An analysis into male homicide patterns found that areas of extreme inequality experienced increased violence (Gamlin and Hawkes 2018). In a culture with a strong machismo focus, sociocultural processes which significantly impacted a man's capabilities of providing for their families and exercising their masculinity led to increased acts of violence in attempts to regain respect and social status (Gamlin and Hawkes 2018). Migration of Mexican individuals across the United States – Mexico border commonly

consists of these young males who are attempting to find work and improve their financial outcomes. Presumed migrants found deceased along the border are also individuals who likely faced additional levels of structural violence, including the lack of the time and money necessary to cross legally.

These patterns of structural violence are also paralleled in the Norther Triangle Countries where high levels of poverty, sociopolitical violence, and gang violence have been found to be reasons for migrants to flee (Martinez et al. 2014; Lakhani 2016; Carney 2015). Whereas Mexican nationals crossing the border primarily consist of single males, nationals from Latin and South America are increasingly consisting of family units that consist of various ages and genders (Martinez et al. 2014; Lakhani 2016; Carney 2015). By considering the different biocultural contexts of these two areas, and how violence and stress across the life course is embodied, this thesis will address how various levels of structural violence inflict stress among unidentified migrants recovered along the United States – Mexico border and in turn, help to quantify these different experiences.

Syndemic Theory

As discussed previously, the biocultural approach can be challenging to implement when attempting to assess the effects of culturally defined variables on human biology, when they are composed of multiple, intersecting variables (Dufour 2006; Zuckerman 2012). While traditional biomedical approaches to studying and interpreting disease have treated them as distinct and independent entities, the Syndemic theoretical framework, formally outlined by medical anthropologist Merrill Singer in the early 2000s, serves as a holistic approach that emphasizes the interrelatedness of influences and contexts (Singer et al. 2006; Carney 2015). Syndemic theory is based upon three

basic tenants: 1) both infectious and non-communicable diseases can coexist in populations, increasingly so among systematically marginalized groups; 2) biological synergism should be considered in order to understand the interactions of various biological diseases; and 3) that interpretations of health and disease need to consider potential influences that together create the "social context" (Carney 2015; Claire 2003; Singer 2006)

Consideration of the interactions of the variables discussed throughout Chapters I and II provides increased interpretive power over the data and can elucidate not just the potential interrelatedness of various environments, but how these variables can compound negative health effects (Carney 2015). Following this theoretical guidance, it is predicted that increasingly marginalized groups, i.e., migrants crossing the US-Mexico border in comparison to American citizens, experience various influences of stress that compound and put them at a higher risk for disease and pathology. Not only are there the various macro-level layers of complexity within the social context to consider for this research (e.g., class and gang violence), but there are further microscale influences at the individual level that may together increase the severity of pathologies (e.g. gender).

III. MATERIALS AND METHODS

Samples

The human skeletal remains assessed in this research were comprised of individuals from three samples; willed body donations in the Texas State Donated Skeletal Collection (TXSTDSC); unidentified migrant forensic cases curated at the Forensic Anthropology Center at Texas State (FACTS) under the Operation Identification (OpID) program; and unidentified migrant forensic cases curated at the Pima County Office of the Medical Examiner (PCOME) in Tucson, Arizona. After application of sampling parameters, a total of 76 individuals from the TXSTDSC, 104 individuals from the OpID project, and 32 individuals from the PCOME were included in this assessment.

Texas State Donated Skeletal Collection. Since its origination in 2008, the TXSTDSC has received individuals who donated their bodies or were donated by their legal next-of-kin into the willed body donation program at the Forensic Anthropology Center at Texas State (FACTS) University. Individuals, or their next of kin, complete a questionnaire and submit demographic information. The use of donated bodies and their self-reported information in the TXSTDSC is covered by the Texas Anatomical Gift Act.

Willed body donations are first brought to the outdoor human decomposition facility called the Forensic Anthropology Research Facility (FARF) where they participate in research on decomposition, the post-mortem interval, and other forensic science. After decomposition of soft tissues, skeletal remains are processed and labeled at the Osteology Research and Processing Laboratory (ORPL). Individual donations are then curated and available for osteological studies in the Grady Early Forensic Anthropology Laboratory (GEFARL). Of the 590+ willed body donations in the

TXSTDSC at the time of this study, 384 were processed and available at GEFARL. Of those, 76 were ultimately included in the observable sample for this research (Table 3).

Operation Identification. The Operation Identification (OpID) program, directed by Dr. Kate Spradley, has been based out of FACTS since 2013 and serves to facilitate the recovery, identification, and repatriation of unidentified human remains found near the Texas - Mexico border. Due to an amalgamation of factors, including not following protocols and/or improperly carrying out investigations, underfunding, and the high number of deaths, many deceased migrants remain unidentified in South Texas (Spradley 2014). Through exhumation of unidentified individuals from unmarked graves in Texas border counties and occasional acquisition from collaborators, (e.g., the Forensic Border Coalition and the University of Indianapolis) as of 2019 OpID has worked with the remains of 270 individuals who were curated at ORPL pending identification, while identification and repatriation efforts took place. After application of sampling parameters, 105 OpID individuals were included in this research (Table 4).

Pima County Office of the Medical Examiner Sample. The Pima County Office of the Medical Examiner (PCOME) is situated in Tucson, Arizona and serves as the primary medical examiner's office for southern Arizona. As such, it conducts forensic anthropological assessments on unidentified skeletal individuals, including those presumed to have died while migrating across the Arizona - Mexico border. Unidentified migrants (or Unidentified Border Crossers – UBCs – in PCOME terminology) recovered in the Sonoran Desert of Southern Arizona are commonly found with missing skeletal elements or in poor preservation due to the desert environment and taphonomic conditions. These found bodies also typically enter the PCOME in various states of

decomposition. Many cases are either completely mummified individuals or consist of isolated skeletonized elements. Mummified and partially decomposed individuals require further maceration and processing before data can be collected.

Individuals that remain unidentified after several months with no leads towards identification are released to be cremated. As a result, the PCOME sample size used in this research was dictated on the availability of skeletal remains that fit the criteria for analysis, and the time it took to process the remains, during the timeframe of data collection. To increase the sample size, data collection at the PCOME occurred in two visits - one in the summer of 2018 for three weeks and again the following winter of 2018 for one week.

Every individual that was included in this study from the PCOME exhibited some degree of desiccation and required further maceration. The author fully processed every case (except for one – thank you, Dr. Jen Vollner and undergraduate intern Liz Young) and extracted the vertebral column to assess its suitability for inclusion in this study. Overall, 32 unidentified individuals at the PCOME were assessed and included in this research (Table 5).

Sampling Procedure

To be included in this study, sampled individuals needed to have, at minimum, half of their identifiable thoracic and lumbar vertebrae present and in good enough preservation to allow the scoring of the vertebral neural canals (VNC) and/or vertebral corpus surfaces. Individuals with pathologies and/or medical interventions, e.g. diffuse idiopathic skeletal hyperostosis, spinal fusion surgery, et cetera, in which the VNC and/or vertebral surfaces were unobservable or modified were excluded. All individuals that met

these criteria at the PCOME or OpID were included in this study. Due to a limited timeframe for data collection, not all individuals in the TXSTDSC were assessed for sampling. Individuals in the TXSTDSC with completely intact spinal columns (T1-L5) were prioritized before those with partially intact or partially fused columns. To increase sampling efficiency and efficacy in the TXSTDSC, an effort was made to include a wide variety of individuals, including those of different ancestries, ages, sex, occupation types and SES backgrounds. However, the TXSTDSC sample is biased towards older, white male individuals and as such, an equal representation of each category could not be met. Under these selection parameters, the total observable sample size for this thesis was 212 individuals; 76 individuals from the TXSTDSC, 104 individuals from OpID, and 32 individuals from the PCOME. In total, the author collected 71,687 points of data for this thesis.

Measures and Covariates

Demographic data. The demographic data collected for individuals assessed from the TXSTDSC included: age at death, sex, stature, ancestry, occupation, and childhood socioeconomic status. This information is reported by the individual donor themselves prior to death, or by the next of kin upon the donation of their body, using a standardized donation questionnaire that includes information on medical health and life histories. Ancestry and occupation are self-described. Childhood socioeconomic status was selected from provided levels, ranging from low, low-middle, middle, middle-high, and high. It should be emphasized that both occupation and socioeconomic status for the individuals from the TXSTDSC are self-reported data, as indicated on their personal donation forms, which does not prompt donors with definitions nor scales of

measurement. Primary occupation is typically noted by individuals, as they are not required to detail all occupations. As such, it was not always possible to distinguish which of the four categories from Villotte et al. (2010) classification would be most appropriate. Therefore, these were grouped together as either non-manual worker (Group A) or manual worker (Groups B, C, D). Socioeconomic status is categorized on the forms as childhood status, which is subjective and not verifiable. The demographic data for individuals from the PCOME and OpID samples were collected from reconstructed biological profiles and included estimations of age at death, sex, stature, and ancestry. These biological profiles were completed by forensic anthropologists and skeletal analysts at the PCOME and OpID.

Demographic information has been abbreviated in Tables 3-5 as follows – Sex: Female (F), Male (M), not available (-); Ancestry: Black (B), Hispanic (H), Native American (NA), White (W), Guatemalan (GTM), unknown (Unk), not available (-).

Morphometric Data. The same morphometric data was collected for all individuals assessed in this research when preservation permitted (Figure 8). Data recorded for each thoracic and lumbar vertebra included the vertebral neural canal (VNC) antero-posterior (AP) and transverse (TR) diameters, the AP and TR diameters of both the superior and inferior vertebral surfaces, and the presence/absence of Schmorl's nodes (SNs). These measurements are shown in Figures 9 - 14. The AP and TR diameters were measured in millimeters (mm) using sliding calipers according to Watts' (2011) method, in which AP diameter is the "posterior portion of the vertebral body to the further opposite point of the neural canal, anterior to the spinous process," and the TR diameter is the maximum distance "between the medial surface of the left and right pedicles," (Watts 2011).

For each SN, the following features were recorded: presence/absence, surface (INF: inferior, SUP: superior), aspect (A: anterior, C: central, P: posterior), maximum depth (Figure 11), AP and TR diameters (Figures 15 – 16). When nodes extended across the vertebral surface, all aspects affected were recorded, e.g., AC, ACP, et cetera. Maximum depth of each node was measured using a periodontal probe and all other measurements were collected using a digital sliding caliper. If multiple SNs were found on a single vertebra, they were recorded separately. These measurements were used to calculate the severity of each node, using the Knüsel et al. (1997) classification method (described further below).

Table 3. TXSTDSC: Demographic Information.

	ID #	Sex	Age	Ancestry	Childhood SES	Occupation	Occupation Type
1	D02-2009	M	91	W	Upper Middle	Mechanic	Manual
2	D05-2009	M	61	W	Upper Middle	Field Engineer, Telephone	Manual
3	D08-2010	M	67	H	Lower	Steelworker	Manual
4	D10-2010	F	32	W	Upper Middle	Administrat-or	Non-manual
5	D12-2010	M	54	W	Lower Middle	Handyman	Manual
6	D01-2011	M	40	W	Lower	Unspecified	-
7	D04-2011	F	68	W	Middle	ER Nurse	Manual
8	D15-2011	M	49	W	Lower	Grocery	Manual
9	D19-2011	M	56	W	Lower Middle	Carpentry	Manual
10	D23-2011	F	66	W	Lower Middle	Jailer, Dispatcher	Non-manual
11	D06-2012	M	58	W	Middle	Construction, Plumber	Manual
12	D12-2012	F	64	W	Lower	Home Maker	Non-manual
13	D14-2012	M	85	H	Lower	Truck Driver, Freight	Manual
14	D21-2012	M	42	W	Upper Middle	Real Estate	Non-manual
15	D25-2012	F	44	W	Upper Middle	computer technician	Non-manual
16	D28-2012	M	75	W	Lower Middle	Uniform Rental/self-employed	Non-manual
17	D32-2012	F	47	W	Lower	healthcare	Non-manual
18	D36-2012	F	42	W	Lower Middle	Executive Assistant	Non-manual
19	D38-2012	M	50	W	Lower Middle	Medical Mechanic	Non-manual
20	D39-2012	M	57	W	Middle	Architect	Non-manual
21	D40-2012	F	67	H	Lower	Care Taker	Non-manual
22	D41-2012	M	60	W	Lower Middle	Warehouse work	Manual
23	D45-2012	M	65	W	Lower Middle	Auto Mechanic	Manual
24	D03-2013	F	89	W	Upper Middle	Housewife	Non-manual
25	D08-2013	F	68	W	Lower	Data entry, nurse assistant	Non-manual

Table 3. Continued. TXSTDSC: Demographic Information.

	ID #	Sex	Age	Ancestry	Childhood SES	Occupation	Occupation Type
26	D15-2013	F	55	W	Upper Middle	Office administrat-or	Non-manual
27	D16-2013	M	53	W	Middle	Architect	Non-manual
28	D25-2013	M	62	W	Upper Middle	Insurance Examiner	Non-manual
31	D31-2013	M	64	W	Upper Middle	Truck driver, Food delivery	Manual
32	D32-2013	F	87	W	Upper Middle	Advertising copywriter, housewife	Non-manual
34	D42-2013	F	74	W	Lower Middle	Electronics Test Technician	Non-manual
35	D53-2013	M	65	W	Lower	Constructio n	Manual
36	D55-2013	M	57	W	Lower Middle	Laborer, Tree service	Manual
37	D57-2013	M	54	W	Lower	Restaurant service, auto repair, home repair and maintenance	Manual
38	D03-2014	F	64	W	Upper Middle	Landscape artist	Non-manual
39	D08-2014	M	57	B	Upper middle	Manager, retail	Non-manual
40	D11-2014	F	46	W	Lower Middle	Sale Associate	Non-manual
41	D13-2014	M	29	B	Lower	None	-
42	D16-2014	F	59	W	Lower Middle	Customer Service	Non-manual
43	D19-2014	F	77	W	Lower Middle	Housewife	Non-manual
44	D21-2014	F	23	W	Upper Middle	Student	Non-manual
45	D27-2014	M	56	W	Lower	Usually unemployed	Non-manual
46	D38-2014	M	79	W	Lower	Automobile dealer	Non-manual
47	D56-2014	F	69	W	Lower	Real Estate	Non-manual
48	D65-2014	M	43	W	Upper Middle	Constructio n	Manual
49	D06-2015	M	93	W	Lower Middle	UNK	-

Table 3. Continued. TXSTDSC: Demographic Information.

	ID #	Sex	Age	Ancestry	Childhood SES	Occupation	Occupation Type
50	D08-2015	M	51	W	Lower Middle	Constructi-on	Manual
51	D10-2015	M	66	W	Lower Middle	Computer Program	Non-manual
52	D12-2015	F	40	W	Middle	UNK	-
53	D14-2015	M	70	W	Middle	Computer Programmer	Non-manual
57	D23-2015	M	69	W	Lower	Teacher	Non-manual
58	D24-2015	F	67	W	Lower Middle	Registered Nurse	Non-manual
59	D25-2015	F	68	W	Lower Middle	Teacher	Non-manual
60	D26-2015	F	21	W	Middle	CNA, MA, Phlebotomis t	Non-manual
61	D28-2015	F	76	W	Lower	housewife	Non-manual
62	D30-2015	M	86	W	Middle	Developme-nt	Non-manual
63	D31-2015	F	55	W	Lower middle	homicide investigator/ prosecutor	Non-manual
64	D35-2015	F	69	W	Middle	UNK	-
65	D37-2015	F	55	W	Middle	Sales	Non-manual
66	D38-2015	F	77	W	Lower	Cashier	Non-manual
67	D39-2015	M	85	W	Lower middle	Air Force	Manual
68	D41-2015	F	57	W	Middle	secretary	Non-manual
69	D52-2015	M	22	W	Middle	UNK	-
70	D60-2015	F	49	W	Middle	Teacher	Non-manual
71	D66-2015	F	56	W	Lower	Homemaker	Non-manual
72	D68-2015	M	62	W	Upper Middle	Financial Officer	Non-manual
73	D02-2016	M	73	W	Lower	Reporter, Analyst, Film Reviewer	Non-manual
74	D02-2016	M	73	W	Lower	Reporter, Analyst, Film Reviewer	Non-manual
75	D16-2016	M	71	W	Lower	Finance	Manual
76	D61-2016	F	74	W	Lower	-	-

Table 4. Operation Identification: Demographic Information.

	Catkey	Sex	Estimated Age	Estimated Ancestry
1	OpID-0362	M	34-66	-
2	OpID-0363	M	23-46	-
3	OpID-0365	F	17-30	-
4	OpID-0367	M	16-25	-
5	OpID-0368	M	27-69	-
6	OpID-0372	M	25-40	-
7	OpID-0377	F	30-50	Unk
8	OpID-0378	F	34-67	H
9	OpID-0379	F	25-40	H
10	OpID-0381	M	28.1-48.1	Unk
11	OpID-0384	F	30-55	H
12	OpID-0388	M	50-66	-
13	OpID-0389	M	30-50	H
14	OpID-0390	M	18-22	H
15	OpID-0391-A	M	25-57	H
16	OpID-0392	M	16-25	H
17	OpID-0393	M	18-30	H
18	OpID-0395	M	17-25	H
19	OpID-0397	M	32-50	Unk
20	OpID-0398	M	35-50	H
21	OpID-0399	M	25-57	H
22	OpID-0401-C	F	15-21	H
23	OpID-0404	F	-	H
24	OpID-0406	F	23-30	-
25	OpID-0408	M	35-56	H
26	OpID-0409	M	23-36	Unk
27	OpID-0470	M	32.5-61.2	H
28	OpID-0411	F	-	H
29	OpID-0412	F	25-60	H
30	OpID-0414	F	25-50	H
31	OpID-0415	M	24-36	-
32	OpID-0416	F	26-50	H
33	OpID-0417	F	35-50	H
34	OpID-0418	M	28-56	-
35	OpID-0419	M	34-56	-
36	OpID-0421	M	20-29	Unk
37	OpID-0422	M	18-26	-
38	OpID-0422	M	18-26	-
39	OpID-0423	M	29-54	-

Table 4. Continued. Operation Identification: Demographic Information.

	Catkey	Sex	Estimated Age	Estimated Ancestry
40	OpID-0426	M	28-42	H
41	OpID-0427	M	15-23	H
42	OpID-0429	F	30-58	H
43	OpID-0437	M	45+	H
44	OpID-0446	F	25-42	H
45	OpID-0448	F	17-26	H
46	OpID-0451	M	16-20	-
47	OpID-0455	F	-	H
48	OpID-0462	M	-	-
49	OpID-0464	F	21-32	H
50	OpID-0465	F	-	H
51	OpID-0467	M	29.3-50.5	GTM
52	OpID-0468	Unk	18-Dec	Unk
53	OpID-0469	M	18-36	-
54	OpID-0471	M	23-46	H
55	OpID-0473	F	28-54	H
56	OpID-0475	F	15-22	H
57	OpID-0476	M	26-42	B
58	OpID-0482	M	15-25	H
59	OpID-0486	M	24-37	H
60	OpID-0487	F	15-20	H
61	OpID-0488	M	29-50	W
62	OpID-0490	F	16-25	H
63	OpID-0491	M	28-45	Unk
64	OpID-0492	M	30-57	W
65	OpID-0495	F	21-33	Unk
66	OpID-0500	M	28-49	H
67	OpID-0503	M	20-45	H
68	OpID-0504	M	20-40	H
69	OpID-0505	F	35-69	H
70	OpID-0506	F	24-58	H
71	OpID-0508	F	15-22	H
72	OpID-0511	M	25-45	H
73	OpID-0513	M	-	H
74	OpID-0514	F	31-57	B
75	OpID-0517	M	-	-
76	OpID-0520	M	-	-
77	OpID-0522	M	-	H
78	OpID-0528	M	-	H
79	OpID-0531	M	-	H

Table 4. Continued. Operation Identification: Demographic Information.

	Catkey	Sex	Estimated Age	Estimated Ancestry
80	OpID-0535	M	-	H
81	OpID-0606	M	29-56	Unk
82	OpID-0609	M	14-17	H
83	OpID-0611	M	15-25	H
84	OpID-0612	F	25-45	H
85	OpID-0615	M	20-31	H
86	OpID-0617	M	15-26	H
87	OpID-0619	M	34-72	W
88	OpID-0623	M	31-60	H
89	OpID-0627	M	27-46	H
90	OpID-0629	M	27-45	Unk
91	OpID-0630	M	42-82	H
92	OpID-0635	-	-	-
93	OpID-0637	M	14-19	H
94	OpID-0638	F	30-61	H
95	OpID-0639	-	-	-
96	OpID-0640	-	-	-
97	OpID-0642	M	30-60	H
98	OpID-0643	M	25-42	H
99	OpID-0644	-	-	-
100	OpID-0645	M	17-27	H
101	OpID-0647	-	-	-
102	OpID-0652	M	25-39	H
103	OpID-0655	-	-	-
104	OpID-0667	M	23-36	H

Table 5. Pima County Office of the Medical Examiner: Demographic Information.

	Individual	Sex	Estimated Age	Age Range	Estimated Ancestry
1	17-1742	M	Adult-Pre 30	18-22	W
2	17-2006	M	Adult-Pre 60	25-55	ANA
3	17-2290	M	Adult-Pre 50	30-45	ANA
4	17-2885	M	Adult-Pre 50	25-45	Likely ANA
5	17-3171	M	Adult-Pre 50	23-50	Likely ANA
6	18-0200	M	Adult-Pre 40	25-35	ANA
7	18-0656	M	Adult-Pre 60	25-55	ANA
8	18-0698	M	Adult-Pre 30	18-25	Likely ANA
9	18-0783	M	Adult-Pre 70	35-60	ANA
10	18-0814	M	Adult-Pre 80	40-70	W
11	18-0960	M	Adult-Pre 50	30-45	ANA
12	18-0968	M	Adult-Pre 50	25-45	Likely ANA
13	18-1003	M	Adult-Pre 70	30-65	Likely ANA
14	18-1032	Unk	Adult-Pre 40	18-30	Unk
15	18-1117	M	Adult-Pre 70	35-65	ANA
16	18-1251	M	Adolescent	13-16	ANA
17	18-1467	M	Adult-Pre 50	25-50	Likely ANA
18	18-1494	M	Adult-Pre 40	28-38	ANA
19	18-1558	M	Adult-Pre 60	25-50	ANA
20	18-1722	F	Adult-Pre 30	17-24	ANA
21	18-2046	M	Adult-Pre 20	15-19	ANA
22	18-2004	F	Adult-Pre 40	20-40	ANA
23	18-2213	M	Adult-Pre 50	30-50	Unk
24	18-2409	M	Adult-Pre 60	30-60	Unk
25	18-2626	M	Adult-Pre 70	30-65	Likely ANA
26	18-1923	M	Adult-Pre 50	29-45	ANA
27	18-2531	M	Adult-Pre 50	24-40	Likely ANA
28	18-2618	-	-	-	-
29	18-2669	M	Adult-Pre 50	28-42	ANA/W
30	18-2761	M	Adult-Pre 60	30-60	ANA
31	18-2985	-	-	-	-
32	18-3142	M	-	-	Unk

Sample	ID	Vert	VNC AP (mm)	VNC TR (mm)	SN?	Surface	Aspect	Depth (mm)	SN AP (mm)	SN TR (mm)	Superior Surface AP (mm)	Superior Surface TR (mm)	Inferior Surface AP (mm)	Inferior Surface TR (mm)
TXSTDSC	D66-2015	T1	21.65	17.80	N	-	-	-	-	-	-	-	-	-
TXSTDSC	D66-2015	T2	17.60	19.00	N	-	-	-	-	-	15.50	25.20	17.65	27.90
TXSTDSC	D66-2015	T3	16.35	19.10	N	-	-	-	-	-	18.00	24.34	20.41	23.46
TXSTDSC	D66-2015	T4	16.19	19.60	N	-	-	-	-	-	21.26	23.11	23.81	23.20
TXSTDSC	D66-2015	T5	16.18	19.90	Y	INF	CP	3.00	14.00	4.60	23.20	23.82	23.87	27.37
TXSTDSC	D66-2015	T6	16.50	20.21	Y	INF	CP	3.00	14.60	7.40	25.83	25.95	28.44	30.00
TXSTDSC	D66-2015	T7	16.85	20.57	Y	SUP	C	3.00	10.60	4.60	27.60	28.00	30.90	31.15
TXSTDSC	D66-2015	T7	16.85	20.57	Y	INF	C	2.50	13.30	20.80	27.60	28.00	30.90	31.15
TXSTDSC	D66-2015	T8	17.30	20.80	Y	SUP	C	2.50	6.50	14.70	30.25	31.43	31.20	33.00
TXSTDSC	D66-2015	T8	17.30	20.80	Y	INF	C	3.00	15.40	18.20	30.25	31.43	31.20	33.00
TXSTDSC	D66-2015	T9	17.60	21.40	Y	SUP	C	3.00	11.60	8.30	31.10	31.39	31.30	34.50
TXSTDSC	D66-2015	T9	17.60	21.40	Y	INF	C	1.00	10.50	12.50	31.10	31.39	31.30	34.50
TXSTDSC	D66-2015	T10	17.90	21.85	Y	SUP	C	1.00	10.00	3.70	30.50	33.88	31.90	37.45
TXSTDSC	D66-2015	T10	17.90	21.85	Y	INF	CP	3.00	12.00	18.20	30.50	33.88	31.90	37.45
TXSTDSC	D66-2015	T11	18.50	24.50	Y	SUP	CP	2.50	13.60	9.80	31.15	36.56	29.37	39.60
TXSTDSC	D66-2015	T11	18.50	24.50	Y	INF	C	2.50	10.50	19.80	31.15	36.56	29.37	39.60
TXSTDSC	D66-2015	T12	20.55	26.00	Y	SUP	C	3.00	9.60	20.60	29.80	39.75	29.70	38.20
TXSTDSC	D66-2015	T12	20.55	26.00	Y	INF	C	2.50	10.00	18.60	29.80	39.75	29.70	38.20
TXSTDSC	D66-2015	L1	21.80	27.80	Y	SUP	C	1.50	9.50	20.00	32.35	39.60	32.63	44.10
TXSTDSC	D66-2015	L1	21.80	27.80	Y	INF	C	2.00	14.90	18.10	32.35	39.60	32.63	44.10
TXSTDSC	D66-2015	L2	21.75	28.34	Y	SUP	C	4.00	10.40	18.20	33.00	44.50	33.50	42.98
TXSTDSC	D66-2015	L2	21.75	28.34	Y	INF	C	4.00	12.50	5.00	33.00	44.50	33.50	42.98
TXSTDSC	D66-2015	L3	21.13	28.20	Y	SUP	C	2.00	7.70	30.00	34.67	42.20	33.55	45.56
TXSTDSC	D66-2015	L3	21.13	28.20	Y	INF	C	1.50	6.40	17.30	34.67	42.20	33.55	45.56
TXSTDSC	D66-2015	L4	21.22	29.80	Y	SUP	C	1.50	5.40	13.80	32.50	45.50	32.46	45.90
TXSTDSC	D66-2015	L5	24.80	30.60	Y	SUP	C	3.00	4.80	5.40	32.56	47.90	30.52	46.40

Figure 8. Example of data collection spreadsheet.

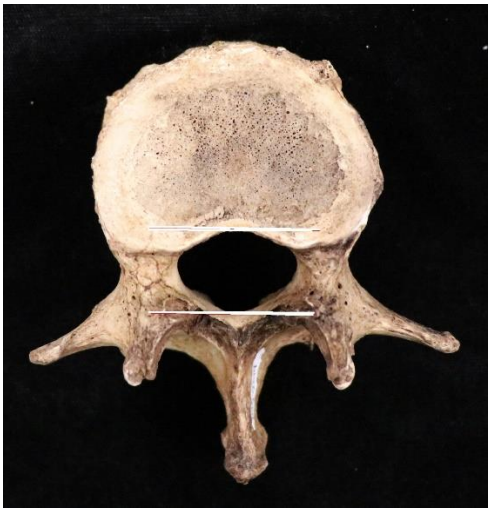

Figure 9. VNC AP diameter (sup view).

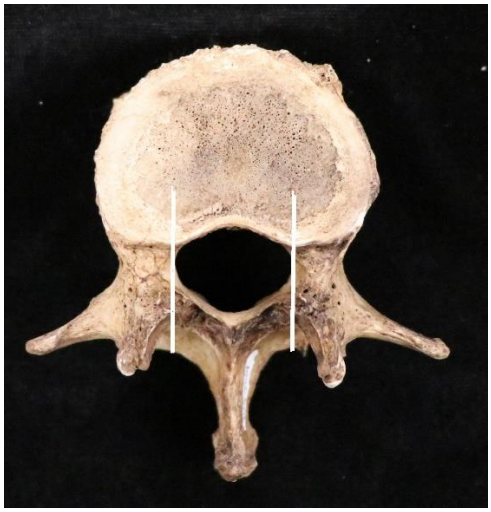

Figure 10. VNC TR diameter (sup view).

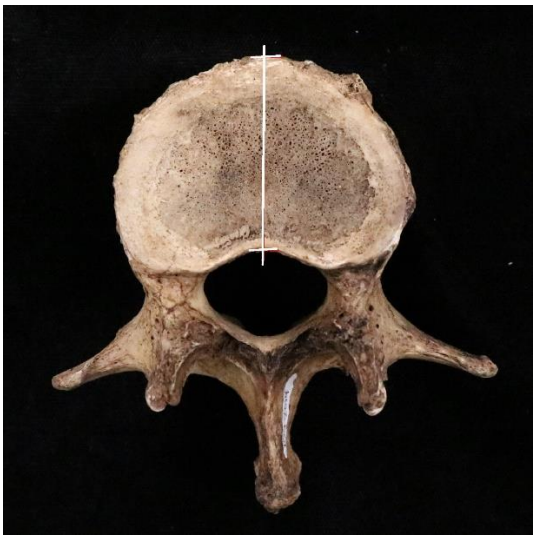

Figure 11. Vertebral surface AP diameter (sup view).

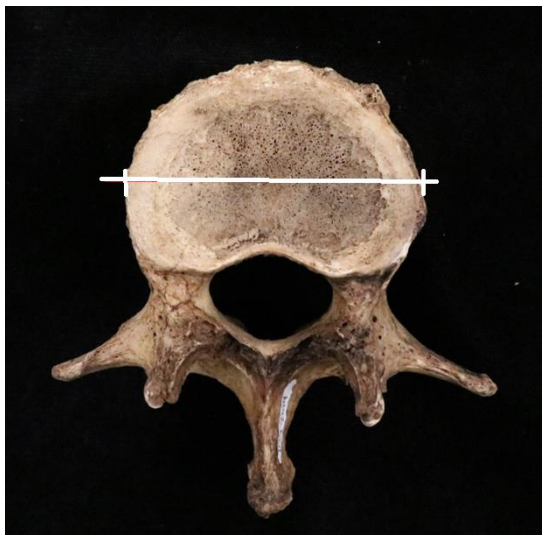

Figure 12. Vertebral surface TR diameter. (sup view).

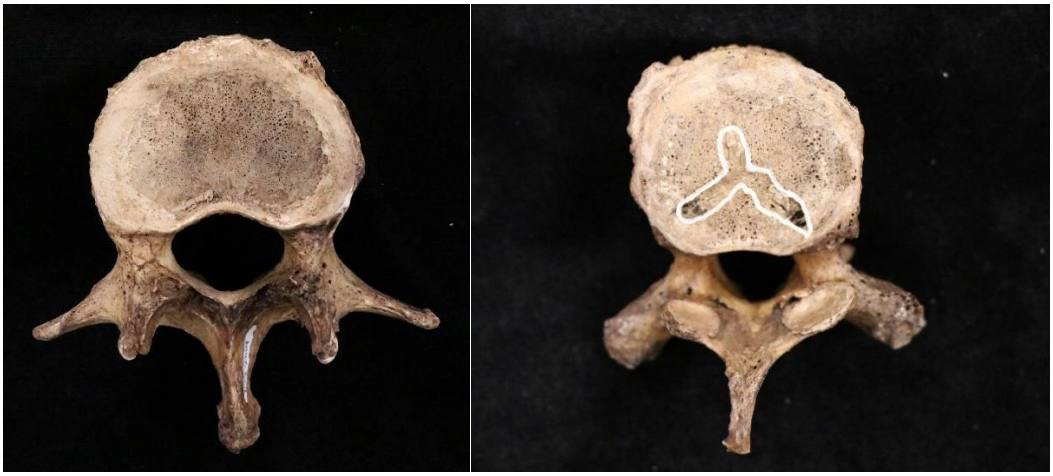

Figure 13. Absence sup (left) and presence inf (right) of SN.

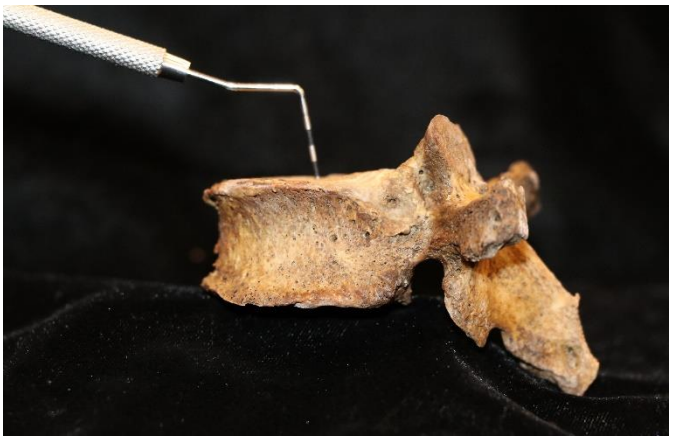

Figure 14. Measuring depth of SN (left lateral)

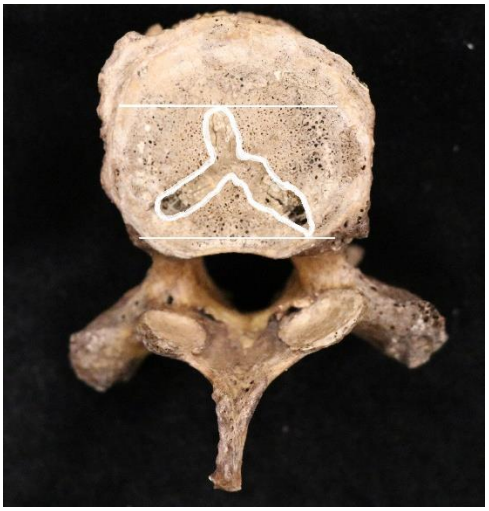

Figure 15. AP width of SN (inf view).

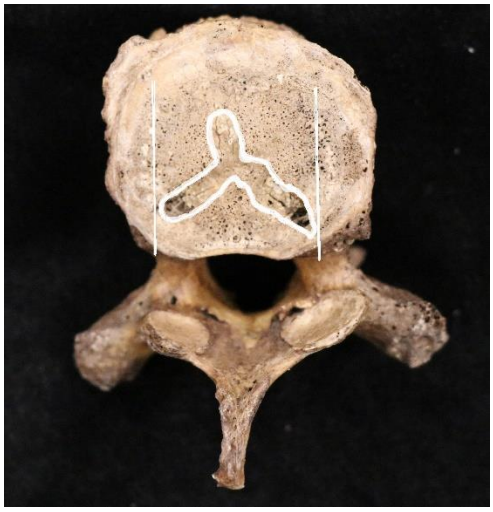

Figure 16. TR width of SN (inf view).

Calculating Severity. The severity of each SN was calculated, using Knüsel et al.'s (1997) classification method, where 'Stage 1' indicates a SN with a depth less than 2mm and length less than half of the vertebral body's anteroposterior length, and 'Stage 2' indicates a SN with a depth more than 2mm and length more than half of the vertebral body's anteroposterior length. Vertebrae with no present SNs were classified as 'Healthy'. However, during data collection, it was determined that this analysis did not sufficiently calculate the severity of SNs that were oriented along the transverse, rather than anteroposterior, axis of the vertebral surface because it did not account for the maximum size of the SN relative to the surface. Therefore, the severity was ultimately calculated using either the AP or TR diameters of the SN relative to the vertebral surface depending on the orientation of the SN and the most severe stage was used.

Confounding variables and limitations. Age was collected when available for all individuals included in this study and presented in Tables 3-5. However, it was not used as a variable in analysis due to inconsistencies in measuring and presenting age at death between the three samples. While age is presented as chronological age at death in the TXSTDSC, in the other samples age is estimated based on various techniques and the available skeletal material and presented in much broader categories that do not allow for direct comparison.

Poor preservation of skeletal remains that exhibited moderate to severe fragility and destruction of the vertebral surfaces and/or VNC prevented proper measurements. In some cases, the destruction was minimal enough that the measurement could be estimated (and was noted as being), but in other cases, the poor preservation was severe enough that the measurement was omitted. The presence of further skeletal pathologies

and medical interventions that prevented the observation of vertebra surfaces and obscured SN scoring, excluded vertebrae from being included in this study. Additionally, individuals who had been cremated or were infants, or lacked mature and fused vertebrae, were also omitted from analysis.

Statistical Analysis

Basic descriptive statistics were carried out in Excel ® while all other analyses were carried out in JMP Pro ® 13 as described below (unless noted otherwise).

VNC AP, TR, and AP/TR Ratio Dimensions x Sex. The VNC AP, TR and ratio dimensions (dependent variable) were tested for statistical significance against sex (independent variable) in all three samples. Individuals with an indeterminate sex were excluded from these analyses. First, an F-Test Two-Sample for Variances was run for each vertebra (T1-L5) to determine if the variances were equal or unequal between females and males. If the p-value was significant ($p<0.05$), then the variances were determined to be unequal. If the p-value was insignificant ($p>0.05$), then the variances were determined to be equal. The appropriate t-Test: Two-Sample Assuming (Un)Equal Variance test was then conducted for each vertebra (T1-L5) to test for significance ($p<0.05$) between the VNC AP, TR, or Ratio dimensions of females and males.

VNC AP, TR, and AP/TR Ratio Dimensions x SES Levels. The VNC AP, TR and ratio dimensions (dependent variables) were also tested for statistical significance ($p<0.05$) against SES levels (independent variable) in all three samples using an ANOVA: Two-Factor without Replication. Statistical analyses were originally completed using five SES levels (Low, Low-Middle, Middle, Upper-Middle, Upper). However, due to varying sample sizes of each SES (e.g., Upper, n = 2 versus Middle, n = 20) the SES

levels were collapsed into two groups, Lower (Low and Low-Middle) and Upper (Middle, Upper-Middle, Upper). This adjustment is referred to as "grouped SES" whereas "SES" signifies the five original SES levels.

Schmorl's Nodes. Descriptive statistics were employed to assess inter and intra sample distributions of SNs and their severity along the spinal column. These distributions were tested for significance against demographic information including sex (TXSTDSC, OpID, PCOME), occupation type (TXSTDSC), and SES (TXSTDSC). Differences in severity calculations, as discussed under "Calculating Severity", was also tested for significance using a Pearson's chi-squared test to assess the efficacy and reliability of the Knüsel et al. (1997) method.

VNC AP, TR, and AP/TR Ratio Dimensions x SN Association. The VNC AP, TR, and ratio dimensions (continuous variables) were tested for an association correlation with the presence/absence of SNs (binary variable). In the TXSTDSC sample, T1-T4 were excluded due to absence of SN data and the analysis was completed for T5-L5. The resulting R-values spanned from -1 to 1 with +/- 0.01-0.19 indicating a very weak correlation, 0.2-0.39 indicating weak, 0.40-.59 indicating moderate, 0.6-0.79 indicating strong, and 0.8-1 indicating very strong, and 0 corresponding to no association. This analysis was also completed for T6 - L2 in the OpID sample and T7 - L5 in the PCOME sample.

IV. RESULTS

Results will be presented in the order described under "Statistical Analysis" in the Materials and Methods chapter; first comparatively across the samples, then examined in detail for each sample individually.

VNC AP, TR, and AP/TR Ratio Dimensions x Sex

Comparative. Results from the t-Tests indicate significant differences between the VNC dimensions of females and males in all three samples (Table 6). The vertebrae which exhibit significant differences between the sexes varies between the samples, however. In the TXSTDSC, the lumbar vertebrae between females and males vary in AP, TR, and ratio dimensions. In the OpID sample, females and males primarily differ in the TR diameters of the thoracic and upper lumbar regions, as well as the AP and ratio dimensions of the lower lumbar. In the PCOME sample, differences between females and males are limited to TR diameters and ratio dimensions sporadically in the thoracic region.

An ANOVA: Two-Factor without Replication was completed to test for significant difference in the VNC AP diameters between the three samples and found a significant difference between them at the $p<0.05$ level, $F(2, 16) = 83.107$, $p = 2.1E\text{-}13$, with the TXSTDSC having the largest AP diameters across the spinal column, followed by the PCOME and OpID samples (Figure 17). Stratifying these results by sex reveals that while females in the TXSTDSC are larger than males in the AP diameters, males are larger than females in the PCOME and OpID samples (Figure 18).

Table 6. T-test results, testing for significant differences between the VNC dimensions of females and males in each of the samples.

	TXSTDSC			***OpID***			***PCOME***		
	VNC AP	VNC TR	VNC Ratio	VNC AP	VNC TR	VNC Ratio	VNC AP	VNC TR	VNC Ratio
T1	0.557	0.008	0.720	0.148	0.074	0.852	0.767	0.505	0.570
T2	0.274	0.211	0.294	0.138	**0.006**	0.557	0.767	0.083	0.080
T3	0.552	0.216	0.227	0.114	**0.001**	0.199	0.907	**0.024**	**0.049**
T4	0.974	0.258	0.233	0.166	**0.000**	0.101	0.305	0.066	0.573
T5	0.643	0.150	**0.039**	0.231	**0.001**	0.077	0.264	0.080	0.740
T6	0.670	0.304	0.111	0.865	**0.001**	**0.003**	0.910	0.057	**0.075**
T7	0.607	0.640	0.318	0.275	**0.006**	0.111	0.530	**0.047**	0.306
T8	0.699	0.765	0.890	0.088	**0.004**	0.279	0.103	0.070	0.790
T9	0.620	0.520	0.909	0.063	**0.006**	0.153	0.278	0.053	0.198
T10	0.773	0.461	0.723	0.200	**0.031**	0.523	0.963	**0.034**	**0.006**
T11	0.474	0.360	0.990	0.322	0.114	0.602	0.172	0.092	**0.013**
T12	0.611	0.190	0.583	0.945	**0.018**	0.162	0.992	0.098	0.093
L1	0.447	**0.028**	**0.006**	0.816	**0.014**	0.861	0.918	0.101	0.102
L2	**0.007**	**0.040**	**0.000**	0.066	**0.042**	**0.029**	0.958	0.060	0.090
L3	**0.023**	**0.026**	**0.000**	0.066	0.395	**0.029**	0.622	0.108	0.061
L4	0.272	0.203	**0.008**	**0.011**	0.895	**0.004**	0.879	0.354	0.335
L5	0.721	0.546	0.286	**0.019**	0.399	**0.002**	0.830	0.397	0.216

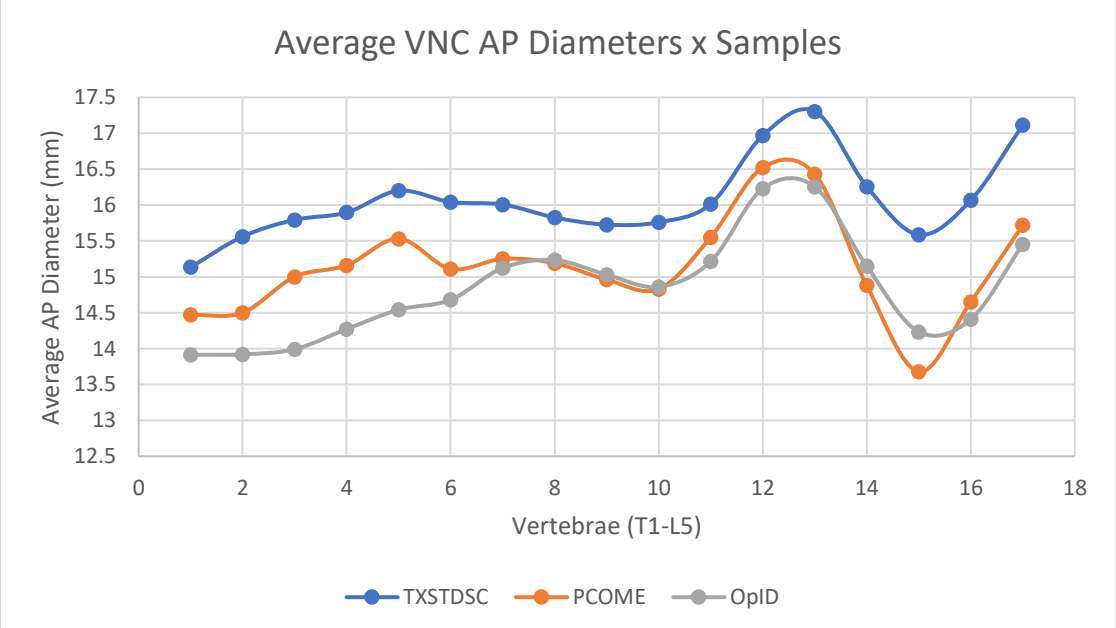

Figure 17. Average vertebral neural canal (VNC) anteroposterior (AP) diameters among all individuals in the TXSTDSC, PCOME, and OpID samples.

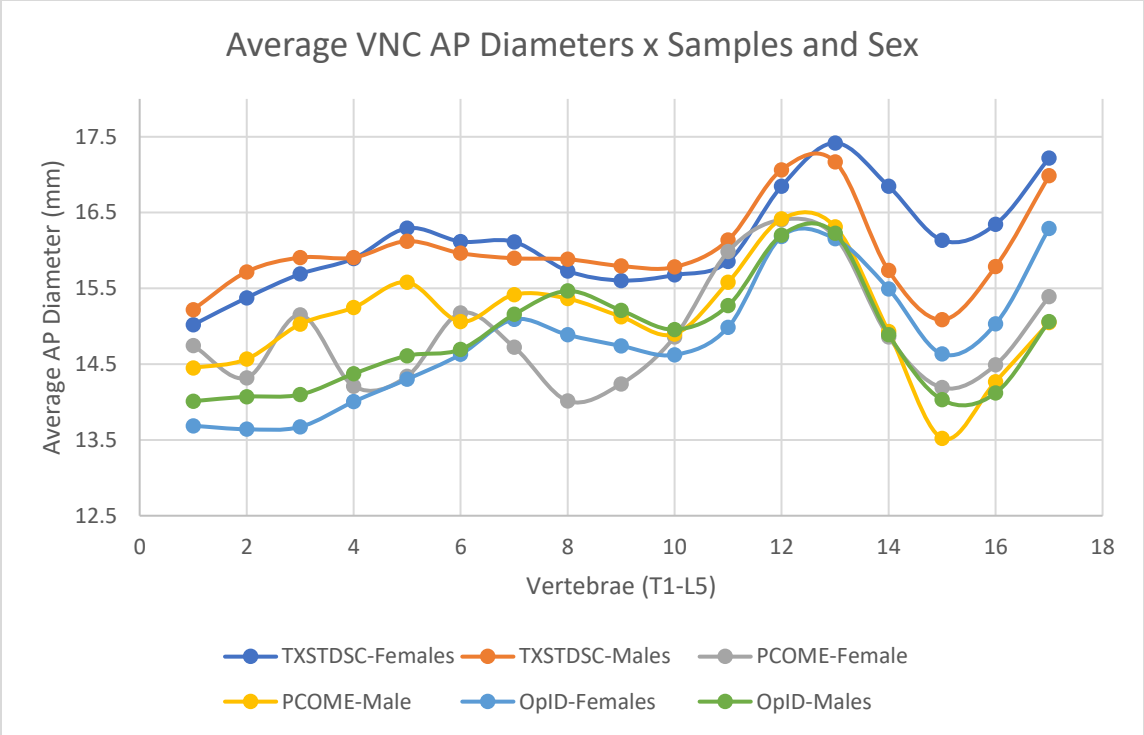

Figure 18. Female and male average vertebral neural canal (VNC) anteroposterior (AP) diameters among individuals in the TXSTDSC, PCOME, and OpID samples.

An ANOVA: Two-Factor without Replication was completed to test for significant difference in the VNC TR diameters between the three samples and found a significant difference at the $P<0.05$ level, $F(2, 16) = 92.713$, $p = 4.85E\text{-}14$, with the TXSTDSC having the largest TR diameters across the spinal column, followed by the PCOME and OpID (Figure 19). Stratifying these results by sex reveals that males on average are larger than females in each sample, and that the PCOME and OpID males are more similar to each other than two their female counterparts (Figure 20).

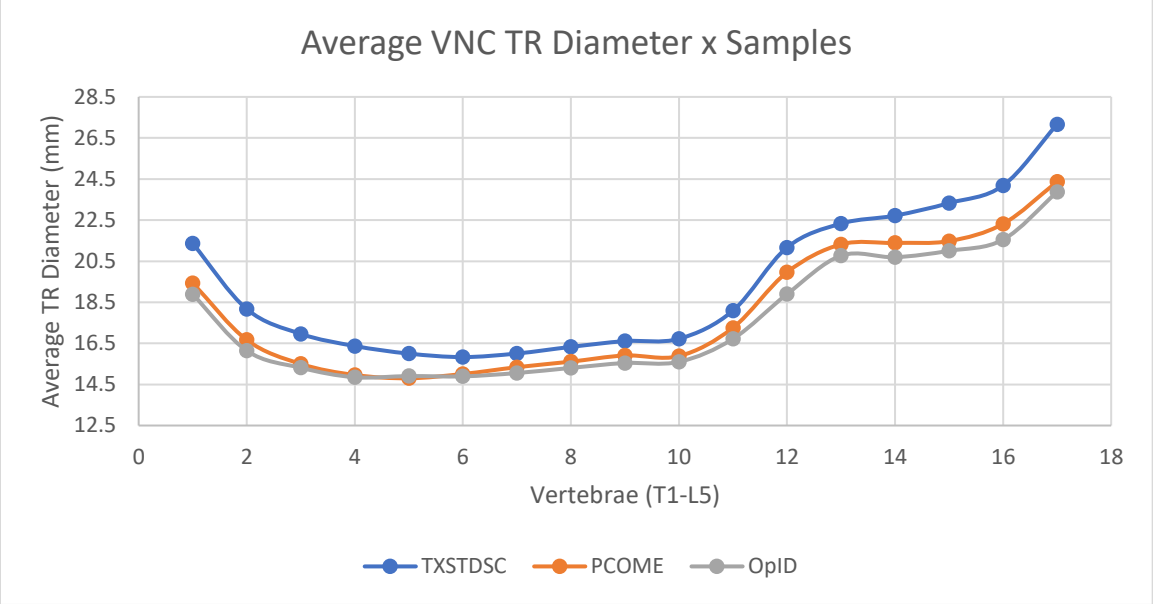

Figure 19. Average vertebral neural canal (VNC) transverse (TR) diameters among all individuals in the TXSTDSC, PCOME, and OpID samples.

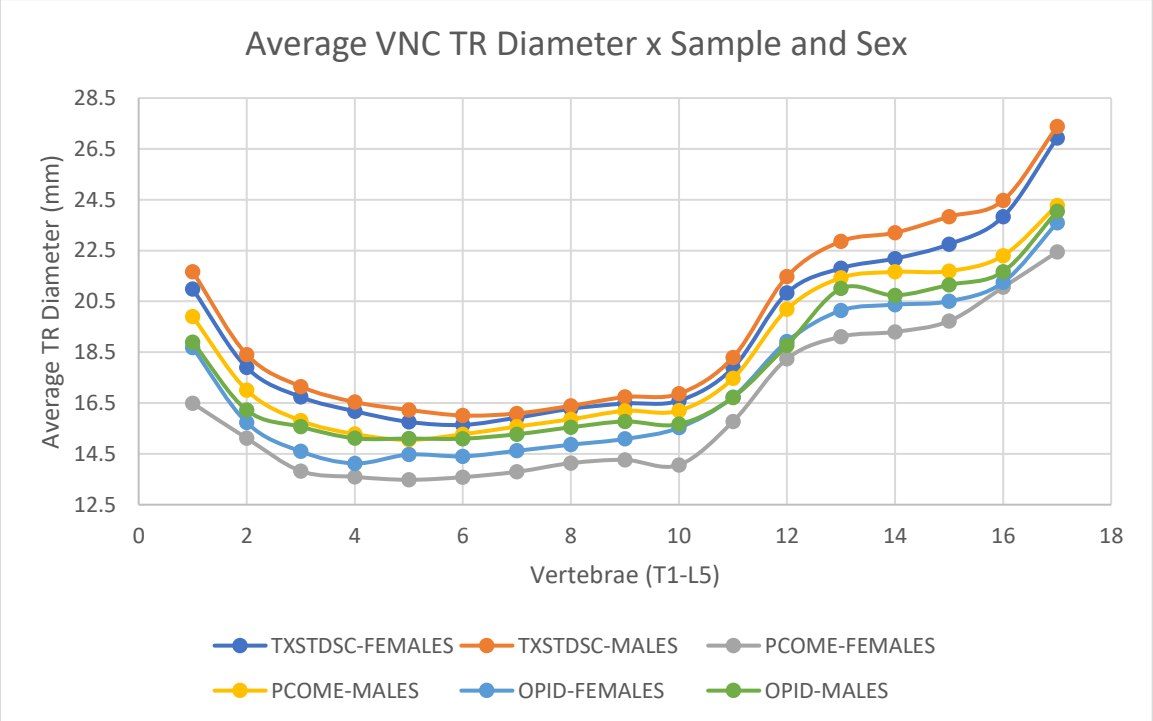

Figure 20. Female and male average vertebral neural canal (VNC) transverse (TR) diameters among individuals in the TXSTDSC, PCOME, and OpID samples.

An ANOVA: Two-Factor without Replication was completed to test for significant differences in the VNC AP/TR ratios between the three samples and found no significant difference at the $p<0.05$ level, $F(2, 16) = 0.068$, $p = 0.934$, with all three

samples having a relatively similar AP/TR ratio throughout the entire spinal column (Figure 21). However, when stratified by sex, an ANOVA analysis indicated a significant difference, F (5, 16) = 34.201, p = 1.56E-18 (Figure 22).

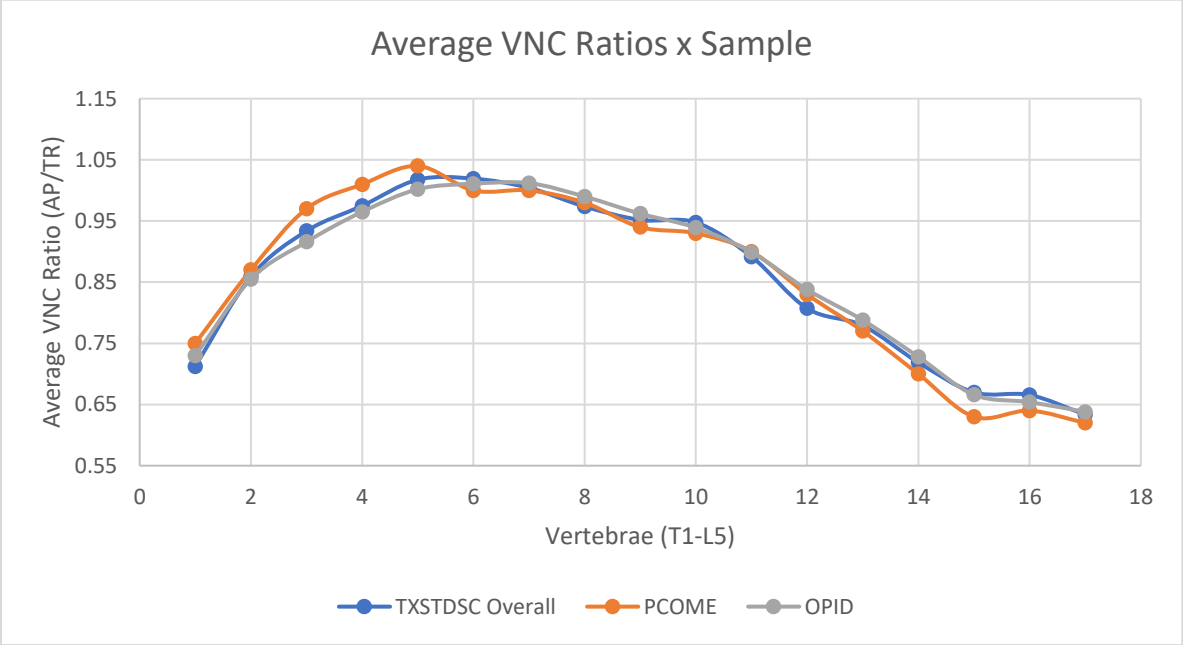

Figure 21. Average Overall VNC ratios (AP diameter/TR diameter) among individuals from TXSTDSC, PCOME, and OpID.

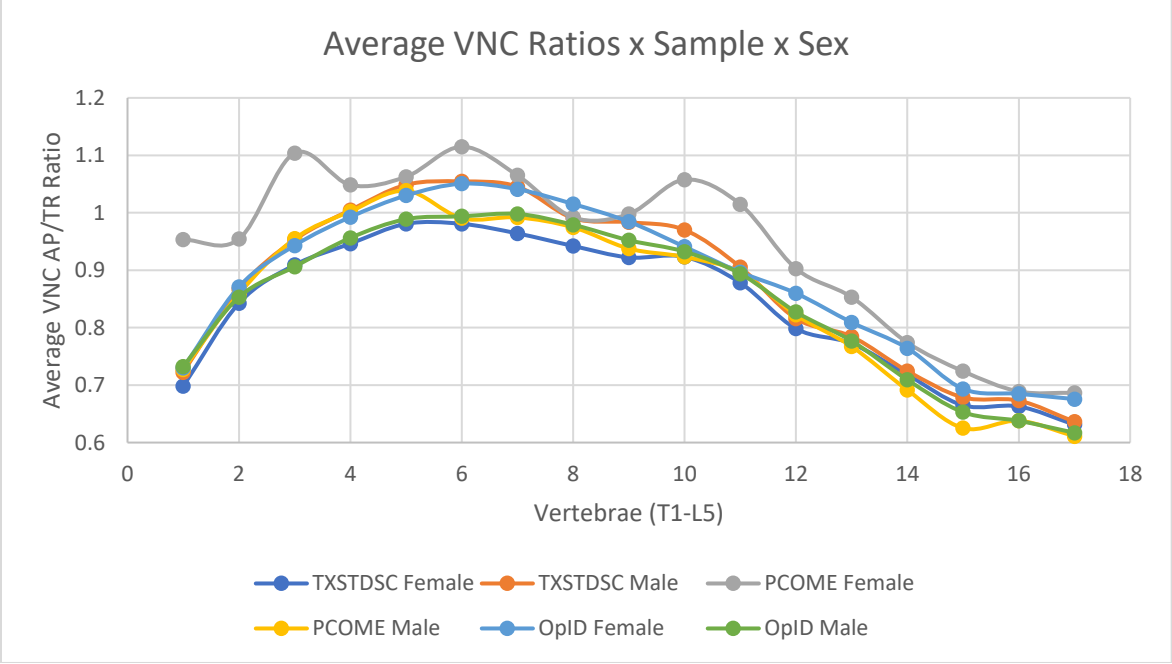

Figure 22. Average overall VNC ratios (AP diameter/TR diameter) among individuals from all the samples.

Texas State Donated Skeletal Collection Sample. The significant difference between females (n = 38) and males (n = 38) as shown in Table 6 are more closely examined in this section. Females have smaller upper and lower thoracic AP diameters, but larger middle thoracic and lumbar AP diameters, in comparison to males (Figure 23). TR diameters in females are smaller than males throughout the spinal column (Figure 24). Therefore, the difference in AP/TR ratios reflects the larger AP diameters of females and larger TR diameters of males (Figure 25).

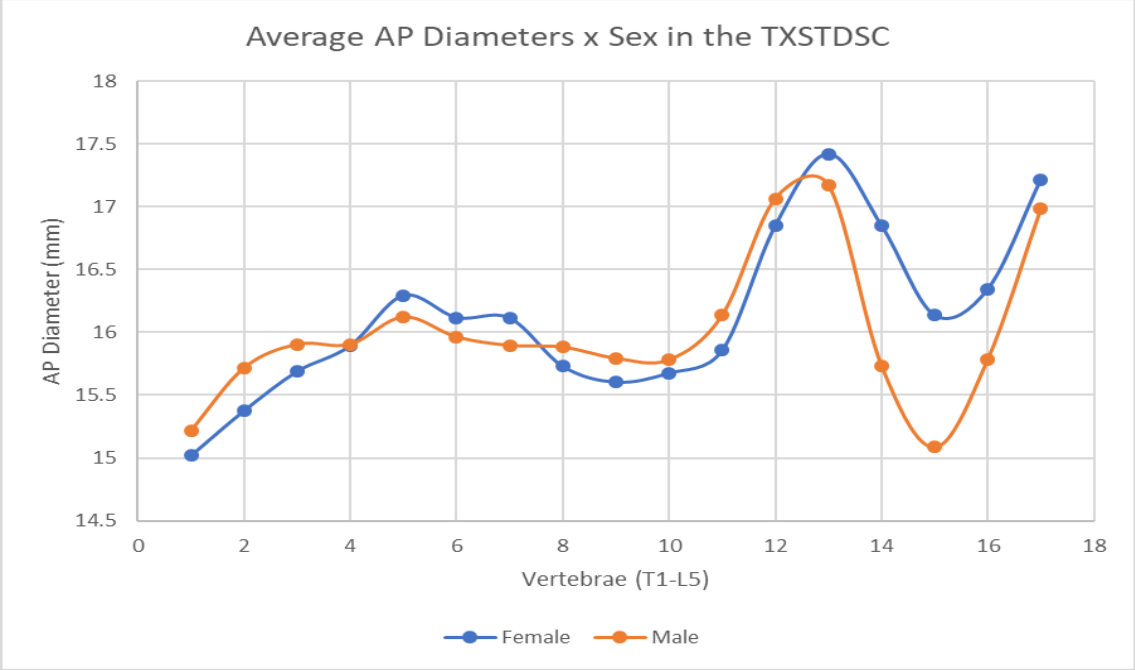

Figure 23. The average vertebral neural canal anteroposterior diameter throughout the spinal column (T1-L5) among females and males in the TXSTDSC sample.

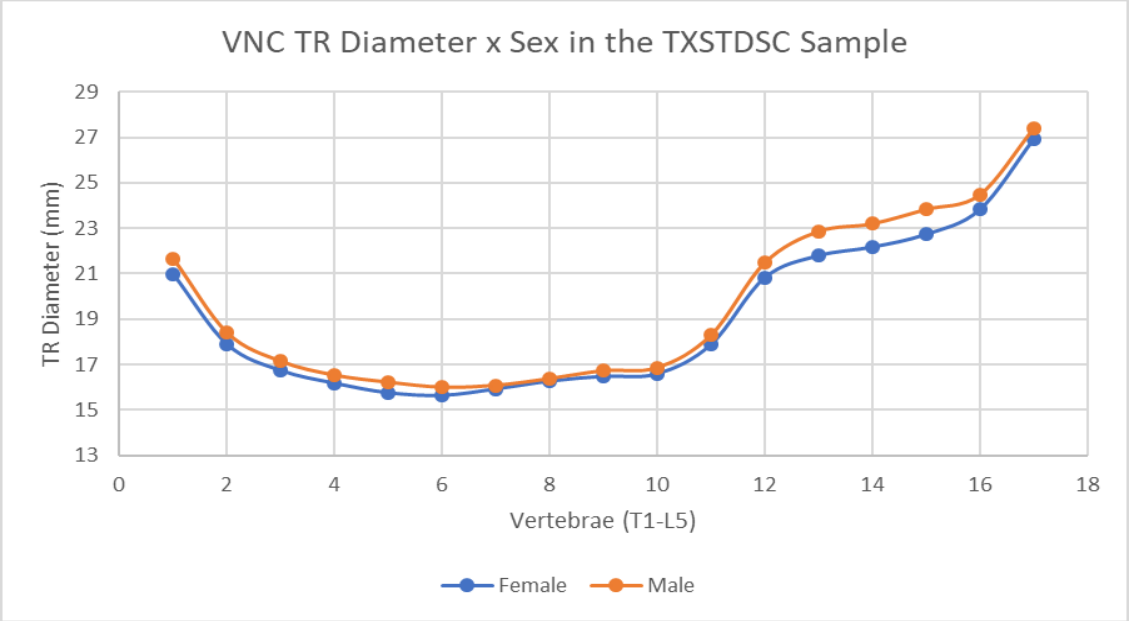

Figure 24. The average vertebral neural canal anteroposterior diameter throughout the spinal column (T1-L5) among females and males in the TXSTDSC sample.

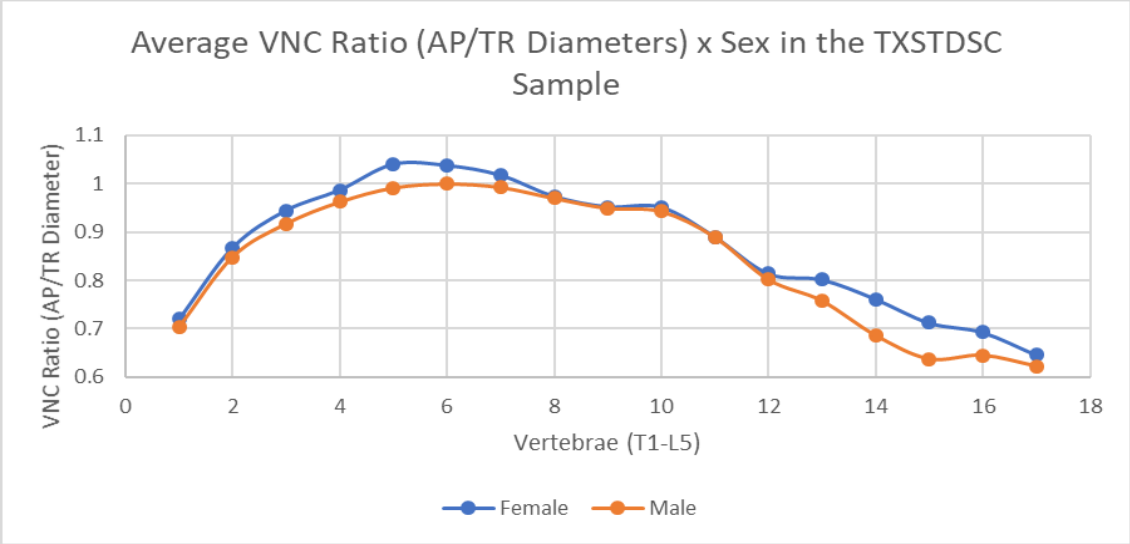

Figure 25. Average VNC ratio (AP/TR diameters) by sex along the spinal column (T1-L5) in the TXSTDSC.

Operation Identification Sample. Females (n = 30) have smaller AP diameters than males (n = 67) throughout the spinal until the lumbar region, where they are larger (Figure 26). Females and males are most significantly different in the TR diameters where females are significantly larger through the thoracic, with exception of T11, and

the upper lumbar regions (Figure 27). The ratio dimensions are significantly different in the lumbar region (Figure 28).

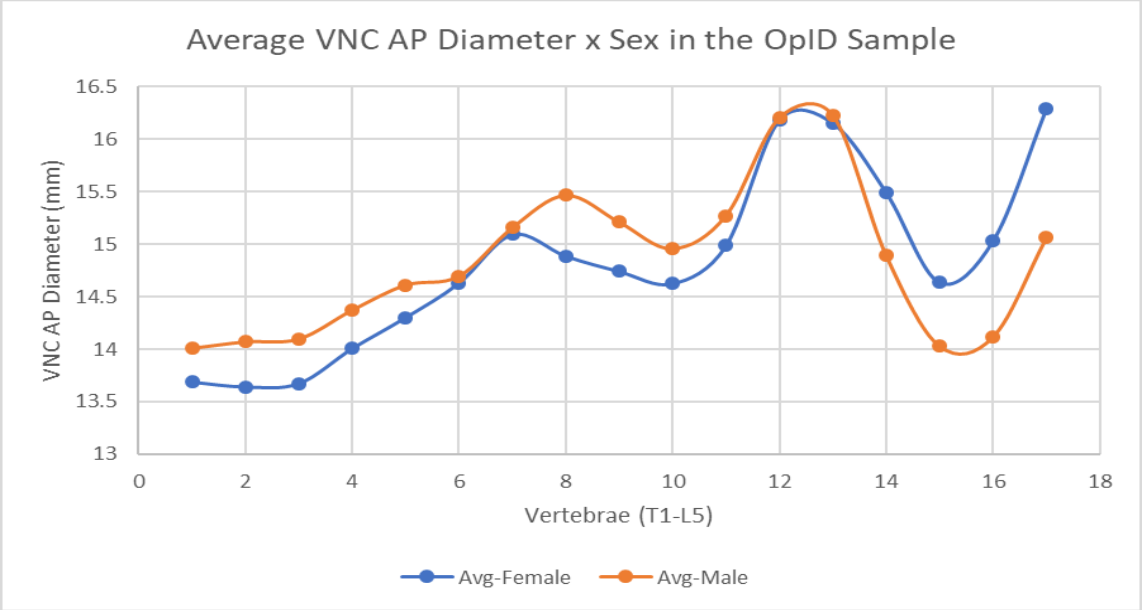

Figure 26. The average vertebral neural canal anteroposterior diameter throughout the spinal column (T1-L5) among females and males in the Operation Identification sample.

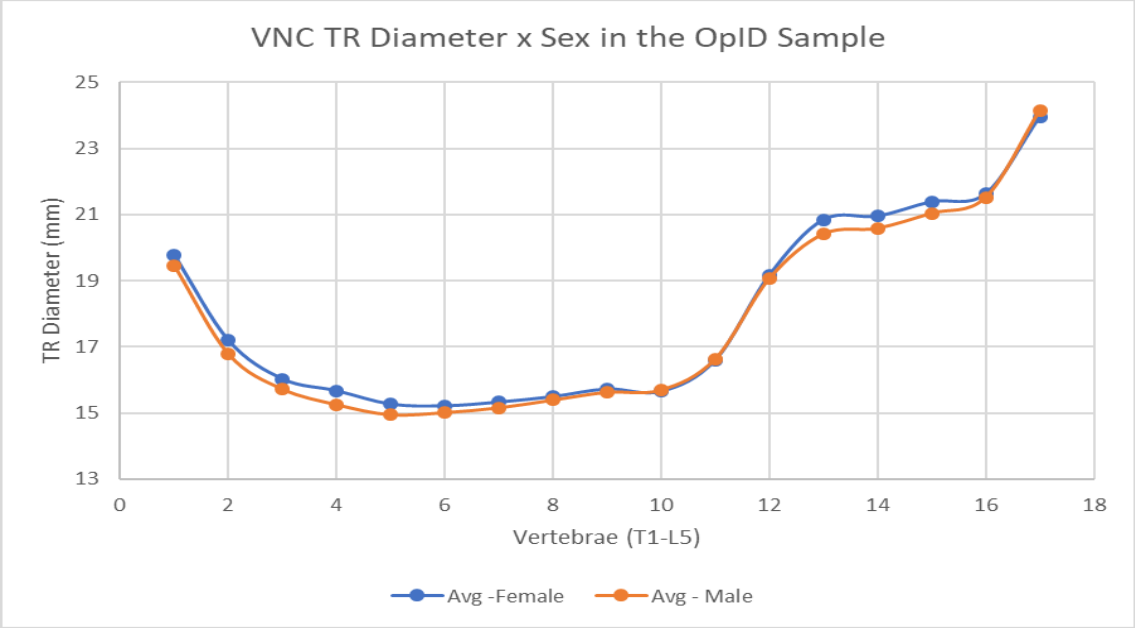

Figure 27. Average VNC TR Diameter along the spinal column (T1-L5) among females and males in the Operation Identification sample.

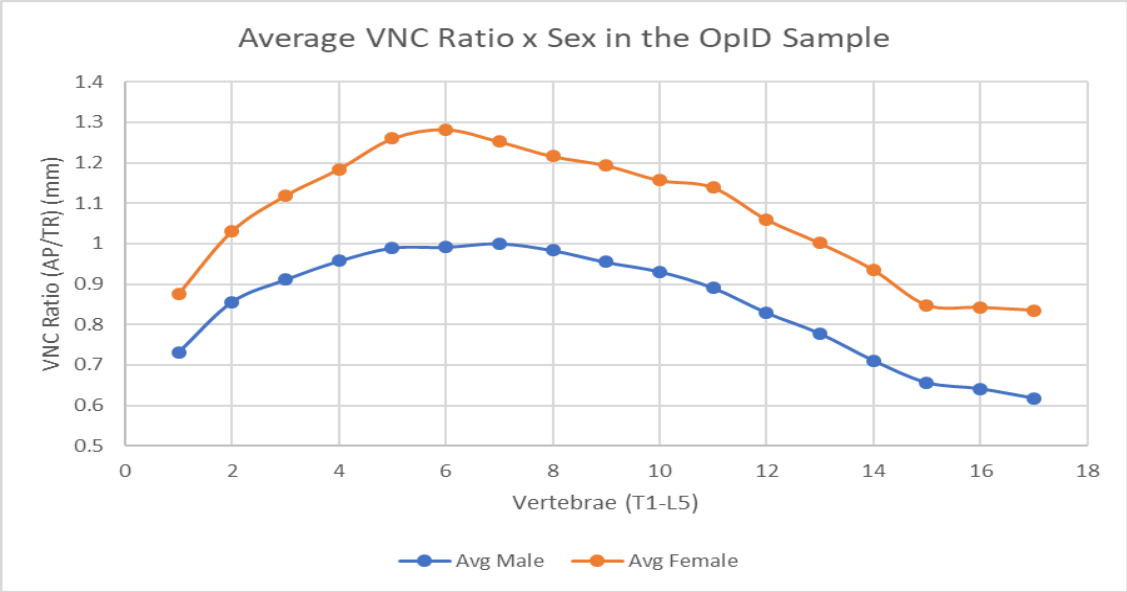

Figure 28. Average VNC ratios (AP/TR diameters) along the spinal column (T1-L5) among females and males in the Operation Identification sample.

Pima County Office of the Medical Examiner Sample. No statistically significant difference was found between the average VNC AP diameters of T1-L5 in males (n=30) and females (n=2) from the PCOME sample (Table 29). However, females are visually smaller on average in the upper and middle thoracic vertebrae in comparison to males. Females have significanlty smaller TR diameters in T3, T7, T10 when compared to males (Figure 30). Females have larger ratios, indicating their TR are larger than AP diameters, but this is only significantly different from males in T3, T6, T10, and T11 (Figure 31).

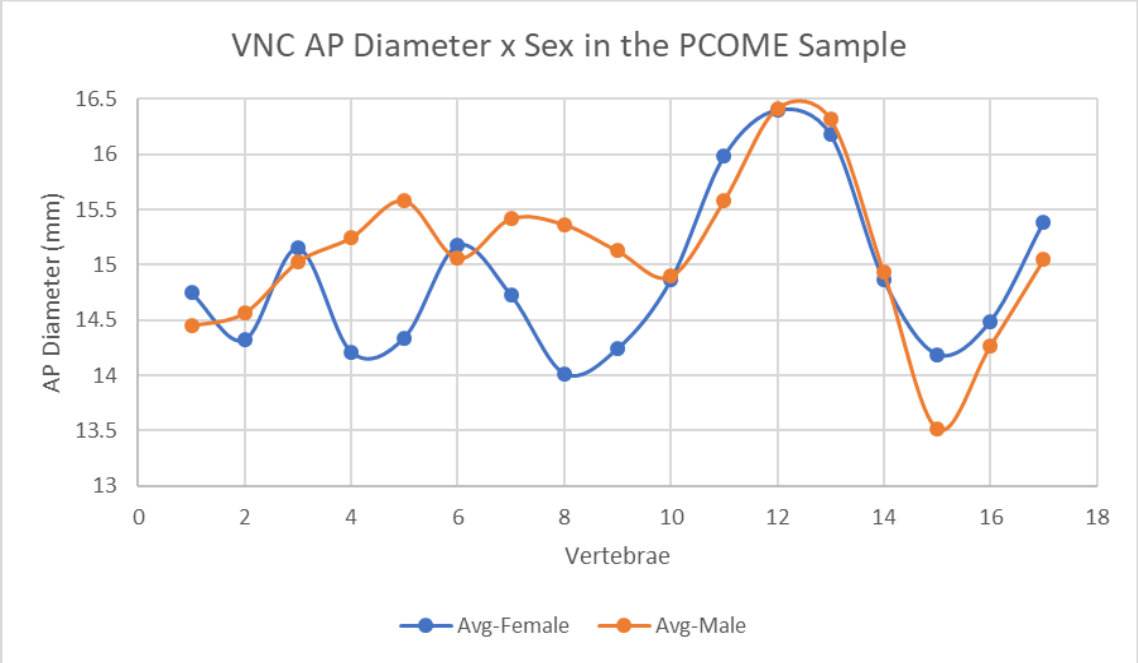

Figure 29. The average vertebral neural canal anteroposterior diameter throughout the spinal column (T1-L5) among females and males in the PCOME sample.

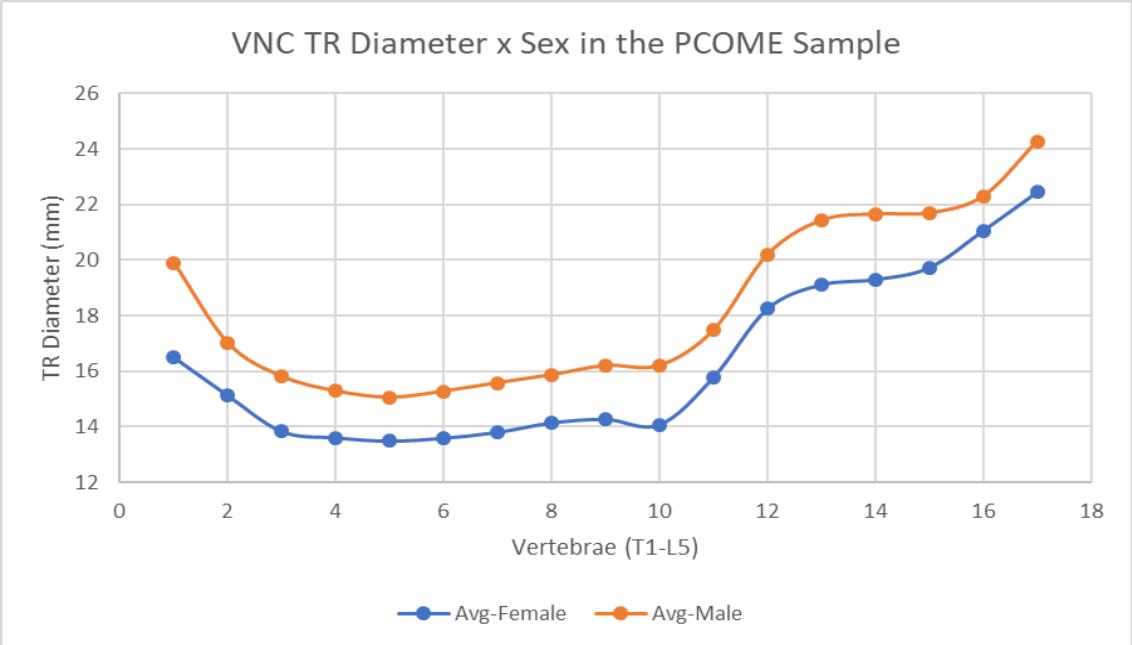

Figure 30. The average vertebral neural canal transverse diameter throughout the spinal column (T1-L5) among females and males in the PCOME sample.

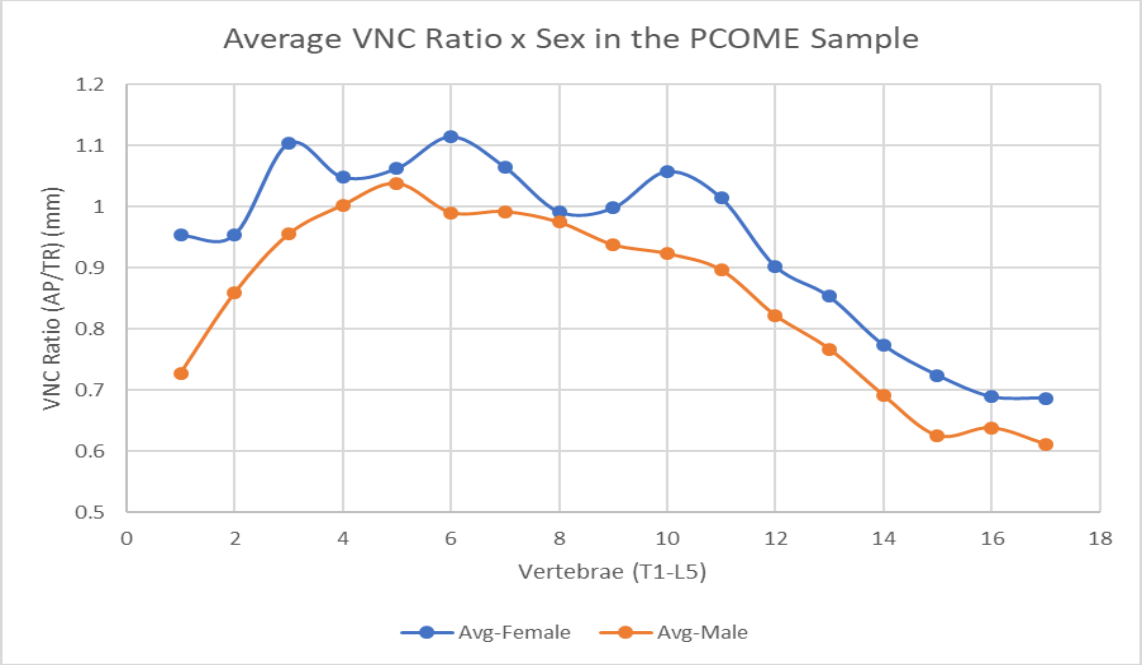

Figure 31. Average VNC ratio by sex along the spinal column for females and males in the PCOME sample.

VNC AP, TR, and AP/TR Ratio Dimensions x SES Levels

Comparative. Assessing VNC AP and TR diameters, with the TXSTDSC stratified by grouped SES levels, reveals that all SES levels are larger than the PCOME and OpID samples in both measurements (Figure 32 and Figure 33). Comparison of VNC ratios between samples and the TXSTDSC stratified by grouped SES levels reveals that ratios are similar throughout the spinal column (Figure 34), but an ANOVA testing for difference between the SES levels in the TXSTDSC samples found significant differences at the $p<0.05$ level, $F(4, 16) = 32.774$, $p = 1.12E-11$ (Figure 34).

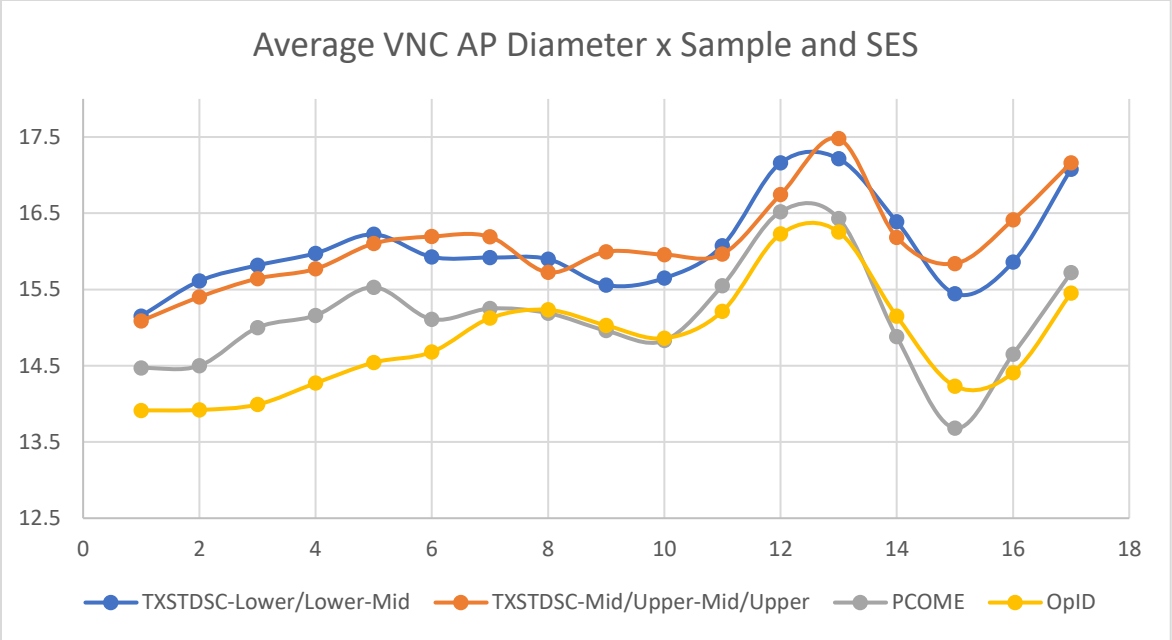

Figure 32. Average vertebral neural canal (VNC) anteroposterior (AP) diameters among individuals from the PCOME and OpID and by grouped socioeconomic status (SES) among individuals in the TXSTDSC.

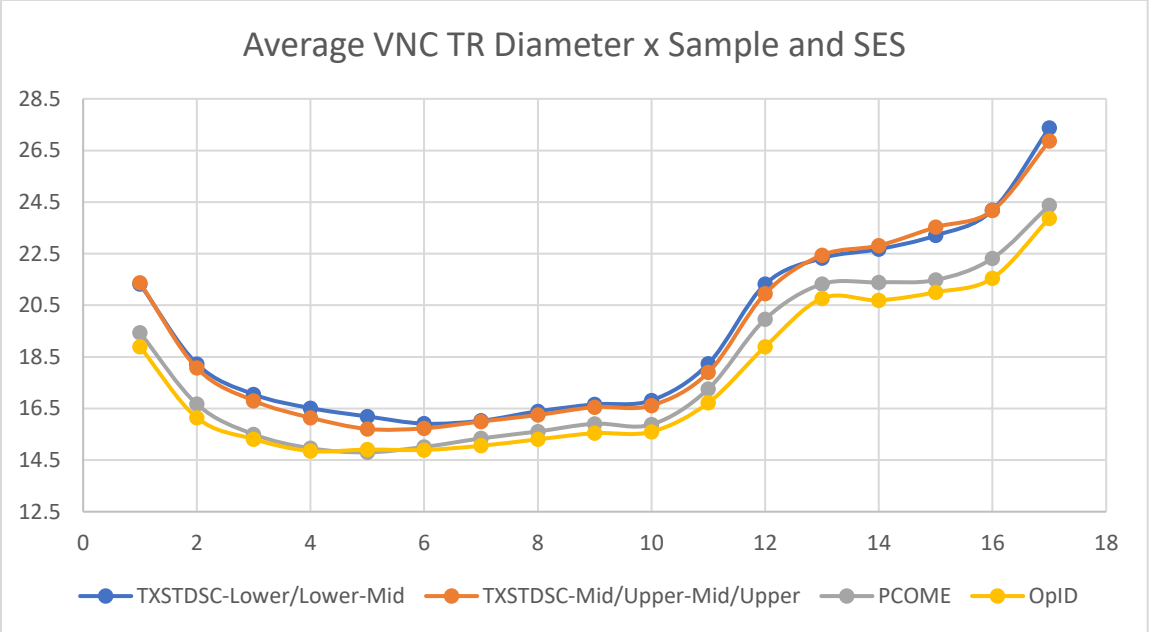

Figure 33. Average vertebral neural canal (VNC) transverse (TR) diameters among individuals from PCOME and OpID and grouped SES among individuals in the TXSTDSC.

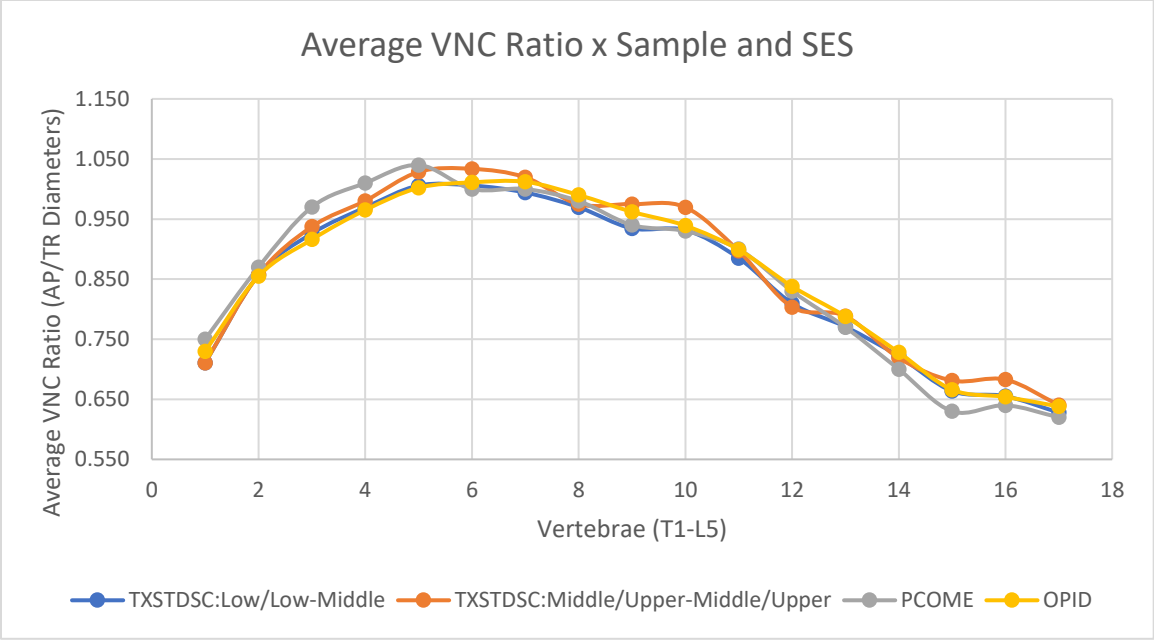

Figure 34. Average Overall VNC ratios (AP diameter/TR diameter) among individuals from PCOME, OpID, and TXSTDSC SES levels.

VNC AP Diameters x Grouped SES Level, TXSTDSC. The ANOVA: Two-Factor without Replication was conducted to assess VNC AP diameters of individuals in the TXSTDSC sample stratified by grouped SES indicated a significant difference between the groups (p = 1.07E-10) (Figure 35).

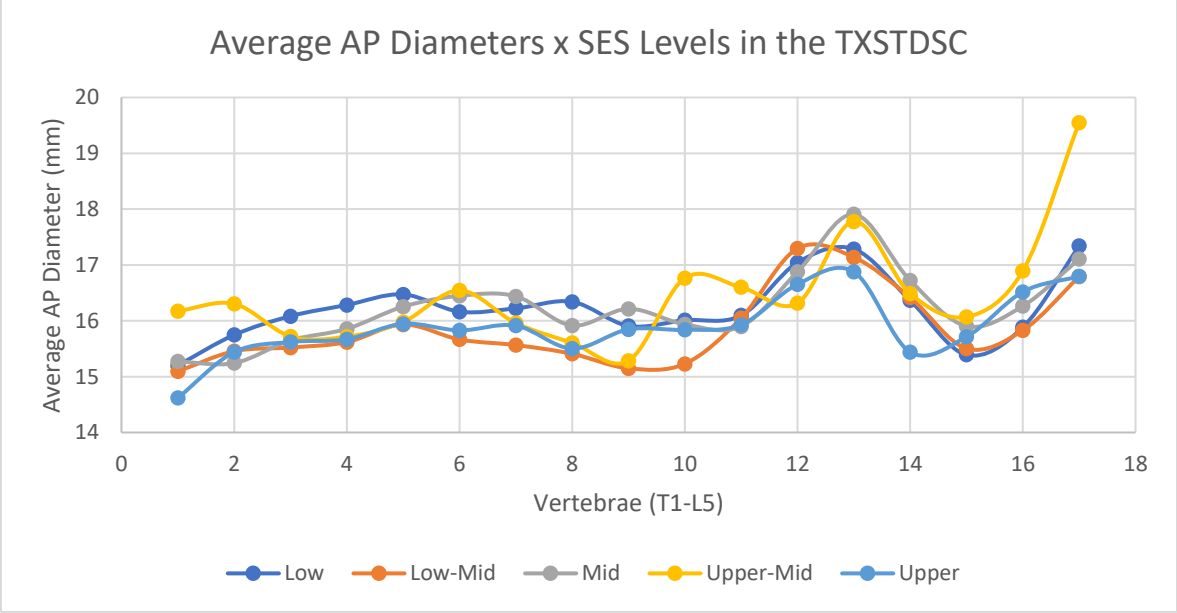

Figure 35. Average vertebral neural canal (VNC) anteroposterior (AP) diameters by socioeconomic status (SES) among individuals in the TXSTDSC.

VNC TR Diameters x SES Level, TXSTDSC. The ANOVA Two-Factor Without Replication indicated a significant different between the VNC TR diameters of SES groups (L, LM, M, UM, U), p = 9.02E-16. The average VNC TR diameters (T1-L5) were calculated and show the significant difference in the lower thoracic through the lumbar (Figure 36).

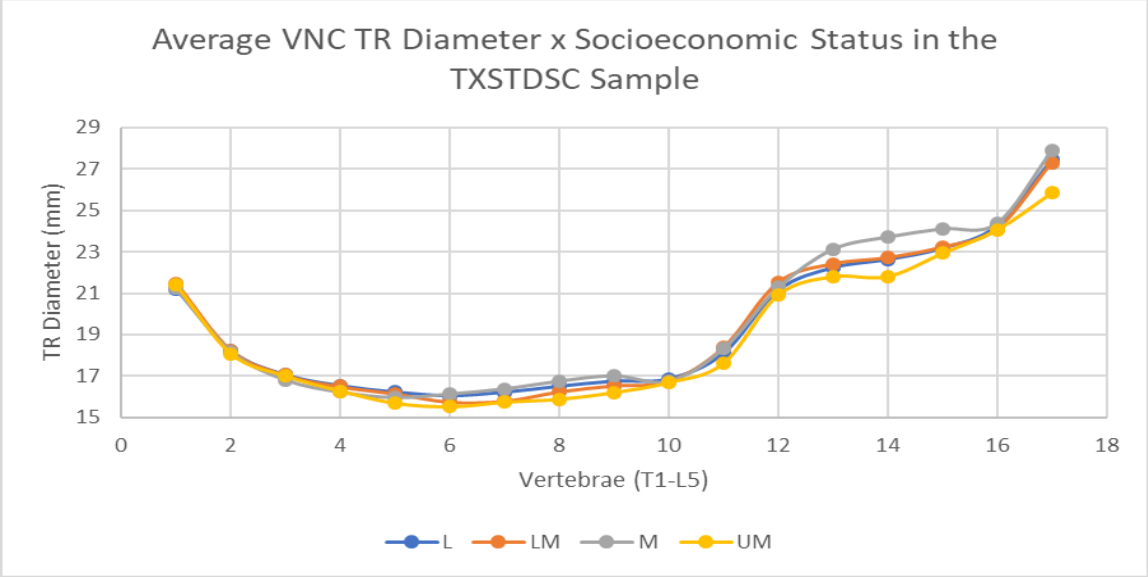

Figure 36. The average vertebral neural canal TR diameter throughout the spinal column (T1-L5) among Lower (L), Low-Middle (LM), Middle (M), and Upper-Middle (UM) socioeconomic status levels in the TXSTDSC sample.

VNC Ratio Dimensions x Grouped SES Level, TXSTDSC. There is a significant difference between SES and VNC ratios, with individuals in the Upper groups exhibiting increasingly larger VNCs respectively. This indicates that the TR diameter is larger relative to the AP diameter, meaning there is more growth occurring throughout the adolescent period.

The average VNC AP/TR ratio dimensions were calculated for each SES level and plotted (Figure 37). To account for sample size differences between the levels, and to further distinguish differences in VNC morphology, the SES levels were collapsed in

"Grouped SES" levels and tested for significant differences (Figure 38). An ANOVA analysis indicated a significant difference using $p<0.05$, $F(6, 16) = 22.417$, $p = 2.36E\text{-}16$.

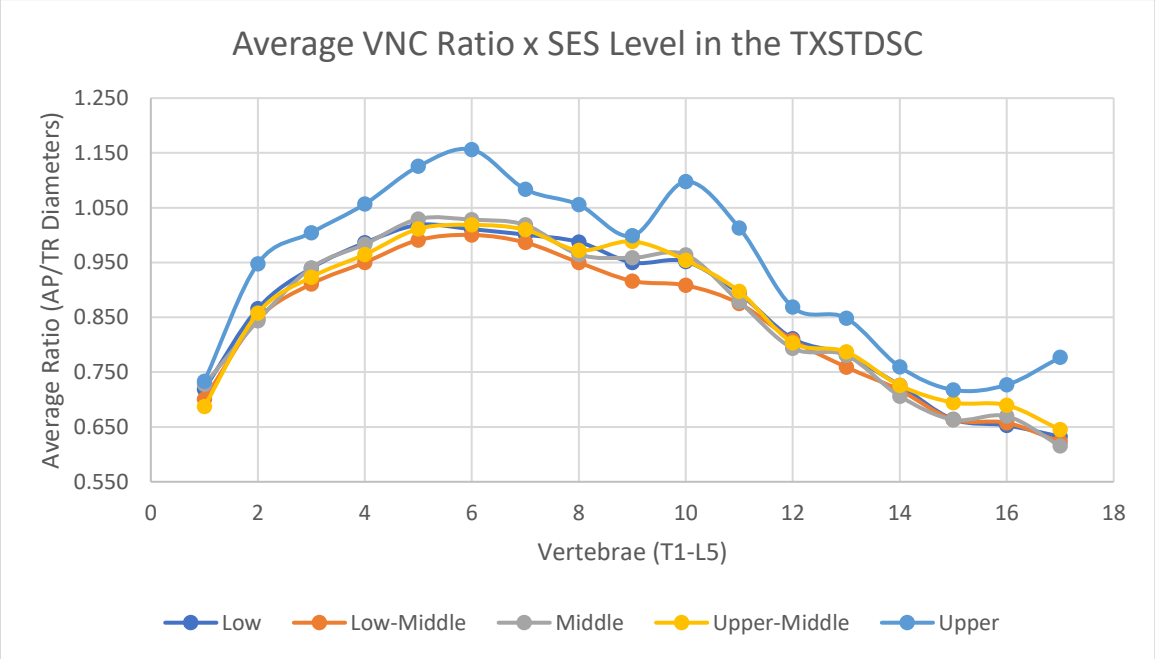

Figure 37. Average VNC ratios (AP diameter/TR diameter) by SES among individuals in the TXSTDSC.

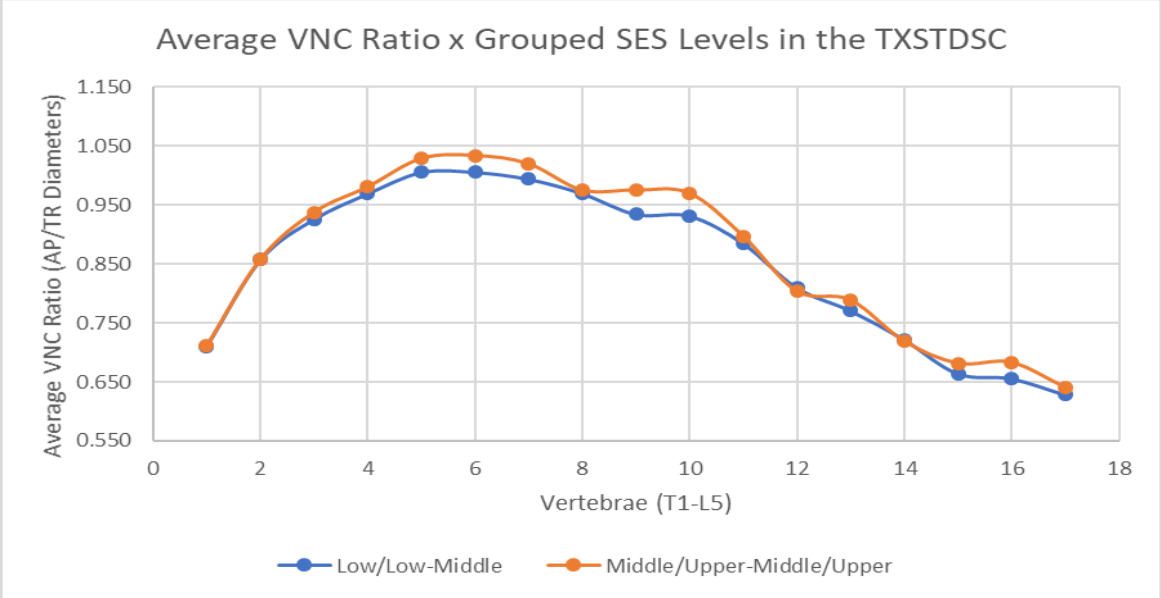

Figure 38. Average VNC ratios (AP diameter/TR diameter) by grouped SES among individuals in the TXSTDSC.

Schmorl's Nodes

In total, 676 Schmorl's nodes were observed across 4,430 total vertebrae (Table 7).

*Prevalence x Samples***.** A Chi-square test was conducted to test for a significant difference between the total number of Schmorl's nodes at each vertebra by individuals and by sample. T1-T2 were not included in this test to avoid error because all three samples exhibited zero present SNs on these vertebrae.

The Chi-square test for T3-L5 indicated that there is no statistical difference between the three samples, X^2 (2, N = 699) = 0.35.1049, p = 0.1669. While the presence/absence of SNs is not significant between the three samples, the distributions of SNs occur differently along the spinal column in the three samples (Figure 39). None of the samples exhibited SNs in the upper thoracic (T1-T3). The presence of SNs in the TXSTDSC sample follows a bimodal distribution, with the most SNs occurring at T8 and T11 and decreasing through the lower lumbar. The presence of SNs in the PCOME sample also follows a bimodal distribution, but with the most SNs occurring at T9 and L2. This peak at L2 is unique to the PCOME sample, where the TXSTDSC and OpID samples exhibit decreasing SNs through the lumbar. From T4-L5, the presence of SNs in the OpID sample follows a left skewed bell curve with most SNs occurring on the middle-lower thoracic vertebrae (T7-T10) and decreasing through the lower lumbar.

Table 7. Count data of present Schmorl's nodes relative to vertebrae assessed across all samples.

	TXSTDSC		*OpID*		*PCOME*	
Vertebra	***SN Count***	***Vert Count***	***SN Count***	***Vert Count***	***SN Count***	***Vert Count***
T1	0	88	0	106	0	56
T2	0	88	0	106	0	56
T3	0	88	0	106	1	56
T4	1	88	0	106	1	56
T5	7	90	2	106	1	56
T6	16	89	8	107	6	58
T7	29	95	29	110	9	57
T8	46	101	39	116	13	60
T9	39	97	38	116	14	61
T10	32	97	25	111	7	58
T11	54	102	26	111	7	58
T12	43	99	23	115	8	60
L1	30	95	16	111	7	59
L2	23	92	17	110	12	59
L3	12	91	15	109	9	59
L4	8	90	13	111	4	58
L5	6	89	4	107	2	56
TOTAL	**346**	**1579**	**225**	**1868**	**105**	**983**

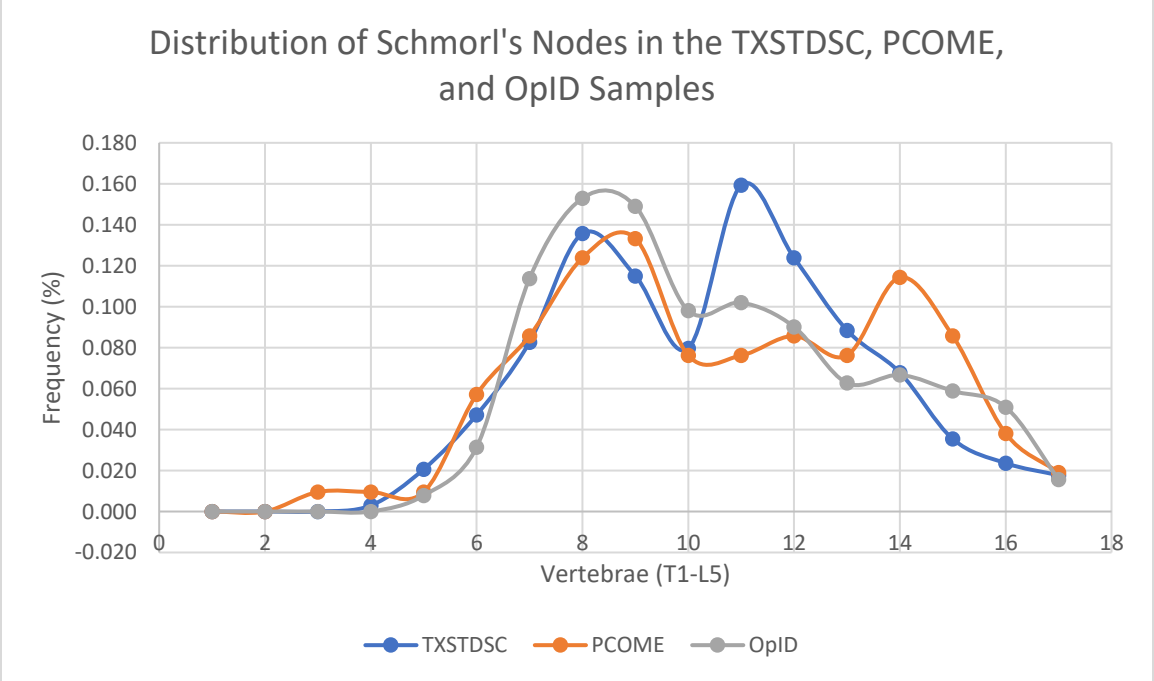

Figure 39. Distribution of SNs along the spinal column across the samples.

Texas State Donated Skeletal Collection

Prevalence and severity. In total, 346 SNs were recorded among 55 (72.37%, N = 76) individuals in the TXSTDSC sample with 21 affected individuals identifying as female (60.0%, n = 35) and 33 (80.49%, n = 41) as male (Table 8). SNs followed a bimodal distribution among both sexes (Figure 40) with females experiencing more SNs around the thoracolumbar junction (T8-L2) than males. A Chi-square statistic was completed and revealed there is no statistical significance between affected females and males in this sample, X^2 (1, N = 76) = 0.6607, p = 0.4163 at p < 0.05.

Table 8. Distribution of SNs by sex in the TXSTDSC.

	Affected	**Unaffected**	**Total**
Females	21	12	33
Males	33	8	41
Total	55	20	**76**

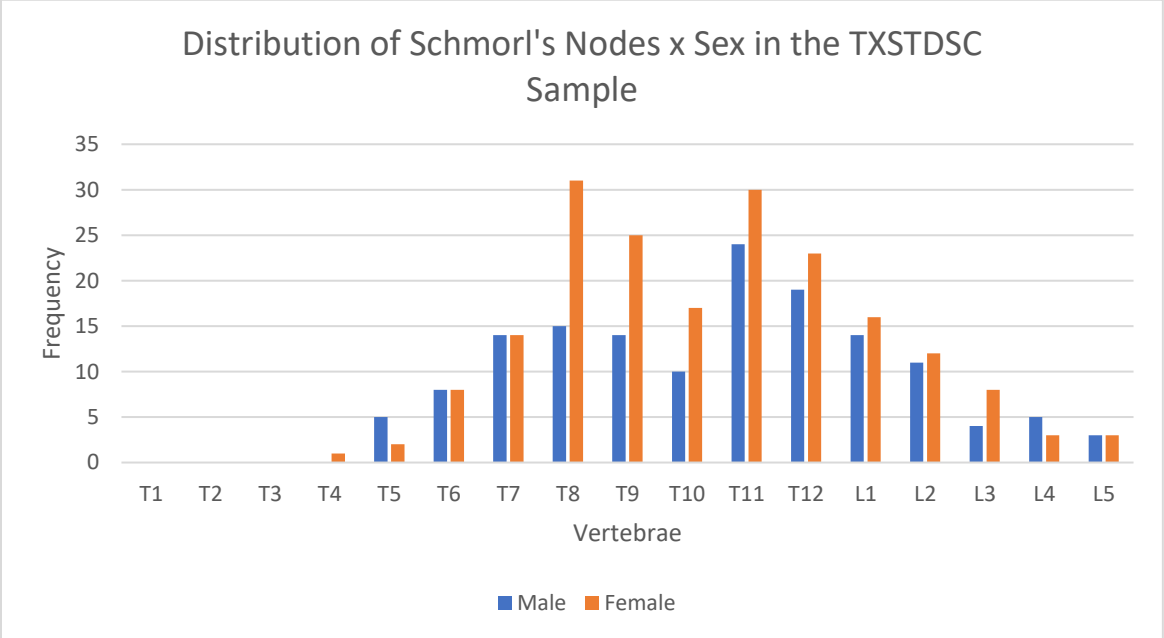

Figure 40. Distribution of SNs along the spinal column in males and females of the TXSTDSC.

Schmorl's nodes were predominately Stage 1 (n = 287) compared to Stage 2 (n = 52). While Stage 1 SNs are distributed through the spinal column, Stage 2 nodes are concentrated in the middle thoracic vertebrae (Figure 41).

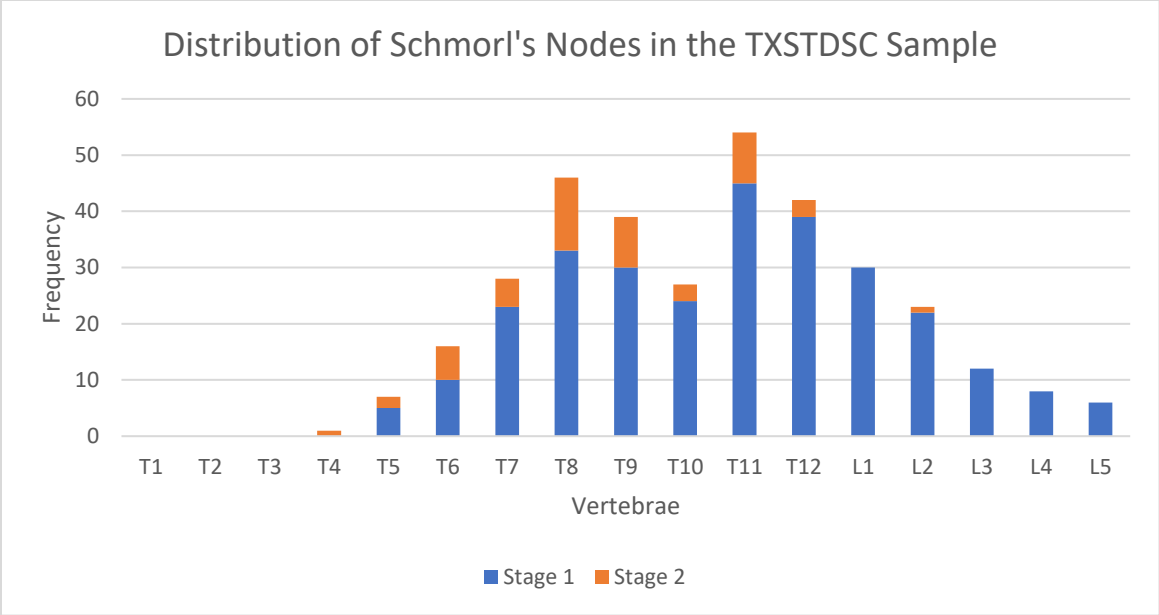

Figure 41. Distribution of SNs along the spinal column stratified by severity in the TXSTDSC sample.

Prevalence and Manual Labor. The chi-square test of independence was performed to examine the relationship between the presence/absence of SNs and manual/non-manual labor among individuals in the TXSTDSC with documented occupation information. Donations without this information were excluded from this test. The relation between these variables was not significant, X^2 (2, N = 66), p = 0.666062 at p < .05. Occupation type, as defined for the purpose of this research, does not significantly contribute to the occurrence of SNs (Table 9).

Table 9. Frequency of observed individuals from the TXSTDSC with documented occupation and the presence/absence of SNs.

	MANUAL	**NON-MANUAL**	**UNKNOWN**	**TOTAL**
NO SN	5	13	2	20
SN	16	32	7	55
TOTAL	21	45	9	75

Prevalence and SES. An ANOVA: single factor was completed to test for a significant association between presence/absence of SN and documented SES level and found no

significance, F (1, 71) = 5.318, p = 0.174. Donations that lacked documented SES information were excluded from this test.

Table 10. Frequency of observed individuals from the TXSTDSC with documented childhood SES and the presence/absence of SNs.

	LOW	LOW-MIDDLE	MIDDLE	UPPER-MIDDLE	UPPER	TOTAL
NO SN	6	7	4	6	2	25
SN	15	14	9	8	0	46
TOTAL	21	21	13	14	2	71

Operation Identification

Prevalence and sex. In total, 225 SNs were recorded among 47 individuals (45.19%, N = 104) in the OpID sample with 9 affected females (45%, n = 20) and 36 affected males (53.73%, n = 67). A chi-square test of indepence was conducted to test for a significance association between sex and presence/absence of SN and found no significant difference, , X^2 (2, N = 87), p = 0.493 at p < .05. Individuals with no estimated sex or an unknown designation were excluded from this test. While there is no statically significant difference in prevalence between females and males, there is a visible difference in the distribution of SN between the sexes (Figure 42) in which females experience SN in the lower thoracic through lumbar vertebrae and males exhibit SN primarily in the middle thoracic.

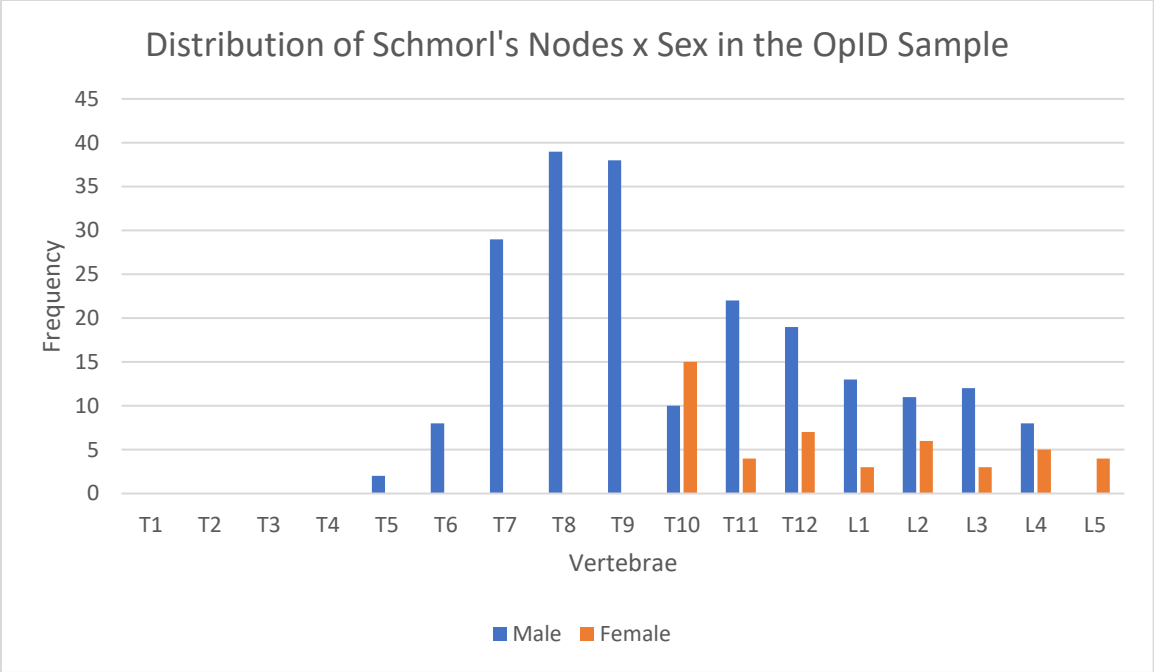

Figure 42. Distribution of SNs along the spinal column in males and females of the OpID sample.

Pima County Office of the Medical Examiner

Prevalence and sex. In total, 105 SNs were recorded among 22 individuals (68.75%, N = 32) in the PCOME sample with 1 affected females (50%, n = 2) and 21 affected males (65.63%, n = 32). A chi-square test of indepence could not be completed due to the small female sample size (n < 5). However, there is a bimodal distribution among the males, with SN increasingly present above and below the thoracolumbar junction (Figure 43).

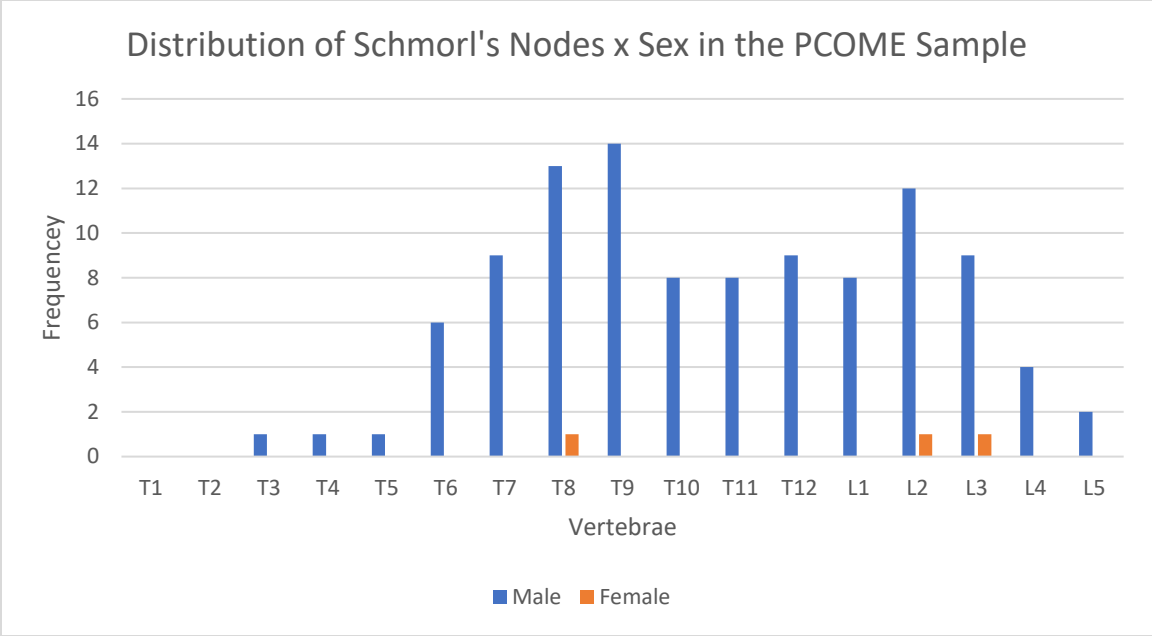

Figure 43. Figure 29. Distribution of SNs along the spinal column in males and females of the PCOME sample.

Reliability of the Knüsel et al. (1997) method. Calculating servity using Knusel et al. (year)'s method resulted in 288 of the 346 (83.24%) SNs classifying as Stage 1 (83.24%) and 58 (16.76%) SNs classifying as Stage 2. Alternatively, when the transverse measurements are used rather than the anteroposterior dimensions as used in Knusel's method, 312 (90.17%) SNs classify as Stage 1 and 34 (9.83%) SNs classify as Stage 2. A chi-square test of goodness of fit was performed to examine is these two methods resulted in similar classifications. Classifications were not similarly distributed, X^2 (2, N=346) = 0.007206, $p < .05$.

VNC AP, TR, and AP/TR Ratio Dimensions x SN Association

A biserial correlation analysis was completed to test the correlation between the presence/absence of SNs with VNC dimensional data, including VNC AP, TR and AP/TR ratio sizes, among individuals from each sample. In the TXSTDSC, T1, T2, T3, and T4 were not included in these analyses because there were limited SN that occurred

on these vertebrae. T1-T5 were not included in the analyses for the PCOME and OpID samples. The resulting coefficients (Table 11-13) indicate the strength of the association between VNC AP, TR and Ratio sizes and the presence/absence of SNs, where -1 indicates a negative association, +1 indicates a positive association, and 0 indicates no association. These was completed for every vertebra, example Figure 44.

TXSTDSC Sample. This analysis was conducted on all individuals in the TXSTDSC regardless of demographic data and the results indicate primarily weak negative correlations between the presence of SN and VNC AP, TR and ratio sizes. The presence of SNs was most strongly correlated with both the VNC AP and VNC ratio in the lower thoracic through lower lumbar vertebrae (highlighted in red in Table 11). The negative R-value indicates a negative correlation, meaning that the presence of SNs was more strongly associated with smaller VNC AP and VNC ratio values. This negative correlation can be more easily viewed in Figure 44, in which the absence of SN is associated with a larger diameter on average than the presence of SN.

Vertebrae in which the R-value indicates a positive correlation, meaning that the presence of SNs was more strongly associated with larger dimensions, included T8-T9, L4 (VNC-AP), thoracolumbar junction T12-L1 (VNC-TR), and middle thoracic T8-T10 (VNC ratio).

OpID Sample. The presence of SN was significantly correlated with larger VNC AP diameters of the middle thoracic (T7-T9), but smaller VNC TR diameters of the same region and the lumbar vertebrae (Table 12).

PCOME Sample. The presence of SN was most significantly correlated to larger AP and TR diameters in the middle thoracic (T6 – T10), but smaller AP and TR diameters in the lower vertebral column. Correlation strengths can be viewed across samples in Figure 45.

Table 11. R-value results from biserial correlation analysis between the VNC dimensional data and the presence/absence of SNs among individuals in the TXSTDSC sample.

Vertebra	*VNC-AP*	*VNC-TR*	*VNC-Ratio*
T5	**-0.189**	**0.101**	**-0.189**
T6	-0.055	**-0.151**	0.068
T7	**-0.165**	-0.092	-0.092
T8	0.076	-0.067	**0.152**
T9	0.081	0.019	0.072
T10	-0.097	-0.093	**0.124**
T11	**-0.140**	-0.091	-0.008
T12	**-0.253**	0.083	**-0.281**
L1	-0.090	0.050	**-0.137**
L2	**-0.235**	-0.063	**-0.204**
L3	**-0.370**	**-0.255**	**-0.223**
L4	**0.315**	-0.010	**0.313**
L5	**-0.237**	**-0.128**	**-0.150**

Table 12. R-value results from biserial correlation analysis between the VNC dimensional data and the presence/absence of SNs among individuals in the OpID sample.

Vertebra	*VNC-AP*	*VNC-TR*	*VNC-Ratio*
T6	0.065	**-0.143**	**0.205**
T7	**0.123**	**-0.105**	**0.164**
T8	**0.182**	**-0.106**	0.006
T9	**0.198**	**0.115**	0.035
T10	0.056	**0.133**	-0.082
T11	0.041	-0.040	0.079
T12	-0.043	0.025	-0.024
L1	0.035	-0.055	-0.056
L2	0.084	**-0.108**	0.021
L3	0.039	**-0.167**	-0.025
L4	0.048	**-0.216**	**0.106**
L5	0.090	**-0.106**	**-.120**

Table 13. R-value results from biserial correlation analysis between the VNC dimensional data and the presence/absence of SNs among individuals in the PCOME sample.

Vertebra	*VNC-AP*	*VNC-TR*	*VNC-Ratio*
T6	**0.552**	**0.275**	**0.263**
T7	**0.187**	**0.232**	-0.021
T8	**0.179**	**0.100**	0.043
T9	0.005	**0.166**	**-0.227**
T10	**0.199**	0.006	**0.154**
T11	0.083	**-0.301**	**0.375**
T12	**-0.169**	0.053	**-0.227**
L1	-0.016	0.035	-0.083
L2	0.012	0.069	-0.069
L3	**-0.125**	0.001	**-0.167**
L4	**-0.124**	0.019	**-0.151**
L5	-0.039	-0.079	0.028

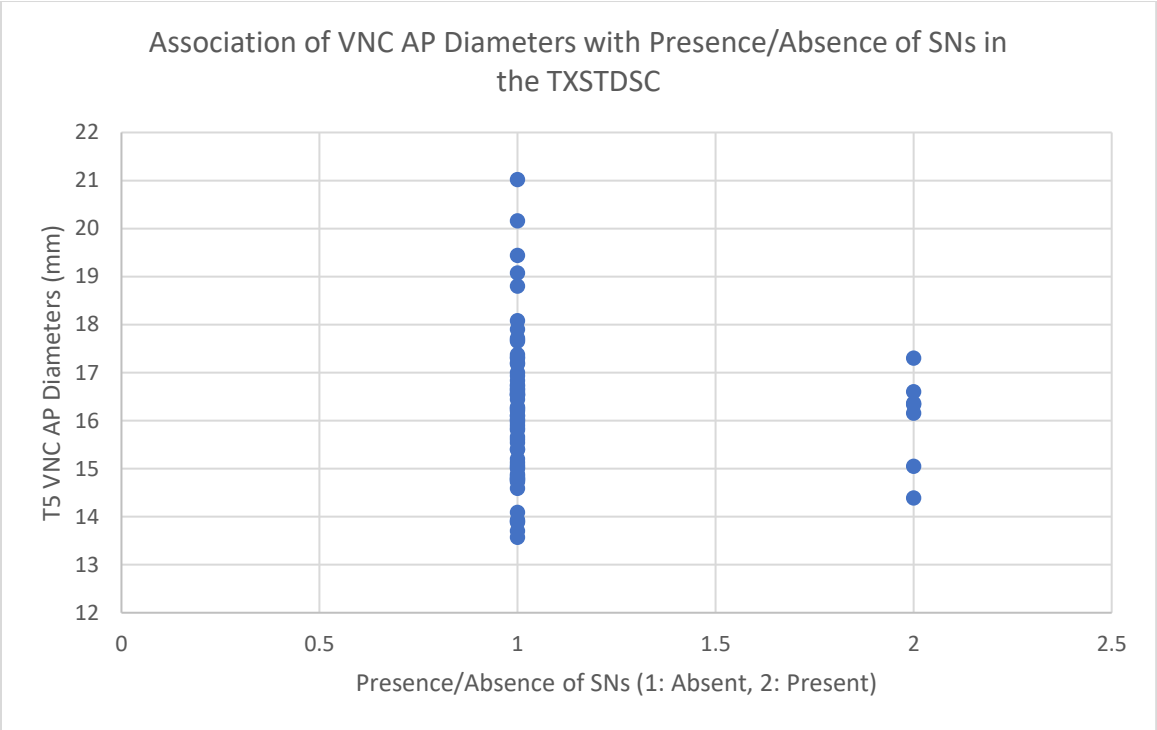

Figure 44. Testing the correlation between presence/absence of SN and VNC AP diameter in the T5 vertebrae of individuals in the TXSTDSC.

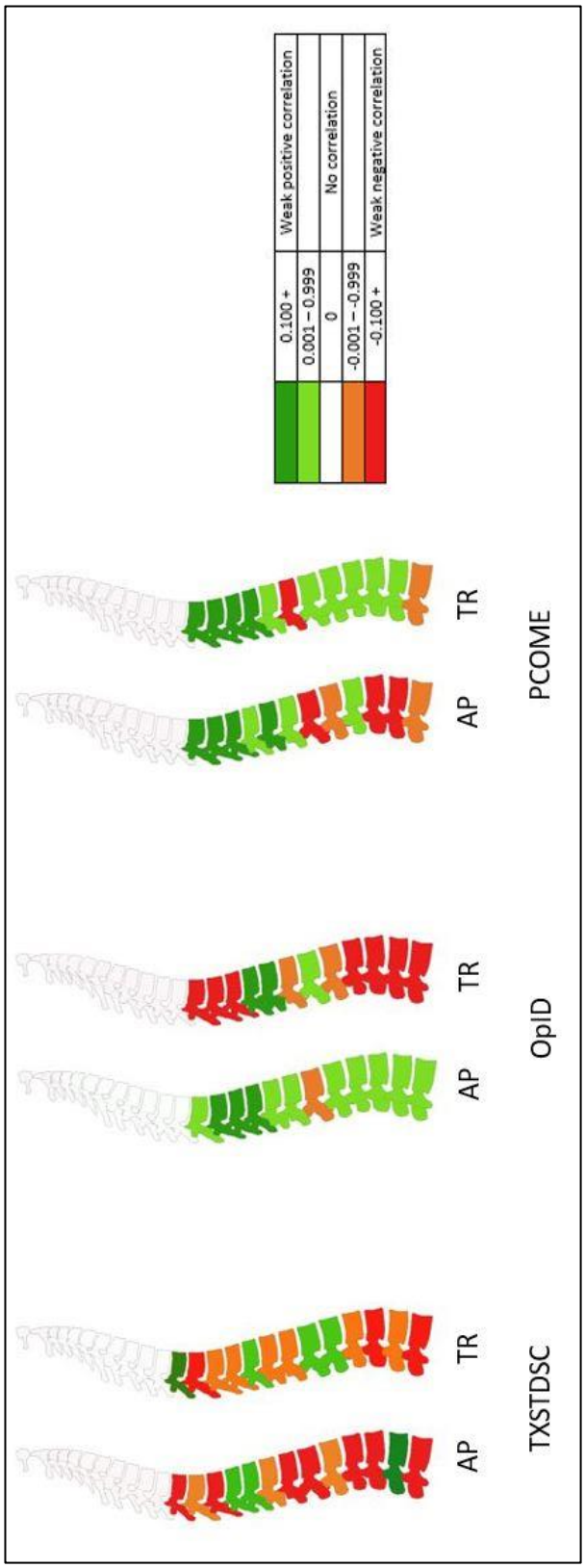

Figure 45. Correlation between VNC dimensions and presence of SNs among the samples.

V. DISCUSSION

Overall, the results from this research indicate that differences in development impact how life history manifests. More specifically, the analysis of vertebral neural canal (VNC) asymmetry and Schmorl's nodes (SNs) suggests that increased stress resulting in developmental plasticity of vertebral elements results in a predisposition to increased susceptibility of other vertebral pathologies in adulthood. The analysis of VNC asymmetry and SNs among modern American nationals from the Texas State Donated Skeletal Collection (TXSTDSC) and unidentified and presumed migrants curated under the Operation Identification (OpID) project and at the Pima County Office of the Medical Examiner (PCOME) further suggests that developmental plasticity and resulting adult health trajectories, as related to the spinal column, embody differently depending on the biosocial context in which an individual lived. The remainder of this chapter will discuss the findings of this research in more detail as related to research questions presented in Chapter I (Introduction). Following this discussion, broader implications and future directions of this research will be presented and considered.

Vertebral Neural Canal Asymmetry

1. Does VNC asymmetry significantly correlate with sex? Differences between the VNC diameters of individuals from the three samples could indicate several things. Males, followed by females, in the TXSTDSC sample exhibited the largest average VNC AP diameters, followed by PCOME males, PCOME females, OpID males, and OpID females. While this difference was significant in the lumbar vertebrae between females and males in the TXSTDSC and OpID samples, there was no significant difference between females and males in the PCOME. Females and males from the same samples

exhibited the most similar VNC AP diameters, with the exception of females and males in the PCOME sample where females appear to have a lower outlier in the middle thoracic vertebrae compared to their male counterparts. However, this is likely due to small sample size (n = 2). Otherwise, in each sample, females generally have smaller AP diameters in the upper and lower thoracic vertebrae but are generally larger in the middle thoracic and lumbar vertebrae. Despite these visual differences, the AP diameters were only statistically significant different between the sexes in L2-L3 in the TXSTDSC and L4-L5 in the OpID sample.

Embodiment of stress in the VNC AP diameters, which reach maturation throughout the entire spinal column by 5 years of age, can be indicative of acute periods of stress in infancy and early childhood, typically from biological pathologies and malnutrition, as well as more chronic stressors, such as low SES. The lack of difference between the sexes indicates that females and males likely experienced these acute periods of stress similarly in infancy and early childhood, a period in which their immune systems were developing regardless of sex. Various types of stress experienced during this time that impact energy allotment for skeletal development would indirectly stunt the growth of VNC AP diameters. For individuals from all samples, low socioeconomic status, malnutrition, and lack of access to healthcare could all contribute to this developmental disruption regardless of sex.

The distribution of VNC TR diameters by sex appears more systematic than the VNC AP diameters, with males consistently exhibiting larger diameters in each sample. Differences in the TR diameters between samples follow this a similar pattern to the AP diameters, the only difference being that the OpID and PCOME males are more similar

and larger than the females from these samples. While females and males are grouped closely in all three samples, with females on average smaller than males, this difference was only significant in the TXSTDSC and OpID samples. The TR diameters were statistically significantly different between females and males in L1-L3 in the TXSTDSC while they were significantly different in T2-T10, T12-L2 in the OpID sample.
The size differences between females and males could be a consequence of sexual dimorphism. However, when size was controlled for by analyzing the VNC AP/TR ratio, it was commonly found that the ratio was significant alongside significant VNC AP and TR diameters, meaning that there was overall a significant difference in asymmetry between the sexes.

The TR diameter reaches maturation later than the AP diameter meaning that embodiments of stress in the VNC TR diameters are indicative of chronic periods of stress experienced through adolescence. Acute periods of stress are likely not embodied in this larger window of time if the individual is otherwise healthy. Significant difference of the VNC TR diameter between males and females indicates a difference in later childhood stress experiences. In light of known gender inequality across the samples, this difference could reflect the embodiment of gendered inequality. More specifically, this difference in the OpID sample could be indicative of differential gender experiences in a society were females start working at a younger age than males (personally communication, Yasmín Díaz, 2019).

Occupation was not explicitly tested in relation to VNC dimensions due to the assumption that VNC are directly related to developmental processes and experiences in

early childhood, whereas occupation would not have been an influence on embodiment until later in life.

2. Does VNC asymmetry significantly correlate with self-reported childhood socioeconomic status (SES) in the TXSTDSC? The assessment of VNC AP diameters by SES in the TXSTDSC revealed significant size differences between the levels with larger average AP diameters in higher SES levels compared to lower average AP diameters in lower SES levels. The assessment of VNC TR diameters and VNC AP/TR ratios were also found to be significantly different between the SES levels, with larger dimensions VNC dimensions in the higher SES levels and smaller dimensions in the smaller SES levels. These results indicate that the TXSTDSC exhibits socioeconomic inequalities in which lower SES contributes to increased stress during both early childhood and adolescence as shown in the VNC AP and TR diameters respectively. A number of influences associated with lower SES, e.g., lack of medical care, poor quality food, etc., could contribute to this stress which disrupted VNC development.

Interestingly, VNC asymmetry in the SES levels commonly overlapped among documented Americans in the TXSTDSC. This could be indicative of how arbitrary levels fail to highlight the variation of experiences within each SES level. However, the VNC dimensions of lower SES individuals in the TXSTDSC was comparable to both the PCOME and OpID individuals, with smaller average VNC AP and TR diameters among unidentified migrants than the upper SES levels in the TXSTDSC. These results indicate that the unidentified migrants experienced stress, even more so, than the documented Americans of low SES, and that this was embodied through smaller VNC sizes.

Schmorl's Nodes (SNs)

3. Do SNs significantly correlate with sex? SNs were found to be more prevalent among males in comparison to females in both the TXSTDSC and the OpID samples, but with not statistically significant difference. Significance was not tested in the PCOME sample because of the small female sample size. Regardless of sex, the distribution of SNs followed specific patterns in the three samples. In the TXSTDSC and PCOME samples, SNs followed a bimodal distribution, while in the OpID sample, they exhibited an increased presence in the middle thoracic.

These results do not support the proposed hypotheses that SNs result strictly from biomechanical forces and increasing load pressures around the thoracolumbar junction and lumbar vertebrae. If this were the case, as argued in previous studies, the distribution of SNs in all samples would be opposite and exhibit a right skewed distribution. Due to the systematic patterns in distributions, the presence of SNs is likely not due to differences in gendered experiences or traumatic injury.

4. Do SNs significantly correlate with self-reported occupation type in the TXSTDSC? The presence and severity of SNs was also not found to be related to occupation type in the TXSTDSC, with no significant difference between the presence/absences and severity of SNs and self-reported manual labor positions versus non-manual positions. These findings are significant, because while SNs have traditionally been utilized by bioarchaeologists to be indicators of traumatic injury, these findings further suggest that the presence of SNs should be interpreted in conjunction with morphological growth and development data, i.e. VNC asymmetry.

VNC Asymmetry and SN Correlation

5. Are VNC asymmetry and SNs significantly associated? In general, the strongest R-values from the biserial correlation analysis indicate that the presence of SNs is most significantly associated with smaller VNC dimensions (AP, TR, and AP/TR ratio). The presence of SNs was more commonly associated with the AP widths and the AP/TR ratio than the TR widths, with the AP and ratio patterns nearly mirroring each other.

In accordance with DOHaD, these results indicate that the presence of SNs is significantly correlated with earlier episodes of stress rather than later episodes of stress as captured in the AP and TR diameters respectively. The mirroring of these results in the AP/TR ratio, which accounts for potential bias in size difference due to sexual dimorphism, further supports this interpretation. This correlation can be interpreted to signify that the embodiment of stress in the vertebrae in both infancy/early childhood and adolescence contributed to smaller VNC dimensions, and that this was associated with the presence of SNs later in life.

6. How do patterns in VNC and SNs compare between the three samples? When assessing the correlation between VNC asymmetry and SNs between the VNC AP and TR diameters, a change over time can be seen in each of the samples (Figure 45). In the TXSTDSC, both smaller VNC AP and TR diameters around the thoracolumbar junction is correlated with the presence of SN, but less so in the TR diameters. This indicates that there was more stress embodied in the AP diameters during early childhood than later in the TR diameters through adolescence.

While this change over time is visible in the TXSTDSC however, the difference between VNC AP and TR diameters and their correlation with SNs is more significant in

the OpID and PCOME samples. In the OpID sample, there is nearly no correlation between the VNC AP diameter and presence of SNs. However, there is a correlation between decreased VNC TR diameters and SNs. This indicates that there was a change in the stress experience over time, with stress increasing through adolescence and resulting in disruptions to VNC TR diameters. The correlation between smaller TR diameters and presence of SNs in both the middle thoracic and lumbar vertebrae shows that this correlation is significant beyond the biomechanical influences alone (which would be experienced the most in the lumbar vertebrae). The PCOME sample exhibits a pattern opposite that of the OpID sample, in which smaller VNC AP were correlated with the presence of SNs while there is no correlation with the TR diameter. This indicates a change in the stress experienced over time, with high levels of stress in infancy and early childhood, seen in the AP diameters, decreasing over time in adolescence, seen in the TR diameters. These results further suggest that developmental plasticity and resulting adult health trajectories, as related to the spinal column, embody differently depending on the biosocial context in which an individual lived.

Broader Implications

In addition to other skeletal indicators of stress that have been traditionally utilized to examine developmental plasticity, e.g., linear enamel hypoplasia and stature, this research suggests that VNC asymmetry is another reliable marker to assess developmental plasticity. VNC asymmetry can be used in conjunction with other pathologies for more longitudinal studies to better investigate how the DOHaD Hypothesis correlates with these markers. While epigenetics in bioarchaeology have been examined through a focus on macroscopic, nonmetric skeletal variants to study kinship,

population movement, and biodistance, a focus on indicators of stress could be an alternative mode of research. This research further supports the proposition that SNs have a developmental or epigenetic origin, and that they should be viewed as such considering the DOHaD Hypothesis and Life Course Theory.

Future Directions

Future directions with this research should include a genetic component and would preferably consist of a diachronic assessment of an intact population that has undergone trauma, e.g., migration, sociopolitical violence, etc. In this way, a direct assessment can be made on the reliability of skeletal indicators of stress as proxies for the underlying epigenome. The results of this research would improve recognition and interpretation of skeletal markers of stress as embodied on the migrant skeleton, for example, by shedding light on potential hidden heterogeneity in embodiment pathways. In addition to bodies found along the US-Mexico border, these SN and VNC stress profiles may have applicability in other regions experiencing large numbers of migrant deaths, such as the Mediterranean, providing insight into area of origin and expediting the identification process. The right of the dead to be identified is a declared human right by the United Nations. Improving identification services will assist governmental compliance when it comes to positive identification of the migrant dead, saving time and money in this process, as well as protecting cultural memory and heritage through repatriation of the deceased.

APPENDIX

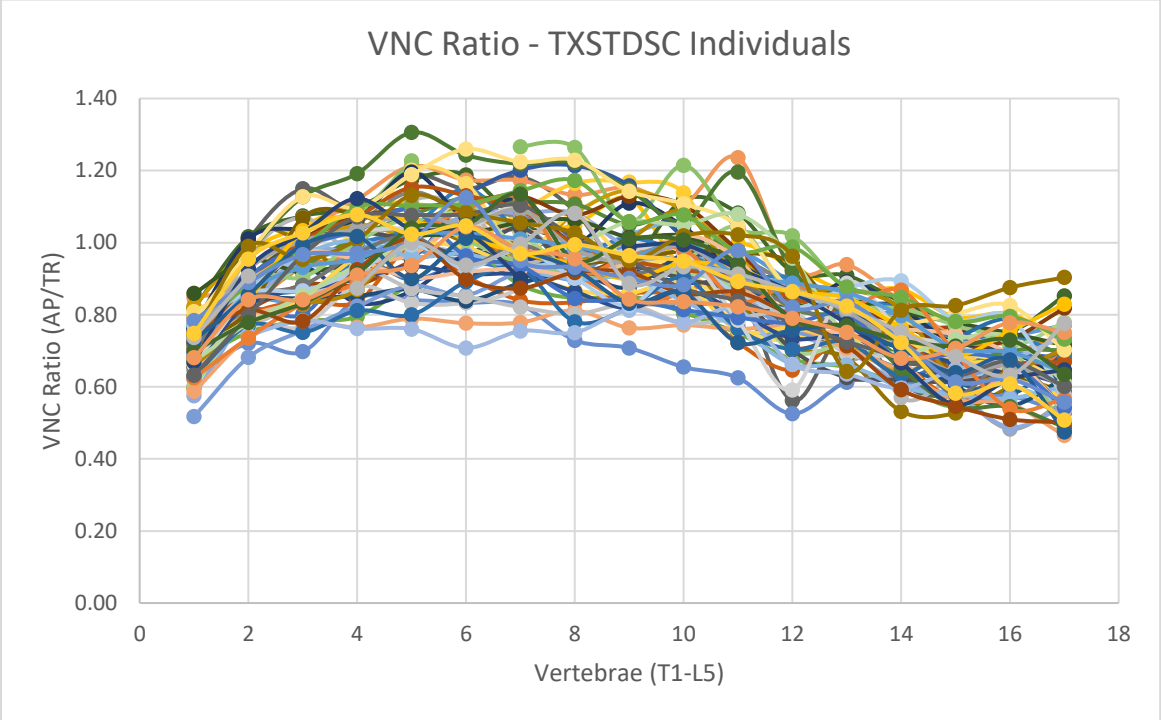

Figure 46. VNC ratios along the spinal column (T1-L5) from all individuals in the TXSTDSC sample.

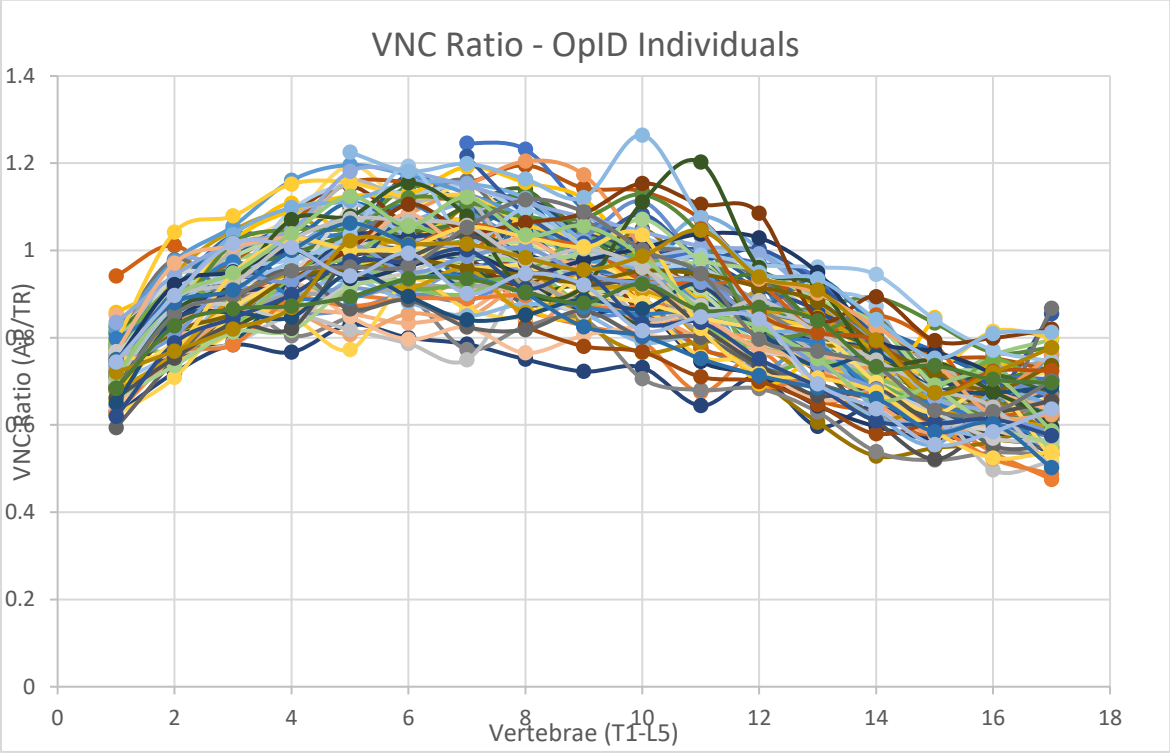

Figure 47. VNC ratios along the spinal column (T1-L5) from all individuals in the OpID sample.

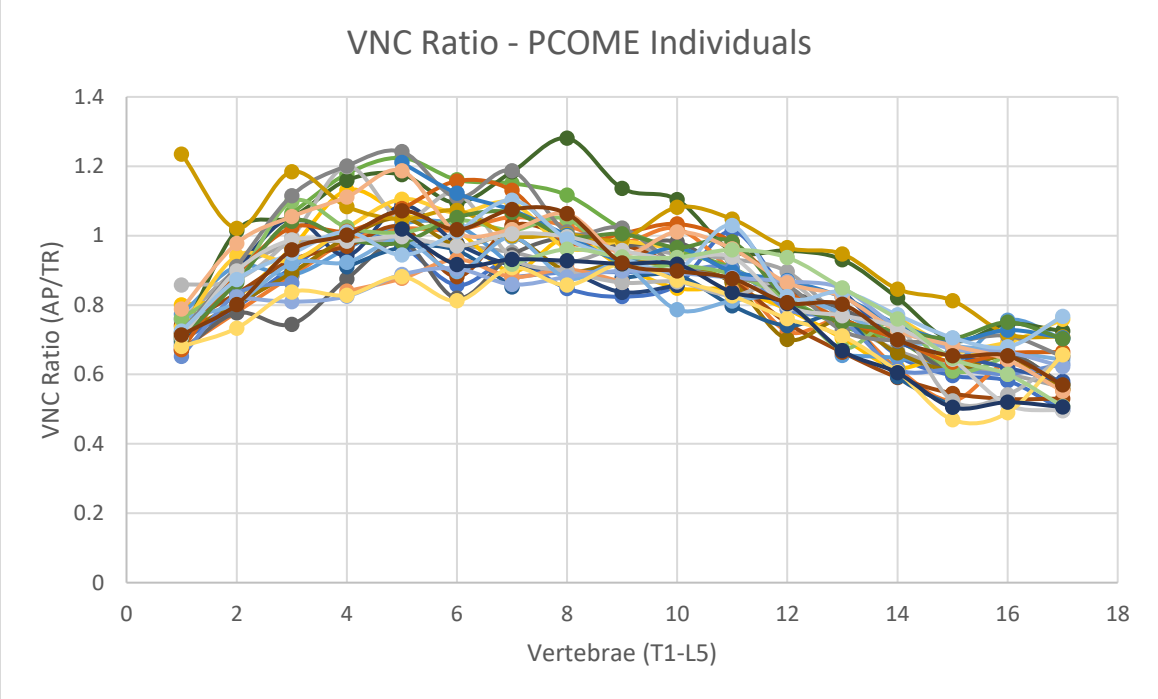

Figure 48. VNC ratios along the spinal column (T1-L5) from all individuals in the PCOME sample.

Table 14. Results from the F-Test Two-Sample for Variances and t-Test: Two-Sample Assuming (Un)Equal Variances tests for each VNC AP diameter (T1-L5) in the TXSTDSC.

Vertebra	**Test**	**Significance**
T1	F-Test Two-Sample for Variances. t-Test: Two-Sample Assuming Unequal Variances.	Significant, F (32, 35) = 0.4918, **p = 0.0245.** Not significant, females (M = 15.019, SD = 1.2719) and males (M = 15.219, SD = 2.5865), t (-0.591) = 1.9996, p = 0.5568.
T2	F-Test Two-Sample for Variances. t-Test: Two-Sample Assuming Unequal Variances.	Significant, F (32, 36) = 0.5256, **p = 0.0362.** Not significant, females (M = 15.376, SD = 1.1389) and males (M = 15.718, SD = 2.1668), t (-1.104) = 1.9977, p = 0.2739.
T3	F-Test Two-Sample for Variances. t-Test: Two-Sample Assuming Unequal Variances.	Significant, F (32, 34) = 0.4192, **p = 0.0085.** Not significant, females (M = 15.689, SD = 1.2957) and males (M = 15.906, SD = 3.0908), t (-0.599) = 2.0025, p = 0.5516.
T4	F-Test Two-Sample for Variances. t-Test: Two-Sample Assuming Unequal Variances.	Significant, F (32, 36) = 0.4668, **p = 0.017.** Not significant, females (M = 15.894, SD = 1.3711) and males (M = 15.906, SD = 2.9374), t (-0.033) = 1.999, p = 0.9736.
T5	F-Test Two-Sample for Variances. t-Test: Two-Sample Assuming Unequal Variances.	Significant, F (32, 35) = 0.5111, **p = 0.0311.** Not significant, females (M = 16.293, SD = 1.5373) and males (M = 16.123, SD = 3.0079), t (0.4628) = 1.999, p = 0.6452.
T6	F-Test Two-Sample for Variances. t-Test: Two-Sample Assuming Unequal Variances.	Not significant, F (30, 36) = 0.5546, p = 0.0537. Not significant, females (M = 16.118, SD = 1.476) and males (M = 15.964, SD = 2.6612), t (0.4279) = 1.9977, p = 0.6702.
T7	F-Test Two-Sample for Variances. t-Test: Two-Sample Assuming Equal Variances.	Not significant, F (32, 36) = 0.725, p = 0.1833. Not significant, females (M = 16.114, SD = 2.4788) and males (M = 15.897, SD = 3.4188), t (0.5173) = 1.9966, p = 0.6067.
T8	F-Test Two-Sample for Variances. t-Test: Two-Sample Assuming Unequal Variances.	Not significant, F (32, 35) = 0.8863, p = 0.3688. Not significant, females (M = 15.73, SD = 2.4624) and males (M = 15.884, SD = 2.7782), t (-0.338) = 1.9971, p = 0.6989.
T9	F-Test Two-Sample for Variances. t-Test: Two-Sample Assuming Equal Variances.	Not significant, F (32, 36) = 0.6752, p = 0.1351. Not significant, females (M = 15.604, SD = 15.795) and males (M = 15.975, SD = 2.938), t (-0.499) = 1.9966, p = 0.6195.
T10	F-Test Two-Sample for Variances. t-Test: Two-Sample Assuming Equal Variances.	Not significant, F (32, 36) = 0.8701, p = 0.3489. Not significant, females (M = 15.676, SD = 15.782) and males (M = 15.782, SD = 2.4117), t (-0.29) = 1.9966, p = 0.7728.

Table 14. Continued. Results from the F-Test Two-Sample for Variances and t-Test: Two-Sample Assuming (Un)Equal Variances tests for each VNC AP diameter (T1-L5) in the TXSTDSC.

Vertebra	**Test**	**Significance**
T11	F-Test Two-Sample for Variances. t-Test: Two-Sample Assuming Equal Variances.	Not significant, F (32, 36) = 0.7702, p = 0.2318. Not significant, females (M = 15.856, SD = 2.2193) and males (M = 16.137, SD = 2.8815), t (-0.721) = 1.9966, p = 0.4736.
T12	F-Test Two-Sample for Variances. t-Test: Two-Sample Assuming Equal Variances.	Not significant, F (32, 36) = 2.0627, p = 1.7793. Not significant, females (M = 16.848, SD = 4.2119) and males (M = 17.065, SD = 2.0419), t (-0.512) = 1.9966, p = 0.6106.
L1	F-Test Two-Sample for Variances. t-Test: Two-Sample Assuming Equal Variances.	Not significant, F (32, 37) = 0.5219, p = 0.0341. Not significant, females (M = 17.418, SD = 1.2698) and males (M = 17.17, SD = 2.4328), t (0.766) = 1.9971, p = 0.4465.
L2	F-Test Two-Sample for Variances. t-Test: Two-Sample Assuming Equal Variances.	Not significant, F (32, 37) = 0.5715, p = 0.0578. Significant, females (M = 16.848, SD = 1.9549) and males (M = 15.735, SD = 3.4208), t (2.7838) = 1.996, **p = 0.007.**
L3	F-Test Two-Sample for Variances. t-Test: Two-Sample Assuming Equal Variances.	Not significant, F (32, 37) = 0.9337, p = 0.4256. Significant, females (M = 16.136, SD = 3.3662) and males (M = 15.087, SD = 3.6051), t (2.3242) = 1.996, **p = 0.0232**.
L4	F-Test Two-Sample for Variances. t-Test: Two-Sample Assuming Equal Variances.	Not significant, F (31, 37) = 0.7717, p = 0.2354. Not significant, females (M = 16.345, SD = 3.6743) and males (M = 15.787, SD = 4.7616), t (1.1084) = 1.9966, p = 0.2717.
L5	F-Test Two-Sample for Variances. t-Test: Two-Sample Assuming Equal Variances.	Not significant, F (31, 35) = 0.7928, p = 0.2608. Not significant, females (M = 17.218, SD = 6.1415) and males (M = 16.985, SD = 7.747), t (0.3585) = 1.9977, p = 0.7212.

Table 15. ANOVA results from average VNC AP diameters across all three samples.

Anova: Two-Factor Without Replication

SUMMARY	*Count*	*Sum*	*Average*	*Variance*		
TXSTDSC	17	273.249	16.07347	0.326461		
PCOME	17	257.43	15.14294	0.480422		
OpID	17	252.505	14.85324	0.51383		
T1	3	43.519	14.50633	0.3737		
T2	3	43.981	14.66033	0.6925		
T3	3	44.784	14.928	0.815689		
T4	3	45.328	15.10933	0.661269		
T5	3	46.271	15.42367	0.694892		
T6	3	45.831	15.277	0.483997		
T7	3	46.381	15.46033	0.228102		
T8	3	46.249	15.41633	0.126332		
T9	3	45.715	15.23833	0.179554		
T10	3	45.449	15.14967	0.27869		
T11	3	46.778	15.59267	0.161365		
T12	3	49.714	16.57133	0.138876		
L1	3	49.989	16.663	0.315817		
L2	3	46.285	15.42833	0.531864		
L3	3	43.499	14.49967	0.961542		
L4	3	45.125	15.04167	0.801464		
L5	3	48.286	16.09533	0.794556		
ANOVA						
Source of Variation	*SS*	*df*	*MS*	*F*	*P-value*	*F crit*
Rows	13.8198	2	6.909901	83.10711	2.13E-13	3.294537
Columns	18.47078	16	1.154424	13.88454	4.05E-10	1.971683
Error	2.660625	32	0.083145			
Total	34.95121	50				

Table 16. ANOVA results from average VNC TR diameters across all three samples.

Anova: Two-Factor Without Replication

SUMMARY	***Count***	***Sum***	***Average***	***Variance***		
TXSTDSC	17	329.367	19.37453	12.73912		
PCOME	17	307.22	18.07176	9.89564		
OpID	17	300.013	17.64782	8.731761		
T1	3	59.699	19.89967	1.689876		
T2	3	50.986	16.99533	1.10555		
T3	3	47.775	15.925	0.815269		
T4	3	46.181	15.39367	0.71941		
T5	3	45.71	15.23667	0.442217		
T6	3	45.735	15.245	0.261849		
T7	3	46.401	15.467	0.238197		
T8	3	47.244	15.748	0.275404		
T9	3	48.063	16.021	0.293863		
T10	3	48.2	16.06667	0.340854		
T11	3	52.072	17.35733	0.477701		
T12	3	60.024	20.008	1.292224		
L1	3	64.421	21.47367	0.628452		
L2	3	64.806	21.602	1.057852		
L3	3	65.817	21.939	1.509417		
L4	3	68.059	22.68633	1.839092		
L5	3	75.407	25.13567	3.15394		
ANOVA						
Source of Variation	*SS*	*df*	*MS*	*F*	*P-value*	*F crit*
Rows	27.53113	2	13.76557	92.71289	4.85E-14	3.294537
Columns	497.1132	16	31.06957	209.2576	9.59E-28	1.971683
Error	4.751207	32	0.148475			
Total	529.3955	50				

Table 17. ANOVA results from average VNC AP/TR ratios across all three samples.

Anova: Two-Factor Without Replication

SUMMARY	***Count***	***Sum***	***Average***	***Variance***		
TXSTDSC Overall	17	14.5601	0.856476	0.018479		
PCOME	17	14.58	0.857647	0.021194		
OPID	17	14.5929	0.858406	0.017791		
T1	3	2.1923	0.730767	0.000354		
T2	3	2.584	0.861333	6.03E-05		
T3	3	2.8197	0.9399	0.000758		
T4	3	2.9498	0.983267	0.00056		
T5	3	3.0596	1.019867	0.000365		
T6	3	3.0303	1.0101	9.37E-05		
T7	3	3.0162	1.0054	3.71E-05		
T8	3	2.9437	0.981233	6.76E-05		
T9	3	2.8538	0.951267	0.000121		
T10	3	2.8166	0.938867	7.75E-05		
T11	3	2.6908	0.896933	2E-05		
T12	3	2.4752	0.825067	0.000255		
L1	3	2.337	0.779	8.1E-05		
L2	3	2.1468	0.7156	0.000204		
L3	3	1.9659	0.6553	0.000484		
L4	3	1.9596	0.6532	0.000164		
L5	3	1.8917	0.630567	8.84E-05		
ANOVA						
Source of Variation	*SS*	*df*	*MS*	*F*	*P-value*	*F crit*
Rows	3.21E-05	2	1.61E-05	0.068081	0.93432	3.294537
Columns	0.91187	16	0.056992	241.5759	9.91E-29	1.971683
Error	0.007549	32	0.000236			
Total	0.919451	50				

Table 18. ANOVA results from average VNC AP/TR ratios x sex across all three samples.

Anova: Two-Factor Without Replication

SUMMARY	***Count***	***Sum***	***Average***	***Variance***		
TXSTDSC Female	17	14.23507	0.837357	0.015216		
TXSTDSC Male	17	14.85794	0.873997	0.021469		
PCOME Female	17	15.99488	0.940875	0.021005		
PCOME Male	17	14.4506	0.850035	0.021308		
OpID Female	17	14.98249	0.881323	0.017715		
OpID Male	17	14.40719	0.847482	0.018102		
T1	6	4.562913	0.760485	0.009068		
T2	6	5.24657	0.874428	0.001632		
T3	6	5.769944	0.961657	0.005313		
T4	6	5.950243	0.991707	0.001364		
T5	6	6.149149	1.024858	0.001088		
T6	6	6.185163	1.030861	0.00271		
T7	6	6.105672	1.017612	0.001501		
T8	6	5.893901	0.982317	0.000584		
T9	6	5.777813	0.962969	0.000904		
T10	6	5.747424	0.957904	0.002698		
T11	6	5.485568	0.914261	0.002503		
T12	6	5.026751	0.837792	0.001421		
L1	6	4.763048	0.793841	0.001059		
L2	6	4.381677	0.73028	0.001039		
L3	6	4.039026	0.673171	0.001168		
L4	6	3.985473	0.664246	0.000502		
L5	6	3.857835	0.642972	0.00097		
ANOVA						
Source of Variation	***SS***	***df***	***MS***	***F***	***P-value***	***F crit***
Rows	0.12101	5	0.024202	34.20064	1.56E-18	2.328721
Columns	1.780423	16	0.111276	157.2478	1.81E-53	1.771557
Error	0.056612	80	0.000708			
Total	1.958045	101				

Table 19. Results of ANOVA testing for significance differences between average VNC AP diameters and sex and grouped SES levels (i.e., female-lower SES, female-upper SES, male-lower SES, male-higher SES) in the TXSTDSC.

Anova: Two-Factor Without Replication

SUMMARY	*Count*	*Sum*	*Average*	*Variance*		
TXST-Low-Middle	17	14.181	0.834176	0.016649		
TXST-Middle	17	14.768	0.868706	0.020042		
TXST-Upper-Middle	17	14.7562	0.868012	0.018053		
TXST-Upper	17	15.501	0.911824	0.02399		
T1	4	2.862	0.7155	0.000111		
T2	4	3.477	0.86925	0.001182		
T3	4	3.777	0.94425	0.00156		
T4	4	3.956	0.989	0.001931		
T5	4	4.1282	1.03205	0.002368		
T6	4	4.181	1.04525	0.002612		
T7	4	4.099	1.02475	0.001916		
T8	4	3.934	0.9835	0.000892		
T9	4	3.874	0.9685	0.001804		
T10	4	3.906	0.9765	0.004428		
T11	4	3.631	0.90775	0.001289		
T12	4	3.219	0.80475	0.000367		
L1	4	3.189	0.79725	0.002136		
L2	4	2.923	0.73075	0.000477		
L3	4	2.742	0.6855	0.000762		
L4	4	2.721	0.68025	0.000424		
L5	4	2.587	0.64675	0.001346		
ANOVA						
Source of Variation	*SS*	*df*	*MS*	*F*	*P-value*	*F crit*
Rows	0.051617	3	0.017206	32.77449	1.12E-11	2.798061
Columns	1.234543	16	0.077159	146.9765	3.87E-35	1.859167
Error	0.025199	48	0.000525			
Total	1.311359	67				

Table 20. Results from the F-Test Two-Sample for Variances and t-Test: Two-Sample Assuming (Un)Equal Variances tests for each VNC TR diameter (T1-L5) in the TXSTDSC.

Vertebra	**Test**	**Significance**
T1	F-Test Two-Sample for Variances. t-Test: Two-Sample Assuming Unequal Variances.	Not significant, F (32, 35) = 0.3740, **p = 0.0034.** Not significant, females (M = 17.8941, SD = 1.1428) and males (M = 18.4075, SD = 1.8823), t (-1.7307) = 1.997, p = 0.0882.
T2	F-Test Two-Sample for Variances. t-Test: Two-Sample Assuming Equal Variances.	Not significant, F (32, 36) = 0.60703, p = 0.0809. Not significant, females (M = 16.7388, SD = 1.36277) and males (M = 17.1494, SD = 2.21375), t (-1.26236) = 1.9977, p = 0.2114.
T3	F-Test Two-Sample for Variances. t-Test: Two-Sample Assuming Equal Variances.	Not significant, F (32, 35) = 0.6156, p = 0.0879. Not significant, females (M = 16.7388, SD = 1.3628) and males (M = 17.1494, SD = 2.2138), t (-1.2488) = 1.997, p = 0.2162.
T4	F-Test Two-Sample for Variances. t-Test: Two-Sample Assuming Equal Variances.	Not significant, F (32, 36) = 0.456, p = 0.0145. Not significant, females (M = 32, 35, SD = 1.0780) and males (M = 16.5381, SD = 2.3641), t (-1.1429) = 1.9989, p = 0.2575.
T5	F-Test Two-Sample for Variances. t-Test: Two-Sample Assuming Equal Variances.	Not significant, F (32, 35) = 0.5403, p = 0.0434. Not significant, females (M = 15.7584, SD = 1.2436) and males (M = 16.2303, SD = 2.3019), t (-1.4587) = 1.9989, p = 0.1497.
T6	F-Test Two-Sample for Variances. t-Test: Two-Sample Assuming Unequal Variances.	Not significant, F (30, 36) = 0.4629, **p = 0.0183.** Not significant, females (M = 15.6403, SD = 1.3539) and males (M = 16.0089, SD = 2.9244), t (-1.0368) = 1.9989, p = 0.3039.
T7	F-Test Two-Sample for Variances. t-Test: Two-Sample Assuming Unequal Variances.	Not significant, F (32, 36) = 0.4158, **p = 0.00753.** Not significant, females (M = 15.925, SD = 1.2541) and males (M = 16.0903, SD = 3.01584), t (-0.4704) = 2.0003, p = 0.6397.
T8	F-Test Two-Sample for Variances. t-Test: Two-Sample Assuming Unequal Variances.	Not significant, F (32, 36) = 0.4329, p = 0.01009. Not significant, females (M = 16.2725, SD = 1.5849) and males (M = 16.3894, SD = 3.6612), t (-0.3007) = 1.9996, p = 0.7647.

Table 20. Continued. Results from the F-Test Two-Sample for Variances and t-Test: Two-Sample Assuming (Un)Equal Variances tests for each VNC TR diameter (T1-L5) in the TXSTDSC.

Vertebra	Test	Significance
T9	F-Test Two-Sample for Variances. t-Test: Two-Sample Assuming Unequal Variances.	Not significant, F (32, 36) = 0.3394, **p = 0.0015.** Not significant, females (M = 32, 36, SD = 1.3670) and males (M = 16.7369, SD = 4.02798), t (-0.64711) = 2.0025, p = 0.5202.
T10	F-Test Two-Sample for Variances. t-Test: Two-Sample Assuming Equal Variances.	Not significant, F (32, 36) = 0.2221, p = 2.6E-05. Not significant, females (M = 16.5906, SD = 0.8767) and males (M = 16.8658, SD = 3.9479), t (-0.7434) = 2.0076, p = 0.4607.
T11	F-Test Two-Sample for Variances. t-Test: Two-Sample Assuming Unequal Variances.	Not significant, F (32, 36) = 0.3140, **p = 0.0008.** Not significant, females (M = 17.8819, SD = 1.6664) and males (M = 18.2939, SD = 5.3062), t (-0.9225) = 2.0032, p = 0.3602.
T12	F-Test Two-Sample for Variances. t-Test: Two-Sample Assuming Unequal Variances.	Not significant, F (32, 36) = 0.4357, **p = 0.0106.** Not significant, females (M = 20.8313, SD = 2.5004) and males (M = 21.4772, SD = 5.7393), t (-1.3253) = 1.9996, p = 0.19.
L1	F-Test Two-Sample for Variances. t-Test: Two-Sample Assuming Unequal Variances.	Not significant, F (32, 37) = 0.4029, **p = 0.0058.** Significant, females (M = 21.8025, SD = 2.2381) and males (M = 22.8614, SD = 5.5536), t (-2.2573) = 1.9989, p = 0.0275.
L2	F-Test Two-Sample for Variances. t-Test: Two-Sample Assuming Equal Variances.	Not significant, F (32, 37) = 0.4923, **p = 0.0236.** Significant, females (M = 22.1847, SD = 2.7147) and males (M = 23.2005, SD = 5.51381), t (-2.1007) = 1.9977, p = 0.0396.
L3	F-Test Two-Sample for Variances. t-Test: Two-Sample Assuming Equal Variances.	Not significant, F (32, 37) = 0.4437, p = 0.0117. Significant, females (M = 22.7466, SD = 2.4475) and males (M = 23.8332, SD = 5.5159), t (-2.2881) = 1.9983, p = 0.0255.
L4	F-Test Two-Sample for Variances. t-Test: Two-Sample Assuming Equal Variances.	Not significant, F (31, 37) = 0.7747, p = 0.2387. Not significant, females (M = 23.8335, SD = 3.5878) and males (M = 24.472, SD = 4.6314), t (-1.2869) = 1.9966, p = 0.2026.
L5	F-Test Two-Sample for Variances. t-Test: Two-Sample Assuming Equal Variances.	Not significant, F (31, 36) = 1.0029, p = 0.4932. Not significant, females (M = 26.9232, SD = 9.0189) and males (M = 27.3744, SD = 8.9931), t (-0.6137) = 1.9971, p = 0.546.

Table 21. Results from the ANOVA Two-Factor without Replication test for VNC TR diameter in vertebrae (T1-L5) by SES groups (L, LM, M, UM, U) in the TXSTDSC.

Anova: Two-Factor Without Replication

SUMMARY	*Count*	*Sum*	*Average*	*Variance*		
Row 1	17	330.5582	19.4446	12.32361		
Row 2	17	330.2758	19.42799	12.89318		
Row 3	17	334.3339	19.6667	14.27398		
Row 4	17	323.3545	19.02086	11.50104		
Row 5	17	308.355	18.13853	14.2726		
Column 1	5	107.6056	21.52112	0.215654		
Column 2	5	89.97702	17.9954	0.1732		
Column 3	5	83.59779	16.71956	0.337891		
Column 4	5	80.41511	16.08302	0.462862		
Column 5	5	78.24269	15.64854	0.682339		
Column 6	5	77.77715	15.55543	0.538131		
Column 7	5	78.85802	15.7716	0.417477		
Column 8	5	80.15356	16.03071	0.610664		
Column 9	5	81.76988	16.35398	0.471112		
Column 10	5	82.35447	16.47089	0.436084		
Column 11	5	88.89146	17.77829	0.632282		
Column 12	5	103.7645	20.75291	1.111417		
Column 13	5	110.7207	22.14414	0.553892		
Column 14	5	112.8359	22.56717	0.588092		
Column 15	5	116.063	23.21259	0.325519		
Column 16	5	120.1298	24.02595	0.175192		
Column 17	5	133.7209	26.74419	1.285584		
ANOVA						
Source of Variation	*SS*	*df*	*MS*	*F*	*P-value*	*F crit*
Rows	24.99463	4	6.248657	36.10982	9.02E-16	2.515318
Columns	1033.156	16	64.57222	373.1508	9.4E-57	1.804179
Error	11.07494	64	0.173046			
Total	1069.225	84				

Table 22. Results from the F-Test Two-Sample for Variances and t-Test: Two-Sample Assuming (Un)Equal Variances tests for each VNC AP/TR ratio (T1-L5) in the TXSTDSC.

Vertebra	**Test**	**Significance**
T1	F-Test Two-Sample for Variances. t-Test: Two-Sample Assuming Equal Variances.	Not significant, F (32, 35) = 0.70573, p = 0.1649. Not significant, females (M = 0.71972, SD = 0.0034) and males (M = 0.70229, SD = 0.0049), t (1.10257) = 1.9971, p = 0.2743.
T2	F-Test Two-Sample for Variances. t-Test: Two-Sample Assuming Equal Variances.	Not significant, F (32, 36) = 1.3064, p = 0.2211. Not significant, females (M = 0.8674, SD = 0.0063) and males (M = 0.8483, SD = 0.0048), t (1.0572) = 1.9966, p = 0.2943.
T3	F-Test Two-Sample for Variances. t-Test: Two-Sample Assuming Equal Variances.	Not significant, F (32, 34) = 1.1806, p = 0.3191. Not significant, females (M = 0.9444, SD = 0.0088) and males (M = 0.9174, SD = 0.0074), t (1.2188) = 1.9977, p = 0.2274.
T4	F-Test Two-Sample for Variances. t-Test: Two-Sample Assuming Equal Variances.	Not significant, F (32, 36) = 0.9474, p = 0.4417. Not significant, females (M = 0.9865, SD = 0.0067) and males (M = 0.9623, SD = 0.0070), t (1.2041) = 1.9966, p = 0.2329.
T5	F-Test Two-Sample for Variances. t-Test: Two-Sample Assuming Equal Variances.	Not significant, F (32, 35) = 1.8655, **p = 0.0391.** Not significant, females (M = 1.0397, SD = 0.0115) and males (M = 0.9916, SD = 0.0062), t (2.1047) = 1.9971, p = 0.0392.
T6	F-Test Two-Sample for Variances. t-Test: Two-Sample Assuming Unequal Variances.	Not significant, F (30, 36) = 1.0509, p = 0.4404. Not significant, females (M = 1.0372, SD = 0.0091) and males (M = 0.9997, SD = 0.0086), t (1.6142) = 1.9977, p = 0.1114.
T7	F-Test Two-Sample for Variances. t-Test: Two-Sample Assuming Equal Variances.	Not significant, F (32, 36) = 1.1735, p = 0.3218. Not significant, females (M = 1.0171, SD = 0.0108) and males (M = 0.9927, SD = 0.0092), t (1.0069) = 1.9966, p = 0.3176.
T8	F-Test Two-Sample for Variances. t-Test: Two-Sample Assuming Unequal Variances.	Not significant, F (32, 35) = 1.789, p = 0.2658. Not significant, females (M = 0.9734, SD = 0.0104) and males (M = 0.9702, SD = 0.0083), t (0.1391) = 1.9971, p = 0.8898.
T9	F-Test Two-Sample for Variances. t-Test: Two-Sample Assuming Equal Variances.	Not significant, F (32, 36) = 0.8766, p = 0.3567. Not significant, females (M = 0.9519, SD = 0.0075) and males (M = 0.94933, SD = 0.0086), t (0.1154) = 1.9966, p = 0.9085.

Table 22. Continued. Results from the F-Test Two-Sample for Variances and t-Test: Two-Sample Assuming (Un)Equal Variances tests for each VNC AP/TR ratio (T1-L5) in the TXSTDSC.

Vertebra	Test	Significance
T10	F-Test Two-Sample for Variances. t-Test: Two-Sample Assuming Equal Variances.	Not significant, F (32, 36) = 1.1607, p = 0.3331. Not significant, females (M = 0.9512, SD = 0.01) and males (M = 0.9428, SD = 0.00862), t (0.3567) = 1.9966, p = 0.7225.
T11	F-Test Two-Sample for Variances. t-Test: Two-Sample Assuming Equal Variances.	Not significant, F (32, 36) = 2.5380, p = 0.0042. Not significant, females (M = 0.8899, SD = 0.0097) and males (M = 0.8896, SD = 0.0038), t (0.0128) = 2.0076, p = 0.9899.
T12	F-Test Two-Sample for Variances. t-Test: Two-Sample Assuming Equal Variances.	Not significant, F (32, 36) = 2.3923, p = 0.0067. Not significant, females (M = 0.8137, SD = 0.0119) and males (M = 0.8016, SD = 0.005), t (0.5507) = 1.9966, p = 0.5837.
L1	F-Test Two-Sample for Variances. t-Test: Two-Sample Assuming Equal Variances.	Not significant, F (32, 37) = 1.1437, p = 0.3469. Not significant, females (M = 0.8013, SD = 0.0044) and males (M = 0.7574, SD = 0.0039), t (2.8326) = 1.9960, p = 0.0061.
L2	F-Test Two-Sample for Variances. t-Test: Two-Sample Assuming Equal Variances.	Not significant, F (32, 37) = 1.0845, p = 0.4047. Not significant, females (M = 0.7614, SD = 0.0044) and males (M = 0.6853, SD = 0.0041), t (4.8329) = 1.9960, p = 8.2e-06.
L3	F-Test Two-Sample for Variances. t-Test: Two-Sample Assuming Equal Variances.	Not significant, F (32, 37) = 1.4004, p = 0.1648. Not significant, females (M = 0.7124, SD = 0.0052) and males (M = 0.6343, SD = 0.0037), t (4.8962) = 1.9960, p = 6.5E-06.
L4	F-Test Two-Sample for Variances. t-Test: Two-Sample Assuming Equal Variances.	Not significant, F (31, 37) = 1.1232, p = 0.3666. Not significant, females (M = 0.6927, SD = 0.0058) and males (M = 0.6439, SD = 0.0051), t (2.7268) = 1.9966, p = 0.0082.
L5	F-Test Two-Sample for Variances. t-Test: Two-Sample Assuming Equal Variances.	Not significant, F (31, 36) = 1.5203, p = 0.1165. Not significant, females (M = 0.6461, SD = 0.0105) and males (M = 0.6217, SD = 0.0069), t (1.0766) = 1.9971, p = 0.2856.

Table 23. Results from the ANOVA Two-Factor without Replication test for average VNC TR diameter by sex and grouped SES levels in the TXSTDSC (T1-L5).

Anova: Two-Factor Without Replication

SUMMARY	*Count*	*Sum*	*Average*	*Variance*		
Row 1	17	328.0895	19.29938	12.85542		
Row 2	17	323.2819	19.01658	11.21948		
Row 3	17	332.0514	19.53244	12.41293		
Row 4	17	337.4447	19.84969	15.35356		
Column 1	4	85.26321	21.3158	0.137798		
Column 2	4	72.78089	18.19522	0.123048		
Column 3	4	67.97977	16.99494	0.092113		
Column 4	4	65.47508	16.36877	0.047458		
Column 5	4	64.07638	16.0191	0.090828		
Column 6	4	63.4666	15.86665	0.038217		
Column 7	4	64.25826	16.06456	0.018363		
Column 8	4	65.56313	16.39078	0.015762		
Column 9	4	66.67206	16.66801	0.029038		
Column 10	4	67.0642	16.76605	0.015894		
Column 11	4	72.48213	18.12053	0.046587		
Column 12	4	84.91462	21.22866	0.135084		
Column 13	4	89.8667	22.46668	0.636065		
Column 14	4	91.22205	22.80551	0.556217		
Column 15	4	93.74192	23.43548	0.779153		
Column 16	4	96.9742	24.24355	0.256235		
Column 17	4	109.0663	27.26658	0.668311		
ANOVA						
Source of Variation	*SS*	*df*	*MS*	*F*	*P-value*	*F crit*
Rows	6.366274	3	2.122091	21.70828	5.01E-09	2.798061
Columns	824.77	16	51.54812	527.3198	2.92E-48	1.859167
Error	4.692238	48	0.097755			
Total	835.8285	67				

Table 24. Results from the ANOVA Two-Factor without Replication testing for significance between VNC ratios and SES levels in the TXSTDSC sample.

Anova: Two-Factor Without Replication

SUMMARY	*Count*	*Sum*	*Average*	*Variance*		
Row 1	17	14.591	0.858	0.019		
Row 2	17	14.265	0.839	0.017		
Row 3	17	14.565	0.857	0.020		
Row 4	17	14.630	0.861	0.018		
Row 5	17	15.970	0.939	0.024		
Column 1	5	3.567	0.713	0.000		
Column 2	5	4.362	0.872	0.002		
Column 3	5	4.719	0.944	0.001		
Column 4	5	4.940	0.988	0.002		
Column 5	5	5.176	1.035	0.003		
Column 6	5	5.214	1.043	0.004		
Column 7	5	5.099	1.020	0.001		
Column 8	5	4.930	0.986	0.002		
Column 9	5	4.812	0.962	0.001		
Column 10	5	4.876	0.975	0.005		
Column 11	5	4.558	0.912	0.003		
Column 12	5	4.085	0.817	0.001		
Column 13	5	3.957	0.791	0.001		
Column 14	5	3.635	0.727	0.000		
Column 15	5	3.403	0.681	0.001		
Column 16	5	3.397	0.679	0.001		
Column 17	5	3.293	0.659	0.004		
ANOVA						
Source of Variation	*SS*	*df*	*MS*	*F*	*P-value*	*F crit*
Rows	0.105	4	0.0262	60.909	3.99217E-21	2.515
Columns	1.523	16	0.0952	221.068	1.33669E-49	1.804
Error	0.028	64	0.0004			
Total	1.656	84				

Table 25. Results from t-Test for VNC AP diameter and sex in the PCOME sample.

Vertebra	Test	Significance
T1	F-Test Two-Sample for Variances. t-Test: Two-Sample Assuming Equal Variances.	Not significant, F (2, 21) = 2.2304, p = 0.1509. Not significant, females (M = 14.745, SD = 3.727) and males (M = 14.45, SD = 1.6707), t (0.2998) = 2.0796, p = 0.7673.
T2	F-Test Two-Sample for Variances. t-Test: Two-Sample Assuming Equal Variances.	Not significant, F (2, 21) = 0.3175, p = 0.4206. Not significant, females (M = 14.32, SD = 0.405) and males (M = 14.567, SD = 1.2755), t (-0.3006) = 2.0796, p = 0.7666.
T3	F-Test Two-Sample for Variances. t-Test: Two-Sample Assuming Equal Variances.	Not significant, F (2, 22) = 0.1778, p = 0.3224. Not significant, females (M = 15.15, SD = 0.3698) and males (M = 15.027, SD = 2.0802), t (0.1174) = 2.0739, p = 0.9076.
T4	F-Test Two-Sample for Variances. t-Test: Two-Sample Assuming Equal Variances.	Not significant, F (2, 23) = 0.004, p = 0.5777. Not significant, females (M = 14.21, SD = 1.2168) and males (M = 14.21, SD = 1.2168), t (-1.0502) = 2.0687, p = 0.3045.
T5	F-Test Two-Sample for Variances. t-Test: Two-Sample Assuming Equal Variances.	Not significant, F (2, 23) = 4.2597, p = 0.18953. Not significant, females (M = 14.34, SD = 3.4322) and males (M = 15.58, SD = 1.7696), t (-1.1434) = 2.0595, p = 0.2637.
T6	F-Test Two-Sample for Variances. t-Test: Two-Sample Assuming Unequal Variances.	Not significant, F (2, 25) = 4.2597, p = 0.1813. Not significant, females (M = 15.175, SD = 3.3541) and males (M = 15.061, SD = 1.7696), t (0.1148) = 2.0595, p = 0.9095.
T7	F-Test Two-Sample for Variances. t-Test: Two-Sample Assuming Equal Variances.	Not significant, F (2, 21) = 4.2417, p = 0.1854. Not significant, females (M = 14.725, SD = 3.9481) and males (M = 15.417, SD = 2.1288), t (-0.6363) = 2.0555, p = 0.5302.
T8	F-Test Two-Sample for Variances. t-Test: Two-Sample Assuming Unequal Variances.	Not significant, F (2, 24) = 0.004, p = 0.4279. Not significant, females (M = 14.015, SD = 0.3961) and males (M = 15.365, SD = 1.2053), t (-1.6947) = 2.0639, p = 0.1031.
T9	F-Test Two-Sample for Variances. t-Test: Two-Sample Assuming Equal Variances.	Not significant, F (2, 25) = 4.2597, p = 0.3209. Not significant, females (M = 14.24, SD = 1.2168) and males (M = 15.127, SD = 1.1843), t (-1.1083) = 2.0595, p = 0.2783.
T10	F-Test Two-Sample for Variances. t-Test: Two-Sample Assuming Equal Variances.	Not significant, F (2, 24) = 0.004, p = 0.4285. Not significant, females (M = 14.86, SD = 0.3872) and males (M = 14.897, SD = 1.1748), t (-0.0466) = 2.0639, p = 0.9632.
T11	F-Test Two-Sample for Variances. t-Test: Two-Sample Assuming Unequal Variances.	Not significant, F (2, 23) = 0.004**, p = 0.0203.** Not significant, females (M = 15.985, SD = 0.0013) and males (M = 15.579, SD = 1.8897), t (1.4106) = 2.0739, p = 0.1723.
T12	F-Test Two-Sample for Variances. t-Test: Two-Sample Assuming Equal Variances.	Not significant, F (2, 22) = 0.0628, p = 0.1954. Not significant, females (M = 16.405, SD = 0.1104) and males (M = 16.415, SD = 1.7595), t (-0.0104) = 2.0739, p = 0.9918.

Table 25. Continued. Results from t-Test for VNC AP diameter and sex in the PCOME sample.

Vertebra	**Test**	**Significance**
L1	F-Test Two-Sample for Variances. t-Test: Two-Sample Assuming Equal Variances.	Not significant, F (2, 24) = 0.3055, p = 0.4142. Not significant, females (M = 16.18, SD = 0.9522) and males (M = 16.313, SD = 3.1164), t (-0.1041) = 2.0639, p = 0.9179.
L2	F-Test Two-Sample for Variances. t-Test: Two-Sample Assuming Equal Variances.	Not significant, F (2, 25) = 0.1917, p = 0.3346. Not significant, females (M = 14.86, SD = 0.605) and males (M = 14.929, SD = 3.1555), t (-0.0539) = 2.0595, p = 0.9575.
L3	F-Test Two-Sample for Variances. t-Test: Two-Sample Assuming Equal Variances.	Not significant, F (2, 26) = 0.5561, p = 0.5372. Not significant, females (M = 14.19, SD = 1.8818) and males (M = 13.522, SD = 3.3836), t (0.4995) = 2.0555, p = 0.6216.
L4	F-Test Two-Sample for Variances. t-Test: Two-Sample Assuming Equal Variances.	Not significant, F (2, 25) = 0.0133, p = 0.0838. Not significant, females (M = 14.49, SD = 0.045) and males (M = 14.269, SD = 3.9843), t (0.1539) = 2.0595, p = 0.8789.
L5	F-Test Two-Sample for Variances. t-Test: Two-Sample Assuming Equal Variances.	Not significant, F (2, 22) = 0.0122, p = 0.0869. Not significant, females (M = 15.39, SD = 0.0578) and males (M = 15.049, SD = 4.7391), t (0.217) = 2.0739, p = 0.8302.

Table 26. T-Test results from VNC TR diameters of T1-L5 between males and females in the PCOME sample.

Vertebra	Test	Significance
T1	F-Test Two-Sample for Variances. t-Test: Two-Sample Assuming Unequal Variances.	Not significant, F (2, 21) = 18.015, **p = 0.0004.** Not significant, females (M = 16.49, SD = 23.805) and males (M = 19.895, SD = 1.3214), t (-0.984) = 12.706, p = 0.505.
T2	F-Test Two-Sample for Variances. t-Test: Two-Sample Assuming Equal Variances.	Not significant, F (2, 21) = 2.573, p = 0.1244. Not significant, females (M = 15.115, SD = 4.7125) and males (M = 17.006, SD = 1.8315), t (-1.821) = 2.0796, p = 0.0829.
T3	F-Test Two-Sample for Variances. t-Test: Two-Sample Assuming Equal Variances.	Not significant, F (2, 22) = 3.5491, p = 0.0735. Not significant, females (M = 13.825, SD = 15.805) and males (M = 15.805, SD = 1.0966), t (-2.423) = 2.0739, p = 0.0241.
T4	F-Test Two-Sample for Variances. t-Test: Two-Sample Assuming Equal Variances.	Not significant, F (2, 23) = 2.174, p = 1545. Not significant, females (M = 13.595, SD = 2.9041) and males (M = 15.279, SD = 1.3358), t (-1.928) =2.0687, p = 0.0663.

Table 26. Continued. T-Test results from VNC TR diameters of T1-L5 between males and females in the PCOME sample.

Vertebra	Test	Significance
T5	F-Test Two-Sample for Variances. t-Test: Two-Sample Assuming Equal Variances.	Not significant, F (2, 25) = 1.6154, p = 0.2159. Not significant, females (M = 13.48, SD = 2.1632) and males (M = 15.049, SD = 1.3391), t (-1.823) = 2.0595, p = 0.0804.
T6	F-Test Two-Sample for Variances. t-Test: Two-Sample Assuming Unequal Variances.	Not significant, F (2, 25) = 0.6215, p = 0.5618. Not significant, females (M = 13.585, SD = 0.8321) and males (M = 15.27, SD = 1.3388), t (-1.997) = 2.0595, p = 0.0568.
T7	F-Test Two-Sample for Variances. t-Test: Two-Sample Assuming Equal Variances.	Not significant, F (2 26) = 0.3239, p = 0.4256. Not significant, females (M = 13.795, SD = 0.4512) and males (M = 15.575, SD = 1.3933), t (-2.082) = 2.0555, p = 0.0473.
T8	F-Test Two-Sample for Variances. t-Test: Two-Sample Assuming Unequal Variances.	Not significant, F (2, 24) = 0.4834, p = 0.5062. Not significant, females (M = 14.135, SD = 0.7565) and males (M = 15.861, SD = 1.5648), t (-1.896) = 2.0639, p = 0.0701.
T9	F-Test Two-Sample for Variances. t-Test: Two-Sample Assuming Equal Variances.	Not significant, F (2, 25) = 0.5217, p = 0.5229. Not significant, females (M = 14.265, SD = 0.8845) and males (M = 16.193, SD = 1.6954), t (-2.035) = 2.0595, p = 0.0526.
T10	F-Test Two-Sample for Variances. t-Test: Two-Sample Assuming Equal Variances.	Not significant, F (2, 24) = 0.643, p = 0.5692. Not significant, females (M = 14.065, SD = 1.0805) and males (M = 16.198, SD = 1.6802), t (-2.253) = 2.0639, p = 0.0337.
T11	F-Test Two-Sample for Variances. t-Test: Two-Sample Assuming Equal Variances.	Not significant, F (2, 23) = 0.3303, p = 0.4287. Not significant, females (M = 15.77, SD = 0.5832) and males (M = 17.467, SD = 1.7655), t (-1.759) = 2.0687, p = 0.0919.
T12	F-Test Two-Sample for Variances. t-Test: Two-Sample Assuming Equal Variances.	Not significant, F (2, 22) = 0.9356, p = 0.6556. Not significant, females (M = 18.24, SD = 2.0402) and males (M = 20.187, SD = 2.1805), t (-1.788) = 2.0739, p = 0.0875.
L1	F-Test Two-Sample for Variances. t-Test: Two-Sample Assuming Equal Variances.	Not significant, F (2, 24) = 0.9932, p = 0.6707. Not significant, females (M = 19.11, SD = 3.38) and males (M = 21.426, SD = 3.4031), t (-1.706) = 2.0639, p = 0.1009.

Table 26. Continued. T-Test results from VNC TR diameters of T1-L5 between males and females in the PCOME sample.

Vertebra	Test	Significance
L2	F-Test Two-Sample for Variances. t-Test: Two-Sample Assuming Equal Variances.	Not significant, F (2, 25) = 0.8961, p = 0.6467. Not significant, females (M = 19.3, SD = 2.3762) and males (M = 21.658, SD = 2.6517), t (-1.974) = 2.0595, p = 0.0595.
L3	F-Test Two-Sample for Variances. t-Test: Two-Sample Assuming Equal Variances.	Not significant, F (2, 26) = 0.8879, p = 0.6449. Not significant, females (M = 19.725, SD = 21.692) and males (M = 21.692, SD = 2.6031), t (-1.665) = 2.0555, p = 0.1079.
L4	F-Test Two-Sample for Variances. t-Test: Two-Sample Assuming Equal Variances.	Not significant, F (2, 25) = 0.2368, p = 0.369. Not significant, females (M = 21.055, SD = 0.7813) and males (M = 22.298, SD = 3.2998), t (-0.945) = 2.0595, p = 0.3535.
L5	F-Test Two-Sample for Variances. t-Test: Two-Sample Assuming Equal Variances.	Not significant, F (2, 23) = 0.2178, p = 0.3547. Not significant, females (M = 22.445, SD = 1.8625) and males (M = 24.275, SD = 8.5517), t (-0.864) = 2.0687, p = 0.3967.

Table 27. T-Test results for VNC AP/TR ratio for T1-L5 among males and females in the PCOME sample.

Vertebra	Test	Significance
T1	F-Test Two-Sample for Variances. t-Test: Two-Sample Assuming Equal Variances.	Not significant, F (2, 22) = 64.851, p = 7e-08. Not significant, females (M = 0.9532, SD = 0.1593) and males (M = 0.7272, SD = 0.0025), t (0.8003) = 12.706, p = 0.5703.
T2	F-Test Two-Sample for Variances. t-Test: Two-Sample Assuming Equal Variances.	Not significant, F (2, 22) = 1.8591, p = 0.1872. Not significant, females (M = 0.9542, SD = 0.009) and males (M = 0.8581, SD = 0.0048), t (1.8341) = 2.0739, p = 0.0802.
T3	F-Test Two-Sample for Variances. t-Test: Two-Sample Assuming Equal Variances.	Not significant, F (2, 23) = 1.3917, p = 0.2507. Significant, females (M = 1.1039, SD = 0.0129) and males (M = 0.9551, SD = 0.0093), t (2.0801) = 2.0687, p = 0.0488.
T4	F-Test Two-Sample for Variances. t-Test: Two-Sample Assuming Equal Variances.	Not significant, F (2, 24) = 0.2008, p = 0.3417. Not significant, females (M = 1.0484, SD = 0.0025) and males (M = 1.002, SD = 0.0126), t (0.5709) = 2.0639, p = 0.5734.

Table 27. Continued. T-Test results for VNC AP/TR ratio for T1-L5 among males and females in the PCOME sample.

Vertebra	Test	Significance
T5	F-Test Two-Sample for Variances. t-Test: Two-Sample Assuming Equal Variances.	Not significant, F (2, 27) = 0.0458, p = 0.1677.Not significant, females (M = 1.0626, SD = 0.0005) and males (M = 1.0384, SD = 0.0101), t (0.3355) = 2.0518, p = 0.7398.
T6	F-Test Two-Sample for Variances. t-Test: Two-Sample Assuming Unequal Variances.	Not significant, F (2, 27) = 0.4174, p = 0.4761. Significant, females (M = 1.115, SD = 0.0036) and males (M = 0.9905, SD = 0.0086), t (1.8518) = 2.0518, p = 0.075.
T7	F-Test Two-Sample for Variances. t-Test: Two-Sample Assuming Equal Variances.	Not significant, F (2, 29) = 0.9185, p = 0.6539. Not significant, females (M = 1.0652, SD = 0.0085) and males (M = 0.9919, SD = 0.0092), t (1.043) = 2.0452, p = 0.3056.
T8	F-Test Two-Sample for Variances. t-Test: Two-Sample Assuming Unequal Variances.	Not significant, F (2, 27) = 0.0314, p = 0.1394. Not significant, females (M = 0.992, SD = 0.0003) and males (M = 0.9747, SD = 0.0087), t (0.2584) = 2.0518, p = 0.798.
T9	F-Test Two-Sample for Variances. t-Test: Two-Sample Assuming Equal Variances.	Not significant, F (2, 28) = 0.033, p = 0.1428. Not significant, females (M = 0.9979, SD = 0.9376) and males (M = 0.9376, SD = 0.004), t (1.3182) = 2.0484, p = 0.1981.
T10	F-Test Two-Sample for Variances. t-Test: Two-Sample Assuming Equal Variances.	Not significant, F (2, 27) = 0.2897, p = 0.405. Significant, females (M = 1.0578, SD = 0.0012) and males (M = 0.9231, SD = 0.004), t (2.955) = 2.0518, p = 0.0064.
T11	F-Test Two-Sample for Variances. t-Test: Two-Sample Assuming Equal Variances.	Not significant, F (2, 26) = 0.5969, p = 0.553. Significant, females (M = 1.0148, SD = 0.0022) and males (M = 0.8964, SD = 0.0037), t (2.6774) = 2.0555, p = 0.0127.
T12	F-Test Two-Sample for Variances. t-Test: Two-Sample Assuming Equal Variances.	Not significant, F (2, 25) = 2.0914, p = 0.1611. Not significant, females (M = 0.9029, SD = 0.0079) and males (M = 0.8223, SD = 0.0038), t (1.7446) = 2.0595, p = 0.0933.
L1	F-Test Two-Sample for Variances. t-Test: Two-Sample Assuming Equal Variances.	Not significant, F (2, 27) = 4.1323, p = 0.0524. Not significant, females (M = 0.8531, SD = 0.0177) and males (M = 0.7671, SD = 0.0043), t (1.6954) = 2.0518, p = 0.1015.

Table 27. Continued. T-Test results for VNC AP/TR ratio for T1-L5 among males and females in the PCOME sample.

Vertebra	Test	Significance
L2	F-Test Two-Sample for Variances. t-Test: Two-Sample Assuming Equal Variances.	Not significant, F (2, 28) = 2.6884, p = 0.1127. Not significant, females (M = 0.774, SD = 0.0104) and males (M = 0.6915, SD = 0.0039), t (1.7591) = 2.0484, p = 0.0895.
L3	F-Test Two-Sample for Variances. t-Test: Two-Sample Assuming Equal Variances.	Not significant, F (2, 29) = 3.5172, p = 0.0712. Not significant, females (M = 0.7242, SD = 0.0157) and males (M = 0.6251, SD = 0.0045), t (1.9457) = 2.0452, p = 0.0614.
L4	F-Test Two-Sample for Variances. t-Test: Two-Sample Assuming Equal Variances.	Not significant, F (2, 28) = 0.292, p = 0.4066. Not significant, females (M = 0.689, SD = 0.0015) and males (M = 0.6379, SD = 0.0052), t (0.9808) = 2.0484, p = 0.3351.
L5	F-Test Two-Sample for Variances. t-Test: Two-Sample Assuming Equal Variances.	Not significant, F (2, 25) = 0.1414, p = 0.2898. Not significant, females (M = 0.6866, SD = 0.001) and males (M = 0.611, SD = 0.0068), t (1.2682) = 2.0595, p = 0.2164.

Table 28. Results from the F-Test Two-Sample for Variances and t-Test: Two-Sample Assuming (Un)Equal Variances tests for each VNC AP diameter in vertebra (T1-L5) in OpID.

Vertebra	Test	Significance
T1	F-Test Two-Sample for Variances. t-Test: Two-Sample Assuming Equal Variances.	Not significant, F (26, 51) = 0.891, p = 0.386. Not significant, females (M = 14.219, SD = 1.620) and males (M = 14.915, SD = 0.955), t (-1.461) = 1.99, p = 0.148.
T2	F-Test Two-Sample for Variances. t-Test: Two-Sample Assuming Equal Variances.	Not significant, F (25, 51) = 1.33, p = 0.192. Not significant, females (M = 14.187, SD = 1.707) and males (M = 14.663, SD = 1.279), t (-1.499) = 1.99, p = 0.138.
T3	F-Test Two-Sample for Variances. t-Test: Two-Sample Assuming Equal Variances.	Not significant, F (25, 52) = 1.676, p = 0.0611. Not significant, females (M = 14.219, SD = 1.6198) and males (M = 14.641, SD = 0.967), t (-1.598) = 1.99, p = 0.114.
T4	F-Test Two-Sample for Variances. t-Test: Two-Sample Assuming Equal Variances.	Not significant, F (25, 53) = 1.3457, p = 0.183. Not significant, females (M = 14.5656, SD = 1.286) and males (M = 14.915, SD = 0.955), t (-1.398) = 1.99, p = 0.166.

Table 28. Continued. Results from the F-Test Two-Sample for Variances and t-Test: Two-Sample Assuming (Un)Equal Variances tests for each VNC AP diameter in vertebra (T1-L5) in OpID.

Vertebra	Test	Significance
T5	F-Test Two-Sample for Variances. t-Test: Two-Sample Assuming Equal Variances.	Not significant, F (27, 54) = 1.706, p = 0.2044. Not significant, females (M = 14.830, SD = 1.513) and males (M = 15.152, SD = 1.161), t (-1.207) = 1.99, p = 0.231.
T6	F-Test Two-Sample for Variances. t-Test: Two-Sample Assuming Unequal Variances.	Not significant, F (27, 55) = 2.626, **p = 0.0013.** Not significant, females (M = 15.171, SD = 2.718) and males (M = 15.2298, SD = 1.035), t (-0.171) = 2.028, p = 0.865.
T7	F-Test Two-Sample for Variances. t-Test: Two-Sample Assuming Equal Variances.	Not significant, F (29, 59) = 1.6159, p = 0.062. Not significant, females (M = 15.092, SD = 2.24) and males (M = 15.414, SD = 1.387), t (-1.099) = 1.987, p = 0.275.
T8	F-Test Two-Sample for Variances. t-Test: Two-Sample Assuming Unequal Variances.	Significant, F (29, 61) = 1.66**, p = 0.013.** Not significant, females (M = 14.887, SD = 2.56) and males (M = 15.465, SD = 1.286), t (-1.748) = 2.018, p = 0.088.
T9	F-Test Two-Sample for Variances. t-Test: Two-Sample Assuming Equal Variances.	Not significant, F (29, 61) = 1.614, p = 0.061. Not significant, females (M = 14.741, SD = 1.642) and males (M = 15.209, SD = 1.017), t (-1.883) = 1.987, p = 0.063.
T10	F-Test Two-Sample for Variances. t-Test: Two-Sample Assuming Equal Variances.	Not significant, F (29, 60) = 0.926, p = 0.422. Not significant, females (M = 14.623, SD = 1.237) and males (M = 14.956, SD = 1.336), t (-1.290) = 1.988, p = 0.200.
T11	F-Test Two-Sample for Variances. t-Test: Two-Sample Assuming Equal Variances.	Not significant, F (29, 60) = 0.964, p = 0.471. Not significant, females (M = 14.986, SD = 1.569) and males (M = 15.272, SD = 1.627), t (-0996) = 1.988, p = 0.322.
T12	F-Test Two-Sample for Variances. t-Test: Two-Sample Assuming Equal Variances.	Not significant, F (30, 60) = 0.729, p = 0.177. Not significant, females (M = 16.184, SD = 1.169) and males (M = 16.203, SD = 1.604), t (-0.0697) = 1.987, p = 0.945.
L1	F-Test Two-Sample for Variances. t-Test: Two-Sample Assuming Equal Variances.	Not significant, F (30, 65) = 1.289, p = 0.198. Not significant, females (M = 16.154, SD = 2.220) and males (M = 16.225, SD = 1.722), t (-0.233) = 1.986, p = 0.816.

Table 28. Continued. Results from the F-Test Two-Sample for Variances and t-Test: Two-Sample Assuming (Un)Equal Variances tests for each VNC AP diameter in vertebra (T1-L5) in OpID.

Vertebra	Test	Significance
L2	F-Test Two-Sample for Variances. t-Test: Two-Sample Assuming Equal Variances.	Not significant, F (30, 64) = 1.352, p = 0.159. Not significant, females (M = 15.493, SD = 2.601) and males (M = 14.891, SD = 1.923), t (1.862) = 1.986, p = 0.066.
L3	F-Test Two-Sample for Variances. t-Test: Two-Sample Assuming Equal Variances.	Not significant, F (29, 63) = 1.191, p = 0.279. Not significant, females (M = 14.634, SD = 2.350) and males (M = 14.030, SD = 1.974), t (1.862) = 1.987, p = 0.0658.
L4	F-Test Two-Sample for Variances. t-Test: Two-Sample Assuming Equal Variances.	Not significant, F (29, 63) = 1.280, p = 0.208. Significant, females (M = 15.030, SD = 2.891) and males (M = 14.118, SD = 2.258), t (2.595) = 1.987, p = 0.011.
L5	F-Test Two-Sample for Variances. t-Test: Two-Sample Assuming Equal Variances.	Not significant, F (28, 62) = 0.646, p = 0.107. Significant, females (M = 16.292, SD = 2.388) and males (M = 15.305, SD = 3.695), t (2.389) = 1.987, p = 0.019.

Table 29. Results from the F-Test Two-Sample for Variances and t-Test: Two-Sample Assuming (Un)Equal Variances tests for each VNC TR diameter vertebra (T1-L5) in the OpID sample.

Vertebra	Test	Significance
Overall	F-Test Two-Sample for Variances. t-Test: Two-Sample Assuming Equal Variances.	Not significant, F (17, 17) = 0.9854, p = 0.4885. Not significant, females (M = 17.996, SD = 8.2811) and males (M = 17.788, SD = 8.4034), t (32) = 0.2103, p = 0.4174.
T1	F-Test Two-Sample for Variances. t-Test: Two-Sample Assuming Equal Variances.	Not significant, F (26, 51) = 0.54286, p = 0.37572. Not significant, females (M = 19.3965, SD = 1.78846) and males (M = 20.0067, SD = 2.02634), t (-1.8145) = 1.9921, p = 0.0736.
T2	F-Test Two-Sample for Variances. t-Test: Two-Sample Assuming Equal Variances.	Not significant, F (25, 51) = 0.5369, p = 05025. Significant, females (M = 16.358, SD = 1.3579) and males (M = 17.174, SD = 1.3749), t (-2.8566) = 1.9925, p = 0.0056.
T3	F-Test Two-Sample for Variances. t-Test: Two-Sample Assuming Equal Variances.	Not significant, F (25, 52) = 1.7327, p = 0.0.3056. Significant, females (M = 15.18, SD = 1.3564) and males (M = 16.165, SD = 1.1526), t (-3.6661) = 1.9921, p = 0.0005.

Table 29. Continued. Results from the F-Test Two-Sample for Variances and t-Test: Two-Sample Assuming (Un)Equal Variances tests for each VNC TR diameter vertebra (T1-L5) in the OpID sample.

Vertebra	Test	Significance
T4	F-Test Two-Sample for Variances. t-Test: Two-Sample Assuming Equal Variances.	Not significant, F (25, 54) = 1.7245, p = 0.0.2846. Significant, females (M = 14.69, SD = 1.4309) and males (M = 15.696, SD = 1.1926), t (-3.6916) = 1.9913, p = 0.0004.
T5	F-Test Two-Sample for Variances. t-Test: Two-Sample Assuming Equal Variances.	Not significant, F (27, 54) = 0.1.1219, p = 0.0.3526. Significant, females (M = 14.473, SD = 1.4282) and males (M = 15.382, SD = 1.273), t (-3.3504) = 1.9905, p = 0.0012.
T6	F-Test Two-Sample for Variances. t-Test: Two-Sample Assuming Equal Variances.	Not significant, F (27, 54) = 1.6979, p = 0.2547. Significant, females (M = 14.402., SD = 1.7229) and males (M = 15.367, SD = 1.4001), t (-3.3591) = 1.6639, p = 0.0012.
T7	F-Test Two-Sample for Variances. t-Test: Two-Sample Assuming Equal Variances.	Not significant, F (29, 59) = 1.2143, p = 0.0.262. Significant, females (M = 14.621, SD = 2.3203) and males (M = 15.531, SD = 1.9109), t (-2.8059) = 1.9879, p = 0.0062.
T8	F-Test Two-Sample for Variances. t-Test: Two-Sample Assuming Equal Variances.	Not significant, F (29, 69) = 0.0.7491, p = 0.2029. Significant, females (M = 14.862, SD = 1.6385) and males (M = 15.799, SD = 2.1874), t (-2.9289) = 1.9873, p = 0.0043.
T9	F-Test Two-Sample for Variances. t-Test: Two-Sample Assuming Equal Variances.	Not significant, F (29, 61) = 0.6099, p = 0.0765. Significant, females (M = 15.088., SD = 1.5539) and males (M = 16.034, SD = 2.5478), t (-2.9076) = 1.9873, p = 0.0061.
T10	F-Test Two-Sample for Variances. t-Test: Two-Sample Assuming Unequal Variances.	Significant, F (29, 60) = 0.442, **p = 0.0105.** Significant, females (M = 15.528, SD = 1.2625) and males (M = 16.189, SD = 2.8423), t (-2.1931) = 1.9908, p = 0.0313.
T11	F-Test Two-Sample for Variances. t-Test: Two-Sample Assuming Unequal Variances.	Significant, F (29, 60) = 0.5648, **p = 0.00091.** Not significant, females (M = 16.734, SD = 1.5952) and males (M = 17.279, SD = 3.6643), t (-1.5997) = 1.9905, p = 0.1137.
T12	F-Test Two-Sample for Variances. t-Test: Two-Sample Assuming Unequal Variances.	Significant, F (29, 60) = 0.5648, **p = 0.0238.** Significant, females (M = 18.861, SD = 1.8315) and males (M = 19.713, SD = 3.657), t (-2.417) = 1.9921, p = 0.0181.

Table 29. Continued. Results from the F-Test Two-Sample for Variances and t-Test: Two-Sample Assuming (Un)Equal Variances tests for each VNC TR diameter vertebra (T1-L5) in the OpID sample.

Vertebra	Test	Significance
L1	F-Test Two-Sample for Variances. t-Test: Two-Sample Assuming Equal Variances.	Not significant, F (31, 65) = 0.6775, p = 0.1214. Significant, females (M = 20.139, SD = 1.8942) and males (M = 21.003, SD = 2.796), t (-2.4991) = 1.9855, p = 0.0142.
L2	F-Test Two-Sample for Variances. t-Test: Two-Sample Assuming Equal Variances.	Not significant, F (31, 64) = 0.5766, p = 0.2416. Significant, females (M = 20.37., SD = 1.9893) and males (M = 21.061, SD = 2.52), t (-2.0617) = 1.9858, p = 0.042.
L3	F-Test Two-Sample for Variances. t-Test: Two-Sample Assuming Equal Variances.	Not significant, F (30, 63) = 0.8053, p = 0.2647. Not significant, females (M = 21.187, SD = 2.1297) and males (M = 21.486, SD = 2.6446), t (-0.8554) = 1.9864, p = 0.3946.
L4	F-Test Two-Sample for Variances. t-Test: Two-Sample Assuming Equal Variances.	Not significant, F (30, 63) = 0.8614, p = 0.3359. Not significant, females (M = 21.956, SD = 2.8618) and males (M = 22.008, SD = 3.3221), t (-0.1326) = 1.9861, p = 0.8948.
L5	F-Test Two-Sample for Variances. t-Test: Two-Sample Assuming Equal Variances.	Not significant, F (30, 63) = 0.5714, p = 0.4086. Not significant, females (M = 24.375, SD = 4.9453) and males (M = 24.805, SD = 5.3938), t (-0.8471) = 1.9864, p = 0.3992.

Table 30. Results from the F-Test Two-Sample for Variances and t-Test: Two-Sample assuming (Un)Equal Variances tests for each VNC Ratio for vertebra (T1-L5) in the OpID sample.

Vertebra	Test	Significance
T1	F-Test Two-Sample for Variances. t-Test: Two-Sample Assuming Equal Variances.	Not significant, F (25, 52) = 0777, p = 0.254. Not significant, females (M = .7336, SD = .00384) and males (M = .7305, SD = .00494), t (0.1868) = 1.9921, p = 0.852.
T2	F-Test Two-Sample for Variances. t-Test: Two-Sample Assuming Equal Variances.	Not significant, F (24, 52) = 1.743, p = 0.170. Not significant, females (M = 0.866, SD = 0.00671) and males (M = 0.85554, SD = 0.00488), t (0.58947) = 1.993, p = 0.557.
T3	F-Test Two-Sample for Variances. t-Test: Two-Sample Assuming Equal Variances.	Not significant, F (24, 53) = 0.905, p = 0.4085. Not significant, females (M = 0.93374, SD = 0.00475) and males (M = 0.91098, SD = 0.00524), t (1.296) = 1.9921, p = 0.1988.

Table 30. Continued. Results from the F-Test Two-Sample for Variances and t-Test: Two-Sample Assuming (Un)Equal Variances tests for each VNC Ratio for vertebra (T1-L5) in the OpID sample.

Vertebra	Test	Significance
T4	F-Test Two-Sample for Variances. t-Test: Two-Sample Assuming Equal Variances.	Not significant, F (24, 54) = 1.05841, p = 0.41805. Not significant, females (M = 0.99185, SD = 0.00751) and males (M = 0.95721, SD = 0.00709), t (1.66172) = 1.99167, p = 0.10069.
T5	F-Test Two-Sample for Variances. t-Test: Two-Sample Assuming Equal Variances.	Not significant, F (26, 55) = 0.4812, p = 0.4812. Not significant, females (M = 1.0293, SD = 0.0084) and males (M = 0.9899, SD = 0.0084), t (1.7899) = 1.990, p = 0.077.
T6	F-Test Two-Sample for Variances. t-Test: Two-Sample Assuming Equal Variances.	Not significant, F (26, 55) = 08615, p = 0.349. Significant, females (M = 1.05651, SD = 0.00687) and males (M = 0.99201, SD = 0.00797), t (3.10442) = 1.99045, p = 0.0026.
T7	F-Test Two-Sample for Variances. t-Test: Two-Sample Assuming Equal Variances.	Not significant, F (28, 59) = 0.775, p = 0.237. Not significant, females (M = 1.0382, SD = 0.00853) and males (M = 1.001, SD = 0.01101), t (1.61) = 1.988, p = 0.111.
T8	F-Test Two-Sample for Variances. t-Test: Two-Sample Assuming Equal Variances.	Not significant, F (28, 62) = 1.210, p = 0.2645. Not significant, females (M = 1.0074, SD = 0.0102) and males (M = 0.9839, SD = 0.008), t (1.0891) = 1.987, p = 0.279.
T9	F-Test Two-Sample for Variances. t-Test: Two-Sample Assuming Equal Variances.	Not significant, F (27, 62) = 0.8209, p = 0.248. Not significant, females (M = 0.9823, SD = 0.006) and males (M = 0.9547, SD = 0.0073), t (1.4414) = 1.9876, p = 0.153.
T10	F-Test Two-Sample for Variances. t-Test: Two-Sample Assuming Equal Variances.	Not significant, F (28, 61) = 0.6098, p = 0.0797. Not significant, females (M = 0.944, SD = 0.0056) and males (M = 0.9311, SD = 0.00913), t (0.6421) = 1.988, p = 0.5225.
T11	F-Test Two-Sample for Variances. t-Test: Two-Sample Assuming Equal Variances.	Not significant, F (28, 60) = 0.5596, p = 0.2741. Not significant, females (M = 0.9028, SD = 0.008) and males (M = 0.8912, SD = 0.0099), t (0.5235) = 1.988, p = 0.60199.
T12	F-Test Two-Sample for Variances. t-Test: Two-Sample Assuming Equal Variances.	Not significant, F (29, 61) = 0.62801, p = 0.08928. Not significant, females (M = 0.8568, SD = 0.0053) and males (M = 0.8293, SD = 0.0084), t (1.41121) = 1.987, p = 0.1617.
L1	F-Test Two-Sample for Variances. t-Test: Two-Sample Assuming Equal Variances.	Not significant, F (29, 66) = 1.006, p = 0.4761. Not significant, females (M = 0.8082, SD = 0.0063) and males (M = 0.7777, SD = 0.0062), t (1.735) = 1.9858, p = 0.0861.
L2	F-Test Two-Sample for Variances. t-Test: Two-Sample Assuming Equal Variances.	Not significant, F (30, 65) = 0.9653, p = 0.4718. Significant, females (M = 0.6884, SD = 0.0041) and males (M = 0.6558, SD = 0.0044), t (2.2141) = 1.9864, p = 0.0293.

Table 30. Continued. Results from the F-Test Two-Sample for Variances and t-Test: Two-Sample assuming (Un)Equal Variances tests for each VNC Ratio for vertebra (T1-L5) in the OpID sample.

Vertebra	Test	Significance
L3	F-Test Two-Sample for Variances. t-Test: Two-Sample Assuming Equal Variances.	Not significant, F (29, 64) = 0.9197, p = 0.4144. Significant, females (M = 0.6884, SD = 0.0043) and males (M = 0.6558, SD = 0.0044), t (2.2141) = 1.9864, p = 0.0293.
L4	F-Test Two-Sample for Variances. t-Test: Two-Sample Assuming Equal Variances.	Not significant, F (29, 62) = 0.8478, p = 0.3217. Significant, females (M = 0.6808, SD = 0.0032) and males (M = 0.6405, SD = 0.0038), t (2.9744) = 1.987, p = 0.0038.
L5	F-Test Two-Sample for Variances. t-Test: Two-Sample Assuming Equal Variances.	Not significant, F (29, 60) = 1.3041, p = 0.1938. Not significant, females (M = 0.6736, SD = 0.00748) and males (M = 0.6171, SD = 0.0057), t (3.1464) = 1.9876, p = 0.00226.

Table 31: ANOVA of VNC Ratio x SES levels in the TXSTDSC sample.

Anova: Two-Factor Without Replication

SUMMARY	***Count***	***Sum***	***Average***	***Variance***
PCOME	17	14.58	0.857647	0.021194
OPID	17	14.5929	0.858406	0.017791
TXST-Low	17	14.608	0.859294	0.019293
TXST-Low-Middle	17	14.181	0.834176	0.016649
TXST-Middle	17	14.768	0.868706	0.020042
TXST-Upper-Middle	17	14.7562	0.868012	0.018053
TXST-Upper	17	15.501	0.911824	0.02399
T1	7	5.0579	0.722557	0.00023
T2	7	6.07	0.867143	0.00062
T3	7	6.609	0.944143	0.001024
T4	7	6.918	0.988286	0.001135
T5	7	7.1932	1.0276	0.001336
T6	7	7.203	1.029	0.00173
T7	7	7.113	1.016143	0.001087
T8	7	6.892	0.984571	0.000457
T9	7	6.729	0.961286	0.001024
T10	7	6.727	0.961	0.002629
T11	7	6.326	0.903714	0.000671
T12	7	5.697	0.813857	0.000382
L1	7	5.528	0.789714	0.001184
L2	7	5.078	0.725429	0.000367
L3	7	4.703	0.671857	0.00081
L4	7	4.664	0.666286	0.000532
L5	7	4.479	0.639857	0.000777

ANOVA						
Source of Variation	***SS***	***df***	***MS***	***F***	***P-value***	***F crit***
Rows	0.056	6	0.009333	22.41706	2.36E-16	2.194516
Columns	2.152226	16	0.134514	323.0813	5.93E-76	1.749954
Error	0.039969	96	0.000416			
Total	2.248195	118				

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