THE EFFECTS OF GROWTH DISRUPTION ON ADULT SKELETAL MORPHOLOGY

By

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ANOVA	Analysis of Variance
AP	Anteroposterior
CBTS	Conservative bilateral tooth stress. An individual is considered "stressed" using this criteria if they have bilateral defects on their anterior dentition.
HL	Harris lines or radiopaque transverse lines
LEH	Linear enamel hypoplasia. This coding scheme does not recognize hypoplastic pitting. Only linear defects are scored as hypoplasias.
ML	Mediolateral
MNSE	Minimum number of stress events. This score was an attempt to approximate the number of individual stress events responsible for a particular type of stress indicator. For Harris lines, this score was calculated using the number of Harris lines present in a single bone. For teeth, the age ranges for multiple defects were considered.
RMA	Reduced major axis regression
SKH	Skeletal height
STH	Sitting height
TS	Tooth stress. This is the most lenient way of scoring enamel defects in this study. Any linear or pit defect is recorded as an indicator of stress, regardless of whether or not it is bilateral.
VBH	Vertebral body height
VNC	Vertebral neural canal

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Skeletal markers of growth disruption, enamel defects and radiopaque transverse lines, were compared to osteometric data to determine whether or not the stresses associated with the skeletal marker had an effect on adult stature, sexual dimorphism and proportions. Black males and females from the Terry and Hamann-Todd collections were used as the sample population. Previous research on the process of catch-up growth suggests that in all but the most extreme cases, the negative effects of growth disruption are erased; however, anthropologists use trends in adult morphology to estimate the relative environmental stress on populations. Resampling statistics determined that stressed females were significantly shorter than their unstressed counterparts when stress was determined by the presence or absence of enamel defects. Significant differences in stature were not found in males, nor were they found when Harris lines were used to determine stress. While significant differences in sexual dimorphism were not found between stressed and unstressed groups, it is notable that stress had a visible effect in the female skeletal sample, but not the male. This finding is in stark contrast to most theories regarding sexually differentiated responses to stress which presume that female growth is canalized while male morphology varies more in accordance with the environment. The crural index in males was the only proportional index investigated that was significantly related to

enamel defects. All other proportional differences observed were subtle. The data from this skeletal sample do not support the predicted models for how the environment affects human growth and morphology.

CHAPTER 1
INTRODUCTION

Studies of historic and archaeological populations use stature and sexual dimorphism as indicators of health status in a population. The recurrent fluctuations in mean height that are found through the analysis of historical records can be correlated with economic or environmental conditions during childhood (Komlos, 1994; Steckel, 1995, 1999; Steegmann, 1985, 1991). Similarly, osteological reports on skeletal populations often include measures of stature or sexual dimorphism with paleopathological data, providing information on the health of a population (Cohen and Armelagos, 1984; Goodman and Martin, 2002; Steckel and Rose, 2002). The theoretical basis for these studies rests on the idea that adult height is a product of both genetic potential and environmental influences: tall individuals may be closer to reaching their genetic potential in light of favorable environmental or economic circumstances. Changes in sexual dimorphism should also reflect physiological responses to environmental stress in that males and females do not respond equally to stressors (Stini, 1985).

It has been argued that growth stunting may be considered an adaptive advantage among the poor, where individuals are able to maintain a normal weight for height because it takes fewer resources to feed a smaller body than a larger one (Nickens, 1976; Seckler, 1980, 1982). However, those who consider stunting to be a major public health concern point out that there is nothing adaptive about malnourishment and infection, both known to contribute to stunting. Instead, stunting is used as an indicator for observing the more serious health effects of growth disruption such as impaired cognitive development and reduced fitness (Martorell, 1989; Waterlow, 1988a).

This research investigates whether or not there is a correlation between skeletal indicators of growth disruption and adult stature and proportions. The human skeleton records episodes of

growth disruption in the form of developmental defects in dental enamel and radiopaque transverse lines in long bones. Indicators of growth arrest serve as an independent variable for comparison with skeletal measurements to determine whether or not the stress associated with observable markers has any effect on adult stature and skeletal proportions.

Research suggests that the stunting associated with growth disruption may be erased depending on the severity, timing and duration of the metabolic insult (Cameron, 2002; Tanner, 1981). Growth disruptions are often only episodic and the human body experiences catch-up growth once the conditions which caused the growth disruption are removed. Stature as an indicator of environmental stress can be seen in juveniles prior to the completion of growth. For example, in undernourished children, skeletal age may be retarded in comparison to chronological age. This is an indicator that a normal growth trajectory has been disrupted. However, once skeletal growth is complete, growth disruption is much harder to detect. Anthropologists use secular trends in adult stature to estimate the relative environmental stress on populations. If catch-up is indeed complete after episodes of growth disruption, then fluctuations in stature should only occur under extreme circumstances, such as chronic (as opposed to episodic) undernourishment.

The null hypothesis in this study is that no metric differences will be observed between the size and shape of individuals who have experienced growth disruption compared to those who have not, due to the corrective process of catch-up growth. In addition, three alternative hypotheses will be tested.

The first alternative hypothesis proposes that individuals with independent indicators of growth disruption or stress will have a smaller estimated stature than those individuals without such indicators of stress. Studies documenting secular changes in stature claim that smaller

stature is an indicator of nutritional deprivation (Steckel, 1995). Such studies would suggest that indicators of growth disruption should be positively associated with smaller stature relative to individuals without such stress markers. If support is found for this hypothesis, then evidence suggests that catch-up growth following growth disruption is not always complete.

The second alternative hypothesis tested states that because female growth is believed to be canalized, or less affected by stress conditions than male growth, males and females with evidence of stress will exhibit less sexual dimorphism than those with no evidence of stress. Theoretically, changes in sexual dimorphism should also reflect physiological responses to environmental stress. The work of Stini (1969, 1985) has shown that the long-term effects of nutritional deprivation and other environmental stressors have a greater effect on males than females. Females, probably as an adaptation to the demands of lactation and gestation, store fat and nutrient reserves which are beneficial during periods of nutritional stress (Stini, 1982). As a result, female body size is canalized in comparison to male body size. While there is always some degree of size dimorphism in human populations, dimorphism increases when males are able to reach their genetic potential and decreases during periods of nutritional stress.

There are many ways to measure sexual dimorphism. Ruff (1987) states that changes in the degree of sexual dimorphism in long bone lengths may be more appropriate than cross-sectional dimensions for addressing questions of how nutrition or general health influences human morphology. Because the focus of this research is in how growth disruption, the product of ill health and/or malnutrition, affects sexual dimorphism, it is the dimorphism of long bone lengths and overall stature that will be analyzed here.

The third alternative hypothesis tested states that proportional differences will be observed between those with independent evidence of growth disruption and those without.

Bogin's (1999) studies of Japanese children have led to interesting conclusions about the relationship between genetics and environment. Bogin believes that genetic differences between Japanese and Europeans can explain differences in size, but that environmental factors may be "powerful determinants of body proportion" (Bogin, 1999:241). By attempting to control for genetic factors and using independent indicators of stress, this research provides a foundation for teasing out the effects of the environment on body size and proportion.

Proportional differences are measured in terms of relative trunk-to-limb ratios and differences in proximal and distal limb lengths. Stini (1985) and Tanner (1963) both state that proportional differences in human form do not occur as a product of growth disruption, but a number of animal experiments suggest that testing an alternative hypothesis is worthwhile (Williams and Hughes, 1975; Wilson and Osbourn, 1960). Studies of catch-up growth demonstrate that the process differentially affects body segments based on the stage of growth at which catch-up occurs; at least until a normal growth trajectory is resumed (Fleagle et al., 1975; Tanner, 1981). If proportional differences can be correlated with the timing of stress events, results may inform the understanding of mechanisms of catch-up growth.

While the process of catch-up growth has been well documented in animal models and in studies of human growth, very few of these studies follow individuals through to adulthood in any animal but laboratory rats. Assumptions are made about the environmental conditions of past populations based on stature estimations. Yet, there is no consensus on how catch-up growth affects adult human morphology.

The goal of this research is to bridge the empirical gap between the morphometric data collected by biological anthropologists and longitudinal studies of human growth. Osteological analyses assume a relationship between stature and stress. The importance of this research is that

it tests an assumption which has directed research in biological anthropology. This assumption has been used to measure human health and explain some aspects of human variation. For example, researchers investigating secular trends in stature are assuming an environmental effect. Meadows Jantz and Jantz (1999) state "environmental forces, such as nutrition and disease, are the usual causes of secular change in overall size." In short, all avenues of research assume that the environment affects human biology; the ways in which it is affected are not as clearly understood.

Chapters 2 and 3 provide background information for understanding growth, growth disruption and indicators of such disruption that appear in skeletal materials. Chapter 4 outlines the analytical approaches used to address the hypotheses outlined and Chapters 5 through 8 report the results of those analyses. In Chapter 9, the implications of these analyses are discussed.

CHAPTER 2
ADULT STATURE AND PROPORTIONS

Human Growth

Like all primates, humans have extended periods of growth in comparison to other mammals. From the gestational stage through maturity, there is a common pattern of growth in normal, healthy human children (Bogin, 1999; Eveleth and Tanner, 1990). Human growth can be divided into five basic stages: the prenatal period, infancy, childhood, the juvenile period, and adolescence. Although growth is continuous until maturity, growth rates are not constant.

In humans, our most rapid rate of growth occurs prenatally, particularly during the period just before birth (Stinson, 2000). Successful growth during the prenatal period is often measured by birth weight. Robson (1978) estimates that as much as 66% of the total variation in birth weight may be due to the prenatal environment as opposed to genetic factors, although others argue that it is less (Stinson, 2000). Infancy is the period from birth to approximately three years of age. The end of the infant stage is most often associated with the age at weaning[1] (Bogin, 1999; Stinson, 2000). The human growth rate slows throughout infancy.

Unlike the typical mammalian pattern, primates are among those animals which experience an extended growth period between weaning and puberty. Extended growth is believed to be correlated to an animal's need to socialize prior to reaching reproductive age (Bogin, 2003). Non-primate mammals exhibiting extended growth include animals such as wolves that live in complex social groups. In humans, this post-weaning period may be divided further into childhood and juvenile periods. During childhood (three to seven years), the growth rate remains constant and it decelerates during the juvenile period (seven to eleven years).

[1] Some sources consider infancy to end at age five or six based on the eruption of the first permanent tooth (Schultz 1969).

Adolescence is the period from around eleven years of age until the cessation of growth. It is during this period that a growth spurt occurs (Bogin, 1999). Although non-human primates have a life cycle more similar to humans than that of non-primate mammals, humans represent an extreme of the primate spectrum. For example, there has been much discussion regarding whether or not the adolescent growth spurt seen in humans is unique to the human species or whether other primates experience a similar growth spurt (Bogin, 1999; Leigh, 1996). Leigh (1996) demonstrated that humans are not unique among primates in experiencing an adolescent growth spurt in weight; nevertheless, the human growth spurt does occur at a later age than would be expected given the trajectory of other primates. Moreover, non-human primates do not appear to experience a spurt in skeletal growth of the magnitude or duration of growth spurts seen in humans (Bogin, 1999).

Throughout growth there are considerable changes in body proportions as well as in growth rates. In early growth, the brain and head are proportionally larger in comparison to the rest of the body. For example, by age five, the brain is already 90% of its adult weight despite the fact that linear growth does not cease for at least another ten years (Tanner, 1988). As an individual grows, relative limb length increases in comparison to the rest of the body (Bogin, 1999).

Bone formation begins *in utero* due to the ossification of preexisting connective tissues. The flat bones of the cranial vault, the mandible and the clavicle are referred to as intramembranous bone because ossification occurs within sheet-like membranes. Most of the bones included in growth studies (including vertebrae and limbs) are referred to as endochondral bone because ossification begins in masses of hyaline cartilage, referred to as cartilage anlagen, which are in the location and approximate shape of the future bone.

Blood vessels and osteoblasts (bone forming cells) from the periosteum begin to invade the center of the cartilage model during the second or third intrauterine month (Steele and Bramblett, 1988). This becomes known as the primary center of ossification and bones develop in the direction of their cartilaginous ends. In human long bones, secondary centers of ossification develop in the epiphyses. There are approximately 450 ossification centers at birth. As a general rule, most primary centers of ossification develop before birth and most secondary centers develop after birth (White and Folkens, 2005). The cartilaginous band that resides between the epiphysis and diaphysis is referred to as the epiphyseal plate. As new chondrocytes undergo cell division, older ones adjacent to the shaft ossify. Long bones lengthen through this process while the epiphyseal plates are active until maximum length is reached when the diaphysis and epiphysis unite. The timing of epiphyseal closure is variable; however, in general, females mature one to two years earlier than males. The distal epiphysis of the humerus is the first epiphyseal plate to fuse in human long bones. This may begin at as early as nine years in females. The humeral head, one of the last plates to fuse, may not be complete until as late as 24 years in males (Buikstra and Ubelaker, 1994).

As a bone lengthens, the diameter of the shaft also increases as osteoblasts form compact bone beneath the periosteum. Osteoclasts dissolve bone adjacent to the marrow cavity, increasing both the cross sectional diameter of the shaft and the size of the medullary cavity. Even after adolescence, this process slowly continues throughout the life of an individual.

Growth Disruption

Stature and proportions, such as sitting height, can be used as indicators of environmental stress prior to the completion of growth. For example, in undernourished children, skeletal age

may be retarded in comparison to chronological age, an indicator that a normal growth trajectory has been disrupted.

> A child's growth rate reflects, perhaps better than any other single index, his state of health and nutrition; and often indeed his psychological situation also. Similarly, the average values of children's heights and weight reflect accurately the state of a nation's public health and the average nutritional status of its citizens, when appropriate allowance is made for differences, if any, in genetic potential. (Eveleth and Tanner, 1990:1)

Longitudinal and cross-sectional studies of growth have been used to create standards or references for growth to help identify growth disruption (Tanner, 1986). Ideally, these standards are population specific and are based on data from healthy individuals in a given population (Eveleth and Tanner, 1990). Limiting standards to healthy individuals is an attempt to control for environmental effects, and population specific studies attempt to control for genetic effects. For example, comparisons of growth trajectories between children of African and European descent demonstrate differences in axial and appendicular growth; black children have shorter trunks, longer legs, and longer forearms than their white counterparts (Hamill et al., 1973; Nyati et al., 2006).

For an individual child, mid-parental height (defined as the average height of an individual's mother and father) can be used to estimate a target range for growth as well as to predict a child's final height (Tanner, 1986). Simple height-for-age formulae are problematic due to genetic effects. For example, because of variations in average population heights, the World Health Organization (WHO) relies more heavily on weight-for-height data than height-for-age data (WHO, 1986). Because stunting is cumulative and catch-up growth can erase growth stunting over time, it is difficult to make assumptions about growth disruption based simply on height-for-age data. An example given in the Bulletin of the World Health Organization (WHO, 1986) used the following example: if a four year old is found to have a greater height-for-age disparity than a two year old, it is not necessarily because the four year old

is more malnourished. Statural growth is cumulative so the process of deprivation and subsequent retardation may have just had more time to affect the four year old.

Microbial infections have been found to have a significant effect on human height (Beard and Blaser, 2002). Not only are there are significant associations between bacterial infections and growth, but historical data find height reductions when microbial transmission rates are high. While growth is said to reflect health and nutrition (Eveleth and Tanner, 1990), the two are inexorably intertwined. For example, *Helicobacter pylori* infections, one of the most common infections in humans worldwide, have been found to have a negative association with growth when coupled with iron deficiency anemia (Choe et al., 2000). Deficiencies in iron, vitamin A, vitamin D, protein, thiamine, ascorbic acid, riboflavin, niacin, zinc, folic acid, and vitamin B12 are just a few of the nutrients that have been associated with inhibited antibody formation (Calder and Jackson, 2000; Scrimshaw et al., 1968). Scrimshaw et al. (1968) found that not only were the consequences of infection more severe when malnutrition was an issue, but often infectious diseases made minor nutritional deficiencies much worse.

Malina (1987) defines six classes of nutrients: carbohydrates, fats, proteins, vitamins, minerals and water. Carbohydrates provide energy; proteins are primarily responsible for the growth, maintenance and repair of tissue; and vitamins and minerals serve regulatory functions. When caloric intake is too low, protein will be used for basic energy needs. Once the basic needs are met, only then does the body devote resources to skeletal growth. There are numerous cases where nutritionally deprived children are of normal weight-for-height, but are stunted in height-for-age compared to adequately nourished children (Eveleth and Tanner, 1990). It is in this sense that "height at a particular age reflects an individual's history of net nutrition" (Steckel, 1995:1910).

Researchers have attempted to tease out the different dietary components to see which have the greatest effect on growth. Observations of growth patterns in developing countries have led researchers to conclude that low protein has the most impact on growth of any nutritional deficiency (Martorell and Habicht, 1986; Stini, 1971). Nutritional supplementation projects in developing countries have served as natural experiments supporting this claim (Silventoinen, 2003). However, caloric as well as protein deficiencies have been found to negatively affect collagen production, a protein which accounts for approximately 90% of a bone's organic material. Vitamin D and calcium deficiencies have long been associated with poor mineralization of bone, but evidence suggests that deficiencies in a number of micronutrients may also affect growth (i.e., zinc, phosphorus, magnesium, vitamin A, and iron) (Allen, 1994, 1995; Prentice and Bates, 1994). An attempt to correlate dietary protein, as evidenced through stable isotope analysis, with femoral length in a South African bioarchaeological sample proved to be unsuccessful (Pfeiffer and Sealy, 2006). Thus, in most human populations, dietary deficiencies probably involve multiple nutrients making it difficult to parse out the effects of various dietary components. For example, low protein diets are often low in energy and important micronutrients (Allen, 1994).

Stini's research on sex differences in response to nutritional stress led him to conclude that males suffered more as a consequence of protein deprivation, leading to a decrease in sexual dimorphism in protein deprived populations (Stini, 1969, 1973, 1975a, 1975b, 1982). This finding is believed to be the linked to the greater fat and nutrient reserves found in females. These fat and nutrient reserves are thought to be an adaptation for the increased metabolic demands of lactation and gestation in producing offspring. Energetic stress results in lower fecundity for women, but the same patterns have not been found in men (Bribiescas, 2006;

Ellison, 2003). Spermatogenesis requires a negligible energetic investment. Testosterone levels drop during periods of energetic stress, but this does not affect fecundity and testosterone levels quickly rebound when conditions improve. Testosterone affects muscle anabolism and male physiology favors reproductive function at the expense of building muscle during periods of energetic stress (Bribiescas, 2001, 2006; Ellison, 2003). As a result of these physiological differences, males experience a greater reduction of lean body mass during periods of nutritional inadequacy than do their female counterparts. When periods of starvation occur during growth, the reduction in body mass is accompanied by reduced skeletal growth (Stini, 1975a). On the other hand, Stinson (1985) did not find irrefutable support for the hypothesis of sex differences in environmental sensitivity, likely due to sex-biases in parental investment. Equality in stress levels of male and female children should not be assumed.

Severe undernutrition leads to stunted adult size, but some researchers have questioned whether or not stunting "matters (provided the brain escapes any lasting effect)" (Eveleth and Tanner, 1990:195). Furthermore, Eveleth and Tanner suggest an adaptive advantage to small size under adverse conditions, because smaller body size requires fewer resources (Márquez and del Ángel, 1997; Nickens, 1976; Seckler, 1980; Stini, 1975a). Assertions regarding the "adaptive" nature of stunting are controversial because of the inherent costs and questionable benefits to stunted individuals. Some researchers argue that small size as a result of deprivation cannot have adaptive advantages due to the negative processes, such as infection and malnutrition, which cause growth stunting (Gopalan, 1988; Martorell, 1989). A review of data on the subject found that smaller individuals were not able to perform more work relative to body size, nor did they experience reproductive advantages during times of scarcity (Stinson, 1992). Likewise, mortality profiles differentially favor taller individuals (Saunders and Hoppa,

1993), and the conditions that lead to stunting also negatively affect cognitive development (Colombo et al., 1988; Martorell, 1989).

Catch-up Growth

Growth disruptions are often only episodic and the human body experiences a process called "catch-up growth" once the adverse conditions which caused the growth disruption are improved (Cameron, 2002; Tanner, 1981). Catch-up may be achieved through an increase in the rate of growth or by postponement of growth completion, thereby lengthening the growth period. In a growing individual these processes can be recorded in the skeleton; however, once skeletal growth is complete, it is more difficult to ascertain whether an individual experienced disrupted growth or subsequent catch-up growth. While catch-up growth has been observed clinically in pediatric case studies and has been recreated in laboratory experiments on non-human animals, the mechanism behind catch-up growth is poorly understood. Tanner (1963) theorized that catch-up growth is a systemic, neuroendocrinologial process that recognizes a mismatch between the present and potential size and then adjusts the growth rate through a "target seeking" process. Tanner admitted that the hypothesis he presented was speculative, but thought that growth experiments of the time were hampered by the lack of appropriate theoretical models under which to work (Tanner, 1963). In contrast, Williams and Hughes (1975) concluded that Tanner's hypothesis was too simple. Because catch-up growth appeared to affect different body parts in different ways, these researchers felt that the resultant growth after rehabilitation was the product of an interaction between the catch-up stimulus and the normal growth stimuli (Williams and Hughes, 1975).

Baron et al. (1994) suggested that the primary mechanism of catch-up growth resided at the growth plate rather than in the central nervous system. Their hypothesis was based on an

experiment in which growth was artificially suppressed in a single growth plate. Rabbits were locally administered glucocorticoid unilaterally in the proximal tibia. The growth suppressant was removed prior to growth completion. The experimental tibia never attained the full size of the control; however, the growth rate in the experimental tibia surpassed the rate of growth in the control side. Left and right femoral lengths were not statistically different offering further support for the conclusion that catch-up growth was localized to a single epiphyseal plate. While this experiment does not rule out the possibility that a regulating mechanism exists at the systemic level, it does suggest there may be a more localized response in individual bones.

Due to the long human growth period and the difficulty in controlling for confounding variables in human populations, animal experiments have proved useful in providing models of catch-up growth. Mammalian models of growth are often tested on mice or rats likely because of their relative availability, low cost and short life-span. However, the utility of applying non-primate mammal models to humans is hampered most notably by their differences in life cycles. These differences are incredibly important when trying to apply the timing of nutritional insults in animal models to humans. First, it is likely that the early postnatal life of a mouse is analogous to prenatal stages in a human. An excellent case in point is a study by Grove et al. (2005), which investigated the development of metabolic systems in relation to overfed and underfed animals by performing experiments on both rodents and primates (*Macaca fuscata*). They found that the neurons for transmitting hormonal signals that modify feeding and energy expenditure develop in week three postnatally for rodents and in the third trimester for primates (Grove et al., 2005). In addition, rodents do not have a juvenile period comparable to humans (Bogin, 1999). Because human infants are even more altricial at birth than non-human primates, growth periods in young rats and mice may be comparable to earlier prenatal growth in human

analogues. In addition, the presence of the adolescent growth spurt in humans cannot be matched in the rat; even compared to other primates, the absolute growth at puberty is greater in humans (Bogin, 1999).

Another criticism of using rat models for studying human growth is that skeletal growth in humans and rats may be too different to be compared. Tanner (1963) claims rats do not experience full epiphyseal closure. In contrast, Martin et al. (2003) not only documented the rates of epiphyseal closure in rats, but they suggest that the rates are subject to environmental pressures in a manner similar to those in humans. Rat bone does differ from humans, however, in that there is no secondary remodeling of cortical bone. The process of bone remodeling removes and replaces older bone thereby repairing microscopic fatigue damage (Martin et al., 1998). Due to the short lifespan and small size of rodents, remodeling would be a worthless energetic expenditure. Martin et al. (2003) concluded that rat models are useful for studies of growth, but not for studies of aging.

Genetically, the breeding populations of rodents and non-human primates used for laboratory research are fundamentally different. The breeding of rodents for experimental research is controlled in an attempt to create a genetically defined animal model, thereby eliminating a certain degree of variation between individuals. In captive primate populations, where selective breeding occurs, the attempt is generally to maintain genetic diversity within those populations (Ribeiro Andrade et al., 2004). While the ultimate effect of the relative genetic variability between primates and rodents may be small, it is important to recognize that the genetic effect on growth is likely to be a greater confounding variable in non-human primate studies than it is in rodent experiments.

The majority of catch-up growth studies performed on rats or mice focus on the timing and duration of nutritional insults (Farnum et al., 2003; Widdowson and McCance, 1963; Williams and Hughes, 1975). While the results of these studies have varied, they have proven useful in establishing two facts. First, there is evidence that there are critical stages during growth at which the effect of growth disruption is greater. Second, catch-up growth follows three possible trajectories: a rapid growth spurt when conditions improve, an extended period of growth, or, in extreme cases, both the rate and duration of growth may increase (Boersma and Wit, 1997).

The most important limitation of catch-up growth studies on non-human primates is also one of the major hurdles in conducting longitudinal studies of human growth – the time it takes for an individual to reach full adulthood. As such, not all growth studies of non-human primates have followed subjects through to growth completion. For example, Fleagle and colleagues (Fleagle et al., 1975; Fleagle and Samonds, 1975) demonstrated that the response to growth arrest was not uniform throughout the body of *Cebus albifrons*. When resources are limited, they appear to be channeled from the most mature skeletal elements toward those with the most growing left to do. However, because the animals used in this study were not followed through adulthood, it could not be determined whether or not the effects of protein deficiency were permanent (Fleagle et al., 1975; Fleagle and Samonds, 1975). While the findings of this study were very provocative and have been frequently cited in subsequent studies of catch-up growth, data on those animals are not available past two years of age. It was believed at the completion of the study that the animals had reached a normal growth trajectory, but *Cebus albifrons* does not reach physical maturity until around the age of five years. With the exception of rats, who are relatively short-lived, catch-up growth studies rarely follow individuals experiencing growth arrest to the age of skeletal maturity.

There is disagreement regarding the degree to which catch-up growth occurs in humans. The results of catch-up studies differ, likely due to variation in the duration, severity and timing of the insults, as well as in the triggers initiating catch-up growth. Numerous examples exist which prove that catch-up growth can and does occur in human populations, although the degree of recovery varies (Delgado et al., 1987; Johnston and MacVean, 1995; Li et al., 2004; Steckel, 1987). For example, height data recorded for American slaves provide notable evidence for catch-up growth. The average heights of American slave children were around the first centile compared to modern standards. However, young adult males were in the 25th centile and females were in the 30th centile based on the same standards (Steckel, 1987). This pattern of growth is unusual and is thought to reflect improved nutrition during growth. Young children were weaned from breast milk relatively early and were fed a poor diet. Rates of infant and childhood mortality were high among American slaves, due in part to the fact that it was not until children entered the work force, by the ages of eight to twelve, that they received a worker's allocation of meat. It was at these ages that the process of catch-up began and height records suggest that growth continued until 19 years in females and 21 years in males (Steckel, 1987). Martorell et al. (1994) compiled evidence of other cases in which catch-up occurred, but notes that evidence suggests catch-up is more effective when the conditions improve at younger ages. Stinson (2000) points out that in studies of catch-up growth at the population level, increased mortality of smaller individuals may contribute to the appearance of catch-up growth. The surviving population may no longer include its shortest members.

While the potential for catch-up growth is apparent, Martorell et al. (1994) found that retarded growth in early childhood resulted in shorter adults in most cases. Li et al. (2004) analyzed longitudinal data from the 1958 British birth cohort and confirmed this position.

Disadvantaged children experienced sizeable height deficits (two to three centimeters) at age seven years. Catch-up growth erased some, but not all of this deficit, resulting in adults that were one centimeter shorter on average (Li et al., 2004). When growth is stunted, the growth trajectory changes, lengthening the period in which growth can occur (Golden, 1994). However, unless an individual's environment changes and there is sufficient time left for growth, stunting will persist into adulthood. Age at menarche is used as a marker for determining maturation in human populations. Data on populations worldwide compiled by Eveleth and Tanner (1990) found that menarche was delayed in chronically undernourished and high altitude populations; however, in most cases, Martorell et al. (1994) did not find that the delay was long enough to fully compensate for growth stunting. In fact, Martorell et al. (1994) find some evidence that for those whom experience accelerated growth late in the growth period; maturation may be triggered, leading to short adult stature. The conclusion of these researchers is that, although complete catch-up is possible, for most stunted children the environment does not change, leading to shorter adults (Golden, 1994; Martorell et al., 1994).

Timing of Growth Disruptions

Because the rate of growth is not constant from infancy through adulthood, it has been suggested that the relative sensitivity to episodes of growth disruption also varies. Eveleth and Tanner (1990) believe that growth is most sensitive to nutritional deprivation during times of peak growth velocity: infancy and adolescence. Experiments on animal models where test subjects were nutritionally deprived during various stages of growth do provide some evidence for the differential effects of growth disruption in relation to body segments which grow at a faster or slower rate. In rats, it has been found that the effects of undernutrition become progressively less important with age (Widdowson and McCance, 1963). Tanner's (1962)

synthesis of research on animal husbandry found that animal shape was affected by both the timing and intensity of insult; faster growing body segments appeared to suffer the most from nutritional insult. Wilson and Osbourn (1960) found that the rate of growth in late maturing regions of the body was most affected by growth insults in mammals and birds. However, because these regions had greater time to recover, the growth disruption is not evident in the adult form.

In the developing world, research suggests that poor environmental circumstances can cause a normal growth trajectory to falter by six months of age, if not earlier (Waterlow, 1988b). In studies of secular trends in stature, Hauspie et al. (1996) and Stinson (2000) conclude that the vast majority of differences seen in adult height can be explained by growth disruptions prior to six years of age. Not only are growth disturbances greatest in the first two to three years of life, but nutritional stresses are also greatest during this time period (Beaton et al., 1990; Martorell and Habicht, 1986; Martorell et al., 1995).

Stinson (2000) lists the cultural norms surrounding breastfeeding as one of the most important culturally mediating factors associated with growth. Weaning is a particularly dangerous time of life because food is a source of pathogens and children no longer have the immunities provided by mother's milk; rates of mortality tend to spike at this phase of life. To compound the problem, the nutritional needs are great during this phase of life because the growth velocity is high (Martorell et al., 1994).

Because growth appears to be more sensitive to environmental circumstances in early childhood (Hauspie et al., 1996; Martorell and Habicht, 1986; Martorell et al., 1995; Stinson, 2000; Wadsworth et al., 2002), and legs are the fastest growing region of the body from birth until puberty (Dangour, 2001; Floyd, 2007; Gunnell, 2002; Tanner, 1962), it stands to reason that

individuals who experience growth disruption in childhood should have proportionally shorter lower limbs. Numerous studies on human proportions have found support for this hypothesis, including studies of secular change.

Luo and Karlberg (2000) examined longitudinal data to see how different growth phases interacted, leading to adult shortness. The shortest adults experienced subnormal growth during multiple growth phases. When growth data are compared with midparental height, the relationship between genetics and height was not apparent until after infancy. Luo and Karlberg (2000) found that most cases of growth faltering during and after childhood were erased through catch-up growth, but that the same is not true during the fetal and infant stages. In a subsequent study, Luo et al. (2001) found that adult shortness was associated with subnormal growth in any growth phase. They concluded that short adult stature in the developing world was primarily due to environmental factors rather than genetics because the stature data were adjusted by mid-parental height, a proxy for the genetic effect.

Despite being a period of rapid growth, adolescence is not considered to be a peak period of permanent growth stunting. Growth during adolescence correlates more closely with genetics than with environmental circumstances (Frisancho et al., 1980; Johnston et al., 1976; Stinson, 2000). During adolescence, large strides can be made in growth under seemingly poor environmental circumstances. Adolescent growth patterns are more similar between individuals of similar genetic backgrounds than between individuals of similar socioeconomic classes (Frisancho et al., 1980; Johnston et al., 1976). However, the physical demands of this rapid growth phase are great and the adolescent growth spurt would mask growth rate changes associated with stunting during this phase, making it harder to detect. One study found that although adolescent and early childhood growth are statistically independent, short adolescents

experience more stunting than their taller peers during periods of hardship (Hermanussen, 1997). Fogel (1986) suggests that growth disruptions in adolescence lead to a delay in growth while it is the early disruptions which have a permanent effect. Studies on adolescent growth disruption indicate that growth is not immune to environmental privation during adolescence, but the ultimate effects are not as easy to detect.

Experimental research on the effect of temperature, physical activity and nutrition on bone elongation supports the theory that the early postnatal growth is the most critical phase (Serrat et al., 2009). When catch-up growth occurs, it generally does so by extending the growth phase or increasing the rate of growth. In other words, growth disruptions change ontogenetic trajectories through heterochronic processes. Allometric differences are due to the fact that different body segments have different growth profiles (Vrba, 1996).

Adult Proportions

Fleagle and Samonds (1975) did not suggest that the effects of growth disruption seen in *Cebus albifrons* were permanent, but their findings raised the question of whether or not proportional differences can be seen in individuals who have experienced catch-up growth as opposed to those who have never suffered growth disruption. Some, but not all, of the catch-up growth studies using rats have found proportional differences in final form. For example, Williams and Hughes (1975) found that catch-up in nose to rump length of the experimental rat group neared adult length in the control group. However, tail length in the experimental group never attained control levels.

Early studies of growth disruption suggest that although adult body size can be affected by growth disruption, changes in proportions would be rare if they were to occur at all (Stini, 1985; Tanner, 1963). It is widely accepted that human proportions (i.e., limb-to-trunk ratios, etc.) appear to be under strong genetic control (Bailey et al., 2007; Bogin and Rios, 2003; Eveleth and

Tanner, 1990; Hamill et al., 1973; Holliday, 1997; Mueller, 1986; Turan et al., 2005; Warren et al., 2002), and that these generally conform to the geographic expectations of Bergmann's and Allen's rules (Baker, 1988; Hiernaux et al., 1975; Holliday and Falsetti, 1995; Katzmarzyk and Leonard, 1998; Kurki et al., 2008; Roberts, 1953; Roberts, 1973; Ruff, 1991; Ruff, 1994). However, deviations in ecogeographic patterning also have been attributed to environmental effects such as health care and nutrition (Katzmarzyk and Leonard, 1998; Kurki et al., 2008). Differences in proportions within populations suggest environmental pressures affect human proportions.

Several studies have found that lower limb length is more sensitive to environmental change than trunk length, which in turn leads to differences in proportions. With an improved developmental environment, lower limb length increases at a greater rate than relative sitting height (Bogin and Rios, 2003; Bogin et al., 2002; Dangour, 2001; Floyd, 2007, 2008; Fredriks et al., 2005; Frisancho et al., 2001; Katzmarzyk and Leonard, 1998; Li et al., 2007; Malina et al., 2004; Stinson, 2000; Tanner, 1992; Wadsworth et al., 2002). Other studies have found differences in relative lower limb length based on factors such as altitude or activity pattern (Bailey et al., 2007; Theintz et al., 1993). Heart disease and associated risk factors are inversely related to lower limb length while the relationship to trunk length is not as strong (Davey-Smith et al., 2001). Fredriks et al. (2005) state that relative lower limb length may be a more sensitive indicator of environmental circumstances than height.

The relationship of proximal and distal limb elements is another proportional measure that has been associated with environmental effects. The length of the tibia has proven to be more variable than other limb segments, and distal elements tend to be more variable than proximal limb segments (Holliday and Ruff, 2001; Smith and Buschang, 2004). Secular allometric trends

have shown that as the environment improves the relative length of the tibia increases (Jantz and Jantz 1999; Jantz and Owsley 1984; Meadows and Jantz 1995). Longitudinal studies also demonstrate the relative sensitivity of the femur to environmental change (Smith and Buschang, 2004).

Secular Trends in Adult Height

Liu et al. (2004) estimate the heritability of stature to be above 0.75 while recognizing that nutrition and disease play a role in determining adult height. Adult stature is a complicated indicator of growth performance because it is difficult to determine what an individual's ultimate height could be under ideal conditions. Estimates of stature can be made based on mid-parental height, but there is a great deal of variation between individuals. Therefore, secular trends in adult height are compiled at the population level where individual genetic effects are canceled out and differences in height can be attributed to the environment (Steckel, 1995). Secular trends in stature refer to short term changes in growth and development rather than changes at the evolutionary level (Ruff et al., 1984). Looking at intergenerational differences within family lines is another means by which individual variation is controlled in secular studies (Damon, 1969; Floyd, 2007, 2008).

Secular studies use historic data or skeletal samples to observe trends or fluctuations in human morphology over time. In particular, military, medical, prison and slave records have provided a wealth of data for observing these trends historically (Fogel, 1986; Komlos, 1994; Steegmann, 1985; Steegmann and Haseley, 1988; Van Wieringen, 1986). Availability of height data on the average population has become more available since the mid 18th century (Steckel, 1995). The general trend over the last 100 years is that people are growing larger, faster and that physical maturation occurs at a younger age (Eveleth and Tanner, 1990; Lieberman, 1982). This being said, it is important to realize that the average European American is only a few

centimeters taller than they were 100 years ago; heights have fluctuated cyclically rather than steadily increasing (Fogel, 1986; Steckel, 1995). The fact that fluctuations in height correspond to the economic, nutritional, and social circumstances at the time of a specific birth cohort is of particular interest in secular studies of adult height. On the other hand, expectations of stunting were not met in one study of a mid 19th century poorhouse raising the question of the degree of severity in conditions needed for stunting to occur (Steegmann, 1991).

Presumably, secular increases in height can be attributed to the fact that today we are subjected to fewer growth inhibitors than in the past and are therefore more likely to reach our genetic potential (Lieberman, 1982; Stini, 1975a). Secular change is often attributed to changes in nutrition, but there are numerous other factors that have been associated with changes in height. Improvements in hygiene and sanitation have reduced exposure to infectious agents, allowing children to utilize nutrients for growth. Height has also been associated with urban/rural residence patterns, reduced family sizes and occupation. It has been suggested that selection for taller mates has led to increased heights (Hauspie et al., 1996; Huss-Ashmore et al., 1982; Malina, 1979; Steegmann, 1985). As populations have migrated, heterosis has become a factor, but Malina (1979) suggests that the effect of heterosis is small at best in human populations.

By correlating birth dates, height and historical data, researchers find that nutrition has the greatest impact on height from the prenatal period through early childhood (Fogel, 1986; Hauspie et al., 1996; Hermanussen, 1997; Steegmann, 1985). More specifically, the greatest secular changes are seen in lower limb length rather than trunk length (Floyd, 2007; Himes, 1979; Malina et al., 2004; Stinson, 2000). In addition, it appears that the distal limb is affected more than the proximal limb segment (Floyd, 2008; Meadows Jantz and Jantz, 1999). Thus, the

results of secular studies follow the same trends found in studies correlating human proportions and environmental stress.

Multiple studies have found that secular increases are greater in males than females (Engerman, 1994; Himes, 1979; Meadows Jantz and Jantz, 1999; Kuh et al., 1991). As discussed previously, some researchers have observed differential response by males and females to negative environmental conditions, which may explain the findings in secular studies (Stini, 1969, 1973, 1975a, 1982). The theoretical basis for this finding will be discussed in further detail in the following section on sexual dimorphism.

Within the last 50 years, researchers have questioned whether the secular trends observed from the mid 1800's through the mid 1900's were still occurring. Most evidence suggests that the secular trend has slowed if not stopped. Damon (1969) analyzed data on Harvard families from 1870-1965 and found that the intergenerational change in males was initially strong, but then declined. In fact, Damon suggested that the secular increase in height has peaked. Other researchers have concluded that the secular trend has slowed or stopped in developed countries, but persists in the developing world (Hauspie et al., 1996; Roche, 1979).

If stature can be used to track environmental stress in human populations, and failure to reach genetic potential can be attributed to environmental stressors, the most reasonable mechanism for stunted growth must involve growth disruption and incomplete catch-up growth. Therefore, it is desirable to investigate how growth disruption and catch-up growth affect adult morphology. Secular studies of adult form rely on understanding how growth disruption affects the end product, and unfortunately there are many assumptions that have not been tested. For example, many studies of growth disruption in humans do not follow individuals from birth through to growth completion (Checkley et al., 1998; Liu et al., 1998; Nabarro et al., 1988;

Smith and Buschang, 2004); thereby ignoring potential catch-up. The longitudinal studies on growth that have been completed have concentrated on looking at patterns of growth rather on the effects of growth disruption (Garn and Rohmann, 1966; Maresh, 1955). As such, even when environmental effects can be assumed, a direct relationship between the environment, growth disruption and catch-up growth cannot be systematically tested. Knowledge of the environment can only be gleaned through factors like social status (Kuh et al., 1991) or maternal smoking during pregnancy (Li et al., 2004). While this information is useful, it does not provide a full picture of the environmental circumstances, nor does it provide a record of chronic or acute stress events at the level of the individual.

Sexual Dimorphism

There are many ways to measure sexual dimorphism. Although in living populations stature is probably the most common measurement of size, simply because of the ease of measurement, the type of measurement considered is often determined by the questions being asked of the data. Ruff (1987) suggests that cross-sectional studies of diaphyses are evidence of the mechanical forces applied to bone, reflecting activity patterns during life. On the other hand, Ruff states that changes in the degree of sexual dimorphism in long bone lengths may be more appropriate for addressing questions of how nutrition or general health influences human morphology. While size and shape are correlated in considerations of human form, Ruff (1987) makes an interesting proposal for how to best measure human sexual dimorphism.

It is also important to consider the scale on which to consider changes in sexual dimorphism. Studies of change in the degree of sexual dimorphism in anatomically modern human populations provide information on a secular rather than an evolutionary level. While some forces may act at the evolutionary and secular scale, others do not appear to be operative on both levels. Evolutionary forces change the genetic composition of a species and are used to

explain the differences observed between species or over spans of time representing hundreds if not thousands of generations. The time depth of secular changes may be generational or even longer, but fluctuations in secular trends suggest that they are not genetic changes.

The degree of sexual dimorphism in *Homo sapiens* has not been consistent throughout time and has been attributed to many factors including genetics, sexual selection, activity patterns and nutrition. The trend has been that sexual dimorphism declined from the Upper Paleolithic to the present (Borgognini Tarli and Repetto, 1997; Brace, 1973; Brace and Ryan, 1980; Frayer, 1980; Frayer, 1981; Frayer and Wolpoff, 1985; Meiklejohn et al., 1984). These decreases have been associated with changes in subsistence or technology, most notably at the end of the Upper Paleolithic when large scale extinctions led to hunting of smaller game (Brace and Ryan, 1980; Frayer, 1980, 1981). While the length of both male and female limb segments declined at that time, the decline was greater in males, therefore reducing the degree of sexual dimorphism (Frayer, 1981).

Researchers have also hypothesized that a further reduction in sexual dimorphism occurred as humans shifted from a hunting and gathering economy to agriculture (Armelagos and Van Gerven, 1980; Boyd and Boyd, 1989; Frayer, 1980; Frayer and Wolpoff, 1985; Hinton and Carlson, 1979; Kennedy et al., 1987; Lazenby, 2002; Ruff, 1987; Wolfe and Gray, 1982). In contrast to data available for the Upper Paleolithic – Mesolithic transition, which strongly support a decrease in sexual dimorphism, data for a possible reduction in sexual dimorphism after the shift to agriculture are more equivocal, despite larger samples (Vick, 2005). These changes have helped inform researchers investigating the processes controlling sexual dimorphism.

While recognizing the primacy of heritability in the determination of stature (estimated to be above 0.75), Liu et al. (2004) recognized that nutrition and disease also play a role in determining adult height; likewise, variation in sexual dimorphism leads researchers to conclude that genetic as well as environmental factors influence sexual dimorphism. Eveleth (1975) found that the degree of sexual dimorphism varied between populations; but did not meet the expectations of the nutritional hypothesis – that the most dimorphic populations should have the highest quality diet. As such, Eveleth concluded that there must be a genetic factor controlling the level of sexual dimorphism in each population. Gustafson and Lindenfors (2004) indicates that both male and female stature is phylogenetically controlled; however, there is no evidence that the degree of sexual dimorphism is significantly more or less pronounced in populations that are larger in stature. The results of this study indicate a genetic component to sexual dimorphism while challenging the notion that relative sexual dimorphism is a byproduct of overall size.

Above the species level, Rensch's rule states that sexual dimorphism increases with body size in taxa where males are the larger sex (Rensch, 1959). This relationship may not be as strong in primates as in other taxa (Frayer and Wolpoff, 1985); nevertheless, Leutenegger and Cheverud (1985) found that variation in body weight could explain 83% of the variance in weight dimorphism among primates suggesting body weight is the major factor contributing to sexual dimorphism in body size. Among hominoids, orangutans and gorillas, the largest members, are the most sexually dimorphic; however, reports of relative dimorphism within chimpanzees and humans can differ depending on the method or element used to make the measurement (Lovejoy et al., 1989; McHenry, 1991; Richmond and Jungers, 1995).

Male-male competition is a common explanation for how sexual dimorphism develops. Because there is greater reproductive variance in males (Chagnon, 1979; Trivers, 1972), larger

body size may offer an advantage to males engaged in aggressive encounters, allowing them to pass their genes to subsequent generations more effectively. Male-male competition is greatest among primates where females assume the largest share of energy investment in the success of offspring (Trivers, 1972). Based on the theory of sexual selection, sexual dimorphism should be greater in polygynous rather than monogamous societies because there is more competition for access to females. Gray and Wolfe (1980) tested this hypothesis in humans and found no significant correlation between sexual dimorphism and mating pattern. Gaulin and Boster (1992) reanalyzed the data of Alexander et al. (1979) and found that sexual selection did not explain variation in sexual dimorphism when applied to cross-cultural studies in humans. However, they do not rule out the possibility that sexual selection has an effect in human populations, suggesting that perhaps human marriage practices have not been stable through time, thus obscuring any observable effects (Gaulin and Boster, 1992).

Ethnographic studies also show that the quality of parental investment varies depending on the gender of the offspring. Because the variance in reproductive success is generally greater for males, parents who can afford to invest in their offspring have a potentially greater return by investing in males (Hrdy, 1990). Rivers (1982) studied survival rates under conditions of famine and disaster and found that while females may have a natural advantage under times of stress, males often receive cultural advantages that may more than make up for any differential survival rates. Culture thus may affect the degree of sexual dimorphism manifest in a population.

Holden and Mace (1999) found that sexual dimorphism in stature is negatively correlated with the amount of work women perform. Likewise, female juvenile mortality rates are higher than those for juvenile males in areas where females contribute less to subsistence. These patterns follow geographical patterns of sexual dimorphism and may suggest that more resources

are allocated toward female children when there are economic returns for such investments (Holden and Mace, 1999).

In addition, biomechanical data suggest that reductions in the division of labor, as seen with the transition to agriculture, lessen the sexual differences found in the cross-sectional properties of bone (Ruff, 1987). Holden and Mace (1999) compared populations in the *Ethnographic Atlas* (Murdock, 1967) with regard to marriage practices, subsistence and the division of labor.

> [They] concluded that in contemporary humans, neither hunting nor agriculture has any effect on sexual dimorphism. [Instead] It is the amount of subsistence work done by men and women, rather than the type of subsistence practiced, which has an effect on sexual dimorphism in different societies. (p. 42)

As women contribute more to the subsistence economy, it appears that the degree of sexual dimorphism is reduced. Holden and Mace (1999) hypothesize that these females are taller due to improved nutrition during growth, but mild to moderate physical activity has also been found to enhance linear growth (Torun and Viteri, 1994). Consequently, there may be biomechanical and nutritional explanations for this phenomenon. Holden and Mace (1999) used stature as their only measurement of dimorphism while Ruff (1987) used only the cross-sectional properties of bone.

Biologists may also view sexual dimorphism as a product of optimal biomass distribution for the species. When conditions select for large males, it is advantageous for the female of the species to be as much smaller as possible while still being able to achieve reproductive success (Bramblett, 1994). The optimum female size is large enough to bear the physical demands of labor, but small enough to reduce the metabolic demands associated with large size. By considering sexual dimorphism as an optimal biomass distribution, DeVore and Washburn (1963) propose that males and females may be better able to utilize their resources if they fill

different ecological niches. If niche divergence amplifies with increased sexual dimorphism, then the selective pressures affecting males and females are progressively more different.

Perhaps most importantly to the issue of growth disruption, a number of studies have shown that sexual dimorphism in stature can decrease when people are under nutritional stress and increase under conditions of optimal nutrition (Brauer, 1982; Gray and Wolfe, 1980; Lieberman, 1982; Stini, 1969, 1982; Wolañski and Kasprzak, 1976). The theoretical basis for this is found in the fact that males and females experience differential success in dealing with stressors like starvation and disease due to hormonal and metabolic differences (Ortner, 2003; Stini, 1969). The greater fat and nutrient reserves characteristic of human females are thought to be an adaptation for the increased metabolic demands of lactation and gestation in producing offspring. As a result of these physiological differences, males experience a greater reduction of lean body mass during periods of nutritional inadequacy than their female counterparts. When periods of starvation occur during growth, the reduction in body mass is accompanied by reduced skeletal growth (Stini, 1975a). Studies of secular trends in adult height have found that male increases in stature are greater than for females as the environment improves (Himes, 1979; Meadows Jantz and Jantz, 1999; Kuh et al., 1991). The resulting change in sexual dimorphism conforms to the theoretical expectations based on the differential response to stressors of males and females.

The Metabolic and Disease Implications of Growth Disruption

In modern America, obesity is more of a health risk than malnutrition; however, studies suggest that obesity may actually be triggered by early metabolic insults. Studies of growth disruptions and subsequent catch-up growth have been divided between those that focus on soft-tissue or metabolic changes and those that measure bony responses to catch-up growth. These studies can be broadly divided into those that focus on length and those that focus on weight.

Catch-up growth of skeletal tissue (length) attempts to return an individual to a normal growth trajectory; on the other hand, catch-up in weight deficiencies appear to be controlled by a different mechanism that may overshoot the normal growth trajectory leading to obesity (Hindmarsh, 2004).

Hales and Ozanne (2003) attribute the effects of early growth disruption on metabolic disorders to what they term the "thrifty phenotype hypothesis." During periods of nutritional deficiency the body naturally diverts scant resources to the areas of greatest need. Resources are diverted away from the pancreas to support the brain. When deprivation occurs in early development the stress causes alterations to the metabolic programming of an individual. The result is "thrifty" metabolic functioning programmed to subsist on fewer resources. Thrifty functioning becomes a problem when nutritional resources become available. It has been found that individuals with a lower than normal birth weight (an indicator of a less than optimal fetal environment) can experience metabolic alterations that make them prone to conditions such as obesity and Type II diabetes (Cameron and Demerath, 2002). Recent research has made strides in coordinating growth statistics with evidence of metabolic changes in both animal models and humans, focusing on early growth disruption (particularly in the fetal or early postnatal periods) and subsequent problems of obesity. (Cameron and Demerath, 2002; Grove et al., 2005).

The bulk of this chapter has focused on studies of hard tissues because the applicability to skeletal biology and bioarchaeological populations; however, from a public health perspective, the most important reason to concentrate on skeletal catch-up growth is the role it serves as an indicator of more serious health problems in growing children. While the mechanism controlling catch-up growth in length and weight seem to be entirely different, they are similar in that both are physiological responses to growth disruption. Recent research has investigated the

relationships between metabolic disorders, weight, and length. As mentioned earlier, studies have found that lower limb length appears to be the component on human height most closely associated with environmental stress – the better the environment, the longer the legs (Li et al., 2007). This relationship has been used as an explanation for why shortened relative lower limb length has been associated with a range of medical conditions including cancer (Gunnell et al., 1998b), coronary heart disease (Davey-Smith et al., 2001; Gunnell et al., 1998a; Han et al., 1997; Lawlor et al., 2004), diabetes (Davey-Smith et al., 2001; Lawlor et al., 2004), and other metabolic disorders (Davey-Smith et al., 2001; Han et al., 1997).

CHAPTER 3
SKELETAL INDICATORS OF GROWTH DISRUPTION

Enamel Defects

Enamel develops in increments, beginning at the apex and laying down successive layers of enamel during mineralization. The pattern and timing of dental development appear to be under strong genetic control, with less environmental effect than that seen in skeletal development (Garn and Rohmann, 1966). Surface enamel on the anterior teeth forms between approximately 0.8 and 5.2 years of age, but continues on the third molars to roughly 11.3 years (Reid and Dean, 2006). Age estimates for the formation of surface enamel are summarized in Table 3-1. During growth, any disruption caused by metabolic insults may stop enamel formation just as they may stop bone growth. However, unlike bone, enamel is acellular and does not continue to remodel in adulthood; therefore, enamel provides an excellent source for investigating growth disruptions from adult skeletal material.

Enamel hypoplasias are a type of developmental defect characterized by a deficiency in enamel thickness due to a disruption during amelogenesis, defined as the process of enamel formation (Goodman and Rose, 1990). These are formed when ameloblasts stop secreting enamel matrix earlier than normal (Hillson, 1996). Possible etiological factors for enamel hypoplasias include heredity, local trauma, or systemic metabolic disturbance (Goodman and Rose, 1990; Suckling, 1989). The majority of hypoplastic defects are caused by growth disruptions such as illness and nutritional deficiencies (Hillson and Bond, 1997). Hereditary defects are considered to be rare and can often be identified due to the fact that they are generally severe and all of the teeth in a set are likely to be affected (Buikstra and Ubelaker, 1994; Goodman and Rose, 1990). Conversely, local trauma will only affect a single or a few adjacent teeth. It is reasonable to assume dental defects are due to metabolic stress, rather than trauma, if

they are bilateral in nature (Floyd, 2007; Goodman and Rose, 1990; Hillson, 1996). Responding to nutritional stress, disease, and other physiological insults, enamel defects are considered to be sensitive, albeit non-specific, indicators of stress (Floyd, 2007; Goodman et al., 1987; Goodman and Rose, 1990; Hillson, 1996; Littleton, 2005; May et al., 1993). Enamel hypoplasias do not take long to form and may represent a single, short-term systemic insult rather than long-term chronic stress (Suckling, 1989).

Surface defects that are macroscopically visible may appear in the form of lines, pits, or the complete absence of enamel. The variation in appearance of enamel defects has been attributed to the severity of insult (Suckling, 1989), the duration of the metabolic stress (Blakey and Armelagos, 1985; Hutchinson and Larsen, 1988), the position of the defect on the tooth (Hillson and Bond, 1997), or may be related to other unknown factors.

When scoring hypoplasias, not all researchers choose to include hypoplastic pitting because of distinct differences in the way pits and lines are formed (Steckel et al., 2006). Linear hypoplasias form when matrix secretion ceases along perikyma grooves. Perikyma grooves, also referred to as imbricational lines, mark the successive layers of enamel formation by particular ameloblasts. King et al. (2002) define linear enamel hypoplasias as "a greater than expected spacing between neighboring pairs of perikymata." Pit defects do not occur along an associated perikyma groove on the tooth surface and are formed when only small clusters of ameloblasts stop forming enamel (Hillson, 1996; Hillson and Bond, 1997). Cross-sectional studies have found that the associated perikyma groove may not demonstrate any surface defects. While hypoplastic pits represent a cessation of enamel formation, they are poorly understood and cannot be aged by macroscopic means (Hillson and Bond, 1997).

The surface of any tooth only represents a fraction of the total developmental history recorded in the enamel. By sectioning a tooth and observing it microscopically, the record of growth disruption is more complete. Microscopically, the incremental lines of growth in enamel, referred to as striae of Retzius, can be observed. Accentuated striae of Retzius, also referred to as Wilson bands, are considered to be a more sensitive indicator of growth disruption than enamel hypoplasias and may take less time to form (Larsen, 1997; Rose et al., 1985). Simpson (2001) has also found differences in the age at which Wilson bands and hypoplasias form. Wilson bands most commonly form between 12 and 30 months, whereas hypoplasias are more common after 25 months of age. Most surface defects in enamel are associated with Wilson bands. Where surface defects exist independent of Wilson bands, it is suggested that the surface defect may not be due to physiological stress (Goodman and Rose, 1990). Microscopic examination is also important for observing the hidden appositional zone of enamel. Enamel deposition begins at the cusp and the first layers become covered by subsequent layers of enamel matrix. In molars, the hidden appositional zone may account for between 40 and 50% of the total enamel. In anterior dentition, the appositional zone may account for 15 – 20% of enamel (Hillson and Bond, 1997).

Studies indicate that the frequency of hypoplastic defects is greater in the anterior dentition (Condon and Rose, 1992a; Goodman and Armelagos, 1985). This fact, in combination with the percentage of hidden enamel in the posterior dentition, is the reason that many studies using enamel hypoplasias concentrate on the anterior dentition (Martin et al., 2008; Reid and Dean, 2000; Santos and Coimbra, 1999). Goodman and Rose (1990) recommend that epidemiological studies focus on the maxillary central incisors and mandibular canines given that they are the teeth most sensitive to enamel defects; alternatively, Wright (1997) recommends considering

each tooth independently and using the posterior dentition as a marker of stress severity. In consideration of these recommendations, individuals selected for inclusion in this study were not removed from the sample if posterior teeth were missing. When enamel defects on the posterior dentition were present, they were considered supporting evidence to the defects in the anterior dentition.

The anterior dentition provides a record of chronological growth disruption between birth and seven years of age, the period during which enamel forms (Goodman et al., 1980). Studies have found that the timing of enamel formation is more closely associated with chronological age than most other skeletal elements (White, 2000). While there is population variation in enamel formation (Reid and Dean, 2006), it is possible to age hypoplasias based on the position of the defect on the tooth crown with a fair degree of accuracy. Amelogenesis begins at the occlusal tip of the tooth; however, growth does not occur in a simple linear fashion (Hillson and Bond, 1997). Research into dental development has produced methods for estimating ages (i.e., aging) of hypoplasia formation (Condon and Rose, 1992b; Goodman et al., 1980; Goodman and Rose, 1990; Reid and Dean, 2000). The ability to accurately age a growth disturbance provides an additional line of reference for investigating the effects of growth disruption on adult morphology.

Harris Lines

Harris lines, also known as radiopaque transverse lines, are areas where trabeculae are deposited in a transverse plane perpendicular to the diaphysis of a long bone as a result of disruption in normal metabolic function during growth. The term "Harris line" is a nod to one of the early researchers of radiopaque transverse line formation (Park, 1964). Harris lines can be seen through radiographs (see Figure 3-1), and medical researchers have been able to document their occurrence in association with known episodes of stress such as disease, nutritional

deficiencies, and even psychological stress (Mays, 1995). For the bioarchaeologist, Harris lines are often used to document changes in health over time. An increased frequency of Harris lines may indicate inadequate nutrition or increased levels of disease or infection in a population (Larsen, 2002).

As an indicator of stress, Harris lines are useful in that the presence of such lines in an adult indicate that some form of growth disruption occurred. However, individual variation in the formation of such lines and cortical remodeling mean that researchers need to be cautious when using these indicators to describe the health insults an individual has experienced.

Understanding the formation of Harris lines is important for understanding the visual presentation of a disruption in skeletal growth. At the growth plate, the cartilage matrix is oriented along the longitudinal axis in which the bone is growing. The orientation of cartilage serves as the template for subsequent trabeculae formation (Martin et al., 1998). Park (1964) uses an analogy of a dam and pond to describe how Harris lines are formed through growth disruption. When growth disruption occurs, the epiphyseal cartilage becomes a thin atrophic layer that serves as a dam, behind which bone is laid down in along the transverse plane. Once growth resumes, the osteoblastic activity is temporarily blocked by the dam. It is the recovery that causes the transverse area to thicken creating a radiopaque line in the bone. Without growth disruption, trabeculae are oriented longitudinally. Based on what is known of this process, the relative thickness of a Harris line can be attributed more to the growth arrest recovery than to the growth arrest itself (Park, 1964). The growth arrest event is only important in that it creates the dam. Periods of intense growth without a prior growth disruption do not produce such lines, but the line seen at epiphyseal fusion is equated to growth disruption without subsequent recovery (Park, 1964).

What we know of the process of Harris line formation allows us to more correctly interpret differences in what appears to be the relative "severity" of Harris lines. A thick, pronounced Harris line compared to a thin Harris line would tell you less about the disruption event and more about the recovery phase (and/or subsequent cortical remodeling). Through the process of resorption and cortical remodeling, thicker lines are more likely to persist than thin lines. The number or relative frequency of Harris lines in an individual may be better evidence that said individual experienced more growth disruption events, but interpretations need be cautious due to complicating factors, to be discussed below.

Park (1964) found that growth disruption needed to be complete, or near complete, for line formation to occur. The severity of insult needed for line formation is best described by Garn et al. (1968), who were able to compare radiographs with longitudinal data from the Fels Research Institute. Because the participants in this research program were subject to regular health reviews, radiographic data could be compared to health data to determine the cause of specific Harris lines and record the association between traumatic events and the formation of lines. Garn et al. (1968) found a statistically significant but low-order association between health insults and the formation of lines. In ten percent of cases where new lines formed, they could not be explained by any insult recorded in the health data. In other cases, lines were associated with illnesses such as whooping cough, chickenpox, minor surgeries, and, in one interesting case, with a "routine" small pox vaccination (Garn et al., 1968:73), which would lead one to believe that even relatively minor insults have the ability to produce Harris lines. Therefore, it is hard to make assumptions about the types of stress which led to the formation of a Harris line, particularly in individuals without medical records.

There appears to be a range of individual variability with regard to the formation of Harris lines: some individuals seem to be more prone to forming lines than others. Garn et al. (1968) found more Harris lines in juvenile males than females, possibly a product of greater male susceptibility to the negative effects of physiological stress. But, female lines persisted into adulthood at a higher rate than those lines found in males (Hummert and Van Gerven, 1985). These points need to be considered when comparing differential effects of stress in males and females.

Unlike teeth, bone continues to remodel over the course of an individual's lifetime. As a result, Park stated that Harris lines were "only occasionally encountered in the bones of adults" (Park, 1964). Moreover, Park (1964) believed that lines formed later in the growth phase would be more likely to persist into adulthood. Subsequent studies on the persistence of Harris lines have changed this view; Garn (1968) found that Harris lines formed in early childhood were more likely to persist than those created later in the growth phase. A more recent study found that Harris lines were most commonly formed in the first year of life and in adolescence, both periods of rapid skeletal growth (Alfonso et al., 2005). As a product of cortical remodeling, older individuals are less likely than younger individuals to have visible Harris lines (Grolleau-Raoux et al., 1997). Therefore the ability to determine any associated effects of Harris line formation are likely clouded as a product of advanced age. By understanding these limitations, researchers are better able to interpret the results of studies.

Harris lines represent a metabolic disruption which affects the longitudinal growth of bone and the subsequent growth recovery. As such, researchers have attempted to correlate long bone length with the presence of Harris lines. The results of these studies have provided conflicting results. Goodman and Clarke (1981) compared the Harris lines in tibiae (based on

presence or absence) with tibial length. For females and a combined sex sample, tibiae with Harris lines were significantly longer than those without. A more recent study found no significant difference in the length of long bones with and without Harris lines (Nowak and Piontek, 2002). These conflicting results are no clearer in published studies of juvenile morphology and Harris lines. Blanco et al. (1974) found living children with Harris lines were significantly shorter than those without; however Mays (1995) found no relation between femur length and the presence of Harris lines. Obviously the results of these studies have not answered the question of the relationship between how growth disruptions affect bone length. Likely complicating the scenario is the fact that the density of a Harris line is not determined by the severity of insult so much as the quality of recovery. Because the thickest lines are likely to persist the longest in light of cortical remodeling, Harris lines present in adults may not represent those with the greatest insults, but instead those with the greatest recovery.

Comparisons of Harris lines with other indicators of growth disruption have also shed light on how Harris lines form. Linear enamel hypoplasias, like Harris lines, are a non-specific indicator of stress that can be aged in terms of when they formed in an individual. Comparisons of linear enamel hypoplasias and Harris lines do not suggest a one-to-one relationship. Alfonso et al. (2005) found that in the archaeological populations they studied the rates of linear enamel hypoplasias peaked between the ages of three to five. On the other hand Harris lines were most commonly formed in the first year and during adolescence. Weaning is known to be a period of stress and the enamel hypoplasias data reflect this stress. However, the ages of peak Harris line formation appear to be more strongly associated with phases of peak bone growth more so than with periods of stress as indicated by the linear enamel hypoplasias. And, while Harris lines have been positively associated with stress events in some cases (Garn et al., 1968), the data

presented by Alfonso et al. (2005), among others, suggest that Harris lines are not nearly as reliable an indicator of stress.

Garn et al. (1968) made the important discovery that Harris lines maintained "dimensional stability" (Garn et al., 1968:72). Simply put, the process of bone remodeling did not cause the lines to migrate along the bone shaft. Where they were laid down, they remained. This was an important discovery, because it laid the groundwork for later studies which have allowed researchers to age Harris lines (Byers, 1991; Hunt and Hatch, 1981). Now, with a degree of reliability, x-rays allow us to determine when a person experienced the metabolic insult which produced a specific Harris line.

Once researchers were able to accurately age Harris lines, they were then able to determine the periods during growth which lines were most likely to form. Park (1964), whose research focused on juveniles, assumed that the process of remodeling meant that the lines seen in adult individuals were formed late in the growth phase and therefore had less time to undergo remodeling. Garn et al. (1968) reached a different conclusion. By measuring the distance of a line from the epiphysis, he concluded that the most persistent lines are formed in infancy or even in fetal life.

There are obviously many variables that need to be considered when using Harris lines as an indicator of growth disruption. But, regardless of the variability in formation and obliteration due to resorption, they provide a line of evidence for growth disruption that is not otherwise available, and one that can be associated with certain phases of growth. Therefore, the Harris lines can be a useful tool in osteological analysis when used with an understanding of how they are formed and resorbed.

Other Skeletal Indicators of Growth Disruption

Although enamel defects and Harris lines may be the most well studied indicators of growth disruption, they are certainly not the only evidence available in the skeleton. For example, in anthropometric studies of living children, head circumference is used as an indicator of substandard growth and development. Stoch and Smythe (1976) found significant differences in head circumference between a control group and those undernourished during infancy, the period during which head growth is most rapid. One longitudinal study on head circumference found that catch-up occurred in the majority of cases, but that small head circumference persists into adulthood for many individuals (Brandt et al., 2003). Therefore, in an adult skeletal sample, the growth environment for those with average head circumferences cannot be assumed.

Clark et al. (1986) suggested that vertebral neural canals could be used as an indicator of disrupted growth. Because vertebral neural canals cease growth in early childhood while the bodies of the vertebrae continue to grow, the relationship of these two measurements can indicate early childhood growth disruption. Small neural canals with normal vertebral body sizes indicate growth disruption in early childhood followed by a period of catch-up growth. If both measurements are small, it would be an indicator of chronic stress (Clark 1988). Rewekant (2001) built on this study by observing differences in a vertebral canal index (sagittal diameter/transverse diameter) in two archaeological populations of divergent socio-economic statuses. Smaller vertebral canal indices were more often associated with the more "unhealthy" population; however, statistically significant differences were only found in the male sample. Rewekant (2001) does not provide data that would inform whether smaller vertebral canal indices are tied to smaller sagittal vertebral canal diameters or larger transverse diameters in the group of lower socio-economic status. Clark (1988) suggests that the transverse diameter of vertebral neural canals complete growth later than the sagittal diameter and that the relationship

of these variables can thereby indicate whether growth disruption occurred early or late in growth, but he does not suggest that a particular vertebral neural canal index is indicative of anything in particular.

At a historic Maya site where approximately half of the skeletal sample had active or remodeled porotic hyperostosis and/or cribra orbitalia, researchers suggest that anemia may have some effect on adult stature. Those individual who displayed the porous lesions associated with anemia were found to be approximately 0.5 centimeters shorter on average than those who did not. However, the results were not found to have statistical significance (Cohen et al., 1997). Although Garn et al. (1968) discovered that Harris lines could be associated with a recovery from chronic or acute anemia, in adults the anemia which caused the lesion could have occurred during a phase of life before or after the completion of growth, whether or not the lesion is active or healed. While the relationship between stature and anemia during the growth phase is an interesting one, the evidence available in an adult skeleton does not provide the information necessary for inclusion in this study. In addition, the number of individuals sampled with evidence of anemia was too small to be statistically significant.

Vertebral neural canal size and head circumference are both, in part, a product of overall body size; as such, assumptions of independence are violated in any statistical interpretation of how these variables are related to overall body size. To test how meaningful these variables are as indicators of growth disruption, they are compared to linear enamel hypoplasias and Harris lines in Chapter 8.

Table 3-1. Mean estimates for ages of enamel formation

		Age in years [a]
Upper dentition		
	I^1	1.1 - 4.2
	I^2	1.8 - 4.8
	C	1.7 - 4.8
	P^3	2.0 - 6.0
	P^4	2.5 - 6.0
	M^1	1.1 - 3.0
	M^2	4.4 - 6.4
	M^3	9.3 - 11.3
Lower Dentition		
	I_1	0.8 - 3.4
	I_2	1.0 - 3.8
	C	1.4 - 5.2
	P_3	1.0 - 6.0
	P_4	2.0 - 7.0
	M_1	1.0 - 3.1
	M_2	4.2 - 6.2
	M_3	9.3 - 11.3

[a] All estimates based on Reid and Dean (2006) except for the premolars which are based on Goodman et al. (1980).

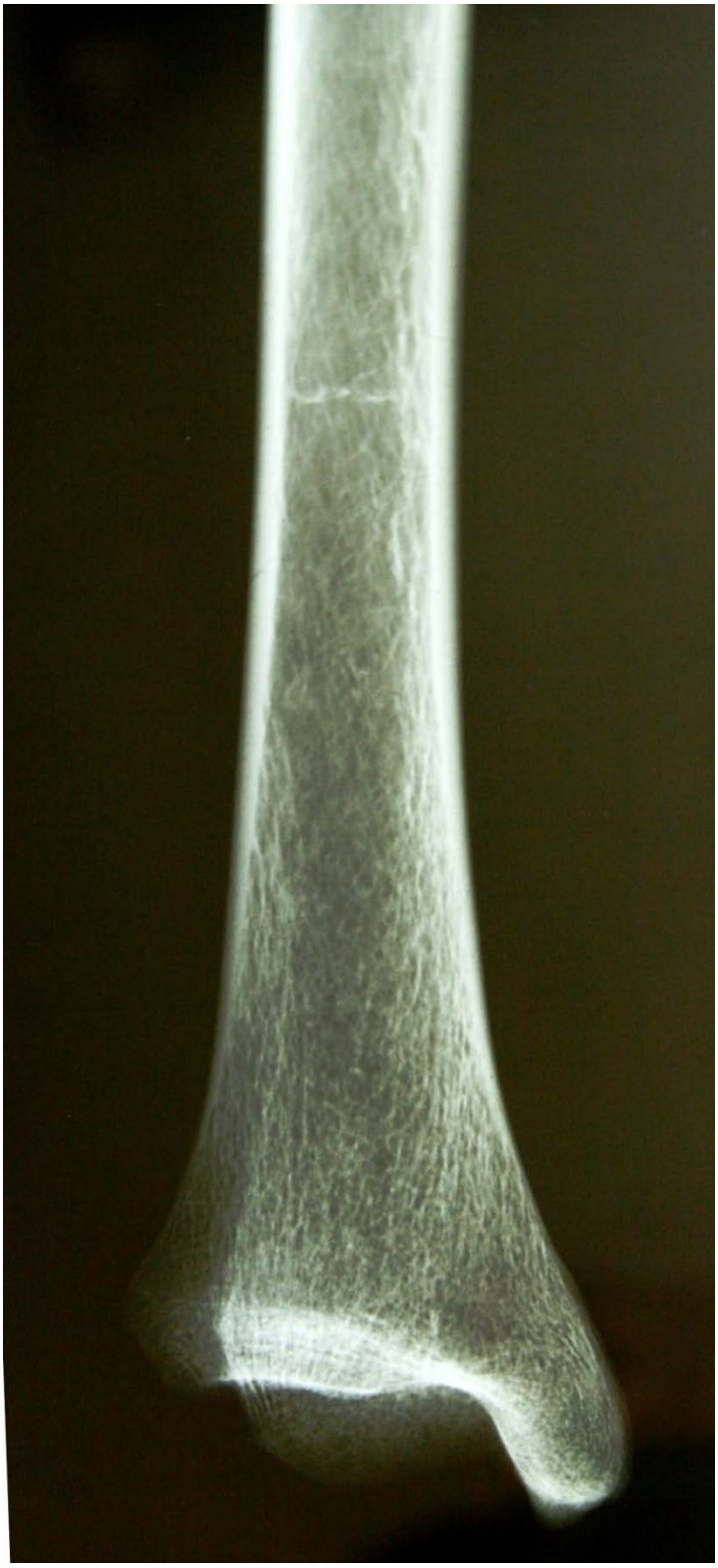


Figure 3-1. Radiograph of a tibia with a Harris line

CHAPTER 4
MATERIALS AND METHODS

The purpose of this chapter is to introduce the materials and methods used to address the hypotheses of this study. The "materials" section describes the collections from which data was obtained as well as the demographic composition of the research sample. The "methods" section outlines the techniques used to obtain measurements and stress data on the skeletal material as well as the statistical procedures used to analyze this data.

Materials

To address the hypotheses outlined, it was necessary to obtain data from a skeletal series of adult males and females, both with and without skeletal indicators of growth disruption. Data was collected from the Terry Collection at the Smithsonian Institution and the Hamann-Todd collection at the Cleveland Museum of Natural History. The Terry and Hamann-Todd collections were compiled from anatomical specimens in the St. Louis, MO and Cleveland, OH areas respectively. In both cases, the majority of the cadavers were unclaimed bodies in morgues or hospitals or those that were given to the state by family members (Hunt and Albanese, 2005). Comparisons of the Terry and Hamann-Todd collections conclude that the two populations are osteometrically similar (Isçan, 1990).

The nature of this collection provides several advantages for this study. First, the combined number of individuals found in these collections insures a large sample size for testing the hypotheses of this project. Second, morgue records allow for reasonable control over ancestry, age, secular trends and any other extenuating circumstances that the historical records may illuminate.

For inclusion in this study, all individuals must be of like ancestry. This criterion attempts to control for the genetic component of human morphology. It is recognized that there are

differences in average stature, growth rates, sexual dimorphism, and proportions in different populations (Eveleth, 1975, 1986; Eveleth and Tanner, 1990). For example, Hamill et al. (1973) found that European Americans tend to have longer trunks and African Americans tend to have longer lower limbs despite socioeconomic conditions. As such, ancestry cannot be ignored in any investigation of growth disruption.

Both the Terry and the Hamann-Todd collections divide their skeletal collections into "white" and "black" samples based on what they term ethnic origins or ethnicity respectively. Today, race is recognized as a cultural construct rather than a biological reality by anthropologists and it is important to recognize problems with making such population distinctions; most notably, that the standards for making such distinctions are not always uniform (AAPA, 1996; Cartmill, 1998). The "white" individuals in these collections are presumably Americans of European descent. While there is a great degree of variability within Europe, the situation becomes even more complicated when considering the ancestry of "black" Americans. The supposition is that these individuals are of African descent, but Africa has the highest degree of genetic diversity of any continent (Jorde et al, 2000). In addition, there is a high degree of genetic admixture in African-American populations. Historical records confirm that nearly all slaves brought to North America were from the coast and interior of western Africa although a few were from Mozambique or Madagascar. Unlike the Portuguese and Dutch, English slave traders did not have ties with specific ports and purchased slaves from Senegal to Angola. The Dutch traders concentrated on the ports in Angola and it is estimated that a quarter of slaves in the colonies originated in this area of Africa (Wright, 1990). Slave routes changed over the course of time and because slaves were treated as chattel rather than people, genealogical information is not available for early African-Americans. Recent studies of modern African-

American populations suggest an average of 20.9% European ancestry and 2.7% Native-American ancestry, although these numbers vary geographically (Reiner et al., 2005). Furthermore, according to the rule of hypodescent (the "one drop rule"), which has been standard practice throughout most of American history, a child of a mixed union would be racially classified as belonging to the race of the parent that is considered to be socially inferior. Therefore, an individual might be classified "black" even if the representation of African ancestry is minimal.

Despite the inherent problems with any label of ancestry, these collections provide the best resource available for controlling genetic variation within my sample. To knowingly ignore such labels would be irresponsible given what is known about variations in human growth across populations (Eveleth and Tanner, 1990; Silventoinen, 2003). Due to the paucity of white females in the two collections, this research focused on those individuals classified as "black," despite the diversity assumed with this label. The Terry collection houses 1,728 individuals and the Hamann-Todd collection houses 3,100 human skeletons. To streamline the data collection procedure, the written records associated with each collection were used to establish ancestry, thereby determining which individuals to pull for analysis.

A second criterion for inclusion in this study is age at death. All individuals must be adults with united epiphyses to insure that maximum height was attained prior to death. However, it is important that there is minimal, if any, osteoporotic bone loss in the vertebrae. Bone mass peaks around the age of 18 years, and, shortly thereafter, the trabecular structure begins to diminish. The age-related changes become particularly pronounced after the age of 50 years although the relative severity of such changes is related to other factors such as sex and mechanical stress (Agarwal et al., 2004; Atkinson, 1967; D'Ippolito et al., 1999; Larsen, 1997). Age was

determined through autopsy records. The youngest individual in this sample was a female who had reached the age of 19 years (and full epiphyseal closure) before death and the oldest individual was 35 years of age (see Table 4-1). By limiting the sample to a maximum age of thirty five, I hope to avoid osteoporotic fracturing that may not be macroscopically visible. While the individuals in the Terry and Hamann-Todd collections were indigents, likely exposed to heavy labor, African-Americans are less susceptible to osteoporotic fracturing than their neighbors of European descent (Mensforth and Latimerm, 1989; Robling and Stout, 2000). Unfortunately, vertebral body heights continue to grow slightly through early adulthood (Clark, 1988). In a population exposed to manual labor, an individual is likely accumulating microfractures in their vertebrae before they finish growth. Individuals with macroscopically visible pathologies that would affect height were excluded from the sample.

The presence of dentition is a third consideration for inclusion in this study. Enamel defects are an important line of evidence for addressing the hypotheses outlined. Edentulous individuals were immediately excluded from analysis. Very few individuals retained all of their teeth, so it was necessary to determine, on a case-by-case basis, whether the dentition provided enough data for analysis. The individual with the lowest total number of teeth (N=16) had one of each tooth type (e.g. central maxillary incisor, lateral mandibular incisor) in the anterior dentition. The presence of posterior dentition was considered to be less important for inclusion in the study for two reasons: 1) the posterior dentition are not as susceptible to developing enamel defects as the anterior dentition (Condon and Rose, 1992a; Goodman and Armelagos, 1985), and 2) the amount of hidden appositional enamel means that they provide less information in macroscopic observations of surface enamel (Hillson and Bond, 1997).

An additional advantage in using anatomical collections, like the Terry and Hamann-Todd collections, is that it is possible to control for secular trends due to the availability of year-of-birth and year-of-death data. The dates of birth for individuals in this sample range from 1885 to 1934. Secular changes in morphology have been documented in these collections (Jantz and Meadows Jantz, 2000; Meadows Jantz and Jantz, 1999); therefore trends found in the results will be compared to the date of birth data to make sure that the results are not potentially confounded by the secular changes (see Table 4-1).

Tuberculosis was the leading cause of death among these individuals (42.6%). The use of antibiotics to effectively treat human diseases did not become a widespread practice until the 1950's (Lederberg, 2000), after the deaths of most individuals analyzed in this study. As a result, many of these individuals died from diseases that are treated with antibiotics today and likely suffered a higher infectious load during growth than the average child born today.

Methods

Once individuals were chosen for analysis, a quick survey of each skeleton was conducted to determine if the skeletal biology of the individual was consistent with the records and if all elements appeared to be from the same individual. It was not necessary that each bone antimere was present, but one of each bone had to be available for measurement. For example, if a left and right tibia were not available, the individual was not excluded from analysis, but one tibia was necessary for inclusion. Individuals with pathologies that affected skeletal height were immediately excluded. Most of excluded individuals had pathological curvature of the spine or long bones, or exhibited severe arthritic lipping and/or compression of the vertebral bodies. Other pathologies were recorded for each individual. The teeth were examined to determine whether there was enough evidence available to make a statement regarding the presence or absence of enamel defects. This is the phase of analysis where the majority of potential

candidates were excluded for either lack of teeth or questionable association of the available teeth.

Measurements

Each individual was measured to obtain estimates of stature and proportional indices. Cranial measurements and vertebral neural canal measurements were also taken to see how they relate to stress. The measurements collected for each individual are summarized in Table 4-2 and include the maximum and bicondylar length of the femur, maximum tibial length, maximum humeral length, and maximum length of the radius - measurements standard in any postcranial osetometric analysis. In addition, the height of the cranium (basion – bregma), maximum height of the vertebral bodies (C2-L5), anterior height of S1, and the articulated height of the calcaneus and talus were included so that skeletal height (SKH) and an estimated living stature could be calculated using the revised Fully technique (Raxter et al., 2006). While reliable regression formulae are available for calculating estimated stature based on long bone measurements, they do not provide independence when individual bone lengths are compared to estimated stature as a means of determining proportional differences (see a list of proportional indices in Table 4-3).

In anthropomorphic studies, sitting height to standing height ratios are used as a measure of proportional differences. In this study, skeletal trunk height (STH), as defined by Holliday (1997), will be included to estimate relative trunk height to stature proportions. Skeletal trunk height is found by summing dorsal body heights of the thoracic vertebrae, lumbar vertebrae, and the ventral height of the sacrum.

The femur, tibia and talus-calcaneus measurements were made using a standard osteometric board. Head circumference from the Terry collection was obtained by using a tape measure placed at opisthocranion and held just above the brow ridge in a manner simulating the anthropometric techniques used in children (Zerran, 2007). Head circumference measurements

from the Hamann-Todd collection were acquired by referencing the autopsy records from the collection's database. Cranial height, length and breadth were measured with spreading calipers and all remaining measurements were made with sliding calipers. Autopsies were performed on the individuals in both the Terry and Hamann-Todd collections; consequently, most individuals had their calotte removed or, in some cases, the skull was bisected sagittally. Skulls in the Hamann-Todd collection were wired into approximate anatomical alignment, but skulls in the Terry collection were articulated using wax prior to being measured. With the exception of head circumference and talus-calcaneus height, all measurements were collected using the landmarks and techniques described by Buikstra and Ubelaker (1994). For the sided elements, both sides were measured and an average was used for the calculation of stature.

Linear Enamel Defects

Surface defects in the enamel were recorded using the dental data collection form (Figure 4-1). Enamel defects were observed macroscopically and recorded when the defect could be felt by rubbing a fingernail across the surface of the tooth. Because many of the teeth in these collections were glued into the alveolar sockets, a 10X hand loupe was used to insure that the enamel surface was not compromised. Because so much of the enamel in molars is unobservable without sectioning and the majority of hypoplasias are found on the incisors and canines, hypoplastic analyses in this study concentrate primarily on the anterior dentition (Condon and Rose, 1992b; Goodman and Armelagos, 1985; Hillson, 1996; Steckel et al., 2006), although all defects were recorded where they were encountered.

Each tooth was assigned an LEH code based on the standards outlined in The Global History of Health Project: Data Collection Codebook (Steckel et al., 2006). By these guidelines, teeth are ranked from zero to three based on the relative number of hypoplastic lines observable. A description of how these codes are assigned can be found in Figure 4-1. Hypoplastic lesions

can be described as lines, bands or pits, and each defect was documented accordingly. Dental caries and opacities were also recorded for each tooth.

For each developmental defect in tooth enamel, the distance from the cemento-enamel junction was recorded (Figure 4-2). For wide band defects, distance was measured from the near, middle and occlusal edge of the defect. These measurements were used to find the age at which the defect was formed using the methodology provided by Goodman et al. (1980). This method was chosen over the Read and Dean (2000) method because the latter relies on crown heights for age estimations. In these samples, numerous teeth were worn or broken so that accurate crown heights could not be obtained. Ritzman et al. (2008) and Martin et al. (2008) compared these techniques and found the largest difference to be in that the Goodman et al. (1980) method consistently underestimates age at formation for the earliest forming hypoplasias. While the difference is statistically significant, it is only a difference of 1 to 4 months – a time frame that is not meaningful for the purposes of this analysis (Martin et al., 2008). For this analysis, hypoplasias were broadly divided into two categories: those that formed in infancy (during the first three years of life), and those that formed during childhood (approximately three to seven years). When a defect was estimated to have been formed at approximately three years of age, that defect was placed in the "child" rather than the "infant" category. Hypoplastic pits were not assigned an age estimate given that pit defects do not form along the imbricational line associated with their location on the crown surface; as such, they cannot be aged correctly based on macroscopic analysis (Hillson and Bond, 1997). Bands were aged based on the location of the occlusal edge of the defect, using the standards outlined in Buikstra and Ubelaker (1994).

Dental defects were coded in a number of ways for data analysis. Tooth stress (TS) was recorded based on presence or absence (scored zero or one) of any enamel defect in an

individual. This coding scheme does not differentiate between lines, bands or pits. Nor does it take into consideration whether or not a dental defect is bilateral or on the anterior or posterior dentition. To further differentiate between individuals with different types of hypoplastic lesions, the linear enamel hypoplasias (LEH) stress code was created. If an individual has hypoplastic pitting, in the absence of any linear enamel defect, they are removed from analysis using the LEH score. This criterion is used, in part, to satisfy those individuals who do not believe that pitting should be scored as a hypoplastic lesion. A more conservative scoring procedure (CBTS) was used in which an individual was considered "stressed" only when an individual had bilateral enamel defects on their anterior dentition. Many of the individuals deemed "stressed" based on TS criteria and "unstressed" by CBTS criteria were removed from further analysis. However, eleven individuals were coded differently depending on the criteria used. Because the CBTS scoring criterion is the most conservative scoring technique employed in this analysis, it is considered to be the standard. A summary of these coding schemes can be found in Table 4-4.

In an attempt to distinguish between chronic and acute stress, an estimate of the minimum number of stress events (MNSE) was calculated for each individual. The MNSE score was based on the number of hypoplastic defects in a single tooth and then the anterior teeth were compared using the calculated age estimates for enamel defect formation. For example, if a single defect was present on all the maxillary, anterior teeth, but age ranges suggest they did not pertain to the same stress event, then the MNSE score would be equivalent to the number of age ranges represented. This minimum number was based on defects found on canines and incisors. If a defect was not bilateral, it was excluded from analysis due to the fact that trauma can cause lines on teeth that are not attributed to systemic stress (Hillson, 1996). If the antimere was not

present, a tooth was tentatively retained for analysis and evidence from other teeth (including posterior dentition) determined whether or not it would be included in the analysis. Data were also collected summing the number of lines for each individual based on CBTS criteria as well as the number of teeth affected in each individual.

Harris Lines

Radiographs were taken of the distal tibiae to score observable radiopaque transverse lines (also known as Harris lines). For inclusion, lines had to be visible to the naked eye and extend half-way across the bone (standards outlined by Garn et al. (1968)). The distal tibiae are used for this type of analysis because they have been shown to exhibit a relatively high frequency of Harris lines when compared to other skeletal elements (Garn et al., 1968; Park, 1964). Because of this, most Harris line research is performed on tibiae, including methods for estimating age at formation (Byers, 1991; Garn et al., 1968; Goodman and Clark, 1981; Hummert and Van Gerven, 1985).

For consistency, the left tibia was x-rayed unless it was unavailable or determined to be the inferior choice due to reactive bone formation or postmortem cracks in the bone. Individuals from the Terry collection were radiographed using 35 x 43 cm mammography film where approximately the distal three quarters of five tibiae could be placed on a single film. The x-ray equipment used while collecting data at the Cleveland Museum of Natural History was a Faxitron cabinet unit with internal dimensions of approximately 16 x 18.25 inches. The unit has a central projection and uses 10 x 12 inch film. This meant that the medial malleolus was only visible on the film of the smallest individuals, although most tibiae could be oriented within the cabinet in such a way to film the entire length of the diaphysis. Byers (1991) technique for estimating the age at which a Harris line formed requires a measurement from the distal end of the bone. To correct for this problem, foil was taped to each bone at a measurement determined

to be the middle of bone length. Harris lines were then measured from the center point and the distance from the distal end was then calculated.

Byers (1991) technique for age estimation was used because all individuals in this study are adults. Unfortunately, Byers' (1991) figures suggest that his calculations rely on a tibial length that includes the intercondylar tubercles. The standard osteometric technique for measuring the length of the tibia is to measure the distance from the articular surface of the lateral condyle to the medial malleolus – a measurement that excludes the intercondylar eminences (Bass, 1995; Buikstra and Ubelaker, 1994). Figure 4-3 shows the difference between these measurements as well as the method for calculating the timing of Harris line formation. Different techniques for measuring the tibia have caused problems in the past (see Jantz et al., 1994). Because tibial length was collected using the standard technique in this study, a correction of 2.46mm was added to the tibia length before Byers age calculations were performed. This correction was the difference found when measuring the tibia by including and excluding the intercondylar eminences of black individuals from the Terry collection (Waxenbaum et al., 2006).

Due to equipment failure and collection error, radiographs were obtained for only 183 of the 204 individuals used in this study. Those 183 individuals were divided based on the presence or absence of Harris lines. An estimated age at formation was calculated to determine whether or not the age at which a stress event occurs has any effect on adult stature and proportions.

Statistical Analyses

Linear enamel hypoplasias and Harris lines are the two primary markers of growth disruption in this study; as such, tests of independence are necessary to evaluate the relationship between these variables. In this case, it was important to determine whether Harris lines and linear enamel hypoplasias are recording the same stress events. To address the question of

independence, a two-tailed, two-by-two contingency table (a Fisher's Exact test) was created using data on the presence or absence of linear enamel hypoplasias and Harris lines.

Stress markers allow for the data to be grouped into two samples: those with and without a particular stress marker. Analysis of variance (ANOVA) was the principal analytical approach employed to test whether or not dimensional variables are statistically different between stressed and unstressed groups. ANOVAs were also used to compare osteometric data with meristic data on age at formation for both Harris lines and linear enamel hypoplasias. Subsequent analyses of age at formation correlate adult metrics and proportional indices with age data to examine whether the age of the inferred stressor has an effect on adult form.

An ANOVA is appropriate only where assumptions of normality and homoscedasticity are met. Shapiro-Wilk's test for normality and Levine's test of error variance were used prior to performing any ANOVA. Where the assumptions of normality and homoscedasticity were not met, the non-parametric Mann-Whitney *U*-test was performed. All descriptive statistics, ANOVAs, and Mann-Whitney *U*-tests were performed using SPSS Statistics Grad Pack 17.0. The results of an ANOVA were considered to be significant at the $p < 0.05$ level.

When results were found to be near significant ($0.05 < p < 0.10$), a resampling procedure, using Resampling Stats 5.0.2 software, was employed as an independent test. In these tests, the populations being compared were first pooled, and then resampled with replacement 10,000 times according to the original sample sizes. For each resampling, a mean difference was calculated. Probabilities were determined by how many times the resampling procedure yielded a mean difference greater to or equal to the original difference. Resampling statistics provides a reliable, conservative method for obtaining probabilities irrespective of distributional assumptions (Simon, 2000).

Two-way factorial analyses of variance were used to compare osteometric data of the stressed and unstressed population broken down by males and females. It is understood that statistically significant differences in osteometric data are found between males and females, but with a two-way factorial analysis of variance it is possible to observe any interactions between the variables of sex and stress. This makes it possible to determine if the effect of growth disruption affects males more than females.

To address the question of whether or not proportional indices (listed in Table 4-3), expressed as ratios, differ in relation to stress, it was first necessary to apply Pearson's correlation to determine whether or not the components of these ratios are independent. These correlations were expected to be significant because long bone length is related to overall body size; therefore, to qualify the results of ANOVAs between stressed and unstressed groups it was necessary to analyze differences in scaling. Reduced major axis (RMA) regressions were conducted on the log-transformed components of ratios in stressed, unstressed and a pooled sample using RMA: Software for Reduced Major Axis Regression (v. 1.17) (Bohonak, 2004). Reduced major axis regressions were chosen over least squares regressions due to the fact that error is assumed in both the X and Y variables. Using reduced major axis regressions to interpret shape has recently been supported by Smith (2009). Using the correlation coefficient from the regressions found using the Bohonak software, RMA2 software (Cole, 1997) was then used to test whether or not the scaling differences between groups were statistically significant. Where RMA slopes were not different, intercept differences were tested by fitting a regression line through the pooled dataset and counting the number of stressed and unstressed individuals that fall above and below the line. This information was then placed in a 2x2 contingency table and Fisher's exact test was used to test the difference (Tsutakawa and Hewett, 1977).

The degree of sexual dimorphism in each group was found using the formula $\ln \bar{X}_M - \ln \bar{X}_F$ for each measurement and proportion, where $\bar{X}_M$ is the male mean and $\bar{X}_F$ is the female mean. The decision to use this method of calculating a score of dimorphism was based on Smith (1999), who demonstrated that compared to other methods for finding dimorphism ratios, this method is believed to have fewer problematic statistical properties.

A dimorphism score is a valuable tool for measuring the value of dimorphism between populations, but, given that the score offers no measure of dispersion, traditional statistical techniques cannot be used to determine whether or not there is a significant difference in sexual dimorphism between stressed and unstressed populations. To address that question, a bootstrap procedure was employed using Resampling Stats 5.0.2 software. The null hypothesis was that no difference in sexual dimorphism exists between stressed and unstressed groups; the alternative (one-tailed) hypothesis is that sexual dimorphism is greater in unstressed groups. The stressed and unstressed, male and female populations were pooled and resampled with replacement using the original sample sizes. In each resampling, the log difference between male and female means was calculated for the stressed and unstressed groups. A difference in dimorphism was then found by subtracting the stressed measure of dimorphism from the unstressed sample. Probabilities were determined by counting the number of times the resampling mean difference was greater or equal to the empirical measure of difference in sexual dimorphism which was calculated by subtracting the original dimorphism score in the stressed sample from the unstressed score. Each resampling procedure was run with 1,000 iterations except where probabilities approached significance. In that instance, the test was run with 10,000 iterations. Because the alternative hypothesis was directional, the probability was calculated as 1-*p* where the empirical difference in dimorphism was negative. In these cases, the

original alternative hypothesis is not tested; instead, 1-*p* tests the alternative hypothesis that sexual dimorphism is greater in the stressed group.

Table 4-1. Description of sample population

		Age at death				Year of birth			
	N	Mean	Min.	Max	S.D.	Mean	Min.	Max	S.D.
Male	107	28.52	20	35	4.342	1902.17	1885	1928	7.167
Female	97	27.31	19	35	4.251	1904.62	1888	1934	6.826
Total	204	27.95	19	35	4.331	1903.33	1885	1934	7.097

Table 4-2. Skeletal measurements recorded

Skeletal element	Measurements
Cranium	Cranial height (br- ba)
	Maximum cranial length (gl - op)
	Maximum cranial breadth (eu - eu)
	Head circumference
Cervical vertebrae (C2-C7)	Maximum anterior body height
Thoracic vertebrae (T1-T12)	Maximum anterior body height
	Maximum posterior body height
	Mediolateral vertebral neural canal diameter
	Anteroposterior vertebral neural canal diameter
Lumbar vertebrae (L1-L5)	Maximum anterior body height
	Maximum posterior body height
	Mediolateral vertebral neural canal diameter
	Anteroposterior vertebral neural canal diameter
Sacrum	Maximum anterior height of S1
Femur	Maximum length (right)
	Maximum length (left)
	Bicondylar length (right)
	Bicondylar length (left)
Tibia	Length (right)
	Length (left)
Fibula	Maximum length (right)
	Maximum length (left)
Talus-calcaneus	Height (right)
	Height (left)
Humerus	Maximum length (right)
	Maximum length (left)
Radius	Maximum length (right)
	Maximum length (left)
Ulna	Maximum length (right)
	Maximum length (left)
	Physiological length (right)
	Physiological length (left)
Clavicle	Maximum length (right)
	Maximum length (left)

Table 4-3. Description of proportional indices

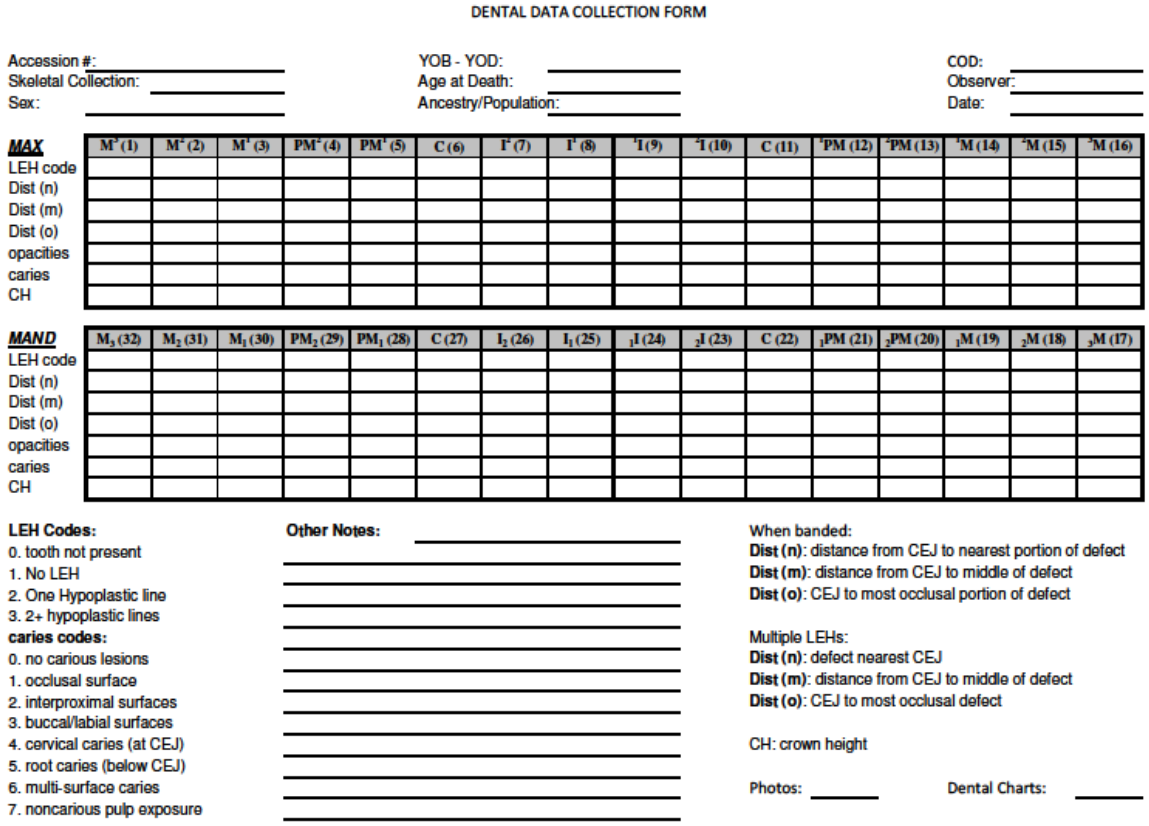
Index	Calculation [a]
Crural	(tibial length / max. femoral length) * 100
Humerofemoral	(max. humeral length / max. femoral length) * 100
Radiohumeral	(max. radial length / max. humeral length) * 100
Sitting Height	(skeletal trunk height / skeletal height) * 100
Intermembral	((max. humeral length + max. radial length) / (max. femoral length + tibial length)) * 100

[a] the average of left and right measurements were used when both were available.

Table 4-4. Coding dental enamel defects

Code	Criteria
CBTS [a]	Individuals are scored as "stressed" (1) only when defects are on the anterior dentition and they are found bilaterally. Otherwise, individuals are scored as "unstressed" (0).
TS	Individuals are scored based on the presence (1) or absence (0) of any enamel defect. This is the most lenient of all the scoring procedures.
LEH	Individuals are scored based on the presence (1) or absence (0) of linear enamel defect(s). Hypoplastic pitting is not scored.

[a] The CBTS scoring procedure is considered to be the standard for this analysis.



DENTAL DATA COLLECTION FORM

Accession #: _____ YOB - YOD: _____ COD: _____
Skeletal Collection: _____ Age at Death: _____ Observer: _____
Sex: _____ Ancestry/Population: _____ Date: _____

MAX	M^3 (1)	M^2 (2)	M^1 (3)	PM^2 (4)	PM^1 (5)	C (6)	I^2 (7)	I^1 (8)	^{1}I (9)	^{2}I (10)	C (11)	^{1}PM (12)	^{2}PM (13)	^{1}M (14)	^{2}M (15)	^{3}M (16)
LEH code																
Dist (n)																
Dist (m)																
Dist (o)																
opacities																
caries																
CH																

MAND	M_3 (32)	M_2 (31)	M_1 (30)	PM_2 (29)	PM_1 (28)	C (27)	I_2 (26)	I_1 (25)	$_1$I (24)	$_2$I (23)	C (22)	$_1$PM (21)	$_2$PM (20)	$_1$M (19)	$_2$M (18)	$_3$M (17)
LEH code																
Dist (n)																
Dist (m)																
Dist (o)																
opacities																
caries																
CH																

LEH Codes:
0. tooth not present
1. No LEH
2. One Hypoplastic line
3. 2+ hypoplastic lines

caries codes:
0. no carious lesions
1. occlusal surface
2. interproximal surfaces
3. buccal/labial surfaces
4. cervical caries (at CEJ)
5. root caries (below CEJ)
6. multi-surface caries
7. noncarious pulp exposure

Other Notes: _____

When banded:
Dist (n): distance from CEJ to nearest portion of defect
Dist (m): distance from CEJ to middle of defect
Dist (o): CEJ to most occlusal portion of defect

Multiple LEHs:
Dist (n): defect nearest CEJ
Dist (m): distance from CEJ to middle of defect
Dist (o): CEJ to most occlusal defect

CH: crown height

Photos: _____ Dental Charts: _____

Figure 4-1. Dental data collection form

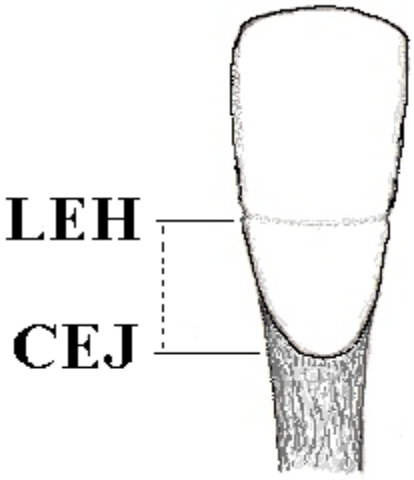


Figure 4-2. Measuring hypoplastic teeth

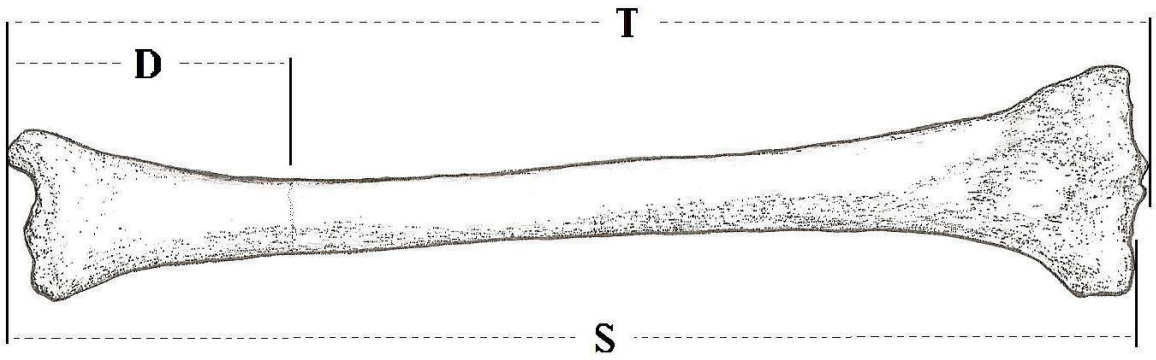


Figure 4-3. Aging radiopaque transverse lines. T = total length of the tibia as defined by Byers (1991); S = the standard technique for measuring tibial length; D = the distance from the Harris line to the distal end of the bone. Age at formation is calculated by using the formula 1.15(T – 2.33D) X 100/T. Tables in Byers (1991) allow for conversion of that percentage to an age range.

CHAPTER 5
RESULTS: STATURE AND STRESS

Of the 204 individuals analyzed in this study, radiographs were available for 183 of them. A G-test of independence was performed to determine whether or not Harris lines and hypoplastic defects should be considered independent variables. The G-test was chosen because it is considered to be a Model I design where individuals are grouped by common experiences that represent treatment effects (Sokal and Rohlf, 1995). The results of this test allow us to accept the null hypothesis that Harris lines and hypoplastic defects are independent ($G = 0.867$, $p = 0.352$). The same result is found when the population if divided by sex (female $G = 0.733$, $p = 0.392$; male $G = 0.206$, $p = 0.65$).

The osteometric variables were tested for normality and homoscedasticity to determine the appropriate statistical method for analysis. The osteometric variables in question proved to be normally distributed (see Table 5-1). However, the individual measurements of the summed variables (SKH and STH) were not normally distributed in every case. This was particularly true of the dorsal height measurements of the thoracic and lumbar vertebrae which were used to find skeletal trunk height.

Stature and Enamel Defects

The following analyses describe difference in stature, between those individuals with and without skeletal indicators of growth disruption. Stature in skeletal populations is estimated by various means; consequently, skeletal height (SKH), stature (as calculated using the revised Fully technique without age), sitting height (STH) and the long bone measurements most commonly used to estimate stature (maximum femur and tibia lengths) were all considered. For the purposes of this study, an individual is considered to be "stressed" if they exhibit bilateral enamel defects in the anterior dentition (CBTS). All stature estimates exhibited

homoscedasticity, as determined by Levine's test of homogeneity. One-way analyses of variance (ANOVAs) were performed to determine whether there were significant differences in stature estimates between stressed and unstressed groups. No significant differences between groups were found in the male sample; however, females had near significant ($0.05 < p < 0.10$) differences for all measures (Table 5-2). Resampling statistics were used as an independent test of near significant results of ANOVAs. The results of these tests were significant for every case: SKH ($p = 0.0406$), Fully ($p = 0.0414$), STH ($p = 0.0296$), maximum femur ($p = 0.0466$) and tibia ($p = 0.0271$). Unstressed groups were larger than stressed groups for all measurements among females. As an exemplar, boxplots of the differences in tibia length can be seen in Figure 5-1.

There are many methods for coding dental enamel defects. For the purpose of comparison to other studies, supplementary coding techniques were employed and the data were analyzed accordingly. The coding criterion for tooth stress (TS) is based on the presence or absence of any hypoplastic lesion in an individual. Both hypoplastic pitting and lines have equal weight and a lesion need not be bilateral for inclusion. One-way analyses of variance (ANOVAs) were performed to determine whether there are significant differences in stature estimates between stressed and unstressed groups divided by TS criterion. No significant differences between groups were found in the male sample; however, females had near significant differences ($0.05 < p < 0.10$) for sitting height and tibia length (Table 5-3). Resampling statistics were used as an independent test of near significant results of ANOVAs and the results of these tests were significant in both cases: STH ($p = 0.0000$) and Tibia length ($p = 0.0335$). A p value of 0.0000, as in the case of STH, means that none of the resampling iterations matched the magnitude of the observed difference.

Because some researchers do not score pitting when scoring enamel hypoplasias, the linear enamel hypoplasia (LEH) code was created. Using this criterion, individuals with hypoplastic pitting in the absence of linear hypoplasias were removed from further analysis. However, this was true of only one individual in the sample, a female. As such, the results for the male sample coded by TS and LEH criteria are the same. One-way analyses of variance (ANOVAs) were performed on the female sample to determine whether there are significant differences in stature estimates between stressed and unstressed groups divided by LEH criterion. Near significant differences were found for the ANOVAs of sitting height and tibia length (Table 5-4). When resampling statistics were used, both sitting height and tibia length differences were found to be significantly different ($p = 0.0365$ and $p = 0.0299$ respectively).

Stature and Harris Lines

Individuals were divided into two groups based on the presence or absence of Harris lines and one-way ANOVAs were performed to determine whether there were any differences in stature estimates between these groups. The results of these ANOVAs (summarized in Table 5-5) show that there are no significant differences between groups, indicating no significant relationship between the existence of Harris lines and differences in stature estimates for the individuals examined. However, it is interesting to note that for all the measurements examined, those with Harris lines exhibit longer dimensions, on average, than those without.

A series of two-way ANOVAs were performed to determine whether or not there was any interaction effect between Harris lines and dental enamel defects as influences on stature. The ANOVA indicated no significant interaction (Table 5-6).

Stature and the Minimum Number of Stress Events

Stature estimates were compared to the minimum number of stress events (MNSE) score calculated for each individual based on the distribution of enamel defects as described in the

methods section. Males in this sample had a MNSE score between zero and five; females had a score between zero and four. Levine's test of homoscedasticity found that female skeletal height and revised Fully stature estimates had unequal error variances, violating the assumptions of the ANOVA; a Kruskal-Wallis test was performed on these data to evaluate the differences between groups based on the MNSE score. Differences in female skeletal height were found to be insignificant, χ^2 (4, 97) = 3.240, p = 0.518. The same was true of the female revised Fully stature estimates, χ^2 (4, 97) = 3.392, p = 0.494. One-way ANOVAs were conducted on the remaining cases (Table 5-7). In no instance do differences in the minimum number of stress events have a significant relationship to the stature estimates in question; however, an apparent trend toward decreased stature with increased number of stress events is notable in the female sample.

Stature estimates were also compared to the number of Harris lines in an individual (none, one, or more than one). In the female sample, no individual had more than one Harris line, so this analysis was confined to males. One-way ANOVAs were conducted and in no instance do differences in the number of Harris lines have a significant relationship to the stature estimates in question. Although the differences are not significant it is notable that average stature estimates are greatest in those with two or more Harris lines (Table 5-8).

Stature and the Age at which Stress Events Occur

Based on the relative location of a linear enamel hypoplasia, individuals in this study were divided into broad groups based on age of occurrence of dental stress events: a) infant stress events (birth to three years), b) childhood stress events (three to seven years), and c) those individuals who experienced stress during both infancy and childhood. These ages were calculated using the method of Goodman et al. (1980). The stature estimates for each of these groups were compared using ANOVAs to discover whether or not the age at which a stress event occurred had any effect on adult stature.

Due to unequal error variances in female measurements, as determined by Levine's test of homoscedasticity, Kruskal-Wallis tests were performed on this sample to evaluate differences in skeletal height (χ^2 (3, 97) = 2.773, p = 0.428), revised Fully stature estimates (χ^2 (3, 97) = 2.863, p = 0.413), and tibia length (χ^2 (3, 97) = 5.274, p = 0.153) between groups based on the age of the stress event. These results indicate that there are no significant locational differences in these measurements based on the age at which stress events occur. For all other indices, ANOVAs yield no significant results for comparisons of samples based on age of stress events; however, near significant ($0.05 < p < 0.10$) differences were found in male tibia lengths (Table 5-9). Two-way comparisons between samples based on the age of stress events were made using resampling statistics. For most of these comparisons, groups were not found to be significantly different; however, tibia length was found to be significantly different between those with only infant stress and those with only childhood stress (p = 0.0079). The results of the comparisons made using resampling statistics are summarized in Table 5-10. Although the results of these ANOVAs were insignificant, a pattern was observable in the female data. Mean lengths were highest among the unstressed and lowest when stress occurred in both infancy and childhood for all measurements. A second set of ANOVAs were performed comparing individuals with infant stress markers and those without any dental stress. Individuals who only formed enamel defects during childhood were excluded from analysis to isolate the effects of stress in infancy – possibly the most critical period of growth disruption. This investigation yielded significant results in tibia length among females (Table 5-11). Tibia length is significantly shorter in females who form enamel defects in infancy compared to an unstressed sample (p = 0.043).

Using Byers technique for aging Harris lines, individuals in this study were divided into three broad groups based on age of occurrence of stress events: a) infancy (birth to three years),

b) childhood (three to seven years), and c) those individuals who experienced stress during both infancy and childhood. No individual had visible Harris lines that formed during the juvenile or adolescent period. The stature estimates for each of these groups were compared using ANOVAs to discover whether or not the age at which a stress event occurred had any effect on adult stature. The ANOVAs yielded no significant or meaningful results given that the vast majority of Harris lines in this sample occurred during infancy. Only two females and four males exhibited Harris lines during childhood (Table 5-12).

Table 5-1. Descriptive statistics and normality of osteometric variables

Variable	Sex	N	Mean	S.D.	Min	Max	Statistic [a]	*p*
Humerus (mm) [b]	F	97	312.3	15.24	280.00	349.00	0.989	0.615
	M	107	339.9	18.57	296.50	382.50	0.991	0.668
Radius (mm) [b]	F	97	237.6	13.63	202.50	276.00	0.989	0.613
	M	107	266.2	14.03	234.00	301.00	0.994	0.942
Femur-bic (mm) [b]	F	97	437.2	25.15	380.00	490.50	0.987	0.462
	M	107	475.1	25.68	410.50	530.50	0.980	0.099
Femur-max (mm) [b]	F	97	442.9	25.48	386.50	495.50	0.983	0.258
	M	107	479.2	26.03	412.50	537.00	0.981	0.138
Tibia (mm) [b]	F	97	366.8	22.50	319.00	420.00	0.986	0.400
	M	107	402.3	25.01	344.00	472.00	0.994	0.902
SKH (cm)	F	97	148.0	6.38	135.09	162.62	0.985	0.351
	M	107	159.8	6.60	146.31	175.07	0.983	0.192
STH (cm)	F	97	45.0	2.34	39.70	50.10	0.989	0.607
	M	107	48.0	2.12	41.44	54.53	0.988	0.431

[a] Normality tests performed using the Shapiro-Wilk statistic

[b] Description of paired elements refers to the average

Table 5-2. ANOVA –Stature estimates by dental enamel defects (CBTS)

		N	Mean	S.D.	Levine's	F	*p*
Male							
	Skeletal height (SKH)						
	Unstressed	42	159.82	6.853			
	Stressed	65	159.74	6.486	0.405	0.004	0.948
	Revised Fully stature						
	Unstressed	42	172.16	6.893			
	Stressed	65	172.05	6.538	0.427	0.007	0.932
	Sitting height (STH)						
	Unstressed	42	47.96	2.260			
	Stressed	65	48.02	2.033	0.846	0.015	0.904
	Max. femur length						
	Unstressed	42	481.50	26.115			
	Stressed	65	478.75	25.884	0.993	0.285	0.594
	Tibia length						
	Unstressed	42	401.29	24.410			
	Stressed	65	403.15	25.337	0.758	0.143	0.706
Female							
	Skeletal height (SKH)						
	Unstressed	43	149.27	6.672			
	Stressed	54	147.04	6.018	0.711	2.982	0.087 [a]
	Revised Fully stature						
	Unstressed	43	161.56	6.691			
	Stressed	54	159.29	6.061	0.752	3.054	0.084 [a]
	Sitting height (STH)						
	Unstressed	43	45.50	2.285			
	Stressed	54	44.62	2.325	0.672	3.472	0.066 [a]
	Max. Femur length						
	Unstressed	43	448.23	26.411			
	Stressed	54	439.46	24.702	0.886	2.837	0.095 [a]
	Tibia length						
	Unstressed	43	371.86	22.271			
	Stressed	54	363.07	21.943	0.927	3.788	0.055 [a]

[a] Near significant ($0.05 < p < 0.10$)

Table 5-3. ANOVA –Stature estimates by dental enamel defects (TS)

		N	Mean	S.D.	Levine's	F	*p*
Male							
	Skeletal height (SKH)						
	Unstressed	37	159.97	6.875			
	Stressed	70	159.66	6.499	0.478	0.053	0.819
	Revised Fully stature						
	Unstressed	37	172.29	6.914			
	Stressed	70	171.99	6.551	0.497	0.047	0.828
	Sitting height (STH)						
	Unstressed	37	48.03	1.950			
	Stressed	70	47.98	2.210	0.476	0.013	0.909
	Max. femur length						
	Unstressed	37	481.76	26.700			
	Stressed	70	478.81	25.583	0.838	0.311	0.578
	Tibia length						
	Unstressed	37	401.57	25.256			
	Stressed	70	402.87	24.848	0.969	0.066	0.798
Female							
	Skeletal height (SKH)						
	Unstressed	36	149.27	6.660			
	Stressed	61	147.29	6.147	0.753	2.209	0.141
	Revised Fully stature						
	Unstressed	36	161.57	6.666			
	Stressed	61	159.54	6.193	0.791	2.312	0.132
	Sitting height (STH)						
	Unstressed	36	45.56	2.301			
	Stressed	61	44.68	2.316	0.902	3.232	0.075 [a]
	Max. femur length						
	Unstressed	36	447.86	26.784			
	Stressed	61	440.69	24.901	0.797	1.776	0.186
	Tibia length						
	Unstressed	36	372.42	22.606			
	Stressed	61	363.75	21.838	0.993	3.471	0.066 [a]

[a] Near significant ($0.05 < p < 0.10$)

Table 5-4. ANOVA – Proxies for stature by dental enamel defects (LEH) in females

	N	Mean	S.D.	Levine's	F	*p*
Skeletal height (SKH)						
Unstressed	36	149.27	6.660			
Stressed	60	147.29	6.199	0.845	2.182	0.143
Revised Fully stature						
Unstressed	36	161.57	6.666			
Stressed	60	159.53	6.245	0.880	2.293	0.133
Sitting height (STH)						
Unstressed	36	45.56	2.301			
Stressed	60	44.67	2.333	0.854	3.290	0.073 [a]
Max. femur length						
Unstressed	36	447.86	26.784			
Stressed	60	440.53	25.082	0.851	1.825	0.180
Tibia length						
Unstressed	36	372.42	22.606			
Stressed	60	363.55	21.963	0.973	3.588	0.060 [a]

[a] Near significant ($0.05 < p < 0.10$)

Table 5-5. ANOVA –Stature estimates by the presence or absence of Harris lines

		N	Mean	S.D.	Levine's	F	*p*
Male							
	Skeletal height (SKH)						
	Absent	56	159.62	6.926			
	Present	40	160.67	6.619	0.613	0.555	0.458
	Revised Fully stature						
	Absent	56	171.91	6.989			
	Present	40	173.01	6.641	0.637	0.599	0.441
	Sitting height (STH)						
	Absent	56	47.96	1.976			
	Present	40	48.20	2.340	0.411	0.290	0.591
	Max. femur length						
	Absent	56	480.32	27.403			
	Present	40	482.63	25.820	0.944	0.173	0.678
	Tibia length						
	Absent	56	401.71	25.796			
	Present	40	405.80	25.588	0.652	0.589	0.445
Female							
	Skeletal height (SKH)						
	Absent	56	147.82	6.631			
	Present	31	148.47	6.230	0.701	0.201	0.655
	Revised Fully stature						
	Absent	56	160.13	6.661			
	Present	31	160.71	6.269	0.718	0.157	0.693
	Sitting height (STH)						
	Absent	56	44.94	2.422			
	Present	31	45.27	2.237	0.385	0.377	0.541
	Max. femur length						
	Absent	56	441.86	26.551			
	Present	31	445.10	25.513	0.992	0.305	0.582
	Tibia length						
	Absent	56	365.18	22.820			
	Present	31	369.71	21.125	0.737	0.829	0.365

Table 5-6. Interaction effects of Harris lines (HL) and enamel defects (CBTS) on stature

		F	*p*
Male			
	Skeletal Height (SKH)		
	HL	0.478	0.491
	CBTS	0.162	0.689
	HL * CBTS	0.013	0.910
	Revised Fully Stature		
	HL	0.518	0.473
	CBTS	0.145	0.704
	HL * CBTS	0.013	0.909
	Sitting Height (STH)		
	HL	0.124	0.725
	CBTS	0.233	0.631
	HL * CBTS	0.995	0.321
	Max. Femur Length		
	HL	0.194	0.661
	CBTS	0.002	0.964
	HL * CBTS	0.045	0.833
	Tibia Length		
	HL	0.432	0.512
	CBTS	0.717	0.399
	HL * CBTS	0.127	0.722
Female			
	Skeletal Height (SKH)		
	HL	0.309	0.580
	CBTS	1.483	0.227
	HL * CBTS	0.000	0.997
	Revised Fully Stature		
	HL	0.255	0.615
	CBTS	1.510	0.223
	HL * CBTS	0.000	0.993
	Sitting Height (STH)		
	HL	0.655	0.421
	CBTS	2.914	0.092
	HL * CBTS	0.092	0.762
	Max. Femur Length		
	HL	0.366	0.547
	CBTS	1.339	0.251
	HL * CBTS	0.132	0.717

Table 5-6. Continued

	F	*p*
Tibia Length		
HL	1.114	0.294
CBTS	2.389	0.126
HL * CBTS	0.007	0.934

Table 5-7. ANOVA – Stature by minimum number of stress events (MNSE)

		N	Mean	S.D.	Levine's	F	*p*
Male							
	Skeletal height (SKH)						
	0	42	159.82	6.853			
	1	19	159.36	6.305			
	2	27	160.99	7.036			
	3	15	158.53	5.428			
	4	3	160.32	7.214			
	5	1	149.47	.	0.437	0.788	0.561
	Revised fully stature						
	0	42	172.16	6.893			
	1	19	171.69	6.452			
	2	27	173.32	7.037			
	3	15	170.79	5.465			
	4	3	172.56	7.264			
	5	1	161.85	.	0.460	0.785	0.563
	Sitting height (STH)						
	0	42	47.96	2.260			
	1	19	48.06	1.842			
	2	27	48.20	1.907			
	3	15	47.66	2.359			
	4	3	49.03	2.440			
	5	1	44.39	.	0.889	0.850	0.517
	Maximum femur length						
	0	42	481.50	26.115			
	1	19	475.21	26.749			
	2	27	484.89	28.133			
	3	15	474.27	21.382			
	4	3	477.67	18.583			
	5	1	451.00	.	0.592	0.744	0.592
	Maximum tibia length						
	0	42	401.29	24.410			
	1	19	401.21	23.827			
	2	27	405.56	27.392			
	3	15	402.47	24.480			
	4	3	407.33	29.771			
	5	1	373.00	.	0.797	0.404	0.845
Female							
	Skeletal height (SKH)						
	0	43	149.27	6.672			
	1	26	146.85	4.745			
	2	18	147.95	7.114			
	3	9	146.13	7.603			
	4	1	143.85	.	0.022 [a, b]	.	.

Table 5-7. Continued

		N	Mean	S.D.	Levine's	F	*p*
Revised fully stature							
	0	43	161.56	6.691			
	1	26	159.09	4.831			
	2	18	160.26	7.169			
	3	9	158.31	7.506			
	4	1	155.75	.	0.031 [a, b]	.	.
Sitting height (STH)							
	0	43	45.50	2.285			
	1	26	44.46	1.949			
	2	18	44.81	2.662			
	3	9	44.51	2.884			
	4	1	46.43	.	0.210	1.063	0.379
Maximum femur length							
	0	43	448.23	26.411			
	1	26	441.08	21.583			
	2	18	443.00	29.100			
	3	9	431.00	23.696			
	4	1	410.00	.	0.330	1.402	0.240
Maximum tibia length							
	0	43	371.86	22.271			
	1	26	362.92	18.560			
	2	18	366.83	25.551			
	3	9	358.00	24.990			
	4	1	345.00	.	0.242	1.344	0.260

[a] Significant ($p < 0.05$)

[b] The assumptions of the F-test are violated where conditions of homoscedasticity are not met.

Table 5-8. ANOVA – Stature estimates by number of Harris lines

	N	Mean	S.D.	Levine's	F	*p*
Male						
Skeletal height (SKH)						
No Harris lines	56	159.62	6.926			
One Harris line	30	160.39	6.238			
Two or more Harris lines	10	161.50	7.964	0.508	0.375	0.688
Revised Fully stature						
No Harris lines	56	171.91	6.989			
One Harris line	30	172.73	6.276			
Two or more Harris lines	10	173.84	7.947	0.560	0.395	0.675
Sitting height (STH)						
No Harris lines	56	47.96	1.976			
One Harris line	30	47.94	2.280			
Two or more Harris lines	10	48.97	2.466	0.852	1.031	0.361
Maximum femur length						
No Harris lines	56	480.32	27.403			
One Harris line	30	482.33	22.934			
Two or more Harris lines	10	483.50	34.539	0.197	0.093	0.912
Tibia length						
No Harris lines	56	401.71	25.796			
One Harris line	30	405.53	24.895			
Two or more Harris lines	10	406.60	28.968	0.802	0.298	0.743

Table 5-9. ANOVA – Stature estimates by age of enamel defect

	N	Mean	S.D.	Levine's	F	*p*
Male						
Skeletal height (SKH)						
No stress	42	159.82	6.853			
Infant stress	10	163.30	6.875			
Childhood stress	18	157.97	6.258			
Infant & childhood stress	37	159.63	6.277	0.597	1.418	0.242
Revised Fully stature						
No stress	42	172.16	6.893			
Infant stress	10	175.68	6.986			
Childhood stress	18	170.31	6.340			
Infant & childhood stress	37	171.92	6.293	0.571	1.432	0.238
Sitting height (STH)						
No stress	42	47.96	2.260			
Infant stress	10	48.50	1.996			
Childhood stress	18	48.07	2.060			
Infant & childhood stress	37	47.86	2.063	0.986	0.247	0.863
Maximum femur length						
No stress	42	481.50	26.115			
Infant stress	10	491.40	26.850			
Childhood stress	18	468.89	27.685			
Infant & childhood stress	37	480.14	23.535	0.921	1.840	0.144
Tibia length						
No stress	42	401.29	24.410			
Infant stress	10	416.60	21.854			
Childhood stress	18	392.89	24.726			
Infant & childhood stress	37	404.51	25.067	0.993	2.148	0.099 [b]
Female						
Skeletal height (SKH)						
No stress	43	149.27	6.672			
Infant stress	13	148.04	4.231			
Childhood stress	19	147.09	5.669			
Infant & childhood stress	22	146.40	7.251	0.039 [a, c]	.	.
Revised Fully stature						
No stress	43	161.56	6.691			
Infant stress	13	160.27	4.314			
Childhood stress	19	159.37	5.741			
Infant & childhood stress	22	158.64	7.266	0.045 [a, c]	.	.

Table 5-9. Continued

	N	Mean	S.D.	Levine's	F	*p*
Sitting height (STH)						
No stress	43	45.50	2.285			
Infant stress	13	44.65	2.214			
Childhood stress	19	44.65	2.280			
Infant & childhood stress	22	44.57	2.527	0.684	1.138	0.338
Maximum femur length						
No stress	43	448.23	26.411			
Infant stress	13	446.23	17.234			
Childhood stress	19	441.26	24.299			
Infant & childhood stress	22	433.91	28.294	0.082	1.633	0.187
Tibia length						
No stress	43	371.86	22.271			
Infant stress	13	362.69	11.071			
Childhood stress	19	367.11	23.664			
Infant & childhood stress	22	359.82	25.284	0.027 [a, c]	.	.

[a] Significant ($p < 0.05$)

[b] Near significant ($0.05 < p < 0.10$)

[c] The assumptions of the F-test are violated where conditions of homoscedasticity are not met.

Table 5-10. Results of resampling for male tibia length by age of the enamel defect

Age categories compared	*p*
No stress – infant stress	0.9674
No stress – childhood stress	0.1052
No stress – infant and childhood stress	0.7180
Infant stress – childhood stress	0.0079 [a]
Infant stress – infant and childhood stress	0.0856
Childhood stress – infant and childhood stress	0.9500

[a] Significant ($p < 0.05$)

Table 5-11. ANOVA – Stature estimates by infant enamel defect

	N	Mean	S.D.	Levine's	F	*p*
Male						
Skeletal height (SKH)						
No stress	42	159.82	6.853			
Infant stress	47	160.41	6.510	0.474	0.173	0.679
Revised Fully stature						
No stress	42	172.16	6.893			
Infant stress	47	172.72	6.555	0.491	0.151	0.698
Sitting height (STH)						
No stress	42	47.96	2.260			
Infant stress	47	47.99	2.045	0.838	0.004	0.951
Maximum femur length						
No stress	42	481.50	26.115			
Infant stress	47	482.53	24.418	0.672	0.037	0.848
Tibia length						
No stress	42	401.29	24.410			
Infant stress	47	407.09	24.702	0.879	1.236	0.269
Female						
Skeletal height (SKH)						
No stress	43	149.27	6.672			
Infant stress	34	147.25	6.211	0.760	1.845	0.178
Revised Fully stature						
No stress	43	161.56	6.691			
Infant stress	34	159.48	6.246	0.789	1.937	0.168
Sitting height (STH)						
No stress	43	45.50	2.285			
Infant stress	34	44.67	2.385	0.484	2.414	0.124
Maximum femur length						
No stress	43	448.23	26.411			
Infant stress	34	439.76	24.414	0.703	2.085	0.153
Tibia length						
No stress	43	371.86	22.271			
Infant stress	34	361.62	20.836	0.699	4.250	0.043 [a]

[a] Significant ($p < 0.05$)

Table 5-12. ANOVA – Stature estimates by age of Harris lines

	N	Mean	S.D.	Levine's	F	*p*
Male						
Skeletal height (SKH)						
No stress	56	159.62	6.926			
Infant stress	36	161.47	6.384			
Childhood stress	1	156. 70	.			
Infant & childhood stress	3	152.33	4.149	0.304	2.044	0.113
Revised fully stature						
No stress	56	171.91	6.989			
Infant stress	36	173.81	6.410			
Childhood stress	1	169.06	.			
Infant & childhood stress	3	164.71	4.233	0.318	2.018	0.117
Sitting height (STH)						
No stress	56	47.96	1.976			
Infant stress	36	48.31	2.350			
Childhood stress	1	49.06	.			
Infant & childhood stress	3	46.53	2.310	0.590	0.798	0.498
Maximum femur length						
No stress	56	480.32	27.403			
Infant stress	36	485.67	25.019			
Childhood stress	1	461.00	.			
Infant & childhood stress	3	453.33	18.930	0.382	1.678	0.177
Tibia length						
No stress	56	401.71	25.796			
Infant stress	36	408.53	25.073			
Childhood stress	1	384.00	.			
Infant & childhood stress	3	380.33	20.526	0.472	1.588	0.198
Female						
Skeletal height (SKH)						
No stress	56	147.82	6.631			
Infant stress	29	148.08	6.152			
Childhood stress	2	154.18	5.917	0.806	0.933	0.398
Revised fully stature						
No stress	56	160.13	6.661			
Infant stress	29	160.31	6.205			
Childhood stress	2	166.41	5.639	0.783	0.901	0.410
Sitting height (STH)						
No stress	56	44.94	2.422			
Infant stress	29	45.18	2.283			
Childhood stress	2	46.56	0.733	0.286	0.511	0.602
Maximum femur length						
No stress	56	441.86	26.551			
Infant stress	29	443.72	25.794			
Childhood stress	2	465.00	7.071	0.291	0.772	0.465

Table 5-12. Continued

	N	Mean	S.D.	Levine's	F	*p*
Tibia length						
No stress	56	365.18	22.820			
Infant stress	29	368.62	21.333			
Childhood stress	2	385.50	10.607	0.535	0.954	0.389

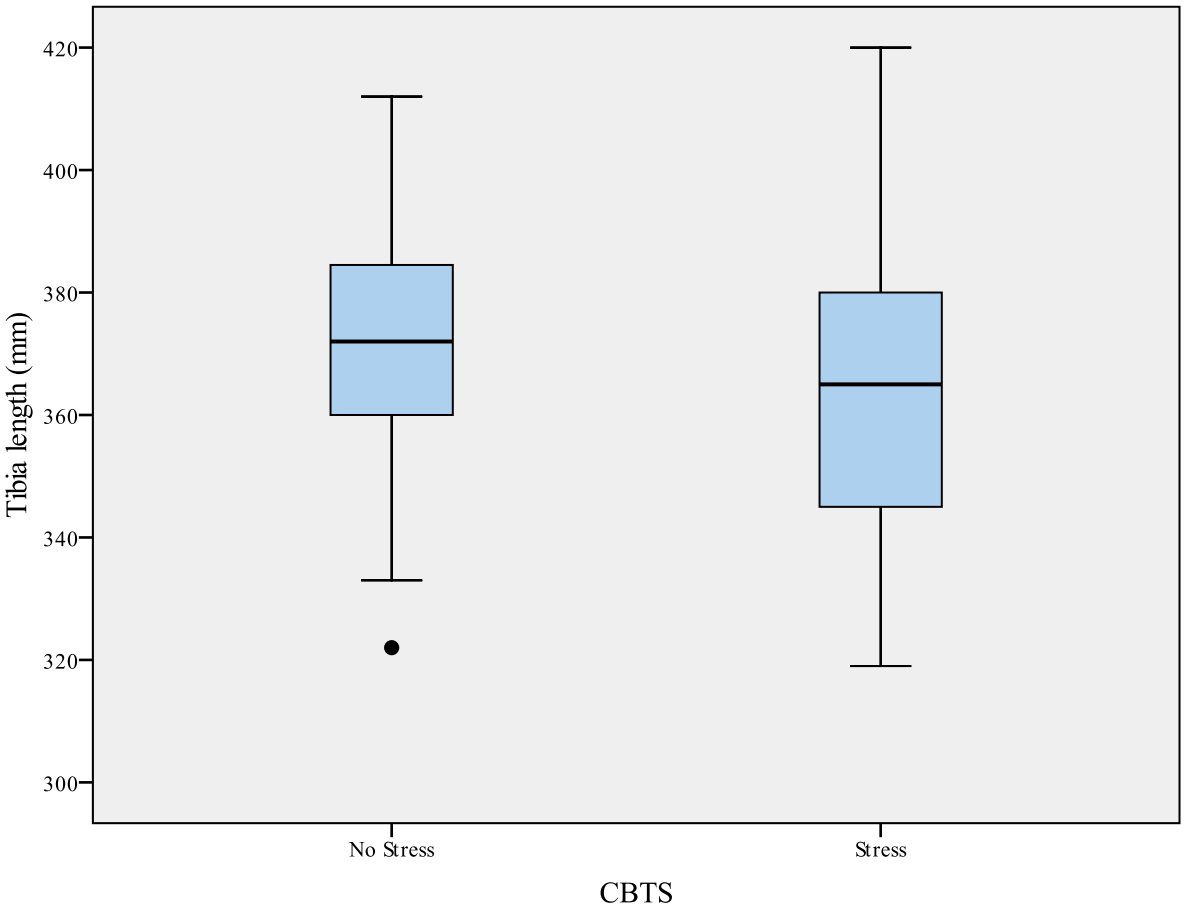


Figure 5-1. Boxplot of female tibia length divided by CBTS stress codes. Differences in tibia length between stressed and unstressed groups were found to be significant (p = 0.0271). This boxplot is used as an example of the distributional differences between stressed and unstressed groups.

CHAPTER 6
RESULTS: SEXUAL DIMORPHISM AND STRESS

The relative degree of sexual dimorphism between the two groups was found using the formula $\ln \bar{X}_M - \ln \bar{X}_F$ for stature estimates and long bone length, where $\bar{X}_M$ is the male mean and $\bar{X}_F$ is the female mean. The population was then divided based on the presence or absence of stress markers based on the CBTS and HL criteria described in the Methods section. The results of these calculations can be observed in Table 6-1 and Table 6-2.

In this study, the null hypothesis states that there are no metric differences between individuals who have experienced growth disruption compared to those who have not. The alternative hypothesis with regards to sexual dimorphism is that unstressed populations will have higher rates of sexual dimorphism due to the fact that males are more likely to reach their genetic potential under favorable circumstances. A corollary with this alternative hypothesis is that female size should be similar in stressed and unstressed populations due to fat and nutrient reserves which help females buffer the negative effects of nutritional stress (Stini, 1969, 1982, 1985).

In Chapter 5, the results indicate that where significant differences in mean stature and long bone estimates occur, these differences are attributed to the female portion of the sample, not the males. Expectations based on Stini's (1969, 1982, 1985) findings were that the opposite pattern would be observed. The mean measures suggest that female measurements vary more in accordance with stress than male measurements conducted in this study.

A comparison of sexual dimorphism scores across various measurements shows that for both stressed and unstressed populations, the greatest amount of sexual dimorphism is found in the radius and in tibia length in stressed individuals. The smallest dimorphism scores are found in sitting height estimates.

Differences in sexual dimorphism scores between stressed and unstressed groups vary depending on the type of stress marker used. Larger differences in sexual dimorphism scores are found by using CBTS markers than are found by using HL markers (Table 6-3). In addition, when the population is divided by CBTS criterion, sexual dimorphism is greater in the stressed group than in the unstressed group for every measure, the opposite of expectations based on Stini's (1969, 1982, 1985) findings. When the population is divided by HL markers the same was true only of stature estimates. It is important to note that the differences in dimorphism scores are an order of magnitude smaller for the population divided by HL criterion than in the population divided by CBTS criterion and, as such, they are likely less meaningful.

Because sexual dimorphism, as it is calculated here, is found by taking the difference of two means, there is no measure of dispersion associated with the sexual dimorphism score; consequently, traditional statistical analyses cannot be completed on the raw data that would allow us to reject the null hypothesis that claims no differences between stressed and unstressed groups. The (one-tailed) alternative hypothesis is that sexual dimorphism is greater in unstressed groups. To address that question, a bootstrap procedure was employed using Resampling Stats 5.0.2 software. The stressed and unstressed, male and female populations were pooled, and then resampled with replacement using the original sample sizes. In each resampling, the log difference between male and female means was calculated for the stressed and unstressed groups. A difference in dimorphism was then found by subtracting the stressed measure of dimorphism from the unstressed sample. Probabilities were determined by counting the number of times the resampling mean difference was greater or equal to the empirical measure of difference in sexual dimorphism which was calculated by subtracting the original dimorphism score in the stressed sample from the unstressed score. Each resampling procedure was run with

1,000 iterations except where probabilities approached significance (in the case of radius length partitioned by CBTS). In that instance, the test was run with 10,000 iterations. Because the alternative hypothesis was directional, the probability was calculated as 1-*p* where the empirical difference in dimorphism was negative. This procedure tests a second alternative hypothesis – that sexual dimorphism is greater in the stressed sample. In no case did the statistical analysis support the first or second alternative hypothesis (at the $p < 0.05$ significance level); i.e., the null hypothesis - no metrically observable difference between the size and shape of individuals who have experienced growth disruption and those who have not - could not be rejected. The results of this procedure are summarized in Table 6-3.

Table 6-1. Sexual dimorphism scores for unstressed individuals

	Stress Marker	$\overline{X}_M$	$\overline{X}_F$	ln $\overline{X}_M$	ln $\overline{X}_F$	Sexual Dimorphism
Skeletal height (SKH)	CBTS	159.822	149.266	5.074	5.006	0.068
	HL	159.616	147.821	5.073	4.996	0.077
Revised Fully Stature	CBTS	172.162	161.556	5.148	5.085	0.064
	HL	171.914	160.128	5.147	5.076	0.071
Sitting height (STH)	CBTS	47.964	45.497	3.870	3.818	0.053
	HL	47.961	44.940	3.870	3.805	0.065
Maximum femur length [a]	CBTS	48.123	44.792	3.874	3.802	0.072
	HL	47.978	44.143	3.871	3.787	0.083
Bicondylar femur length [a]	CBTS	47.701	44.191	3.865	3.789	0.076
	HL	47.571	43.563	3.862	3.774	0.088
Tibia length [a]	CBTS	40.126	37.170	3.692	3.615	0.077
	HL	40.171	36.486	3.693	3.597	0.096
Humerus length [a]	CBTS	33.989	31.592	3.526	3.453	0.073
	HL	34.098	31.124	3.529	3.438	0.091
Radius length [a]	CBTS	26.637	24.101	3.282	3.182	0.100
	HL	26.657	23.724	3.283	3.166	0.117

[a] This is the average of the left and right elements where both are available.

Table 6-2. Sexual dimorphism scores for stressed individuals

	Stress Marker	$\overline{X}_M$	$\overline{X}_F$	ln $\overline{X}_M$	ln $\overline{X}_F$	Sexual Dimorphism
Skeletal height (SKH)	CBTS	159.736	147.037	5.074	4.991	0.083
	HL	160.665	148.472	5.079	5.000	0.079
Revised Fully Stature	CBTS	172.049	159.289	5.148	5.071	0.077
	HL	173.011	160.707	5.153	5.080	0.074
Sitting height (STH)	CBTS	48.015	44.618	3.872	3.798	0.073
	HL	48.199	45.265	3.875	3.813	0.063
Maximum femur length [a]	CBTS	47.788	43.883	3.867	3.782	0.085
	HL	48.185	44.452	3.875	3.794	0.081
Bicondylar femur length [a]	CBTS	47.391	43.336	3.858	3.769	0.089
	HL	47.776	43.889	3.867	3.782	0.085
Tibia length [a]	CBTS	40.292	36.281	3.696	3.591	0.105
	HL	40.546	36.982	3.702	3.610	0.092
Humerus length [a]	CBTS	33.985	30.944	3.526	3.432	0.094
	HL	34.088	31.419	3.529	3.447	0.082
Radius length [a]	CBTS	26.612	23.482	3.281	3.156	0.125
	HL	26.718	23.892	3.285	3.174	0.112

[a] This is the average of the left and right elements where both are available.

Table 6-3. Differences in sexual dimorphism between unstressed and stressed populations [a]

Measurement	CBTS markers	*p*	HL markers	*p*
Skeletal height (SKH)	-0.0145	0.128	-0.0022	0.472
Revised Fully Stature	-0.0135	0.121	-0.0027	0.464
Sitting height (STH)	-0.0206	0.080	0.0022	0.407
Maximum femur length [b]	-0.0135	0.214	0.0027	0.392
Bicondylar femur length [b]	-0.0130	0.215	0.0031	0.411
Tibia length [b]	-0.0283	0.064	0.0042	0.399
Humerus length [b]	-0.0206	0.090	0.0098	0.247
Radius length [b]	-0.0251	0.061	0.0048	0.368

[a] Difference measured as the unstressed score minus the stressed score.

[b] This is the average of the left and right elements where both are available.

CHAPTER 7
RESULTS: PROPORTIONS AND STRESS

The following analyses describe differences in proportional indices, expressed as ratios, between those individuals with and those without skeletal indicators of growth disruption. The components of these ratios were significantly correlated for all proportions, whether the data were divided by gender or combined across genders (Table 7-1). This result means that although ratios are dimensionless, they are not independent of body size and allometric factors need to be considered when investigating differences between groups.

Proportions and dental enamel defects

Individuals were determined to be either "stressed" or "unstressed" based on the presence or absence of bilateral dental enamel defects in the anterior dentition (CBTS) by the criteria described in the Methods section. All proportions, except for the crural index in males, exhibited homoscedasticity, as determined by Levine's test of homogeneity. For those proportions that were found to be homogenous, one-way analyses of variance (ANOVAs) were performed to determine whether there were significant differences in proportional indices between stressed and unstressed groups. In no case were significant differences between groups observed (Table 7-2). In the case of the male crural index, the Mann-Whitney test found no significant differences in the crural index between the stressed and unstressed groups of males ($p = 0.080$).

Because researchers code enamel defects using different methods, different coding techniques were used to form a basis of comparison with other studies. The coding criterion for tooth stress (TS) is based on the presence or absence of any hypoplastic lesion, regardless of the type or whether or not a lesion is found on its antimere. One-way ANOVAs were performed to determine whether there are significant differences in the distribution of proportional differences between stressed and unstressed groups divided by TS criterion. No significant differences

between groups are found; however, males have near significant differences in crural indices (Table 7-3). Resampling statistics were used as an independent test of the near significant results of ANOVAs and the differences were determined to be insignificant ($p = 0.9618$); however, the resampling procedure is directional and tests the alternative hypothesis that the unstressed values are greater than the stressed values. In this case, $1 - p < 0.05$, which means there is a significant difference between groups, but the difference is not in the direction proposed. In the case of the male crural index, values are significantly higher in the stressed group.

Because some researchers do not score pitting when scoring enamel hypoplasias, the linear enamel hypoplasia (LEH) code was created. Using this criterion, individuals with hypoplastic pitting in the absence of linear hypoplasias were removed from further analysis. However, this was only true of one female in the sample; as a result, the male sample coded by TS and LEH criteria are equivalent. One-way ANOVAs were performed on the female sample to determine whether there are significant differences in proportional indices between stressed and unstressed groups divided by LEH criterion. No significant or near significant differences were observed (Table 7-4).

Proportions and Harris lines

Individuals were divided into two groups based on the presence or absence of Harris lines (see Methods section) and one-way ANOVAs were performed to determine whether there were any differences in proportional indices between these groups. The results of these ANOVAs (summarized in Table 7-5) show that there are no significant differences between groups, indicating no relationship between the existence of Harris lines and differences in proportional indices for the individuals examined.

A series of two-way ANOVAs were performed to determine whether or not there was any interaction effect between Harris lines and dental enamel defects in their potential influence on

proportions. The results show that no interaction effect exists between these indicators of stress (Table 7-6).

Scaling

Correlations suggest that the components of ratios are not independent of body size (see Table 7-1). Because of this fact, a scaling analysis is necessary to qualify the results of the preceding ANOVAs. For example, if a stressed population is found to be larger than an unstressed one for any given measurement, there is a scaling effect that needs to be considered. A non-zero slope indicated that size influenced proportions in five of eight cases (see Figures 7-1 through 7-4). In three of eight regressions, the confidence interval for the slope included zero, signifying that the effect of skeletal height on proportions in those instances was negligible at best (Table 7-7). Based on the magnitude of the correlation coefficient it can be inferred that skeletal height is moderately related to the intermembral index in males and females and the humerofemoral index in females. For the remaining proportional indices, less than nine percent ($R^2 \leq 0.085$) of the variance can be explained by skeletal height. The components of the ratios were examined to see whether the numerator or denominator in each proportional ratio has greater effect on how proportional ratios are scaling (Figure 7-5 and Figure 7-6). The components of the ratios follow a pattern consistent with the negative slope exhibited when scaling ratios. A negative slope in Figures 7-1 through Figure 7-4 suggests that the variable in the denominator of a ratio is changing relative to size at a faster rate than the variable in the numerator.

Scaling Differences Based on Enamel Defects

Reduced major axis (RMA) regressions were used to analyze how the components of ratios scale to one another and to see whether or not scaling differs between groups designated

"stressed" or "unstressed" based on the presence or absence of enamel defects. Log-transformed components of ratios were scaled to one another using the RMA software (Bohonak 2004).

Differences in scaling exist between groups based on stress categories, but also vary by gender. The majority of RMAs find that the components of ratios scale isometrically to one another. For males, these indices include: the unstressed crural index, stressed and unstressed humerofemoral indices, stressed and unstressed radiohumeral indices, stressed and unstressed sitting height indices and the stressed intermembral index. Female isometric indices include: stressed and unstressed crural indices, the unstressed radiohumeral index, the unstressed sitting height index, and the stressed intermembral index. Three ratios are positively allometric, indicating that the numerator of the ratio scales at a faster rate than the denominator. These include the stressed male crural index, the stressed female radiohumeral index and the stressed female sitting-height index. The stressed and unstressed humerofemoral index in females and the unstressed male and female intermembral indices were found to be negatively allometric (Table 7-8).

RMA2 software (Cole, 1997) was used to test whether the scaling of skeletal dimensions between stressed and unstressed groups was significantly different (Table 7-9). Minor differences in slope and intercept values are noted in Table 7-8 and Table 7-9; these differences reflect calculation differences between the Bohonak (2004) and Cole (1997) software and are likely due to rounding errors that are exaggerated in log transformed data. These differences are considered negligible and do not influence interpretation. RMA2 analyses find significant differences in slope between stressed and unstressed groups for the humerofemoral index and the intermembral index in males. For these indices, the length of the humerus (or upper limb length) is scaling faster than femoral (or lower limb length) in the stressed group.

In cases where RMA slopes were not significantly different, intercept differences were tested by fitting a regression lined through the combined dataset and counting the number of individuals with and without enamel defects that fall above and below the line. This information was then placed in a 2x2 contingency table and Fisher's exact test was used to test the difference (Tsutakawa and Hewett, 1977). In no case were intercept differences significant. In other words, for any given value of X, the value of Y is not significantly different between samples.

These RMA regressions are represented graphically in Figure 7-7 through Figure 7-16. The results of the ANOVAs between stressed and unstressed groups suggest that there are no significant differences in proportional indices between individuals grouped by the presence or absence of dental enamel defects. An investigation into the question of scaling suggests that allometric differences between such groups are subtle.

Scaling Differences Based on Harris Lines

Reduced major axis (RMA) regressions were also conducted to determine whether or not scaling differs between those with and without Harris lines. Log-transformed components of ratios were scaled to one another using the RMA software of Bohonak (2004) in Table 7-10.

Once again, differences in scaling exist between groups based on the presence or absence of Harris lines and also vary by gender. Male proportional ratios scale isometrically for those with and without Harris lines in all but the crural index. In this case males are positively allometric with tibial length increasing at a faster rate than femoral length. Females vary greatly. For females without Harris lines, the crural index, humerofemoral index, and the intermembral index scale isometrically. Females with Harris lines scale isometrically for the crural index, the radiohumeral index and the sitting-height index. Radiohumeral and sitting height indices scale positively in females without Harris lines. Humerofemoral and intermembral indices scale negatively in females with Harris lines, with femoral (or lower limb) length increasing at a faster

rate than humerus (or upper limb) length. The length of the radius increases at a faster rate than the humerus and sitting height increases at a faster rate than skeletal height.

RMA2 software (Cole, 1997) was used to test whether the allometric differences between those with and without Harris lines were significantly different (summarized in Table 7-11). The only significant differences in slope are for female humerofemoral indices and the intermembral indices. These are also the only instances where those individuals with Harris lines exhibit negative allometry. No intercept differences were found using Fisher's exact test.

RMA regressions representing the relationship between Harris lines and the scaling of proportional indices are depicted graphically in Figure 7-17 through Figure 7-26. ANOVAs suggest that there are no significant differences in proportional indices between those with and without Harris lines. RMA regressions suggest that there are some scaling differences between groups, but that those differences are subtle. Large differences in slope as well as high r^2 values for the regressions are necessary to interpret a scaling difference between groups as significant. R^2 values for these regressions range from 0.29 to 0.84 with a median value of 0.735.

Proportions and the Minimum Number of Stress Events

Using the aging techniques for enamel hypoplasias described in the Methods section, a minimum number of stress events (MNSE) score was calculated for each individual. Levine's test of homoscedasticity found that male crural indices had unequal error variances, violating the assumptions of the ANOVA; a Kruskal-Wallis test was performed on this sample to evaluate the differences in crural indices between groups based on the MNSE score. The test was insignificant, $\chi 2\ (4, 65) = 3.13$, $p = 0.54$. One-way ANOVAs were conducted on the remaining cases (Table 7-12). In no instance do differences in the minimum number of stress events appear to affect the proportional indices in question.

Proportions were also compared to the number of Harris lines (none, one, or more than one). In the female sample, no individual had more than one Harris line, so this analysis was confined to males. Due to unequal error variances in male crural indices, as determined by Levine's test of homoscedasticity, a Kruskal-Wallis test was performed on this sample to evaluate differences in crural indices between groups based on the age of the stress event. The test was insignificant, $\chi 2$ (2, 96) = 1.244, p = 0.537. For all other indices, one-way ANOVAs were conducted and in no instance do differences in the number of Harris lines have a significant relationship to the proportions in question; however, near significant results were obtained for the intermembral index (Table 7-13). Resampling statistics were performed as an independent test for ANOVAs with near significant results. A significant difference in intermembral indices exists between those individuals with no Harris lines and those with one Harris line (p = 0.0336). However, when those with no Harris lines and those with two or more lines were compared, the differences were insignificant (p = 0.7798). Similarly, intermembral indices in those with one Harris line and those with two or more Harris lines were not significantly different (p = 0.986).

Proportions and the Age at which Stress Events Occur

Based on the relative location of a linear enamel hypoplasia, individuals in this study were divided into broad groups based on age of occurrence of dental stress events: a) infant stress events (birth to three years), b) childhood stress events (three to seven years), and c) those individuals who experienced stress during both infancy and childhood. The proportional indices of these groups were compared using ANOVAs to discover whether or not the age at which a stress event occurred had any effect on adult proportions.

Due to unequal error variances in male crural indices, as determined by Levine's test of homoscedasticity, a Kruskal-Wallis test was performed on this sample to evaluate differences in crural indices between groups based on the age of the stress event. The test was insignificant, $\chi 2$

(2, 65) = 1.37, $p = 0.51$. For all other indices, ANOVAs yield no significant results for comparisons of samples based on age of stress events (Table 7-14).

A second set of ANOVAs were performed comparing individuals with infant stress markers and those without any dental stress (Table 7-15). This investigation did not find any significant results, but for the female radiohumeral index, a near significant score was obtained ($p = 0.097$). Resampling statistics were performed as an independent test for ANOVAs with near significant results and in this case the results were found to be significant ($p = 0.0447$). For the male crural index, which did not pass the test for homogeneity of variance, the result of the Mann-Whitney *U* test is near significant, = -1.87, $p = 0.062$. Resampling statistics were used as an independent test of the near significant results of the Mann-Whitney *U* test. The difference was determined to be insignificant ($p = 0.9885$); however, the resampling procedure was directional and tests the alternative hypothesis that the unstressed values are greater than the stressed values. In this case, $1 - p < 0.05$, which means there is a significant difference between groups, but the difference is not in the direction proposed. The male crural index is significantly higher in those with infant stress than in the unstressed group.

Using Byers technique for aging Harris lines, individuals in this study were divided into three broad groups based on age of occurrence of stress events: a) infancy (birth to three years), b) childhood (three to seven years), and c) those individuals who experienced stress during both infancy and childhood. No individual had visible Harris lines that formed during the juvenile or adolescent period. The stature estimates for each of these groups were compared using ANOVAs to discover whether or not the age at which a stress event occurred had any effect on proportions. The ANOVAs yielded no significant or meaningful results given that the vast

majority of Harris lines in this sample occurred during infancy. Only two females and four males exhibited Harris lines during childhood (Table 7-16).

Table 7-1. Pearson correlations of the components of ratios (N = 204)

	Radius	Humerus	Femur (max)	Femur (bic)	Tibia	STH	SKH
Radius	1.000	0.915 [a]	0.868 [a]	0.873 [a]	0.916 [a]	0.683 [a]	0.909 [a]
Humerus	0.915 [a]	1.000	0.906 [a]	0.907 [a]	0.909 [a]	0.694 [a]	0.923 [a]
Femur (max)	0.868 [a]	0.906 [a]	1.000	0.997 [a]	0.928 [a]	0.651 [a]	0.949 [a]
Femur (bic)	0.873 [a]	0.907 [a]	0.997 [a]	1.000	0.929 [a]	0.649 [a]	0.953 [a]
Tibia	0.916 [a]	0.909 [a]	0.928 [a]	0.929 [a]	1.000	0.667 [a]	0.946 [a]
STH	0.683 [a]	0.694 [a]	0.651 [a]	0.649 [a]	0.667 [a]	1.000	0.783 [a]
SKH	0.909 [a]	0.923 [a]	0.949 [a]	0.953 [a]	0.946 [a]	0.783 [a]	1.000

[a] Correlation is significant at the 0.01 level (2-tailed).

Table 7-2. ANOVA – Proportional indices by dental enamel defects (CBTS)

	N	Mean	S.D.	Levine's	F	*p*
Male						
Crural index						
Unstressed	42	83.37	1.852			
Stressed	65	84.31	2.405	0.048[a, b]	.	.
Humerofemoral index						
Unstressed	42	70.69	2.015			
Stressed	65	71.13	2.136	0.574	1.156	0.285
Radiohumeral index						
Unstressed	42	78.39	2.333			
Stressed	65	78.36	2.429	0.812	0.003	0.958
Sitting height index						
Unstressed	42	30.03	1.127			
Stressed	65	30.08	1.110	0.716	0.053	0.818
Intermembral index						
Unstressed	42	68.76	1.869			
Stressed	65	68.82	1.596	0.670	0.037	0.847
Female						
Crural index						
Unstressed	43	83.00	2.288			
Stressed	54	82.70	2.745	0.378	0.349	0.556
Humerofemoral index						
Unstressed	43	70.60	2.114			
Stressed	54	70.58	2.166	0.731	0.003	0.953
Radiohumeral index						
Unstressed	43	76.30	2.076			
Stressed	54	75.87	2.228	0.937	0.953	0.331
Sitting height index						
Unstressed	43	30.50	1.247			
Stressed	54	30.35	1.092	0.285	0.391	0.533
Intermembral index						
Unstressed	43	68.01	1.652			
Stressed	54	67.93	1.675	0.886	0.050	0.823

[a] Significant ($p < 0.05$)

[b] The F-test cannot be performed if conditions of homoscedasticity are not met.

Table 7-3. ANOVA –Proportions by dental enamel defects (TS)

		N	Mean	S.D.	Levine's	F	*p*
Male							
	Crural index						
	Unstressed	37	83.42	1.896			
	Stressed	70	84.21	2.373	0.116	3.067	0.083 [a]
	Humerofemoral index						
	Unstressed	37	70.84	2.090			
	Stressed	70	71.02	2.104	0.741	0.165	0.685
	Radiohumeral index						
	Unstressed	37	78.37	2.401			
	Stressed	70	78.38	2.388	0.969	0.001	0.981
	Sitting height index						
	Unstressed	37	30.04	1.053			
	Stressed	70	30.06	1.149	0.618	0.008	0.929
	Intermembral index						
	Unstressed	37	68.88	1.943			
	Stressed	70	68.75	1.569	0.418	0.141	0.708
Female							
	Crural index						
	Unstressed	36	83.23	2.337			
	Stressed	61	82.60	2.650	0.631	1.378	0.243
	Humerofemoral index						
	Unstressed	36	70.84	2.046			
	Stressed	61	70.44	2.184	0.712	0.794	0.375
	Radiohumeral index						
	Unstressed	36	76.38	2.055			
	Stressed	61	75.87	2.218	0.628	1.259	0.265
	Sitting height index						
	Unstressed	36	30.54	1.328			
	Stressed	61	30.34	1.052	0.078 [a]	0.659	0.419
	Intermembral index						
	Unstressed	36	68.19	1.625			
	Stressed	61	67.83	1.674	0.838	1.016	0.316

[a] Near significant ($0.05 < p < 0.10$)

Table 7-4. ANOVA – Proportional indices by dental enamel defects (LEH) in females

	N	Mean	S.D.	Levine's	F	*p*
Crural index						
Unstressed	36	83.23	2.337			
Stressed	60	82.58	2.667	0.612	1.461	0.230
Humerofemoral index						
Unstressed	36	70.84	2.046			
Stressed	60	70.45	2.200	0.675	0.730	0.395
Radiohumeral index						
Unstressed	36	76.38	2.055			
Stressed	60	75.95	2.152	0.742	0.938	0.335
Sitting height index						
Unstressed	36	30.54	1.328			
Stressed	60	30.33	1.059	0.085 [a]	0.709	0.402
Intermembral index						
Unstressed	36	68.19	1.625			
Stressed	60	67.88	1.641	0.943	0.760	0.386

[a] Near significant ($0.05 < p < 0.10$)

Table 7-5. ANOVA – Proportional indices by the presence or absence of Harris lines (HL)

		N	Mean	S.D.	Levine's	F	*p*
Male							
	Crural index						
	Absent	56	83.72	2.159			
	Present	40	84.13	2.297	0.956	0.797	0.374
	Humerofemoral index						
	Absent	56	71.11	2.224			
	Present	40	70.77	1.941	0.455	0.631	0.429
	Radiohumeral index						
	Absent	56	78.23	2.54			
	Present	40	78.41	2.282	0.592	0.124	0.725
	Sitting height index						
	Absent	56	30.07	1.013			
	Present	40	30.02	1.289	0.153	0.046	0.830
	Intermembral index						
	Absent	56	68.98	1.832			
	Present	40	68.56	1.488	0.306	1.423	0.236
Female							
	Crural index						
	Absent	56	82.67	2.534			
	Present	31	83.24	2.734	0.446	0.944	0.334
	Humerofemoral index						
	Absent	56	70.56	2.139			
	Present	31	70.78	2.283	0.597	0.197	0.658
	Radiohumeral index						
	Absent	56	76.22	2.194			
	Present	31	76.04	2.187	1.000	0.145	0.704
	Sitting height index						
	Absent	56	30.41	1.097			
	Present	31	30.50	1.141	0.722	0.132	0.718
	Intermembral index						
	Absent	56	68.06	1.64			
	Present	31	67.99	1.757	0.849	0.038	0.845

Table 7-6. Interaction effects of Harris lines (HL) and enamel defects (CBTS) on proportions

		F	*p*
Male			
	Crural Index		
	HL	0.396	0.531
	CBTS	5.030	0.027 [a]
	HL * CBTS	1.118	0.293
	Humero-femoral Index		
	HL	0.901	0.345
	CBTS	1.556	0.215
	HL * CBTS	0.505	0.479
	Radio-humeral Index		
	HL	0.067	0.796
	CBTS	0.074	0.786
	HL * CBTS	0.411	0.523
	Sitting Height Index		
	HL	0.141	0.709
	CBTS	0.019	0.890
	HL * CBTS	1.054	0.307
	Intermembral Index		
	HL	1.668	0.200
	CBTS	0.066	0.797
	HL * CBTS	0.540	0.464
Female			
	Crural Index		
	HL	1.258	0.265
	CBTS	0618	0.434
	HL * CBTS	0.565	0.454
	Humero-femoral Index		
	HL	0.261	0.611
	CBTS	0.064	0.801
	HL * CBTS	0.179	0.673
	Radio-humeral Index		
	HL	0.086	0.771
	CBTS	1.111	0.295
	HL * CBTS	0.023	0.880
	Sitting Height Index		
	HL	0.252	0.617
	CBTS	1.030	0.313
	HL * CBTS	0.154	0.696

Table 7-6. Continued

	F	*p*
Intermembral Index		
HL	0.021	0.884
CBTS	0.160	0.690
HL * CBTS	0.003	0.959

[a] Significant ($p < 0.05$)

Table 7-7. Scaling proportional indices to size (SKH)

	Slope	R^2
Crural index		
Male	0.089	0.069
Female	0.028 [a]	0.005
Humerofemoral index		
Male	-0.042 [a]	0.017
Female	-0.126	0.143
Radiohumeral index		
Male	-0.048 [a]	0.018
Female	0.081	0.058
Intermembral index		
Male	-0.099	0.085
Female	-0.101	0.150

[a] 95% C.I. includes zero

Table 7-8. RMA regressions of log-transformed components of ratios divided by dental enamel defects (CBTS)

	N	RMA Intercept	RMA Slope	95% CI Lower	95% CI Upper	R^2	Scaling [a]
Crural index							
Male (combined)	107	-0.309	1.139	1.045	1.233	0.819	Positive
Unstressed	42	-0.274	1.116	0.989	1.243	0.873	Isometric
Stressed	65	-0.337	1.157	1.025	1.288	0.798	Positive
Female (combined)	97	-0.200	1.072	0.964	1.179	0.758	Isometric
Unstressed	43	-0.143	1.038	0.892	1.184	0.801	Isometric
Stressed	54	-0.236	1.093	0.929	1.257	0.710	Isometric
Humerofemoral index							
Male (combined)	107	-0.145	0.997	0.898	1.097	0.734	Isometric
Unstressed	42	0.081	0.862		1.001	0.747	Isometric
Stressed	65	-0.285	1.081	0.943	1.219	0.744	Isometric
Female (combined)	97	0.104	0.845	0.754	0.935	0.725	Negative
Unstressed	43	0.158	0.813	0.682	0.943	0.742	Negative
Stressed	54	0.078	0.861	0.728	0.993	0.695	Negative
Radiohumeral index							
Male (combined)	107	-0.053	0.965	0.864	1.066	0.706	Isometric
Unstressed	42	-0.231	1.082	0.886	1.277	0.681	Isometric
Stressed	65	0.030	0.911	0.791	1.031	0.727	Isometric
Female (combined)	97	-0.396	1.185	1.066	1.304	0.759	Positive
Unstressed	43	-0.246	1.086	0.907	1.264	0.729	Isometric
Stressed	54	-0.492	1.249	1.081	1.417	0.767	Positive
Sitting height index							
Male (combined)	107	-0.658	1.061	0.902	1.221	0.398	Isometric
Unstressed	42	-0.738	1.098	0.834	1.361	0.435	Isometric
Stressed	65	-0.597	1.034	0.827	1.241	0.371	Isometric
Female (combined)	97	-0.977	1.212	1.035	1.388	0.489	Positive
Unstressed	43	-0.794	1.128	0.856	1.400	0.416	Isometric
Stressed	54	-1.125	1.280	1.036	1.524	0.532	Positive
Intermembral index							
Male (combined)	107	0.016	0.908	0.832	0.984	0.815	Negative
Unstressed	42	0.180	0.824	0.704	0.944	0.792	Negative
Stressed	65	-0.084	0.960	0.862	1.058	0.835	Isometric
Female (combined)	97	0.054	0.884	0.808	0.960	0.822	Negative
Unstressed	43	0.177	0.820	0.715	0.925	0.835	Negative
Stressed	54	-0.021	0.923	0.809	1.036	0.806	Isometric

[a] Variables are considered to be isometric when the confidence interval includes a slope of 1.0.

Table 7-9. Comparison of RMA regressions (RMA2) divided by dental enamel defects (CBTS)

	N	Intercept	Slope	95% CI lower	upper	Scale	t	df	*p*	Intercept Diffs.
Male										
Crural index										
Unstressed	42	-0.274	1.116	0.996	1.250	Iso.				
Stressed	65	-0.336	1.156	1.032	1.295	Pos.	0.454	53	0.326	0.326
Humerofemoral index										
Unstressed	42	0.079	0.864	0.736	1.014	Iso.				
Stressed	65	-0.283	1.080	0.950	1.260	Iso.	2.241	49	0.015 [a]	.
Radiohumeral index										
Unstressed	42	-0.231	1.081	0.904	1.294	Iso.				
Stressed	65	0.032	0.910	0.798	1.038	Iso.	1.587	48	0.060 [b]	0.324
Sitting height index										
Unstressed	42	-0.734	1.096	0.864	1.390	Iso.				
Stressed	65	-0.597	1.034	0.848	1.261	Iso.	0.381	51	0.352	0.843
Intermembral index										
Unstressed	42	0.186	0.821	0.710	0.949	Neg.				
Stressed	65	-0.084	0.960	0.866	1.063	Iso.	1.806	47	0.039 [a]	.
Female										
Crural index										
Unstressed	43	-0.146	1.040	0.903	1.196	Iso.				
Stressed	54	-0.231	1.091	0.939	1.266	Iso.	0.481	50	0.317	0.543
Humerofemoral index										
Unstressed	43	0.154	0.815	0.695	0.956	Neg.				
Stressed	54	0.078	0.860	0.738	1.002	Iso.	0.499	50	0.310	0.837
Radiohumeral index										
Unstressed	43	-0.241	1.082	0.919	1.274	Iso.				
Stressed	54	-0.490	1.249	1.092	1.428	Pos.	1.391	48	0.085 [b]	1.000
Sitting height index										
Unstressed	43	-0.784	1.123	0.885	1.426	Iso.				
Stressed	54	-1.127	1.281	1.060	1.548	Pos.	0.881	48	0.191	1.000
Intermembral index										
Unstressed	43	0.182	0.818	0.720	0.929	Neg.				
Stressed	54	-0.019	0.922	0.816	1.042	Iso.	1.395	50	0.085 [b]	1.000

[a] Significant ($p < 0.05$)

[b] Near significant ($0.05 < p < 0.10$)

Table 7-10. RMA regressions of log-transformed components of ratios divided by the presence or absence of Harris lines

	N	RMA Intercept	RMA Slope	95% CI Lower	Upper	R^2	Scaling [a]
Crural index							
Male (combined)	96	-0.340	1.157	1.021	1.293	0.812	Positive
Absent	56	-0.286	1.125	1.001	1.250	0.836	Positive
Present	40	-0.423	1.207	1.020	1.394	0.778	Positive
Female (combined)	87	-0.134	1.031	0.915	1.148	0.724	Isometric
Absent	56	-0.177	1.057	0.912	1.202	0.749	Isometric
Present	31	-0.031	0.970	0.762	1.178	0.681	Isometric
Humerofemoral index							
Male (combined)	96	-0.139	0.994	0.890	1.098	0.738	Isometric
Absent	56	-0.139	0.995	0.853	1.136	0.730	Isometric
Present	40	-0.142	0.995	0.833	1.158	0.754	Isometric
Female (combined)	87	0.117	0.837	0.742	0.932	0.724	Negative
Absent	56	0.009	0.902	0.776	1.028	0.738	Isometric
Present	31	0.364	0.688	0.552	0.824	0.728	Negative
Radiohumeral index							
Male (combined)	96	-0.077	0.981	0.864	1.098	0.661	Isometric
Absent	56	-0.037	0.955	0.794	1.115	0.620	Isometric
Present	40	-0.140	1.022	0.847	1.198	0.727	Isometric
Female (combined)	87	-0.396	1.186	1.055	1.318	0.736	Positive
Absent	56	-0.347	1.154	1.008	1.300	0.787	Positive
Present	31	-0.564	1.298	0.988	1.608	0.603	Isometric
Sitting height index							
Male (combined)	96	-0.566	1.020	0.853	1.186	0.365	Isometric
Absent	56	-0.382	0.936	0.744	1.129	0.437	Isometric
Present	40	-0.811	1.130	0.818	1.443	0.294	Isometric
Female (combined)	87	-0.984	1.215	1.034	1.396	0.526	Positive
Absent	56	-1.050	1.245	1.024	1.466	0.578	Positive
Present	31	-0.853	1.155	0.825	1.485	0.433	Isometric
Intermembral index							
Male (combined)	96	0.037	0.897	0.818	0.976	0.815	Negative
Absent	56	0.045	0.894	0.785	1.002	0.802	Isometric
Present	40	0.017	0.906	0.787	1.025	0.841	Isometric
Female (combined)	87	0.058	0.882	0.794	0.970	0.785	Negative
Absent	56	-0.043	0.935	0.818	1.052	0.791	Isometric
Present	31	0.268	0.772	0.636	0.908	0.784	Negative

[a] Variables are considered to be isometric when the confidence interval includes a slope of 1.0.

Table 7-11. Comparison of RMA regressions (RMA2) divided by Harris lines

	N	Intercept	Slope	95% C.I. Lower	95% C.I. Upper	Scale	t	d.f.	p	Intercept Diffs.
Male										
Crural index										
Absent	56	-0.269	1.114	0.998	1.244	Iso				
Present	40	-0.376	1.179	1.011	1.374	Pos	0.611	44.213	0.272	0.214
Humerofemoral index										
Absent	56	-0.095	0.969	0.841	1.116	Iso				
Present	40	-0.172	1.013	0.862	1.190	Iso	0.426	47.140	0.336	0.301
Radiohumeral index										
Absent	56	-0.026	0.947	0.801	1.120	Iso				
Present	40	-0.132	1.017	0.858	1.205	Iso	0.608	48.656	0.273	0.308
Sitting height index										
Absent	56	-0.383	0.937	0.764	1.148	Iso				
Present	40	-0.917	1.179	0.899	1.546	Iso	1.380	46.127	0.087 [b]	0.682
Intermembral index										
Absent	56	0.076	0.878	0.767	1.004	Iso				
Present	40	-0.031	0.931	0.818	1.061	Iso	0.648	49.192	0.260	0.210
Female										
Crural index										
Absent	56	-0.192	1.066	0.930	1.222	Iso				
Present	31	-0.086	1.004	0.811	1.242	Iso	0.496	35.239	0.311	0.507
Humerofemoral index										
Absent	56	0.015	0.898	0.782	1.033	Iso				
Present	31	0.369	0.685	0.563	0.834	Neg	2.337	36.615	0.013 [a]	0.500
Radiohumeral index										
Absent	56	-0.339	1.148	1.012	1.302	Pos				
Present	31	-0.541	1.282	1.011	1.624	Pos	0.856	33.114	0.199	1.000
Sitting height index										
Absent	56	-0.976	1.211	1.015	1.445	Pos				
Present	31	-0.894	1.174	0.885	1.557	Iso	0.195	35.917	0.423	0.180
Intermembral index										
Absent	56	-0.040	0.933	0.824	1.057	Iso				
Present	31	0.279	0.766	0.643	0.914	Neg	1.902	36.506	0.033 [a]	.

[a] significant ($p < 0.05$).

[b] near significant ($0.05 < p < 0.10$)

Table 7-12. ANOVA – Proportional indices by minimum number of stress events (MNSE)

	N	Mean	S.D.	Levine's	F	*p*
Male						
Crural index						
0	42	83.37	1.852			
1	19	84.51	1.650			
2	27	83.78	2.241			
3	15	84.93	3.244			
4	3	85.21	3.452			
5	1	82.59	.	0.001 [a, b]	.	.
Humerofemoral index						
0	42	70.69	2.015			
1	19	71.11	1.766			
2	27	70.66	2.331			
3	15	72.24	2.174			
4	3	70.30	0.508			
5	1	69.96	.	0.385	1.551	0.181
Radiohumeral index						
0	42	78.39	2.333			
1	19	78.63	2.0151			
2	27	78.36	2.925			
3	15	77.86	2.183			
4	3	78.38	1.460			
5	1	80.82	.	0.256	0.384	0.859
Sitting height index						
0	42	30.03	1.127			
1	19	30.19	1.328			
2	27	29.96	1.097			
3	15	30.06	1.002			
4	3	30.58	0.417			
5	1	29.70	.	0.331	0.242	0.943
Intermembral index						
0	42	68.76	1.869			
1	19	68.84	1.435			
2	27	68.56	1.719			
3	15	69.47	1.532			
4	3	67.72	1.439			
5	1	69.28	.	0.767	0.836	0.527
Female						
Crural index						
0	43	83.00	2.288			
1	26	82.44	3.123			
2	18	82.73	2.089			
3	9	83.22	3.065			
4	1	84.04	.	0.266	0.313	0.869

Table 7-12. Continued

	N	Mean	S.D.	Levine's	F	*p*
Humerofemoral index						
0	43	70.60	2.114			
1	26	70.30	2.262			
2	18	70.54	1.894			
3	9	70.98	2.229			
4	1	74.67	.	0.611	1.111	0.356
Radiohumeral Index						
0	43	76.30	2.076			
1	26	75.93	2.566			
2	18	75.70	1.973			
3	9	75.85	1.891			
4	1	77.65	.	0.371	0.431	0.786
Sitting height index						
0	43	30.50	1.247			
1	26	30.28	1.198			
2	18	30.28	1.001			
3	9	30.45	0.887			
4	1	32.28	.	0.525	0.837	0.505
Intermembral index						
0	43	68.01	1.652			
1	26	67.78	1.683			
2	18	67.82	1.490			
3	9	68.11	1.652			
4	1	72.07	.	0.694	1.725	0.151

[a] significant ($p < 0.05$).

[b] The assumptions of the F-test are violated where conditions of homoscedasticity are not met.

Table 7-13. ANOVA – Proportional indices by number of Harris lines

	N	Mean	S.D.	Levine's	F	*p*
Male						
Crural index						
no Harris lines	56	83.72	2.159			
one Harris line	30	84.05	2.588			
two or more Harris lines	10	84.39	1.095	0.031[a, c]	.	.
Humerofemoral index						
no Harris lines	56	71.11	2.224			
one Harris line	30	70.47	1.953			
two or more Harris lines	10	71.65	1.692	0.614	1.519	0.224
Radiohumeral index						
no Harris lines	56	78.23	2.540			
one Harris line	30	78.31	2.379			
two or more Harris lines	10	78.70	2.048	0.772	0.155	0.857
Sitting height index						
no Harris lines	56	30.07	1.013			
one Harris line	30	29.90	1.161			
two or more Harris lines	10	30.36	1.636	0.121	0.641	0.529
Intermembral index						
no Harris lines	56	68.98	1.832			
one Harris line	30	68.26	1.385			
two or more Harris lines	10	69.44	1.508	0.261	2.578	0.081[b]

[a] significant ($p < 0.05$)

[b] near significant ($0.05 < p < 0.10$)

[c] The assumptions of the F-test are violated where conditions of homoscedasticity are not met.

Table 7-14. ANOVA – Proportional indices by age of enamel defect

	N	Mean	S.D.	Levine's	F	*p*
Male						
Crural index						
No stress	42	83.37	1.852			
Infant stress	10	84.93	1.377			
Childhood stress	18	83.93	1.980			
Infant & childhood stress	37	84.32	2.789	0.000 [a, b]	.	.
Humerofemoral index						
No stress	42	70.69	2.015			
Infant stress	10	71.20	1.087			
Childhood stress	18	71.05	1.925			
Infant & childhood stress	37	71.15	2.462	0.113	0.392	0.759
Radiohumeral index						
No stress	42	78.39	2.333			
Infant stress	10	78.91	1.972			
Childhood stress	18	78.63	2.495			
Infant & childhood stress	37	78.09	2.526	0.966	0.414	0.743
Sitting height index						
No stress	42	30.03	1.127			
Infant stress	10	29.72	0.990			
Childhood stress	18	30.46	1.410			
Infant & childhood stress	37	29.99	0.944	0.289	1.162	0.328
Intermembral index						
No stress	42	68.76	1.869			
Infant stress	10	68.87	0.770			
Childhood stress	18	68.99	1.620			
Infant & childhood stress	37	68.73	1.764	0.163	0.111	0.953
Female						
Crural index						
No stress	43	83.00	2.288			
Infant stress	13	81.49	3.159			
Childhood stress	19	83.22	2.607			
Infant & childhood stress	22	82.95	2.504	0.605	1.45	0.233
Humerofemoral index						
No stress	43	70.60	2.114			
Infant stress	13	70.03	2.655			
Childhood stress	19	70.46	1.693			
Infant & childhood stress	22	71.00	2.230	0.398	0.588	0.624
Radiohumeral index						
No stress	43	76.30	2.076			
Infant stress	13	75.03	2.008			
Childhood stress	19	76.52	2.492			
Infant & childhood stress	22	75.81	2.021	0.791	1.58	0.200

Table 7-14. Continued

	N	Mean	S.D.	Levine's	F	*p*
Sitting height index						
No stress	43	30.50	1.247			
Infant stress	13	30.16	1.226			
Childhood stress	19	30.36	1.205			
Infant & childhood stress	22	30.45	0.935	0.586	0.296	0.828
Intermembral index						
No stress	43	68.01	1.652			
Infant stress	13	67.52	1.813			
Childhood stress	19	67.88	1.546			
Infant & childhood stress	22	68.22	1.720	0.936	0.503	0.681

[a] significant ($p < 0.05$)

[b] The assumptions of the F-test are violated where conditions of homoscedasticity are not met.

Table 7-15. ANOVA – Proportional indices by infant enamel defect

	N	Mean	S.D.	Levine's	F	*p*
Male						
Crural index						
No stress	42	83.37	1.852			
Infant stress	47	84.45	2.554	0.016 [a, c]	.	.
Humerofemoral index						
No stress	42	70.69	2.015			
Infant stress	47	71.16	2.230	0.522	1.108	0.296
Radiohumeral index						
No stress	42	78.39	2.333			
Infant stress	47	78.26	2.423	0.905	0.064	0.802
Sitting height index						
No stress	42	30.03	1.127			
Infant stress	47	29.93	0.950	0.232	0.187	0.666
Intermembral index						
No stress	42	68.76	1.869			
Infant stress	47	68.76	1.599	0.533	0.000	0.999
Female						
Crural index						
No stress	43	83.00	2.288			
Infant stress	35	82.41	2.812	0.396	1.053	0.308
Humerofemoral index						
No stress	43	70.60	2.114			
Infant stress	35	70.64	2.406	0.330	0.005	0.944
Radiohumeral index						
No stress	43	76.30	2.076			
Infant stress	35	75.52	2.022	0.735	2.816	0.097 [b]
Sitting height index						
No stress	43	30.50	1.247			
Infant stress	35	30.34	1.044	0.248	0.351	0.555
Intermembral index						
No stress	43	68.01	1.652			
Infant stress	35	67.96	1.762	0.711	0.017	0.896

[a] significant ($p < 0.05$)

[b] near significant ($0.05 < p < 0.10$)

[c] The assumptions of the F-test are violated where conditions of homoscedasticity are not met.

Table 7-16. ANOVA – Proportions by age of Harris line

	N	Mean	S.D.	Levine's	F	*p*
Male						
Crural index						
No stress	56	83.72	2.159			
Infant stress	36	84.15	2.412			
Childhood stress	1	83.39	.			
Infant & childhood stress	3	84.20	0.920	0.200	0.299	0.826
Humerofemoral index						
No stress	56	71.11	2.224			
Infant stress	36	70.55	1.880			
Childhood stress	1	73.51	.			
Infant & childhood stress	3	72.43	1.693	0.457	1.547	0.208
Radiohumeral index						
No stress	56	78.23	2.540			
Infant stress	36	78.60	2.285			
Childhood stress	1	75.63	.			
Infant & childhood stress	3	77.03	1.653	0.478	0.876	0.456
Sitting height index						
No stress	56	30.07	1.013			
Infant stress	36	29.94	1.236			
Childhood stress	1	31.31	.			
Infant & childhood stress	3	30.58	2.056	0.218	0.754	0.523
Intermembral index						
No stress	56	68.98	1.832			
Infant stress	36	68.42	1.466			
Childhood stress	1	70.40	.			
Infant & childhood stress	3	69.60	1.391	0.365	1.330	0.269
Female						
Crural index						
No stress	56	82.67	2.534			
Infant stress	29	83.26	2.821			
Childhood stress	2	82.91	1.123	0.332	0.483	0.618
Humerofemoral index						
No stress	56	70.56	2.139			
Infant stress	29	70.86	2.331			
Childhood stress	2	69.62	1.177	0.540	0.399	0.672
Radiohumeral index						
No stress	56	76.22	2.194			
Infant stress	29	75.97	2.213			
Childhood stress	2	76.93	2.171	0.963	0.247	0.782
Sitting height index						
No stress	56	30.41	1.097			
Infant stress	29	30.52	1.172			
Childhood stress	2	30.22	0.684	0.693	0.134	0.875

Table 7-16. Continued

	N	Mean	S.D.	Levine's	F	*p*
Intermembral index						
No stress	56	68.06	1.640			
Infant stress	29	68.03	1.805			
Childhood stress	2	67.33	0.724	0.435	0.179	0.836

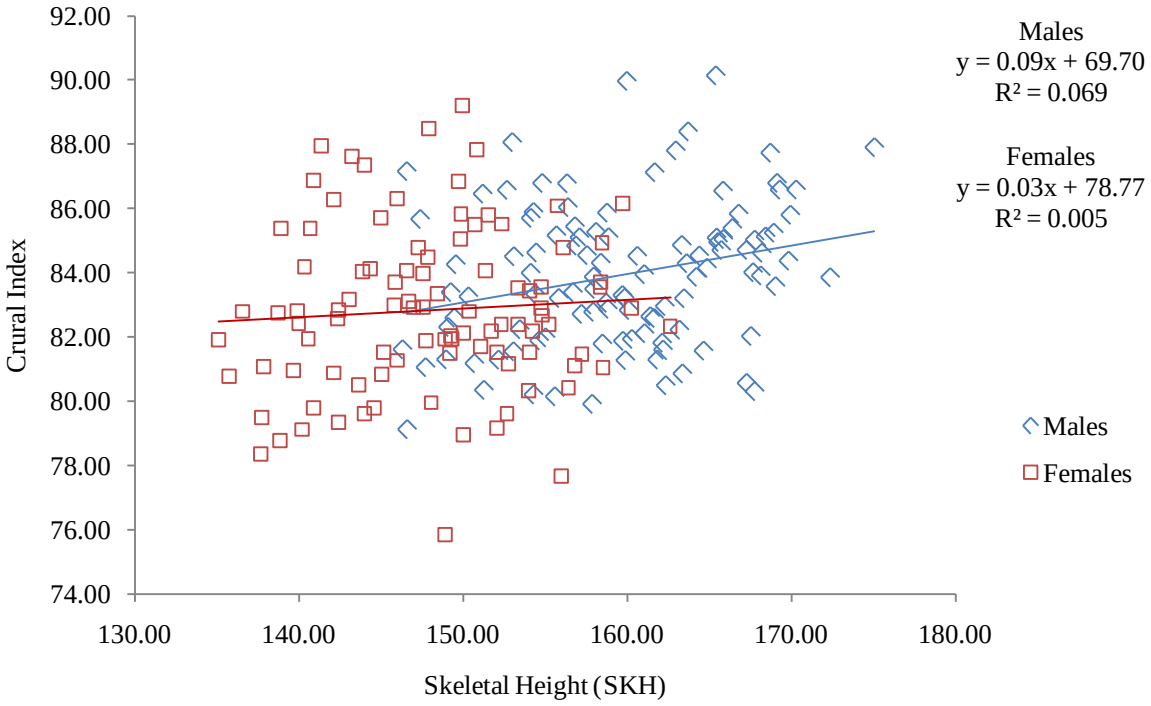


Figure 7-1. Scaling crural index to size

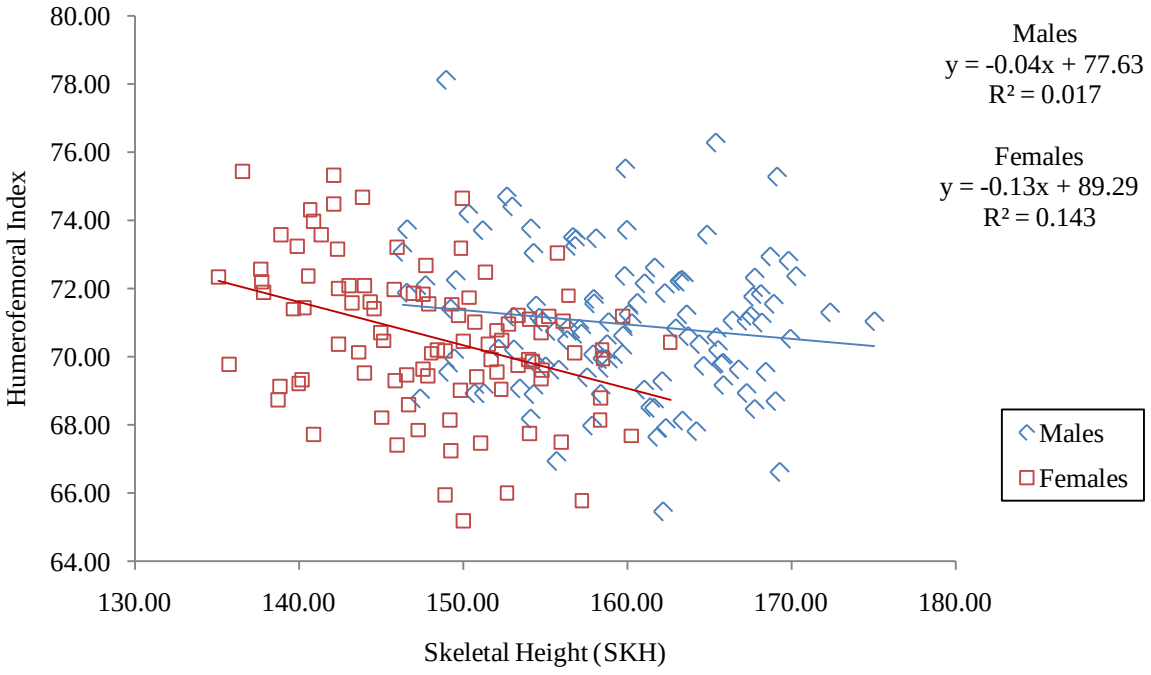


Figure 7-2. Scaling humerofemoral index to size

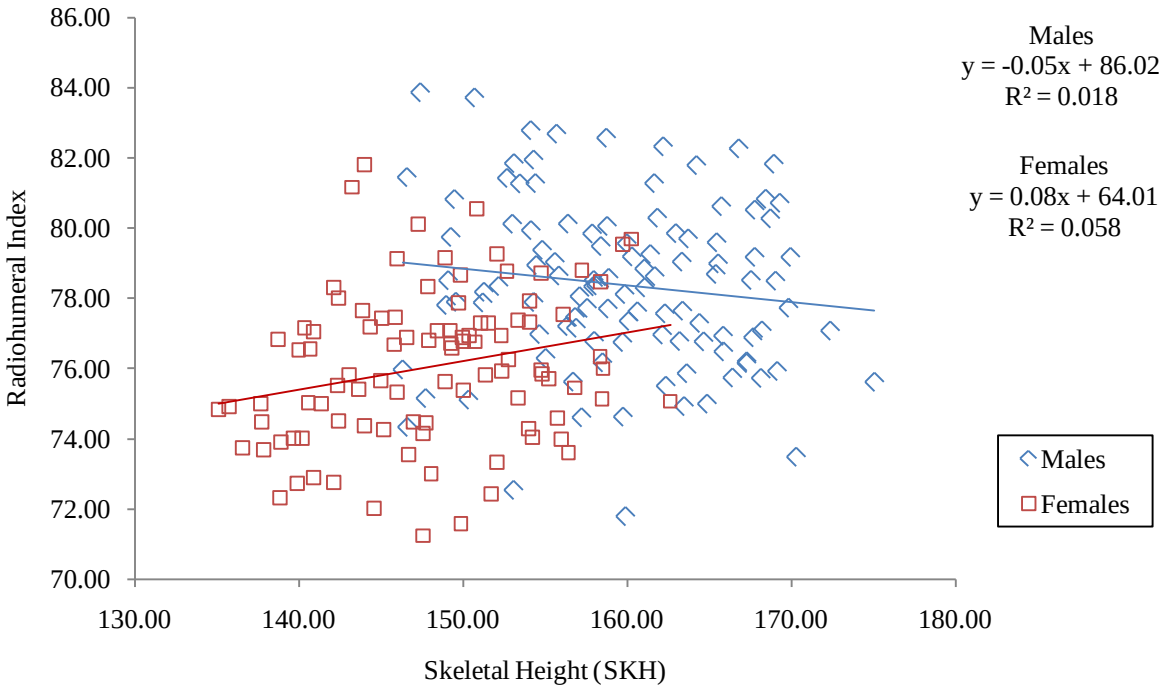


Figure 7-3. Scaling radiohumeral index to size

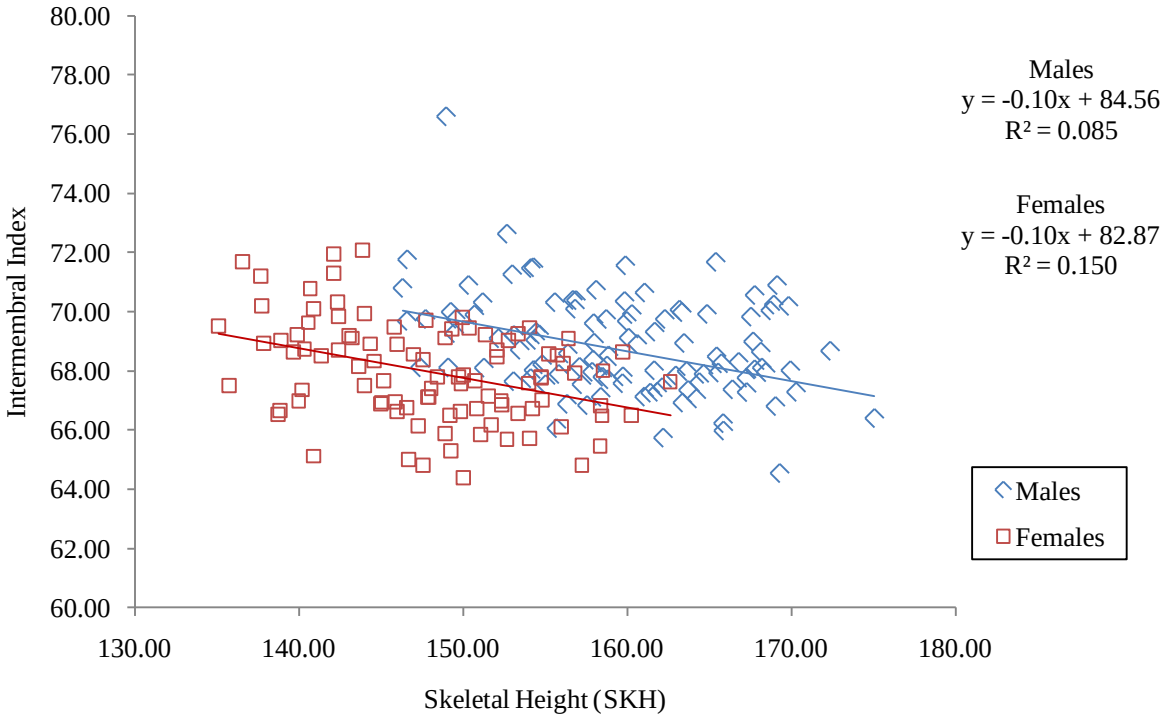


Figure 7-4. Scaling intermembral index to size

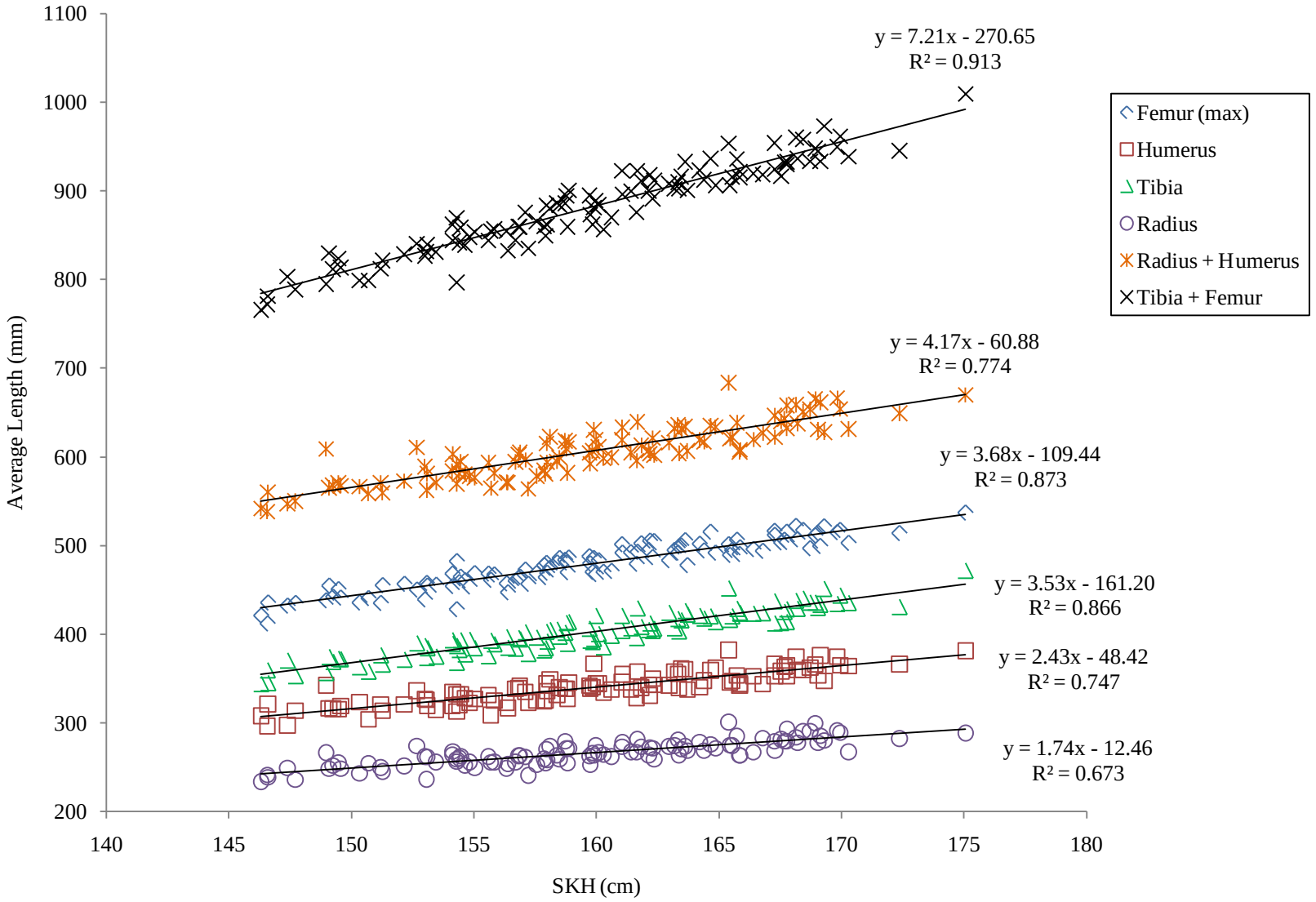


Figure 7-5. Components of proportional ratios and long bone lengths scaled to skeletal height (male)

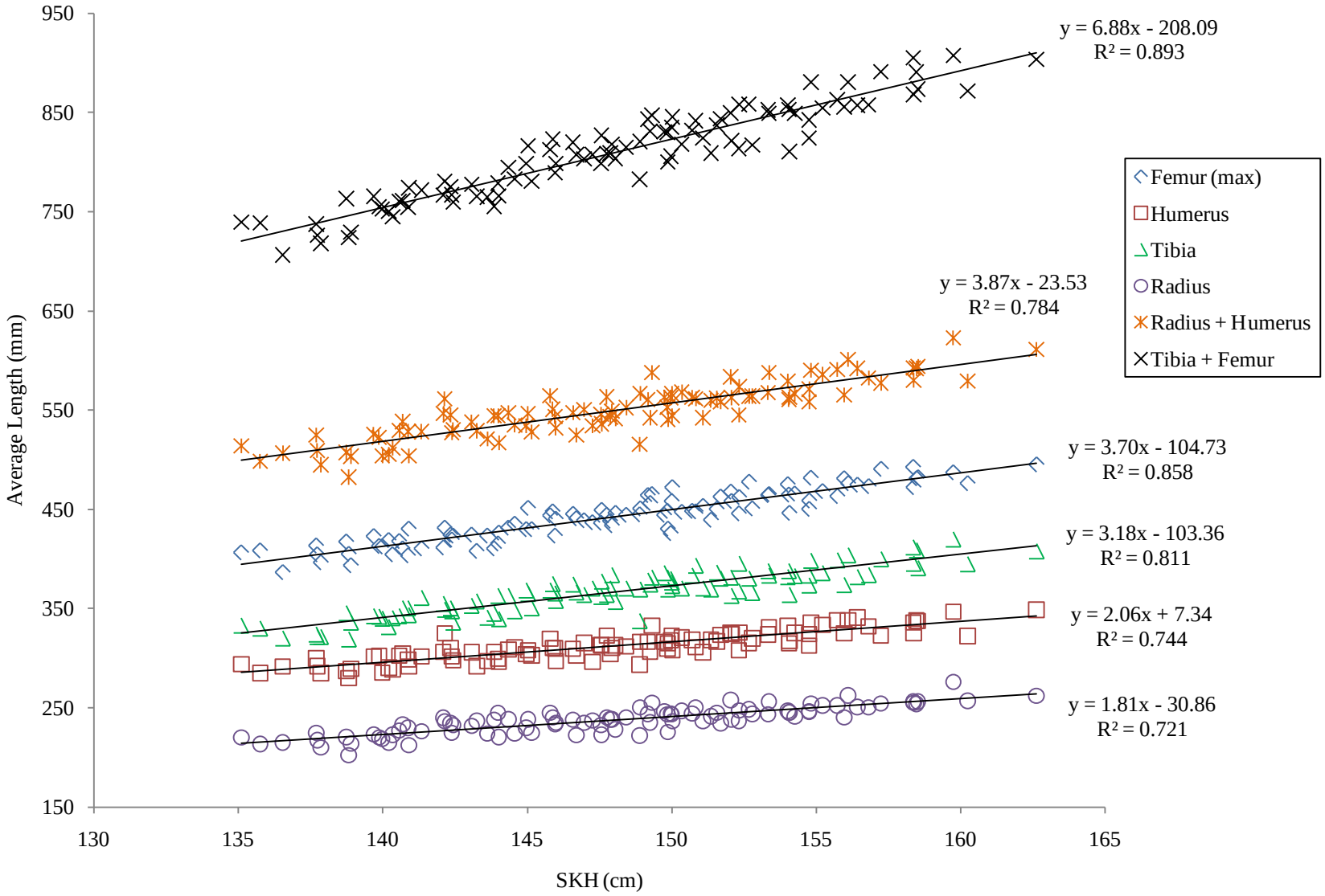


Figure 7-6. Components of proportional ratios and long bone lengths scaled to skeletal height (female)

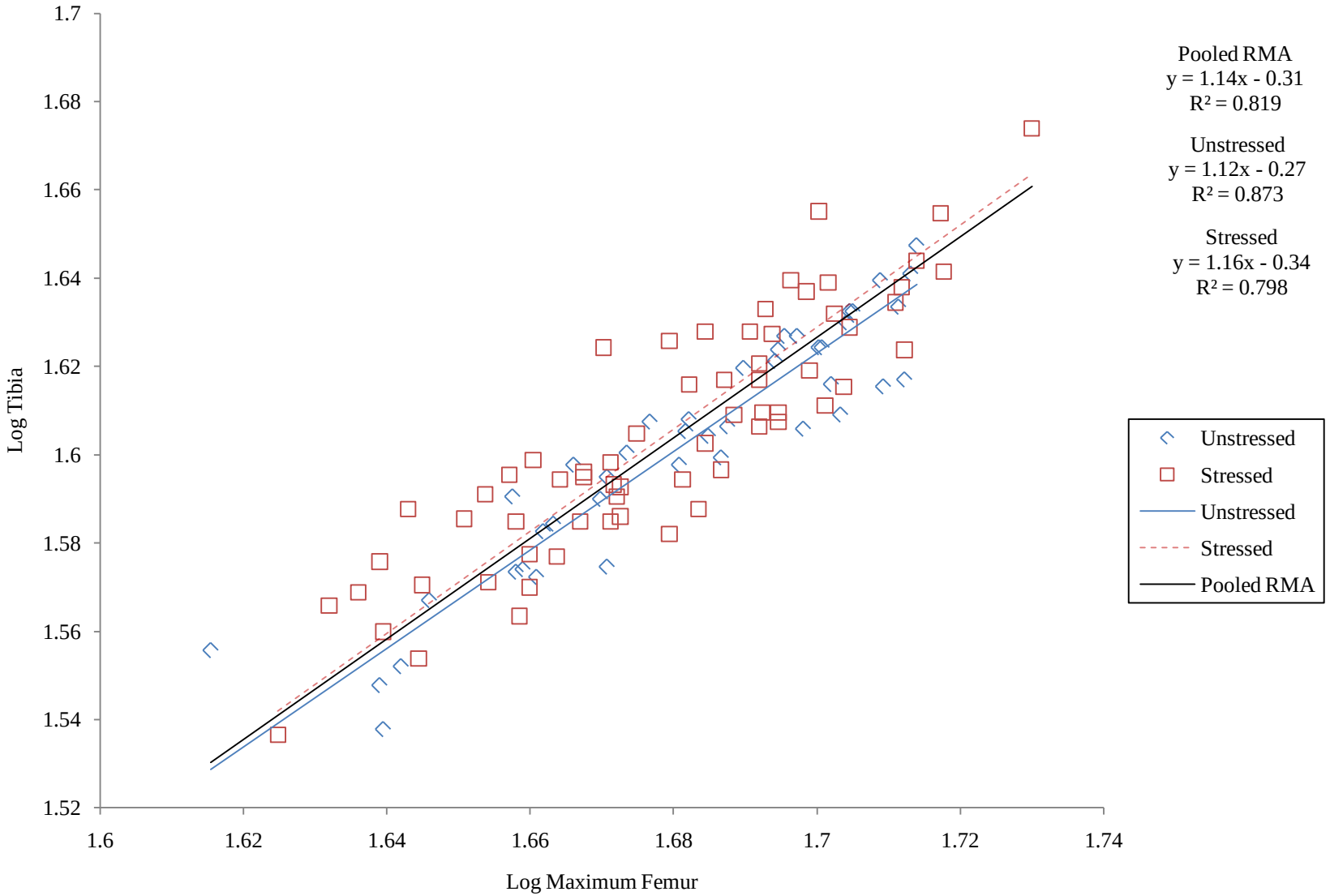


Figure 7-7. RMA for crural indices divided by dental enamel defects (male)

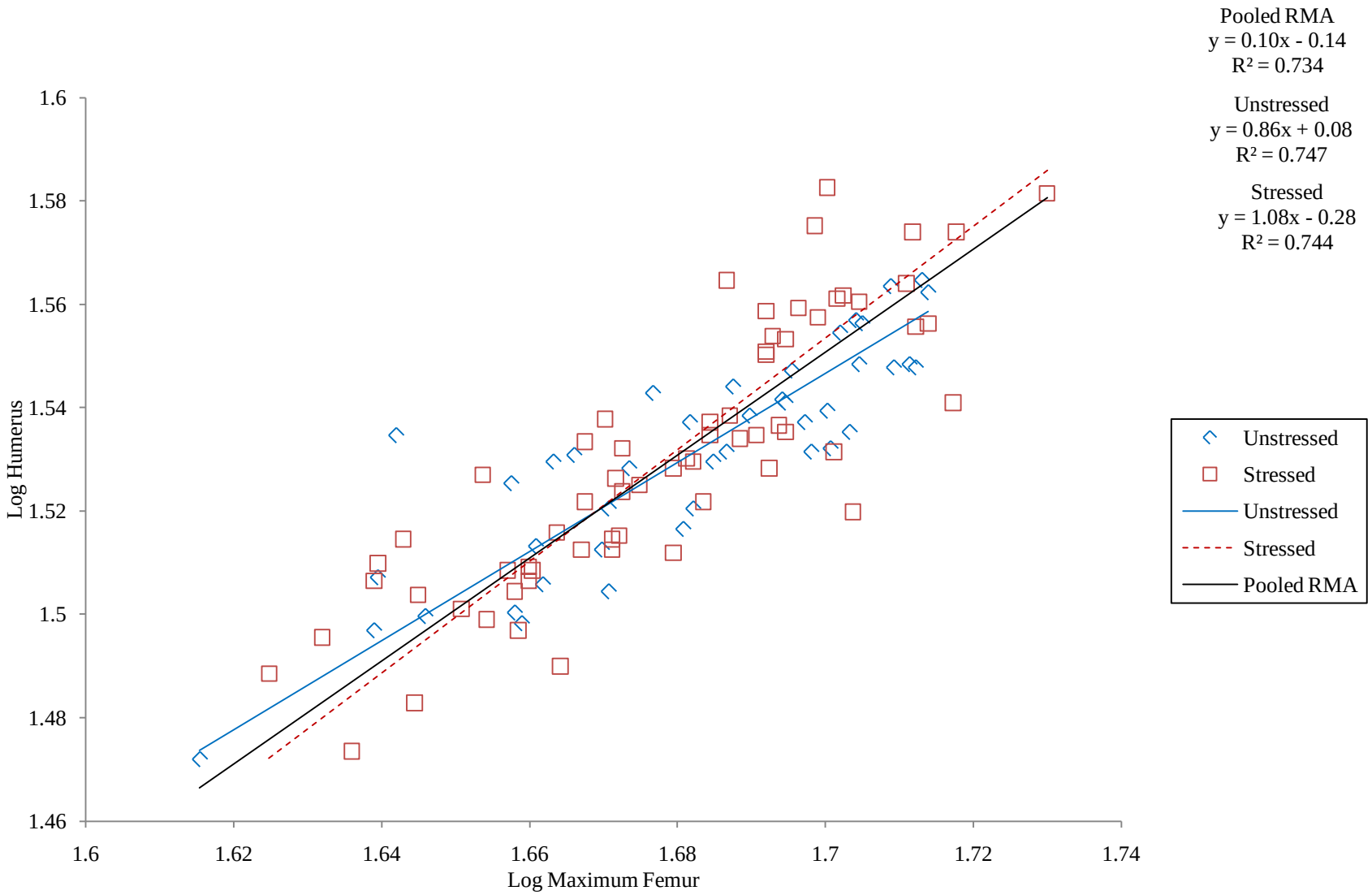


Figure 7-8. RMA for humerofemoral indices divided by dental enamel defects (male)

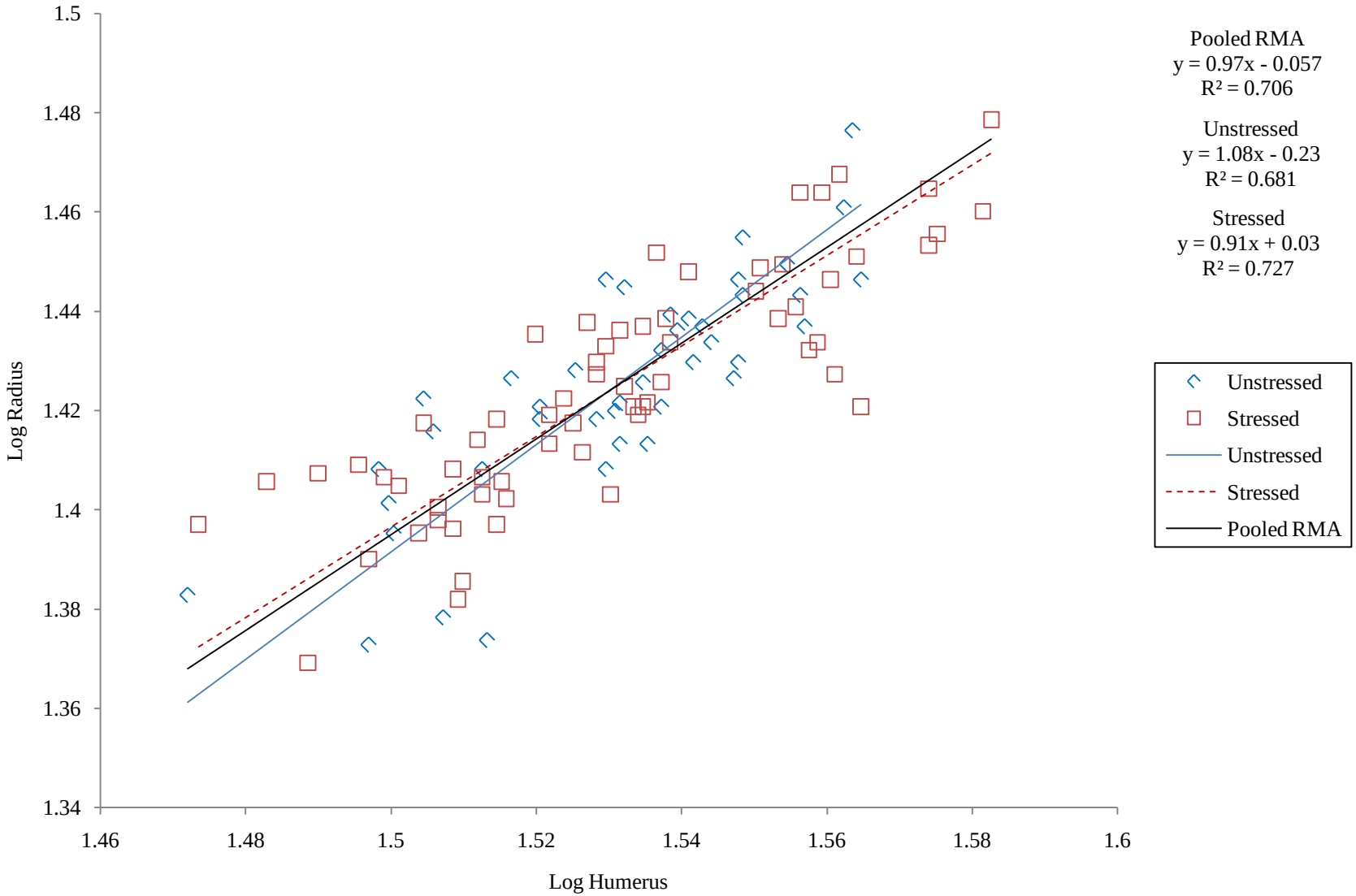


Figure 7-9. RMA for radiohumeral indices divided by dental enamel defects (male)

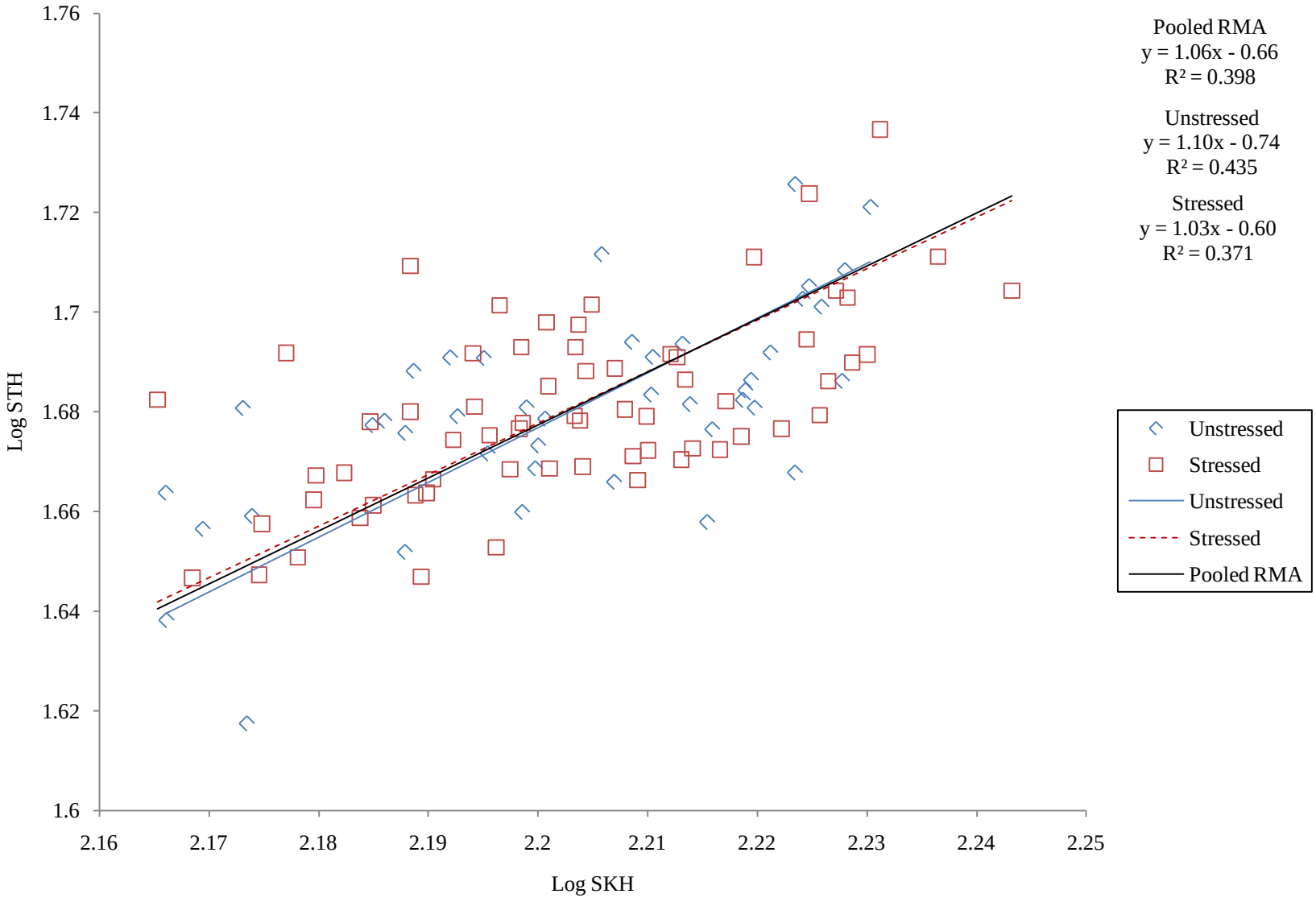


Figure 7-10. RMA for sitting height indices divided by dental enamel defects (male)

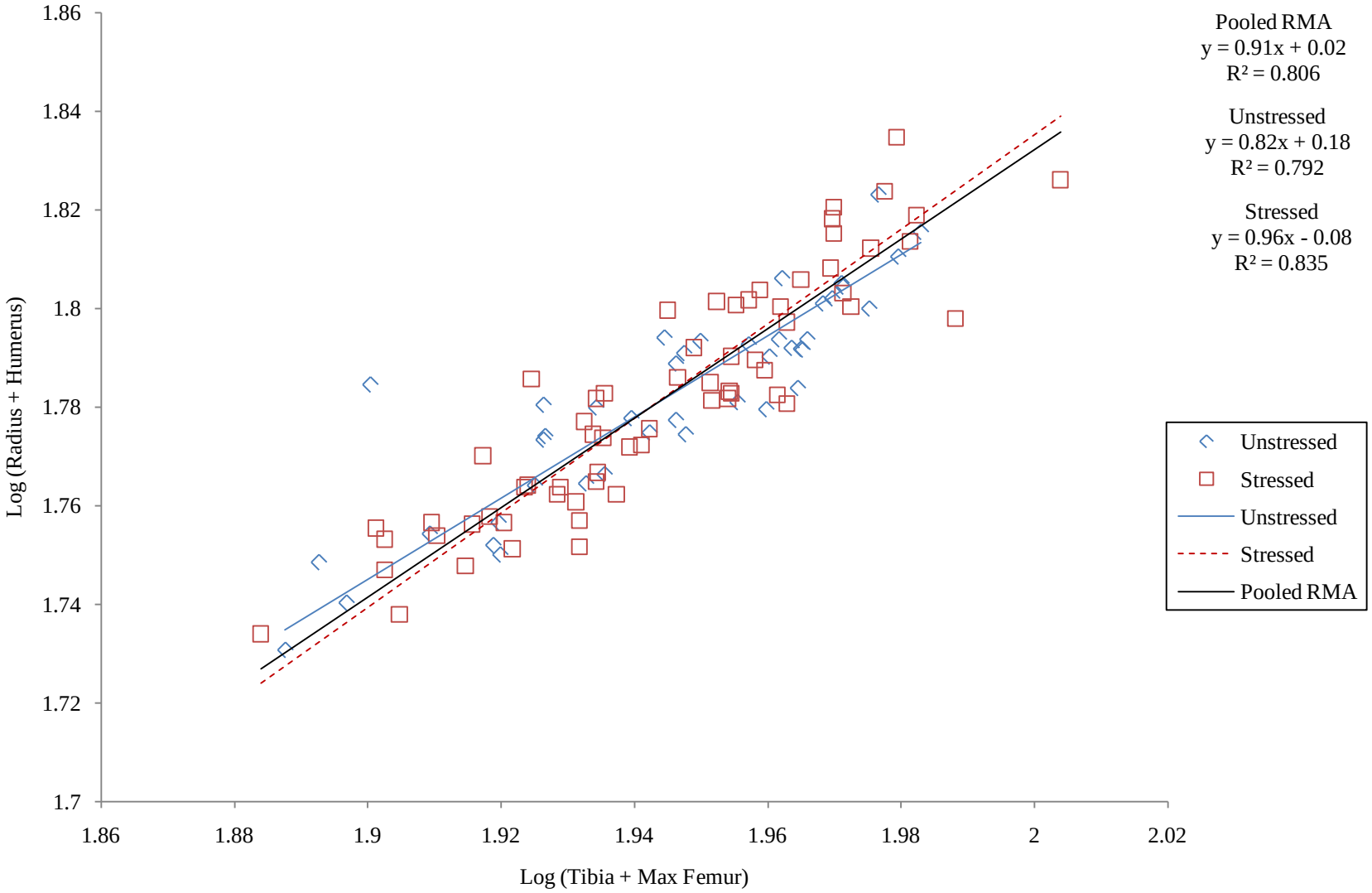


Figure 7-11. RMA for intermembral indices divided by dental enamel defects (male)

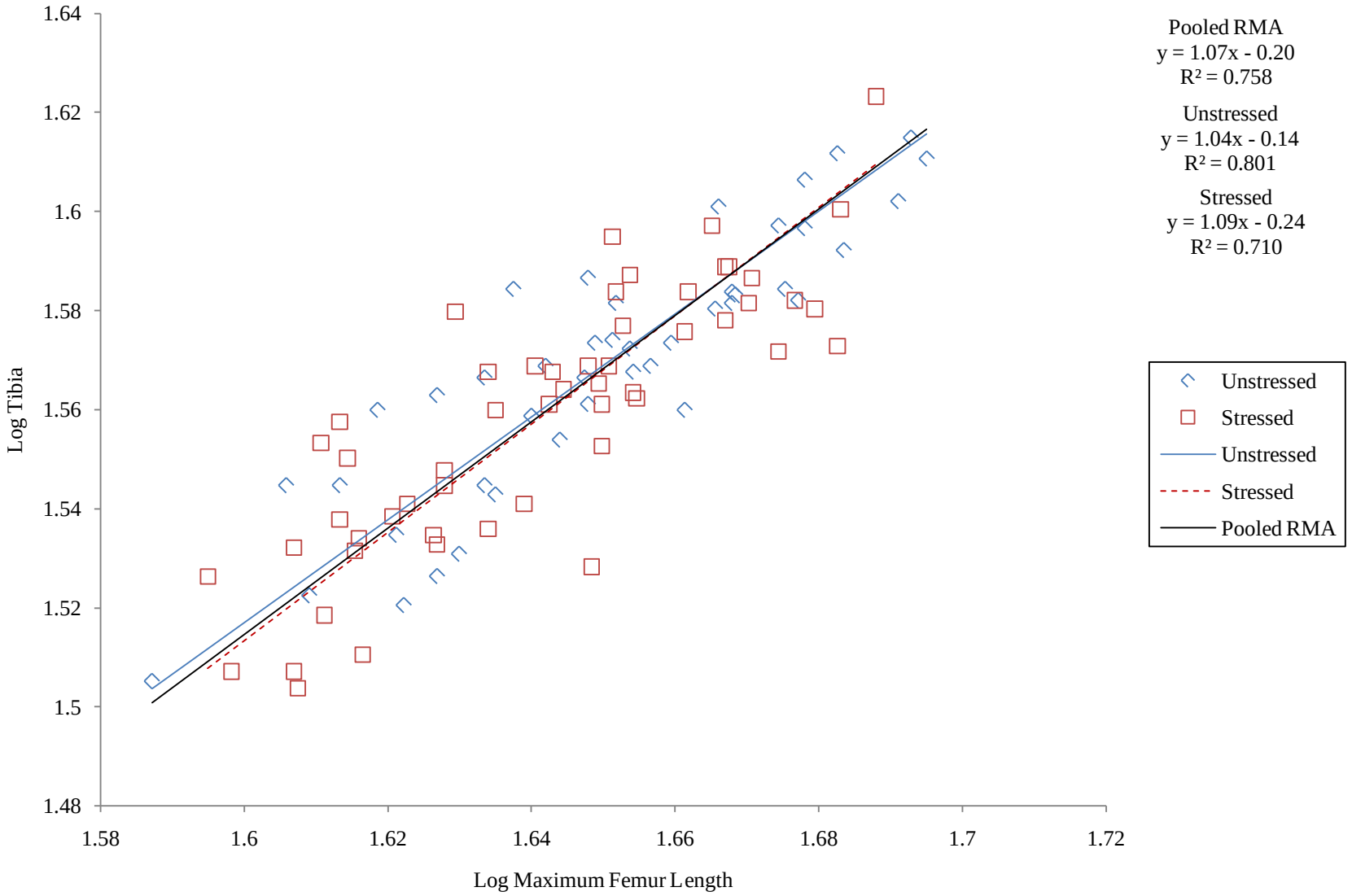


Figure 7-12. RMA for crural indices divided by dental enamel defects (female)

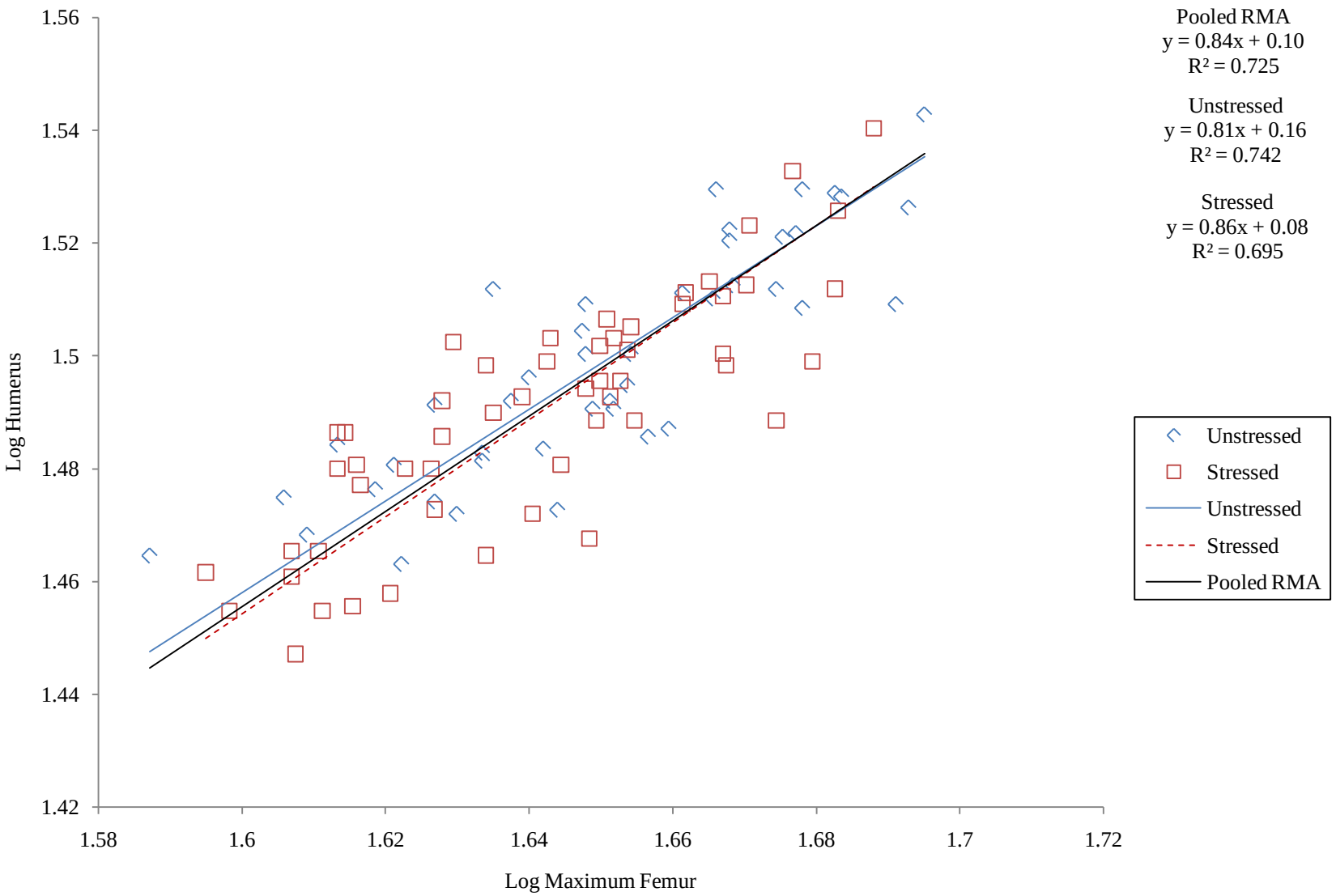


Figure 7-13. RMA for humerofemoral indices divided by dental enamel defects (female)

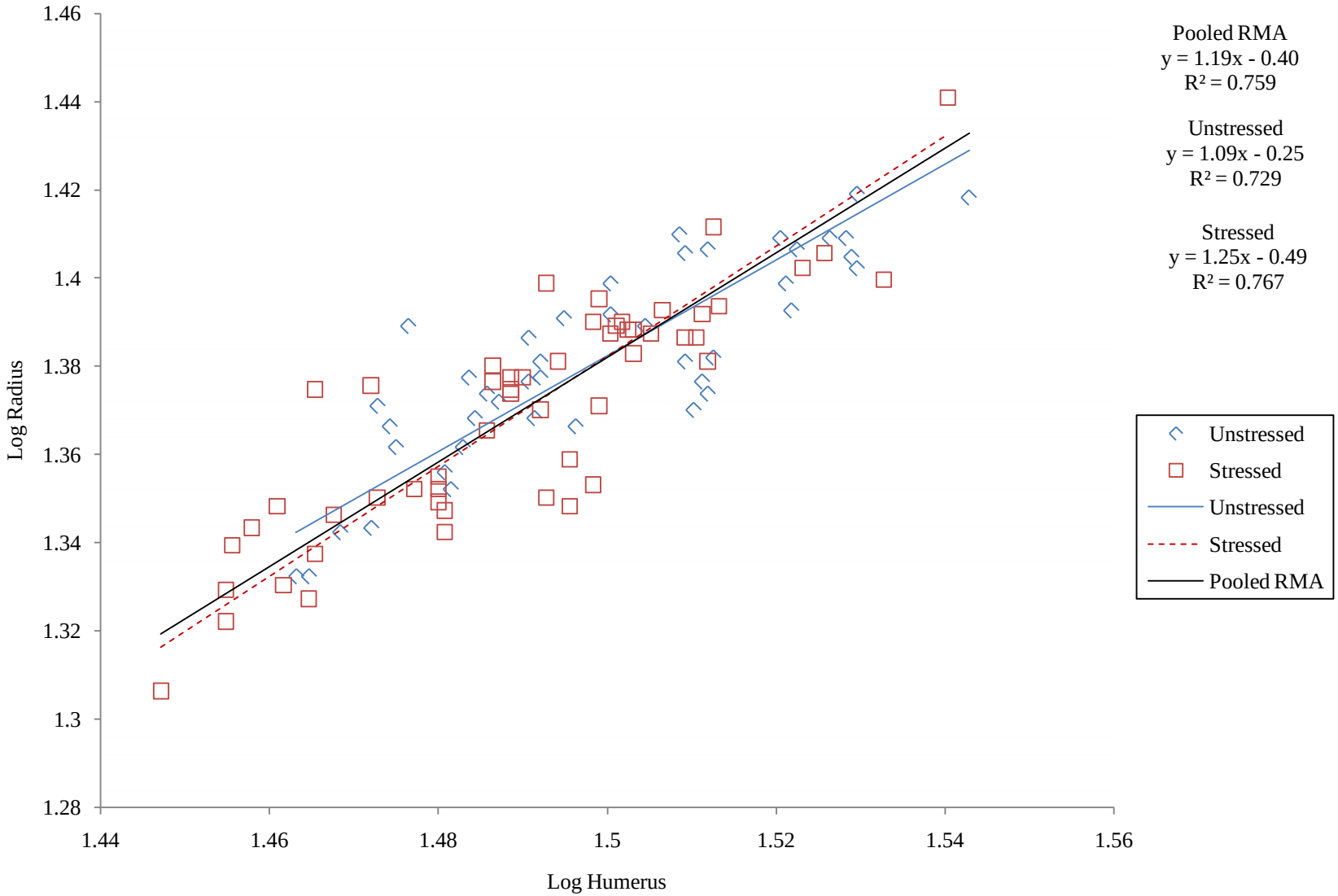


Figure 7-14. RMA for radiohumeral indices divided by dental enamel defects (female)

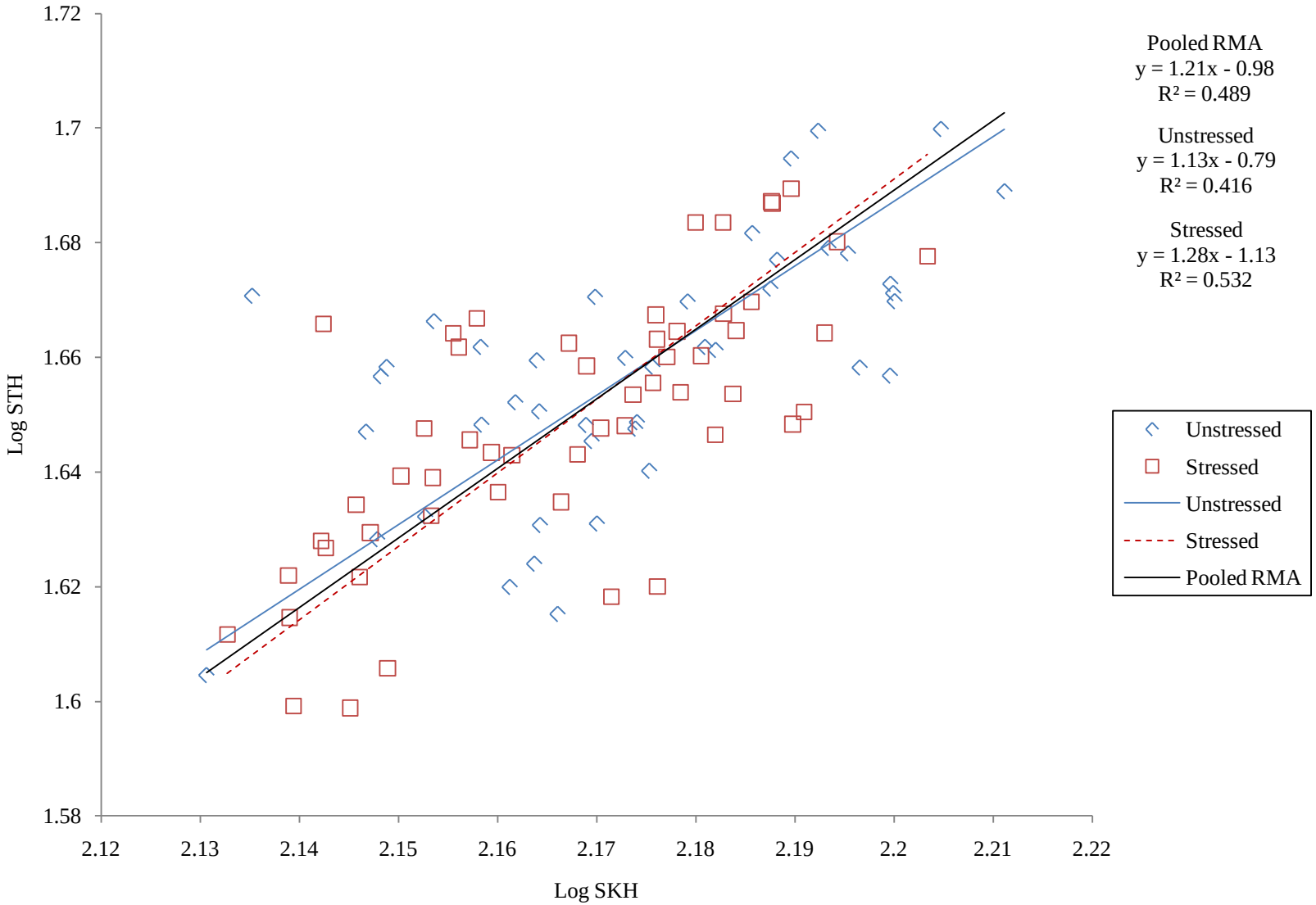


Figure 7-15. RMA for sitting height indices divided by dental enamel defects (female)

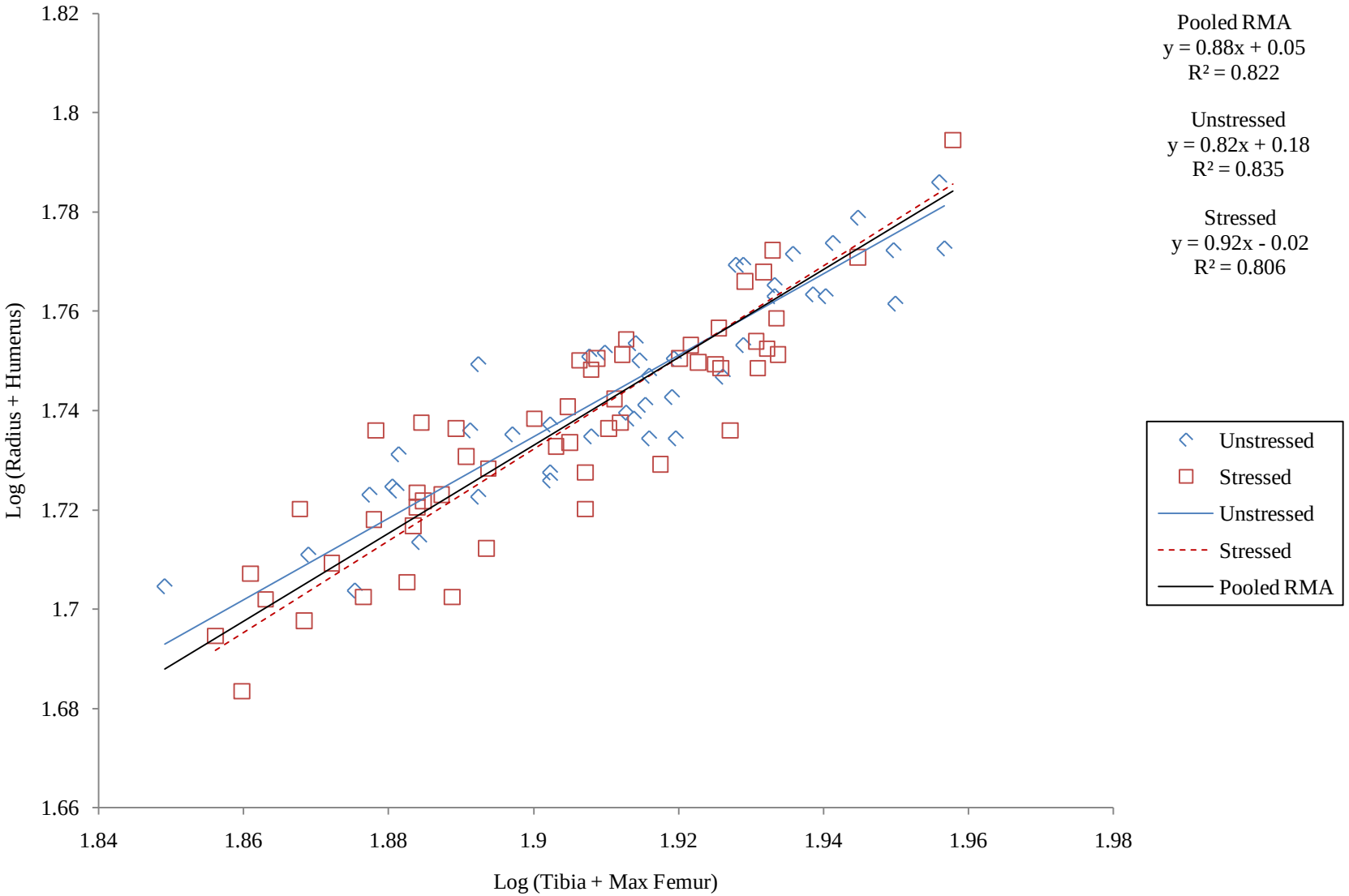


Figure 7-16. RMA for intermembral indices divided by dental enamel defects (female)

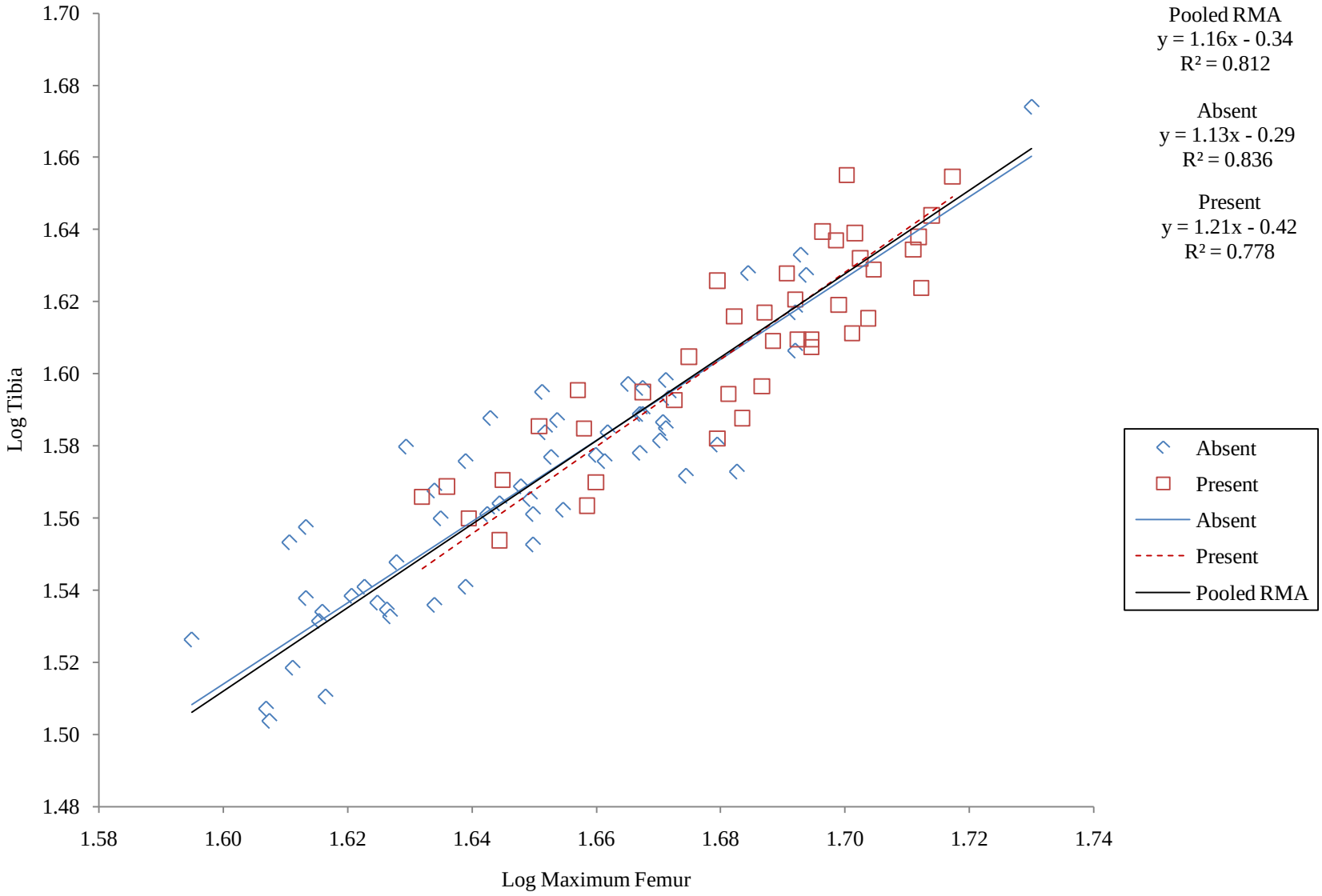


Figure 7-17. RMA for crural indices divided by Harris lines (male)

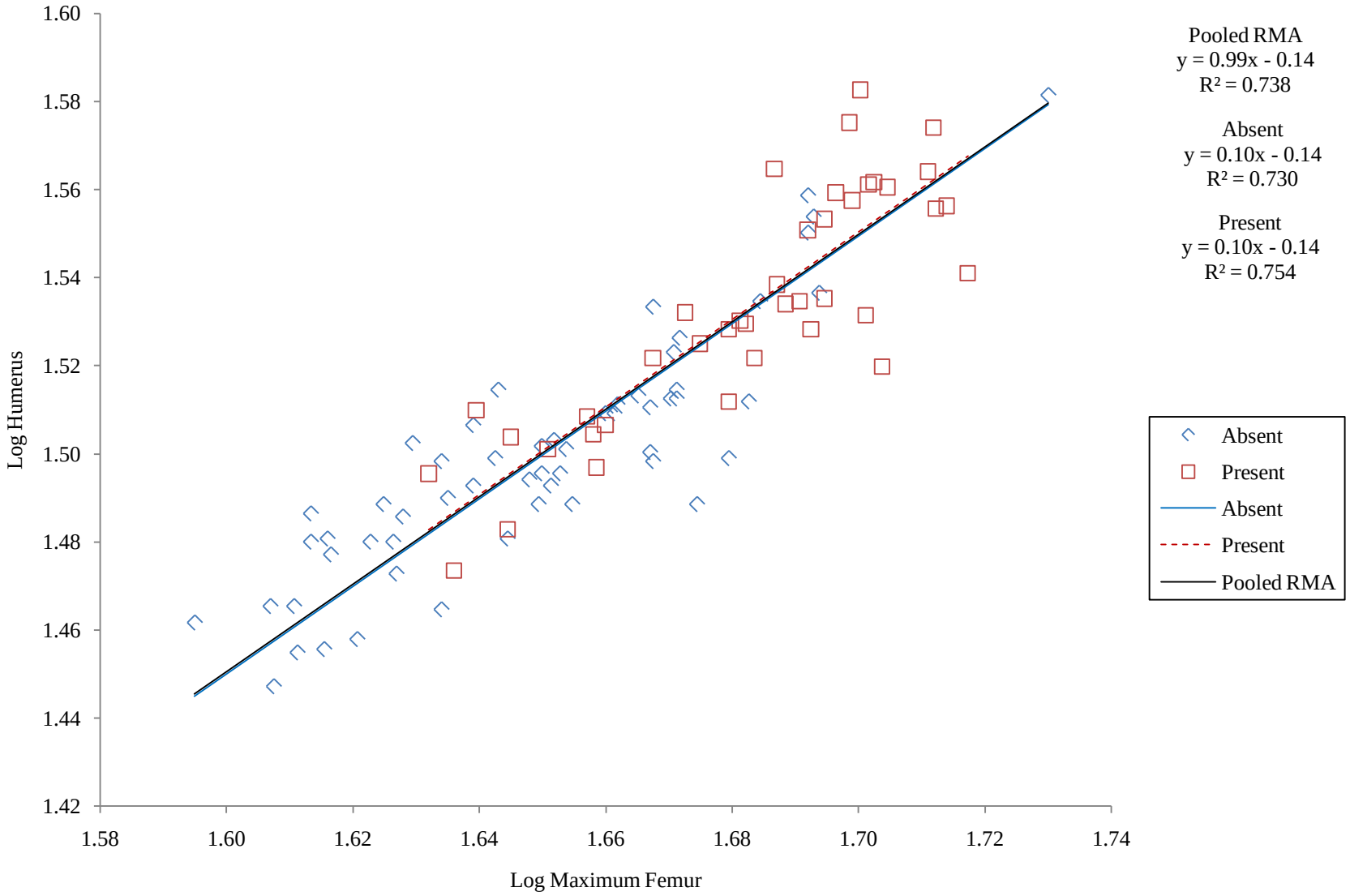


Figure 7-18. RMA for humerofemoral indices divided by Harris lines (male)

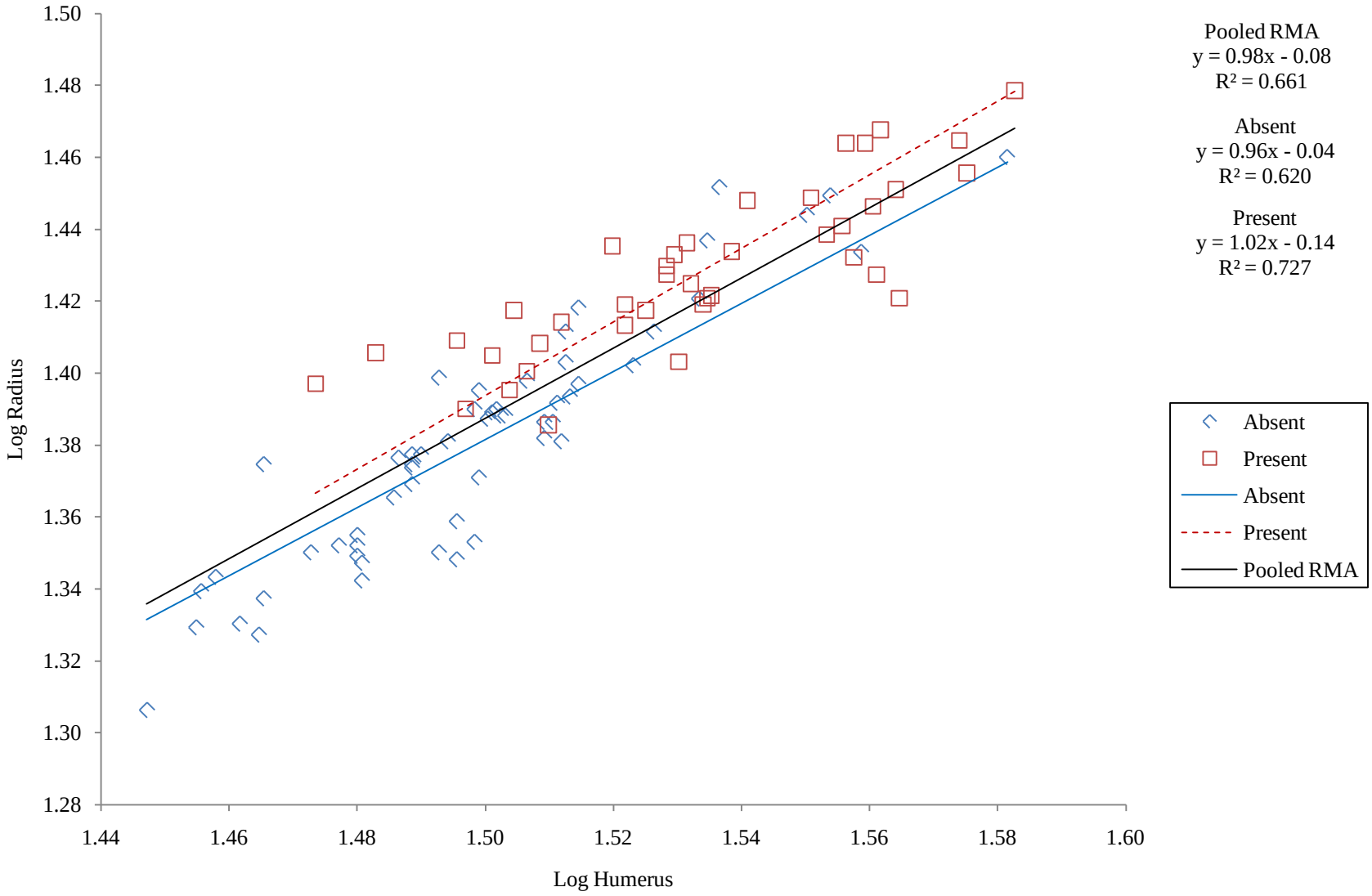


Figure 7-19. RMA for radiohumeral indices divided by Harris lines (male)

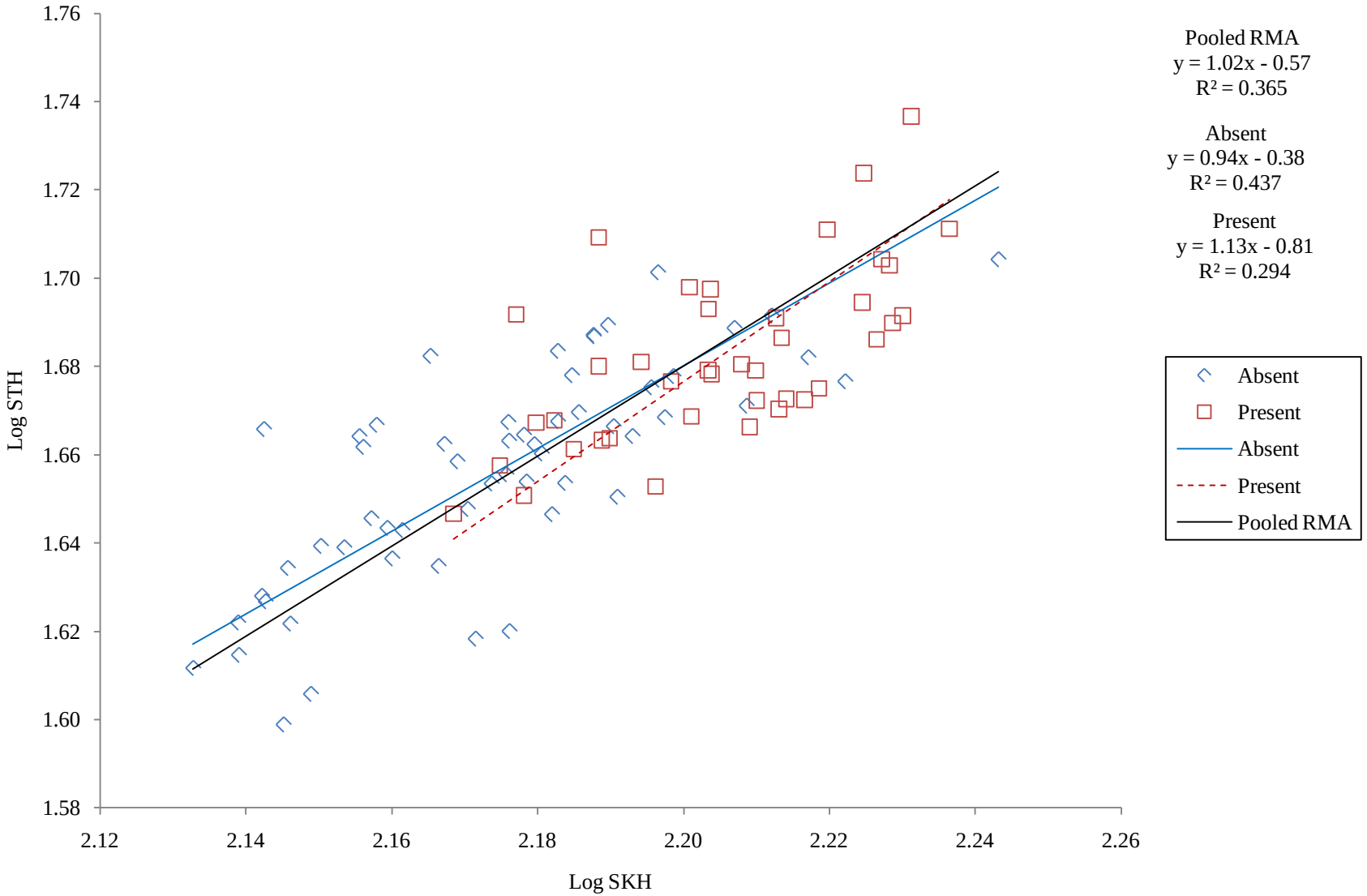


Figure 7-20. RMA for sitting height indices divided by Harris lines (male)

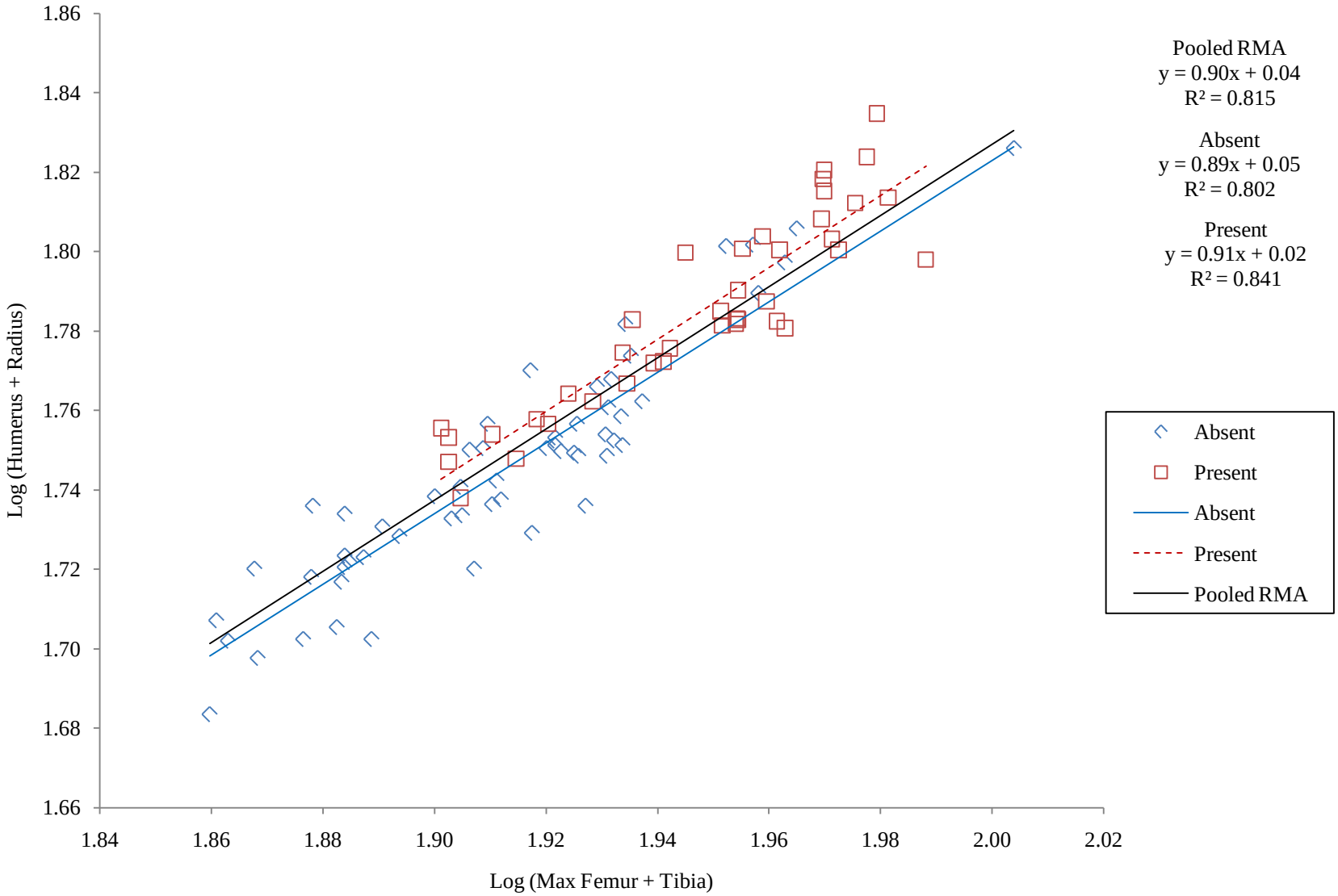


Figure 7-21. RMA for intermembral indices divided by Harris lines (male)

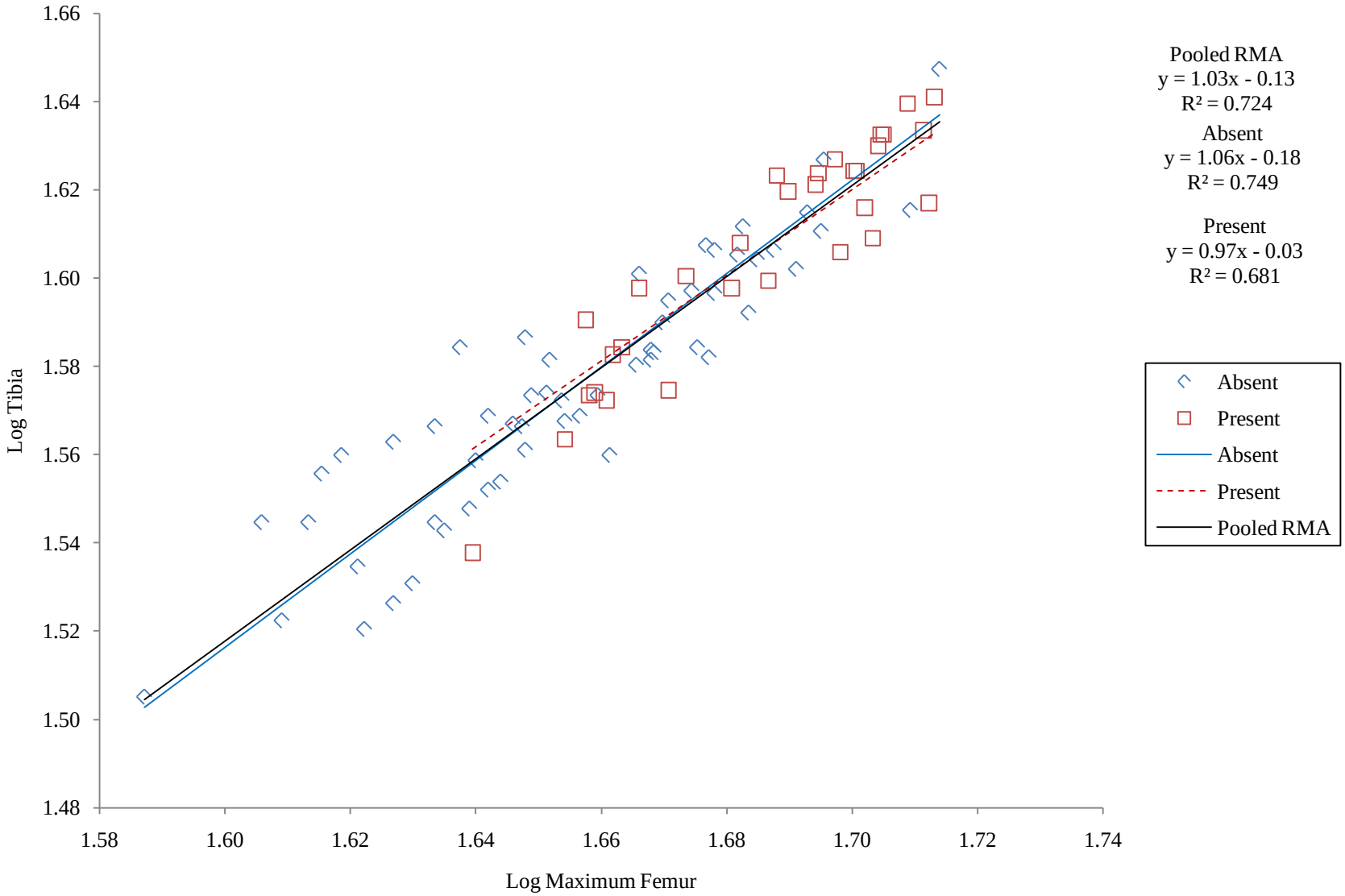


Figure 7-22. RMA for crural indices divided by Harris lines (female)

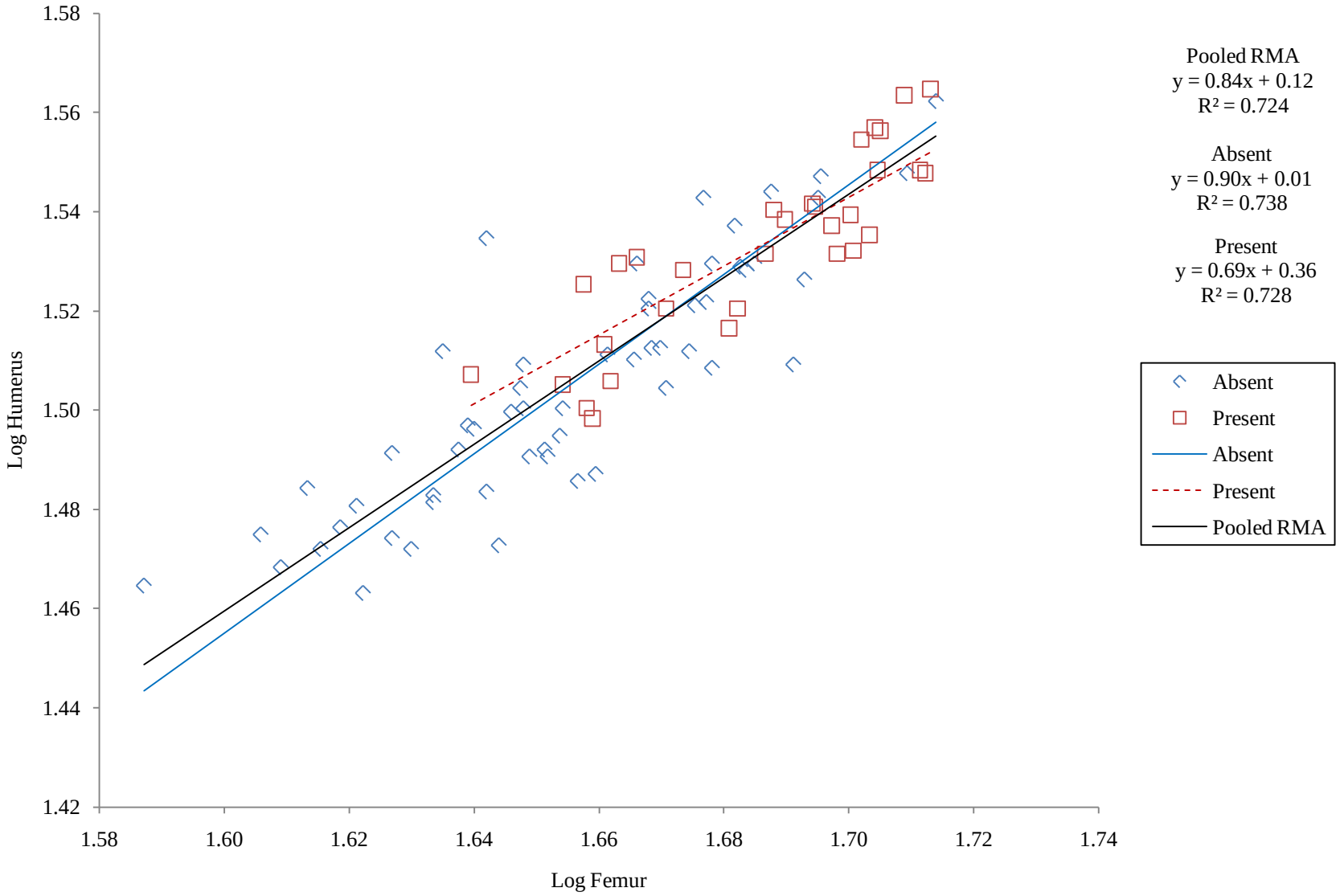


Figure 7-23. RMA for humerofemoral indices divided by Harris lines (female)

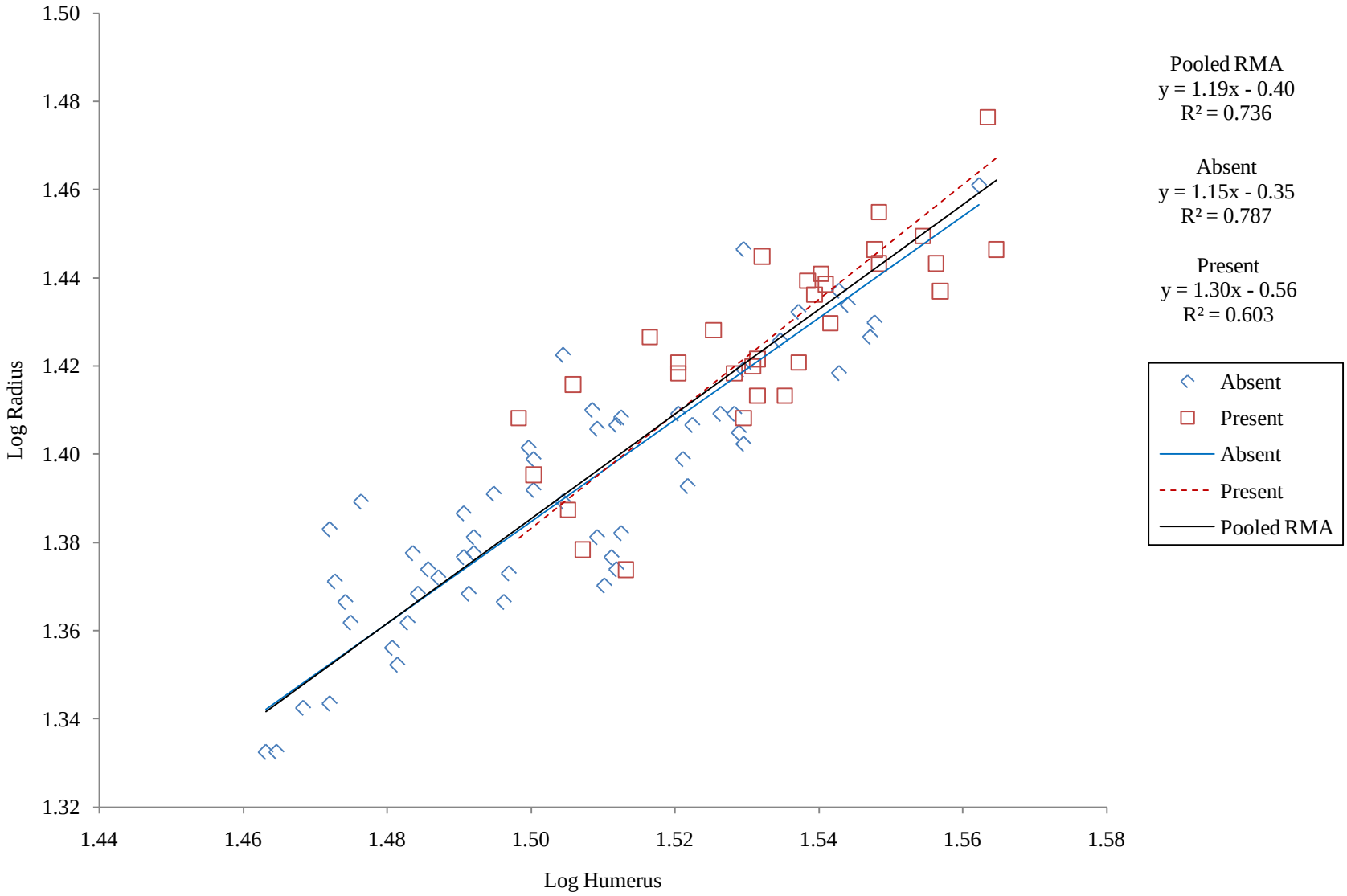


Figure 7-24. RMA for radiohumeral indices divided by Harris lines (female)

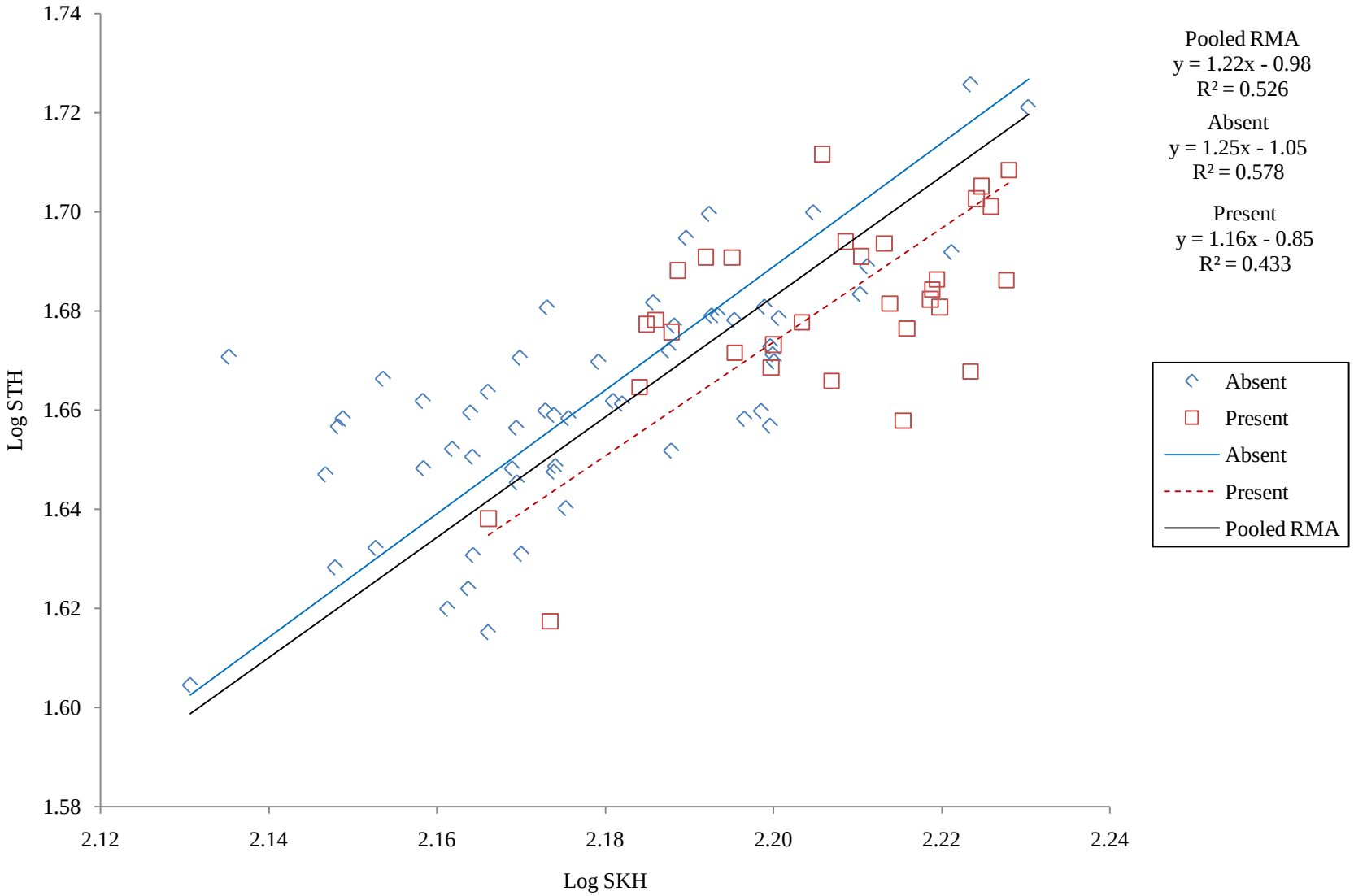


Figure 7-25. RMA for sitting height indices divided by Harris lines (female)

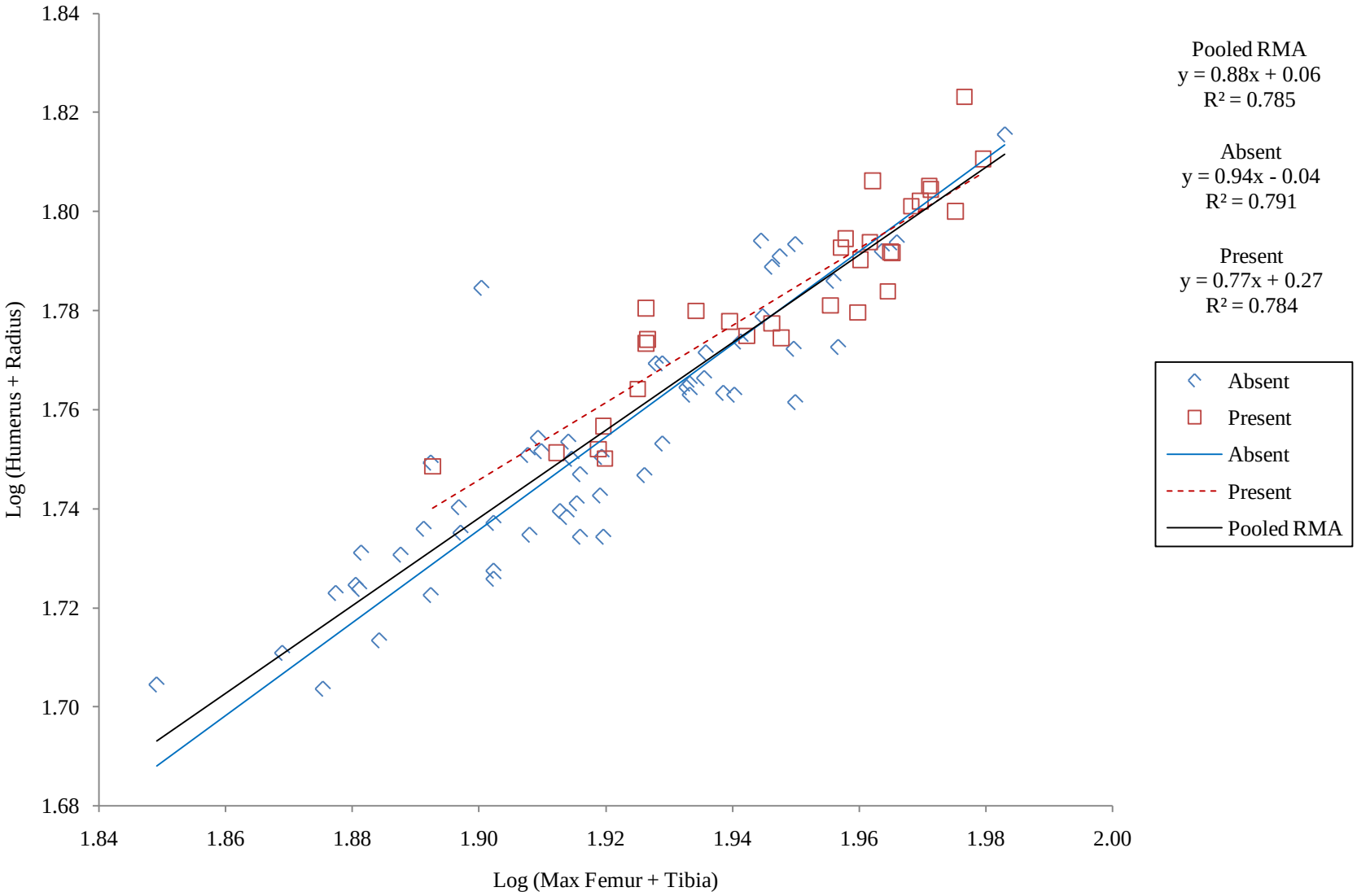


Figure 7-26. RMA for intermembral indices divided by Harris lines (female)

CHAPTER 8
RESULTS: TRADITIONAL AND NON-TRADITIONAL MARKERS OF GROWTH DISRUPTION IN THE ADULT SKELETON

Linear enamel hypoplasias and Harris lines are standard markers used to assess growth disruption in the adult skeleton. It has been suggested that the diameter of vertebral neural canals provide a similar line of evidence in that they are affected by stressors and cease growth early in life, thus preserving a marker of early stress events. Deficits in head circumference measurements, a common marker of growth disruption in anthropometric studies of living children, may also persist into adulthood in some individuals (Brandt et al., 2003). This chapter compares head circumference and vertebral neural canal shape to linear enamel hypoplasias and Harris lines to see if there is a significant relationship between these variables.

Vertebral Neural Canals and Traditional Stress Markers

The anteroposterior diameters of the lumbar vertebrae (L1-L4) are 70% complete at birth (Jeffrey et al., 2003). The mediolateral (transverse) neural canal diameters and vertebral body heights continue to grow through childhood, and are thus more likely to experience catch-up before completing growth in the case of growth disruption. Small anteroposterior vertebral neural canal diameters have been associated with low birth weight, maternal smoking and protein deficient childhood diets (Clark, 1988; Jeffrey et al., 2003). This investigation will determine whether vertebral neural canal (VNC) size is associated with other skeletal stress markers and whether or not VNC size is associated with adult stature and proportions.

To determine the relationship between vertebral neural canal size and other more traditional stress markers, individuals were divided into groups based on the presence or absence of enamel defects as well as Harris lines. These groups were tested for normality and homoscedasticity and one-way ANOVAs were performed on those groups meeting these assumptions. Where assumptions were not met, non-parametric Mann-Whitney *U* tests were

performed. ANOVAs were performed on anteroposterior (AP) and mediolateral (ML) diameters and the vertebral canal index (AP/ML) on L1, averaged thoracic and averaged lumbar measurements. The ANOVAs were performed on a combined sex sample as well as on samples divided by sex.

ANOVAs dividing the sample by enamel defects (CBTS) yielded significant results for the anteroposterior diameters of L1 and the averaged lumbar samples for the combined sex and male samples (Table 8-1). Mann-Whitney *U* tests yielded significant results for the averaged thoracic anteroposterior diameters in both the male and combined sex samples (Table 8-2). Significant results were found for mediolateral diameters in the case of the male averaged lumbar sample alone. The vertebral canal index measures were found to be significantly different for L1 (male and combined sex). In no case were female vertebral canal sizes, in any dimension, significantly related to enamel defects (CBTS).

Unlike the ANOVAs for enamel defects, ANOVAs dividing the sample by the presence or absence of Harris lines (HL) only yielded significant results for the female samples. In addition, the significant results ($p < 0.05$) were for the mediolateral diameters of the averaged thoracic and lumbar measurements. These ANOVAs are summarized in Table 8-3 and the nonparametric tests are summarized in Table 8-4. Near significant ($0.05 < p < 0.10$) results were found for the vertebral canal index of L1 among females which prompted the use of resampling statistics as an independent test. The resampling procedure tested the alternative hypothesis that the vertebral neural canal index was greater in the unstressed group. No support was found for the alternative hypothesis in question ($p = 0.9774$). However, the inverse relationship is significant ($1 - p = 0.0226$). The vertebral canal index of L1 among females is significantly greater in the stressed

sample. This means that the AP diameter of the L1 VNC is relatively greater than the ML diameter among stressed females.

Because of the sex differences found in the one-way ANOVAs, two-way ANOVAs were performed to determine whether or not there was any interaction effect between sex and the traditional stress markers. No significant results were found, but enamel defects (CBTS) and sex were found to have a near significant interaction effect for the averaged lumbar anteroposterior diameters ($p = 0.079$). Near significant results were also found for an interaction effect between Harris lines and sex for L1 and the averaged thoracic vertebral canal indices ($p = 0.094$ and $p = 0.057$, respectively).

Clark (1988) suggests that the relationship between vertebral neural canals (VNC) and vertebral body heights (VBHs) is a critical one to examine. If VNC and VBH measurements are reduced, Clark considers this to be an indicator of chronic growth disruption. If VNC measurements are reduced but VBH measurements are not, then catch-up growth may have occurred. ANOVAs were performed on vertebral body heights divided by the presence or absence of enamel defects (CBTS) and Harris lines (HL). When assumptions of normality or homoscedasticity were not met, Mann-Whitney U tests were performed to test the difference between groups. The results of these tests, summarized in Tables 8-5 through 8-8, demonstrate that there are no significant differences in vertebral body height in samples grouped by the presence or absence of enamel defects or Harris lines.

Reduced major axis regressions (RMAs) of VNC measurements over VBH were performed to see if there were significant scaling differences between those with traditional indicators of stress (enamel defects and Harris lines) and those without. A few significant differences were found in the slopes of RMAs in females. When the sample was divided by

enamel defects (CBTS), the significant differences were found in AP diameters of the lumbar vertebrae (L1 and averaged) (see Table 8-9). When the sample was divided by the presence or absence of Harris lines (HL), near significant differences ($0.05 < p < 0.10$) in slope were found in the transverse diameters of the lumbar vertebrae (averaged and L1) (see Table 8-10). Significant ($p < 0.05$) results suggest that VNC size increases at a faster rate than VBH in stressed groups.

Intercept differences were also found for females in the averaged mediolateral measurement of the thoracic and lumbar vertebrae when the sample was divided by HL. In males, intercept differences were found for the averaged mediolateral measurements of the thoracic vertebrae whether the sample was divided by CBTS or HL markers. Near significant intercept differences were found for the anteroposterior diameters of the averaged lumbar canals in males. The significant intercept differences suggest that for a given VBH, VNC size is significantly different. Where significant differences in slope occur, intercept differences are meaningless and are therefore not calculated. Figures 8-1 through 8-8 graph the reduced major axis regressions in which significant or near significant differences in slopes or intercepts were found. It is important to note that in most of these regressions the R^2 values are low, suggesting that allometry can only explain part of the observed variation.

Vertebral Neural Canals and Stature

Previous studies have determined that tall individuals tend to have wider vertebrae, and hence wider mediolateral measurements of their vertebral neural canals (Clark et al., 1985; Porter et al., 1987; Porter and Pavitt, 1987); therefore independence should not be assumed between skeletal height and mediolateral vertebral neural canal diameters. In contrast, the same research found that anteroposterior measures of the vertebral neural canals were independent of tibial length or other anthropometrics (Clark et al., 1985; Porter et al., 1987).

Skeletal height (SKH) and the anteroposterior measurements of L1, the average lumbar and the average thoracic vertebral neural canals were all found to have normal distributions using the Shapiro-Wilk test. Pearson's correlations and the non-parametric Kendall's tau correlations were performed comparing the anteroposterior vertebral canal of the averaged thoracic vertebrae and SKH, L1 and SKH, the averaged lumbar vertebrae and SKH. All correlations were found to be highly significant with the exception of the anteroposterior dimensions of the averaged lumbar canals among females (see Table 8-11). The correlation between skeletal height and anteroposterior vertebral neural canal diameters suggest that larger VNC diameters may be an effect of body size despite previously published results (Clark et al., 1985; Porter et al., 1987).

As a heuristic exercise, the relationship between vertebral canal size and height was further illustrated by dividing the population into quartiles based on the anteroposterior diameter of L1 and comparing skeletal height. Box plots show that skeletal height is greater in those individuals in the highest quartile for L1 anteroposterior diameter than those in the lowest quartile (Figures 8-9 and 8-10). However, because VNC diameters may be dependent on overall body size, anteroposterior diameters were size standardized by dividing the VNC measurements by vertebral body height. Size-standardized VNC measurements and SKH cannot be considered independent variables; therefore, the size-standardized box plots compare maximum femur length between the highest and lowest quartiles in males and females (see Figures 8-11 and 8-12). Interpretation of these results is difficult because femur length is also correlated with body size.

Head Circumference and Traditional Stress Markers

Head circumference, an anthropometric indicator of stunted growth in children, is also a measurement typically included in autopsy reports. Two methods were used for acquiring head circumference measures in this study. All head circumference measurements of individuals from

the Terry collection were collected by approximating the anthropometric technique used in growing children (Zerran, 2007). A flexible tape measure was placed around the head from opisthocranion to a point just above the brow ridge. This measurement was recorded in millimeters for each individual. Wax (approximating the width of the saw blade) was used to hold the skulls together for measurement in specimens where either the calotte was removed or the skull was hemisected. At the Hamann-Todd collection, hemisected skulls are all reconstructed with wire and head circumference measures were difficult to obtain accurately. Due to this complication and the easy access to autopsy records in the Hamann-Todd database, I decided to obtain the Hamann-Todd head circumference measurements from the collection's records. While I can vouch for the consistency of my own data collection techniques, a cursory inspection of the Hamann-Todd data caused me to question the reliability of head circumference measures provided in the autopsy records.

Because both skeletal height and head circumference are not assumed to be dependent on overall body size, correlations were performed to examine this relationship in the study sample. Among the males and females measured in the Terry collection, a Pearson's correlation found that head circumference and skeletal height were significantly correlated (male $p = 0.011$; female $p = 0.028$). In the Hamann-Todd collection, a Pearson's correlation between head circumference and skeletal height was found to be insignificant ($p = 0.631$) in the male sample. Because head circumference in the female sample is not normally distributed, a Kendall's tau correlation was performed and was not found to be significant ($p = 0.097$). The results of these correlations are summarized in table 8-11. The fact that head circumference and skeletal height were not significantly correlated in the Hamann-Todd sample cause me to question the integrity of

autopsy reports; accordingly, they have been removed from all analyses comparing head circumference data to traditional stress markers.

Data from the Terry collection was tested for normality and homoscedasticity. Where these assumptions were met, one-way ANOVAs were performed to determine whether or not there was a significant difference in head circumference between groups with and without traditional stress markers (enamel defects and Harris lines). Mann-Whitney *U* tests were used on all non-parametric data.

No significant difference was found in the male sample when divided by dental enamel defects or Harris lines, or in the female sample when divided by enamel defects (CBTS) (Table 8-12). The female sample divided by Harris lines was not found to meet the assumptions of an ANOVA and so a Mann-Whitney *U* test was performed. The difference in head circumference when the population was divided by the presence or absence of Harris lines was found to be insignificant (= -1.166 $p = 0.244$).

Table 8-1. ANOVAs of vertebral neural canal measurements by dental enamel defects (CBTS)

	N	Mean	S.D.	Shapiro-Wilk	Levine's	F	*p*
Combined Sex							
L1 - anteroposterior							
Unstressed	84	17.68	1.458				
Stressed	119	17.17	1.369				
Total	203	17.38	1.426	0.411	0.507	6.557	0.011 [a]
L1 - mediolateral							
Unstressed	85	21.00	1.945				
Stressed	119	21.01	1.737				
Total	204	21.00	1.822	0.505	0.104	0.001	0.971
L1 -AP/ML							
Unstressed	84	0.85	0.073				
Stressed	119	0.82	0.072				
Total	203	0.83	0.073	0.466	0.761	6.171	0.014 [a]
Thoracic (average) - anteroposterior							
Unstressed	85	15.23	1.205				
Stressed	116	14.84	1.028				
Total	201	15.00	1.120	0.044 [a, c]	.	.	.
Thoracic (average) - mediolateral							
Unstressed	85	16.76	1.413				
Stressed	115	16.72	1.378				
Total	200	16.74	1.390	0.120	0.544	0.050	0.823
Thoracic (average) - AP/ML							
Unstressed	85	0.92	0.073				
Stressed	114	0.90	0.063				
Total	199	0.91	0.068	0.561	0.337	4.050	0.046 [a]
Lumbar (average) - anteroposterior							
Unstressed	84	16.92	1.574				
Stressed	119	16.46	1.584				
Total	203	16.65	1.592	0.126	0.400	4.237	0.041 [a]
Lumbar (average) - mediolateral							
Unstressed	85	22.99	2.020				
Stressed	119	22.59	1.729				
Total	204	22.75	1.861	0.114	0.027 [a, c]	.	.
Lumbar (average) - AP/ML							
Unstressed	85	0.74	0.061				
Stressed	119	0.73	0.057				
Total	204	0.74	0.059	0.432	0.624	1.252	0.264
Male							
L1 - anteroposterior							
Unstressed	41	17.93	1.568				
Stressed	65	17.18	1.286				
Total	106	17.47	1.442	0.814	0.147	7.244	0.008 [a]

Table 8-1. Continued

	N	Mean	S.D.	Shapiro-Wilk	Levine's	F	*p*
L1 - mediolateral							
Unstressed	42	21.89	1.570				
Stressed	65	21.71	1.672				
Total	107	21.78	1.628	0.418	0.915	0.316	0.575
L1 -AP/ML							
Unstressed	41	0.82	0.064				
Stressed	65	0.79	0.060				
Total	106	0.80	0.063	0.897	0.981	4.468	0.037 [a]
Thoracic (average) - anteroposterior							
Unstressed	42	15.45	1.319				
Stressed	63	14.86	0.993				
Total	105	15.10	1.164	0.106	0.046 [a, c]	.	.
Thoracic (average) - mediolateral							
Unstressed	42	17.44	1.276				
Stressed	63	17.23	1.369				
Total	105	17.31	1.330	0.174	0.881	0.644	0.424
Thoracic (average) - AP/ML							
Unstressed	42	0.90	0.074				
Stressed	62	0.88	0.052				
Total	104	0.88	0.062	0.251	0.019 [a, c]	.	.
Lumbar (average) - anteroposterior							
Unstressed	41	17.37	1.635				
Stressed	65	16.50	1.618				
Total	106	16.84	1.672	0.592	0.272	7.250	0.008 [a]
Lumbar (average) - mediolateral							
Unstressed	42	23.80	1.807				
Stressed	65	23.12	1.674				
Total	107	23.39	1.752	0.403	0.256	3.990	0.048 [a]
Lumbar (average) - AP/ML							
Unstressed	42	0.73	0.054				
Stressed	65	0.72	0.052				
Total	107	0.72	0.053	0.726	0.901	1.869	0.174
Female							
L1 - anteroposterior							
Unstressed	43	17.44	1.318				
Stressed	54	17.16	1.476				
Total	97	17.28	1.408	0.358	0.584	0.999	0.320
L1 - mediolateral							
Unstressed	43	20.12	1.894				
Stressed	54	20.16	1.416				
Total	97	20.15	1.636	0.146	0.055 [b]	0.012	0.913

Table 8-1. Continued

	N	Mean	S.D.	Shapiro-Wilk	Levine's	F	*p*
L1 -AP/ML							
Unstressed	43	0.87	0.072				
Stressed	54	0.85	0.071				
Total	97	0.86	0.072	0.736	0.919	1.560	0.215
Thoracic (average) - anteroposterior							
Unstressed	43	15.02	1.054				
Stressed	53	14.81	1.078				
Total	96	14.90	1.066	0.329	0.927	0.851	0.359
Thoracic (average) - mediolateral							
Unstressed	43	16.10	1.223				
Stressed	52	16.10	1.123				
Total	95	16.10	1.163	0.341	0.590	0.000	0.999
Thoracic (average) - AP/ML							
Unstressed	43	0.94	0.064				
Stressed	52	0.93	0.062				
Total	95	0.94	0.063	0.599	0.804	0.995	0.321
Lumbar (average) - anteroposterior							
Unstressed	43	16.48	1.396				
Stressed	54	16.40	1.554				
Total	97	16.44	1.479	0.417	0.433	0.078	0.781
Lumbar (average) - mediolateral							
Unstressed	43	22.19	1.911				
Stressed	54	21.95	1.583				
Total	97	22.06	1.731	0.097 [b]	0.168	0.453	0.503
Lumbar (average) - AP/ML							
Unstressed	43	0.75	0.066				
Stressed	54	0.75	0.057				
Total	97	0.75	0.061	0.645	0.311	0.003	0.953

[a] Significant ($p < 0.05$)

[b] Near significant ($0.05 < p < 0.10$)

[c] The assumptions of an ANOVA are violated where conditions of normality and homoscedasticity are not met.

Table 8-2. Nonparametric tests of VNC measurements by enamel defects (CBTS)

	N	Mean Rank	Sum of Ranks	Mann-Whitney U	Wilcoxon W	Z	*p* (2-tailed)
Combined Sex							
Thoracic (average) - AP							
Unstressed	85	112.61	9571.5				
Stressed	116	92.50	10729.5	3943.5	10729.5	-2.421	0.015 [a]
Lumbar (average) - ML							
Unstressed	85	108.70	9239.5				
Stressed	119	98.07	11670.5	4530.5	11670.5	-1.268	0.205
Male							
Thoracic (average) - AP							
Unstressed	42	61.32	2575.5				
Stressed	63	47.45	2989.5	973.5	2989.5	-2.286	0.022
Thoracic (average) - AP/ML							
Unstressed	42	57.89	2431.5				
Stressed	62	48.85	3028.5	1075.5	3028.5	-1.501	0.133

[a] Significant ($p < 0.05$)

Table 8-3. ANOVAs of vertebral neural canal measurements by Harris lines (HL)

	N	Mean	S.D.	Shapiro-Wilk	Levine's	F	*p*
Combined Sex							
L1 - anteroposterior							
Unstressed	111	17.44	1.510				
Stressed	71	17.41	1.304				
Total	182	17.43	1.429	0.411	0.161	0.023	0.881
L1 - mediolateral							
Unstressed	112	21.09	1.855				
Stressed	71	20.82	1.765				
Total	183	20.99	1.820	0.505	0.383	0.921	0.338
L1 -AP/ML							
Unstressed	111	0.83	0.072				
Stressed	71	0.84	0.074				
Total	182	0.83	0.073	0.466	0.993	0.765	0.383
Thoracic (average) - anteroposterior							
Unstressed	109	15.07	1.120				
Stressed	71	14.90	1.145				
Total	180	15.00	1.129	0.044 [a, c]	.	.	.
Thoracic (average) - mediolateral							
Unstressed	110	16.78	1.384				
Stressed	69	16.58	1.426				
Total	179	16.70	1.400	0.120	0.828	0.874	0.351
Thoracic (average) - AP/ML							
Unstressed	109	0.91	0.066				
Stressed	69	0.91	0.071				
Total	178	0.91	0.068	0.561	0.434	0.007	0.935
Lumbar (average) - anteroposterior							
Unstressed	111	16.79	1.643				
Stressed	71	16.55	1.563				
Total	182	16.70	1.612	0.126	0.535	0.964	0.327
Lumbar (average) - mediolateral							
Unstressed	112	22.97	1.789				
Stressed	71	22.57	1.951				
Total	183	22.81	1.859	0.114	0.869	2.052	0.154
Lumbar (average) - AP/ML							
Unstressed	112	0.74	0.057				
Stressed	71	0.74	0.058				
Total	183	0.74	0.057	0.432	0.732	0.180	0.671

Table 8-3. Continued

	N	Mean	S.D.	Shapiro-Wilk	Levine's	F	*p*
Male							
L1 - anteroposterior							
Unstressed	55	17.61	1.462				
Stressed	40	17.42	1.444				
Total	95	17.53	1.450	0.814	0.916	0.395	0.531
L1 - mediolateral							
Unstressed	56	21.82	1.773				
Stressed	40	21.64	1.498				
Total	96	21.75	1.658	0.418	0.105	0.261	0.611
L1 -AP/ML							
Unstressed	55	0.81	0.064				
Stressed	40	0.81	0.060				
Total	95	0.81	0.062	0.897	0.449	0.037	0.847
Thoracic (average) - anteroposterior							
Unstressed	54	15.17	1.130				
Stressed	40	15.03	1.251				
Total	94	15.11	1.178	0.106	0.539	0.317	0.575
Thoracic (average) - mediolateral							
Unstressed	55	17.27	1.420				
Stressed	39	17.28	1.284				
Total	94	17.27	1.358	0.174	0.471	0.002	0.964
Thoracic (average) - AP/ML							
Unstressed	54	0.89	0.063				
Stressed	39	0.88	0.064				
Total	93	0.89	0.064	0.251	0.925	1.194	0.277
Lumbar (average) - anteroposterior							
Unstressed	55	17.01	1.652				
Stressed	40	16.83	1.721				
Total	95	16.94	1.675	0.592	0.773	0.282	0.597
Lumbar (average) - mediolateral							
Unstressed	56	23.46	1.762				
Stressed	40	23.36	1.775				
Total	96	23.42	1.759	0.403	0.576	0.069	0.793
Lumbar (average) - AP/ML							
Unstressed	56	0.73	0.054				
Stressed	40	0.72	0.048				
Total	96	0.73	0.051	0.726	0.740	0.139	0.711

Table 8-3. Continued

	N	Mean	S.D.	Shapiro-Wilk	Levine's	F	*p*
Female							
L1 - anteroposterior							
Unstressed	56	17.27	1.550				
Stressed	31	17.39	1.122				
Total	87	17.31	1.407	0.358	0.036 [a, c]	.	.
L1 - mediolateral							
Unstressed	56	20.36	1.647				
Stressed	31	19.77	1.516				
Total	87	20.15	1.618	0.146	0.705	2.725	0.102
L1 -AP/ML							
Unstressed	56	0.85	0.074				
Stressed	31	0.88	0.068				
Total	87	0.86	0.073	0.736	0.985	3.913	0.051 [b]
Thoracic (average) - anteroposterior							
Unstressed	55	14.97	1.111				
Stressed	31	14.74	0.989				
Total	86	14.89	1.068	0.329	0.889	0.872	0.353
Thoracic (average) - mediolateral							
Unstressed	55	16.30	1.173				
Stressed	30	15.68	1.054				
Total	85	16.08	1.165	0.341	0.257	5.892	0.017 [a]
Thoracic (average) - AP/ML							
Unstressed	55	0.93	0.065				
Stressed	30	0.95	0.059				
Total	85	0.94	0.063	0.599	0.685	2.564	0.113
Lumbar (average) - anteroposterior							
Unstressed	56	16.58	1.619				
Stressed	31	16.20	1.272				
Total	87	16.44	1.508	0.417	0.224	1.264	0.264
Lumbar (average) - mediolateral							
Unstressed	56	22.48	1.693				
Stressed	31	21.54	1.690				
Total	87	22.14	1.742	0.097 [b]	0.503	6.161	0.015 [a]
Lumbar (average) - AP/ML							
Unstressed	56	0.75	0.060				
Stressed	31	0.76	0.063				
Total	87	0.75	0.061	0.645	0.606	1.408	0.239

[a] Significant ($p < 0.05$)

[b] Near significant ($0.05 < p < 0.10$)

[c] The assumptions of an ANOVA are violated where conditions of normality and homoscedasticity are not met.

Table 8-4. Nonparametric tests of vertebral neural canal measurements by Harris lines (HL)

	N	Mean Rank	Sum of Ranks	Mann-Whitney U	Wilcoxon W	Z	*p* (2-tailed)
Combined Sex							
Thoracic (average) - AP							
Unstressed	109	92.92	10128.0				
Stressed	71	86.79	6162.0	3606.0	6162.0	-0.771	0.441
Female							
L1 - AP							
Unstressed	56	42.920	2403.5				
Stressed	31	45.950	1424.5	807.5	2403.5	-0.536	0.592

Table 8-5. ANOVAs of vertebral body height measurements by dental enamel defects (CBTS)

	N	Mean	S.D.	Shapiro-Wilk	Levine's	F	*p*
Male							
L1 - VBH							
Unstressed	42	26.36	1.387				
Stressed	65	26.10	1.457				
Total	107	26.21	1.429	0.324	0.817	0.846	0.360
Lumbar (average)- VBH							
Unstressed	42	27.92	1.504				
Stressed	65	27.84	1.504				
Total	107	27.87	1.498	0.361	0.822	0.075	0.784
Thoracic (average) - VBH							
Unstressed	42	20.30	0.883				
Stressed	65	20.36	0.862				
Total	107	20.34	0.867	0.055 [b]	0.741	0.124	0.726
Female							
L1 - VBH							
Unstressed	43	25.41	1.678				
Stressed	54	25.35	1.205				
Total	97	25.38	1.426	0.071 [b]	0.027 [a, b]	.	.
Lumbar (average)- VBH							
Unstressed	43	27.17	1.553				
Stressed	54	27.01	1.205				
Total	97	27.08	1.365	0.318	0.092 [b]	0.344	0.559
Thoracic (average) - VBH							
Unstressed	43	18.95	0.939				
Stressed	54	18.69	0.787				
Total	97	18.80	0.863	0.461	0.119	2.191	0.142

[a] Significant ($p < 0.05$)

[b] The assumptions of an ANOVA are violated where conditions of normality and homoscedasticity are not met.

Table 8-6. ANOVAs of vertebral body height measurements by Harris lines (HL)

	N	Mean	S.D.	Shapiro-Wilk	Levine's	F	*p*
Male							
L1 - VBH							
Unstressed	56	26.08	1.512				
Stressed	40	26.41	1.308				
Total	96	26.22	1.433	0.324	0.456	1.244	0.268
Lumbar (average)- VBH							
Unstressed	56	27.68	1.605				
Stressed	40	28.18	1.348				
Total	96	27.89	1.516	0.361	0.161	2.634	0.108
Thoracic (average) - VBH							
Unstressed	56	20.31	0.887				
Stressed	40	20.36	0.836				
Total	96	20.33	0.862	0.055 [b]	0.624	0.062	0.803
Female							
L1 - VBH							
Unstressed	56	25.40	1.555				
Stressed	31	25.43	1.202				
Total	87	25.41	1.432	0.071 [a]	0.222	0.006	0.939
Lumbar (average)- VBH							
Unstressed	56	27.07	1.484				
Stressed	31	27.18	1.131				
Total	87	27.11	1.363	0.318	0.254	0.116	0.734
Thoracic (average) - VBH							
Unstressed	56	18.85	0.855				
Stressed	31	18.78	0.971				
Total	87	18.83	0.893	0.461	0.678	0.152	0.697

[a] Near significant ($0.05 < p < 0.10$)

Table 8-7. Nonparametric tests of vertebral body height measurements by dental enamel defects (CBTS)

	N	Mean Rank	Sum of Ranks	Mann-Whitney U	Wilcoxon W	Z	*p* (2-tailed)
Male							
Thoracic (average) - VBH							
Unstressed	42	52.74	2215.0				
Stressed	65	54.82	3563.0	1312.0	2215.0	-0.338	0.735
Female							
L1 - VBH							
Unstressed	43	49.1	2111.5				
Stressed	54	48.92	2641.5	1156.5	2641.5	-0.033	0.974
Lumbar (average)- VBH							
Unstressed	43	49.74	2139.0				
Stressed	54	48.41	2614.0	1129.0	2614.0	-0.232	0.816

Table 8-8. Nonparametric tests of vertebral body height measurements by Harris lines (HL)

	N	Mean Rank	Sum of Ranks	Mann-Whitney U	Wilcoxon W	Z	*p* (2-tailed)
Male							
Thoracic (average) - VBH							
Unstressed	56	47.93	2684.0				
Stressed	40	49.30	1972.0	1088.0	2684.0	-0.238	0.812
Female							
L1 - VBH							
Unstressed	56	43.89	2458.0				
Stressed	31	44.19	1370.0	862.0	2458.0	-0.053	0.958

Table 8-9. Comparison of RMA regressions (RMA2) divided by dental enamel defects (CBTS)

	N	Intercept	Slope	95% CI lower	95% CI upper	Scale	t	df	*p*	Intercept Diffs.
Male										
L1 - anteroposterior										
Unstressed	41	-1.202	1.727	1.300	2.294	Pos.				
Stressed	65	-0.713	1.375	1.076	1.756	Pos.	1.235	50	0.111	0.549
L1 - mediolateral										
Unstressed	42	-0.637	1.391	1.035	1.870	Pos.				
Stressed	65	-0.612	1.375	1.074	1.760	Pos.	0.062	51	0.475	0.552
Avg. thoracic - anteroposterior										
Unstressed	42	-1.358	1.947	1.435	2.643	Pos.				
Stressed	63	-0.825	1.526	1.191	1.955	Pos.	1.257	50	0.107	0.168
Avg. thoracic - mediolateral										
Unstressed	42	-0.961	1.684	1.240	2.288	Pos.				
Stressed	63	-1.106	1.789	1.417	2.260	Pos.	0.319	50	0.375	0.047 [a]
Avg. lumbar - anteroposterior										
Unstressed	41	-1.401	1.826	1.359	2.453	Pos.				
Stressed	65	-1.421	1.826	1.431	2.330	Pos.	0.000	50	0.500	0.076 [b]
Avg. lumbar - mediolateral										
Unstressed	42	-0.698	1.435	1.072	1.921	Pos.				
Stressed	65	-0.583	1.348	1.051	1.729	Pos.	0.331	51	0.371	0.117
Female										
L1 - anteroposterior										
Unstressed	43	-0.358	1.138	0.858	1.509	Iso.				
Stressed	54	-1.239	1.762	1.350	2.299	Pos.	2.290	50	0.013 [a]	.
L1 - mediolateral										
Unstressed	43	-0.731	1.448	1.111	1.888	Pos.				
Stressed	54	-0.701	1.429	1.088	1.876	Pos.	0.073	50	0.471	0.539
Avg. thoracic - anteroposterior										
Unstressed	43	-0.710	1.476	1.126	1.936	Pos.				
Stressed	53	-1.019	1.722	1.305	2.273	Pos.	0.808	50	0.212	1.000
Avg. thoracic - mediolateral										
Unstressed	43	-0.801	1.571	1.182	2.089	Pos.				
Stressed	52	-0.801	1.579	1.193	2.089	Pos.	0.024	49	0.490	0.680
Avg. lumbar - anteroposterior										
Unstressed	43	-0.905	1.480	1.103	1.986	Pos.				
Stressed	54	-1.875	2.158	1.642	2.837	Pos.	1.906	49	0.031 [a]	.
Avg. lumbar - mediolateral										
Unstressed	43	-0.776	1.480	1.099	1.993	Pos.				
Stressed	54	-0.995	1.632	1.245	2.138	Pos.	0.492	49	0.312	0.306

[a] Significant ($p < 0.05$)

[b] Near significant ($0.05 < p < 0.10$)

Table 8-10. Comparison of RMA regressions (RMA2) divided by Harris lines (HL)

	N	Intercept	Slope	95% CI lower	95% CI upper	Scale	t	df	*p*	Intercept Diffs.
Male										
L1 - anteroposterior										
Unstressed	55	-0.795	1.440	1.111	1.867	Pos.				
Stressed	40	-1.196	1.714	1.283	2.291	Pos.	0.912	47	0.183	0.405
L1 - mediolateral										
Unstressed	56	-0.645	1.400	1.070	1.831	Pos.				
Stressed	40	-0.696	1.429	1.035	1.971	Pos.	0.098	47	0.461	0.100
Avg. thoracic - anteroposterior										
Unstressed	54	-1.021	1.684	1.286	2.206	Pos.				
Stressed	40	-1.441	2.000	1.465	2.730	Pos.	0.848	47	0.200	0.538
Avg. thoracic - mediolateral										
Unstressed	55	-1.172	1.842	1.427	2.378	Pos.				
Stressed	39	-1.089	1.778	1.297	2.437	Pos.	0.178	46	0.430	0.093 [b]
Avg. lumbar - anteroposterior										
Unstressed	55	-1.192	1.680	1.294	2.181	Pos.				
Stressed	40	-1.812	2.095	1.548	2.836	Pos.	1.124	47	0.133	0.147
Avg. lumbar - mediolateral										
Unstressed	56	-0.533	1.320	1.015	1.717	Pos.				
Stressed	40	-0.910	1.571	1.148	2.151	Pos.	0.865	47	0.196	0.147
Female										
L1 - anteroposterior										
Unstressed	41	-0.792	1.444	1.115	1.871	Pos.				
Stressed	65	-0.728	1.400	1.014	1.933	Pos.	0.155	39	0.439	1.000
L1 - mediolateral										
Unstressed	42	-0.496	1.286	1.000	1.654	Iso.				
Stressed	65	-1.094	1.700	1.273	2.270	Pos.	1.499	40	0.071 [b]	0.502
Avg. thoracic - anteroposterior										
Unstressed	42	-0.866	1.600	1.222	2.095	Pos.				
Stressed	63	-0.568	1.364	0.987	1.885	Iso.	0.779	40	0.220	0.377
Avg. thoracic - mediolateral										
Unstressed	42	-0.765	1.550	1.185	2.028	Pos.				
Stressed	63	-0.411	1.261	0.886	1.795	Iso.	0.956	38	0.173	0.041 [a]
Avg. lumbar - anteroposterior										
Unstressed	41	-1.348	1.792	1.375	2.334	Pos.				
Stressed	65	-1.421	1.833	1.284	2.617	Pos.	0.106	39	0.458	0.507
Avg. lumbar - mediolateral										
Unstressed	42	-0.558	1.333	1.024	1.736	Pos.				
Stressed	65	-1.297	1.833	1.325	2.537	Pos.	1.563	39	0.063 [b]	0.042 [a]

[a] Significant ($p < 0.05$)

[b] Near significant ($0.05 < p < 0.10$)

Table 8-11. Correlations between vertebral neural canal measures and skeletal height (SKH)

		Average thoracic - AP	L1 - AP	Average lumbar - AP
Combined Sex				
	N	201	204	203
	Pearson Correlation	0.291	0.300	0.287
	Sig. (2-tailed)	0.000 [a]	0.000 [a]	0.000 [a]
	Kendall's tau	0.192	0.190	0.182
	Sig. (2-tailed)	0.000 [a]	0.000 [a]	0.000 [a]
Female				
	N	96	97	97
	Pearson Correlation	0.308	0.350	0.165
	Sig. (2-tailed)	0.002 [a]	0.000 [a]	0.107
	Kendall's tau	0.183	0.211	0.070
	Sig. (2-tailed)	0.008 [a]	0.002 [a]	0.307
Male				
	N	105	106	106
	Pearson Correlation	0.323	0.349	0.361
	Sig. (2-tailed)	0.001 [a]	0.000 [a]	0.000 [a]
	Kendall's tau	0.224	0.231	0.263
	Sig. (2-tailed)	0.001 [a]	0.000 [a]	0.000 [a]

[a] Significant ($p < 0.05$)

Table 8-12. Correlations between head circumference and skeletal height

Collection	Sex	Correlation coefficient [a]	p
Terry	Male	0.345	0.011 [b]
	Female	0.270	0.028 [b]
Hamann-Todd	Male	0.082	0.631
	Female	0.187	0.097

[a] Pearson's correlations were performed in all cases except the Hamann-Todd females. Because head circumference measures among Hamann-Todd females were non-parametric, Kendall's tau correlations were used.
[b] Significant ($p < 0.05$)

Table 8-13. ANOVAs of head circumference measurements by traditional stress markers

	N	Mean	S.D.	Shapiro-Wilk	Levine's	F	*p*
Divided by dental enamel defects (CBTS)							
Female							
Unstressed	29	510.66	14.659				
Stressed	25	513.00	17.682				
Total	54	511.74	16.016	0.587	0.705	0.284	0.596
Male							
Unstressed	28	526.14	17.016				
Stressed	38	529.32	15.141				
Total	66	527.97	15.914	0.053	0.997	0.637	0.428
Divided by Harris lines							
Female							
Unstressed	33	509.64	12.180				
Stressed	20	514.55	20.985				
Total	53	511.49	16.062	0.587	0.009 [a, b]	.	.
Male							
Unstressed	37	529.49	14.521				
Stressed	28	525.71	17.841				
Total	65	527.86	16.014	0.053	0.203	0.883	0.351

[a] Significant ($p < 0.05$)

[b] The assumptions of an ANOVA are violated where conditions of normality and homoscedasticity are not met.

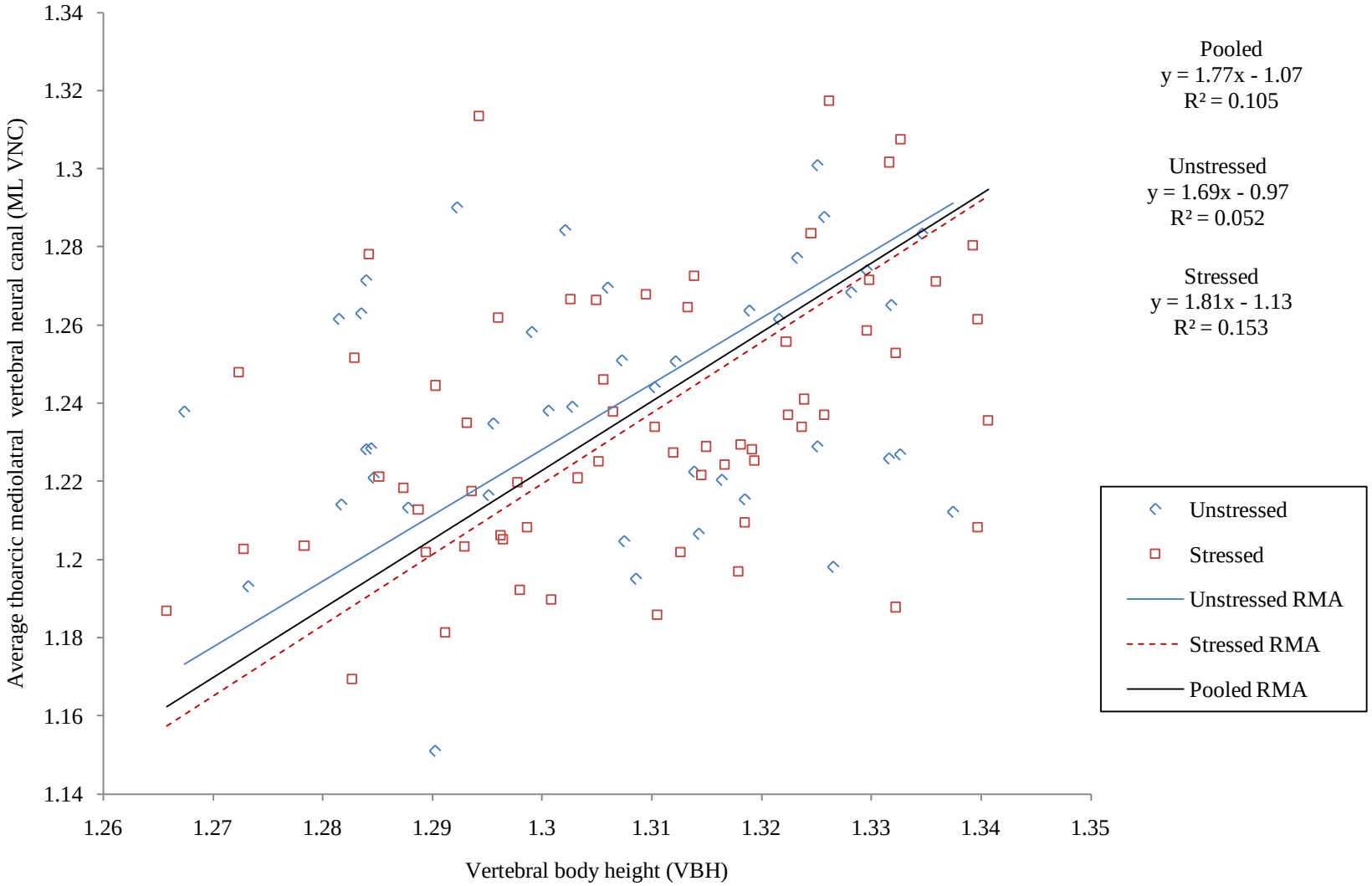


Figure 8-1. Reduced major axis regressions of mediolateral vertebral neural canal diameters of the averaged thoracic vertebrae over vertebral body heights in males divided by enamel defects (CBTS).

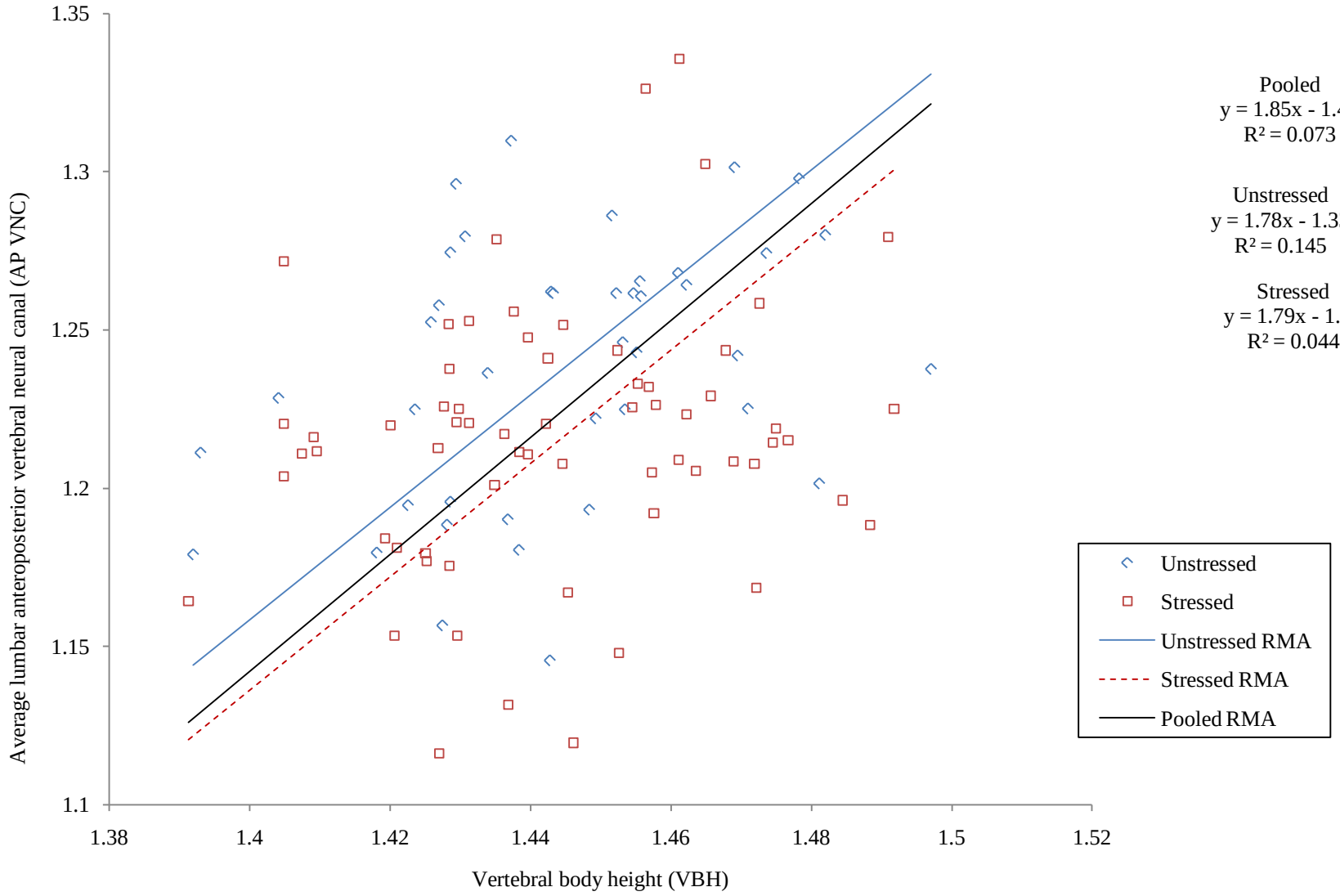


Figure 8-2. Reduced major axis regressions of anteroposterior vertebral neural canal diameters of the averaged lumbar vertebrae over vertebral body heights in males divided by enamel defects (CBTS).

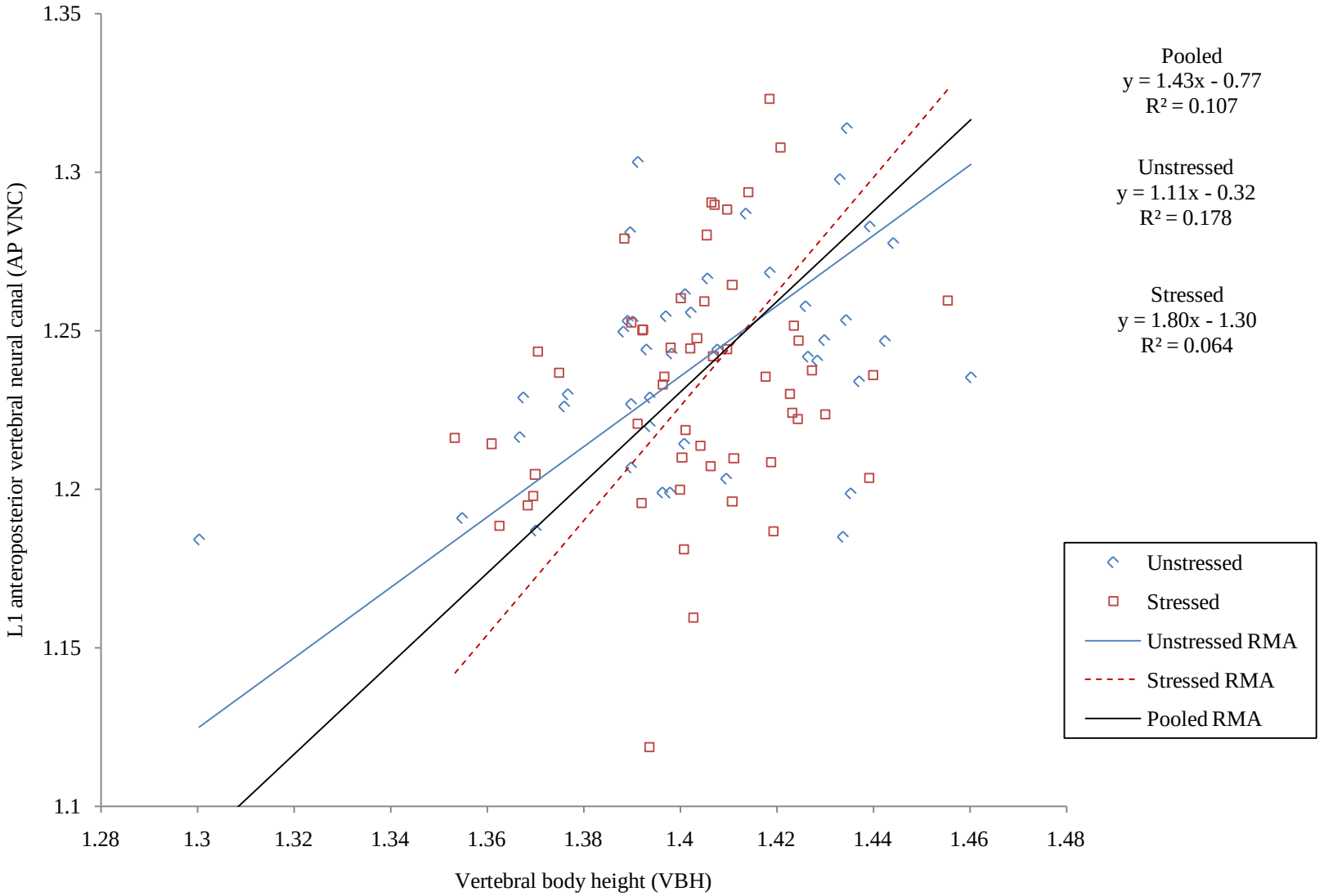


Figure 8-3. Reduced major axis regressions of anteroposterior vertebral neural canal diameters of L1 over vertebral body heights in females divided by enamel defects (CBTS).

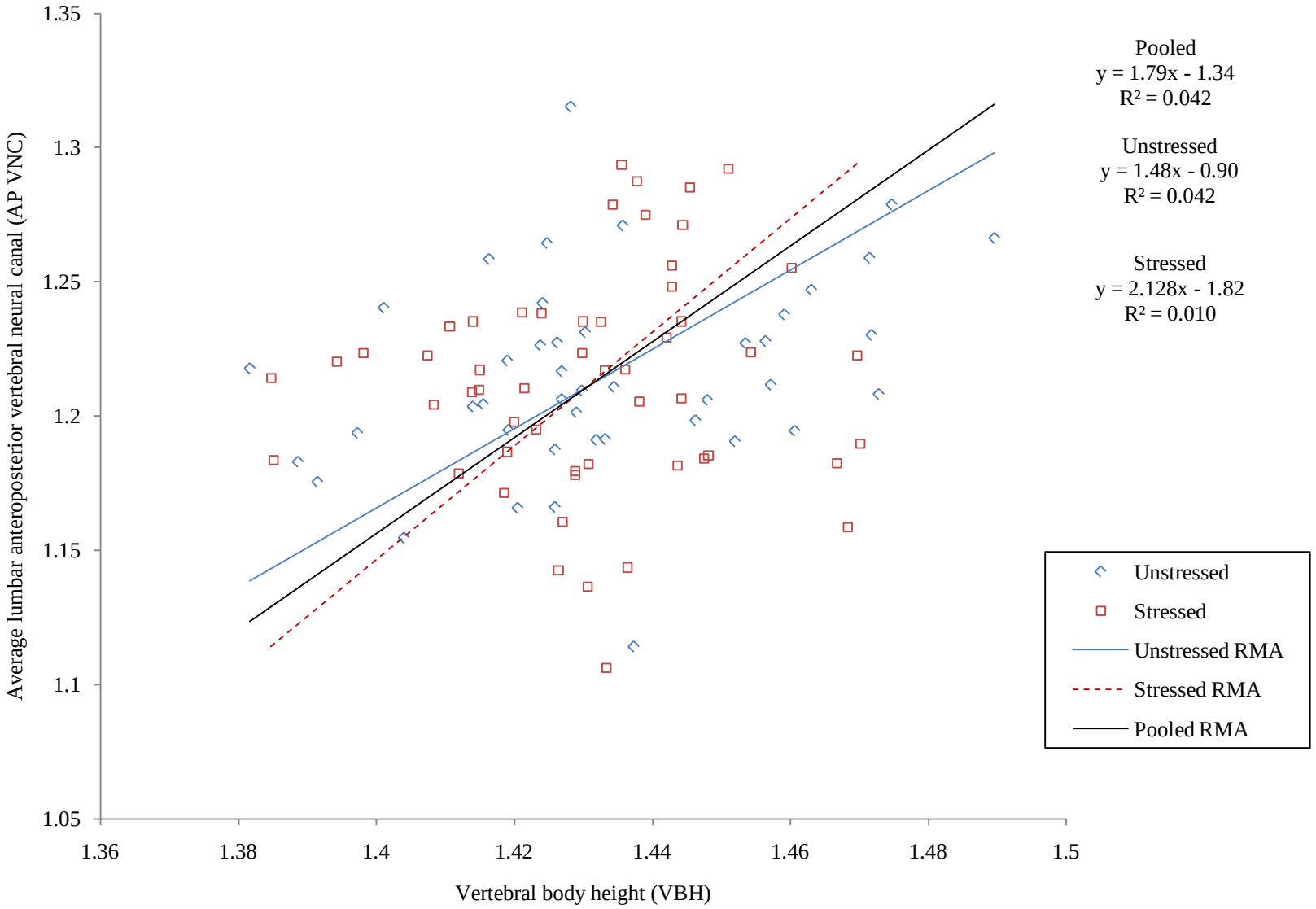


Figure 8-4. Reduced major axis regressions of transverse vertebral neural canal diameters of the averaged lumbar vertebrae over vertebral body heights in females divided by enamel defects (CBTS).

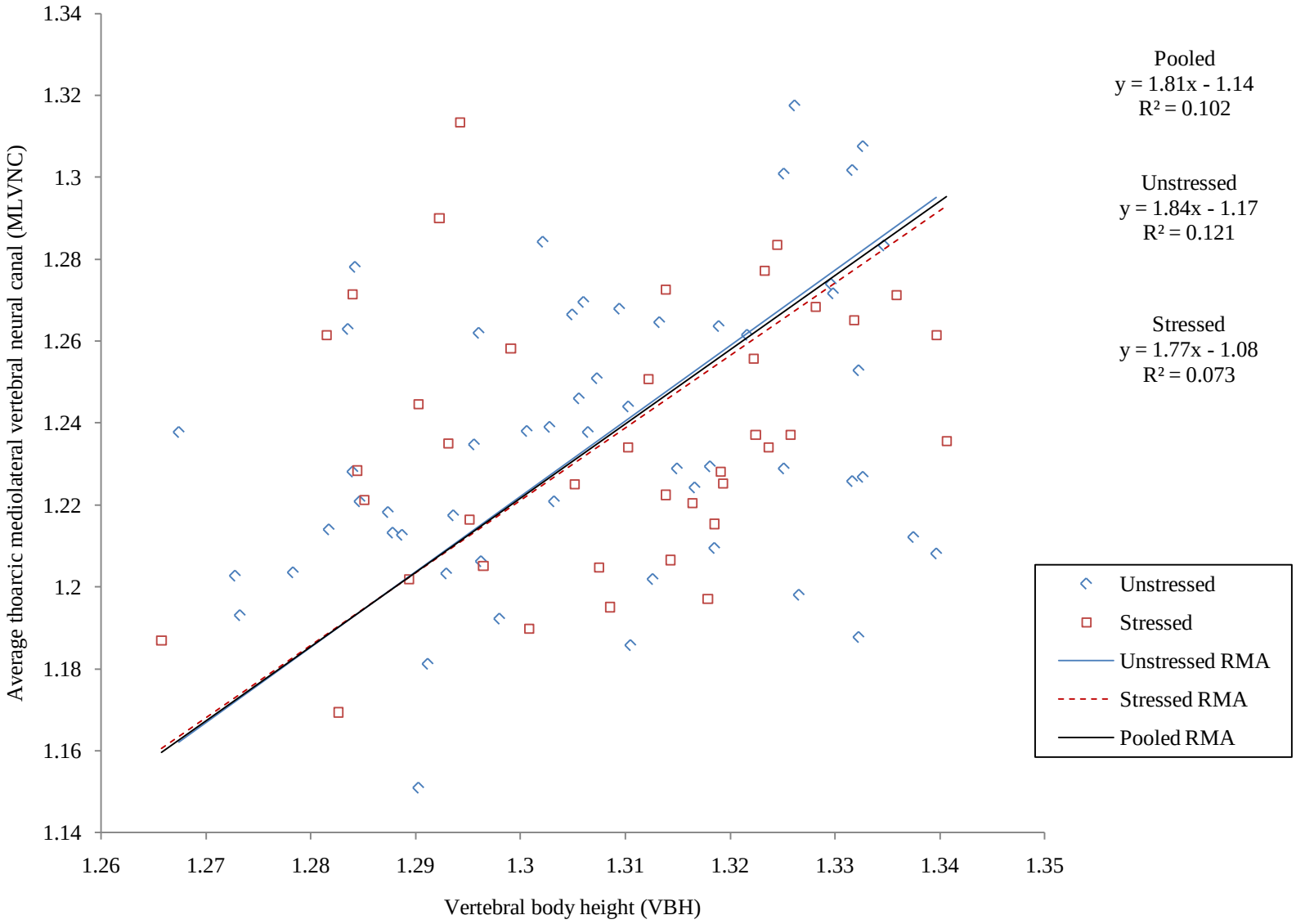


Figure 8-5. Reduced major axis regressions of mediolateral vertebral neural canal diameters of the averaged thoracic vertebrae over vertebral body heights in males divided by Harris lines (HL).

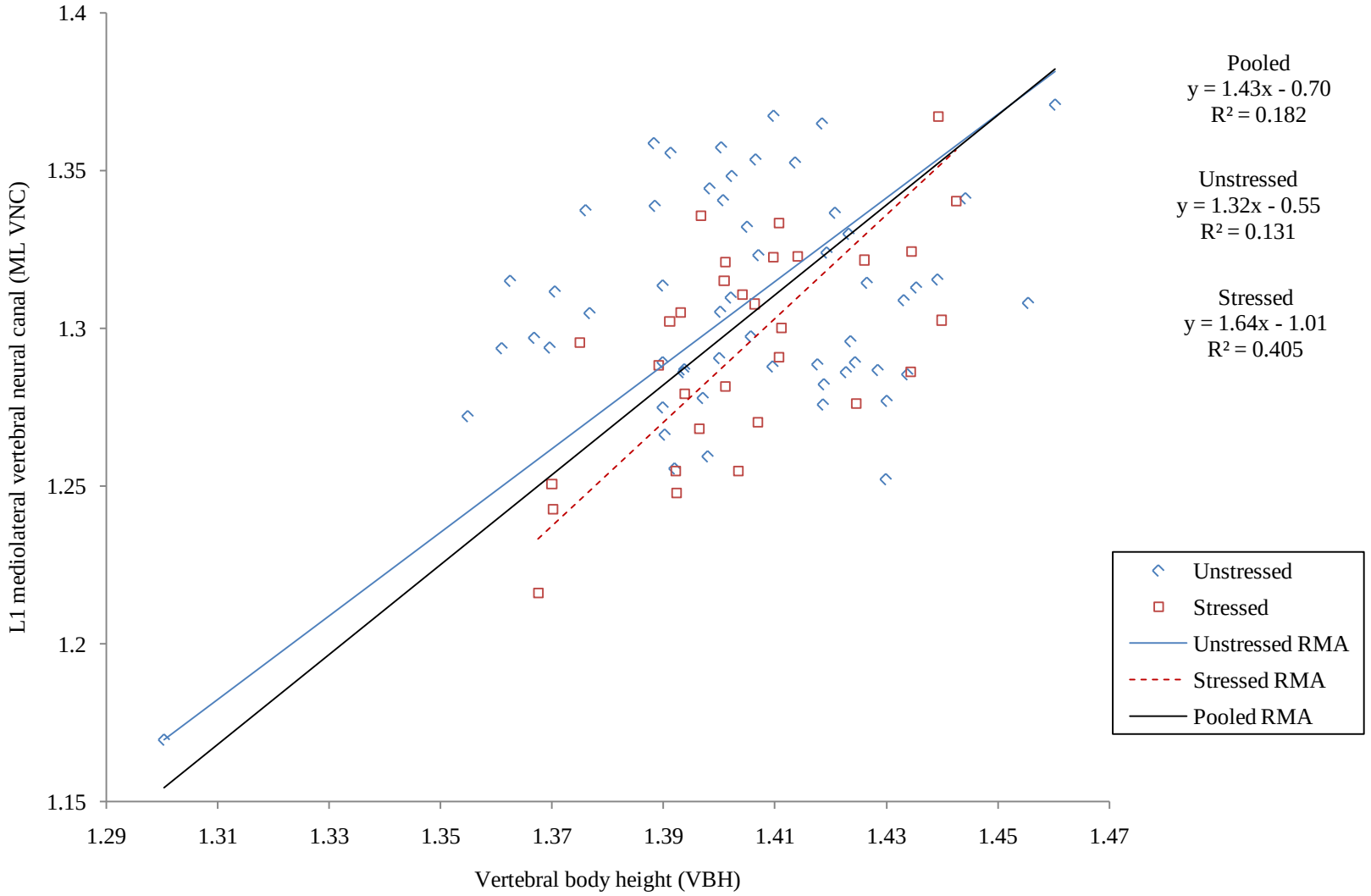


Figure 8-6. Reduced major axis regressions of mediolateral vertebral neural canal diameters of L1 over vertebral body heights in females divided by Harris lines (HL).

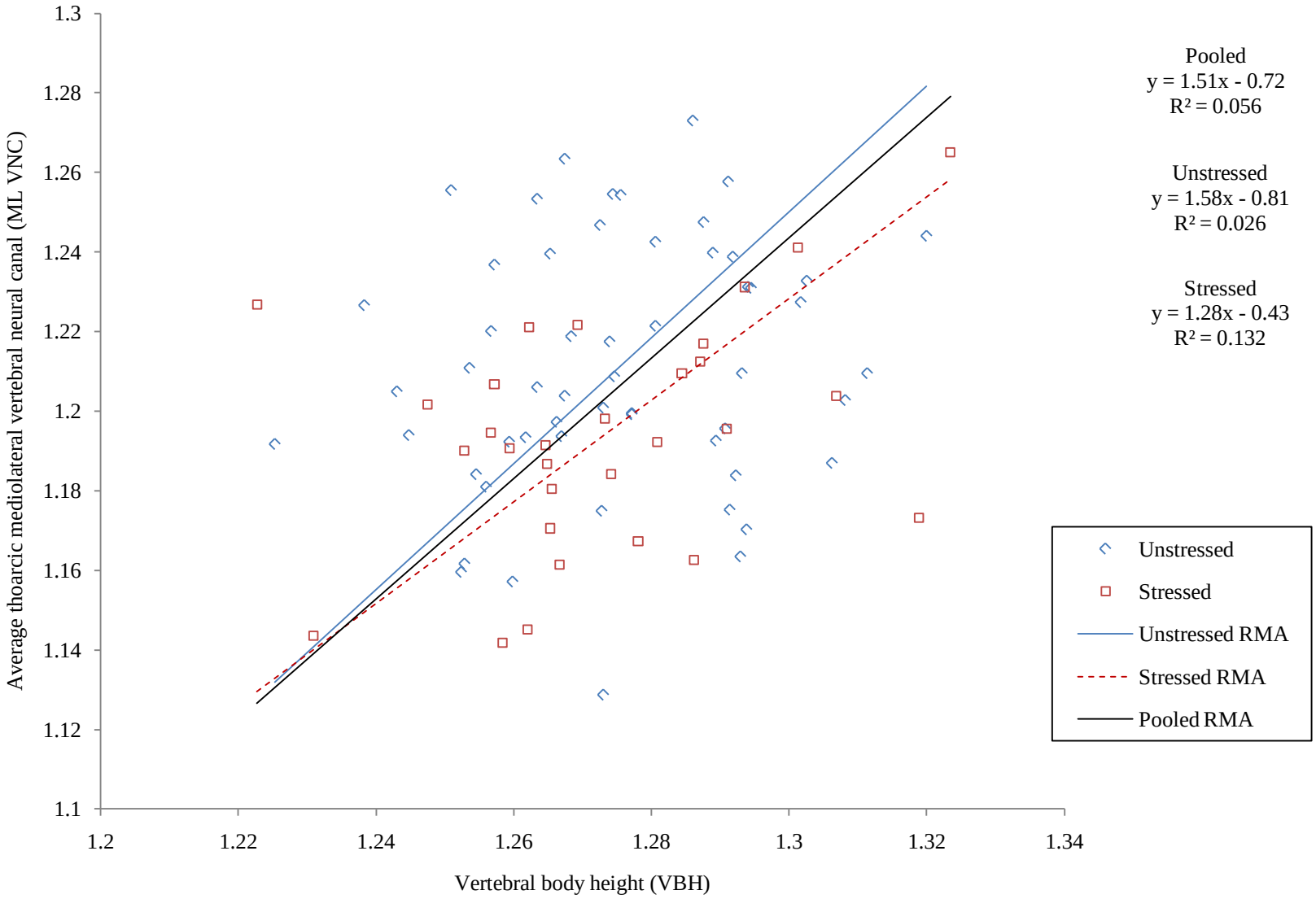


Figure 8-7. Reduced major axis regressions of transverse vertebral neural canal diameters of the averaged thoracic vertebrae over vertebral body heights in females divided by Harris lines (HL).

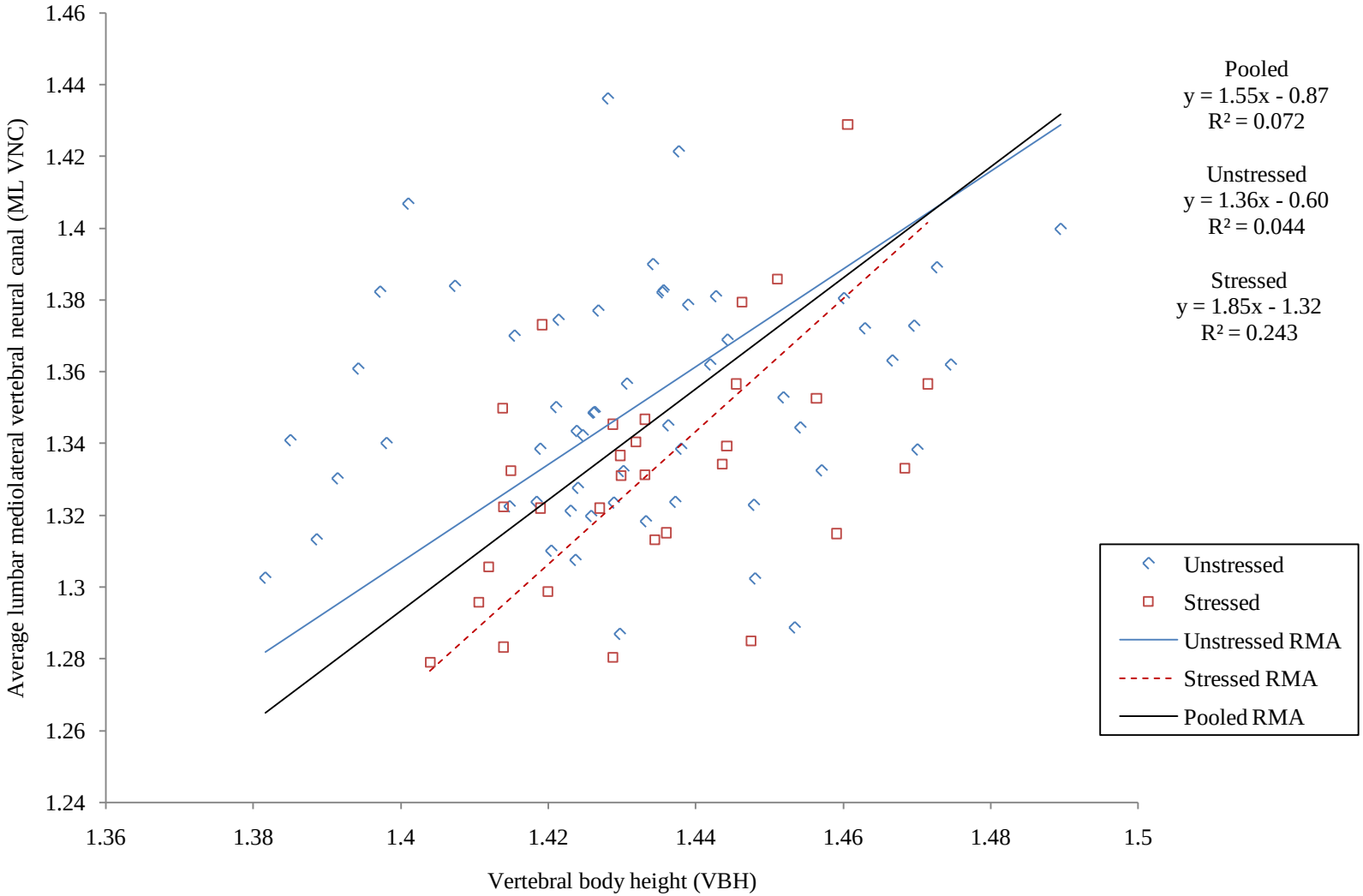


Figure 8-8. Reduced major axis regressions of mediolateral vertebral neural canal diameters of the averaged lumbar vertebrae over vertebral body heights in females divided by Harris lines (HL).

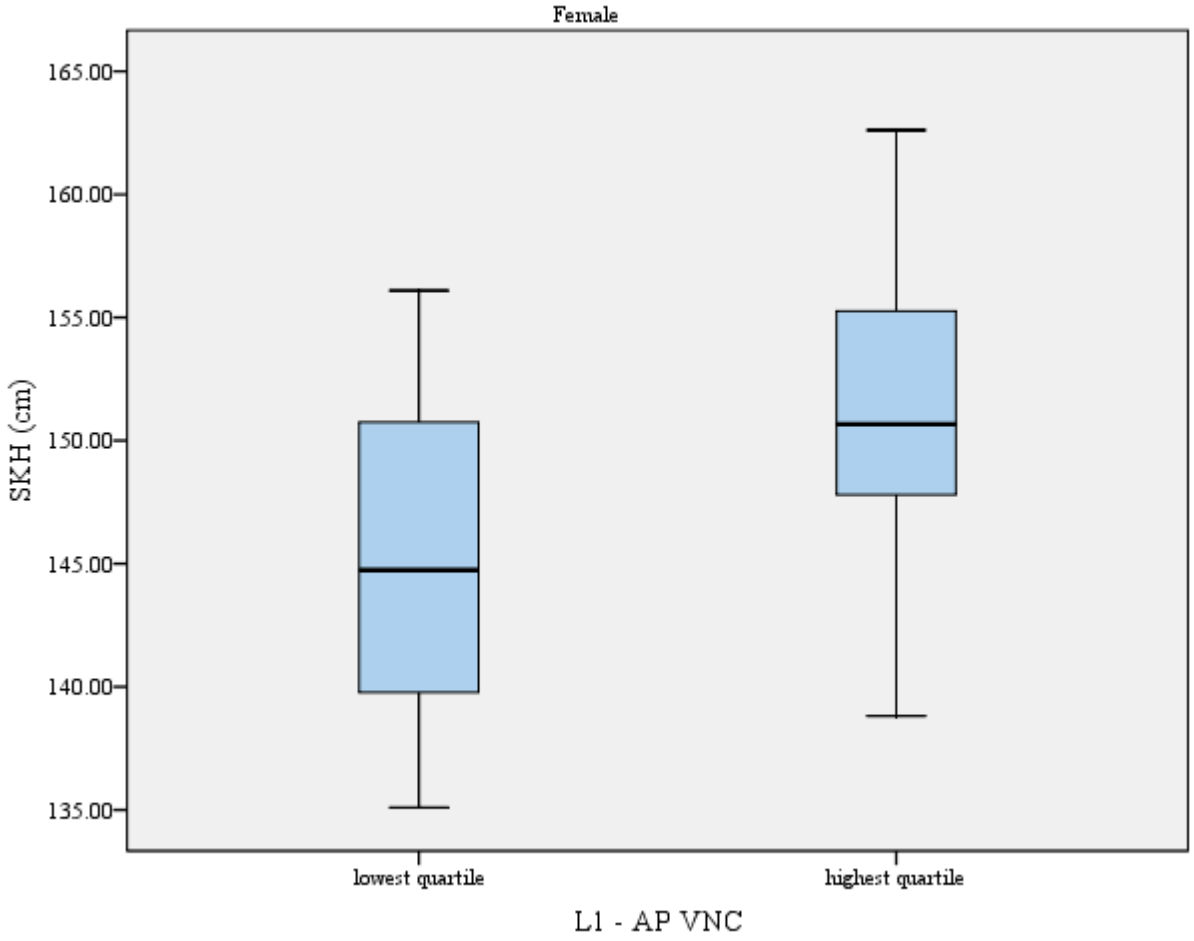


Figure 8-9. Box plots of female skeletal height (SKH) divided by highest and lowest quartiles of vertebral neural canal anteroposterior diameters.

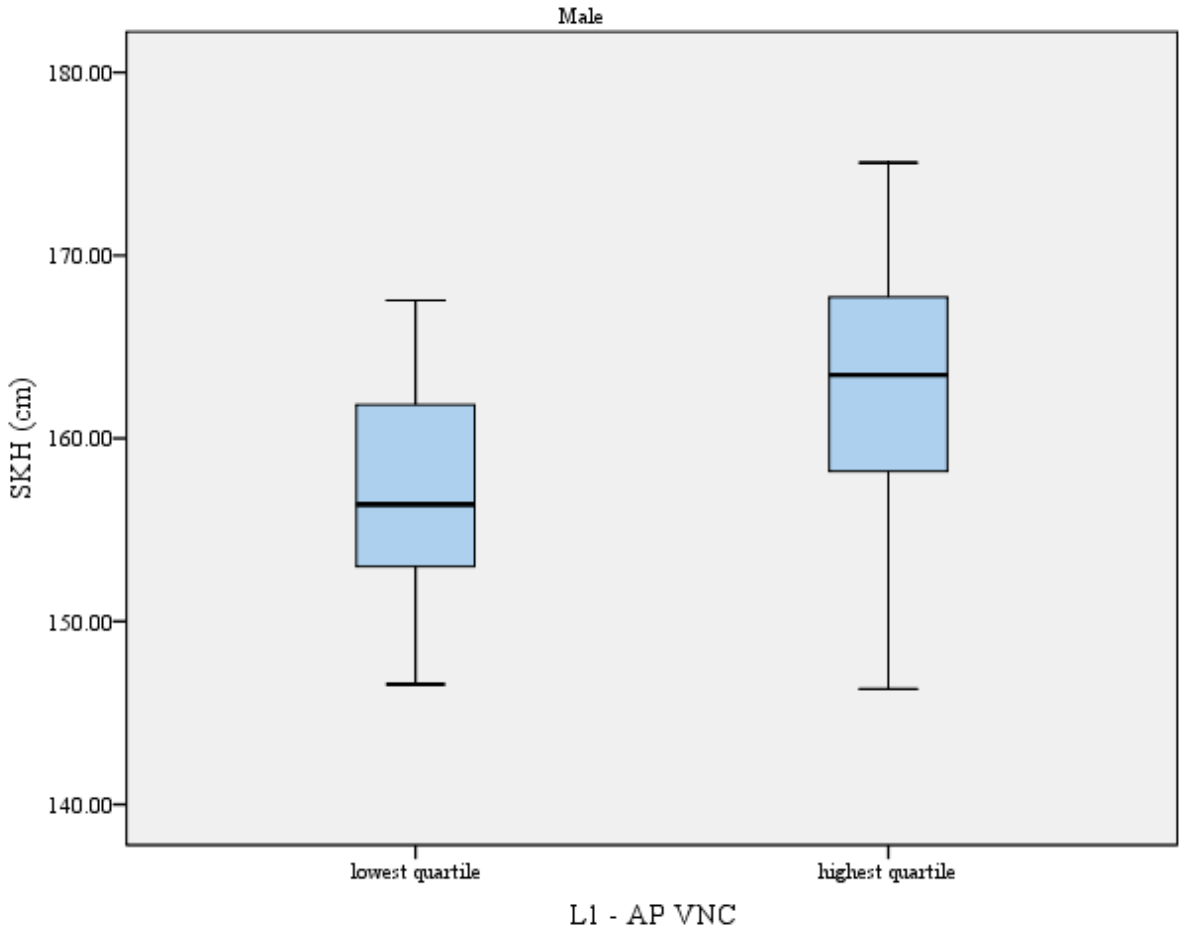


Figure 8-10. Box plots of male skeletal height (SKH) divided by highest and lowest quartiles of vertebral neural canal anteroposterior diameters.

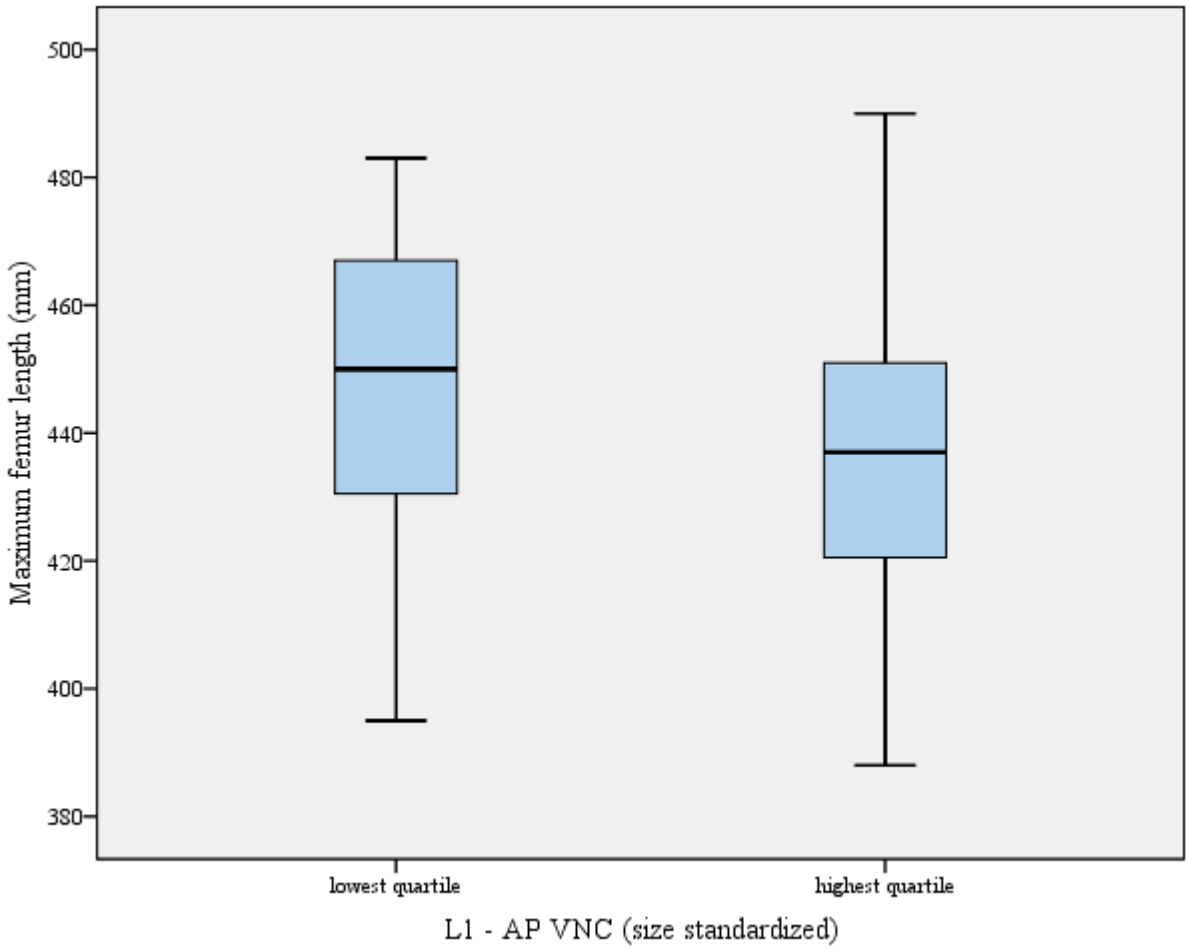


Figure 8-11. Box plots of female femur length divided by highest and lowest quartiles of vertebral neural canal anteroposterior diameters size standardized using vertebral body heights.

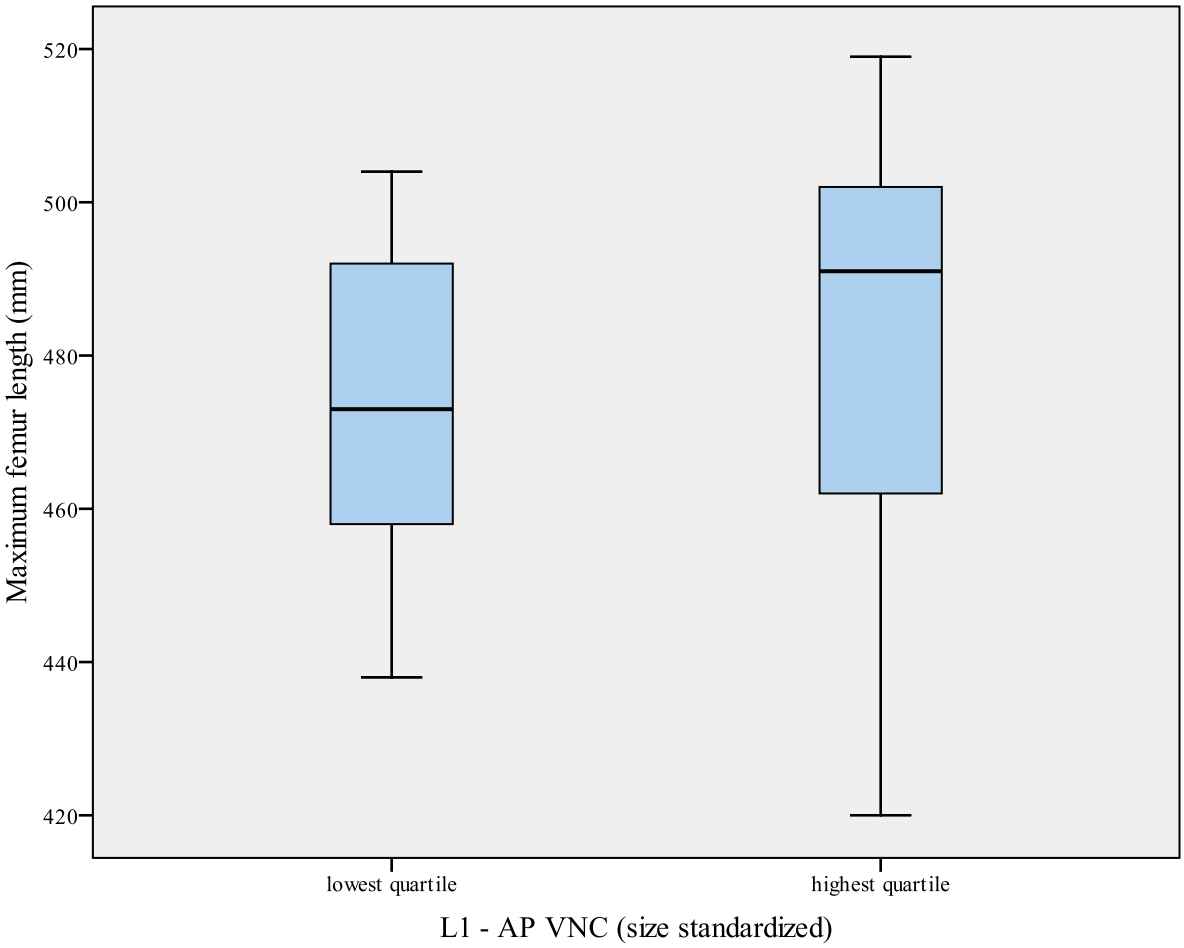


Figure 8-12. Box plots of male maximum femur length divided by highest and lowest quartiles of vertebral neural canal anteroposterior diameters size standardized using vertebral body heights.

CHAPTER 9
DISCUSSION

This study uses the presence or absence of indicators of growth arrest as an independent variable for comparison with skeletal measurements to determine whether or not the observable stress markers have any association with the skeletal morphology of adults. The null hypothesis is that there are no metrically observable difference between the size and shape of individuals who have experienced growth disruption to those who have not, due to the corrective process of catch-up growth. Three alternative hypotheses were tested with this research:

H_1: Individuals with independent indicators of growth disruption or stress have a smaller estimated stature than those individuals without such indicators of stress.

H_2: Because female growth is believed to be canalized, or less affected by stress conditions than male growth, males and females with evidence of stress have a lesser degree of sexual dimorphism than those without any evidence of stress.

H_3: Proportional differences are observable between those individuals with independent evidence of growth disruption and those without.

Stature

When the osteometric data associated with stature estimates were divided by the presence or absence of Harris lines to address the first alternative hypothesis, no significant difference was found. However, when dental defects (CBTS) were used as the stress indicator, support was found for the first alternative hypothesis in the case of females, but not in the case of males. When dental enamel defects were coded by the TS criterion (any enamel defect) or LEH criterion (any linear defect) the results were significant for females in sitting height (STH) and tibia length.

Support for the first alternative hypothesis suggests that catch-up growth following growth disruption is not always complete. We can therefore reject the null hypothesis as it applies to females, but not as it applies to the male sample. The fact that significant differences are found

with females, but not with males is of particular interest because it is the opposite of expectations based on how males and females are believed to respond to stress. Since females have greater fat and nutrient reserves, thought to be an adaptation for the increased demands of lactation and gestation, many researchers have hypothesized that they account for a canalized growth trajectory in females. Due to this physiological difference between the sexes, males are thought to experience a greater reduction in lean body mass than females during periods of stress, and reduced body mass is accompanied by reduced skeletal growth during severe stress events (Stini, 1975a). In considering the physiological differences between the sexes it is also important to note that Guatelli-Steinberg (1999) found that males and females are equally likely to form enamel hypoplasias. The observed frequency of Harris lines between the sexes vary among archaeological populations (e.g. Goodman and Clark, 1981; Hummert and Van Gerven, 1985; Martin, 1985; Rathbun, 1987).

The relationship between stature and stress can be interpreted several ways. The first explanation I will consider does not challenge prior notions of sex differentiated responses to growth disruption; instead, I hypothesize that a sex difference in parental investment may favor male children over female children, assisting the process of catch-up growth in males. Culturally, males may be better attended. In situations where all may suffer, male children may recover sooner or more completely. In research on the demographics of natural disasters and famines, females appear to have some biological advantage over their male counterparts (Macintyre, 2002); however, there are many famines in which females die at a higher rate during infancy and childhood (Agarwal, 1990; Dyson, 1991a, 1991b; Kidane, 1989; Mariam, 1986). In famines, it has been suggested that the most feasible explanation for this trend is that female babies and children have less access to food than male children (Neumayer and Plümper, 2007).

In other natural disasters, such as a flood, male success over females may be attributed to the survival skills that are more often taught to male children such as tree climbing, running and swimming (Neumayer and Plümper, 2007).

Whether or not preferential feeding of male children during times of stress characterized African-Americans during the 1880's to 1930's is a question that could likely be addressed through a historical investigation, but there are also anthropological studies which may be informative. Ethnographic studies show that the quality of parental investment varies depending on the gender of the offspring. The Trivers-Willard hypothesis states that parents should invest more in the offspring with the greatest reproductive variance. Because the variance in reproductive success is generally greater for human males than females, parents who can afford to invest in their offspring have a potentially greater return by investing in males. On the other hand, a parent in poor condition may see a greater return (in terms of reproductive success in the next generation) by investing in female offspring (Hrdy, 1990; Trivers and Willard, 1973). Because the skeletal remains used in this research were of Americans of African descentfrom the early 1900's, most of whom were unclaimed by family members or given to the state at death (Hunt and Albanese, 2005), it is fair to assume that the majority of these individuals were from a low socioeconomic background; according to the Trivers-Willard model, this scenario would favor female offspring. The effect suggested by the Trivers-Willard hypothesis may, however, be most pronounced in non-industrialized area of the world; Keller et al. (2001) found no support for this hypothesis in a recent study of the modern United States. They point out that any investment in male children may be beneficial in terms of reproductive success, thus counteracting the effect suggested by Trivers and Willard. Clutton-Brock (1991) points out that children's work, rather than their direct reproductive success, may play the biggest role in the

inclusive fitness of parents. If males are contributing more to the subsistence economy, it may be more adaptive to invest in male offspring who will help both parents and siblings. Research shows that female juvenile mortality is higher in areas of the world where females contribute little to the subsistence economy (Arnold and Zhaoxiang, 1986; Baigari, 1986; Das Gupta, 1987), and male juvenile mortality is higher than expected where the female contribution is high (Cronk, 1989; Harpending and Pennington, 1991).

A second explanation for why only female growth disruption is associated with reduced stature is that it actually may be an example of female physiological buffering at its finest. The only individuals sampled in this study were adults between the ages of 19 and 35 years at death. If juvenile mortality rates were higher for males than females, the "stressed" females in this study may represent a wider range of stress severity than is observed in adult males. Wood et al. (1992) refer to this type of problem as the "osteological paradox." Differential mortality can affect the demography of a skeletal sample, and the subsequent interpretation of data from those samples. To address the osteological paradox in this study, it would be valuable to analyze historical documents to discern whether or not a sex difference in juvenile mortality existed during this time period.

A third hypothesis for explaining the results of how stress and stature are related in males and females questions the core of the theory regarding physiological differences in stress response between the sexes. The typical explanation for the physiological stress buffering in females is always related to fat stores for the demands of lactation and gestation (Rivers, 1982; Stini, 1969). Greulich's (1976) study of secular changes among males and females remarked on the "biological superiority" of females, but also found that females were more variable than males in most morphological characteristics. While females do have the ability to retain fat, this

fact seems less relevant during early growth than it is once an individual reaches puberty. Female children simply do not store fat in the way that female adults do. Owens (2002) found that in the United States, males are twice as likely to die from parasitic infections as females. In some areas of the world they are nearly four times as vulnerable as females. However, this trend does not begin until men reach their mid-twenties and may be hormonally related to the immunosuppressant nature of testosterone or may be due simply to exposure risk. While male children are believed to be more vulnerable than their female counterparts, Owens (2002) reports that large differences between male and female mortality rates do not begin until after puberty. Other research reports higher infant mortality in males as well as greater male vulnerability during infancy, while admitting that causal factors are poorly understood (Waldron, 1983). When female mortality is higher, a cultural bias against female children is assumed to be the cause (Holden and Mace, 1999; Neumayer and Plümper, 2007; Waldron, 1983). The majority of "stressed" individuals used in this study developed stress markers in infancy (between birth and 3 years of age) – a time period where lactation and gestation are hardly an issue. Research needs to focus on whether or not a sex differentiated vulnerability to disease and malnutrition exists during the early juvenile years - independently of factors such as accidents and parental investment biases. If such differences exist during the growth period, what are the true causal factors? Physiological differences may only become critical in the later years of development as sex hormones begin to play a role.

Sexual Dimorphism

Resampling statistics were used to test the second alternative hypothesis – that sexual dimorphism is greater in unstressed than stressed groups. No support was found for the alternative hypothesis. This hypothesis was formed on the premise that females have greater resistance to environmental stress than their male counterparts; "stressed" males and females

would be expected to exhibit less sexual dimorphism due to the fact that female size is believed to be canalized while male size varies more with environmental conditions (Stini, 1969, 1985).

Not only were there no significant differences in sexual dimorphism scores between groups, but the differences based on enamel stress markers were in the opposite direction of expectations – raw scores of dimorphism (calculated as $\ln \bar{X}_M - \ln \bar{X}_F$) were greater in the stressed group. The greatest differences in dimorphism, although insignificant at $p < 0.05$, were for tibial ($p = 0.064$) and radial ($p = 0.061$) length. While it is interesting that both these measures are for distal limb elements (see discussion pertaining to proportions), the results are inconsistent. For tibial length, males have slightly longer tibiae among the stressed group and females have slightly longer tibiae in the unstressed group. For radial length, both males and females have slightly larger radii in the unstressed group.

When Harris lines were used as the marker to differentiate stressed and unstressed groups, no compelling results were found. The direction of dimorphism was not consistent and the *p* values found by resampling averaged 0.395 suggesting that the differences observed were not meaningful. Harris line data as it relates to sexual dimorphism could be interpreted two ways: 1) Harris lines are reliable markers of stress and the *p* values found suggest that sexual dimorphism is not different between stressed and unstressed groups, or 2) Harris lines are not a good indicator of stress given that they do not pick up any differences in dimorphism. Given other concerns with Harris lines (see discussion on the reliability of stress markers), it is reasonable to conclude that Harris line data does not contribute to understanding the relationship between sexual dimorphism and stress.

A corollary to the alternative hypothesis regarding dimorphism is that female size should be similar in stressed and unstressed groups while male size should be different. The results

from Chapter 5 found this to be untrue. When dental defects were used as the stress marker, female measurements are significantly larger among the unstressed while male differences are not significant. Furthermore, because stressed females are shorter, dimorphism is actually greater in stressed populations, although not significantly so.

These findings challenge the hypothesis that sexual dimorphism is greater in unstressed groups as well as the theory that female growth is more canalized than in males. There are a few possible interpretations for why no support was found for either the null or alternative hypotheses: 1) female growth is not more canalized than males and therefore dimorphism should not follow the pattern of the alternative hypothesis, 2) cultural buffering favors male children to a greater extent than biological buffering favors females, or 3) the alternative hypothesis is correct, but is not observed due to greater male mortality. Each of these explanations are explored in the discussion of stature differences.

Proportions

The position that body proportions are more heritable than body size (Mueller 1986; Stini 1975) has been challenged by numerous researchers over the past decade who have found that better environmental circumstances lead to longer relative lower limb length (Bogin and Rios, 2003; Bogin et al., 2002; Dangour, 2001; Floyd, 2007, 2008; Fredriks et al., 2005; Frisancho et al., 2001; Li et al., 2007; Malina et al., 2004; Stinson, 2000; Wadsworth et al., 2002) and that the relationship of proximal and distal limb elements is associated with environmental effects. The length of the tibia has proven to be more variable than other limb segments, distal elements tend to be more variable than proximal limb segments (Holliday and Ruff, 2001; Smith and Buschang, 2004), and secular allometric trends have shown that as the environment improves, the relative length of the tibia increases (Meadows Jantz and Jantz 1999; Jantz and Owsley 1984; Meadows and Jantz 1995). Furthermore, because nutritional stress and growth disturbances are

greatest in the first two to three years of life, age of an insult may affect its influence (Beaton et al., 1990; Checkley et al., 1998; Martorell and Habicht, 1986; Martorell et al., 1995).

The results from research on human proportions is what influenced the third alternative hypothesis, that proportional differences would be observable between those with and without indicators of growth disruption. Support for this alternative hypothesis is weak at best. To investigate how stress affected proportions, ANOVAs were performed on common proportional indices (crural index, humerofemoral index, radiohumeral index, sitting height index and intermembral index) to see whether or not these proportions differed based on the presence or absence of stress markers. None of these ANOVAs yielded significant results; however, near significant results were retested using a resampling procedure which yielded a significant result in the case of the male crural index divided by the presence or absence of enamel defects. Interestingly, the observed difference was in the opposite direction of that proposed by the alternative hypothesis. In this study, the crural index was found to be higher among the stressed group. Maximum tibia length was higher and the maximum femur length was lower among the stressed than among the unstressed.

Because proportional indices are ratios and the components of the ratios are not independent, scaling procedures were conducted to qualify the results. Significant differences in scaling were found in male humerofemoral and intermembral indices when the skeletal sample was divided according to presence or absence of enamel defects (CBTS). Figures 7-8 and 7-11 graph these scaling differences where femur (or lower limb) length increases at a slower rate than humerus (or upper limb) length in the stressed group. Significant differences in scaling were found in female humerofemoral indices and intermembral indices when the population was divided according to the presence, absence, and number of Harris lines. Figures 7-23 and 7-26

graph these scaling differences where femur (or lower limb) length increases at a faster rate than humerus (or upper limb) length in the stressed group. These scaling differences are significant, but because the ANOVAs are not, the scaling effects are considered to be subtle. No significant scaling differences were observed for male crural indices where significant differences were found between stressed and unstressed groups.

Proportional indices were also analyzed to see if the number of stress events could be associated with significant differences in proportions. The majority of these comparisons were not significant. Although significant differences in intermembral indices were found between male individuals with no Harris lines and those with only one Harris line, when groups with no Harris lines and those with two Harris lines were compared, there were no significant differences. Similarly, when intermembral indices in those males with one Harris line and those with more than one Harris line were compared, no significant difference was found. Observation of the mean values for these indices do not allow for a simple explanation of these results and suggest that this may be a Type I statistical error (rejection of the null hypothesis when the null hypothesis is true).

In most cases, proportional differences were not significantly associated with the age at which a stress event occurred. The two exceptions are for the radiohumeral index among females and the crural index in males when the sample was divided into stress categories based on the presence or absence of enamel defects. Within the three age groupings, significant differences were found only between those individuals with CBTS enamel defects that occurred during infancy and those without stress during this time period (those individuals who only formed enamel defects during childhood (3-7 years) were removed from this analysis to isolate the effects of infant stress). In the case of the radiohumeral index in females, the distal element

is relatively shorter. On the other hand, significant differences in the male crural index are due to longer distal limb segments among those stressed in infancy.

To summarize, the proportional differences based on stress were subtle and did not follow the trends observed in previous research. No proportional differences were found in sitting height indices suggesting that leg-to-trunk proportions were not different between groups. There is some evidence that distal and proximal limb segments respond differently to stress; however, the direction of variation is not consistent. The expectation was that distal limb segments would be truncated in stressed groups, but in the case of male crural indices the opposite pattern was observed. A significant relationship between age and proportions only exists for one of the many comparisons made; however, the significant difference does follow a pattern that has been suggested in previous studies.

The pattern of these results creates more questions than it answers. The r^2 values of the reduced major axis regressions have a median value of 0.735, but range from 0.29 to 0.84. This means that 16 – 71% of the variation observed can be attributed to an unmeasured variable. R^2 values are particularly low when scaling SKH and STH. One lurking variable that should be considered is genetics. Proportions are known to be strongly influenced by genetics (Mueller 1986; Stini 1975), and proportions are known to vary between populations (Eveleth, 1975, 1986; Eveleth and Tanner 1990; Hamill et al., 1973). Because population admixture among African-Americans can be high (Reiner et al., 2005), it is possible that the genetic signature in this sample is creating too much noise to observe the effects of growth disruption on proportions. To truly observe environmental effects on proportions, it may be necessary to find a more genetically homogenous population with larger sample sizes.

The Reliability of Stress Indicators

To interpret the results of the analyses used in this research, it is first necessary to critique the stress markers used. Dental defects were coded in three ways for data analysis (see Table 4-4 for a summary of these coding criteria). The TS coding criterion was the most lax in that any enamel defect (pit or line, bilateral or not) was recorded as stress, and the LEH coding criterion did not consider hypoplastic pits as enamel stress. The third, and most conservative coding criterion (CBTS) only considered and individual stressed if they had bilateral defects on their anterior dentition. The concern with enamel defect coding criteria is that systemic stress is not the only cause of enamel defects. Using the TS criterion, a traumatic insult to the dentition that affects a single tooth is coded as systemic stress. The CBTS criterion eliminates individuals from analysis who have not retained the antimere of affected teeth. The conservative requirements of this coding criterion severely limited the number of individuals that could be included in this analysis. After selectively removing many of the individuals deemed "stressed" based on TS criteria and "unstressed" by CBTS criteria were removed from further analysis, eleven individuals in this study were coded differently depending on the criteria used.

In Chapter 5, comparisons of stature estimates were performed using all three coding criteria (see Tables 5-2 through 5-4). The results of comparisons using TS and LEH coding criteria were nearly identical given that only one female in this sample had hypoplastic pitting in the absence of any linear enamel defect. The results of comparisons using LEH/TS coding criteria and the CBTS coding criterion were also very similar. Because the CBTS coding criterion is more conservative in recognizing defects as stress, the fear is that stressed individuals will be misrepresented as healthy. If unstressed individuals are expected to have longer measurements, this could artificially lower the unstressed mean. This was not found to be the case. In fact, more near significant differences were found using ANOVAs comparing groups

divided by the CBTS criteria. Because the CBTS scoring criterion is the most conservative scoring technique employed in this analysis, it was considered to be the standard in further analysis.

A G-test performed in Chapter 5 indicated that Harris lines and hypoplastic defects behaved as independent variables. Not only are Harris lines and enamel defects the product of different events during growth, individuals who are deemed stressed based on enamel defects are not necessarily the same individuals who have visible Harris lines. It is for that reason that the two indicators of growth disruption need to be analyzed separately. For example, measurements in the stressed group, as determined by enamel defects, were shorter than in the unstressed group for eight of ten measurements, but for only five of these measurements were the differences determined to be significant (see Table 5-2). On the other hand, utilization of measurements grouped by Harris lines indicated that the bones of stressed individuals were longer in every measurement, though none of these differences were significant (see Table 5-5). It is these differences that cause me to question the reliability of one stress marker over another.

The differences in degree of reliability as stress indicators are likely due to the ways in which these stress markers form and their persistence in the adult skeleton. Enamel defects are caused by a disruption in amelogenesis which, in the majority of cases, is due to illness or nutritional deficiency (Goodman and Rose, 1990; Hillson and Bond, 1997). These markers form at the time of the insult and are indelible once formed. Harris lines also record stress, but a thick Harris line can be attributed more to a growth recovery than to the growth arrest itself (Park, 1964). A growth disruption must occur for a Harris line to form: however, growth disruption without adequate recovery will not leave as thick of a line as an individual who recovers fully. Bone remodeling may mean that the most chronically stressed individuals are those without

observable Harris lines as adults. Martin et al. (1985) caution researchers that while children with poor nutrition are more likely to form Harris lines, there are fewer "remnant scars" among the nutritionally stressed.

Blanco et al. (1974) conducted a study of Harris lines in Guatemalan children less than seven years of age. They found a trend for shorter height for age in children with Harris lines than those without. These differences were more pronounced in male children than among females. On the other hand, Goodman and Clark (1981) found very different results when analyzing Harris lines from the adult skeletal sample at Dickinson Mounds. Tibial length was significantly greater among adults with Harris lines than among those without, particularly in females. Goodman and Clark (1981) suggest that individuals genetically destined to be taller may have greater nutritional needs than shorter individuals and are therefore more likely to form Harris lines when such needs are not met. While taller individuals may be more likely to form Harris lines, there are other possible explanations for individuals with Harris lines being taller. Because thick Harris lines are linked to a positive recovery, it is possible the most severely stressed in any population present as unlined. It is also possible that taller individuals with Harris lines may have undergone the catch-up growth process. Based on Baron et al.'s (1994) experiments in artificially suppressing growth plates, we know that catch-up growth leads to an increase in growth rate (although full size was never attained in these studies). Recent research on catch-up growth in weight has linked obesity with early metabolic insults which suggest the process of catch-up may "overshoot" a normal growth trajectory in weight (Cameron and Demerath, 2002; Grove et al., 2005; Hindmarsh, 2004). While there is no evidence for the same catch-up process in skeletal length, until researchers better understand the process of catch-up growth, this possibility should not be ruled out.

In the investigation into the relationship of sexual dimorphism and stress, differences in dimorphism between stressed and unstressed groups divided by the presence or absence of Harris lines were an order of magnitude smaller than those produced by enamel defects. The corresponding *p* values found that Harris line data was not meaningful. This comparison, in addition to the others listed above, forces the conclusion that 1) Harris lines and enamel defects are not recording the same stress events, 2) the relationship between Harris lines and stress is not well understood, and 3) enamel defects are a far more reliable marker of stress in the human skeleton than are Harris lines.

Chapter 8 investigated the utility of using vertebral neural canal (VNC) shape as a marker for growth disruption as suggested by Clark et al. (1985, 1986, 1988). ANOVAs suggest that there is a significant relationship between vertebral neural canal dimensions and enamel defects (CBTS) in five out nine comparisons made in males. Of particular interest is the fact that differences in anteroposterior dimensions were significantly different in every case for males when the population was divided by enamel defects. In no case are vertebral neural canal sizes significantly related to enamel defects (CBTS) in females (see Table 8-1 and 8-2). Small anteroposterior vertebral neural canal dimensions cease growth earlier than mediolateral dimensions and have been associated with low birth weight, maternal smoking and a protein deficient diet (Clark 1985; Jeffrey, 1988). Rewekant (2001) found smaller vertebral neural canal indices (anteroposterior diameter/ mediolateral diameter) among a stressed archaeological population when compared to a population of higher socioeconomic status. Interestingly, Rewekant (2001) found that the significant differences were only for the male sample – as is the case in this study. These findings raise the question of whether the selective advantage of maintaining a large vertebral canal is greater in females than males.

When vertebral neural canal sizes were compared using Harris lines as the fixed factor, a different trend was observed. Among females there significant differences exist in the mediolateral diameter of the averaged thoracic and averaged lumbar vertebrae as well as in the vertebral canal index of L1 when the population was divided by the presence or absence of Harris lines (see Tables 8-3 and 8-4). In the case of the vertebral canal index, the significant difference is due to larger mediolateral diameters among the unstressed segment of the population. Mediolateral dimensions of the vertebral neural canals continue to grow through childhood and are believed to be more likely to experience catch-up growth than anteroposterior dimensions (Clark 1985, 1988; Jeffrey et al., 2003). Because Harris lines are more closely associated with growth recovery than growth disruption, the fact that there is a relationship between Harris lines and mediolateral VNC dimensions may not be surprising. It is important to note that this response is only observable among females.

The relationship between VNC dimensions and traditional markers of stress suggest there may be utility in using vertebral neural canals as a stress indicator; however, there are several caveats to that conclusion. First, the utility of using a vertebral canal index (anteroposterior diameter/mediolateral diameter) is presumably an attempt to get an understanding of the overall shape of the vertebral neural canal shape, but a small index may be due to a smaller anteroposterior diameter or a larger mediolateral diameter. Given that both measurements are significantly correlated to overall body size, this ratio will always need to be qualified. In this study, anteroposterior and mediolateral dimensions were far more informative than the vertebral neural canal index. While the relationship between vertebral canal dimensions and other indicators is an important one, VNC shape did not prove useful for addressing the hypotheses of this study. Correlations comparing skeletal height and vertebral neural canal shape suggest that

height and VNC shape are not independent variables (see Table8-11), although VNC measurements would not be useful for predicting stature. Therefore, VNC dimensions cannot be used to infer stature differences between stressed and unstressed groups.

Head circumference, a known indicator of stunted growth in children, was also compared to enamel defects and Harris lines. These investigations did not yield any significant relationships in the Hamann-Todd collection (where head circumference was determined from autopsy reports), or in the Terry collection (where head circumference was measured). Regardless of the relationship with other stress markers, head circumference is significantly correlated with skeletal height in the Terry collection which means it cannot be used as an independent stress marker in this study. The fact that head circumference and skeletal height are not significantly correlated in the Hamann-Todd collection only serves to make me wary of the autopsy reports.

In an adult skeletal sample, the available markers of growth disruption are limited, and understanding these limitations is critical to the interpretation. No skeletal marker used in this study records the full picture of stress during growth. Harris lines and enamel hypoplasias form during acute insults to growth. They may represent a single negative episode in an otherwise healthy individual, or they may be an acute mark in a chronically stressed individual; it is not always possible to tell the difference skeletally. Numerous stress markers may certainly be suggestive of a chronically stressed individual; on the other hand, it could be no more than a record of numerous acute events. Steckel (1995) suggests that stature provides a record of chronic stress which can be observed by looking at the secular changes in a population. The nature of the growth disruption (chronic vs. acute) could provide the explanation for why stress markers are not significantly related to stature in men.

Study Limitations: Collection Effect

It is important to consider the "collection effect" that may be influencing the results of this study. The fact that the majority of these individuals were unclaimed by family suggests that they were from the lowest of socioeconomic classes. Therefore, the Hamann-Todd and Terry collections are not necessarily representative of the African-American communities as a whole in Cleveland, OH and St. Louis, MO at the turn of the twentieth century (Hunt and Albanese, 2005). Attempts to assess whether or not this sample is representative have proven inconclusive (Albanese, 2003).

The decision to combine materials from two osteological collections was made based on Işçan's (1990) conclusion that the two populations were osteometrically similar. To further ascertain the similarity of the two populations, a few simple variables were analyzed to determine whether or not the individuals I sampled from the two collections were similar: year of birth and skeletal height. When data for year of birth were compared, a significant difference was observed ($p = 0.016$). The mean year of birth in the Hamann-Todd collection was 1902.77 and the mean for the Terry collection was 1906.09. Because secular trends exhibit increasing height within these collections, skeletal height in the two collections were compared and no significant differences were observed ($p = 0.138$). In addition, there is no correlation between year of birth and skeletal height in the combined sample ($p = 0.342$). Since a secular trend cannot be observed in this sample and the mean difference between the collections is only 3.32 years, the difference in year of birth is therefore notable, but likely unimportant.

Study Limitations: Age Effect

In acquiring the sample population for this study I initially chose individuals between the ages of twenty and thirty-five years in order to analyze the skeletal remains of individuals with fully fused long bones and who were not suffering from any age-related changes that would

affect stature. Because the number of females in the Hamann-Todd and Terry collections is much lower than the males, a few 19 year-old females with fully fused epiphyses were included to increase the final sample size.

Age is not normally distributed within this sample, so nonparametric correlations (Kendall's tau-b) were run comparing age to both skeletal height (SKH) and sitting height (STH) to determine whether or not an age effect was visible in the sample population. Age is not significantly related to SKH, but it is related to STH in males (see Table 9-1). A scatter plot of STH data against age suggests that some of the youngest individuals in the male sample did not attain full vertebral height before death; however, the confidence interval for a linear regression line fit through the data included zero, signifying that the effect of age on STH is negligible (see Figures 9-1 and 9-2). Bass (1995) states that vertebral body heights almost attain their full size by puberty; nevertheless, they do not fully fuse until 17 to 25 years of age. Including males under 25 in my initial analysis is likely the cause of the age effect observed in this sample. While collecting data for this study, individuals were excluded from analysis if the epiphyseal rings of the vertebrae were not present, but they were included if the epiphyseal rings were intact but still in the process of fusing.

To see how this age effect influenced the results of this study, individuals under the age of twenty five were removed from the male sample. After removing these individuals, STH and age were no longer significantly correlated (τ-B = 0.137, p = 0.079). ANOVAs were performed comparing SKH, STH, femur length and tibia length in stressed and unstressed groups (defined by the presence or absence of enamel defects (CBTS)) in this smaller sample (containing only 25 to 35 year old males). None of these results were significant (see Table 9-2) which suggests that any age effect which exists in the original sample population did not significantly influence the

results of this study. This research therefore informs future methodology: in future studies where vertebral height is considered, conservative procedures should exclude males under the age of twenty-five years.

Table 9-1. Nonparametric correlations between age and height

	Skeletal Height (SKH)		Sitting Height (STH)	
	Correlation	Significance (2-	Correlation	Significance (2-
Females	0.092	0.195	0.133	0.060
Males	0.063	0.348	0.175	0.010 [a]

[a] Significant at the 0.01 level (2-tailed)

Table 9-2. ANOVA –Stature estimates (age controlled) by dental enamel defects (CBTS)

		N	Mean	S.D.	Levine's	F	*p*
Male							
	Skeletal height (SKH)						
	Unstressed	30	160.10	6.918			
	Stressed	53	159.74	6.620	0.530	0.055	0.816
	Revised Fully Stature						
	Unstressed	30	172.35	6.955			
	Stressed	53	171.99	6.666	0.527	0.054	0.816
	Sitting height (STH)						
	Unstressed	30	48.50	1.707			
	Stressed	53	48.07	2.0393	0.339	0.939	0.335
	Max. femur length (mm)						
	Unstressed	30	480.50	27.748			
	Stressed	53	477.86	25.167	0.387	0.196	0.659
	Tibia length						
	Unstressed	30	401.38	24.543			
	Stressed	53	403.50	25.495	0.829	0.136	0.714

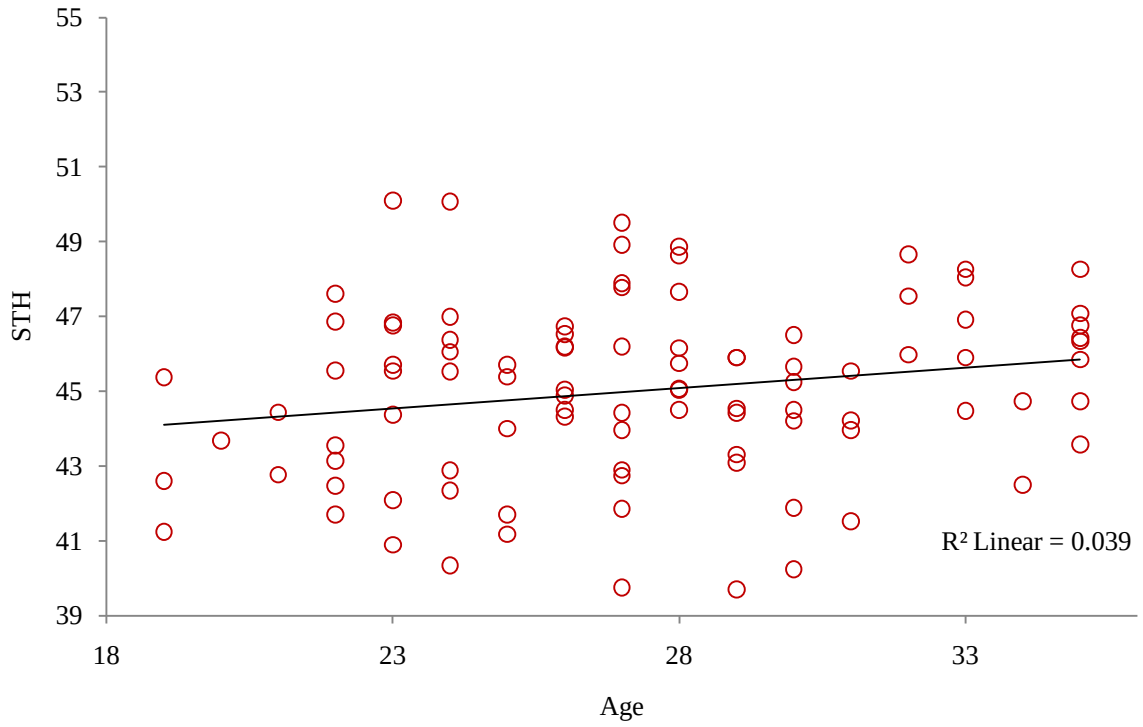


Figure 9-1. Scatter plot of STH by age in females

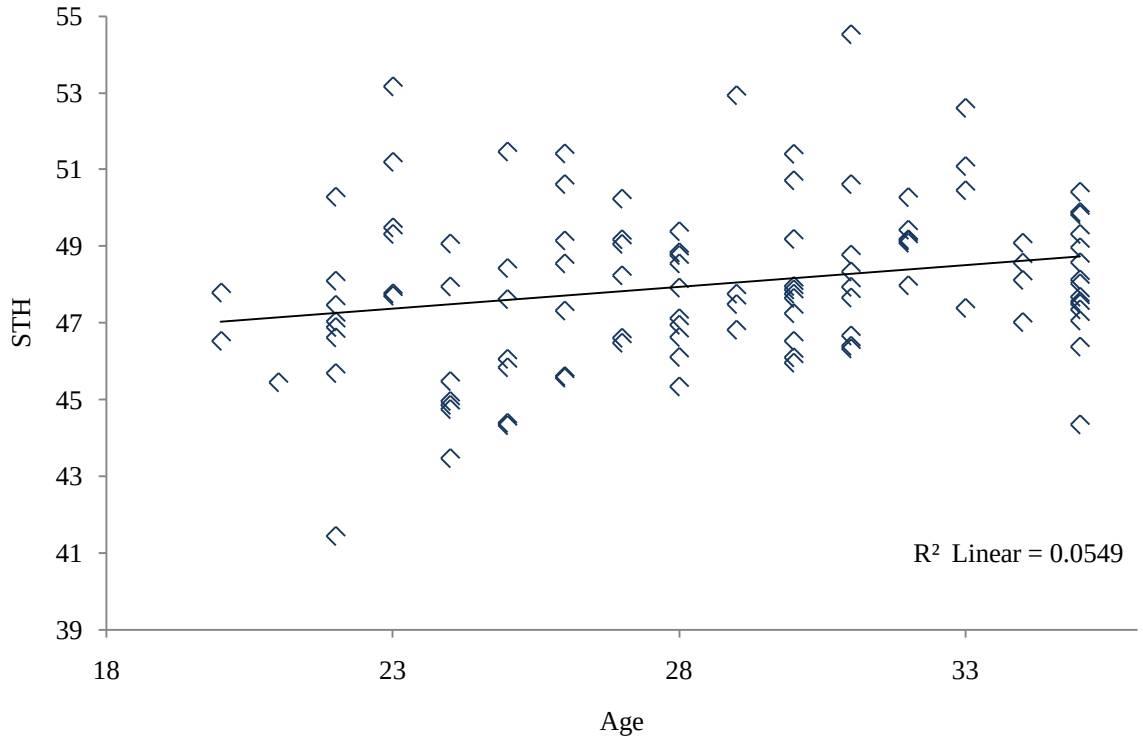


Figure 9-2. Scatter plot of STH by age in males

CHAPTER 10
CONCLUSIONS

The three alternative hypotheses tested in this study were based on previous research on the relationship between growth and stress, and an interest in whether or not these relationships could be observed in a skeletal sample. Skeletal markers of growth disruption, enamel defects and radiopaque transverse lines were compared to osteometric data to determine whether or not the stresses associated with the skeletal marker had an effect on adult stature, sexual dimorphism and proportions.

Previous research on the process of catch-up growth suggests that the negative effects of growth disruption are often erased (e.g. Cameron, 2002; Tanner, 1981); however, anthropologists use trends in adult morphology to estimate the relative environmental stress on populations (Komlos, 1994; Steckel 1995, 1999; Steegman 1985, 1991). The premise behind such studies is that stressed populations are less likely to reach their genetic potential for height. In addition, females are believed to be buffered biologically against stressors, evident in that females tend to have greater fat and nutrient reserves (Stini 1969, 1973, 1975a, 1975b, 1982). In that females are buffered, male size is believed to very more in response to environmental circumstance, thereby decreasing the degree of sexual dimorphism found in stressed populations.

Human proportions vary by population and some researchers have suggested they are under stronger genetic control than is overall body size (Mueller 1986; Stini 1975). However, numerous researchers have found that better environmental circumstances lead to longer relative lower limb length (Bogin and Rios, 2003; Bogin et al., 2002; Dangour, 2001; Floyd, 2007, 2008; Fredriks et al., 2005; Frisancho et al., 2001; Li et al., 2007; Malina et al., 2004; Stinson, 2000; Wadsworth et al., 2002) and that the relationship of proximal and distal limb elements is associated with environmental circumstances (Holliday and Ruff, 2001; Meadows Jantz and

Jantz 1999; Jantz and Owsley 1984; Meadows and Jantz 1995; Smith and Buschang, 2004). Furthermore, because nutritional stress and growth disturbances are greatest in the first two to three years of life, age of an insult may affect its influence (Beaton et al., 1990; Checkley et al., 1998; Martorell and Habicht, 1986; Martorell et al., 1995).

An investigation into the relationship between stature and skeletal markers of stress found that females were significantly shorter than their unstressed counterparts when stress was determined by the presence or absence of enamel defects. Significant differences in stature were not found among males. When Harris lines were used to determine stress, no significant differences were found between stressed and unstressed groups. While significant differences in sexual dimorphism were not found between stressed and unstressed groups, it is notable that stress had a visible affect in the female skeletal sample, but not the male.

Investigations into the relationship between proportions and stress yielded only one significant result. The crural index in males was significantly related to the presence or absence of enamel defects. Interestingly, crural indices were higher among the stressed group. The stressed group had longer tibial lengths and shorter femoral lengths than their unstressed counterparts. Previous researchers found that the distal limb segment is more variable (Holliday and Ruff, 2001; Smith and Buschang, 2004), and that the length of the tibia increases in secular studies where environmental conditions improve (Meadows Jantz and Jantz, 1999; Jantz and Owsley, 1984; Meadows and Jantz, 1995). All other proportional differences observed were subtle.

Enamel defects and Harris lines were the pathological markers observed in the Terry and Hamann-Todd collections with enough frequency to yield statistically significant results as markers for dividing a population into stressed and unstressed groups. While other pathologies

were observed, they were immediately thrown out if they were believed to directly affect stature. Other pathologies could not be tied specifically to the growth period, so they were recorded but not considered in this analysis. Vertebral neural canals were measured and compared to traditional stress markers to test their efficacy as independent markers of stress. A trend between small anteroposterior dimensions of the vertebral canal and enamel defects was evident in males, but ultimately the fact that VNC dimensions were significantly correlated to stature ruled out the use of VNC dimensions as a stress marker of use in this study. A G-test comparing enamel defects and Harris lines concluded that these markers behaved as independent variables, and the results when dividing the population by these two markers were drastically different. The etiology of enamel defects is better understood than that of Harris lines and they are more widely accepted as an indicator of growth disruption (Alfonso et al., 2005). Harris line data in this study can be used as an informative comparison between the two techniques, but the enamel defect data is more useful for comparing the actual relationship between skeletal morphology and stress. Skeletal markers of stress represent acute insults and are an incomplete record of growth disruption. This fact needs to be considered when interpreting how the results of this study can be applied to research on human growth. In addition, it qualifies how effectively knowledge regarding how growth disruption affects morphology can be applied to a bioarchaeological sample.

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