HUMAN SKELETAL GROWTH: OBSERVATIONS FROM ANALYSES OF THREE SKELETAL POPULATIONS

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Submitted to the Faculty of

Arts and Sciences in partial fulfillment

of the requirements for the degree of

Bachelors of Philosophy

University of Pittsburgh

2009

UNIVERSITY OF PITTSBURGH

Faculty of Arts and Sciences

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HUMAN SKELETAL GROWTH: OBSERVATIONS FROM ANALYSES OF THREE SKELETAL POPULATIONS

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This research seeks to illuminate four problems that have long plagued the anthropological study of human skeletal growth. These problems, and their respective research questions, are as follows:

1) Sexual dimorphism: Is there a difference in skeletal growth between males and females?

2) Population variation: Do geographically distinct populations experience different patterns of growth?

3) Mortality bias: Is there a morphological difference between those who die and those who survive?

4) Disease and malnutrition: What are the effects of disease and malnutrition on human skeletal growth?

Subadult individuals from the Hamann-Todd Collection (n=33) in Cleveland, the Luis Lopes Collection (n=44) in Lisbon, Portugal, and the Raymond Dart Collection (n=31) in Johannesburg, South Africa, were analyzed to test these questions. Diaphyseal lengths were measured for all individuals; femora were used for all statistical analyses. The three samples were combined following the analysis of population variation.

ANOVA of femoral length by sex (controlled for age) was used to analyze the degree of sexual dimorphism within the combined sample, and the difference was found to be insignificant ($p=0.367$).

Population variation was investigated using ANOVA; femoral length by sample (controlled for age) was analyzed and found to be insignificant ($p=0.203$).

T-tests of mean femoral length for the combined sample vs. the reported means of Maresh (1955) were conducted for each age category in order to examine the difference between living standards (provided by Maresh, 1955) and their contemporaneous skeletal counterparts. Nine of the 13 age categories exhibited significant results ($p<0.05$).

No significant difference was found between diaphyseal lengths of the pathological sample and the normal sample ($p=0.25$), or between the different pathological categories ($p=0.388$). ANOVA between individual pathological categories and the normal sample showed that only malnutrition had a significant ($p=0.016$) inhibitory effect on growth.

The results of this study indicate that sexual dimorphism in long bone growth is not apparent prior to adolescence, the degree of variation between geographically disparate populations is not significant ($p>0.05$), mortality bias is a significant factor affecting juvenile skeletal remains, and while malnutrition significantly retards skeletal growth, the diseases tested here do not.

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PREFACE

This study began as a precursory investigation of methods of aging skeletal subadults. After extensive review of the literature regarding skeletal juveniles, it became apparent that the field of anthropology was laboring under a tenet that no one had attempted to validate using skeletal material, namely, that disease and malnutrition retard skeletal growth. Skeletal collections were chosen on the basis of the availability of demographic data (age, sex, and cause of death), in order to test three hypotheses:

1) The presence of disease and/or malnutrition retards skeletal growth

2) The organ system upon which a disease acts impacts the amount of skeletal growth retardation

3) The severity of the disease (acute or chronic) differentially affects skeletal growth

After analysis of two skeletal collections (Hamann-Todd, Cleveland Museum of Natural History, Cleveland, and Luis Lopes, Museu Bocage, Lisbon), it became apparent that the nature of the interactions between health status and skeletal growth is much more complex than the tenet has led observers to believe. Another skeletal collection was sought to test the results of the first analysis, and further assumptions about sexual dimorphism, population variation, and mortality bias. The paper that follows is the result of this further analysis.

A note on terminology: Throughout the course of this paper, individuals who have yet to reach the adolescent phase will be referred to variably as subadults, children, and juveniles. These terms do not reflect the life stage of the individuals; they are generalized terms for any person who has not yet reached puberty.

The research presented here would not have been possible without the financial support of the University Honors College, the U.S. Steel Foundation, the Office of Experiential Learning, the University Center for International Studies, and Ms. Georgia Berner. I would like to thank both of my research assistants, Ms. Alexandra R. Klales and Ms. Alanna J. MacCord, for their dedication to both skeletal research and international travel. Lyman Jellema (Cleveland Museum

of Natural History), Diana Carvalho (Museu Bocage), and especially Dr. Hugo Cardoso (formerly of the Museu Bocage), were integral to developing insights into the skeletal collections analyzed. All statistics were provided by James J. Cray, who has my eternal thanks for committing himself to this tedious task. Finally, I would like to thank Dr. Jeffrey Schwartz; without his guidance, roguishness, and encouragement of my inquisitive nature, I would never have been able to complete this project.

Kate MacCord

April 2009

1.0 INTRODUCTION

In the past, anthropological studies of human longitudinal skeletal growth have been conducted in one of three ways: 1) a literature review, 2) a population biology study of growth in living children, or 3) a study of archaeologically-derived skeletal remains (Johnston, 1968; Hummert and van Gerven, 1983; Jantz and Owsley, 1984; Mensforth, 1985; Lovejoy, 1990; Steyn and Henneberg, 1996). Problems implicit in such studies abound: chronological age is based on methodology that is both population-specific and inconsistent between authors, sex is unknown, cause of death is unknown, etc. These problems are overcome by utilizing skeletal collections that provide demographic information for each of its specimens.

Four additional factors affecting growth and skeletal materials are not so easily conquered, these are: 1) sexual dimorphism, 2) population variation, 3) mortality bias, and 4) pathology. Each issue has the potential to bias analyses and interpretations of skeletal growth, and each will be addressed throughout the course of this paper.

1.1 JUVENILE SKELETAL REMAINS

The largest problem facing those who wish to study juvenile skeletal remains is the dearth of materials. Fewer than 4% of the specimens in the Luis Lopes Collection, and only 2% of the specimens within the Hamann-Todd and Raymond Dart Collections were applicable to this

study. It is commonly held that infant's and children's skeletons will be the least well preserved of skeletons excavated from cemeteries because of their small size and higher organic composition (Saunders and Hoppa, 1993). Collections of skeletal juveniles may also be small because childhood mortality is not a common occurrence, and children who do die are likely to be interred. Along with a lack of juvenile skeletal materials, there is also a very small set of skeletal collections which maintain extensive demographic records on their specimens (including age, sex, and cause of death).

1.2 ANIMAL MODELS

The momentum of growth in the individual is subject to a variety of influences, the effects of which can be anticipated only imperfectly (McIntosh, 1957). Factors that complicate the interpretation of growth can be divided broadly into three groups: 1) factors which are hereditary; 2) circumstances of the external environment; and 3) conditions of the internal environment; all three must be taken into account when studying the growth of any organism.

Because human growth studies can employ few controls for their subjects, they generally come to weak and contradictory conclusions; these will be discussed in detail later. Additionally, growth data that comes from skeletal juveniles is necessarily cross-sectional, which makes it impossible to analyze growth rates or individual growth curves.

Animal models are ideal for the study of growth because the aforementioned factors can be largely controlled; however, the applicability to humans of results derived from non-human

models is problematic. There are well-documented differences between animals species used in research in how they utilize, metabolize, and excrete nutrients (Baker, 2008). For instance, β-Carotene is the carotenoid with the greatest vitamin A activity, but its cleavage efficiency in the gut can be four times higher in chicks and rats than in pigs (Baker, 2008); no single animal model perfectly mimics human absorption and metabolism of carotenoids (Lee et al, 1999).

Human children also develop at a slower rate and over a more protracted time period than any model animal, and so children will respond less rapidly to any vitamin or mineral deficiencies than chicks, rats, or mice (Baker, 2008). The slower growth rate also means that a great deal of the nutrition that children receive will be used for maintenance, not just growth.

1.3 SEXUAL DIMORPHISM

Sexually dimorphic growth is a problematic factor to bear in mind when studying juveniles. The existence of different growth rates and patterns of growth between the sexes complicates the interpretation of overall population growth patterns and may cut sample sizes in half if males and females vary significantly; however, it is believed that sexual dimorphism in long bone length does not develop until adolescence (Humphrey, 1998). This is supported by the work of Gindhart (1973), who analyzed a sample of several hundred healthy, white, middle class children from the Fels Institute, and found that males and females show little difference in tibial growth until adolescence. Maresh (1955) studied the growth of all long bones in healthy, white children from the Child Research Council study in Colorado and discovered that sexually dimorphic patterns exist in infancy, but then disappear until adolescence.

Gindhart (1973) also noted statistically significant differences at nearly all ages in the radius. Her findings about the radius reiterate those of Maresh (1955), who reported that longer forearm bones in boys than in girls have been demonstrated at all age periods.

1.4 POPULATION VARIATION

There is much debate about the applicability of growth standards between populations that are geographically, genetically, and socio-economically disparate. Some authors support the application of a single growth standard worldwide, while others promulgate the grave importance of developing and using growth standards that are population-specific. This question will be addressed briefly here, and then in further detail in the Results and Discussion sections.

The use of a single growth standard for children worldwide would provide consistency in methodology which would allow for direct comparison of growth studies. Additionally, a host of research shows that differences in growth between populations are not significant before puberty (Sundick, 1978). The universality of human growth was demonstrated for preschool children raised under good nutritional and environmental conditions, regardless of genetic or ethnic background (Martorell et al, 1974). New growth standards developed from the WHO Multicentre Growth Reference Study (MGRS) (released in 2006) describe how children should grow rather than reference growth in particular localities, intimating a consistent pattern of growth for children worldwide.

This consistency in growth between populations is also seen in several studies of archaeologically derived skeletal populations. Merchant and Ubelaker (1977) found that when compensation was made for methodological variability (i.e. the means of determining age), only minimal differences existed between the Indian Knoll and Arikara rates of long bone growth from birth to at least 9.5 years. Saunders et al (1993) found their sample of juvenile skeletons from 19th century Belleville, Ontario had growth comparable to standards of the living.

Despite the support for uniform growth during childhood, many physical anthropologists still maintain that population-specific growth standards should be developed and utilized. Saunders (2000) reminds us of the importance of choosing skeletal standards that are appropriate for the known or suspected population. It is an unfortunate reality that relative to the number of different human populations, few of these standardized growth profiles have been established. The main point of contention for a single growth reference standard is that reference standards used to assess growth are often based on growth rates for well-nourished children from Western and other developed nations (Maresh, 1955; Gindhart 1973; Saunders and Hoppa, 1993). It is believed that individuals who do not have the advantages of proper nutrition or adequate healthcare will fail to meet the standard set by their well-fed, healthy peers. This question of the effects of disease and malnutrition on linear skeletal growth will be addressed several times throughout the remainder of this paper.

A further problem with using a single growth reference standard is the assumption that all populations have the same potential for attaining stature. This problem may be alleviated simply by employing a single standard until adolescence, at which point population-specific growth

standards could be developed. Adolescence provides a further set of issues (addressed in Methods section), because the timeframe in which individuals reach this phase varies widely between populations and between the sexes (Bogin, 1999). These complications provide an excellent argument for the development of population-specific growth standards.

Saunders' (2000) warning seems appropriate in light of the research of Buzina (1976), whose anthropometric research on three Yugoslav populations aged between birth and 18 years showed significant height differences between populations at all ages. Buzina (1976) attributed these distinctions to both environmental and hereditary disparities.

The feasibility of developing a single international growth standard was further challenged by Eveleth and Tanner (1976), whose extensive review of child growth studies point to differences in achieved height and growth patterns across populations of juveniles. Among the studies reviewed by Eveleth and Tanner (1976) is that of Howe and Schiller (1952), whose work with school-aged children from Stuttgart, Germany, before, during, and after both WWI and WWII provides evidence of differences in height and body weight between the three different schools used in their study. These dissimilarities are not supported by statistical analysis, so the extent of the differences in the measurements is unknown.

Johnston (1968) compared his juveniles (aged birth- five years) to a modern, living population (Maresh, 1955) and found that after the age of two, statistically significant differences were seen in their growth rates. Johnston (1968) did not take into account the secular trend towards

increased height when he compared the Indian Knoll population (over 5,000 years old) to modern children.

1.5 SECULAR TRENDS

“During approximately the last hundred years in industrialized countries, and recently in some developing ones, children have been getting larger and growing to maturity more rapidly” (Tanner, 1962: 150). This statement highlights the phenomenon of a secular trend toward increased height. After analysis of four populations of children in Toronto, Hoppa and Garlie (1998) confirmed Tanner’s (1962) assertion. Cardoso (2008) investigated secular changes in body weight and height of Portuguese boys over the 20th century and concluded that there was a strong trend toward increased height. Hoppa and Garlie(1998) concluded that these positive secular changes, "support the notion of global changes in health and well-being, from the late 19th and early 20th centuries to present day, associated with a variety of socio-economic improvements that are reflected in growth."

In accordance with Hoppa and Garlie (1998), Jantz and Jantz (1999) found that the pattern of secular change is very general and cuts across socioeconomic lines, such that even the severely disadvantaged seem to participate in the secular gains and losses in height. The association between growth and environment will be investigated later in this paper, but one must bear in mind the trend towards increased height over time between children of the same age when choosing skeletal collections from which to draw samples.

1.6 CATCH-UP GROWTH

Catch-up growth is a phenomenon whereby animals that experience growth retardation due to unfavorable conditions (illness or malnutrition) may regain some of their lost stature by an increased growth velocity when conditions again become favorable. The possibility that catch-up growth exists seems to be universally accepted, but the extent to which it influences final form is a matter of much inquiry.

A delicate balance exists between the age at which the insult affects the animal and its duration and/or severity. Growth velocity is most rapid in the postnatal phase (birth through 28 days) and infancy phase (1-36 months), with quick deceleration towards the end of the latter (Bogin, 1999). Thus, the first few years of life represent a period when adverse factors can have a significant and lasting effect on growth. Evidence indicates that severely malnourished young animals are capable of reaching normal adult bone lengths if the growth period is sufficient and conditions become favorable (Himes, 1978). If, however, conditions remain unfavorable, normal adult size may never be achieved.

Similarly, if the period of malnutrition or illness occurs relatively late during the growth period, then bone length will be only mildly affected by the insult because adult size was nearly attained (Himes, 1978). The more mature bone also has a smaller window of catch-up growth, and so may reach a smaller final size.

Catch-up growth may also be applied to circumstances in which one part of the body experiences unusually high growth velocity in order to make up for a retarded velocity in another part. Krishna and Upadhyay (1996) conducted a study of patients with spinal deformities due to

tuberculosis during early childhood in order assess whether there were any compensatory growth mechanisms in patients with stunted spinal growth. They compared a variety of measurements taken on adults who had spinal deformities resultant from childhood illness with normal adults and found that patients with the spinal deformity had significantly shorter mean standing and spine heights compared to the volunteers. However, mean leg length and mean upper limb length were significantly higher than those without spinal deformities. This betokens the existence of a compensatory mechanism.

Catch-up growth is an important factor affecting growth studies, because it may mask the presence of morbidity, and lead to an underestimation of disease and malnutrition within a skeletal population. This growth mechanism is also known to have a differential affect between the sexes, with females generally experiencing more catch-up growth than males (Stini, 1969).

1.7 MORTALITY BIAS

In a pioneering study of juvenile growth in archaeologically derived skeletal populations, Johnston (1968) warns that,

> *…it must be remembered that, no matter how impressive any skeletal information pertaining to immature individuals may appear, and particularly when incremental growth is the frame of reference, some degree of error is introduced by the very fact that the sample is skeletal. It does not represent the normal, healthy population from which it was drawn. The fact that a person died young presupposes illness, injury, or other deficiency which prevented his*

reaching adulthood. However, these factors, limiting as they are, are present in all studies of non-living material, and, if such material is to be of use at all, they must be borne graciously and realized analytically. (249)

In effect, Johnston is alerting future researchers to the possibility of a mortality bias in juvenile skeletal populations. This caveat has been repeated subsequently in the literature (e.g. Sundick (1978), Lovejoy et al (1990), Saunders and Hoppa (1993), Saunders et al (1993), Steyn (1996), and Saunders (2000)).

There are several types of mortality bias that can potentially affect the composition of a skeletal collection: cultural, environmental, and biological. Cultural mortality bias produces differential representations of individuals in cemeteries due to variations in mortuary practices (Saunders and Hoppa, 1993). Environmental mortality bias refers to the differential effects of skeletal preservation which are dependent upon conditions of interment. Both cultural and environmental mortality biases are contributing factors in the dearth of juvenile remains housed in human skeletal collections. Biological mortality bias is the physiological and morphological difference between those who die and those who survive. It is biological mortality bias that will be addressed in the remainder of this paper.

The literature presents conflicting views on the overall effect of biological mortality bias on skeletal samples. Saunders (2000) found that the effects of mortality bias on long bone lengths of juvenile skeletons from archaeological samples were minimal. However, in a review of the literature, Saunders and Hoppa (1993) suggest that skeletal samples are potentially biased, with linear growth of survivors often greater than that of non-survivors. They also remind us that

growth profiles derived from skeletal samples may not be representative of the "true" growth trajectory followed by children who survived to adulthood. These same authors come to the conclusion that, “while the potential for such a bias exists within subadult skeletal collections, the effects are likely to be small at the aggregate level and error introduced by other methodological considerations (ageing, unknown sex, sample size, preservation, quality of excavation) is likely to outweigh any such error in interpretations of past populations” (Saunders and Hoppa, 1993:128). We can see that even within the same publication, authors may change their minds about the effects of mortality bias.

While investigating growth and mortality using children in a rural Gambian village, McGregor et al (1961) concluded that growth curves of children who died were very close to the growth curves of those who survived. A follow-up study of children aged birth through five years in three Gambian villages led McGregor et al (1968; 350) to conclude that “the median heights of children who died were slightly less than those of survivors, but there is no evidence that rates of growth in height were consistently different in the two groups to draw any firm conclusion with respect to height, except that if there was a real difference it was a minor one”. Similarly, Gunnell et al (1998) reviewed data from the Boyd Orr study on diet and health in pre-war Britain and found no significant relationship between childhood height and overall mortality. These studies appear to contradict the existence of mortality bias.

From their study of the Libben population, Lovejoy, et al (1990) concluded that most juvenile deaths are the result of acute conditions that would not affect dental or osteological maturation; therefore mortality bias would not have a significant effect on a juvenile skeletal sample.

Sundick (1978) found no significant difference in the height of children who suffer frequent illnesses when compared to their healthier peers and asserted that archaeological collections of subadult skeletons should not bear a significant difference from skeletons of those who would survive to adulthood. Sundick (1978; 232) goes on to state that "…it may be possible to assume that the subadult skeletons which are present in our archaeological collections are not very different from those who survived in terms of their size. They may just have succumbed to a relatively stressful situation that lasted for a short period of time". Based on these studies, it would appear that biological mortality bias is not a significant factor affecting juvenile skeletal populations.

In contradiction to the findings of McGregor et al (1961, 1968), Lovejoy et al (1990), and Sundick (1978), Cook (1981) reviewed several studies and concluded that children who die before the age of 7 are smaller in size than their living counterparts. Mortality bias has been inferred for adult skeletal samples. Kemkes-Grottenhaler (2005) analyzed a pooled-sample of nearly 3000 skeletons and found that both sexes display a statistically significant inverse relationship between adult height and age-at-death. Kemkes-Grottenhaler concludes that, "...the relationship between body height and longevity is not causal but coincidental: mitigated by diverse environmental factors such as nutrition, socioeconomic stressors, and disease load." (340)

Sundick (1978) and Kemkes-Grottenhaler (2005) bring to light the dilemma that will be the topic of the remainder of the introduction, namely: the effects of disease and malnutrition on skeletal growth.

1.8 FACTORS AFFECTING GROWTH

The effect of environment on growth has long been a source of scientific inquiry. The majority of investigations on this subject, whether through population biology, health sciences, or anthropology, have maintained that growth processes are exceedingly plastic and readily molded by environmental factors (Mensforth, 1985) including disease, malnutrition, and even psychological environment. Widdowson's (1951) studies on mental contentment and growth in German orphanages showed that a child's affective reaction to supervisory personnel influenced weight gain; a similar response was also detected, although to a lesser extent, in stature.

Skeletal growth occurs gradually from the prenatal period, when the cartilaginous anlagen of endochondral bones appear, until maturity with cessation of osteogenesis and epiphyseal closure. Bone growth involves both an increase in volume density and an increase in bone size (longitudinal growth for our purposes). It is likely that these two types of growth are regulated differently and that nutrients have different roles in the two kinds of growth (Hoppe, 2000). Longitudinal growth– that impacts on height– occurs at the cartilaginous plates (epiphyseal plates) which lie between the zones of primary (diaphyses) and secondary (epiphyses) centers of ossification. At these epiphyseal plates, cartilage from the epiphyseal side of the disc is turned into bone through a complex process of proliferation, flattening, hypertrophy and ossification. The newly formed bone is laid down on the diaphyseal side of the epiphyseal plate. This process permits longitudinal extension of the tubular appendicular bones (as well as many others bones of the skeleton).

Growth at the epiphyseal plate ceases before fusion occurs, thus, epiphyseal fusion is the result, not the cause of growth cessation. Nilsson and Baron (2004, 2005) found that senescence is a localized mechanism, intrinsic to the growth plate, and not under the control of systemic signaling. Chondrocytes in the resting zone have a finite proliferative capacity; proliferative exhaustion is followed by epiphyseal fusion. Once the epiphyseal growth plates have become senescent, epiphyseal fusion is mediated largely by estrogens; premature exposure to estrogen leads to premature epiphyseal closure (Nilsson and Baron, 2005).

As bone grows longitudinally, it maintains and renews itself via the alternating processes of resorption (osteoclastic activity) and deposition (osteoblastic activity). These two antagonistic processes also permit constant remodeling of cortical bone, including expansion of the medullary cavity as the bone enlarges.

The constant longitudinal growth of bone throughout childhood, combined with the supposition that all growth process are readily molded by environmental factors, provides the basis for a tenet long maintained in anthropology: namely, disease and malnutrition in childhood retard skeletal growth. Guided by this tenet, anthropologists often use the skeletal growth of an individual as a proxy for their health status. The assumptions of a significant and inhibitory relationship between skeletal growth and disease or malnutrition have existed for many years without rigorous investigation using human skeletal materials, despite an abundance of conflicting literature.

1.8.1 Disease

The connection between disease and growth is complex and there is little clear evidence of the specific effects of various diseases on growth rates and patterns during childhood (Saunders and Hoppa, 1993) A comprehensive study of the effects of disease on skeletal growth requires the researcher to be cognizant of several factors: Is the disease major (i.e. tuberculosis) or minor (i.e. pneumonia)? Is the disease acute or chronic? And, what organ system does the disease principally affect (i.e. respiratory vs. gastrointestinal)?

After a review of the available literature, Tanner (1962; 130) concluded that longitudinal studies conducted on well-nourished children, "...fail to reveal any retardation of growth over a 6-month period in children who suffered throughout it from colds, bronchitis, tonsilitis, measles and pneumonia". Tanner (1962) also cited a study conducted by the Ministry of Health in 1959 of all social classes in England and Wales that detected no difference in weight gain during the first three years between babies who had excellent, average, or poor health records. Hardy (1938) discovered no relationship between frequency or type of illness and retardation in growth rate or final size at maturity. Similarly, Sundick (1978) found that children with more frequent illnesses compare well, as far as height with those who were healthier. Tanner (1978) argues that major illnesses may slow down growth, but that the effects are rarely permanent.

Martorell et al (1975) conducted a study of growth and morbidity in Guatemalan children aged birth through seven years. They discovered that "children less ill with diarrhea had substantially larger increments in length and weight than children who were ill with diarrhea a greater percentage of the time. In contrast, fever and respiratory illnesses did not affect growth rates"

(1296). In a similar study, Condon-Paoloni et al (1977) found that upper and lower respiratory infections did not affect weight or height. However, they also noted that a high frequency of diarrheal infection was found to reduce weight gain, but that gain in height was not affected.

Growth plate chondrogenesis is regulated by endocrine factors, while underlying cellular processes are regulated by paracrine factors. It is believed that the complex interaction of molecular signals is dysregulated during chronic illness (DeLuca, 2006). The events responsible for this disruption may be inflammation, protein/calorie deprivation (which contributes to immunosupression/immunodeficiency), uremia/metabolic acidosis, glucocorticoids, or an impaired GH/ IGF-1 axis (DeLuca, 2006). These factors explain why growth retardation is commonly seen in juvenile patients with illnesses such as rheumatoid arthritis, chronic renal failure, and Crohn's disease.

1.8.2 Malnutrition

The term "malnutrition" is problematic because it describes a set of symptoms rather than a specific cause (Saunders and Hoppa, 1993). Malnutrition results from a variety of factors: i.e. not only an inadequate quantity of food, but also an inadequate quality of food, wherein the levels of vitamins and minerals necessary for maintenance of normal bodily functions are not met.

The significant inhibitory effects of malnutrition on skeletal growth are well-known in the literature. "Malnutrition delays growth" is a frequently repeated statement of JM Tanner (Tanner, 1962; Eveleth and Tanner, 1976; Tanner, 1978). Tanner (1978; 128) states that "children

subjected to an episode of acute starvation recover more or less completely by virtue of their regulative powers, provided the adverse conditions are not too severe and do not last too long. Chronic malnutrition is another matter. Most members of some populations, and some members of all populations, grow to be smaller adults than they should because of chronic undernourishment during most or all of their childhood". McCance (1971; 123) agrees: "growth is a luxury which the undernourished can only afford after their maintenance requirements have been met", and concludes that a lifetime of subnormal nutrition delays growth and produces small adults. In a literature review that included the works of Eveleth and Tanner (1976) and Acheson and Hewitt (1954), Mensforth (1985) found that comparative studies have repeatedly demonstrated that linear long bone growth is a sensitive indicator of differential response to environmental stress.

Acheson and Hewitt (1959) found that starvation impairs endochondral bone growth in rats and results in a narrowing of the epiphyseal plate, the extent of which is related to the degree and duration of the malnutrition. The narrowing of the epiphyseal cartilage is the result of the cessation of mitosis in the cartilaginous proliferative zones and chondrocyte atrophy (Himes, 1978). There is a corresponding decrease in width and cell numbers in the zones of resting cartilage, and maturing cartilage (Himes, 1978). Under conditions of protein-calorie malnutrition, the zone of calcifying cartilage also experiences a decrease in vascular invasion, with concomitant reduction in osteoblastic activity (Himes, 1978); as a consequence, the layer of calcified cartilage directly adjacent to the diaphysis loses its filigree appearance and becomes stout and thick (producing a Harris Line) (Acheson and Hewitt, 1959). Histological studies indicate that narrowing and increased calcification of the growth plate accompany the

deceleration of growth, and that the decrease in osteogenesis occurs later (Acheson and Hewitt, 1959). Thus, fewer cells and less activity produce smaller increments of linear growth under malnourished conditions.

From these sources, a clear image of the detrimental effects of malnutrition on skeletal growth is evident. However, it is interesting to note that the studies which underlie many of Tanner's (and therefore most subsequent authors) opinions on the subject are war-famine studies that recorded the heights of European school children during WWI and WWII (see Lumey et al, 2007, for information on the Dutch Hunger Winter study, and Howe and Schiller, 1952, for studies on Stuttgart school children from pre-WWI to post-WWII). The growth charts show increases in height at all ages from 1920 to 1940, but large decreases in height during both world wars. The increases in height are part of the secular trend towards increased stature discussed earlier in this paper. These temporary decreases in the heights of school children during WWI and WWII are neither uniform throughout the duration of the wars, nor equivalent between age groups. Despite the presence of many more stressors than famine during wartime conditions, these decreases in height are given a simple causality and are attributed to a restriction in food intake. In contrast to Tanner's war famine studies, Wu (1994) found that the Great Depression (a period of renowned belt-tightening) led to no discernible effect on height, and in fact height increase was faster during the 1930s than between 1890 and1945.

In contrast to McCance's (1971) statement about growth as a luxury, Bogin (1979) states that skeletal growth continues to occur even under conditions of malnutrition so severe that there is no weight gain. However, Bogin (1979) does not mention the possibility of depressing the

growth rate. In 1970, Rao and Singh attempted to evaluate the relative merits of anthropometric measurements as indices of nutritional status. Using a sample of over 3,000 children, aged 1-5 years, from low income families in India, Rao and Singh (1970) found minimal differences in height between normal children and those diagnosed with protein-calorie malnutrition.

1.8.3 Disease, Malnutrition, and Heredity

The complex interaction between disease and malnutrition prohibits a simple interpretation of growth; further compounding matters is the influence of heredity on stature. Children suffering from malnutrition are at greater risk of infection than children who receive adequate nutrition. This immunodeficiency caused by a lack of proper nutrition leaves children at risk for the contraction of infectious disease, creating a perpetual cycle of ill health. This all leads to the following questions: can disease, malnutrition and factors of heredity be separated for analysis? And, to what extent does each of these influence skeletal growth?

The possibility of inter-population variation has been discussed previously, and will be addressed again in the Materials, Results, and Discussion sections. However, intra-population variation due to differences in heredity and individual growth patterns is a complicated factor that requires some inspection. Garn (1965) makes an excellent point about growth standards when he writes,

> *As a generalization, the use of averaged height data as incorporated in our conventional "growth" charts does a disservice to children whose parentage is known. It does a disservice to the children of short parents, about whom we worry excessively. It does a further disservice to the children of tall parents, about whom we so often worry too little. We tend to ignore their genetic heritage,*

assuming that they are growing satisfactorily as long as they are above the population mean (917).

By using reference growth standards we ignore the possibility of intra-population variability, and also possibly introduce both a great deal of overestimation of ill-health in children of small-statured parents, and a simultaneous underestimation of ill-health in children of tall parents. According to Mensforth (1985), this is not problematic: "Although individual and population differences in hereditary growth potential have been demonstrated, these constitute a minor source of variation relative to environmental factors (250)."

Many environmental factors are thought to influence the rate of growth (i.e. disease, malnutrition, and even mental contentment), "but in the final analysis most of them hinge upon the level of nutrition, in some areas acting in conjunction with infection" (Eveleth and Tanner, 1976: 241).

2.0 MATERIALS

2.1 HAMANN-TODD COLLECTION

The Hamann-Todd Collection is housed at the Cleveland Museum of Natural History, in Cleveland, Ohio

2.1.1 History

The Hamann-Todd collection was intiated in the 1890s by Dean Hamann as an anatomical and skeletal collection for research and teaching use by the Western Reserve Medical School. Hamann went a long way in establishing the Rocky Mountain mammal sample, but was not able to collect a large sample of human remains (Kern, 2006). Hamann's efforts to revise the anatomical laws of Ohio, together with those of Roger Perkins (director of Cleveland's Division of Health and Western Reserve's professor of preventive health), led to a greater emphasis on the collection and preservation of human remains.

In 1911, new Ohio Code Sections were passed that forced the superintendents of city hospitals, the Cleveland Workhouse, and local mortuaries to notify Western Reserve of unclaimed bodies in their possession. T.W. Todd's arrival at Western Reserve coincided with these legal changes. Unclaimed bodies were sent to the medical school where Todd and his assistants measured and

photographed them and recorded their vital statistics (i.e. age, sex, country of origin) (Kern, 2006); the remains were then embalmed for dissection in anatomy classes. After instructional use, Todd had the remains macerated, labeled, and stored in army surplus-pine ammunition boxes (Kern, 2006). In his dissertation Todd's student, William Cobb, recalls the perils of amassing a large quantity of skeletons:

> *The earlier years were replete with handicaps, many of which gave rise to highly amusing circumstances. On one occasion, when there was no room elsewhere for drying a number of skulls, they were placed singly beneath the chairs in the ampitheater of the old medical school, so that every lecturer had to face this grotesque, grinning audience as well as his less attentive but not as severe, living hearers. (Cobb, 1932)*

By the time Todd died in 1938, he had accumulated more than 3000 skeletons, each associated with a corresponding file containing anthropometric and demographic data taken at the time of death: i.e. name, age, sex, ethnicity, cause of death, and a variety of anthropometric measurements. Many files also include photos taken prior to embalming, radiographs, notes on the results of any autopsies or dissections performed, and hospital records.

Because it is an assemblage of unclaimed bodies from the Cuyahoga County Morgue and city hospitals, the Hamann-Todd collection represents an ethnically heterogeneous collection of the lower socio-economic levels of the city of Cleveland during the early 20th century. Cleveland had become an important industrial city in the 1860s when John D. Rockefeller founded the Standard Oil Company and Samuel Mather began steel production there. By 1880, 28% of the Cleveland

workforce was employed in the steel mills (Ohio History Central). Cleveland workers were hit hard by the Great Depression due to the workforce's heavy reliance on industry; by 1933, roughly one third of the workers in Cleveland were unemployed (Ohio History Central). This heavily industrialized setting is reflected in the health of the collection's subadults: e.g. respiratory disease or infections are indicated in the deaths of 20 out of the 39 (51%) children originally analyzed.

2.1.2 Sample

Despite the large number of individuals Todd initially collected, many subadults were returned to their families for interment. Of the 83 individuals under the age of 18 years that Todd collected, 51.8 % (n=43) were returned. The original subadult collection consisted of 46 males (55.6%) and 37 females (44.6%) (see Table 1). Twenty-eight males (60.9%) and only 15 females (40.5%) were repatriated (see Table 2), leaving the Hamann-Todd Collection with 40 subadult specimens. Of the 40, 39 were suitable for my analysis because of incomplete epiphyseal fusion. The sample was further reduced when the age range was truncated to 12 years (see Methods section for details), leaving 33 from the original collection in the statistical analysis. The composition of the sample included a nearly equal distribution of the sexes, with 17 males (51.5%) and 16 females (48.5%), whose ethnicity was recorded as either white or black. Four white juveniles (12.1%) and 29 black juveniles (87.9%) comprised the sample. Descriptive information is provided in Tables 1, 2, 3, 6, 7, and 10, as well as Graphs 1, 3, 6, and 12.

Table 1. Hamann-Todd specimen frequencies in original collection and sample used: sex and ethnicity separate

	Original		Sample	
Ethnicity	**Frequency**	**Percent**	**Frequency**	**Percent**
W	18	21.70%	4	12.10%
B	65	78.30%	29	87.90%
Total	83	100.00%	33	100.00%

	Original		Sample	
Sex	**Frequency**	**Percent**	**Frequency**	**Percent**
M	46	55.40%	17	51.50%
F	37	44.60%	16	48.50%
Total	83	100.00%	33	100.00%

Table 2. Frequencies and repatriation rates of Hamann-Todd collection by ethnicity, sex, and combined ethnicity and sex of specimens

	Original	Returned	Percent Returned	Sample
MW	11	10	91%	1
MB	35	18	51.40%	16
FW	7	3	42.90%	3
FB	30	12	40%	13
Total	83	43	60%	33
Ethnicity				
W	18	13	72.20%	
B	65	30	46.10%	
Sex				
M	46	28	60.90%	
F	37	15	40.50%	

The disparity of repatriation between blacks and whites within the collection is evident when broken down by race. Todd's collection was comprised of 65 (78.3%) black and 18 (21.7%) white subadults. Thirteen (72.2%) white subadults were repatriated, while only 30 (46.2%) of the black subadults were returned to their families (Table 2), which reduced the subadult collection to 35 (87.5%) black and only 5 (12.5%) white specimens.

Further inequality is noted when the percentage of children repatriated is broken down by sex and race. The original sample was comprised of 11 (13.3%) white males, 35 (42.2%) black

males, 7 (8.4%) white females, and 30 (36.1%) black females (Table 3). Of this original collection, 10 (91%) white males, 18 (51.4%) black males, 3 (42.8%) white females, and 12 (40%) black females were repatriated (Table 3). This disparity between the children returned to their families changed the composition of the collection to 1 (2.5%) white male, 18 (42.5%) black males, 4 (10%) white females, and 18 (45%) black females (Table 3).

Table 3. Hamann-Todd specimen frequencies in original collection and sample used: sex and ethnicity combined

	Original		**Sample**	
	Frequency	**Percent**	**Frequency**	**Percent**
MW	11	13.30%	1	3.00%
MB	35	42.20%	16	48.50%
FW	7	8.40%	3	9.10%
FB	30	36.10%	13	39.40%
Total	83	100.00%	33	100.00%

Age category frequencies are provided in Table 4 and displayed in Graph 1. Age 1 year had the highest frequency of specimens (9), followed by ages 8 and 10 years (both had 4). No specimens were available for ages 0 or 9 years. Years of death for individuals range from 1917 to 1937, and are shown in Graph 2.

Table 4. Distribution of specimens in each age category by sample

Age	LL	HT	RD
0	5	0	11
1	7	9	5
2	6	1	2
3	1	2	2
4	6	3	3
5	3	2	0
6	2	3	1
7	1	1	3
8	1	4	1
9	3	0	0
10	3	4	0
11	5	2	0
12	1	2	3
Total	**44**	**33**	**31**

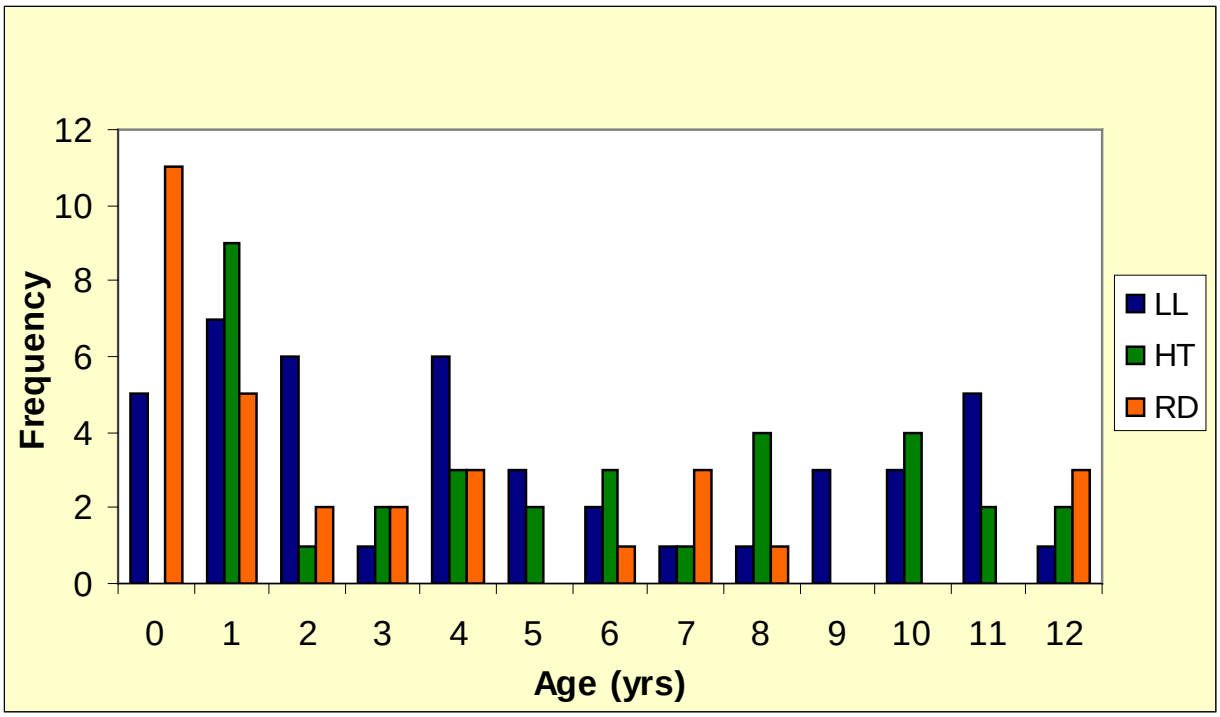

Figure 1. Distribution of specimens in each age category by sample: frequency x age category

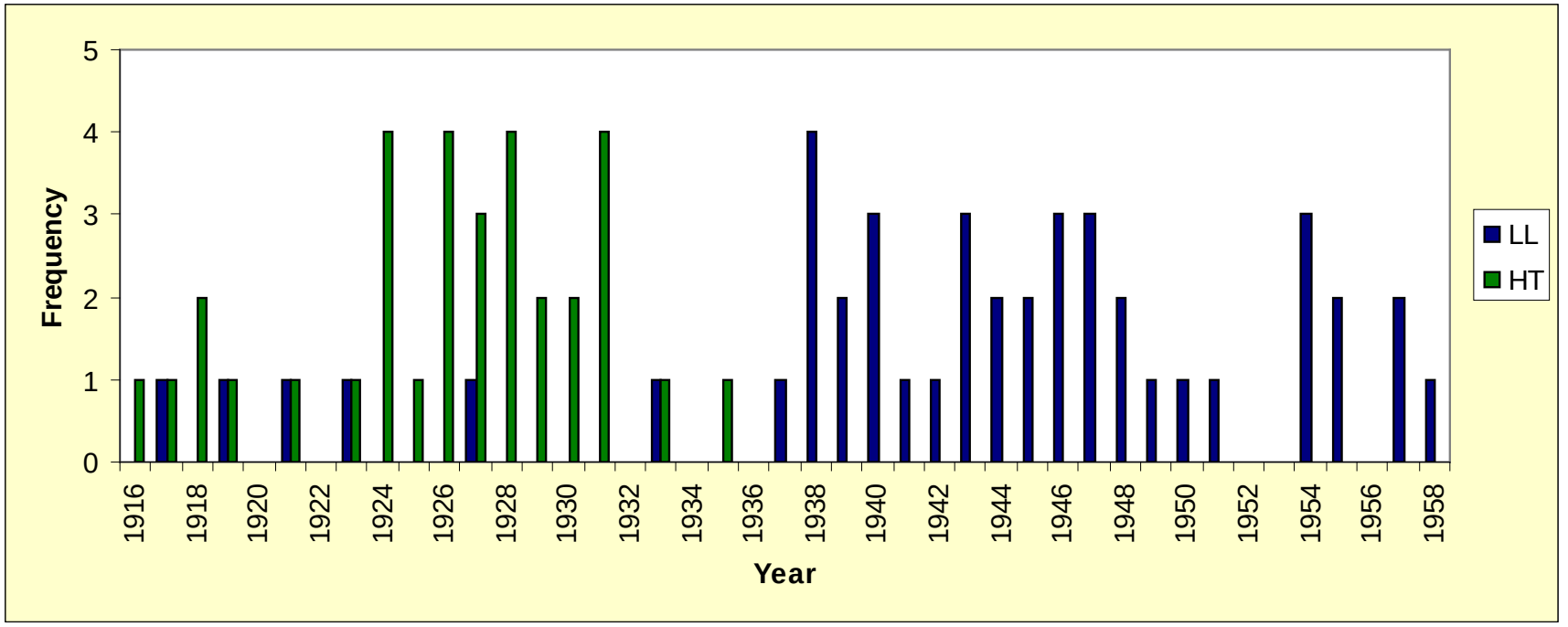

Figure 2. Year of death distribution: frequency x stated year of death. Raymond Dart Collection not shown

Records were available for many of these individuals and included: autopsy photos and reports, and hospital records. Not all specimens had each of these records. If present, autopsy photos were observed by the researcher and the nutritional status of the individual was recorded under the subjective categories: normal, malnourished, or severely malnourished. Hospital records provided ante mortem information about the individual's health status, and were recorded along with the cause of death.

2.2 LUIS LOPES COLLECTION

The Luis Lopes Collection is housed at the Museu Bocage, in the National Museum of Natural History in Lisbon, Portugal.

2.2.1 History

The Luis Lopes collection was started in 1981, when the Bocage Museum (a branch of the Nation Museum of Natural History in Lisbon) requested permission from the Lisbon City Hall to collect the remains of individuals destined for communal graves at local cemeteries. Three Lisbon cemeteries provided the majority of the skeletal material: Alto de S. João, Prazeres, and Benfica. Beginning in the early 1980s, it was the practice of these cemeteries to exhume individuals from temporary graves after a period of five years so that the grave could be reused (Cardoso, 2006). Once exhumed, the family had the option of paying a fee to place the remains in an urn that was then enclosed in a block compartment (ossário), or of allowing the remains to be buried in a communal grave (Cardoso, 2006). If no one claimed the remains, or if the fee for

the ossário was not paid, the cemetery issued an order of removal, at which point the remains were either incinerated or placed in a communal grave; it is the latter that the Bocage Museum began to collect and curate in 1981 (Cardoso, 2006). Coffin plates and cemetery registers provided a host of demographic data, including: name of the individual, name of parents, place of birth, age at death (with a precise date), sex, marital status, occupation, occupation of parents, and cause of death.

The collection of skeletal remains ceased in 1991, when the lead technician of the curation process, Luis Lopes, retired. At this point, more than 1,600 skeletons had been collected. Additional collection and curation was reinitiated in 2000 (Cardoso, 2006).

The Luis Lopes collection is comprised of Portuguese nationals who lived in Lisbon during the 19th and 20th centuries (1805-1975); these individuals represent the middle to low socio-economic classes, as evidenced by occupation of the parents and place of residence within Lisbon (Cardoso, 2007). Portugal began industrialization well after the rest of Europe. Indeed, in 1900 the majority of farmers were still practicing subsistence farming (Cardoso, 2007). Only the largest cities, such as Lisbon, had significant industries, the majority of which were small and manufactured traditional products (i.e. tiles, pottery, etc). Urban growth was greatest during the first half of the 20th century due to large migrations of rural farmers into cities in search of work, and Lisbon received the majority of this increase (Cardoso, 2009). This influx led to overcrowding and notoriously poor living conditions for the poor and working classes.

The health conditions in Portugal were among the lowest in Western Europe during the early 20th century (Cardoso, 2007). In 1920, life expectancy at birth was respectively, 35.8 and 40.0 years for men and women (Instituto Nacional de Estatística, 2001: taken from Cardoso, 2007). Even by the 1950s, the living conditions of the working classes were dismal: 43% of families lacked indoor plumbing, 69% were without electric power, and 81% had no toilet (Cardoso, 2009). High infant and childhood mortality also plagued Portugal: by 1900 approximately 50% of children died before they reached 15 years, and infant mortality was estimated at 200 deaths/1000 births (Bandeiro, 1996: taken from Cardoso, 2007). Infant mortality rates remained high until the 1940s. The main causes of death in Lisbon during the first half of the 20th century were infectious or communicable disease (Morais, 2002: taken from Cardoso, 2007).

Aside from being subjected to abysmal living conditions and high mortality rates, children in Lisbon during the early 20th century were exploited as a labor force. At about age 12, children in poor families were sent to work in factories to supplement family income (Cardoso, 2009).

2.2.2 Sample

Forty-four individuals from the Luis Lopes Collection were used for statistical analysis. Descriptive statistics for the sample are provided in Tables 6, 7, 10, 11, and 13, as well as in Graphs 1, 3, 6 and 12. The sample comprises 23 males (52.3%) and 21 females (47.7%) (see Table 5). Individuals were available for each age category. Year 1 had the highest representation (n=7), and the first three age categories combined (ages 0 through 2) make up 41% of the sample. Years of deaths for the sample range from 1917 to 1958 (see Graph 2).

Table 5. Sex distribution by sample

	LL		HT		RD	
SEX	**Frequency**	**Percent**	**Frequency**	**Percent**	**Frequency**	**Percent**
Male	23	52.30%	17	51.50%	17	54.80%
Female	21	47.70%	16	48.50%	14	45.20%
TOTAL	44	100.00%	33	100.00%	31	100.00%

Information available with each of the individuals includes age, sex, cause of death, birth date, death date, cemetery of interment, and nationality.

2.3 RAYMOND DART ANATOMICAL COLLECTION

The Raymond Dart Anatomical Collection is housed in the Department of Anatomy, University of Witswatersrand, in Johannesburg, South Africa.

2.3.1 History

The Raymond Dart collection was begun in 1924 by Professor Raymond Dart of the School of Medicine, University of Witswatersrand. The skeletal remains were prepared from dissection hall cadavers obtained from Transvaal hospitals (Saunders and DeVito, 1991). Hospital administrators provided Dart with demographic data acquired before the deaths of the patients, including: age, sex, and race/tribe (Saunders and DeVito, 1991). Cause of death was added to

each individual's record after autopsy. The subadult individuals in the collection were acquired between 1927 and 1973 (Saunders and DeVito, 1991), although no record of acquisition dates or dates of death could be found.

2.3.2 Sample

Of the 3,000 skeletal individuals housed within the Raymond Dart Collection, only 31 were suitable for my analysis. Descriptive statistics for the sample are provided in Tables 4, 5, 6, 7, 10, 11 and 13, as well as in Graphs 1, 3, 6 and 12. The sample consists of 17 males (54.8%) and 14 females (45.2%). The frequency of individuals within an age category reaches its peak at age 0 (n=11) and rapidly declines after this point. Several of the age categories do not have any specimens (ages 5, 9, 10, and 11). Information available with each specimen includes age, sex, cause of death, and tribe.

Table 6 is a list of the different tribes that comprise the sample, along with their abbreviations and country of origin. Table 7 is a break-down of the sample by tribe. The Sotho and Zulu tribes have the highest frequencies (8 and 6, respectively), and their combined percentages (25.8% and 19.35%, respectively) make up nearly half of the sample (45.15%). All of the tribes are South African, except the Mashona, who are from Zimbabwe.

Table 6. Tribes (with abbreviations) that comprised the sample from the Raymond Dart Collection, along with country of origin

Abbreviation	Tribe	Country
Bush	San ("Bushman")	S. Africa
Fing	Amafengu	S. Africa
Hlub	Hlubi	S. Africa
MixE	Mixed parentage ("coloured")	
N/S	not stated- black S. African of unspecified population group	S. Africa
Ndeb	Ndebele	S. Africa
Shan	Mashona	Zimbabwe
Soto	Sotho	S. Africa
Swaz	Swazi	S.Africa/ Swaziland
Tswa	Tswana	S.Africa/ Botswana
Xosa	Xhosa	S. Africa
Zulu	Zulu	S. Africa

Table 7. Distribution of specimens by tribe in Raymond Dart sample

Tribe	Frequency	Percent
Fing	1	3.23%
Hlub	1	3.23%
Mala	1	3.23%
Mixe	2	6.45%
N/S	4	12.90%
Ndeb	1	3.23%
Shan	2	6.45%
Soto	8	25.80%
Swaz	2	6.45%
Tswa	1	3.23%
Xosa	2	6.45%
Zulu	6	19.35%
Total	31	100.00%

3.0 METHODS

3.1 INTRODUCTION

According to Sundick (1978),

> *…the problems involved in conducting growth studies on skeletal collections are: 1) unknown age of the skeleton, 2) unknown sex of the skeleton, 3) possibility of growth retarding illnesses which led to the presence of the individual in the burial population, 4) secular trends which affect maturational stages and which may make comparisons between populations from different time periods inappropriate, 5) the unavailability in most instances of the proper dental standards to be used in the age determination of an individual from a particular population, 6) scarcity of individuals from the adolescent time periods and 7) the frequent incompleteness of the individual skeletons (228).*

The only difficulty proposed by Sundick (1978) that was not confronted by the methodology of this study is number 5; because chronological ages were known, there was no need to use dental standards to age the specimens.

Three skeletal collections were analyzed: the Hamann-Todd collection (n=33), the Luis Lopes collection (n=44), and the Raymond Dart collection (n=31). The specimens in these collections span approximately 50 years (see Graph 2 for Year of Death data). Individuals were included in the study on the basis of availability of the following: chronological age at death, sex, and cause of death.

3.2 SPECIMEN AGE RANGE

The study initially considered the age range from birth to 18 years, but the upper end of the range was lowered to 12 years for statistical analysis. This truncation was made for three reasons: 1) to exclude the confounding effects of the pubertal growth spurt, 2) to eliminate children who were working in factories (Cardoso, 2007), and 3) to utilize the measurements of Maresh (1955), whose data after age 12 included epiphyseal measurements.

3.3 MEASUREMENTS

The twelve appendicular long bones (paired humeri, radii, ulnae, femura, tibiae, fibulae) of each individual were analyzed when complete and the epiphyses were either unfused or preserved clear epiphyseal lines. Where applicable, diaphyseal lengths were measured in millimeters using an osteometric board and/or sliding calipers. When epiphyses were partially fused or attached via soft-tissue and could not be excluded from the diaphyseal measurement, the length of the diaphysis was determined by subtracting the length of these epiphyses from the total length of

the bone. When the diaphysis was bowed (i.e. as a result of rickets), the condition was noted, and diaphyseal measurements were taken on the osteometric board as well as via a tape measurer in order to determine the maximum length of the curved diaphysis. Measurements taken with a tape measure were used for statistical analyses. Each skeleton was visually inspected for porotic hyperostosis and dental enamel hypoplasias (see sections 3.8.1 and 3.9.1 for diagnostic criteria).

3.4 USE OF FEMORA

Femoral diaphyseal lengths were utilized for all statistical analyses in this study because they were most frequently preserved bones and the effects of pathology are most likely to be evidenced on them.

Growth retardation is most marked in parts of the body where growth is most rapid during times of malnutrition (Stini, 1969); and stress-induced growth retardation is most pronounced in the rapidly growing long bones of the lower limbs (Tanner, 1978). These findings were reiterated by Sciulli (1994), who found that all bones are not equally affected by nutrition and disease stress and that the most rapidly growing ones (legs) are more influenced than others.

3.5 AGE CATEGORIES

Several analyses required the samples to be divided into age groups. This created 13 age categories that covered the age range of the sample (birth through 12 years). Each age category

represents 0.99 years; age category 0 includes all individuals aged birth through 0.99 years, age category 1 includes years 1 through 1.99, and so on.

3.6 CONTEMPORANEOUS LIVING STANDARD: MARESH, 1955

Analysis of mortality bias required data from a population of living children contemporaneous with the three skeletal samples. Mean femoral lengths reported by Maresh (1955) were used. Maresh (1955) used roentenograms to measure the long bone lengths of children in a longitudinal study of healthy, white children from Colorado. The findings were reported in centimeters by 6 months age categories. Each age category was reported as five percentiles (10%, 25%, 50%, 75%, and 95%). The 50% was taken for each 6-month age category and averaged to obtain mean femoral length for each age. Males and females were reported separately, so the mean for each sex for each age category was averaged to produce a mean femoral length that was not sex-specific.

Maresh (1955) reported her findings without correcting the lengths for radiographic enlargement. She calculated magnification from roentenograms of dried bone specimens of infants, children, and adults of between 1.0% and 1.5% at a focal film distance of 2.3m with the bone in direct contact with the film-cassette surface. Mean femoral lengths for each age category were reduced by 1.5% to account for this enlargement; however, the amount of enlargement would have been increased on living children, especially at older ages, because the distance of the bone from the cassette increases with the growth and expansion of soft tissue. There was no data to support

reducing the means by more than 1.5%. These corrected means were then used for comparison against the combined sample.

3.7 PATHOLOGICAL CATEGORIES

Six pathological categories were created from the three combined samples based on a high frequency of occurrence; these categories include: dental enamel hypoplasia (DEH), porotic hyperostosis (PH), tuberculosis (TB), pneumonia (PN), malnutrition (MAL) and gastroenteritis (GE). Three additional categories were created to subsume individuals who fell into two (M2), three (M3), or four (M4) of the pathological categories. A list of the pathological categories and their abbreviations is given in Table 8. Pathology was diagnosed on the basis of recorded cause of death, hospital records, autopsy records, autopsy photos (MAL), and visual inspection (DEH and PH). Each individual within the combined sample was placed into one of these nine pathological categories. If the individual did not fit into any pathological category, it was deemed normal and included in the normative subset of the sample for analysis against the pathologies.

Table 8. Pathological categories and their abbreviations

Pathology	Abbreviation
Dental Enamel Hypoplasia	DEH
Porotic Hyperostosis	PH
Tuberculosis	TB
Pneumonia	PN
Malnutrition	MAL
Gastroenteritis	GE
2 pathologies	M2
3 pathologies	M3
4 pathologies	M4

ANOVA and T-tests were used to analyze the degrees of sexual dimorphism, of population variation, of mortality bias, and of the effects of pathology on the linear growth of the diaphyses.

3.8 POROTIC HYPEROSTOSIS (PH)

Porotic hyperostosis is characterized by an expansion of the cranial diploë with a corresponding thinning of the ectocranial cortical bone. The resulting lesion on the outer table of the skull can appear as benign as pin-prick sized porosity to complete erosion of the cortical layer and exposure of the underlying diploë. This pathology is most often linked with anemia, whether acquired (due to environmental stressors such as parasites, disease, or diet) or genetic. When the

anemic individual has depleted their store of iron, the body responds by increasing the production of red blood cells (erythropoiesis). If this process is extremely rapid, the diploë expand to accommodate the increase in marrow. The resultant hyperplasia of the marrow cavities increases the pressure on the ectocranial cortical bone, causing the skeletally diagnostic thinning and porosity.

Porotic hyperostotic lesions manifest themselves in two areas of the skull: the roof of the eye orbit ("cribra orbitalia") and the calvaria ("cribra crania"). Blom et al (2005) found a strong relationship between vault and orbital lesions: when vault lesions were present with orbital lesions, the orbital lesions tended to be more severe than when they were found alone. These results strengthen the conclusion that the orbital roof is the first expression of porotic hyperostosis, and the cranial vault bones are the second and more serious manifestation of the pathology (Lallo, 1977; Blom et al, 2005).

It is difficult to identify in skeletal materials the causal mechanisms of acquired anemia. Blom et al (2005) found in their Andean sample that environmental stressors, such as parasites and disease, were more likely to be associated with the childhood anemia. There is evidence that dietary practices also heavily influence the incidence of anemia: frequency of porotic hyperostosis is significantly higher in agricultural populations than in hunting a gathering groups (Lallo, 1977), supposedly due to differences in diet.

Iron-deficiency anemia is thought to be linked with infectious disease and, although the connection is a source of great debate (Oppenheimer, 2001), Lallo (1977) discovered a

significant ($p<.001$) association between porotic hyperostosis and infectious disease. Iron-deficiency anemia has also been observed to significantly retard growth if present during the first two years of life (Soliman, 2009).

3.8.1 Diagnosis

Porotic hyperostosis was diagnosed on the appearance of porosity on the intramembranous bones of the ectocranium that was not deemed the result of infection, and recorded as cribra crania or cribra orbitalia, depending upon its location; however, these categories were later combined for statistical analysis. Severity of porotic hyperostosis was recorded using the following scale: slight, mild, moderate, or severe. Severity was not tested for statistical significance.

3.9 DENTAL ENAMEL HYPOPLASIAS (DEH)

Dental enamel hypoplasias are classified into three categories based on their appearance: 1) furrow (linear), 2) pit, and 3) plane. Furrows are the most common form of hypoplasia found in enamel (Hillson and Bond, 1997; Schwartz, 1995) and appear as linear impressions that run along the buccal surface of the tooth, paralleling the occlusal surface. Enamel hypoplasias develop when cells forming the enamel matrix (ameloblasts) are disrupted, causing them to prematurely cease secretion of matrix (Hillson, 1996). Once the stressor has subsided, the ameloblasts resume matrix secretion, leaving an area of thinned enamel in their wake. Thus, factors that initiate enamel hypoplasias are episodic in nature (Hillson and Bond, 1997).

Disruption of the ameloblasts may be caused by an array of stressors that act systemically, including: dietary deficiency, fever, or infection. Since localized trauma to the developing dentition may induce enamel hypoplasias formation, these cases are distinguished from systemic disruptions by the number of teeth affected, the former involving only one or two teeth (Goodman and Armelagos, 1985). Genetic conditions can also cause hypoplastic enamel defects, but this is very rare in most parts of the world (Hillson, 1997). Since it is seldom possible to link enamel hypoplasias with their underlying etiology, they are considered non-specific indicators of systemic stress and health (Hutchinson and Larsen, 1988). Because enamel hypoplasias can only form while the dental crowns are developing, they cannot be remodeled during life (except by abrasive factors), thus making them excellent indicators of an episode of ill-health during infancy and early childhood (Goodman et al, 1984).

3.9.1 Diagnosis

In order to be recorded as a DEH, a groove had to be sufficiently pronounced to catch a fingernail sliding perpendicular to it along the surface of the crown. Each instance of DEH was recorded. On teeth with more than one DEH, each was noted separately. The distance of each DEH from the cemento-enamel junction (CEJ) was measured in order to ascertain the timeframe in which the individual had formed the DEH. The presence of multiple DEHs and the information about the time of formation were not included in the analysis.

4.0 RESULTS

4.1 DESCRIPTIVE

Of the total sample, 108 individuals provided all information necessary for statistical analysis. Descriptive results for these individuals are seen in Tables 6 through 11 and Graphs 1-3. Of these 108 juveniles, 44 (40.7%) were from the Luis Lopes Collection, 33 (30.6%) were from the Hamann-Todd Collection, and 31 (28.7%) were from the Raymond Dart Collection (see Table 9). Age frequencies and distributions for each collection are presented in Graph 1 and in Table 4. The majority of individuals analyzed (58.2%) were under the age of five years. Ages 0 and 1 make up the largest percentages of any of the age categories analyzed with 14.8% and 19.4%, respectively (see Table 10 for the percentages of the combined sample for each age category). Age frequencies for the combined sample are presented in Graph 3.

Table 9. Distribution of specimens by sample

	Frequency	Percent
LL	44	40.7
HT	33	30.6
RD	31	28.7
Total	108	100

Table 10. Distribution of specimens by age category within the combined sample

Age	Frequency	Percent
0	16	14.8
1	21	19.4
2	9	8.3
3	5	4.6
4	12	11.1
5	5	4.6
6	6	5.6
7	5	4.6
8	6	5.6
9	3	2.8
10	7	6.5
11	7	6.5
12	6	5.6
Total	108	100

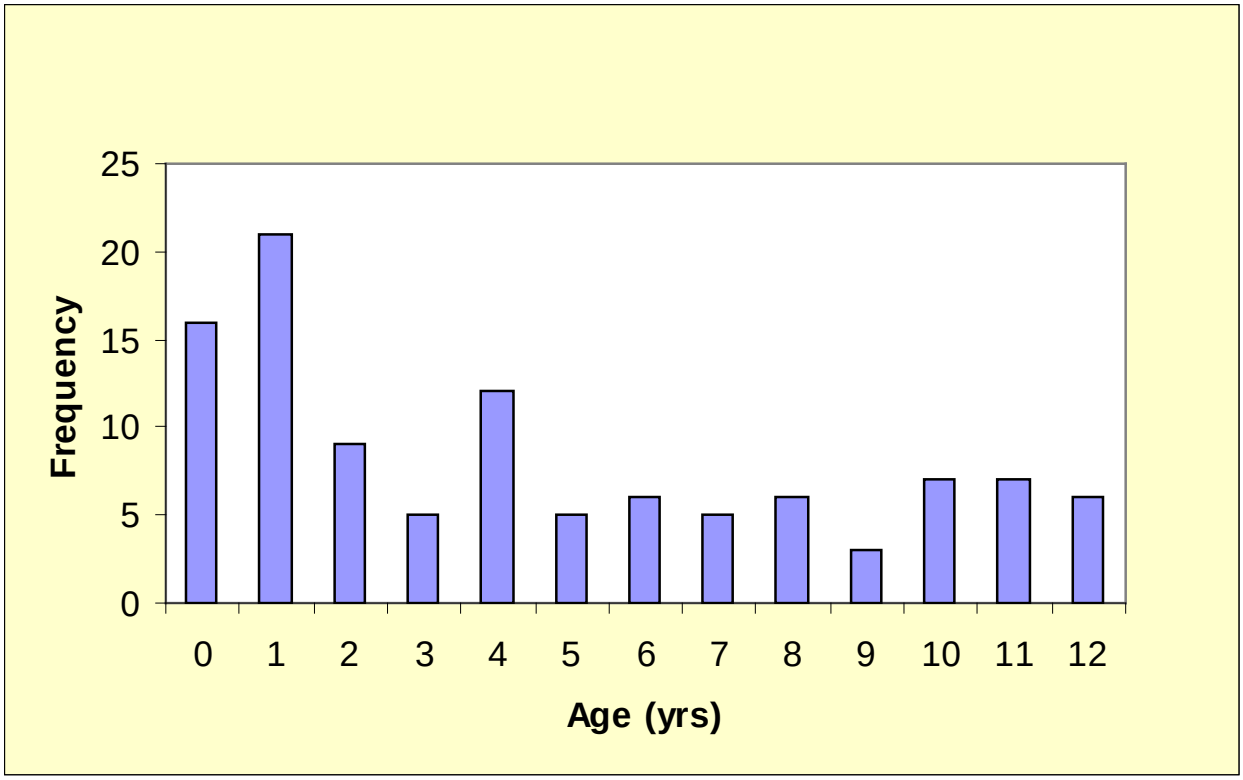

Figure 3. Distribution of specimens by age category within the combined sample: frequency x age category

4.2 SEXUAL DIMORPHISM

The sexes are nearly equally represented: 57 males (52.8%) and 51 females (47.2%) (see Table 12). Males constitute the majority of each sample, however, this majority is very small (as little as one specimen). The frequencies of sex by sample are provided in Tables 11 and 12, as well as in Graph 4. Male and female growth curves are presented in Graph 5. Mean femoral lengths for males and females of each age category are given in Table 13. ANOVA of femoral lengths was conducted on males and females (adjusted for age) in order to determine the degree of sexual dimorphism within the culled sample. ANOVA results show that the degree of sexual

dimorphism is insignificant (p=0.367). The sexes were combined for all further statistical analyses.

Table 11. Distribution of specimens by sex within the combined sample

	Frequency	**Percent**
Male	57	52.8
Female	51	47.2
Total	108	100

Table 12. Sex distribution by age category for each sample and the combined sample

	LL		HT		RD		Combined Sample	
Age	**Male**	**Female**	**Male**	**Female**	**Male**	**Female**	**Male**	**Female**
0	2	3	0	0	8	3	10	6
1	6	1	8	1	1	4	15	6
2	5	1	0	1	0	2	5	4
3	0	1	1	1	2	0	3	2
4	3	3	1	2	2	1	6	6
5	1	2	0	2	0	0	1	4
6	1	1	2	1	1	0	4	2
7	1	0	0	1	2	1	3	2
8	1	0	1	3	0	0	2	3
9	1	2	0	0	0	0	1	2
10	1	2	3	1	0	0	4	3
11	1	4	1	1	0	0	2	5
12	0	1	0	2	1	3	1	6
Total	**23**	**21**	**17**	**16**	**17**	**14**	**57**	**51**

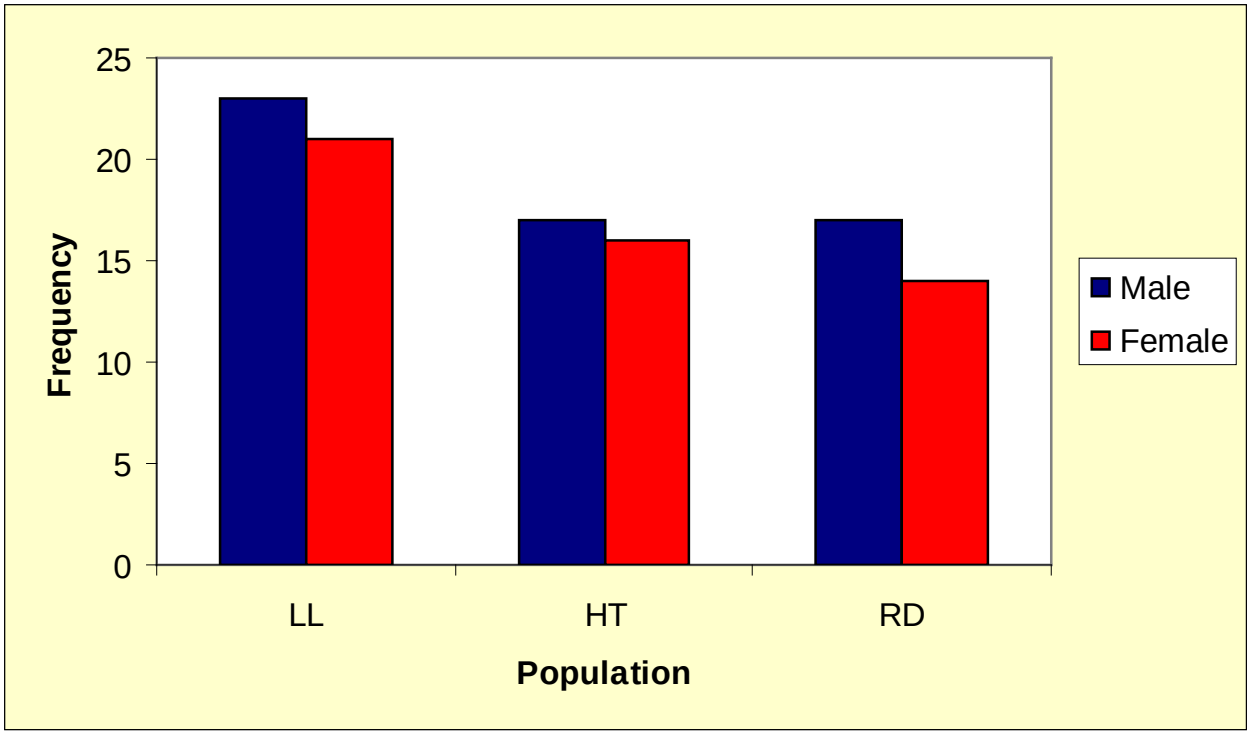

Figure 4. Sex Distribution by sample: frequency x population

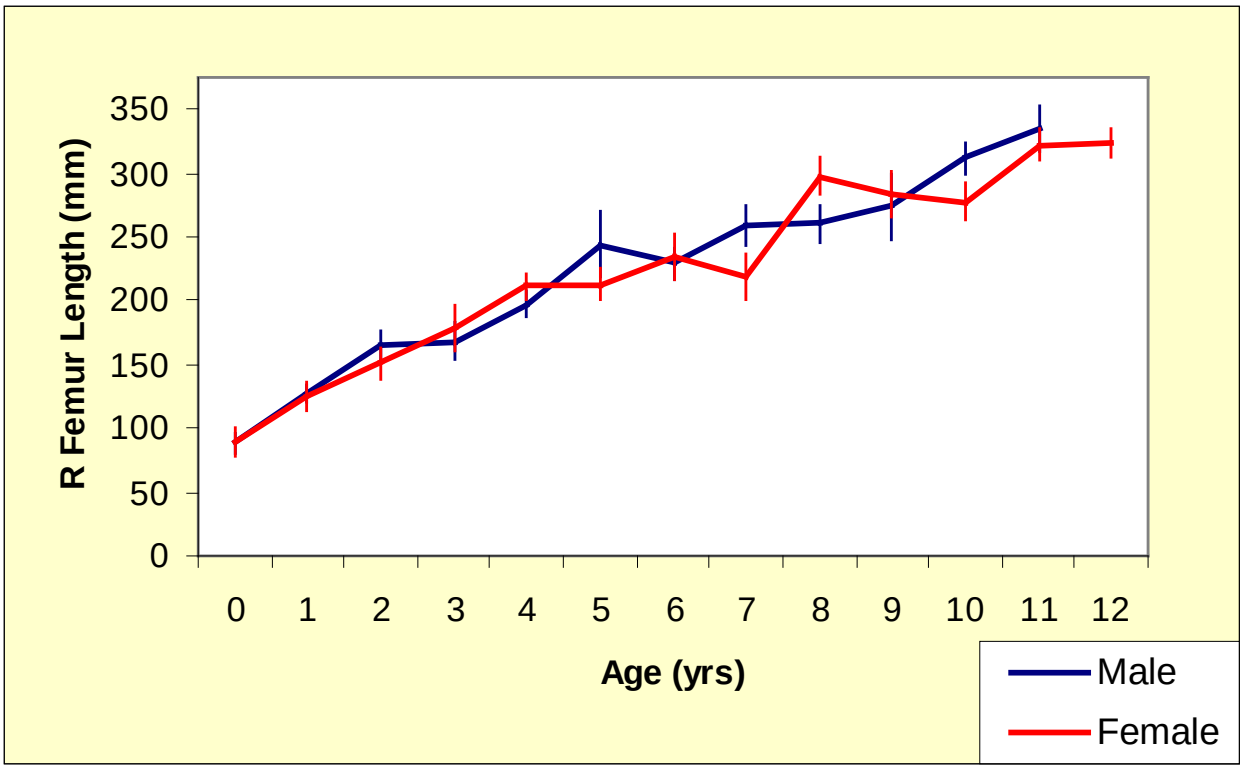

Figure 5. Growth curves for combined sample by sex: mean femoral length (and SE) x age category. Sexes separate.

Table 13. Mean femoral length (with SE and 95% CI) by age category. Sexes separate.

	Male				Female			
			95% CI				95% CI	
Age	**Mean**	**SE**	**Lower**	**Upper**	**Mean**	**SE**	**Lower**	**Upper**
0	88.5	8.18	72.237	104.763	89.33	10.56	68.339	110.328
1	126.87	6.68	113.588	140.145	125.5	10.56	104.505	146.495
2	164.2	11.56	141.201	187.199	151.13	12.93	125.412	176.838
3	168.17	14.93	138.475	197.858	178	18.28	141.636	214.364
4	197.17	10.56	176.172	218.161	211.5	10.56	190.505	232.495
5	244	25.86	192.573	295.427	213	12.93	187.287	238.713
6	229	12.93	203.287	254.713	234	18.28	197.636	270.364
7	259.33	14.93	229.642	289.025	218.75	18.28	182.386	255.114
8	260.67	14.93	230.975	290.358	297.67	14.83	267.975	327.358
9	274	25.86	222.573	325.427	283	18.28	246.636	319.364
10	311.75	12.93	286.037	337.463	277.33	14.93	247.642	307.025
11	335.5	18.28	299.136	371.864	322.2	11.56	299.201	345.199
12					324	10.56	303.005	344.995

4.3 POPULATION VARIATION

A scatter plot of all data points used in the statistical analyses is presented in Graph 6. Growth curves for each collection are offered in Graph 7; femoral length means and SE of each age category for each sample are given in Table 14. ANOVA of femoral lengths between the three

samples (adjusted for age) was conducted in order to determine the amount of population variation. The results show that the degree of population variation was insignificant (p= 0.203) (Table 15). The samples were combined for all other statistical analyses. The growth curve for the combined sample is seen in Graph 8. Femoral length means and SD for the combined sample are given for each age category in Table 16.

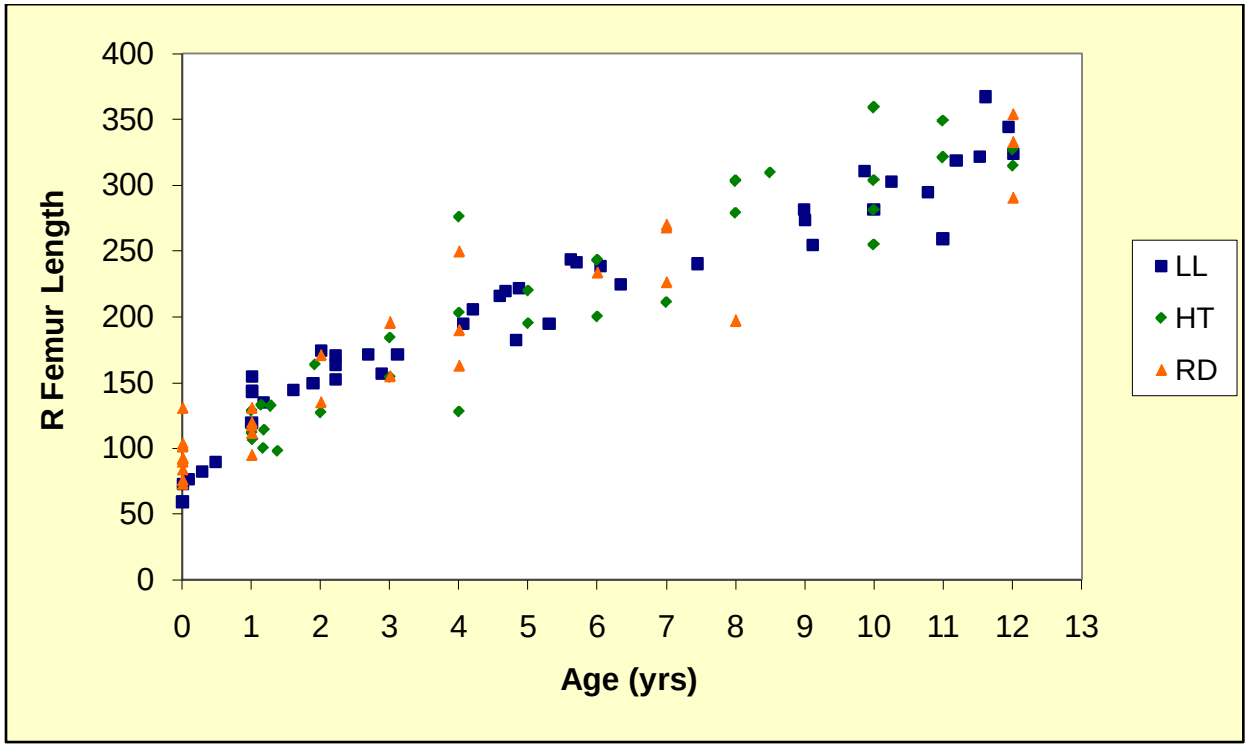

Figure 6. Scatterplot of all data points used in statistical analyses: R femur length x age category

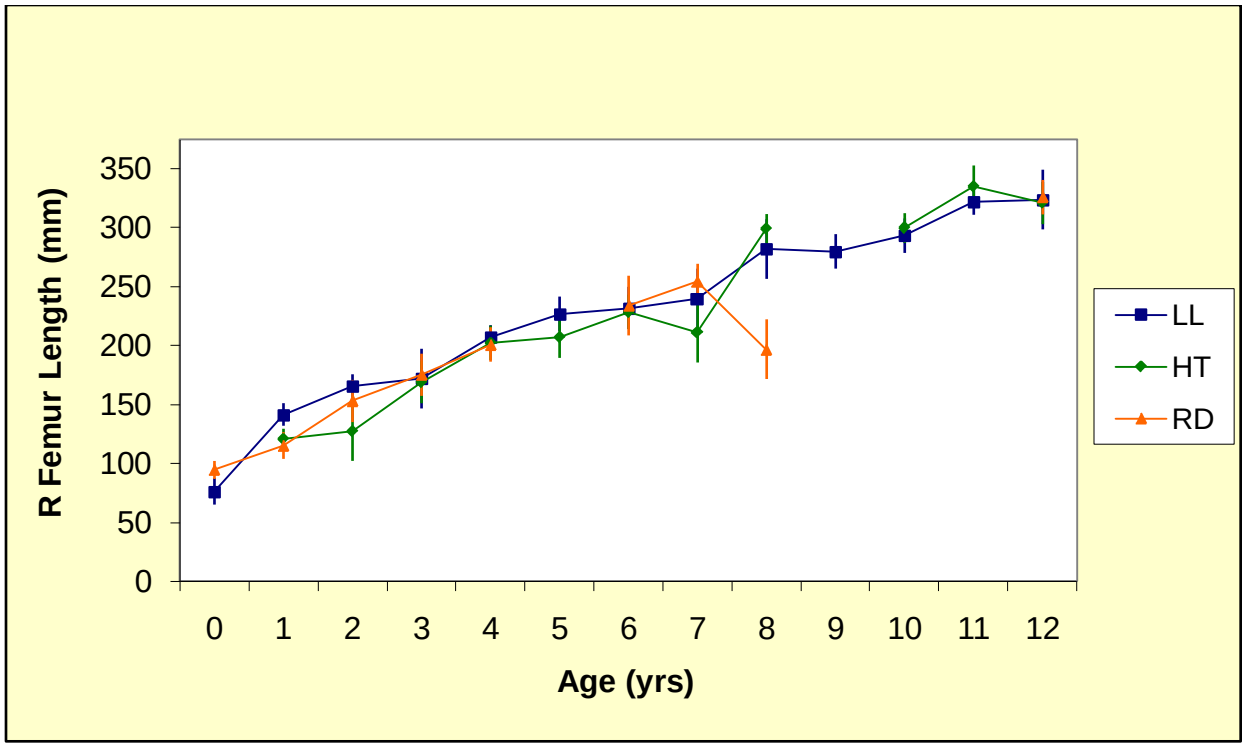

Figure 7. Growth curve by sample: mean femoral length (and SE) x age category for each sample.

Table 14. Mean femoral length (with SE and 95% CI) by age category for each sample

	LL				HTH				RD			
			95% CI				95% CI				95% CI	
Age	**Mean**	**SE**	**Lower**	**Upper**	**Mean**	**SE**	**Lower**	**Upper**	**Mean**	**SE**	**Lower**	**Upper**
0	76.4	11.3	53.83	98.97					94.46	7.64	79.238	109.67
1	141.57	9.58	122.5	160.65	121	8.45	104.18	137.82	115.2	11.33	92.63	137.77
2	165.33	10.3	144.73	185.94	127.5	25.33	77.033	177.97	153	17.91	117.31	188.69
3	172	25.3	121.53	222.47	169	17.91	133.31	204.69	175.25	17.91	139.56	210.94
4	207	10.3	186.4	227.6	202.33	14.63	173.2	231.47	201	14.63	171.86	230.14
5	227	14.6	197.86	256.14	207.5	17.91	171.81	243.19				
6	232	17.9	196.31	267.69	228.67	14.63	199.53	257.8	234	25.33	183.53	284.47
7	240	25.3	189.53	290.47	211	25.33	160.53	261.47	254.83	14.63	225.7	283.97
8	282	25.3	231.53	332.47	299	12.67	273.77	324.23	197	25.33	146.53	247.47
9	280	14.6	250.86	309.14								
10	293.33	14.6	264.2	322.47	299.75	12.67	274.52	324.98				
11	322.4	11.3	299.83	344.97	335	17.91	299.31	370.69				
12	324	25.3	273.53	374.47	321	17.91	285.31	356.69	326	14.63	296.86	355.14

Table 15. Results of ANOVA of femoral length by sample (controlling for age)

df	F	sig
18	1.317	0.203

Levene's Test of Equality of Error Variances			
F	df1	df2	sig
1.796	32	75	0.02

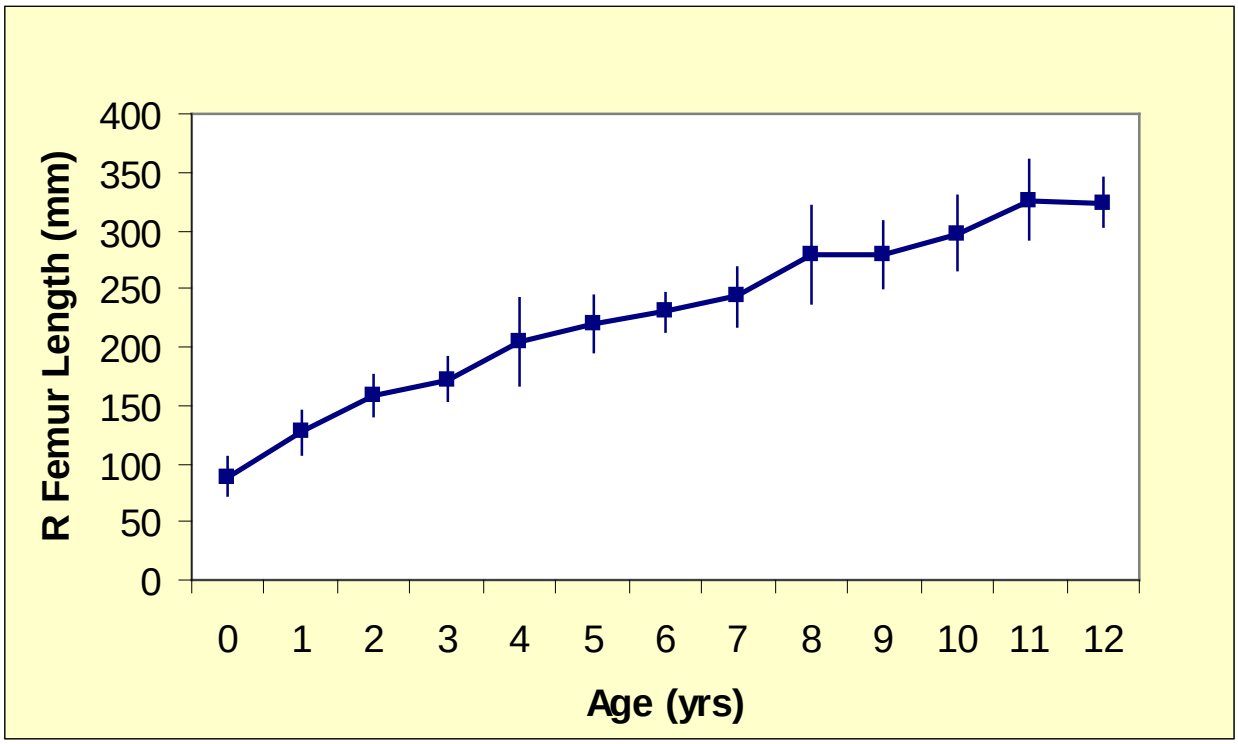

Figure 8. Growth curve of combined sample: mean femoral length (with SD) x age category

Table 16. Mean femoral length (with SD) by age category for combined sample

Age	Mean	SD
0	88.81	16.63
1	126.48	19.3
2	158.39	17.11
3	172.1	18.09
4	204.33	38.38
5	219.2	24.01
6	230.67	16.48
7	243.1	25.78
8	279.17	42.18
9	280	28.48
10	297	32.09
11	326	34.52
12	324	20.78

4.4 MORTALITY BIAS

T-tests of mean femoral length for the combined sample and Maresh's (1955) published means were conducted for each age category in order to identify the presence of mortality bias. The results show that the degree of mortality bias is significant ($p < 0.05$) at nine of the thirteen age categories. Means of the combined sample and Maresh's (1955) along with significance of each

age category are given in Table 17. Graph 9 shows the growth curves for the combined sample and Maresh (1955).

Table 17. Mean femoral lengths for combined sample (with SD) and Maresh (1955). T-test values by age category.

	Combined Sample		Maresh		
Age	**Mean**	**SD**	**Mean**	**df**	**sig**
0	88.81	16.63	97.91	15	0.045
1	126.48	19.3	144.5	20	0.000
2	158.39	17.11	175.08	8	0.019
3	172.1	18.09	201.83	4	0.021
4	204.33	38.38	225.42	11	0.083
5	219.2	24.01	246.64	4	0.063
6	230.67	16.48	270.53	5	0.002
7	243.1	25.78	291.71	4	0.014
8	279.17	42.18	311.9	5	0.116
9	280	28.48	330.81	2	0.091
10	297	32.09	348.69	6	0.005
11	326	34.52	367.31	6	0.019
12	324	20.78	382.77	5	0.001

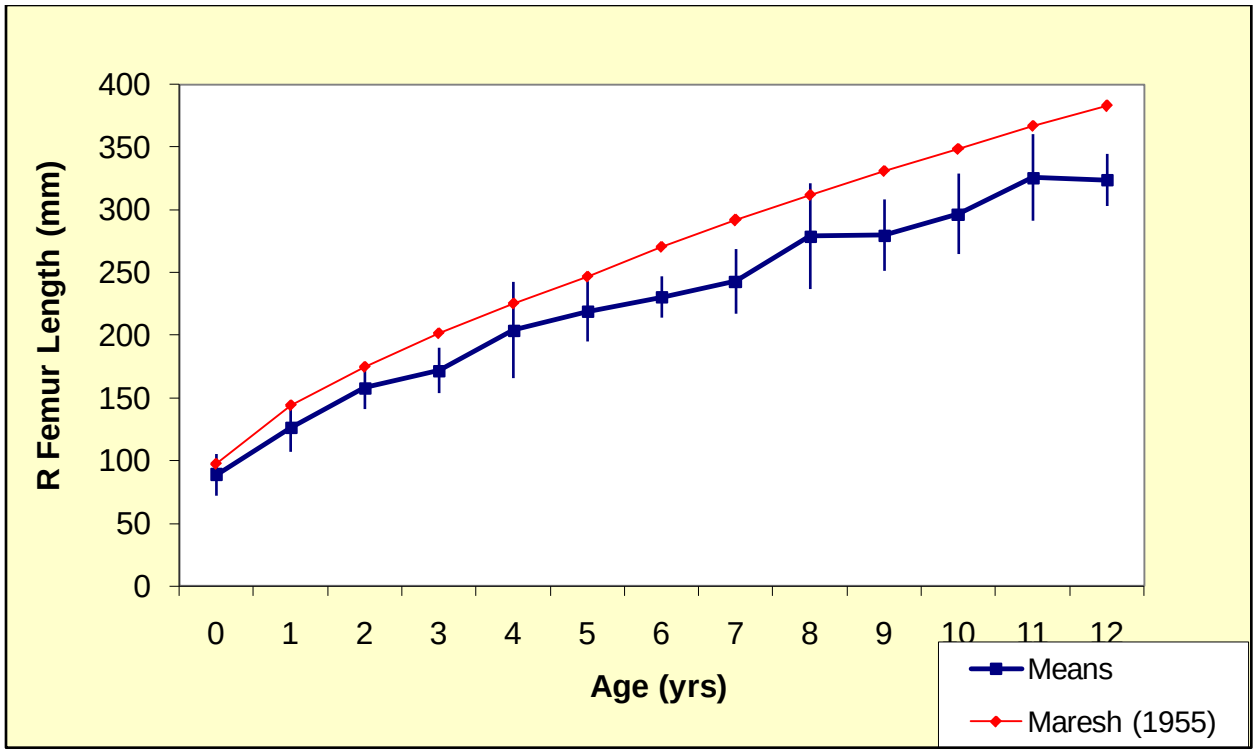

Figure 9. Growth curve of combined sample vs. Maresh (1955): mean femoral length x age category

4.5 PATHOLOGY

An independent T-test was performed to determine the difference between the femoral lengths of individuals included in pathological categories and those included in the normal subset of the sample. The difference between the pathological and normal samples is insignificant (p=0.25) (see Table 18).

Table 18. Result of T-test of femoral length difference by pathology or no pathology

t	df	sig
1.157	109	0.25

ANOVA was used to determine the strength of the difference in femoral lengths between pathological categories (controlling for age). The femoral lengths do not vary significantly (p= 0.388) between the pathological categories (see Table 19). Mean femoral lengths are given for each pathological category in Table 20. The mean femoral lengths and SE are plotted for each pathological category in Graph 10. Graph 11 shows the means and SEs for each pathology plotted with the growth curve for the combined sample.

Table 19. Results of ANOVA of femoral length by pathology (controlling for age)

df	**F**	**sig**
23	1.104	0.388

Levene's Test of Equality of Error Variances			
F	**df1**	**df2**	**sig**
17.95	43	35	0.00

Table 20. Mean femoral length (with SE and 95% CI) by age category for each pathology

	CS*	DEH				PH				TB			
				95% CI				95% CI				95% CI	
Age	**Mean**	**Mean**	**SE**	**Lower**	**Upper**	**Mean**	**SE**	**Lower**	**Upper**	**Mean**	**SE**	**Lower**	**Upper**
0	88.81												
1	126.48					264.50	28.37	206.90	322.10				
2	158.39									168.00	28.37	110.40	225.60
3	172.1												
4	204.33	239.50	28.37	181.90	297.10	190.00	40.13	108.54	271.46	221.00	28.37	163.40	278.60
5	219.2	195.00	40.13	113.54	276.46	195.00	40.13	113.54	276.46	243.00	28.37	185.40	300.60
6	230.67					238.50	28.37	180.90	296.10	238.50	28.37	143.54	306.46
7	243.1					226.50	40.13	145.04	307.96				
8	279.17									279.00	40.13	197.54	360.46
9	280									255.00	40.13	173.54	336.46
10	297					268.50	28.37	210.90	326.10				
11	326					367.00	40.13	285.54	448.46	320.50	28.37	262.90	378.10
12	324					319.67	23.17	272.64	366.70	327.00	40.13	245.54	408.46

*CS= Combined Sample

Table 20. Continued

	CS*	PN				MAL				GE			
				95% CI				95% CI				95% CI	
Age	**Mean**	**Mean**	**SE**	**Lower**	**Upper**	**Mean**	**SE**	**Lower**	**Upper**	**Mean**	**SE**	**Lower**	**Upper**
0	88.81	92.75	20.06	52.02	133.48	76.00	40.13	-5.46	157.46	102.40	17.94	65.97	138.83
1	126.48	126.33	16.38	93.08	159.58	107.50	28.37	49.90	165.10				
2	158.39	164.00	28.37	106.40	221.60								
3	172.1	163.00	28.37	105.40	220.60								
4	204.33	179.00	28.37	121.40	236.60	128.00	40.13	46.54	209.46				
5	219.2												
6	230.67	239.00	40.13	157.54	320.46	200.00	40.13	118.54	281.46				
7	243.1												
8	279.17												
9	280												
10	297												
11	326	321.00	40.13	239.54	402.46								
12	324												

*CS= Combined Sample

Table 20. Continued

	CS*	M2				M3				M4			
				95% CI				95% CI				95% CI	
Age	**Mean**	**Mean**	**SE**	**Lower**	**Upper**	**Mean**	**SE**	**Lower**	**Upper**	**Mean**	**SE**	**Lower**	**Upper**
0	88.81	102.00	40.13	20.54	183.46								
1	126.48	119.00	20.06	78.27	159.73					107.00	40.13	25.54	188.46
2	158.39												
3	172.1	189.75	28.37	132.15	247.35								
4	204.33	181.00	28.37	123.40	238.60								
5	219.2	220.00	40.13	138.54	301.46								
6	230.67	243.00	40.13	161.54	324.46								
7	243.1	211.00	40.13	129.54	292.46								
8	279.17	303.00	40.13	221.54	384.46	307.00	28.37	249.40	364.60				
9	280												
10	297	320.00	28.37	262.40	377.60								
11	326	304.00	28.37	246.40	361.60								
12	324	343.50	28.37	285.90	401.10	315.00	40.13	233.54	396.46				

*CS= Combined Sample

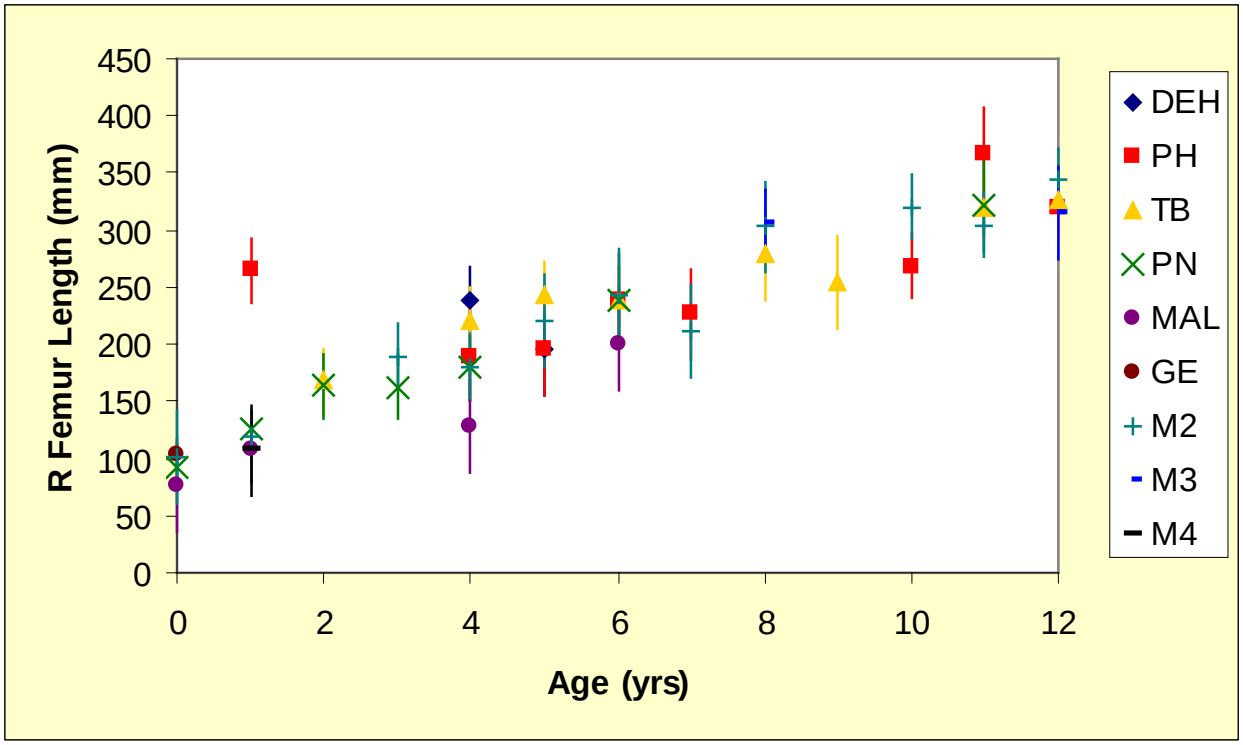

Figure 10. Growth by pathology: mean femoral length (with SE) by age category for each pathology.

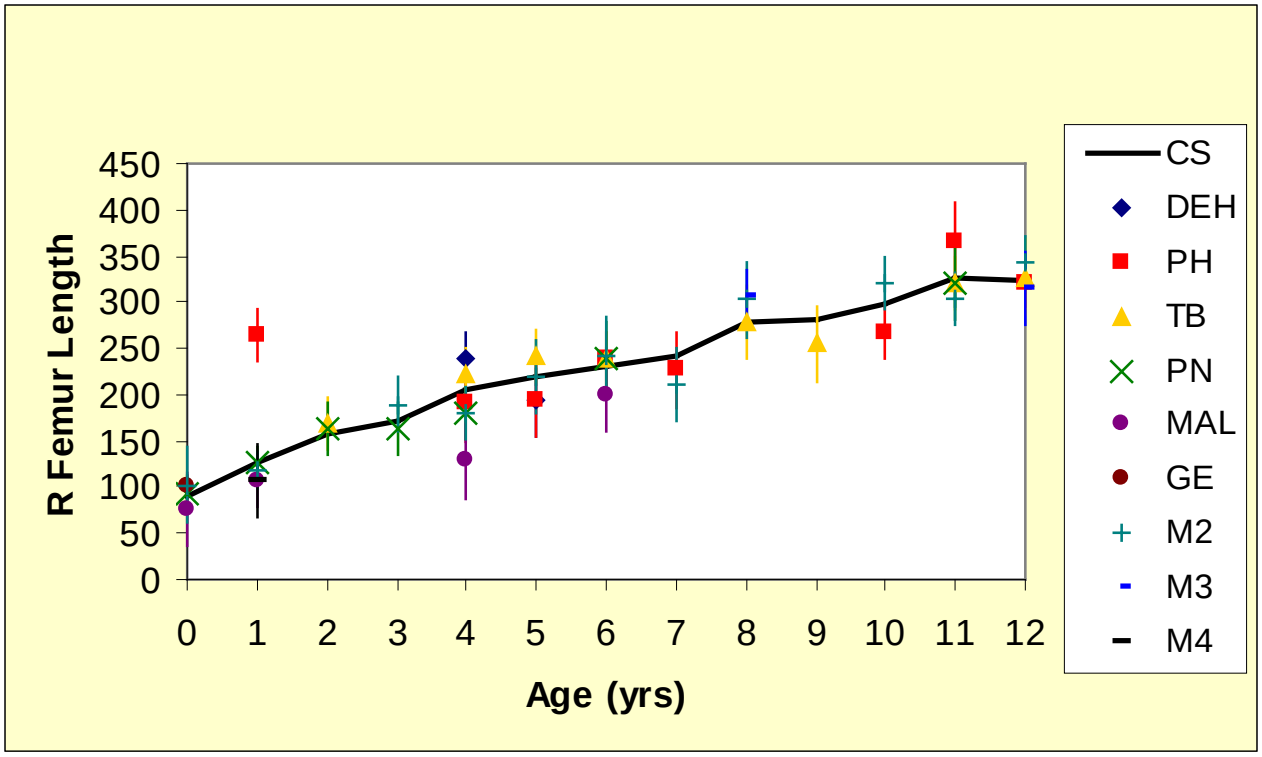

Figure 11. Mean femoral length (with SE) by age category for each pathology- plotted against growth curve for combined sample

Each pathological category was analyzed separately against the normal sample. ANOVA was used to determine the difference between femoral lengths of each individual pathological category against the normative sample (controlling for age). The results are presented in Table 21. DEH, M3 and M4 had too few values for analysis. TB, PN, and M2 had insignificant results (p= 0.154, 0.302, and 0.334, respectively). PH and MAL were the only pathological categories to return significant values (p=0.022 and p=0.016, respectively), however, PN is significantly higher than the normal sample.

Table 21. Results of ANOVA of femoral length for each pathology x normal sample

Pathology	df	F	sig
DEH	0	---	---
PH	5	3.21	0.022*
TB	5	1.771	0.154
PN	6	1.261	0.302
MAL	3	5.008	0.016
GE**			
M2	8	1.196	0.334
M3	0	---	---
M4	0	---	---

** See Table 22

GE was analyzed using a T-test because information was available only for one age category (Age 0). The difference between femoral lengths in the GE category and the normal sample at age 0 is significant (p=0.02) (Table 22); as with the results of the PH analysis, GE femoral lengths are significantly larger than their normal counterparts.

Table 22. Result of T-test for GE: femoral lengths of pathology x normal sample

Pathology	t	df	sig
GE	-2.765	10	0.02

5.0 DISCUSSION

Growth during childhood is assumed to be the best indicator of the health status of the child, consequently nutritional studies of living populations use anthropometric measurements to determine children's health, as do demographic and growth studies of skeletal juveniles. However, childhood growth is a very complex and dynamic process involving the interaction of heredity, and internal and external environmental factors.

The analysis of juvenile skeletal populations requires the observer to bear in mind several issues: sexual dimorphism, population variation, mortality bias, and pathology. Sexual dimorphism must be investigated in order to determine the degree of intra-population variation that is not the result of pathology or heredity; so must population variation. There are three types of mortality bias (discussed in the Introduction), and any of them can alter the composition of and the conclusions drawn from the sample. Disease and malnutrition have long been held to retard skeletal growth, and as such, have been used by anthropologists to interpret health status of individuals and populations via skeletal growth.

Three skeletal collections (Luis Lopes, Hamann-Todd, and Raymond Dart) were analyzed in order to address the issues affecting the analysis of skeletal collections listed previously. These samples were chosen because of their abundant records, which provided at least information on

age, sex, and cause of death for each individual, as well as their close temporal proximity. The overlapping chronology of the collections minimized the problem of a secular trend towards increased height.

5.1 SEXUAL DIMORPHISM

The distribution of sexes within each of the samples is nearly equal (Table 5), with males comprising a slight majority of each sample. The greatest difference between the number of males and females within one collection (RD) is only 3 individuals (Table 12). The three samples were combined for statistical analysis, giving a final sample of 57 males (52.8%) and 51 females (47.2%). For a complete distribution of sex by age category for each sample, see Table 12.

Mean femoral lengths for each age category (Table 13) show that males and females followed nearly identical growth curves. Only at ages 7, 8, and 10 does the mean femur length of one sex not fall within the 95% CI of the other sex; however, this dimorphism does not follow a single trend. At age 7, females fall below the 95% CI of males, but at age 8 they are above the 95% CI of males. At age 10 females again fall below the 95% CI of males. These discrepancies are most likely due to the small sample size.

5.1.1 Conclusion

Within the combined sample, the amount of sexual dimorphism in the femoral lengths of the males and females is insignificant ($p=0.367$). These findings support the idea that sexually

dimorphic patterns of linear skeletal growth do not appear until adolescence (Humphrey, 1998). The problem of sexually dimorphic growth of the forearm bones seen in the works of Maresh (1955) and Gindhart (1973) was not addressed by this analysis, and so remains unchallenged.

5.2 POPULATION VARIATION

Variation in growth between populations is widely debated. Studies on both living and skeletal samples have provided proof for both sides of the argument. Martorell et al (1975), Merchant and Ubelaker (1977), Sundick (1978), and Saunders et al (1993) all found that the growth between children of different populations showed minimal variation. The WHO Multicentre Growth Reference Study (MGRS) (2006) even released an international growth standard that was applicable to all populations.

The three samples in this study were analyzed for the presence of population variation. The populations were geographically discrete, with no admixture. Given the vast differences between the hereditary, social, and geographical components of the samples, a significant degree of population variation would be expected. Growth curves for each population are seen in Graph 7, and a scatterplot of all data points used in this analysis are shown in Graph 6. From a precursory analysis of the scatterplot and population growth curves, no difference between the populations is immediately recognizable. The data points for the three samples form a tight pattern, and the growth curves overlap each other in many places. The only noticeable divergence is in the Raymond Dart collection at age 8 years, where the mean dips well below the Luis Lopes and

Hamman-Todd collections. The mean femoral lengths for each age category by sample bears out this observation (Table 14).

ANOVA of mean femoral lengths between each of the three samples (adjusted for age) shows that the degree of population variation is insignificant ($p= 0.203$) (Table 15). The similarity of growth of three such disparate populations which all had individuals suffering from numerous diseases and nutritional deficits is amazing given the studies by Johnston (1962), Buzina (1976), Eveleth and Tanner (1976), and Bogin (1999), all of whom report that the difference in linear growth between populations is significant.

5.2.1 Conclusion

This result supports the findings of Martorell et al (1975), who demonstrated the universality of human growth for preschool children raised under good nutritional and environmental conditions, regardless of genetic or ethnic background. These data also raise the possibility that the trend Martorell et al (1975) described can be extended beyond preschool-aged children, to include all individuals prior to adolescence. The result of this analysis supports the call for a single international growth standard.

5.3 MORTALITY BIAS

Research directly examining biological mortality bias is rare. McGregor et al (1961; 1968) investigated growth and mortality of children in a rural Gambian village and found that the

difference in growth curves between the children who lived and the children who died were very similar. Most of the literature that deals with mortality bias provides hypotheses and opinions without supporting data (Johnston, 1968; Saunders and Hoppa, 1993; Steyn, 1996; Saunders, 2001).

Mortality bias was analyzed using the combined sample and Maresh's (1955) study of contemporaneous living children (for more on the Maresh sample, see Methods section). T-tests were used to compare the combined sample to Maresh (1955) at each age category. Mean femoral lengths along with significance for each age category are given in Table 17. Mean femoral lengths for the combined sample are smaller than Maresh's (1955) at each age category, and nine of the 13 age categories show significant differences ($p<0.05$) between the combined sample and Maresh (1955). The nine significant age categories form three distinct groupings. It is interesting to note that the ages at which the significant differences appear correspond to phases of rapid growth prior to adolescence.

From age 0 through age 3, the combined sample is significantly smaller than Maresh's (1955). This supports Saunders and Hoppa's (1993) conclusions that the period from birth until about three years of age is the most crucial with respect to stunting. The first three years of life are what Bogin (1999) calls the "infancy stage", which is characterized by the most rapid growth velocity of any of the postnatal stages; introduction of adverse factors during this stage would have a considerable effect on growth (Saunders and Hoppa, 1993; Bogin, 1999). The significant difference between the skeletal samples (whose pathologies are well documented) and the living

sample of healthy, well-fed children seems to support the findings of Saunders and Hoppa (1993) and Bogin (1999).

At ages 4 and 5, the difference between the two samples drops below significant ($p=0.083$ and $p=0.063$, respectively), however, the significance returns again at ages 6 and 7 ($p=0.002$ and $p=0.014$, respectively) (Table 17). Bogin (1999) defines the stage immediately following "infancy" as the "childhood stage", which lasts from about age 3 to age 7. The childhood stage (ages 6 to 7) ends in the mid-growth spurt, in which children experience an increase in growth velocity (Bogin, 1999). As with the infancy stage, the introduction of a factor adversely affecting growth during a time of rapid growth would necessarily produce individuals smaller than their peers who are not experiencing growth retardation.

At ages 8 and 9 there is a return to an insignificant ($p= 0.116$ and $p=0.091$, respectively) difference between the combined sample and Maresh (1955); this changes at 10 years, at which age the combined sample dips significantly ($p<0.05$) below the femoral lengths of Maresh (1955) and stays there for the remainder of the age categories (Table 17). Following childhood is the juvenile stage (Bogin, 1999), which lasts from about age 7 to about age 13 in males, and from approximately age 7 to 10 in females. The juvenile stage is succeeded by the adolescent stage, during which time there is acceleration in the growth rate. At ages 11 and 12, there are more females in the sample than males (5:2, and 6:1, respectively) (Table 12). What may account for some of the difference between the combined sample and Maresh (1955), is that female growth begins to accelerate at an earlier age and there are a larger number of females than males at the older ages.

The non-significant differences in femoral length between the combined and Maresh's (1955) samples occur during periods in which growth is normally slow or decelerating. The childhood phase is much slower than the preceding infancy phase, and the juvenile phase is often accompanied by a pronounced, but short-lived, decrease in rate of growth (Bogin, 1999). The slow growth rate during these ages would give subadults who had experienced depressed growth a period in which catch-up growth would counteract any previous difference in femoral length between them and their peers. This interpretation, however, is dependent upon reading the growth curve for a population as if it was an individual's growth curve. Overall, the effects of catch-up growth within the combined sample would be difficult to support because the sample is cross-sectional and does not reflect any one individual's growth rate.

5.3.1 Conclusion

The results of these analyses suggest that biological mortality bias does exist, and is a significant factor affecting juvenile skeletal remains.

5.4 PATHOLOGY

There is little clear evidence of the specific effects during childhood of various diseases on growth rates and patterns (Saunders and Hoppa, 1993). Most of the literature about the effects of disease and malnutrition on childhood skeletal growth is conflicting (see Introduction for a literature review).

Nine pathological categories were analyzed within the combined sample. These categories are listed in Table 18.

ANOVA of femur length by pathology (controlling for age) was conducted to test strength of the difference between the pathologies (Table 19). The difference between the femoral lengths of each of the pathological categories was not significant (p=0.388). This contradicts the result of Martorell et al (1975), who found that children suffering from diarrhea experienced growth retardation, while respiratory infections had no effect on growth.

Levene's test of quality of error variances was run with the ANOVA of femur length by pathology. The result of the Levene's test is significant (p=0.00) (Table 19). However, ANOVA is a robust statistical tool, and the size of the sample is small, thus, this result most likely reflects the small sample size.

A T-test was used to examine the strength of the difference of femoral lengths between pathological and non-pathological individuals (Table 18). The difference in femoral lengths between individuals in the pathological categories and those in the normal subset of the data was not significant (p=0.25). A precursory examination of the mean femoral lengths (with SE) for each pathological category plotted by age against the growth curve for the combined sample (see Graph 11) shows that many of the pathological categories closely overlay the curve.

Following the surprising results of the pathology T-test, the data was mined for more information. ANOVA was conducted between each pathological category and the normal subset

of the sample (controlling for age) in order to further analyze the effects of malnutrition and disease on skeletal growth (Table 21 and Graphs 10 and 11).

5.4.1 Insufficient Specimen Number: DEH, M3, M4– Descriptive trends:

Three pathological categories (DEH, M3, and M4) lack sufficient specimens in the age categories for statistical analysis. The mean femoral lengths for these three categories are given in Table 20. DEH means are available for ages 4 and 5. At age 4, DEH is well above the combined sample mean, while at age 5 it is below the sample mean. In both cases, the sample mean falls within the 95% CI of DEH. M3 follows a pattern similar to DEH; age 8 is above the combined sample mean, while age 12 is below it, but the combined sample mean falls within the 95% CI of both M3 age groups. A mean femur length was available for M4 only at age 1 and it fell below the combined sample mean for age 1; however, the combined sample mean was within the 95% CI range of M4. There is no distinct trend in the mean femoral lengths of the three pathological categories for which statistical analysis was not possible, which suggests that none of these pathologies significantly influenced the growth of the individuals suffering from them.

5.4.2 Non-Significant Results: TB, PN, M2:

Three pathological categories (TB, PN, and M2) had results that were statistically insignificant ($p<0.05$) (Table 21).

5.4.2.1 TB

Tuberculosis is a chronic disease that is well-documented for causing wasting (Schwenk and Macallan, 2000). Chronic disease is more likely to impact growth because it affects the individual over a greater span of time. The wasting associated with tuberculosis is the effect of nutritional deficiency, which would be expected to amplify any inhibitory effects that the disease has on growth. Despite the consumptive effects on the lungs and body, the individuals within the combined sample who suffered from tuberculosis had femur length comparable to their healthier counterparts ($p=0.154$) (Table 21). This seems to support Martorell et al's (1975) findings that respiratory infections had little effect on growth; however, Martorell et al (1975) focused on minor respiratory infections (i.e. pneumonia), not major and chronic illnesses like tuberculosis.

5.4.2.2 PN

Pneumonia is a relatively minor acute respiratory illness and thus would not be anticipated to have a significant effect on growth. The results of the ANOVA (Table 21) show that this expectation is true, the presence of pneumonia has little effect on femur length ($p=0.302$), which supports the work of Martorell et al (1975).

5.4.2.3 M2

M2 is a broad category that included individuals with any combination of two pathologies; as such, the effects of the pathologies on the femoral lengths would be expected to be amplified. The results of the ANOVA (Table 21) show that the difference in femur length between individuals suffering from two pathologies and those in the normal sample are not significant ($p=0.334$).

5.4.3 Significant Results: MAL, PH, GE

5.4.3.1 MAL

Malnutrition in children has been associated with stunted growth, delayed mental development, blindness, night blindness, and immunodeficiency, among other ailments (Allen, 1995; WHO, 2000). Immunodeficiency resulting from a lack of proper nutrition also leaves children at risk for the contraction of infectious disease. Longitudinal growth retardation has been frequently cited in studies of malnourished children (Howe and Schiller, 1952; Tanner, 1962; McCance, 1971; Eveleth and Tanner, 1976; Himes, 1978; Tanner, 1978; Lumey et al, 2007); however, some evidence from similar studies contradicts these findings (Rao and Singh, 1970). ANOVA conducted on femoral lengths between the malnourished and normal samples indicates that a significant difference ($p=0.016$) exists. These results support the conclusions of earlier authors, that malnutrition has a significant inhibitory effect on longitudinal growth.

5.4.3.2 PH

Porotic hyperostosis is linked to iron-deficiency anemia, which is strongly associated with infectious disease (Lallo, 1977). It is believed that iron-deficiency leaves the sufferer at risk for infection, which in turn depletes stores of iron. Iron-deficiency anemia has also been found to significantly retard growth if manifested during the first two years of life (Soliman, 2009). PH showed a significant difference in femur length ($p=0.022$) (Table 21). This result seems to support the findings of Soliman (2009), except that the mean femoral lengths for individuals with PH are higher in several age categories than the mean femoral lengths of the normal sample (see Table 20). Graph 11 illustrates this point well.

Several variables could account for this curious result. First, instead of retarding growth, porotic hyperostosis accelerates it. This explanation can be quickly discounted because there is no research or literature supporting it. Second, the sample of pathological individuals is biased because of inaccurate records on some specimens. An inaccurate chronological age, in which an older child is placed into a younger category, would easily account for the cases where the individuals with porotic hyperostosis have longer femoral lengths than the normal sample. This is possible; however, it is unlikely that a sufficient amount of error exists in the collections' records to account for these results. The most likely explanation is that porotic hyperostosis does not have a truly significant effect on growth, because, if this was the case, the data would go in one direction: i.e. all individuals suffering from porotic hyperostosis would either be smaller or taller than their healthy counterparts.

5.4.3.3 GE

Gastroenteritis is an inflammation of the GI tract that results in acute diarrhea. It is usually caused by infection and is currently the leading cause of death among children under 5 (King et al, 2003). The disease is acute and affects the nutritional status of the individual. Martorell et al (1975) found that growth in children suffering from diarrhea was substantially smaller than their healthier counterparts.

Gastroenteritis was not included in the ANOVA because the individuals with it were all in one age category (age 0). A t-test was used to analyze the difference in femur lengths at age 0 between individuals with GE and those in the normal sample (Table 22); a significant difference ($p=0.02$) was found. This seems to support the findings of Martorell et al (1975), except that the mean femoral lengths for children with GE were higher than the femoral lengths of the children

in the normal sample (see Table 20). The results of the t-test indicate that children suffering from GE are substantially taller than those who do not have the disease (see Graph 11).

The results of the t-test for GE are unexpected, and there are several scenarios that could account for them. The first scenario has children with GE (a disease that severely affects nutritional status) growing better than children unaffected by the disease. But there is no supporting evidence for this. As with porotic hyperostosis, the higher mean femoral lengths could result from inaccurate record keeping. This is possible. However, the most likely explanation is that the small sample size inordinately skews the data towards individuals with GE, thus yielding a higher mean femoral length than the normal sample. Until further analyses are conducted on larger cohorts, we are left only to concur with the study by Condon-Paoloni et al (1977), who found no great growth discrepancy between individuals with high or low frequencies of diarrheal disease.

5.4.4 Conclusion

Of the six pathologies tested, the only one found to significantly retard skeletal growth is malnutrition ($p=0.016$). These results seem to indicate that many diseases have little effect on skeletal growth, and that children will continue to grow at nearly normal rates under most adverse conditions, except malnourishment.

The nature of the pathology results leads to two further questions: 1) Is the normal sample really normal? And 2) are the categories robust enough to yield firm results?

The normal sample was comprised of children who died of a variety of causes, many of which were sudden and accidental. Thus, it would appear that the normal sample is fairly well representative of children who experienced typical growth. In the absence of extensive medical records on each specimen, we cannot definitively rule out the possibility that these were not healthy individuals during life, and so, the adequacy of the sample can only be judged by the information provided.

In answer to the second question, once the pathological sample was broken into separate pathologies for individual analysis, the sample sizes became quite small. The results of the ANOVA of the individual pathologies are thus tentatively given with the small sample size in mind. Analysis of further collections, providing a more robust sample, would address this problem.

BIBLIOGRAPHY

2000. Turning the tide of malnutrition: responding to the challenge of the 21st century. Geneva: WHO.

2005. Cleveland,Ohio. Ohio History Central. http://www.ohiohistorycentral.org/entry.php?rec=687.

Acheson RM. 1959. Effects of starvation, septacaemia and chronic illness on the growth cartilage plate and metaphysis of the immature rat. J Anat 93:123-130.124.

Allen LH. 1995. Malnutrition and human function: a comparison of conclusions from the INCAP and nutrition CRSP studies. J Nutr 125:1119S-1126S.

Baker DH. 2008. Animal models in nutrition research. J Nutr 138:391-396.

Blom DE, Buikstra JE, Keng L, Tomczak PD, Shoreman E, and Stevens-Tuttle D. 2005. Anemia and childhood mortality: Latitudinal patterning along the coast of pre-Columbian Peru. Am J Phys Anth 127(2):152-169.

Bogin B. 1979. Monthly changes in the gain and loss of growth in weight of children living in Guatemala. Am J Phys Anth 51:287-291.

Bogin B. 1999. Patterns of Human Growth. Cambridge: Cambridge University Press.

Buzina R. 1976. Growth and development of three Yugoslav populations in different ecological settings. Am J Clin Nutr 29:1051-1059.

Cardoso HFV. 2006. Brief Communication: The Collection of Identified Human Skeletons Housed at the Bocage Museum (National Museum of Natural History), Lisbon, Portugal. Am J Phys Anth 129:173-176.

Cardoso HFV. 2007. Environmental effects on skeletal versus dental development: Using a documented subadult skeletal sample to test a basic assumption in human osteological research. Am J Phys Anth 132:223-233.

Cardoso HFV. 2008. Secular changes in body height and weight of Portuguese boys over one century. Am J Hum Biol 20:270-277.

Cardoso H, and Garcia S. 2009. The Not-So-Dark Ages: Ecology for Human Growth in Medieval and Early Twentieth Century Portugal as Inferred From Skeletal Growth Profiles. Am J Phys Anth 138:136-147.

Cobb W. 1932. Human Archives at the Case Western Reserve Department of Anatomy.

Condon- Paoloni D, Cravioto J, Johnston FE, de Licardie ER, and Scholl TO. 1977. Morbidity and growth of infants and young children in a rural Mexican village. Am J Public Health 67:651-656.

Cook DC. 1981. Mortality, age structure and status in the interpretation of stress indicators in prehistoric skeletons: A dental example from the Lower Illinois Valley. In: Chapman R, Kinnes I, and Randsborg K, editors. The Archaeology of Death. Cambridge: Cambridge University Press. p 133-144.

De Luca F. 2006. Impaired growth plate chondrogenesis in children with chronic illness. Pediatr Res 59(5):625-629.

Eveleth PB, and Tanner JM. 1976. Worldwide Variation in Human Growth. Cambridge: Cambridge University Press.

Garn SM. 1965. The applicability of North American growth standards in developing countries. Canad Med Ass J 93.

Gindhart PS. 1973. Growth standards for the tibia and radius in children aged one month through eighteen years. Am J Phys Anth 39:41-48.

Goodman AH, and Armelagos GJ. 1985. Factors affecting the distribution of enamel hypoplasias within the human permanent dentition. Am J Phys Anth 68(4):479-493.

Goodman AH, Armelagos GJ, and Rose JC. 1984. The chronological distribution of enamel hypoplasias from prehistoric Dickson Mounds populations. Am J Phys Anth 65(3):259-266.

Gunnell DJ, Smith GD, Frankel S, Nanchahal K, Braddon FE, Pemberton J, and Peters TJ. 1998. Childhood leg length and adult mortality: follow up to the Carnegie (Boyd Orr) survey of diet and health in pre-war Britain. J Epidemiol Community Health 52:142-152.

Hardy MC. 1938. Frequent illness in childhood, physical growth and final size. Am J Phys Anth 23:241-260.

Hillson S. 1996. Dental Anthropology: Cambridge University Press.

Hillson S, and Bond S. 1997. Relationship of enamel hypoplasia to the pattern of tooth crown growth: A discussion. Am J Phys Anth 104:89-103.

Himes JH. 1978. Bone growth and development in protein-calorie malnutrition. Wld Rev Nutr Diet 28:142-187.

Hoppe C, Mølgaard C, and Michaelsen KF. 2000. Bone size and bone mass in 10-year-old Danish children: Effect of current diet. Osteoporos Int 11:1024-1030.

Hoppa R, and Garlie TN. 1998. Secular changes in the growth of Toronto children during the last century. Ann Hum Biol 25(6):553-561.

Howe PE, and Schiller M. 1952. Growth responses of the school child to changes in diet and environmental factors. J Appl Physiol 5(2):51-61.

Hummert JR, and Van Gerven DP. 1983. Skeletal growth in a Medieval population from Sudanese Nubia. Am J Phys Anth 60:471-478.

Humphrey LT. 1998. Growth Patterns in the Modern Human Skeleton. Am J Phys Anth 105:57-72.

Hutchinson DL, and Larsen CS. 1988. Determination of stress episode duration from linear enamel hypoplasias: a case study from St. Catherines Island, Georgia. Hum Biol 60(1):93-110.

Jantz LM, and Jantz RL. 1999. Secular changes in long bone length and proportion in the United States, 1800-1970. Am J Phys Anth 110(1):57-67.

Jantz RL, and Owlsley DW. 1984. Long bone growth variation among Arikara skeletal populations. Am J Phys Anth 63(1):13-20.

Johnston FE. 1968. Growth of long bones of infants and young children at Indian Knoll. Am J Phys Anth 20:249-254.

Lallo JW, Armelagos GJ, and Mensforth RP. 1977. The role of diet, disease, and physiology in the origin of porotic hyperostosis. Hum Biol 49(3):471-483.

Lumey LH, Stein AD, Kahn HS, van der Pal-de Bruin KM, Blauw GJ, Zybert PA, and Susser ES. 2007. Cohort profile: The Dutch Hunger Winter families study. Int J Epidemiol 36:1196-1204.

Kemkes-Grottenhaler A. 2005. The short die young: The interrelationship between stature and longevity- Evidence from skeletal remains. Am J Phys Anth 128:340-347.

Kern KF. 2006. T. Wingate Todd: Pioneer of Modern American Physical Anthropology. Kirtlandia 55:1-42.

King CK, Glass R, Bresee JS, and Duggan C. 2003. Managing acute gastroenteritis among children: oral rehydration, maintenance, and nutritional therapy. MMWR Recomm Rep 52(RR-16):1-16.

Krishna M, and Upadhyay SS. 1996. Increased limb lengths in patients with shortened spines due to tuberculosis in early childhood. Spine 21(9):1045-1047.

Lee CM, Boileau A, Boileau TW, Williams AW, Swanson KS, Heintz KA, and Erdman JWJ. 1999. Review of animal models in carotenoid research. J Nutr 129(12):2271-2277.

Lovejoy CO, Russell KF, and Harrison ML. 1990. Long bone growth velocity in the libben population. Am J Hum Biol 2:533-541.

Maresh MM. 1955. Linear growth of long bones of extremities from infancy through adolescence; continuing studies. AMA Am J Dis Child 89(6):725-742.

Martorell R, Habicht JP, Yarbrough C, Leichtig A, Klein RE, and Western KA. 1975. Acute morbidity and physical growth in rural Guatemalan children. Am J Dis Child 129(11):1296-1301.

McCance RA. 1971. The effect of malnutrition on growth, metabolism and final form. Indian J Med Res 59(6 suppl):123-131.

McGregor IA, Billewicz WZ, and Thomson AM. 1961. Growth and mortality in children in an African village. Brit Med J 2(2):1661-1666.

McGregor IA, Rahman AK, Thompson B, Billewicz WZ, and Thomson AM. 1968. The growth of young children in a Gambian village. Trans Roy Soc Trop Med and Hyg 62(3):341-352.

McIntosh R. 1957. On growth and development: Parts I and II. Arch Dis Child 32(164):261-278.

Mensforth RP. 1985. Relative tibia long bone growth in the Libben and Bt-5 prehistoric skeletal populations. Am J Phys Anth 68:247-262.

Merchant VL, and Ubelaker DH. 1977. Skeletal growth of the Protohistoric Arikara. Am J Phys Anth 46:61-72.

Nilsson O, and Baron J. 2004. Fundamental limits on longitudinal bone growth: growth plate senescence and epiphyseal fusion. Trends Endocrinol Metab 15(8):370-374.

Nilsson O, and Baron J. 2005. Impact of growth plate senescence on catch-up growth and epiphyseal fusion. Pediatr Nephrol 20:319-322.

Oppenheimer SJ. 2001. Iron and Its Relation to Immunity and Infectious Disease. J Nutr 131(2S-2):633s-635s.

Rao KV, and Singh D. 1970. An evaluation of the relationship between nutritional status and anthropometric measurements. Am J Clin Nutr 23(1):83-93.

Saunders SR. 2000. Subadult skeletons and growth-related studies. In: Katzenberg MA, and Saunders SR, editors. Biological Anthropology of the Human Skeleton: Wiley-Liss. p 135-161.

Saunders SR, and DeVito C. 1991. Subadult Skeletons in the Raymond Dart Anatomical Collection: Research Potential. Hum Evol 6(6):421-434.

Saunders SR, and Hoppa R. 1993. Growth deficit in survivors and non-survivors: Biological mortality bias in subadult skeletal samples. Am J Phys Anth 36(S17):127-151.

Saunders SR, Hoppa R, and Southern R. 1993. Diaphyseal growth in a nineteenth century skeletal sample of subadults from St. Thomas' Church, Belleville, Ontario. Int J Osteoarchaeol 3:265-281.

Schwartz JH. 1995. Skeleton Keys. New York: Oxford University Press.

Schwenk A, and Macallan DC. 2000. Tuberculosis, malnutrition and wasting. Curr Opin Clin Nutr Metab Care 3(4):285-291.

Sciulli PW. 1994. Standardization of long bone growth in children. Int J Osteoarchaeol 4(3):257-259.

Soliman AT, Al Dabbagh MM, Habboub AH, Adel A, Humaidy NA, and Abushahin A. 2009. Linear Growth in Children with Iron Deficiency Anemia Before and After Treatment. J Trop Pediatr E pub ahead of print.

Steyn M, and Henneberg M. 1996. Skeletal growth of children from the Iron Age site at K2 (South Africa). Am J Phys Anth 100:389-396.

Stini WA. 1969. Nutritional stress and growth: Sex difference in adaptive response. Am J Phys Anth 31(3):417-426.

Sundick RI. 1978. Human skeletal growth and age determination. Homo 29:228-249.

Tanner JM. 1962. Growth at Adolescence. Oxford: Blackwell Scientific Publications.

Tanner JM. 1978. Foetus into Man: Physical Growth from Conception to Maturity. Cambridge, MA: Harvard University Press.

Widdowson EM. 1951. Mental contentment and physical growth. Lancet 1(6668):1316-1318.

Wu J. 1994. How severe was the Great Depression? Evidence from the Pittsburgh Region. In: Komlos J, editor. Stature, living standards, and economic development: essays in anthropometric history. Chicago and London: University of Chicago Press. 129-152.