2-4-2022 2:00 PM

The impact of energetic trade-offs on the developmental trajectory and life history strategy of Homo sapiens: The modern human female phenotype

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A thesis submitted in partial fulfillment of the requirements for the Master of Arts degree in Anthropology

Abstract

This study interrogates the relationship between early life environmental variability (measured through birth weight and age at menarche), and adult phenotypic outcomes in female athletes and non-athletes from the United Kingdom. Using anthropometric, and 3D body surface scan analysis, patterns of phenotypic variation were interpreted in a life history context. Significant correlations between birth weight, stature, and bi-iliac breadth were observed. Age at menarche had significant correlations with linear growth and body composition measures in both Pearson and Canonical Correlation analyses. Crural index was found to be negatively correlated with limb segment SA:Vol in opposition to the expectations of Allen's rule, a result that requires further investigation. Overall, variation in age at menarche was related to adult phenotypic variation, specifically linear skeletal growth, which may allow the development of regression equations that estimate life history variables in bioarchaeological populations, which would aid in more accurate interpretations of the past.

Key Words:

Life History Theory, Development Plasticity, Female Health, Athletic Paleobiology, Energetics, Anthropometrics, 3D Body Surface Scan

Summary for Lay Audiences

This study aims to investigate the relationships between variation in early life stress and nutrition and its impact on shaping variation in the adult phenotype (the amalgamation of observable physical characteristics, relating to the environmentally dependent expression of an individual's genetic code). It has been shown that children who are underweight at birth and/or who experience stressful or nutrient-restricted childhoods often have adverse developmental outcomes. This is reflected in their life history strategy (the individual timing and manifestation of specific developmental processes and milestones unique to a species) such that they mature earlier (often resulting in reduced stature), with an underdeveloped beta cell mass, and are more likely to develop a centralized fat distribution. These factors contribute to the manifestation of metabolic and cardiovascular diseases in adulthood. The role of early life variation in the manifestation of the life history strategy itself, and subtle variation in physical characteristics is less well understood.

In the following study, the physical characteristics of modern human females are examined through the use of anthropometrics, 3D body surface scans, and bioimpedance and subsequently compared to the early life indicator variables birth weight and age at menarche to better understand how variation in an individual's life history is reflected in their phenotype as it relates to reproduction, growth, and maintenance processes. Pearson correlation analyses were used to determine the directionality and degree of shared variance between phenotypic characteristics. Canonical correlation analyses were then used to determine the variable contributions of birth weight and age at menarche to variation in linear growth and body composition variable groups. Overall, age at menarche was shown to contribute more to phenotypic variation than birth weight.

This research is relevant both to understanding the effects of early life environmental variation in the manifestation of certain phenotypes in modern humans but also in better understanding the lived experiences of ancient humans. The clarification of the connection between different outcome characteristics in a modern population can aid in the interpretation of variation in skeletal populations, aiding researchers in developing more robust reconstructions of life in the past.

Acknowledgments

I would like to begin by thanking my supervisor Dr. Jay Stock, for this guidance and mentorship during my master's. Without your understanding and patience with me when things were hard, or when my computer(s) decided to self-implode, and your encouragement to "Get'er Done" when the motivation flowed, this project would not have been possible.

I would also like to thank Dr. Andrew Nelson for being my mentor through the later years of my undergrad and my advisor in my master's. Your passions for anthropology, research, and life are contagious and some of my fondest memories and friends have been created in your classrooms. Thank you for always encouraging me and providing opportunities to become a better researcher, academic, and person.

Many thanks to Dr. Kim Clark for encouraging me to apply to the master's program at Western, it was the right choice. Your open-door policy and chats kept me sane. Thanks to Dr. Lisa Hodgetts for seeing something in me and providing me with my first opportunity to do research in Arctic archaeology. Also, thanks to Dr. Murray at the University of Victoria for providing access to the data that was used in this study.

I want to thank my Anthro friends: Lauren, Adrianna, Nicole, Teegan, and Zsofia, whose friendships have followed me through my undergrad into my master's and have always been there for me as cheerleaders, copy editors, and sisters. Thank you also to my life-long friends, Morgan and Holly for your unconditional love and support.

I am thankful to have a family who has always been supportive of my academic and personal interests. Throughout my undergrad, I lived with my Aunt Mary and Uncle Gerry who picked me up from my evening chemistry labs in my first year and made sure I was alive and fed when I was studying through final exams. I can never repay you, and I am sorry I almost burnt down your house.

All the thanks and love to my partner David, who has for seven years aided with many midnight editing sessions and supported me in both school and life. I love you to the moon and back.

Lastly, thank you to my parents, Dr. Burr Atkinson, and Heather McLean, you are the reason I am where I am today. Your passions for the sciences and research are what guided my interest and love for science. Your support of my academic career has been unwavering, and I cannot thank you enough for all that you have done and continue to do.

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1. Introduction

The incorporation of life history theory and energetics into the theoretical understanding of developmental plasticity and their effects on the phenotype has opened the gates for further exploration of the relationship between environmental and human variability. This is a research paradigm that biological anthropologists can employ and benefit from. Through the study of female volunteers from Cambridge, UK, this research aims to validate previously identified relationships and answer new questions regarding how variation in energetic availability in early life (the period encompassing ongoing developmental processes, up to the cessation of growth) environment affects the expression of the life history strategy, the manifestation of the adult female phenotype, and contributes to the generation of phenotypic variation and adaptation in modern populations.

1.1. Project Context

A species' life history strategy describes the broad order and timing of important developmental events (Stearns, 1992). When the life history program of an individual organism is disrupted or changed, this can generate phenotypic variation. Major disruptions to an organism's life history program can be caused by energy availability. Reductions in extrinsic energy resources, increases in physical activity requirements, or the diversion of energy to combat infection are examples of such disruptions. Constriction of the energy budget during the developmental period results in energetic trade-offs which favour immediate survival over future investment in growth, reproduction, or maintenance. This results in variation in phenotypic characteristics. The ability to divert energy to survive energetically stressful periods is an adaptive mechanism known as phenotypic plasticity. These necessary energetic trade-offs can

have secondary effects and influence future health and developmental outcomes as an organism's phenotype becomes increasingly immutable.

Foundational studies by Barker and Hales examining the later life phenotypic outcomes of low birth weight have linked this characteristic to the development of cardiovascular and metabolic diseases (D. J. P. Barker, 1991; D. J. P. Barker et al., 1993; Hales & Barker, 1992). This has been further connected to an energy storage strategy that favours investment in fat mass rather than lean mass and a tendency toward a centralized fat distribution in individuals who experienced early life energy constriction (M. Barker et al., 1997). The relationships that exist between the life history schedule, the timing of stress events, and energy availability during the developmental window can be used to create an explanatory framework for understanding the variation in phenotype and disease outcomes identified by Barker and others. This framework is an increasingly important focus of research in human biology and biological anthropology and more can be done to incorporate this paradigm into research focusing on human variation and adaptability in the past and present.

Correlations between the early life indicator variables birth weight for gestational age (FGA) and age at menarche (first menstruation) and other phenotypic outcomes, such as body composition, stature, and non-communicable disease risk have been identified. However, these relationships have primarily been interrogated separately from one another, resulting in a lack of integration. While these phenotypic relationships are independently important, little research effort has focused on generating a synthesis that acknowledges the complexity of the human phenotype within its environmental context and the role of morphological integration in generating variation outside of health outcomes. Such research would recognize that the

phenotype reflects both complex mechanisms of lifetime environmental adaptation and a previously established evolutionary trajectory that yielded the capacity for plasticity.

The lived experiences of women have been under-represented in both the archaeological and literary record, leading to an androcentric archaeology and biological anthropology. Limitations placed on women (and other non-male identifying groups) that bar them from participating in research and voicing their unique insights and research questions have further compounded this issue (Heath-Stout, 2020). As a result, the majority of the biological research in the 20th century focused on male health outcomes, which in turn informed interpretations of health in the past by bioarchaeologists, with little attention paid to the inherent biological differences between males and females (Holdcroft, 2007). Some headway has been made in recent years (e.g. Macintosh et al., 2017; Veselka et al., 2018), with more focus being placed on female health and adaptation in the past and present. Life history and environmental adaptations differ between male and female humans, with more complex energetic relationships being needed to balance the increased energetic load that reproduction-related processes place on female organisms. It is important to understand how differences in energy needs might manifest as differences in energy allocation strategies and phenotypic outcomes in women. Therefore, this study aims to increase our understanding of female health in the present as well as provided information to aid in constructing more accurate reconstructions of health and lived experiences in the past.

1.2. Project Aims and Research Questions

The field currently lacks a synthesis of information, one which is needed to understand how variation in different traits interact to produce the human phenotype outside of a disease context. Understanding the environment-organism interaction that produces variation in multiple

traits in a living population will create a body of evidence useful for understanding the etiology of variation in a bioarchaeological context. The question that arises is how are these different life history traits and phenotypic characteristics expressed collectively when the programming of the early life environment is taken into consideration?

This project aims to understand and highlight how indicators of early life environmental conditions, specifically birth weight and age at menarche, correlate with other life history variables such as body composition and form to produce subtle forms of phenotypic variation beyond pathological phenotypes. To do so, this study draws on data that was collected by Dr. Stock and Dr. Murray and includes 3D whole-body surface scans, anthropometric measurements, bio-impedance data, and life history information of 104 cis-gendered female volunteers of European descent from Cambridge, UK. This study aims to provide a holistic approach in examining the human female phenotype to understand variation and adaptation.

Many previous studies of life history outcomes have focused on coarse relationships between birth weight and disease risk in later life. This leaves the overarching questions:

1. How do multiple environmental and biological factors combine to produce complex phenotypic outcomes, and what is the adaptive reasoning for these outcomes?
    a. Do the patterns of variation observed in the Cambridge sample validate the previously established trends that associate low birth weight with body composition, specifically a reduction in % lean mass compared to % fat mass?
    b. Gluteal-femoral fat deposits are known to be important energy sinks that are primarily utilized during pregnancy and lactation (Rebuffé-Scrive et al., 1985), therefore, is there a relationship between increased energetic investment in tissue

stores in that region and life history traits such as the age at menarche or birth weight?

2. Does this sample validate previously established correlations between energetic constriction in early life, measured by birth weight, and earlier sexual maturation identified by an earlier age at menarche?
    a. Age at menarche is also known to influence the growth program, with earlier sexual maturation linked to reductions in stature in industrialized populations. Therefore, is there a link between the age of reported menarche and stature in this sample (Mcintyre & Kacerosky, 2011)?
3. Continued energetic restriction in childhood has been linked to reductions in forelimb segment lengths, presumably a result of energetic trade-offs(Pomeroy et al., 2012). Therefore, is the relationship identified between energy constriction and reduction in limb segment length and proportions also reflected in soft tissue investment, namely in limb segment volumes and surface areas?
    a. Do limb segment surface areas and volumes follow the same trends seen in bone length or size reduction under energetic stress?
    b. Lastly, does the relationship between limb volume and surface area and trunk volume and surface area vary with any of the life history variables under study, and if so, how is this interpreted in a life history context?

An additional aim of this study is the validation of the use of a 3D body scanner in the collection and analysis of anthropometric data. If proved accurate and useful, with this tool it is possible to streamline the collection of relevant anthropometric data.

1.3. Chapter Outlines

The following chapter summaries are intended to provide the reader with a brief content overview:

Chapter 1 – Introduction: This chapter serves to provide a broad research context and outline the questions that guide this research.

Chapter 2 – Background: Further research context is provided, including expanding on the definition of life history theory and how it provides the theoretical framework for understanding the role of energetics in shaping phenotypic variation in humans. This chapter also outlines the different developmental windows and their associated life history milestones.

Chapter 3 – Materials and Methods: In this chapter, the protocols for data collection are reviewed along with the statistical tests used for analysis. Additionally, the validation procedures and potential limitations for the use of the Size Stream 3D Body Surface Scanner in studies of human anthropometrics are discussed.

Chapter 4 – Results: The results of Pearson and Canonical Correlation analyses are presented and organized regarding their relevance to the research questions presented in Chapter 1.

Chapter 5 – Discussion: The discussion chapter addresses the role of early life environmental variation in shaping female phenotypic variation in later life and how this was reflected in the study sample. The research questions outlined in Chapter 1 are addressed based on results presented in Chapter 4 and the potential interpretations and implications are discussed.

Chapter 6 – Conclusion: This chapter provides a brief overview of the research findings, potential limitations, and future research avenues.

2. Background

2.1. Human Variation

Humanity can arguably be described as one of, if not the, most successful invasive species on earth. This is due to our unique ability to adapt and thrive during periods of environmental instability (Grove, 2014; Grove et al., 2015a; Grove, 2015; Wells & Stock, 2007). This innate adaptability has allowed anatomically modern humans to proliferate and expand across the globe while other *Homo* species have gone extinct. It has been argued that our success as a species is not solely due to unique genetic adaptations, but rather our ability to modify our growth, development, and physiology in response to cues from the environment (Wells & Stock, 2007). This ability to respond to our environment (without the fixation of characteristics that comes with genetically programmed adaptation) has allowed for higher plasticity in response to stochastic environmental conditions. This plasticity also contributes to the wide range of visible variation in our species, creating both population and individual differences in phenotypic characteristics such as height, limb morphology, and the timing of developmental events.

Phenotypic plasticity, also described as environmentally influenced variation, is well documented in other species (Wells & Stock, 2007). An illustrative example is the delay of metamorphosis and the retention of juvenile characteristics in some species of salamander when aquatic conditions are more favourable (Denoël & Poncin, 2001). However, they retain the ability to transition into their adult form when resources are scarce (Denoël & Poncin, 2001). This shows how different environmental conditions can induce vastly different phenotypic outcomes.

The study of human variation is relevant to biological anthropologists for two main reasons. First, variation reflects environmental conditions, and therefore understanding the

etiology or source of variation in a phenotypic characteristic in a modern group can help in the identification and reconstruction of the environmental context in the past. This is specifically relevant to bioarchaeological studies where multiple lines of evidence must be drawn on to provide a reliable reconstruction of environmental, social, and cultural contexts. Secondly, studying variation should be of interest to anthropologists as it can be linked to negative health outcomes. For example, variation in birth weight has been repeatedly linked to the manifestation of metabolic and cardiovascular disease in adulthood (D. J. P. Barker, 1991; Hales & Barker, 1992). Therefore, understanding the causes of variation which have been linked to diseases can contribute to recommendations to improve health outcomes at both the individual and population levels.

2.2. Phenotypic Plasticity

Plasticity describes the ability of an object or substance to be altered. In biology, this term describes the ability of an organism to alter itself in response to environmental conditions (Stearns, 1989). The concept of plasticity is commonly invoked in the field of neurobiology to describe the brain's ability to alter existing neural networks and create new ones. It can also describe different environmental acclimations, for example, when exposed to high altitude humans increase ventilation and red blood cell production (West, 2006). When some form of stress (e.g., hypoxia, under nutrition, or infection) is experienced that threatens homeostasis, plastic responses which control the allocation of energy are initiated to return the body to homeostasis. This is the central tenet to the study of plasticity in *H. sapiens,* that it is an adaptive response to maintain homeostasis and ensure the immediate survival of the individual. A subset of phenotypic plasticity is developmental plasticity and refers to the phenotypic changes that

occur during critical periods in the developmental window that can permanently change the phenotype or constrict future phenotypic options (Bateson et al., 2004).

Plasticity operates at the level of the phenotype, which is the suite of observable characteristics of an organism. It functions through changes to the expression of genes rather than through changes to the genetic sequence itself. Phenotypic plasticity, therefore, describes the ability of one genotype to produce more than one phenotype, depending on environmental input. Another classic example of phenotypic plasticity in nature is the growth of *Physella virgata*, a species of freshwater snail. Snails grown in the presence of sunfish, a natural predator, had reduced overall growth and more rotund shells compared to snails from the same brood raised in the absence of sunfish (Langerhans & DeWitt, 2002). In the case of the snails raised with the sunfish, they picked up on environmental cues which triggered the expression of an 'anti-predator' phenotype characterized by both rapid growth and hard to penetrate shells (Langerhans & DeWitt, 2002). This specific kind of phenotypic plasticity can also be described as a reaction norm where a threshold for a stimulus is reached causing the expression of the alternate phenotype (Gavrilets & Scheiner, 1993). This kind of plasticity allows organisms to thrive in unstable environments without having to maintain multiple genotypes within the population (Gavrilets & Scheiner, 1993).

One of the earliest engagements with plasticity within a human biology context was seen in the Thrifty Phenotype Hypothesis. This theory stipulates that poor conditions in early life predispose an individual to a thrifty, or energy-sparing, phenotype that prioritizes brain growth and survival at the expense of development in other areas during growth (Hales & Barker, 1992). Under these conditions, individuals can develop the hallmarks of Type-II diabetes, including glucose intolerance, impaired insulin resistance, and a tendency to obesity. In the case that the

resource-scarce environment persists into adulthood, the negative health consequences are less. However, in the case that the nutritional environment improves, there is a predisposition to metabolic diseases (Wells, 2007a, 2009). This theory has been used as an explanation for the higher rates of obesity and metabolic and cardiovascular diseases in some human populations, specifically those of a non-European background who have been recently exposed to a westernized diet.

An extension of the thrifty phenotype hypothesis purposed by Hales and Barker (1992), it has been proposed by some that the phenotypic changes that result from plastic responses to energetic stress during the developmental window represent a form of predictive adaptive response (PAR). That is, the environmental conditions experienced during the developmental window in utero are read as a prediction of the environment to come and the plastic changes to the phenotype are intended to increase advantage and fitness in the future environment (Gluckman et al., 2005).

Where the thrifty phenotype hypothesis connects deficits in the growth of organ systems with manifestations of later-life disease, the PAR theory takes this scenario in another direction and proposes that the phenotypic changes experienced are a deliberate response to adapt to the future environment. When a mismatch occurs, however, between the early life environment and the later-life environment and resource availability, diseased phenotypes occur.

The PAR theory has been highly critiqued due to the suggestion that the phenotypic changes that occur early in life do so in order to increase that individual's adaptation to future environments (Wells, 2007a). Humans in particular are a long-lived species and are likely to experience multiple environments over the course of their lifetimes, therefore, there is a high likelihood of mismatch between the fetal environment and future environments. Therefore, the

selective pressure needed to promote a PAR energetic strategy does not exist. This theory also ignores the direct connection between early thrift causing reductions in the investment and growth of metabolically important tissues, and how that is the factor limiting response to future metabolic load (Wells, 2011).

2.2.1. Mechanisms

The mechanisms which underly phenotypic plasticity are not entirely clear and require further study. However, dominating theories point to epigenetic mechanisms controlling the expression of environmentally dependent traits. The prefix epi- means over, or on top, of. As such, epigenetics is the study of the things which lie on top of, or adjacent to, the genetic sequence. Found in the nucleus of a cell, deoxyribonucleic acid (DNA) is wrapped around protein-ribonucleic acid (RNA) complexes called histones. For DNA to be accessed for transcription (the process of copying a DNA sequence into an RNA sequence to be used for protein synthesis) these histones must be un-wound or moved out of the way to allow the transcription machinery to interact and bind to the area upstream of the gene of interest. Epigenetic markers, such as methyl groups, can alter the interaction between the transcription machinery and the DNA, often blocking the transcription to RNA and therefore reducing or silencing the expression of that gene (Attwood et al., 2002). It is epigenetic mechanisms, such as DNA or histone methylation, that allow for the alteration of the expressed phenotype so that it differs from the genetically encoded genotype (Prokopuk et al., 2015). It is thought that certain environmental conditions can alter the methylation patterns of DNA or other transcriptionally relevant proteins (Prokopuk et al., 2015). The epigenome describes the suite of epigenetic modifications present on an organism's DNA. Researchers examining the epigenomes of children who were conceived or born during the Dutch hunger winter of 1944 reported the first

evidence of environmental effects on methylation patterns (Heijmans et al., 2008). They found that individuals who experience famine early in gestation exhibited decreased methylation of the *IGFII* differentially methylated region (DMR) compared to their non-exposed siblings, showing strong evidence that methylation patterns are environmentally sensitive (Heijmans et al., 2008; Tobi et al., 2012). Individuals whose mothers experienced famine in later gestation did not show hypomethylation of *IGFII DMR* when compared to non-exposed siblings. However, these individuals, who were more likely to experience low birth weight, were shown to have impaired glucose tolerance and were more likely to experience metabolic diseases in later life (Ravelli et al., 1998). Low birth weight has been linked to increased methylation of the *PPARGC1A* loci, which in turn has been linked to the development of insulin resistance when overfed (Brøns et al., 2010). Another phenotypic modification that presented in this cohort was the tendency to acquire fat mass over lean mass (Rogers, 2003). The epigenome, which is influenced by environmental triggers, is now understood to be partially heritable, with epigenetic modifications being retained across more than one generation (Prokopuk et al., 2015). This discovery changes our understanding of how environmental conditions can have lasting impacts on the survivability or fitness of a species or population and how rapid responses to environmental instability can be seen in one generation's time. The hypothalamus-pituitary-adrenal axis (HPAA) has been proposed as a likely control of the regulation of plastic phenotypic responses to environmental stressors (Ponzi et al., 2020). Overall, the results of these studies demonstrate the effect of environmental variation on the epigenetic markers that control phenotypic expression and, consequently, phenotypic variation.

Phenotypic plasticity has been invoked in explanations of the ability of humans to survive in and colonize extreme environments (Grove et al., 2015b; Wells & Stock, 2007). An

illustrative example of human phenotypic plasticity is seen in the inhabitants of high-altitude environments such as the Peruvian Andes. The highland populations here have developed adaptions suited to living in a high altitude, hypoxic environment. Anthropometric studies of Peruvian children have identified increased lung and thoracic volumes reflective of increased chest dimension in highland children compared to children living in lowland regions (de Meer et al., 1995). Additionally, children with lowland ancestry raised in the highlands since birth and early childhood had the same phenotypic adaptations to a hypoxic environment as children with highland ancestry, showing that advantageous adaptions were gained during the developmental period (Frisancho, 2013). Genetic studies have identified positive selection on genes related to cardiovascular function and metabolic homeostasis (Julian & Moore, 2019), however, to date there have been no genes identified that explain the morphological phenotype observed in these populations. While this morphological variation in body form, among other adaptive mechanisms related to increased oxygen availability, is often assumed to represent genetic adaptations, they may be plastic responses to a hypoxic environment during the developmental period. Additionally, plastic responses to systemic stress (a combination of hypoxic, nutritional, and pathogen-related stress) are also observed in limb proportions in children from highland regions in the Andes and Tibet. Here, overall height and arm length are reduced as a result of a reduction in the zeugopod (shin and forearm) segments of the limb, this reflects an energetic trade-off disadvantaging somatic growth (Pomeroy et al., 2012). In the case of the high altitude adapted populations, energetic strategies employed during development favored investment in increased lung capacity and other hypoxia combating mechanisms.

While phenotypic plasticity is not genetically encoded, the capacity for plasticity is and therefore natural selection can affect it. Species (and individual organisms) who can more

quickly adapt to unstable environmental conditions are more likely to outcompete and outlive those who lack the same degree of phenotypic plasticity. Therefore, the capacity for plasticity is beneficial for species experiencing or living in stochastic environments and can provide a selective advantage. In long-lived species, such as humans, diverse environmental conditions are likely to be experienced over the life course, making the ability to adapt without committing to genetic changes a selective advantage (Wells & Stock, 2007). It is hypothesised that this advantage has allowed *H. sapiens* to expand and colonize diverse environments worldwide, effectively making humans the cockroaches of the mammalian world. Understanding how plasticity contributes to the range of human phenotypic variation, how that variation is patterned in predictable ways, and how those patterns, in turn, relate to environmental conditions will allow for better modeling of human health, disease, and adaptation in the past.

2.3. Energetics and Adaptation

The first law of thermodynamics stipulates that energy cannot be created or destroyed, only transformed from one form to another. This theory extends to living organisms when considering them as an energetic system (Atkins, 2010; Hill, 2005). Energy can take many forms, such as heat or light, and is stored in the chemical bonds which join atoms to produce different substances. The processes which allow the acquisition, storage, and transformation of energy are integral to life and can vary dramatically between different organisms. Natural selection, one of the processes which actively directs the evolutionary course, can act on the energetic strategy of a species. Species that are more effective in obtaining and utilizing the energy in their environments are more likely to survive and reproduce. Additionally, the ability of an organism to modify its energy attainment and mobilization strategy to adapt to instances of environmental instability or increases in energy requirements also influences individual and

species level survivorship and fitness. An organism's ability to modify its energetic strategy is a form of phenotypic plasticity.

Energy is the limiting factor and the ultimate driver behind the need for phenotypic plasticity as a survival mechanism. When resources are plentiful, there is no constraint on the energy budget, and barring any intrinsic pathologies, energy can be appropriately allocated to the functions of survival, maintenance, reproduction, and defence (Wells & Stock, 2020). However, when energy is constrained either due to malnutrition, increased physical activity, or infection, trade-offs occur in the allocation of energy to these competing functions to promote immediate survival. Plasticity of the phenotype can therefore be thought of as a way to make the best of a bad situation. Energetic trade-offs are arguably most impactful if they occur during the developmental period: between conception and the cessation of somatic growth. Within this period there are windows of development that are more sensitive to assault than others. Take for example the Dutch Hunger Famine outlined previously. Famine experienced in the later stages of pregnancy, specifically the third trimester, had a greater impact on the fetus's childhood and adult phenotype in the form of body composition and disease risk than if famine was acutely experienced during other windows of neonatal development (Stein, 2004). This is likely a result of the energy budget for brain growth being maintained through the diversion of energy from other areas. There is also the suggestion that placental growth is maintained over growth in other areas as the placenta is integral to infant survival (D. J. P. Barker et al., 1993). This identifies later gestation as an important developmental window for the programming of childhood and adult metabolism. The energetic trade-offs which occur during this window either to improve chances of immediate survival or to preserve integral organ systems can result in negative health consequences in later life.

2.4. Life History Theory

The ability to modify an energetic strategy is a form of phenotypic plasticity, and both concepts as they relate to human adaptability are best understood within the context of life history theory (Wells & Stock, 2020). Life history theory describes the schedule of an organism's developmental processes and the timing of milestones related primarily to reproduction and growth and dictates traits such as age and stature at maturity (Hill, 2005). Different species employ different life history strategies to maximize their fitness and survivorship within their environmental context. These life history strategies fall on an r-K-selection spectrum. In short-lived species, such as mice, their life history strategy favours fast development, a short interval between birth and sexual maturity, investment in reproduction over somatic growth, large litter sizes, and short interbirth intervals (Wells & Stock, 2007). This strategy, referred to as r-selection (Stearns, 1992), promotes population expansion during resource-rich periods but can lead to local extinctions if conditions change quickly (climate, weather, access to resources, etc.) as their fast-paced development strategy does not allow for many plastic adaptive responses (Wells & Stock, 2007). In long-lived species, such as apes, the extension of the developmental window between conception, and sexual maturation and the cessation of somatic growth has increased the amount of time offspring are dependent on their parents compared to other species. However, this also allows for the generation of plastic phenotypic responses to cues of environment instability, potentially increasing survivability and fitness in the long term. This strategy, known as K-selection, requires increased energy and time investments from parents, with an output of fewer, but higher "quality" offspring (Wells & Stock, 2007). Broadly speaking, humans meet many of the criteria for a K-selected species, however, when compared to other extant apes, humans tend to have shorter birth intervals and

more offspring (Galdikas & Wood, 1990; Wells & Stock, 2007). Humans also exhibit a generalist rather than specialist adaption strategy, on top of the uniquely human capacity for cultural adaptation, which allows for success in adapting to stochastic or novel environments.

Generally, the life history strategy of a species is fairly consistent, however, the life history schedule of an individual organism is plastic and is a source of intraspecies variation. For example, humans experience a gestation period of 40 weeks, but this can vary depending on maternal nutrition and fetal body size (length, weight, etc.); it is proposed that the energetic conflict that arises between fetus and mother is what in part drives delivery timing (Nepomnaschy et al., 2020). Variability in weaning also introduces some variation in the length of the human birth interval as lactation suppresses ovulation (Galdikas & Wood, 1990). Initiation of the childhood and adolescent growth spurts can vary between individuals as a result of energy availability, and in females, age at menarche can vary dramatically between individuals and populations based on a variety of extrinsic factors including nutritional history, illness, and psychosocial stress (Mcintyre & Kacerosky, 2011; Prebeg & Bralić, 2000; Tahirovie, 1998). Menarche, age of first sexual activity, and age of first birth are life history variables known to be linked, exhibiting variable presentation between populations (Udry & Cliquet, 1982). Timing of sexual maturation is closely linked to the attainment of adult stature, with women who experience delayed sexual maturation being taller on average due to a delay in the hormonal signaling which arrests long bone growth (Dunsworth, 2020).

The interdependence of energy availability and the capacity for plasticity is what shapes the life history strategy of humans as a species but also on a population and individual level, generating the wide range of observable human variation. Therefore, to understand the source(s) of variation in morphology, the expression of life history milestones, and their connection to

human health, survivorship, and fitness it is essential that we broaden our understanding of the interplay between environmental influences and the patterns of variation which we observe.

2.5. Developmentally Sensitive Windows

There are well-documented and understood forms of phenotypic plasticity which operate throughout the life course to maintain homeostasis. Some phenotypic changes that occur in response to environmental cues are reversible. However, changes that occur during developmentally sensitive windows, such as *in utero* or infancy, can have far-reaching and lasting effects on the phenotype as it becomes increasingly irreversible during development. This fixation of the phenotype is what connects early life environmental influences to the manifestation of later life health and phenotypic outcomes. The life stages referred to hereon follow the descriptions provided by Bogin, 1997.

2.5.1. Prenatal and Infancy Stages

Fetal and infant development establishes the foundation on which the human phenotype is built. The prenatal period spans from conception to birth. Following Bogin's life history stages, infancy covers the period between birth and weaning (Bogin, 1997). These periods represent developmentally integral stages, and therefore, a sensitive window where interactions with the environment can be recorded in the phenotype through plastic changes in an individual's physiology, morphology, or metabolism (Heijmans et al., 2008; Ravelli et al., 1998). This makes it necessary to understand the environmental factors which trigger different phenotypic outcomes to understand the etiology of human variation. The energetic influences during the prenatal and infancy period can be divided into two categories, direct maternal effects on fetal growth, and environmental effects external to the maternal environment.

2.5.1.1. Maternal Effects on Fetal Growth

During embryonic and fetal development, the conditions of the maternal environment can be captured in the growth and development of the offspring and reflected in later life health outcomes (Li, 2018). This development can be shaped by the current and past environmental conditions experienced by the mother (Wells, 2003; Wells & Stock, 2007). Past environmental conditions are important to consider, especially in the context of female development and health because female fetuses develop their oocytes *in utero*. Therefore, the maternal environment can directly impact up to two generations. This can lead to intergenerational inheritance of epigenetic markers which in turn affect the development and life history of multiple generations.

One of the first measurable phenotypic characteristics that capture environmental conditions is birth weight. Birth weight is a plastic phenotypic characteristic modulated by energy availability and has been correlated with phenotypic outcomes at other stages of development. As such, birth weight (relative to gestational age, placenta size, or body length) is used as an indicator of the maternal environment and indicates energy availability to the fetus or stress experienced by the mother during pregnancy.

Birth weight has been interpreted as the result of a conflict between the fetus and the mother over the allocation of energy. It has been suggested that this conflict may explain spontaneous abortion in human females and may represent an adaptive mechanism that prevents pregnancy during periods of stress or adverse environmental circumstances (Nepomnaschy et al., 2020). A study released in 2021 examining the miscarriage rate in the province of Manitoba from 2003 to 2014 reported a prevalence of 11.9% (Strumpf et al., 2021). These metrics likely underestimate spontaneous abortion rates as not all miscarriages are reported and the loss of many ‘chemical pregnancies’ (fertilization without successful implantation) go undetected

(Nepomnaschy et al., 2020). The adaptive benefit of this phenomenon is that it prevents the energetic burden of a pregnancy at a time when energy reserves are constrained (Nepomnaschy et al., 2020).

Outside of nutritional stress during pregnancy, other environmental variables of the mother have been linked to later life outcomes. In a retrospective study of births in Northern Ireland between 1971 and 1986, it was found that there was a positive correlation between maternal and paternal age and the risk of manifestation of childhood Type I diabetes at age 15 (Cardwell et al., 2005). A negative correlation was also identified between birth order and risk of Type 1 diabetes (Cardwell et al., 2005).

If stress, such as undernutrition or infection, is experienced by the mother during critical windows in fetal development it can lead to a lower-than-average weight FGA in an infant. Low birth weight is thought to reflect an energetic trade-off in the mother during times of energetic stress (such as an infection) that favours her survival, maintenance, and future fitness over that of the offspring (Abrams & Meshnick, 2009). Barker's study of infant mortality and subsequent mortality from cardiovascular and obstructive lung disease in same age cohorts, he proposed the fetal origins of disease hypothesis which connects poor early life conditions, as measured by infant mortality, with increased occurrence of disease in adult survivors (D. J. P. Barker, 1991). Building on this, the thrifty phenotype hypothesis connects poor maternal nutrition, and more specifically, fetal malnutrition (which can be caused by placental abnormalities), with an increased incidence of Type II diabetes Mellitus (Hales & Barker, 1992). They proposed that energy constriction leads to a trade-off in the growth and development of the pancreas in favour of immediate survival. The negative consequences of these trade-offs – metabolic and

cardiovascular disease – typically manifest themselves in later adulthood and therefore fall after the selection shadow as they do not directly affect reproduction.

Influences on the phenotype are complex and it is this complexity that leads to the continuous variation we see in many traits. The contribution of the uterine environment, reflected in birth weight, does not in isolation shape aspects of the phenotype or the manifestation of disease in later life. Rather, it is the pattern of growth in early life, rather than isolated measures of body dimensions, that shape later life phenotypic variation (D. J. Barker et al., 1993). The coupling of birth weight to subsequent weight gain in infancy and early childhood has been connected to a variety of phenotypic outcomes. If conditions improve after birth and energy availability is no longer constrained, infants born underweight can experience significant catch-up growth, quickly catching up to their peers in terms of weight and length for age (Ibáñez et al., 2006). However, while catch-up growth leads low birth weight infants to track with their peers in terms of weight and stature, there is a fundamental difference in body composition between those groups. Low birth weight infants are more likely to convert excess energy into subcutaneous fat mass (rather than lean mass) with a centralized distribution (Ibáñez et al., 2006).

Unsurprisingly, birth weight is linked to adult body composition in both males and females. In the Amsterdam Growth and Health Longitudinal Study, birth weight was compared to adult weight, skinfold thickness, and waist-to-hip ratio. It was found that individuals who were underweight at birth were more likely to be overweight in adulthood, with higher fat mass, and a truncal subcutaneous fat distribution (te Velde et al., 2003). Additionally, low birth weight is also correlated with earlier sexual maturation, namely the earlier onset of menstruation in adolescents compared to average-weight infants (Sloboda et al., 2007; Tam et al., 2006).

2.5.1.2. External Environmental Effects

In addition to direct maternal effects, there is also evidence that aspects of the physical external environment can shape phenotypic outcomes. One aspect that has been extensively studied is birth month. A retrospective study of singleton births in Israel between 1998 and 2004 shows a significant pattern of high birth weight infants being born in the summer months (Chodick et al., 2007). The authors suggest this is due to increased sunlight and improved environmental conditions in the later phases of gestation (Chodick et al., 2007). A similar study examining the association between body proportions and birth month in Peru found a significant positive correlation, with tibia length, lower limb length, and stature peaking with those born in November (Pomeroy et al., 2014). The authors interpret this as the result of improved nutritional conditions during the spring and summer in later gestation but indicate that direct causation is hard to determine (Pomeroy et al., 2014). However, these examples illustrate how yearly fluctuations in environmental conditions and resource availability can contribute to the generation of human phenotypic variation.

2.5.2. Childhood and Juvenile Stages

During Childhood, which roughly spans ages three to seven years, human children maintain high rates of both brain and somatic growth which needs to be fueled by nutritionally dense, easily digestible meals, making children dependent on adults for food and resource acquisition (Bogin, 1997). Because of the high energy requirements for growth, Childhood is a developmentally sensitive window, and changes in resource availability or stress levels can alter the phenotype. The Juvenile period spans from the occlusion of the first permanent molars, the achievement of adult brain weight, and the reduction of energy needs for growth to 50% of the

total energy budget, to the start of puberty (Bogin, 1997). This usually occurs two years earlier in females (Bogin, 1997).

2.5.2.1. Skeletal Growth Trade-offs

Because of both the high energy requirements during this developmental period as well as the focus on brain growth, Childhood is a developmentally sensitive period where insults can have long-lasting impacts on the phenotype. If nutritional requirements are not adequately met, energy is diverted away from somatic growth to preserve brain development, also known as brain sparing energy allocation. This energetic strategy preserves the most important functions, putting survival and brain development over other areas of energy allocation such as somatic growth, maintenance, defence, or reproduction. These energetic trade-offs can be seen archaeologically in the form of non-specific stress indicators like Linear Enamel Hypoplasia (LEH) and Harris Lines. LEH generally presents as banding on the enamel of the teeth and results from the deceleration or arrestment of enamel development as a result of some stress on the energetic system and the subsequent recovery from said stress (Temple, 2019). Seen in both deciduous and permanent teeth, the location of the banding can indicate when the stress event occurred and provide an idea of how long it lasted – or at least indicate that the stress was sufficient to impact enamel growth (Temple, 2019). It is possible to see LEH that likely line up with the weaning transition. Weaning can be stressful if undertaken suddenly or if weaning foods do not meet the energetic requirements of the infant. Additionally, the transition itself can be hard, resulting in undernutrition for a period and arrestment of growth in the enamel of the forming adult teeth. LEH can also occur as a result of an illness or stressor that constricts energy availability (Temple, 2014, 2019). Harris Lines are observable in radiographs and present as horizontal lines at the ends of long bones (Alfonso-Durruty, 2011). Like LEH, they represent a

period of stress and recovery affecting growth (Scott & Hoppa, 2015). Unlike LEH, Harris Lines are considerably more non-specific, are often not visible in adults, and do not always correlate with LEH occurrence (Alfonso et al., 2005). Instead, it is thought they correlate with periods of increased growth velocity, and as a result, less emphasis is placed on their importance as a marker of stress (Alfonso et al., 2005; Scott & Hoppa, 2015). This is supported by an experimental study of harris line formation in rabbits exposed to either chronic undernutrition or periodic fasting showed that they occur as a result of accelerations of growth velocity, rather than chronic undernutrition (Alfonso-Durruty, 2011).

Energetic stress during childhood can also be reflected in attained adult stature, limb lengths, and limb ratios. Studies of the growth and development of Peruvian children from the Andean highlands by Pomeroy and colleagues show that chronic stress caused by hypoxia and undernutrition results in a lower overall stature compared to their lowland counterparts (Pomeroy et al., 2012). Additionally, the reduction in stature was not a result of unilateral reductions in body dimensions but rather the reduction of zeugopod lengths. Specifically, tibia and ulna lengths were reduced relative to their lowland peers, however, there was the protection of stylopod and autopod lengths (Pomeroy et al., 2012). This served as evidence against a 'distal blood flow' hypothesis that suggests circulation plays a role in the dissemination of nutrients leading to reduced size in the extremities furthest from the heart. Instead, the differential investment in the growth of limb segments shows energetic buffering of functionally important components (Pomeroy et al., 2012). This same pattern of zeugopod reduction was observed in a rabbit model, with animals exposed to chronic undernutrition during the developmental period demonstrating shorter forelimbs compared to control animals and animals exposed to periodic fasting periods (Alfonso-Durruty, 2011). This experiment illustrates that both periodic and

chronic stress can affect skeletal growth, although the effects of chronic energetic buffering are longer lasting.

2.5.2.2. Energetics, Puberty timing, and Long-Term Outcomes

During the Juvenile phase of development, the hormonal cascades responsible for initiating the process of sexual maturation begin (Bogin, 1997). In females, this is identified by the beginning of breast development, called thelarche. Thelarche beings 2-3 years before menarche, which is the beginning of menstruation (Diaz et al., 2006; Papadimitriou, 2016). Age of thelarche, like menarche, is influenced by the early life environment including nutritional status during critical developmental windows and exposure to other forms of stress (Biro et al., 2018). Age at menarche in the last 50 years has remained fairly stable, with longitudinal studies in the United States reporting a median age of 12.25 years (Biro et al., 2018; Goldberg et al., 2020). Conversely, the age of thelarche has been decreasing (Biro et al., 2013), with one meta-analysis reporting a decrease of 3 months per decade between 1977 to 2013 (Eckert-Lind et al., 2020). Earlier initiation of thelarche has been examined for its impact on the adult phenotype and risk for developing non-communicable diseases. Earlier onset of thelarche and menarche has been associated with a 30% increase in the risk of developing breast cancer in later life (Goldberg et al., 2020). Secular changes in the timing of integral life history traits are noteworthy for biological anthropologists and anthropologists as they reflect changes in social, cultural, and environmental conditions rather than being the result of natural selection and can have important social and biomedical impacts. Therefore, these traits act as important biological markers of external change, and if it is possible to identify those changes or their knock-on effects in the bioarchaeological record it would be informative of the surrounding social and environmental contexts. Additionally, when examining modern populations, being able to identify connections

between early life environment and phenotypic outcomes in later life will help with the identification of secular trends which lead to adverse health outcomes and hopefully provide the knowledge to combat them.

2.5.3. Adolescence

The Adolescent stage begins with puberty, first initiated by a hormonal cascade beginning during the later phase of the Juvenile stage (Bogin, 1997), and is marked by a change in the strategy of energy allocation from one that prioritises investment in somatic growth to one focused on investing in reproduction (Reiches et al., 2013). The adolescent period is characterized by a growth spurt followed by the completion of somatic growth, sexual maturation, and the taking on of adult roles and responsibilities (Bogin, 1997). While there is evidence of genetic control of thelarche and menarche, effect sizes are small (Busch et al., 2018), and studies have shown that the timing of puberty is sensitive to environmental conditions and reflects the energy availability and allocation strategy experienced during fetal development, infancy, and childhood.

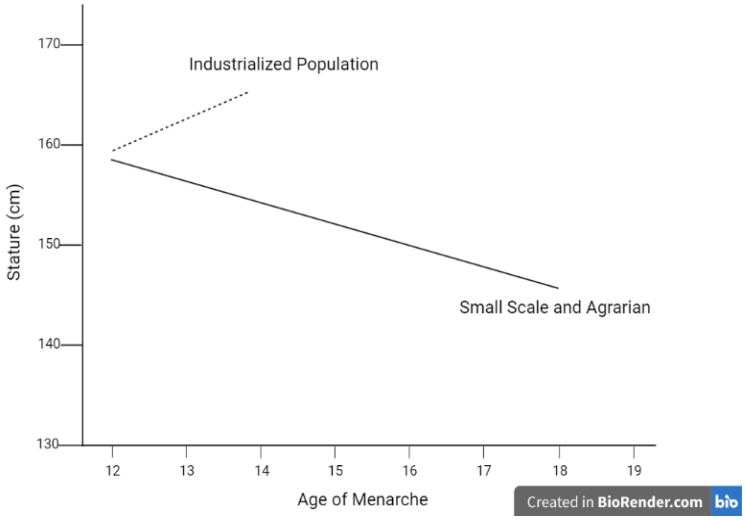
Age at menarche in females is relevant to anthropological studies because, on the population level, the average age at menarche can indicate the quality or abundance of necessary resources experienced both during gestation and childhood and can reflect population-level stress events such as war, disease, or famine (DeWitte & Lewis, 2020; Prebeg & Bralić, 2000; Tahirovie, 1998). Age at menarche also impacts the reproductive life history of an individual and the population level pattern of reproduction, for example, if menarche is delayed, the average age of first birth is also delayed, which has important implications for generational turnover and population expansion (in populations or groups that do not intentionally or artificially delay reproduction). On an individual level, similar to birth weight, early menarche has been linked to

risk factors for non-communicable diseases, for example, some uterine and breast cancers (Werneck et al., 2018), and delayed menarche and leanness have been linked to increased risk of osteoporosis. Therefore, the study of menarche and its relation to the environment and other life history variables is relevant to studies of female health and wellness, a historically under-researched area.

2.5.3.1. Sexual Maturation and Stress

This pattern holds for industrialized populations, however, is not universal. Mcintyre and Kacerosky suggest that the relationship between age at menarche and stature represents a norm of reaction based on environmental conditions experienced during childhood (Figure 1) (Mcintyre & Kacerosky, 2011). Their meta-analysis of studies reporting age at menarche and stature found different patterns of correlation depending on the status of the society as industrialised or 'small-scale and agrarian'(SSA) (Mcintyre & Kacerosky, 2011). In the industrialised populations, there was a significant positive correlation between age at menarche and stature whereas in the SSA populations there was a significant negative correlation (Mcintyre & Kacerosky, 2011). Mcintyre and Kacerosky suggest that this may relate to historical and current resource availability in industrial societies, where individuals have access to the

Figure 1 *Norms of Reaction for Stature and Age at Menarche*



Note. Regression lines demonstrating the relationship between stature and age at menarche in industrialized and small-scale and agrarian populations. Figure adapted from Mcintyre and Kacerosky, 2019.

energetic resources needed to initiate puberty earlier. If there is a delay in the start of puberty (which represents a change in energy investment strategy) due to some form of stress (nutritional, psychosocial, etc.) then the energetic strategy remains focused on somatic growth, allowing for a higher stature to be achieved. In SSA societies, the authors suggest that chronic stress in the form of undernutrition and disease burden during childhood can cause severe perturbation of the growth process, such that both somatic growth and sexual maturation are compromised, causing the negative correlation between stature and menarche observed (Mcintyre & Kacerosky, 2011). This suggests a norm of reaction for height and age at menarche in women that is dependent on resource availability. A reaction norm is a concept related to phenotypic plasticity that proposes that once some environmental stimuli or cue is received, it triggers the expression of an alternative phenotype. In this case, the pattern of growth and energy investment employed is cued by resource availability in childhood and adolescence and causes irreversible changes to the adult phenotype.

Psychosocial stress can also be a powerful influence on age at menarche in an individual. Correlations between family environment and social sources of stress such as paternal parent absence have been linked to earlier sexual maturation in female offspring (Hoier, 2003), however, those findings have been contested (Campbell & Udry, 1995; Hoier, 2003). Additionally, experiencing a traumatic event can delay menarche. In a study of girls from Sibenik, Croatia, who experienced war conditions from 1991 to 1995, there was a significant increase in the average age at menarche from 12.87 ± 0.06 years pre-war, to 13.13 ± 0.10 post-war (Prebeg & Bralić, 2000). In a subgroup of girls who had their homes destroyed, the average age at menarche was 13.53 ± 0.14 years, and in girls who experienced the death of a family member, the average age at menarche increased again to 13.76 ± 0.27 (Prebeg & Bralić, 2000).

This illustrates how the human life history program and phenotypic outcomes are a product of multiple environmental influences and studying them within their context provides more insight into the etiology of phenotypic variation than examining one or two variables in isolation.

2.5.3.2. Reconciling Menarche, Body Composition, and Birth Weight

Individuals born with low birth weight FGA who then experience a resource-rich childhood are more likely to experience an early age at menarche relative to their normal or overweight at birth peers. Age at menarche has also been linked to body composition and is shown to happen earlier in individuals deemed overweight or obese according to their BMI (Ahmed et al., 2009; Rosenfield et al., 2009). Body composition variation has also been linked to birth weight, with high birth weight individuals being more likely to have a higher childhood and adult BMI. If low birth weight predicts early menarche, high birth weight predicts an overweight or obese phenotype, and being overweight or obese in childhood predicts early menarche, how do we reconcile the same phenotypic outcome from different environmental conditions?

The majority of studies linking birth weight to body composition have done so using BMI, which has suggested obesity as a product of high birth weight. However, using BMI as a measure of body composition has received criticism in recent years as BMI groups both lean mass and fat mass together. Practically, this means that if two individuals of the same height and body dimensions are compared, the individual with more lean mass will have a higher BMI compared to the individual with more fat mass because muscle is denser than fat (Rogers, 2003). Additionally, BMI as a measure does not illustrate the distribution of fat and other tissues around the body, which is important when determining disease risk and physical health. For instance, centralized fat deposits are associated with risk for heart and metabolic diseases whereas gluteal-femoral fat deposits in women are associated with energy reserves for reproduction (M. Barker et

al., 1997; Rebuffé-Scrive et al., 1985). When lean mass and fat mass are used in place of BMI to understand the relationship between birth weight and body composition, a different relationship emerges.

More recent studies have examined lean mass and fat mass separately and demonstrated that individuals of low birth weight tend to acquire fat mass over lean mass and that individuals with normal to higher birth weights tend to instead invest in lean mass (Ong et al., 2009; Rogers, 2003). This energetic strategy makes sense within the context of energy restriction as mouse models have shown it takes less upfront energetic investment to deposit fat mass compared to lean mass and that fat mass is more energetically available (Elsukova et al., 2012; X. Li et al., 2010). Furthermore, low birth weight individuals tend to carry excess adipose tissue centrally, a pattern that is linked to both metabolic and cardiovascular disease.

This interpretation clarifies the relationship between birth weight and menarche as the age of onset is closely linked to acquired fat mass (Vizmanos & Martí-Henneberg, 2000). Therefore, low birth weight individuals who tend to store energy reserves in the form of fat over lean muscle are more likely to have a lower BMI and experience an earlier age at menarche (Ong et al., 2009; Rogers, 2003). This relationship, previously clouded by the use of BMI as a measure of body composition, also shows that high birth weight infants who have a greater tendency to build lean mass over fat mass are also less likely to experience early menarche. Overall, this shows the importance of looking at the broader context of growth and development and using multiple life history and growth characteristics to understand phenotypic outcomes, and also of using specific measures of body composition over BMI. This interpretation that separates body composition into lean mass and fat mass explains some of the disagreement in the literature

regarding how birth weight and body composition affect age at menarche and contributes to the risk of non-communicable disease.

2.5.4. Adulthood

Adulthood begins with the cessation of somatic growth and ends with death (Bogin, 1997). It is within this period that a number of the consequences of developmental plasticity become visible, increasing as a female moves into the reproductive shadow (Chmielewski, 2019). As previously mentioned, there is a wide body of evidence that links the conditions of early life with later life non-communicable disease manifestation. This is summarized by the Fetal Origins Hypothesis (D. J. P. Barker et al., 1993). The manifestation of these diseases is not solely determined by birth weight but is likely the result of a combination of environments and conditions experienced during the developmental period lasting from conception onwards. Understanding how multiple factors contribute to a phenotype, and in particular, to a disease state, is essential to understanding both environmental impacts on the phenotype as well as in determining health recommendations or therapeutic targets for the prevention of disease.

Metabolic disease is an umbrella term that describes the manifestation of conditions such as high blood pressure, dysglycemia, obesity, and dyslipidemia (Skyler, 2004). Type II diabetes occurs as a result of acquired insulin insensitivity and abnormal insulin secretion, resulting in the dysregulation of blood glucose levels (Mahler & Adler, 1999; Skyler, 2004). This confers, most significantly, numerous macro and microvascular complications that can cause damage to, or failure of, multiple organ systems. Studies of individuals conceived during the Dutch famine of 1944-1945 have shown that there is a significant relationship between the timing of famine during gestation and the occurrence of metabolic diseases in adulthood. Ravelli and colleagues showed that famine experienced during mid- to late-gestation resulted in glucose intolerance at

age 50, with the most severe glucose intolerance being in individuals who were also deemed obese at that age (Ravelli et al., 1998). As previously noted, individuals who experienced famine later in gestation were more likely to have low birth weight.

Studies of the Dutch Famine cohort have also shown that individuals who experienced famine in early gestation were more likely to be obese (based on BMI) in later life. The tendency toward being overweight or obese has been linked through positive, linear relationships to birth weight, meaning high birth weight individuals were more likely to be obese according to their BMI. However, as discussed previously, the use of BMI as a measure of body condition can cloud phenotypic interpretations linking early life environment and disease risk. This further demonstrates the need for more specificity than identifying variables of interest in studies of phenotypic variation.

Being able to identify the environmental cues and their responses that collectively combine to create the phenotype and generate the vast array of human variation possible will allow for more precise health recommendations and intervention methods when it comes to preventing the non-communicable diseases outlined above. Additionally, being able to identify specific patterns of phenotypic variation and the combinations of environmental cues that cause it will potentially allow for more accurate environmental and health reconstruction of past populations.

2.6. Theoretical Synthesis and Anthropological Applications

2.6.1. Using Modern Populations to Study Variation

While the range of phenotypic variation in modern human populations almost certainly looks different than that which existed in ancient populations, the underlying physiological pathways and possible phenotypic patterns are the same. Therefore, studying the presentation

and etiology of phenotypic variation in modern populations is a productive avenue for biological anthropologists looking to understand the relationships between the environment, and phenotypic and health outcomes. Historically, the biomedical research that has identified relationships between early life influence and later-life phenotype has done so using simple health-related metrics such as stature and BMI. As a result, this has overlooked more subtle trends in human phenotypic variation, such as body composition, tissue distribution patterns, relative limb lengths, crural (lower leg length/upper leg length) and brachial (forearm length/upper arm length) indices, and other body dimensions that would be of interest to biological anthropologists studying human adaptability and health in both modern and archaeological contexts. The more subtle patterns of variation identified through the study of modern populations can be applied to archaeological populations to better understand the social, political, cultural, and environmental contexts of a population and the lived experience of an individual.

The use of variation in modern populations as a proxy for variation in archaeological populations is not a new practice. For example, the regression equations commonly used to estimate variables like height from skeletal remains are the product of the analysis of modern skeletal populations (Trotter & Gleser, 1952). However, these equations are often population-specific and may not provide a perfect representation of ancient populations. The analysis of modern humans as a model for ancient humans has also been employed in studies of energetics and mobility in the emerging field of human athletic paleobiology (e.g. Longman et al., 2020).

2.6.2. Bio-archaeological Applications

It is in the patterns of growth and development that we more clearly see the relationships that exist between the environment, life history, and variation in the phenotype. Research in growth and development in human biology and biological anthropology has attempted to identify

relationships between environmental influence and health outcomes. Influenced by the Barker hypothesis (Hales & Barker, 1992), the majority of biomedical research to date has failed to provide the relevance of early life influence and plasticity outside of the context of understanding health outcomes, and little attention has been paid to how other aspects of the phenotype are impacted, such as subtle patterns in body morphology and physiology. This focus on health outcomes has influenced biological anthropology, placing more emphasis on identifying pathology instead of evidence of plasticity and resilience in human populations. This necessitates a more nuanced approach that incorporates discussion of life history theory and plasticity to increase our understanding of human adaptability as a process that also generates variation.

Historically, there has been a reliance on non-specific indicators of stress such as Harris Lines and LEH by bioarchaeologists in studies of past human health. These stress indicators are taken as evidence of poor health, but nuanced discussion of how bony variation fits within the energetic strategy of an organism has generally been lacking. Recent research by Temple (2019) has taken a life history approach and engaged with discussions of how variation in bony structures are the result of energetic trade-offs and may be evidence of successful buffering during periods of resource scarcity, high energetic demand, or illness. The use of covariates such as age at death, other markers of stress or chronic infection, alongside measures of body size and morphology are needed to correctly interpret the energetic relationships between the individual and their environment and to better understand the causes of biological variation in the archaeological record (Temple, 2019). Through examining variation in enamel microstructures in conjunction with other markers of health in the Japanese Jomon population, Temple compared the PAR hypothesis to the plasticity/constraint hypothesis in their ability to explain how Late/Final Jomon people responded to early life stress and how it impacted their later health. It

was found that individuals with earlier first enamel defects were more likely to have subsequent defects and die earlier compared to those with first defects that formed later (Temple, 2014). This illustrates that early life stress events that required energetic trade-offs did not increase buffering ability and adaptation in later life as would be expected according to the PAR hypotheses (Temple, 2014). Instead, plastic responses in energy allocation favour immediate survival over investment in growth and future maintenance.

Another example of the use of an integrated life history and energetics approach in bioarchaeology is seen in the interpretation of height variation in a population. A reduction in a population's height over time is typically taken as an indicator of poor population health and nutrition (Kemkes-Grottenthaler, 2005). However, using an approach that incorporates considerations of life history and phenotypic plasticity can provide alternative, evidence-based interpretations in specific contexts. For example, recent research on populations in London, England before, during, and following the Black Death (1348-1350 CE) has identified seemingly contradictory evidence of female health. Following the plague and the accompanying reduction in population size, survivorship and other markers of health in London increased; however, female stature decreased while male stature increased (DeWitte & Lewis, 2020). Using a life history approach and incorporating considerations for energetics, plasticity, and population history, DeWitt and Lewis suggest the novel interpretation that reductions in height are a result of increased resource availability in childhood, leading to earlier puberty in females (DeWitte & Lewis, 2020). This has been validated by skeletal evidence of puberty completion, specifically the presence of fusion of the hook of hamate and mineralization of the canine. This interpretation is supported by evidence of secular trends of early menarche in modern industrial populations and the positive relationship between age at menarche and adult height attainment (Mcintyre &

Kacerosky, 2011). This integration of a life history approach in biological anthropology and bioarchaeology is fruitful, but to be able to identify these complex patterns of population and individual variation, health, and life history strategy we must first be able to identify these patterns in living populations. This will allow for a better understanding of the combination of circumstances that leads to the phenotypic variability we see in the archaeological record. It will also allow us to infer the circumstances which would have led to that variability when that evidence may otherwise be invisible on skeletal remains.

3. Methods

3.1. Sample Population

The sample data used in this study were collected in 2016 and 2017 by Dr. Murry and Dr. Stock as part of the ADAPT project with funding from the European Research Council. The anonymized sample is made up of 104 cis-gendered female athletes and non-athletes of European descent from Cambridge, UK. The varsity athlete sub-sample is made up of athletes from the Rowing, Running, and Soccer teams. The control sample is made up of non-varsity females with varying degrees of reported physical activity. Ethics approval for this study was sought and granted by the Human Biology Research Ethics Committee of Cambridge University.

The surface scan information for individuals 205, 259, 274, 301, 304, 305, and 308 was ultimately excluded from the final analyses due to problems with the scan rendering which could not be corrected, namely, baggy clothing causing surface irregularities which would artificially inflate the surface area and volume data. Small irregularities introduced by clothing or body position could be corrected for most individuals through the use of the selection and smoothing tools available in the MeshMixer 1.5 software created by Autodesk.

Expansion of this data set through the recruitment of varsity athletes from The University of Western Ontario was intended for the fall of 2020 and ethics application materials were composed, however, due to provincial closures taking place as a result of the COVID-19 pandemic, this was not possible. As a result, this study is limited to the data set collected by Dr. Murray and Dr. Stock in 2016/2017.

3.2. Data Collection

3.2.1. Survey

Life history information for each participant was collected through a survey which covered information relating to their early life (date of birth and birth weight, if known), history of physical activity during the developmental period, and age of first menstruation. Studies measuring the correlation of recalled and actual age at menarche have been variable, but overall, show moderate to strong correlations (R = 0.60-0.83), with younger individuals more reliably recalling the age of their first period (Biro et al., 2018; Dorn et al., 2006; Koprowski et al., 2001; Must, 2002). Similarly, parent recalled birth weight shows a very strong correlation (R = 0.90) with actual birth weight, making it a suitable metric in epidemiological studies (Shenkin et al., 2017; Walton et al., 2000).

3.2.2. Anthropometric Measures

Anthropometric measures of each participant were taken by Dr. Murray using a stadiometer, scale, sliding calipers, anthropometer, and a tape measure. These measures include stature; body mass; bi-iliac breadth; waist and hip circumference; and humerus, femur, and tibia lengths. Measurement protocol was taken from Standards for Anthropometric Assessment (Norton, 2018).

3.2.3. Bio-electrical Impedance

Bioelectrical impedance analysis (BIA) was also conducted to measure fat and lean muscle mass for each participant. BIA employs the use of a safe electrical current in order to measure the effective resistance of that individual. This method estimates total body water (TBW), which is then used to estimate fat-free mass. The limitation of BIA is that it estimates TBW (and therefore, fat-free mass) using regression equations linking stature and impedance to

TBW rather than measuring it directly. In a study by Wells and Fewtrell (2006), they report that the use of the index of 1/Impedance (1/R) reliably predicts lean mass index and, therefore, their equation was used in this study.

3.2.4. Size Stream 3D Body Surface Scanner

Three-dimensional (3D) body surface scans were taken using the Size Stream 3D Body Scanner, Version 12 (Figure 2). This machine uses a combination of infrared sensors and cameras to capture depth information for two-million surface points and uses this to generate a three-dimensional mesh representing the surface of the participant. From this, the Size Stream Studio software can generate over 240 different body measures, including various lengths, circumferences, and breadths (*Size Stream Body Scanner Assembly and Operation Manual*, 2014). The scan is complete in roughly six seconds and the resulting mesh is constructed in two minutes. This non-invasive measurement system is efficient and safe to use.

3.2.4.1. Validating the Size Stream Body Surface Scanner in Anthropometry

One of the aims of this study was to assess the applicability of the Size Stream 3D Body Scanner in the collection and study of anthropometric measures of human variation. Stature measured with the stadiometer was compared to the auto-generated stature measurement calculated by the Size Stream Studio program for the 100 individuals with paired measures. Shapiro-Wilk normality tests confirmed that both measures were normally distributed. A one-sample t-test examining the difference in the means between the two measurement methods shows there is a significant difference (n =

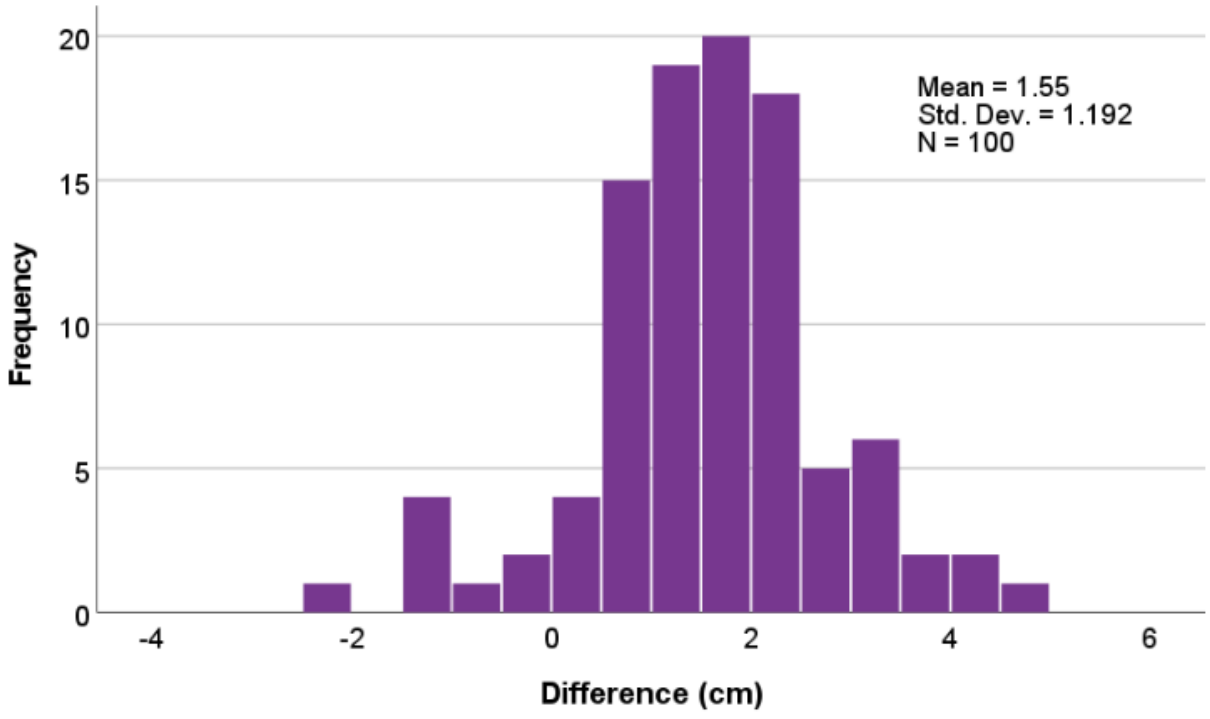
Figure 2 *Size Stream Scanning Booth*

100, $p < 0.001$). This is reflected in a frequency distribution that also shows a right shift indicating that the stadiometer height was on average greater than the Size Stream autogenerated height (Figure 3).

The agreement between the two techniques was then further assessed through the use of a Bland-Altman Plot which displays the upper and lower limits of agreement (Figure 4). This plot shows ten data points that fall outside the limits of agreement. Reanalysis following the removal of these points from the test was not found to improve the agreement between the methods.

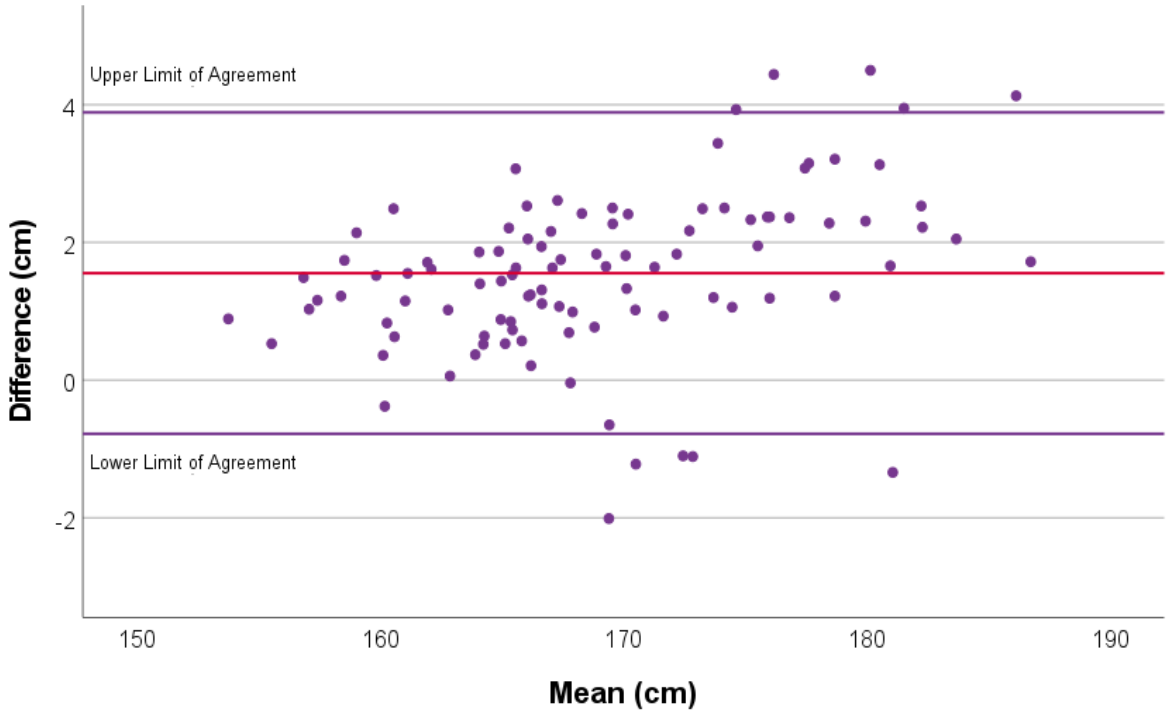
The disagreement between the two methods may be a result of underestimation of stature by the Size Stream or overestimation by the stadiometer, however, the differences are more likely to be a result of differences in measurement protocols rather than an error with the

Figure 3 *Method Based Difference in Stature*



Note. Difference was calculated as stadiometer stature minus Size Stream stature. The right shift in the histogram indicates that on average, the stadiometer stature is larger than the Size Stream stature.

Figure 4 *Bland-Altman Plot Showing Level of Agreement Between Measurement Methods*



Note. The plot depicts the upper and lower limits of agreement based on the unit difference between the two methods of stature measurement. A total of 10 data points fall outside the limits of agreement.

techniques themselves. Specifically, when stature is taken with a stadiometer, the subject is standing with feet close together and back against a vertical surface, however, when scanned in the Size Stream, subjects have their feet spaced slightly apart to allow for volume and surface area measures of the legs to take place. Additionally, subjects are also behind a curtain for privacy, meaning that the researcher cannot observe or correct the subject's posture. These factors in combination may result in the underestimation of stature by the Size Stream 3D Body Scanner, leading to the statistical disagreement between the methods.

It is important to note that the Size Stream was not developed with anthropometry in mind, but rather as a commercial device for tailors and therefore, the measures it takes are not designed to maximize stature. Furthermore, many of the Size Stream measures are non-standard anthropometric variables and are specifically relevant to 3D body scans, so they are not

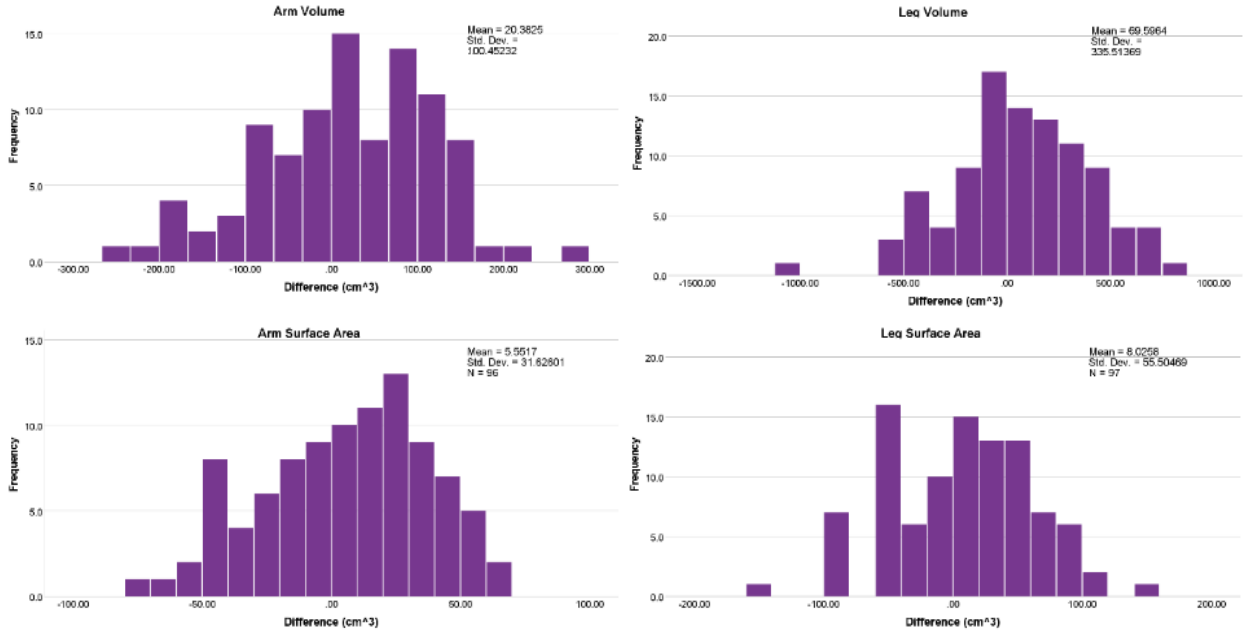
necessarily intended to be comparable to measures taken using standard anthropometric methods. Keeping this in mind, the Size Stream still provides informative measures of body dimension and shows the need for further validation.

It was decided that the absolute measures generated by the Size Stream alongside relative measures of segment lengths, SA, and Vol (e.g., Lower Leg Vol: Upper Leg Vol) would be used in the subsequent analyses with an understanding of the potential limitations caused by the differences in measurement protocols. While there were differences between the two measurement techniques, there is no reason to believe that the proportions generated by the Size Stream were affected (see the next section), therefore, the data generated by the Size Stream can still provide meaningful contributions to this research and to the study of how continuous phenotypic variation is linked to the early life environment.

3.2.4.2. Determining Limb Asymmetry

Before limb volume and surface area data could be used in further analyses, it was prudent to determine if there were significant asymmetries between the limbs as a result of limb dominance, processing errors introduced to the mesh by Size Stream Studio, or during segmentation in MeshMixer 3.5. Interlimb differences in volume and surface area were calculated by subtracting the left-side value from the right-side value and a one-sample t-test was performed. It was determined that there were significant differences between the arm and leg segment pairs in volume but not in surface area. Small changes in surface area result in disproportionally higher changes to volume, therefore, this may explain the difference being found in volume but not surface area. Frequency histograms (Figure 5) of the surface area and volume differences for arm and leg segments showed that there was a positive bias that favoured the right side. Hand dominance data for this sample showed that for n = 104, 92.3% of

Figure 5 *Frequency Histograms of Volume and Surface Area Differences*



Note. The means of all four difference variables are positive, indicating a right-side bias for surface area and volume.

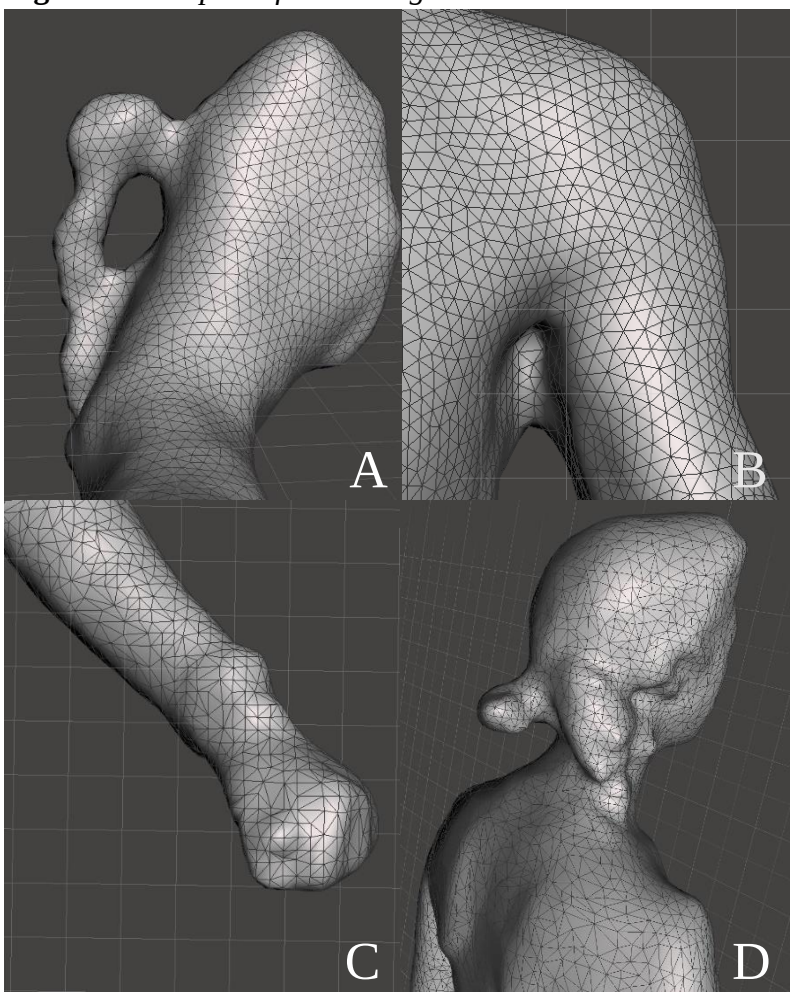
individuals self-identified as right dominant, 5.8% were left-dominant, and 1.9% were ambidextrous. A meta-analysis of handedness in 2020 reported that non-right-handed individuals can account for 9.3% to 18.1% of the population, with best estimates putting non-right-handedness at 10.4% of the population (Papadatou-Pastou et al., 2020). Based on this, it is likely that the asymmetry in limb volumes is a result of side-based dominance rather than the result of an error introduced during scanning or processing. Studies have also shown that athletes that participate in sports with un-even side engagement can exhibit bi-lateral differences in musculature and that non-athletes also show significant side-based differences in musculature (Burdukiewicz et al., 2020). Side-averaged values were used in the subsequent analyses to look at overall proportionality in volume and surface area relationships.

3.3. Data Processing

3.3.1. 3D Scan Clean-Up and Auto-generated Measurement Validation

Scans were loaded into the 3D visualization and editing software MeshMixer 3.5 by Autodesk. Here, it was identified whether the 3D meshes generated using the body scanner software contained any errors. Common errors encountered include ‘pooling’ of the feet (Figure 6A), ‘webbing’ of the underarms and groin areas (Figure 6B), protrusions due to clothing or watches (Figure 6C), and incorrect reconstruction of hair (Figure 6D). The webbing artifact was corrected by removing the triangle polygons making up the webbed portion (using visual landmarks as a guide), and then filling in the resulting holes in the mesh using MeshMixer’s Bridge and Inspector functions. Unusual protrusions, such as the one evident in Figure 6C, were corrected using the sculpting function in MeshMixer, again, using the visible anatomical structures as a guide.

Figure 6 *Examples of Rendering Errors*



Note. A) Pooling of the mesh at the foot, B) armpit webbing error, C) wristwatch causing a volume error, and D) hair rendering error.

3.3.2. Segmentation Protocol

The segmentation protocol employed in this study is modeled after the segmentation protocol presented by McConville and colleagues (1980) and adapted to fit a female model (Figure 7).

The McConville protocol identifies 11 planes of segmentation, seven of which were used in this study. They are identified as follows:

Neck plane: Originates at the superior surface of the right and left clavicles and rises diagonally until it intersects with the vertebral prominens.

Hip plane: Originates at the center of the groin and passes laterally midway between the anterior superior iliac spine and the trochanteric landmarks along the lines of the right and left inguinal ligaments.

Knee plane: passes transversely through the lateral femoral epicondyle landmark.

Ankle plane: originates at the sphyrion landmark and passes transversely through the ankle.

Shoulder plane: originates at the acromion landmark and passes downward through the anterior and posterior scye creases at the level of the axilla.

Elbow plane: originates at the olecranon landmark and passes through the medial and lateral humeral epicondyle landmarks.

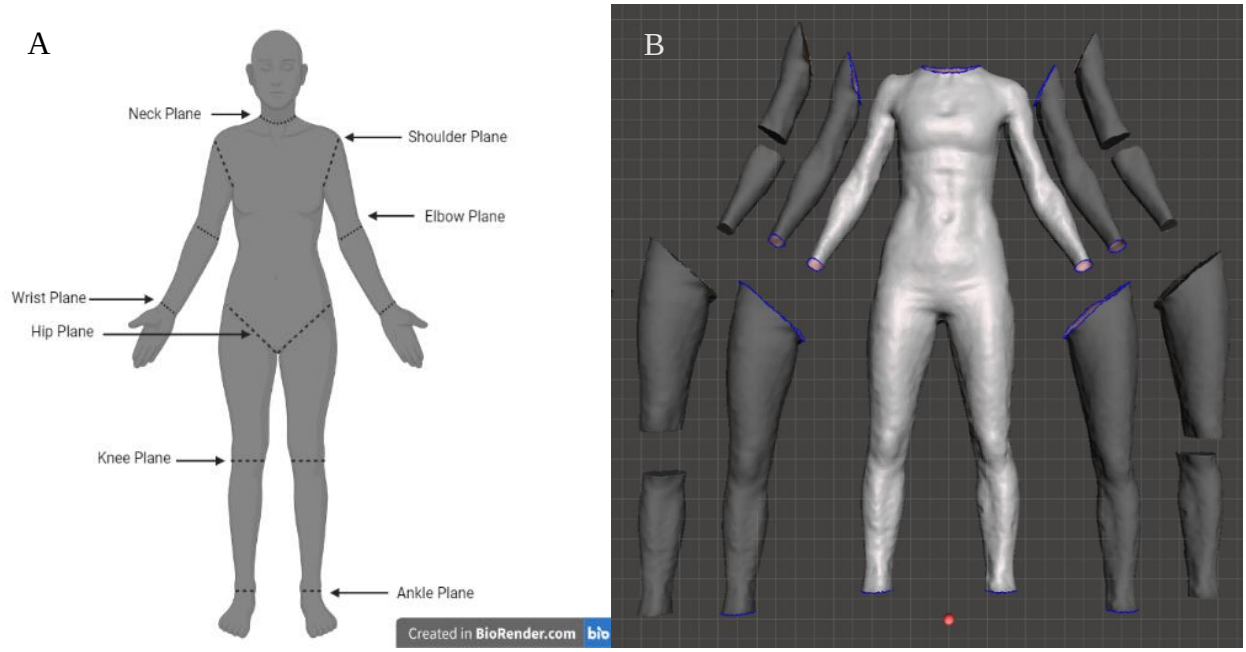
Wrist plane: originates at the ulnar and radial styloid landmark and passes through the wrist perpendicular to the long axis of the forearm

When combined, these planes create 25 segments, nine of which were identified for this study. These segments were:

1. Trunk
2. Right Upper Arm
3. Right Forearm
4. Left Upper Arm
5. Left Forearm
6. Right Upper Leg
7. Right Calf
8. Left Upper Leg
9. Left Calf

Using the identification instructions outlined by McConville and colleagues (McConville et al., 1980), the relevant planes of segmentation were identified visually on the 3D surface scans for each individual. The 3D segments were then highlighted and isolated using the selection function in the program MeshMixer 1.5. Volume and surface area information automatically calculated by the program was then recorded for each segment.

Figure 7 *Example Segmentation Spread*



Note. A) Diagram of segmentation planes used in this study, adapted from McConville and colleagues, 1980. B) Model segmentation of Subject ID 300 in MeshMixer.

3.4. Statistical Tests

3.4.1. Pearson Correlation Analysis and General Linear Modeling

To determine if there were differences between the athlete and non-athlete sub-samples in birth weight or age at menarche, a T-test, and a Mann-Whitney U test were conducted (respectively). Due to the large number of variables and univariate tests performed, a Bonferroni correction was calculated and applied. Correlations that remained significant following the correction were identified. Individuals were identified as either ‘athlete’ or ‘non-athlete’ based on their self-reported participation in a sport. This was then used as a fixed factor in the general linear modeling (GLM) regression analyses to control for the effect that participation in a sport, and the potentially higher energetic demands that entails, may have on relationships between life history and phenotypic measures. All variables analysed in this study can be found summarized in the Appendix.

Interpretation of correlation coefficients varies between fields, with different thresholds for what is considered a moderate or strong correlation (De Muth,

Table 1 *Canonical Correlation Variable Groupings*

Early Life Variables
Age at Menarche
Birth Weight
Linear Growth
Femur Length
Forearm Length
Humerus Length
Stature
Tibia Length
Trunk Length
Body Composition
% Dry Lean Mass
% Fat Mass
Body Mass
Waist Circumference
Body Morphology
Bicep Circumference
Calf Circumference
Chest Circumference
Forearm Circumference
Hip Circumference
Max Stomach Circumference
Mid-Thigh Circumference
Underbust Circumference
Waist:Hip Ratio
Intersegment Size Relationship
Brachial Index
Crural Index
Arm:Trunk SA
Arm:Trunk Vol
Calf:Thigh SA
Calf:Thigh Vol
Forearm:Upper Arm SA
Forearm:Upper Arm Vol
Leg:Trunk SA
Leg:Trunk Vol
Body Breadths
Bi-iliac Breadth
Shoulder Breadth
Thermoregulation
Arm SA:Vol
Leg SA:Vol
Trunk SA:Vol

2014; Overholser & Sowinski, 2008). For the present study, correlation coefficients were interpreted as follows: the R values less than 0.19 were interpreted as weak. The R values between 0.20 and 0.39 were interpreted as fair. The R values between 0.40 and 0.69 were interpreted as moderate, and R values greater than 0.70 were interpreted as strong.

3.4.2. Canonical Correlation Analysis

To understand how the life history variables considered in this study contribute to variation in different areas of the phenotype, canonical correlation analyses (CCA) were performed. This type of analysis is useful in the scenario that independent variables of interest are found to be colinear, which birth weight and age at menarche were found to be in this sample. CCAs compare two sets of variables and measure the associations between them. The CCA measures the correlation between constructed canonical variates that are created by applying a linear equation to the predictor and criterion variables. The resulting equations are created to yield the greatest possible correlation between the two synthetic variables (Sherry & Henson, 2005). This test allows the comparison of groups of variables that are collinear and indicates which variables within the set contribute most strongly to the variation in the other set. Additionally, this test reduces the chance of Type I error as the variables are examined simultaneously (Sherry & Henson, 2005). The variables collected in this study were split into six variable sets based on their shared attributes (Table 1). These groups were: variables relating to linear growth, variables related to body composition, variables related to intersegment size relationships, body breadth variables, variables related to body morphology and form, and variables related to thermoregulation. These six variable sets were compared in CAA to a life history variable set which contained birth weight and age at menarche. A summary of terms related to the CCA, and their respective definitions, can be found in Table 2.

Table 2 *Canonical Correlation Terms and Definitions*

Term (Alternative Name)	Definition
Canonical Correlation Analysis	Examines the correlation between synthetic criterion and predictor variables know as canonical variates.
Canonical Correlation Coefficient	The Pearson r correlation coefficient between the two synthetic variates.
Canonical Function	The number of canonical functions is limited by the number of variables in the smaller of the two variable sets being compared. The first canonical function is the most strongly correlated and all subsequent functions are created with the residual variance not used in the first function. Each function is orthogonal to one another and can be independently analysed.
Canonical Variates	Synthetic variables created from the criterion and predictor variables sets that maximize the correlation between the two synthetic variables.
Canonical Weights (Standardized Canonical Correlation Coefficients)	Variables with larger weights contribute more to the variates.
Cross Loadings	The correlation between an individual variable and the opposite synthetic variate.
Squared Canonical Correlation (Roots, Eigenvalues)	Square of the canonical correlation coefficient and represents that shared variance between the two variable sets.
Canonical Loading (Structure Coefficient)	The correlation between an individual variable and the synthetic variable it is a part of. Indicates which of the variables is integral to the creation of the synthetic variate.

4. Results

4.1. Descriptive Statistics

The summary statistics for the pooled study sample, along with separated athlete and non-athlete sub-groups are presented in Table 1 of the Appendix. In the case of missing data, individuals were removed from tests in a pairwise fashion unless otherwise noted. All characteristics with continuous data were tested for normal distributions using the Shapiro-Wilk test. Body mass, waist circumference, % fat mass, % dry lean mass, age at menarche, and the brachial index did not have normal distributions, however, linear regression analyses are not overly sensitive to deviation from normality when observations per variable are >10 (Schmidt & Finan, 2018). Canonical correlation analysis requires variable distributions to be approximately normal and this was assessed by examining frequency histograms (Sherry & Henson, 2005).

Independent sample T-tests for normally distributed data and Mann-Whitney U tests for non-normal data were performed to determine if there were differences between the athlete and non-athlete sub-groups in the early life variables birth weight and age at menarche (See Table 2, Appendix). It was determined there was no significant difference between the athlete and non-athlete subgroups in terms of their early life variables, therefore, athlete and non-athlete sub-groups were combined to examine the overall trends in adult phenotypic variation and how it relates to early life environment.

4.2. Birth Weight and Correlations with Adult Phenotypic Outcomes

Birth weight, a measure of the maternal environment and energetic availability during fetal development, was compared to the outcome variables representing the adult physical phenotype to first determine whether previously established relationships with stature, body mass, and body composition could be identified in this sample, and second, if the variation in

surface area and volume relationships observed in adulthood were related to variation in the early life environment. Characteristics associated with birth weight are presented in Table 3.

In this sample, birth weight was not found to be significantly associated with any measures of body composition other than body mass, which has a weak positive relationship, reporting an R^2 of 0.048, which increased to 0.051 when sports participation was included in the GLM. Birth weight was found to have the highest correlation with stature, reporting an R^2 value of 0.091. The positive correlations between birth weight and stature, body mass, and bi-iliac breadth are of interest as stature and bi-iliac breadth have been used together in some body mass estimation procedures (Ruff et al., 2005). To further investigate this relationship, a model was tested that combined both stature and bi-iliac breadth. It was found that birth weight shared more variation with stature than bi-iliac breadth, producing an overall R^2 value of 0.104. No significant relationships between birth weight and any of the volume or surface area variables were observed in this sample.

Table 3 *Correlations with Birth Weight (g)*

Characteristics	n	Sig.	R	R^2	Sport**	R^2 with Sport
Age at Menarche	87	0.028	0.236	0.056	No	0.056
Bi-Iliac Breadth (cm)	87	0.020	0.248	0.062	No	0.062
Body Mass (Kg)	87	0.042	0.219	0.048	No	0.051
Shoulder Breadth (cm)	81	0.045	0.223	0.050	No	0.056
Stature (cm)	87	0.005	0.301	0.091	No	0.097
Tibia Length (cm)	85	0.039	0.224	0.050	No	0.052
% Fat Mass	80	0.620	-0.056	0.003	No	0.007
% Dry Lean Mass	80	0.161	0.158	0.025	No	0.032

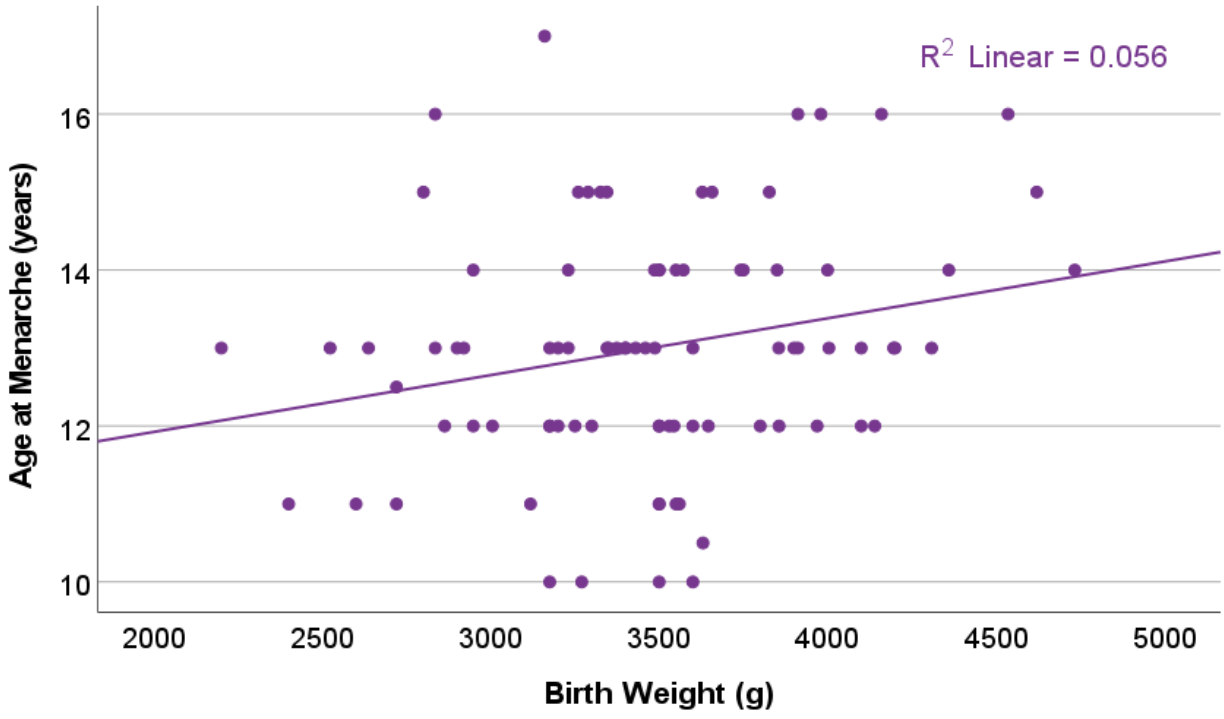
*Remains significant with Bonferroni Correction applied
** Does sport contribute to variance in the dependant variable?

4.3. Birth Weight and Relationship to Age at Menarche

Weight at birth is known to influence the age at menarche in females, with low birth weight individuals often experiencing an earlier age at menarche than their peers. To determine if there was a relationship between the pre-birth environment and later life reproductive strategy, self-reported birth weight was compared to age at menarche.

In line with previous research (Sloboda et al., 2007; Tam et al., 2006), Pearson correlation analyses identified a significant relationship between birth weight and age at menarche (n = 87, R = .236, $p < 0.05$), producing a fair positive correlation (Figure 8). A GLM with sport included as a fixed factor showed that birth weight significantly contributes to a small proportion (6.2%) of the variation in the age at menarche in females, with a non-significant contribution by sport-participation.

Figure 8 *Correlation Between Birth Weight and Age at Menarche*

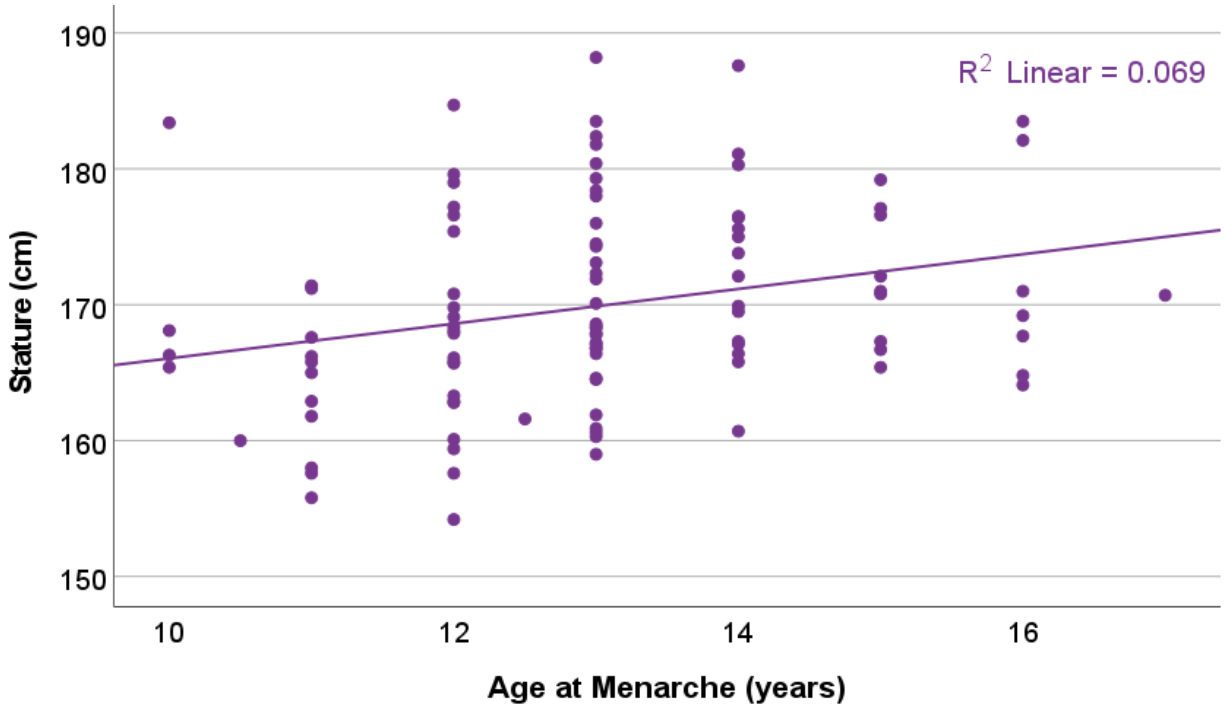


Note. There is a significant, fair relationship between age at menarche and birth weight (n = 87, R = .236, $p = 0.028$).

4.4. Age at Menarche and Impact on Linear Growth

Age at menarche is known to influence the growth program through the initiation of a hormonal cascade that, in addition to driving the development of secondary sexual characteristics, causes the cessation of long bone growth in females. Females who experience menarche earlier than the average tend to be shorter than their peers, whereas those who experience it later tend to be taller. To better understand the relationship between age at menarche and linear growth in this sample the correlation with stature was examined. Linear regression showed that age at menarche accounts for only 6.9% of the variation in stature (Figure 9).

Figure 9 *Correlation Between Age at Menarche and Stature*



Note. There was a significant, fair correlation between age at menarche and stature (n = 104, R = .263, p < 0.01.

Age at menarche was also found to have significant but weak correlations with other measures of linear growth, specifically, humerus, femur, and tibia lengths (Table 4). As expected, these characteristics are also closely correlated with stature. These findings indicate that the variation in birth weight and age at menarche together account for some of the variation in linear growth observed in this sample.

Table 4 *Correlations Between Age at Menarche and Later Life Phenotypic Outcomes*

Characteristics	n	Sig.	R	R^2	Sport**	R^2 with Sport
% Dry Lean Mass	97	0.002	0.309	0.095	No	0.113
% Fat Mass	97	0.010	-0.262	0.069	No	0.091
Birth Weight (g)	87	0.028	0.236	0.056	No	0.062
Femur Length (cm)	102	0.003	0.295	0.087	No	0.088
Humerus Length (cm)	102	0.015	0.241	0.058	No	0.058
Stature (cm)	104	0.007	0.263	0.069	No	0.070
Tibia Length (cm)	102	0.008	0.261	0.068	No	0.068
Waist Circumference (cm)	104	0.036	-0.206	0.042	No	0.045

*Remains significant with Bonferroni Correction applied
** Does sport contribute to variance in the dependant variable?

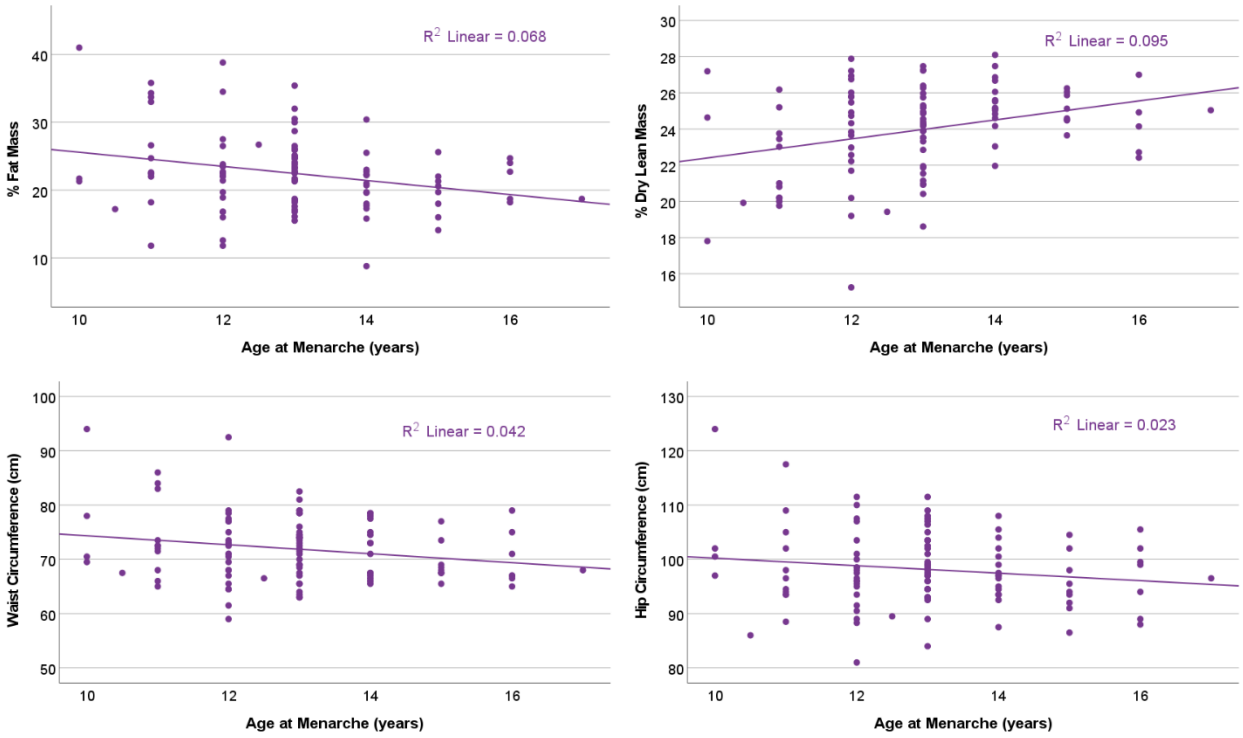
4.5. Age at Menarche and Tissue Investment Strategy

There is evidence to suggest that the investment in different tissue types and their distributions in the body are related to the life history strategy of an organism and that the distribution of lean and fat mass in human females is related to investments in sexual maturation and reproductive potential (Nepomnaschy et al., 2020). To assess this relationship, age at menarche was compared to measures reflecting adult body composition and gluteal-femoral tissue distribution.

Age at menarche was found to have a fair positive correlation with % dry lean mass and a fair negative correlation with % fat mass (Table 4) (Figures 10A & 10B). Additionally, age at menarche was found to have a fair negative correlation with waist circumference, such that as the age at menarche increases for an individual, they were more likely to have a smaller waist

circumference (Figure 10C). However, only 4.2% of the variation in waist circumference is accounted for by age at menarche. There was no correlation between age at menarche and hip circumference or waist to hip ratio, which does not support a direct relationship between age at menarche and investment in gluteal-femoral fat depots (Figure 10D).

Figure 10 *Age at Menarche Plotted Against Measures of Tissue Investment*



4.6. Correlations Within the Adult Phenotype

Previous research in human life history has examined the relationship between early life variables and their influence on the phenotype. However, somewhat neglected is the understanding of how the outcome variables of the phenotype relate to each other. The goal of this analysis was to identify where phenotypic outcome variables covaried and interpret how this might relate to larger patterns of adaptation and normal human variation. Outcome variables were compared using Pearson correlation analyses to determine colinear relationships.

4.6.1. Correlations with Body Composition

Fat mass and lean mass are suggested to be strongly influenced by early life environment, therefore, the relationship of these variables to other life history and phenotypic outcome variables can inform researchers about the conditions experienced during development and the resulting programming of an individual's life history strategy. The significant phenotypic relationships with % fat mass are summarized in Table 5.

Table 5 *Correlations Between % Fat Mass and Other Phenotypic Traits*

Characteristics	n	Sig.	R	R^2	Sport**	R^2 with Sport
% Dry Lean Mass*	97	$p < 0.001$	-0.653	0.426	Yes	0.460
Age of Menarche	97	$p = 0.01$	-0.262	0.069	Yes	0.198
Arm SA/Vol*	91	$p < 0.001$	-0.394	0.155	Yes	0.364
Arm/Trunk Vol	91	$p < 0.05$	-0.234	0.055	Yes	0.110
Calf/Thigh SA*	91	$p < 0.001$	-0.405	0.164	Yes	0.295
Calf/Thigh Vol*	90	$p < 0.001$	-0.509	0.259	Yes	0.321
Forearm/Upper Arm Vol*	90	$p < 0.001$	-0.544	0.296	Yes	0.335
Forearm/Upper Arm SA*	90	$p = 0.001$	-0.349	0.122	Yes	0.179
Leg SA/Vol*	91	$p < 0.001$	-0.536	0.287	Yes	0.450
Body Mass (Kg)*	97	$p < 0.001$	0.413	0.171	Yes	0.364
Hip Circumference (cm)*	97	$p < 0.001$	0.603	0.364	Yes	0.503
Stature (cm)	97	$p < 0.01$	-0.297	0.088	Yes	0.171
Trunk SA/Vol*	91	$p < 0.001$	-0.615	0.378	Yes	0.520
Waist Circumference (cm)*	97	$p < 0.001$	0.62	0.384	Yes	0.535
Waist:Hip	97	$p < 0.05$	0.201	0.040	Yes	0.178
Chest Circumference (cm)*	91	$p < 0.001$	0.508	0.258	Yes	0.406
Underbust Circumference (cm)*	91	$p = 0.001$	0.349	0.122	Yes	0.309
Calf Circumference (cm)*	91	$p < 0.001$	0.379	0.144	Yes	0.247
Forearm Circumference (cm)	91	$p < 0.05$	0.220	0.048	Yes	0.234
Mid-thigh Circumference (cm)*	91	$p < 0.001$	0.471	0.221	Yes	0.432
Max Stomach Circumference (cm)*	92	$p < 0.001$	0.578	0.334	Yes	0.478
Bicep Circumference (cm)*	91	$p < 0.001$	0.516	0.266	Yes	0.473

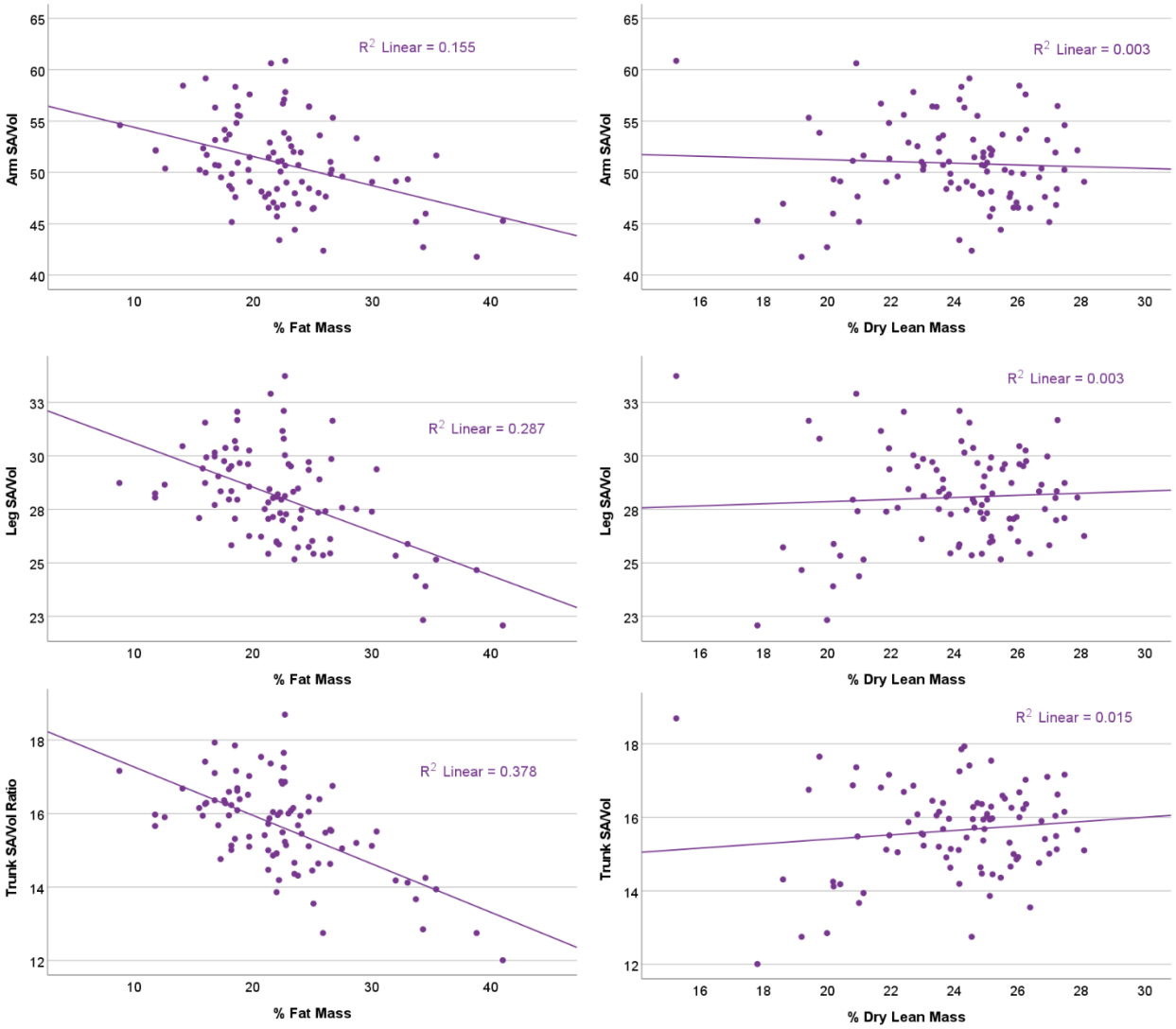
*Remains significant with Bonferroni Correction applied
**Does sport contribute to variance in the dependant variable?

Pearson correlation analyses found that % fat mass was negatively associated with many of the volume and surface area measures presented in this study (Table 5), such that increases in % fat mass were associated with decreases in SA:Vol ratios in the arms, legs, and trunk, and intersegment volume and surface area ratios (e.g., forearm/upper arm volume, forearm/upper arm

surface area, etc.) (Figure 11). This indicates that increases in % fat mass are associated with body-wide increases in volume, which in turn decreases SA:Vol values. This is supported by the correlations observed between % fat mass and measures of limb and trunk circumference, as % fat mass was not found to be correlated with any linear limb and body measures.

Furthermore, increases in the volume and surface area of the proximal limb segments correlated with a reduction in the intra-limb volume and surface area relationships. Using a cylinder model for the limbs, changes in diameter have a larger effect on volume than changes in

Figure 11 *% Fat and Dry Lean Mass Plotted Against Segment SA:Vol Ratios*



length (Cross et al., 2008; Kasabova & Holliday, 2015). In this sample, neither % fat mass nor % dry lean mass was associated with birth weight, but both variables were correlated with age at menarche.

The relationships between % dry lean mass and the various surface area and volume variables examined in this study were positive, meaning that increases in % lean mass were correlated with increases in the arm to trunk volume ratios (Table 6, Figure 12). However, unlike % fat mass, there was no correlation between % dry lean mass and the surface area to volume ratios for the limb and trunk segments (Figure 11).

Lean mass was also negatively correlated with waist to hip ratio (n = 97, R = -.262, p = 0.01), such that increases in % dry lean mass were associated with decreases in the waist to hip ratio (Figure 13). Changes to waist circumference have a larger impact on the waist to hip ratio than changes of the same magnitude to hip circumference. Pairing this with the moderate positive correlation between % fat mass and waist circumference, it can be inferred that the negative relationship between % dry lean mass and waist to hip ratio is a result of reductions in

Table 6 *Correlations Between % Dry Lean Mass and Phenotypic Variables*

Characteristics	n	Sig.	R	R^2	Sport**	R^2 with Sport
% Fat Mass*	97	$p < 0.001$	-0.653	0.426	No	0.427
Arm/Trunk Vol	91	$p < 0.01$	0.294	0.086	No	0.113
Bi-iliac Breadth (cm)	97	$p < 0.01$	0.287	0.082	Yes	0.143
Calf:Thigh SA	91	$p = 0.05$	0.206	0.042	Yes	0.127
Calf:Thigh Vol	90	$p < 0.01$	0.300	0.090	Yes	0.140
Crural Index	95	$p < 0.05$	0.236	0.056	Yes	0.097
Femur Length (cm)*	95	$p < 0.001$	0.561	0.315	Yes	0.362
Forearm/Upper arm SA	90	$p < 0.05$	0.248	0.062	Yes	0.107
Forearm/Upper Arm Vol*	90	$p = 0.001$	0.338	0.114	No	0.151
Humerus Length (cm)*	95	$p < 0.001$	0.627	0.393	No	0.403
Lower Arm Length (cm)*	96	$p < 0.001$	0.396	0.157	No	0.171
Menarche	97	$p < 0.01$	0.309	0.095	Yes	0.170
Stature (cm)*	97	$p < 0.001$	0.749	0.561	No	0.569
Tibia Length (cm)*	95	$p < 0.001$	0.602	0.362	No	0.384
Trunk Length (cm)	90	$p < 0.05$	0.232	0.054	Yes	0.133
Waist:Hip	97	$p = 0.01$	-0.262	0.069	Yes	0.152

*Remains significant with Bonferroni Correction applied
**Does sport contribute to variance in the dependant variable?

waist circumference, rather than increases in hip circumference. This also suggests that the positive relationship observed between % dry lean mass and Arm:Trunk volume ratios is influenced by reductions in trunk volume rather than increases in arm volume (Figure 12). Bi-iliac breadth, a variable commonly used in regression equations to estimate surface area, volume, and body composition, was found to be correlated with % dry lean mass, but not % fat mass (Table 7, Figure 14).

Figure 12 *% Dry Lean Mass Plotted Against Measures of Segment Surface Area and Volume*

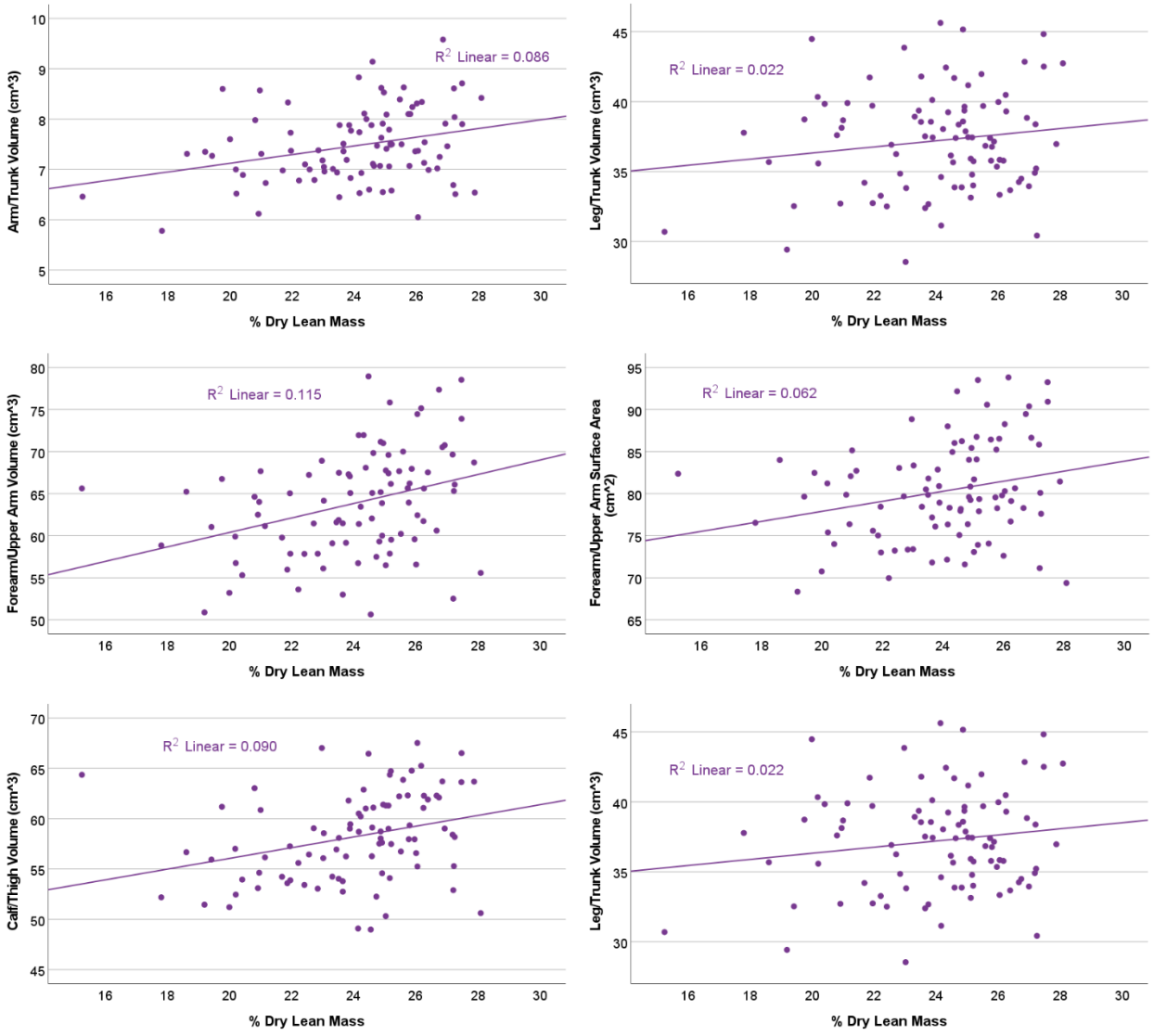
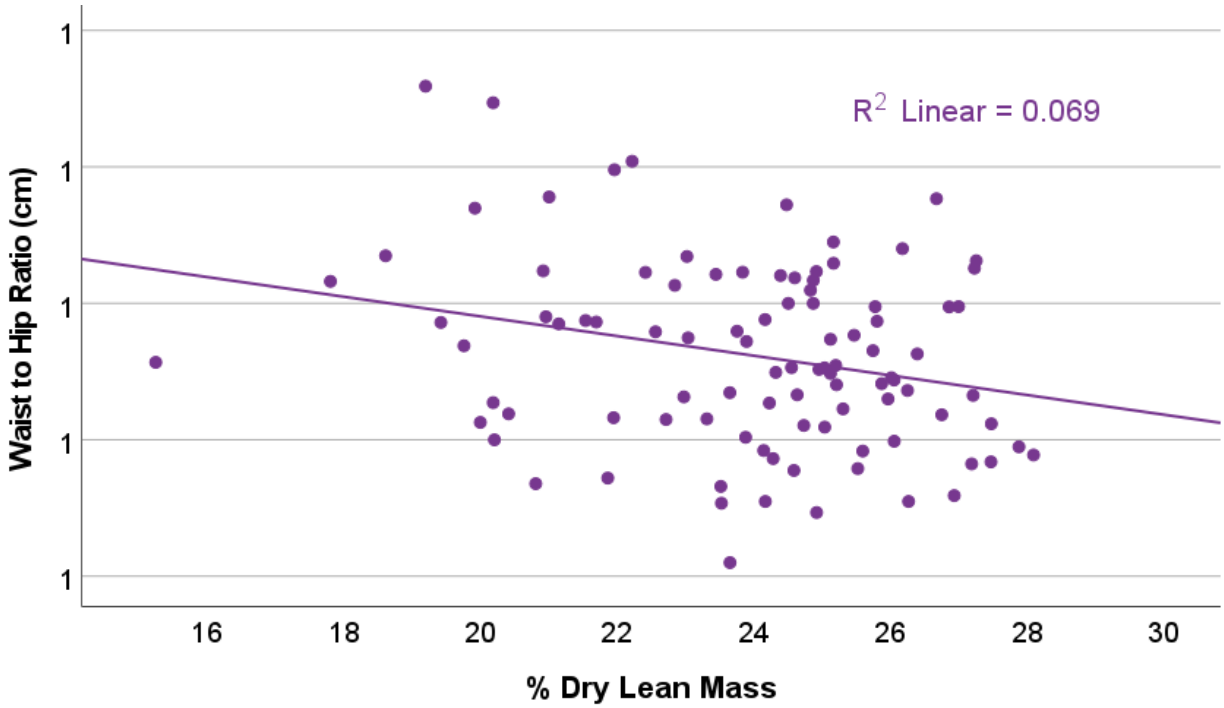
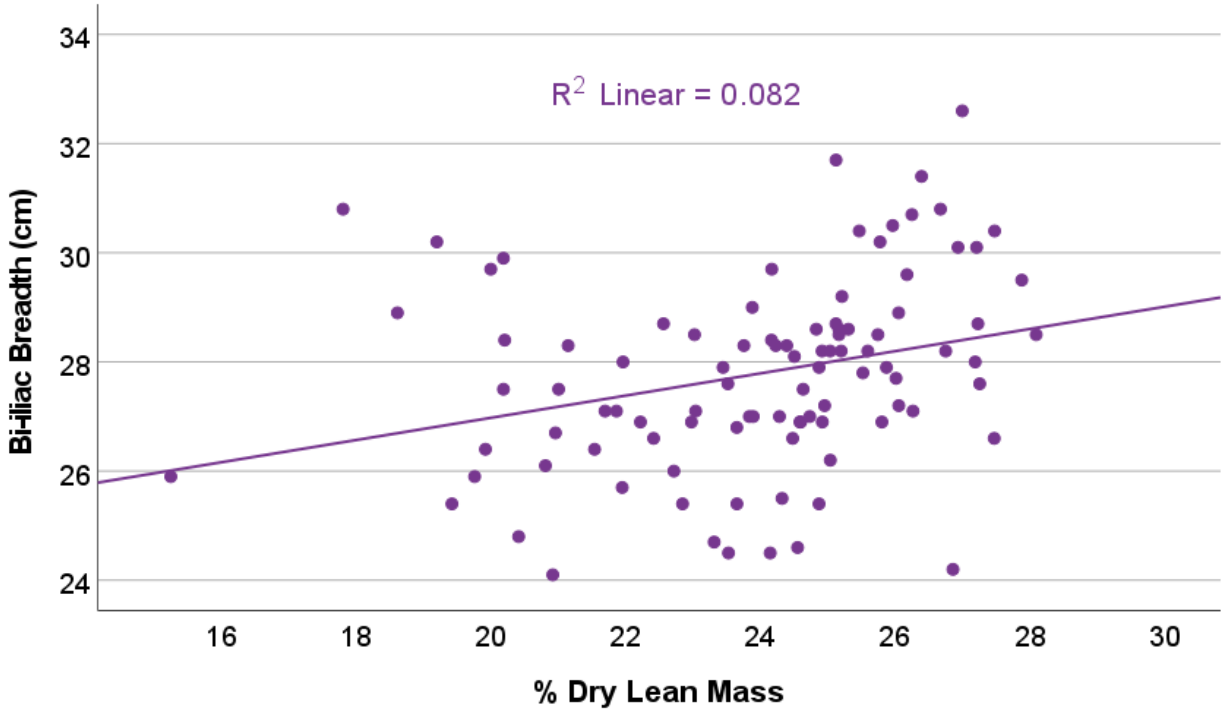


Figure 14 *% Dry Lean Mass Plotted Against Waist:Hip Ratio*



Note. There was a significant, fair negative correlation between % dry lean mass and Waist:Hip ratio (n =97, R = -.262, p = 0.01).

Figure 13 *% Dry Lean Mass Plotted Against Bi-iliac Breadth*



Note. There was a significant, fair correlation between % dry lean mass and bi-iliac breadth (n = 97, R = 0.287, p < 0.01).

Waist and hip circumferences, which are indicators of the tendency toward a centralized mass distribution in the body are also important risk factors in the context of metabolic and cardiovascular diseases. These variables were shown to have moderate to strong correlations with the measures of mass distribution in the limbs and trunk presented in this study (Tables 8 and 9). Both waist and hip circumference showed strong negative correlations with leg, arm, and trunk surface area to volume ratios (Figure 15), such that increases in hip or waist circumferences were associated with either increases in volume or decreases in surface area in the limb segments. Given that both waist and hip circumferences are highly correlated with % fat

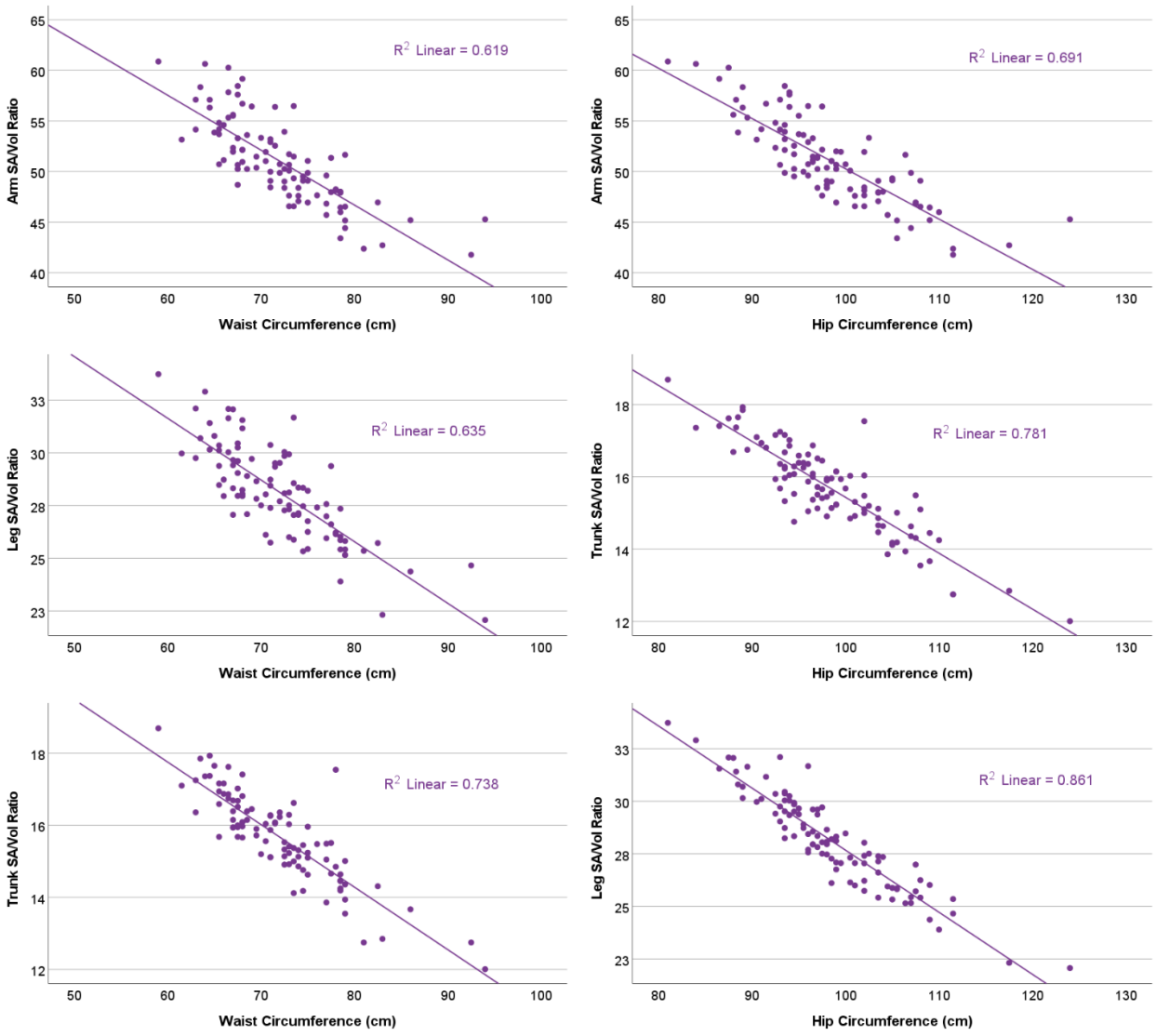
Table 7 *Phenotypic Correlations with Bi-Iliac breadth (cm)*

Characteristics	n	Sig.	R	R^2	Sport**	R^2 with Sport
% Dry Lean Mass	97	$p < 0.01$	0.287	0.082	No	0.083
Arm SA/Vol	97	$p < 0.001$	-0.355	0.126	No	0.127
Arm/Trunk SA	97	$p < 0.05$	-0.227	0.052	No	0.073
Leg SA/Vol	97	$p = 0.001$	-0.328	0.108	No	0.109
Leg/Trunk SA	97	$p < 0.05$	-0.215	0.046	No	0.058
Birth Weight (g)	87	$p < 0.05$	0.248	0.062	No	0.069
Femur Length (cm)	102	$p < 0.05$	0.203	0.041	No	0.043
Hip Circumference (cm)	104	$p < 0.001$	0.391	0.153	No	0.155
Humerus Length (cm)	102	$p = 0.001$	0.323	0.104	No	0.105
Crural Index	102	$p = 0.001$	0.328	0.108	No	0.108
Statue (cm)	104	$p < 0.001$	0.467	0.218	No	0.222
Tibia Length (cm)	102	$p < 0.001$	0.382	0.146	No	0.146
Trunk SA/Vol	97	$p = 0.001$	-0.346	0.120	No	0.123
Waist Circumference (cm)	104	$p < 0.001$	0.418	0.175	No	0.176
Chest Circumference (cm)	97	$p < 0.001$	0.456	0.208	No	0.208
Trunk Length (cm)	96	$p < 0.001$	0.407	0.166	No	0.225
Underbust Circumference (cm)	97	$p < 0.001$	0.456	0.208	No	0.210
Calf Circumference (cm)	97	$p = 0.001$	0.324	0.105	No	0.112
Forearm Circumference (cm)	97	$p < 0.001$	0.381	0.145	No	0.150
Mid-Thigh Circumference (cm)	97	$p < 0.001$	0.349	0.122	No	0.122
Shoulder Breadth (cm)	97	$p < 0.001$	0.376	0.141	No	0.144
Max Stomach Circumference (cm)	98	$p < 0.001$	0.473	0.224	No	0.225
Bicep Circumference (cm)	97	$p = 0.001$	0.340	0.116	No	0.116
Body Mass (Kg)	104	$p < 0.001$	0.502	0.252	No	0.254

*Remains significant after Bonferroni Correction applied
**Does sport contribute to variance in the dependant variable?

mass, the former scenario seems the most likely explanation. Overall, these findings indicate that measures of central body mass distribution, such as waist and hip circumference are highly correlated with mass distribution in other areas of the body, namely limb segments.

Figure 15 *Waist and Hip Circumference Plotted Against Segment SA:Vol Ratios*



Note. These plots illustrate the strong negative correlation between centralized mass distribution and a decrease in body-wide surface area to volume ratios.

Table 8 *Phenotypic Correlations with Waist Circumference (cm)*

Characteristics	n	Sig.	R	R^2	Sport**	R^2 with Sport
% Fat Mass*	97	$p < 0.001$	0.620	0.384	Yes	0.486
Age at Menarche	104	$p < 0.05$	-0.206	0.042	No	0.047
Arm SA/Vol*	97	$p < 0.001$	-0.786	0.618	Yes	0.640
Bicep Circumference (cm)*	97	$p < 0.001$	0.833	0.694	No	0.706
Bi-iliac Breadth (cm)*	104	$p < 0.001$	0.418	0.175	No	0.176
Body Mass (Kg)*	104	$p < 0.001$	0.840	0.706	Yes	0.718
Calf Circumference (cm)*	97	$p < 0.001$	0.624	0.389	No	0.397
Calf/Thigh SA	97	$p < 0.05$	-0.25	0.063	No	0.066
Calf/Thigh Vol	96	$p < 0.01$	-0.28	0.078	No	0.093
Chest Circumference (cm)*	97	$p < 0.001$	0.867	0.752	No	0.752
Crural Index	102	$p < 0.05$	0.221	0.049	No	0.049
Forearm Circumference (cm)*	97	$p < 0.001$	0.714	0.510	Yes	0.551
Forearm/Upper Arm Vol	96	$p = 0.01$	-0.263	0.069	No	0.092
Hip Circumference (cm)*	104	$p < 0.001$	0.836	0.699	No	0.700
Humerus Length (cm)	102	$p < 0.05$	0.195	0.038	No	0.038
Humerus Length (cm)	102	$p < 0.05$	0.195	0.038	No	0.038
Leg SA/Vol*	97	$p < 0.001$	-0.797	0.635	No	0.636
Max Stomach Circumference (cm)*	98	$p < 0.001$	0.841	0.707	No	0.707
Mid-thigh Circumference	97	$p < 0.001$	0.779	0.607	Yes	0.0625
Shoulder Breadth (cm)	98	$p < 0.001$	0.432	0.187	No	0.192
Stature (cm)	104	$p < 0.05$	0.215	0.046	No	0.046
Tibia Length (cm)	102	$p < 0.05$	0.226	0.051	No	0.051
Trunk Length (cm)*	96	$p < 0.001$	0.545	0.297	No	0.301
Trunk SA/Vol*	97	$p < 0.001$	-0.859	0.738	No	0.738
Underbust Circumference (cm)*	97	$p < 0.001$	0.814	0.663	Yes	0.685
Waist:Hip*	104	$p < 0.001$	0.527	0.278	No	0.279

* Remains significant after Bonferroni Correction is applied
**Does sport contribute to variance in the dependant variable?

Table 9 *Phenotypic Correlations with Hip Circumference (cm)*

Characteristics	n	Sig.	R	R^2	Sport**	R^2 with Sport
% Fat Mass*	97	$p < 0.001$	0.603	0.363	Yes	0.503
Arm SA/Vol*	97	$p < 0.001$	-0.831	0.690	Yes	0.750
Bicep Circumference (cm)*	97	$p < 0.001$	0.858	0.736	Yes	0.758
Bi-iliac Breadth (cm)*	104	$p < 0.001$	0.391	0.152	No	0.155
Body Mass (Kg)*	104	$p < 0.001$	0.919	0.844	Yes	0.874
Calf Circumference (cm)*	97	$p < 0.001$	0.699	0.489	No	0.492
Calf/Thigh SA*	97	$p = 0.001$	-0.34	0.115	No	0.135
Calf/Thigh Vol*	96	$p < 0.001$	-0.358	0.128	No	0.144
Chest Circumference (cm)*	97	$p < 0.001$	0.821	0.674	No	0.676
Crural Index	102	$p < 0.01$	0.275	0.075	Yes	0.116
Femur Length (cm)	102	$p < 0.05$	0.229	0.052	No	0.056
Forearm Circumference (cm)*	97	$p < 0.001$	0.748	0.560	Yes	0.621
Forearm/Upper Arm Vol	96	$p < 0.05$	-0.304	0.092	Yes	0.153
Humerus Length (cm)	102	$p < 0.01$	0.308	0.094	Yes	0.146
Leg SA/Vol*	97	$p < 0.001$	-0.928	0.861	Yes	0.871
Leg/Trunk SA	97	$p < 0.05$	0.208	0.043	No	0.050
Leg/Trunk Vol	97	$p = 0.05$	0.2	0.040	No	0.053
Max Stomach Circumference (cm)*	98	$p < 0.001$	0.857	0.734	No	0.735
Mid-thigh Circumference (cm)*	97	$p < 0.001$	0.879	0.773	Yes	0.810
Shoulder Breadth (cm)*	98	$p < 0.001$	0.444	0.197	No	0.203
Stature (cm)*	104	$p < 0.001$	0.379	0.143	Yes	0.201
Tibia Length (cm)*	102	$p < 0.001$	0.371	0.137	No	0.169
Trunk Length (cm)*	96	$p < 0.001$	0.648	0.420	No	0.421
Trunk SA/Vol*	97	$p < 0.001$	-0.884	0.781	No	0.782
Underbust Circumference (cm)*	97	$p < 0.001$	0.724	0.524	Yes	0.547
Waist Circumference (cm)*	104	$p < 0.001$	0.836	0.698	No	0.700

*Remains significant after Bonferroni Correction Applied
**Does sport contribute to variance in the dependant variable?

4.6.2. Stature

Generally speaking, stature is one of the few variables that can be consistently calculated from skeletal remains – either through anatomical methods or regression equations – with relative accuracy (Raxter et al., 2006). Stature also reflects the investment of energy in linear growth and final height can be interpreted as representing a compromise between competing functions. Therefore, phenotypic traits that were significantly correlated with stature were identified through Pearson regression analyses, these values are summarized in Table 10.

Table 10 *Phenotypic Correlations with Stature (cm)*

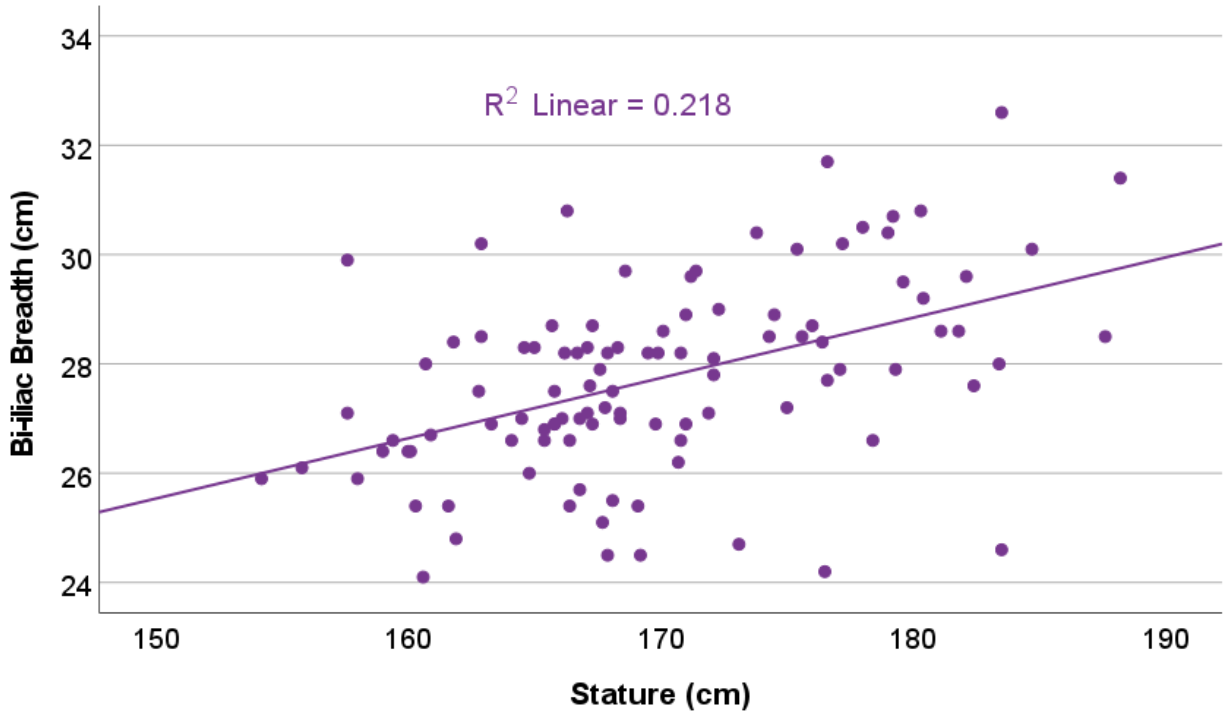
Characteristics	n	Sig.	R	R^2	Sport**	R^2 with Sport
% Dry Lean Mass*	97	$p < 0.001$	0.749	0.561	No	0.569
% Fat Mass	97	$p < 0.01$	-0.297	0.088	Yes	0.171
Age of Menarche	104	$p < 0.01$	0.263	0.069	No	0.070
Arm SA/Vol*	97	$p < 0.001$	-0.384	0.147	Yes	0.191
Bicep Circumference (cm)	97	$p < 0.01$	0.266	0.070	Yes	0.111
Bi-iliac Breadth(cm)*	104	$p < 0.001$	0.467	0.218	No	0.222
Birth Weight (g)	87	$p < 0.01$	0.301	0.090	No	0.970
Body Mass (Kg)*	104	$p < 0.001$	0.571	0.326	No	0.330
Chest Circumference (cm)*	97	$p = 0.001$	0.344	0.118	Yes	0.163
Crural Index*	104	$p < 0.001$	0.478	0.228	No	0.238
Femur Length (cm)*	102	$p < 0.001$	0.716	0.512	No	0.525
Forearm Circumference (cm)*	97	$p < 0.001$	0.438	0.192	No	0.200
Hip Circumference (cm)*	104	$p < 0.001$	0.379	0.143	No	0.147
Humerus Length (cm)*	102	$p < 0.001$	0.798	0.636	No	0.638
Leg SA/Vol*	97	$p = 0.001$	-0.333	0.110	No	0.117
Lower Arm Length (cm)*	103	$p < 0.001$	0.449	0.201	No	0.210
Max Stomach Circumference (cm)*	98	$p = 0.001$	0.344	0.118	Yes	0.181
Mid-Thigh Circumference (cm)*	97	$p < 0.001$	0.358	0.128	No	0.155
Shoulder Breadth (cm)*	98	$p < 0.001$	0.465	0.216	No	0.236
Tibia Length (cm)*	102	$p < 0.001$	0.889	0.790	No	0.791
Trunk Length (cm)*	96		0.626	0.391	Yes	0.448
Trunk SA/Vol*	97	$p < 0.01$	-0.289	0.083	No	0.084
Underbust Circumference (cm)*	97	$p < 0.001$	0.418	0.175	No	0.195
Waist Circumference (cm)	104	$p < 0.05$	0.215	0.046	No	0.046

*Remains significant with Bonferroni correction applied
**Does sport contribute to variance in the dependant variable?

As mentioned above, stature is correlated with both age at menarche and birth weight, with GLM showing that 17.6% of the variation in stature can be explained by those two variables. This study also found that stature was strongly positively correlated with % dry lean mass, with stature explaining 56.1% of the variation (Table 10). Stature had moderate negative correlations with the surface area to volume ratios of the arm, leg, and trunk, which is likely due to increases in segment volume, rather than length. Additionally, it had a moderate positive correlation with bi-iliac breadth, such that stature explained 22% of the variation (Figure 16). Stature and bi-iliac breadth are used together in regression equations to estimate body surface

area (Ruff et al., 2005), therefore, the strong correlations between the surface area to volume ratios, bi-iliac breadth, and stature validate that relationship in this sample.

Figure 16 *Stature Plotted Against Bi-iliac Breadth*



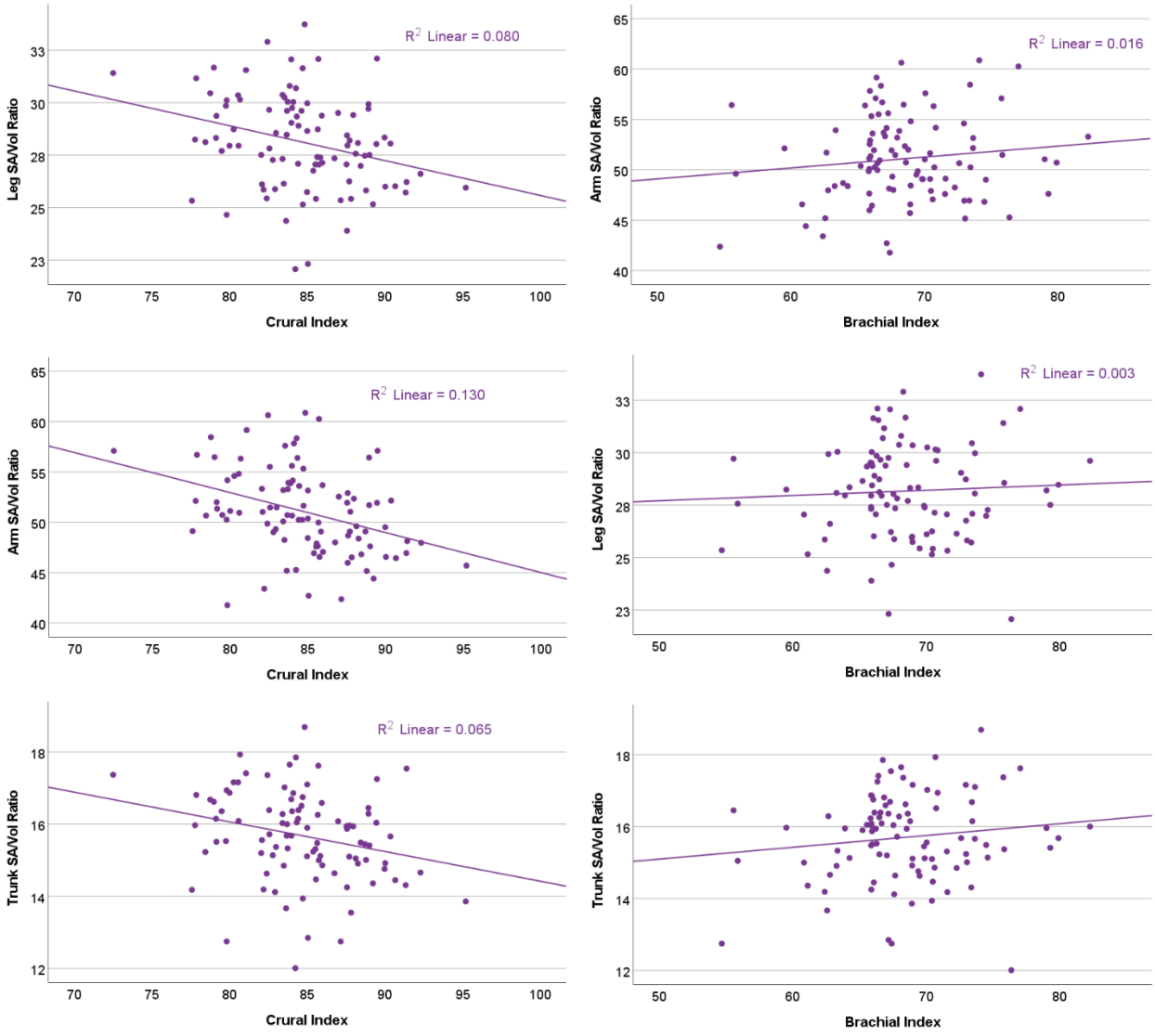
Note. There was a significant, moderate correlation between stature and bi-iliac breadth (n = 104, R = 0.467, p < 0.001).

4.6.3. Limb Segment Correlations

Arm and leg segment lengths are known to correspond to stress experienced during the developmental period, with tibia and radius length showing a reduction under conditions of chronic stress (Pomeroy et al., 2012). However, the existence of a similar relationship has not been explored in limb surface area and volume. In this study, significant fair correlations between the crural index and arm, leg, and trunk surface area to volume ratios were observed (Figure 17).

Increases in the crural index correspond to increases in the length of the lower leg or decreases in the length of the upper leg. In this sample, increases in the crural index were negatively correlated with arm, leg, and trunk surface area to volume ratios (Table 11). In the case of the leg surface area to volume ratio, this correlation suggests that increases in the crural index, likely achieved through increases to the length of the lower leg, rather than decreases in femur length, were associated with increases in the volume of the limb. Increases in limb

Figure 17 *Crural and Brachial Indices Plotted Against Segment SA:Vol Ratios*



Note. Only the crural indices show a relationship to SA:Vol ratios, however, in the opposite direction than what would be expected under Allen's rule (James, 2018).

segment length would cause a small increase in surface area, but a larger increase in segment volume.

Therefore, the negative relationship between crural index and leg, arm, and trunk SA:Vol is likely the result of increases in limb volume achieved through lengthening of the distal limb segment. Interestingly, while there were significant fair relationships observed between brachial index and arm and leg SA:Vol (n = 96, R = -.242, $p < 0.05$ and n = 96, R= -.247, $p < 0.05$, respectively), there was no significant relationship found with trunk SA:Vol (Figure 17).

Table 11 *Phenotypic Correlations with the Crural Index*

Characteristics	N	Sig.	R	R^2	Sport**	R^2 with Sport
Stature (cm)*	102	$p < 0.001$	0.478	0.228	No	0.238
Body Mass (kg)*	102	$p < 0.001$	0.372	0.138	No	0.158
Bi-iliac Breadth (cm)*	102	$p = 0.001$	0.328	0.108	Yes	0.146
Waist Circumference (cm)	102	$p < 0.05$	0.221	0.049	Yes	0.088
Hip Circumference (cm)	102	$p < 0.01$	0.275	0.076	Yes	0.116
% Dry Lean Mass	95	$p < 0.05$	0.236	0.056	No	0.074
Humerus Length (cm)*	102	$p = 0.001$	0.328	0.108	No	0.127
Tibia Length (cm)*	102	$p < 0.001$	0.625	0.391	No	0.399
Lower Arm Length (cm)	101	$p < 0.05$	0.246	0.061	No	0.078
Leg/Trunk Volume (cm^3)	95	$p = 1.00$	0.000	0.000	Yes	0.059
Leg/Trunk Surface Area (cm^2)	95	$p = 0.855$	-0.019	0.000	Yes	0.060
Arm SA/Vol*	95	$p < 0.001$	-0.360	0.130	No	0.150
Leg SA/Vol	95	$p < 0.01$	-0.283	0.080	Yes	0.118
Trunk SA/Vol	95	$p < 0.05$	-0.255	0.065	Yes	0.112
Chest Circumference (cm)*	95	$p = 0.001$	0.350	0.123	Yes	0.159
Trunk Length (cm)*	94	$p < 0.001$	0.382	0.146	Yes	0.193
Underbust Circumference (cm)*	95	$p < 0.001$	0.374	0.140	No	0.158
Forearm Circumference (cm)	95	$p < 0.01$	0.318	0.101	No	0.117
Mid-Thigh Circumference (cm)	95	$p < 0.01$	0.293	0.086	No	0.112
Shoulder Breadth (cm)	96	$p < 0.01$	0.306	0.094	No	0.120
Max Stomach Circumference (cm)	96	$p < 0.05$	0.245	0.060	Yes	0.112
Bicep Circumference (cm)	95	$p < 0.01$	0.312	0.097	No	0.126

*Remains significant with Bonferroni Correction applied
** Does sport contribute to variance in the dependant variable?

4.7. Canonical Correlation Analyses

Previous research has examined the impacts of age at menarche and birth weight on later life phenotype, specifically disease outcomes, however, how both variables contribute to the generation of other forms of phenotypic variation and in what capacity has not been explored. Canonical correlation analyses were performed to determine the relationships between a set of early life history variables, birth weight and age at menarche, and groups of variables that relate to body composition, body morphology, body breadth, thermoregulation, inter- limb and segment relationships, and linear growth (see Table 1). The goal was to determine which variable types are most influenced by the early life environment and which of the early life variables is contributing more to variation in the adult phenotype. Of the six variable sets, only two – linear growth, and body composition – were found to have significant canonical correlations with the early life variable set.

4.7.1. Early Life and Linear Growth Variates

In the canonical correlation performed between the early life history and linear growth sets, one significant function was found (Figure 18). In this function, R_c = .481 with 67.8 % of the variance being shared between the two variates. The Wilk's Lambda statistic was found to be 0.673 and significant at p = 0.005. The other canonical correlation function was not found to be significant. The results of this analysis are summarized in Table 12.

Of the variables which contributed to the linear growth variate, canonical weights indicate that femur length (.903) and trunk length (-.943) were the strongest contributors to the construction of the variate. However, the canonical loading values, which reflect the correlation of each variable with its corresponding variate, reveal that femur length (.673), tibia length (.566), and stature (.545) are more highly correlated with the linear growth variate and contribute

more to the variance used in the construction of the variate compared to trunk length (-.315). The large canonical weight for trunk length compared to the tibia and overall stature may be a result of a lack of collinearity between trunk length and limb lengths. Limb lengths are highly correlated with stature, and the lengths of long bones are commonly used in regression equations to estimate stature from human remains. The cross loadings show that femur length (.324), tibia length (.272), and stature (.262) have moderate correlations with the early life history variate and are likely most influenced by that variate.

Table 12 *CCA for Linear Growth and Early Life Variable Sets*

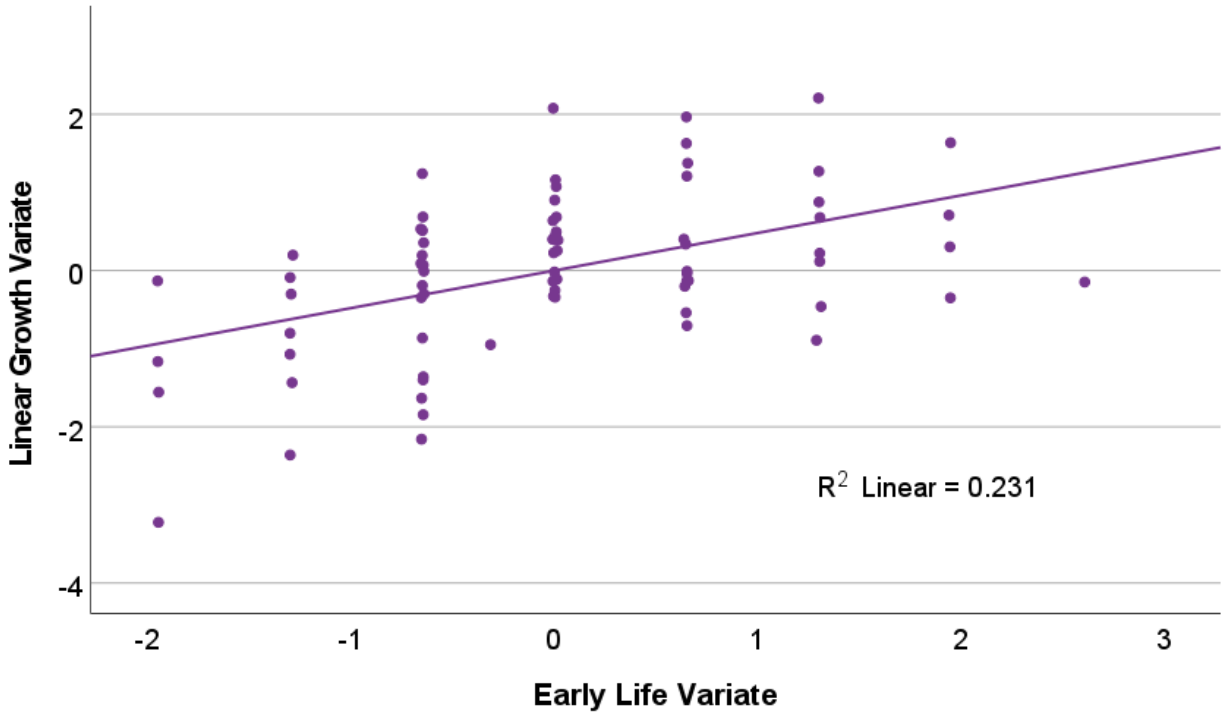
Linear Growth Variable Set				
Variable	Raw Canonical Correlation Coefficient	Standardized Canonical Correlation Coefficient	Canonical Loadings	Cross Loadings
Femur (cm)	.143	.903	.673	.324
Forearm (cm)	.058	-.220	.275	.132
Humerus (cm)	-.131	.099	.468	.225
Stature (cm)	.122	.335	.545	.262
Tibia (cm)	.040	.117	.566	.272
Trunk Length (cm)	-.145	-.943	-.315	-.152

Early Life History Variable Set				
Variable	Raw Canonical Correlation Coefficient	Standardized Canonical Correlation Coefficient	Canonical Loadings	Cross Loadings
Age at Menarche	.650	1.002	1.000	.481
Birth Weight (g)	-.000012	-.006	.271	.130

A comparison of the early life history variable set with the linear growth variable set reveals that compared to birth weight, age at menarche has a larger canonical weight and canonical loading, indicating it has a larger contribution to the construction of the variate and shares more of the variation with the variate than birth weight. Furthermore, a cross loading value of .481 indicates that age at menarche is more strongly correlated with the linear growth variate than birth weight. Overall, these results indicate that between the early life history and

linear growth variable sets, age at menarche, and femur length, tibia length, and stature are most strongly contributing to the canonical function and therefore the relationship between the two variable sets.

Figure 18 *Plot of the CCA Between the Early Life and Linear Growth Variates*



4.7.2. Early life and Body composition Variates

In the canonical correlation performed between the early life history and body composition variable sets, one significant canonical correlation function was found (Figure 19). The results of this analysis are summarized in Table 13.

For this function, R_c = .3741 with 68.3 % of the variance being shared between the two variates. The Wilk's Lambda statistic was found to be 0.803 and significant at $p < 0.05$. The second canonical correlation function was not found to be significant. The standardized canonical correlation coefficient (canonical weight) indicates that of all the variables, % dry lean mass is the strongest contributor with a canonical weight of -.711. The low canonical correlation coefficient for % fat mass is comparatively low; however, this is likely the result of

Table 13 *CCA for Body Composition and Early Life History Variable Sets*

Body Composition Variable Set				
Variable	Raw Canonical Correlation Coefficient	Standardized Canonical Correlation Coefficient	Canonical Loadings	Cross Loadings
Body Mass (kg)	.000365	.004	.304	.113
Waist Circ. (cm)	.054	.335	.583	.216
% Fat Mass	.035	.197	.854	.317
% Dry Lean Mass	-.271	-.711	-.894	-.332

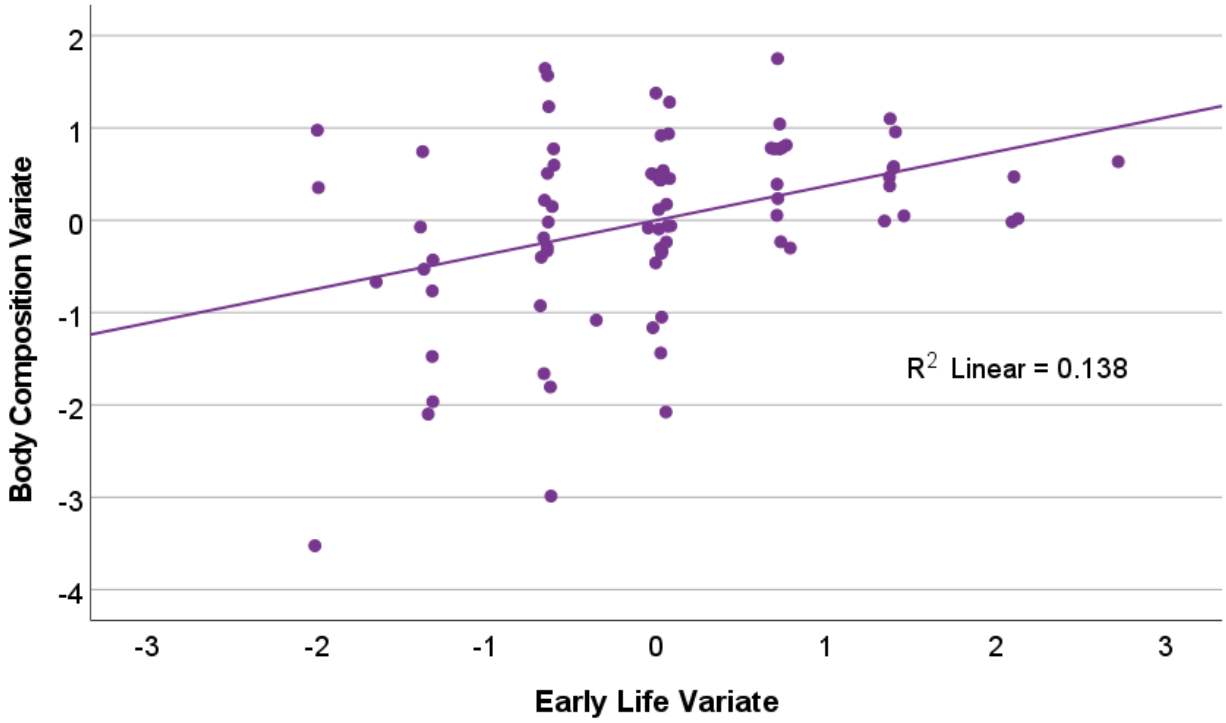
Early Life History Variable Set				
Variable	Raw Canonical Correlation Coefficient	Standardized Canonical Correlation Coefficient	Canonical Loadings	Cross Loadings
Age at Menarche	-.677	-.001	-1.000	-.371
Birth Weight (g)	-.000063	-.031	-.282	-.105

multicollinearity with body mass and waist circumference. This is supported by the high canonical loading values for % fat mass and % dry lean mass (.854 and -.894, respectively), which reflect their high degree of shared variance with the body composition variate and their relative importance to the construction of the variate. Furthermore, cross loading values of .317 and -.332 for % fat mass and % dry lean mass, respectively, indicate a fair correlation of these variables with the life history variate.

In the early life history variate, both age at menarche and birth weight exhibit low canonical weights. However, when contrasted with their canonical loadings (-.282 and -1.000, respectively) it reveals that the low canonical weights are likely the result of collinearity between the two variables. The canonical loading values indicate that of the two variables, age at menarche is more strongly correlated with the life history variate, reflecting a high degree of shared variance and a higher contribution to the construction of the canonical function. This is further supported by a moderate correlation (R = -.317) between the age at menarche variable and the body composition variate. Overall, this indicates that age at menarche has a stronger relationship and influence on body composition variables in adulthood than birth weight in the

sample, reflecting the importance of post-natal environment and energetic availability in this sample.

Figure 19 *Canonical Correlation Between the Early Life and Body Composition Variates*



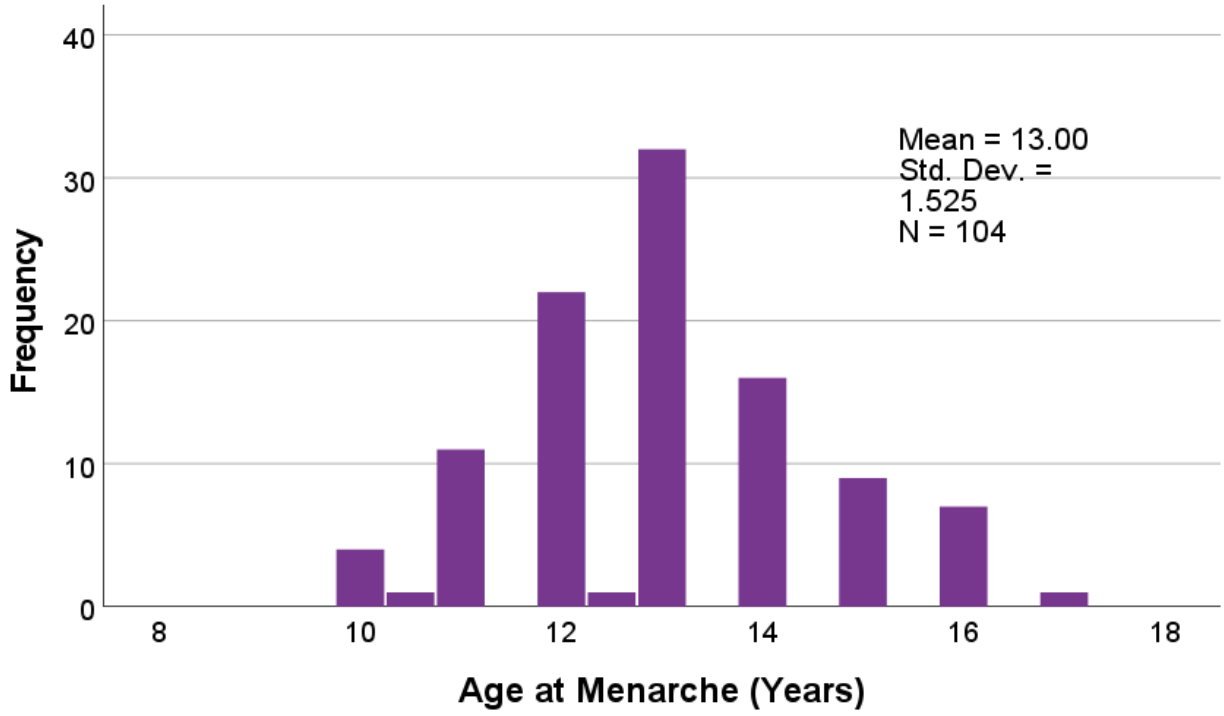
5. Discussion

5.1. Sample Composition

The focus of this study was on the relationship between early life environment and outcomes for adult life history and phenotypic variability. The data, however, were drawn from both athletes and non-athlete groups, making it important to determine whether athletic participation contributed to variations in birth weight or age at menarche. Independent sample t-tests indicated that there was no difference between athlete and non-athlete sub-samples with regard to birth weight and age at menarche; this allowed for these sub-samples to be pooled for further analyses (See Appendix). Previous research on the variability of age at menarche between athlete and non-athlete populations has shown mixed results. Gymnasts have been shown to be shorter and have a later age at menarche, presumably as a result of energetic constriction during the developmental period either as a result of training intensity prior to menarche or as a means to meet aesthetic requirements in leanness- or weight-dependent sports (Baxter-Jones, 2002; Frisch, 1981, 1987). However, other studies have suggested that being short for age compared to one's peers does not persist into adulthood and that more research needs to be done to assess the role of training intensity and catch-up growth in modulating the female phenotype (Erlandson et al., 2008; Klentrou, 2006). The mean ($n = 104$, $\mu = 13$, $SD = 1.525$), and range of variation (10-17 years), in age at menarche reported for this study is situated within the normal range of variation reported for longitudinal studies of female pubertal development and does not reflect a shift to either earlier or later menses (Figure 20) (Biro et al., 2018). The athletes sampled in this study were drawn from soccer, running, and rowing teams. These sports are energetically demanding; however, there is not the same degree of pressure to maintain certain body type aesthetics as there is in sports like gymnastics or dance. Furthermore, few of the athletes in this

study reported starting intensive training before starting menstruation, with athletes reporting starting their training, on average, 2.6 years after their first menses.

Figure 20 *Distribution of Age at Menarche*



5.2. Validating Relationships with Birth Weight and Age at Menarche

One of the first goals of this study was to test relationships with birth weight, age at menarche, and a broad range of phenotypic outcome variables, with a specific interest in previously un-examined traits and trait combinations. Variations in birth weight and age at menarche have independently been linked to variation in linear growth and body composition measures (Gale et al., 2001; Remsberg et al., 2005; te Velde et al., 2003). Furthermore, birth weight and age at menarche are known to be co-linear, with low birth weight predicting an earlier age at menarche (Sloboda et al., 2007; Tam et al., 2006). As expected, birth weight was found to be positively correlated with age at menarche. Building on this, another aim of this

study was to identify the relative importance of both traits to the development of different aspects of the adult phenotype.

5.2.1. Birth Weight and Influence on Body Composition

Previous studies have pointed to a negative correlation between birth weight and fat mass, such that lower birth weight individuals are more likely to have a higher proportion of fat than their normal or large at birth peers (te Velde et al., 2003). Lean mass has also been shown to have a positive correlation with birth weight (Gale et al., 2001; Loos et al., 2002; Rogers, 2003; Ruff, 1994). Overall, this is interpreted as evidence that with decreasing birth weight there is a tendency to store energy in the form of % fat mass, whereas with increasing weight, the tendency is to accumulate % lean mass (Rogers, 2003).

While a significant association between birth weight and adult body mass (kg) was observed, this study found no significant correlation between birth weight and % fat mass or % dry lean mass. The lack of a clear relationship between birth weight and tissue investment strategy in adulthood in this study (when it has been previously identified elsewhere) may be the result of a lack of diversity in the sample populations in terms of weight at birth. Currently, the threshold for considering an infant to have low birth weight is contested and population dependent. In a clinical setting in the UK, 2500 g and 1500 g are the current cut-offs for labeling an infant low birth weight and extremely low birth weight, respectively (Norris et al., 2018; Wells, 2007). However, these standards, developed in the late ’90s, have been criticized for not being based on birth weight FGA and the under-representation at the lower end of the distribution (based on inclusion criteria for stillbirths) (Norris et al., 2018). Recently developed birth curves for the UK that are based on weight FGA identify the threshold for the 2nd percentile at ~2750 g at 40 weeks gestation (Norris et al., 2018). In the present study, seven individuals fall

under the low-birth-weight threshold of 2750 g proposed by Norris and Colleagues, two individuals fall under the clinical cut-off of 2500 g, and none fall under 1500 g. Additionally, 17 of the individuals who participated in this study did not report their weight at birth. Furthermore, gestational age was not reported by the participants in this study, further complicating the ability to assess the relationship between birth weight and phenotypic outcomes. It is possible that none of the individuals assessed in this study were underweight for their gestational age at birth. Other studies have focused on the relationship between birth weight and lean mass specifically in adulthood and have reported a significant positive relationship (Gale et al., 2001; Loos et al., 2002). Therefore, the lack of a strong relationship between birth weight and fat mass is not surprising. The lack of a clear association between birth weight and body composition in this study suggests that the effects of energy constrictions *in utero* (reflected in low birth weight) may not manifest continuously, but rather develop as a result of failing to meet some critical threshold of weight FGA.

5.2.2. Age at Menarche's Relationship to Body Composition

There is debate over the role of body composition (specifically % fat mass) when it comes to the initiation of puberty in women. Studies have identified that individuals who have an earlier age at menarche, tend to have more body fat than their peers with a later age at menarche (Tam et al., 2006). It is contested whether the initiation of puberty and menses are triggered when a critical fat mass is reached or if the timing of these events is shaped by earlier life environment and birth weight (Biro et al., 2018; Frisch, 1987, 1990; Rogers, 2003; Vizmanos & Martí-Henneberg, 2000). The distribution of somatic tissues, like fat, as they relate to age at menarche was of interest here as gluteal-femoral fat depots are important energy sources for fueling pregnancy and lactation (Rebuffé-Scrive et al., 1985; Reiches et al., 2013). Furthermore,

it has been observed that there is a difference in tissue metabolism strategy between young and older adolescent females under energetic stress. In a study by Reiches and colleagues in 2013, it was discovered that young, post-menarcheal adolescent females tended to lose fat mass (reproductive tissue) under energetic stress, while older adolescent females tended to lose lean mass and were instead protective of fat mass. This illustrates a maturity-dependent difference in tissue investment strategy, however, how this strategy differs based on variation in early life energetic conditions is less well understood. It was hypothesized that the relationship between age at menarche and tissue distribution may reflect a tissue investment strategy that favors investment in gluteal-femoral depots (reproductive tissue) when age at menarche is earlier.

In this study, age at menarche was not found to account for any significant variation in measures of tissue distribution in the gluteal-femoral region, specifically, it was not associated with hip circumference. There was also no association with Calf:Thigh volume or surface area, which also captures tissue distribution in the gluteal-femoral region. There was a weak, negative correlation between age at menarche and % fat mass, and a positive correlation between age at menarche and % lean mass, indicating that individuals with an earlier age at menarche were found to have more body fat in adulthood while individuals with a later age at menarche had more lean mass. Furthermore, age at menarche was found to be negatively correlated with waist circumference. Waist circumference was found to be correlated with % fat mass. Abdominal fat is a known risk factor for the development of cardiovascular and metabolic disease in later life (D. J. P. Barker et al., 1993; Bubach et al., 2021; Fall et al., 1995; Lakshman et al., 2009; Remsberg et al., 2005).

While there is evidence here of a relationship between body composition and age at menarche, such that a younger age at menarche was associated with higher % fat mass and lower

% lean mass, the differential investment in the location of reproductively integral tissues based on menarcheal age was not observed. The accumulation of fat in the gluteal-femoral region during pregnancy and the mobilization of said fat during lactation may be a strategy of tissue investment and energy mobilization that is only implemented following conception (Rebuffé-Scrive et al., 1985).

Some considerations are that these findings may be a result of the sample structure which included more athletes than non-athletes. A GLM showed that participation in a sport significantly contributed to variation in % fat and % dry lean mass. Additionally, this study does not capture body composition and tissue distribution before or during puberty, and therefore, cannot speak to the differences in tissue metabolism under energetic stress outlined by Reiches and colleagues and how that may fit into the life history context discussed here (2013). Overall, the trends in body composition and tissue distribution in this study reflect that body mass increases with decreasing birth weight and age at menarche, with fat being accumulated preferentially over lean mass under those conditions.

5.2.3. Birth Weight and Implications for Skeletal Growth

Birth weight was found to be correlated with some measures of linear growth, (stature and trunk, forearm, humerus, and tibia lengths) and measures of body breadth (shoulder and bi-iliac breadths). It has been shown that individuals who are small at birth and continue to experience energy constriction in childhood are more likely to experience reductions in stature compared to their peers (Koziel & Jankowska, 2002; Stock & Migliano, 2009). Furthermore, birth weight has been shown to have a positive relationship with bone quality (Gale et al., 2001).

Age at menarche is also known to influence stature through the timing of hormonal cascades involved in puberty that affect the growth plates of long bones (Cutler, 1997;

Dunsworth, 2020). As previously mentioned, low birth weight individuals may be under-represented in this sample, therefore, the question becomes: are correlations between measures of linear growth and birth weight the result of maternal environmental interactions or rather the result of collinearity with age at menarche? Canonical correlation analyses comparing birth weight and age at menarche with the measures of linear growth showed that of the two variables, age at menarche contributed more to the construction of the early life variate and also to the correlation with the linear growth variate when compared to birth weight. This indicates that while birth weight does have some influence on linear growth, the statistical relationship between birth weight and linear growth is likely being influenced by collinearity with age at menarche, and that age at menarche has a stronger influence on growth outcomes. This points to the importance of the post-uterine environment in determining the expression of plastic adult phenotypic traits such as stature and limb lengths.

Unlike the measures of linear growth, the body breadth measures (bi-iliac and shoulder breadths) reported in this study were significantly correlated with birth weight but not age at menarche, and in the canonical correlation analysis that combined birth weight and age at menarche into an early life variate, there was no correlation found with the body breadth variate. This is interpreted as evidence that birth weight, independent of age at menarche, has a weak but significant influence on measures of body breadth. This is an interesting discovery as bi-iliac breadth is often used in regression equations to estimate body mass (Ruff et al., 2005), and an association between birth weight and body breadth has not been identified in the literature. This correlation may provide an avenue for using the stature and body breadths of skeletal populations to develop estimates of body size (weight, stature, etc.) in early life, which would inform other understandings of human life history trajectories.

5.2.4. Age at Menarche and Implications for Skeletal Growth

The relationships between age at menarche and linear growth are stronger than those associated with birth weight. It is well established in studies of growth velocity and pubertal timing that the hormonal cascades associated with puberty, particularly the increase in estradiol concentration that fuels the development of secondary sexual characteristics in females, are closely tied to the deceleration and eventual cessation of bone deposition activity at the growth plates (Cutler, 1997; Dunsworth, 2020; Kang et al., 2019; McIntyre, 2011). Therefore, it was predicted that age at menarche would be positively correlated with stature and long bone and limb segment lengths. This prediction was supported by the results of both univariate correlation analyses of age at menarche with stature and other linear measures and in canonical correlation analysis comparing the early life variate against a composite linear growth variate. While both birth weight and age at menarche were found to be independently correlated with stature and tibia length, age at menarche was also correlated with both femur and humerus length while birth weight was not. Furthermore, the CCA between early life and linear growth variates demonstrated that in that comparison, variation in the age at menarche was contributing more to the formation of the early life variate and the cross-loading with the linear growth variate. This demonstrates that of the two variables, age at menarche has a stronger connection to the variation in linear growth in this sample.

Estrogen has a biphasic effect on bone growth, with lower doses mediating bone maturation while higher doses promote the cessation of growth (Cutler, 1997; Iravani et al., 2017). Timing of puberty is known to also affect bone strength, alongside length, with earlier age at menarche being correlated with a decrease in bone strength (Cutler, 1997; Dunsworth, 2020). While not captured in this study, an interesting line of questioning for future research would be

to examine the link between bone functional adaptation and age at menarche, to determine if those with a later age at menarche (and a longer growth window) show more variation in bone morphology, than those with an earlier age at menarche as this may have implications for interpretation of activity in the archaeological record.

Interestingly, trunk length (groin to the base of the neck), which is somewhat analogous to sitting height, was not found to be correlated with age at menarche. The predominant skeletal component contributing to trunk length would be the spine, with vertebral bodies, like long bones, being a result of endochondral bone formation (Karaplis, 2008). The effects of estrogen on the cessation of growth are often discussed in terms of long bone formation, but not in the formation of other endochondrally formed bones like the vertebra. Other studies of plasticity in the growth of body segments have shown that when compared to variation in leg length, that sitting height is more highly conserved under energetic pressures, especially in women (Ríos et al., 2020). Therefore, the lack of variation in trunk length, when age at menarche is considered, is not surprising; however, the mechanism behind the difference in the effect of estrogen concentration and pubertal timing on endochondrally formed non-long bones is not well understood and presents an avenue for further research.

In their 2011 meta-analysis of stature and age at menarche, Mcintyre and Kacerosky identified that in industrialized societies, stature showed a positive correlation with age at menarche, whereas they observed a negative relationship in small-scale and agrarian societies. They proposed that this negative relationship is reflective of both poor uterine and childhood environmental conditions. As the energetic burden increases, the portion of the energy budget that can be allotted to reproduction is further constrained, leading to shorter individuals with a later age at menarche (Mcintyre & Kacerosky, 2011). The positive relationship observed in the

industrialized populations was likely the result of a higher quality energetic environment during childhood, which was attributed to increased resource availability. Simply put, the energy was available to invest in both growth and reproduction, and under conditions of energy constriction, investment is made in growth and future reproductive potential over an accelerated reproductive schedule. The relationship between age at menarche and linear growth observed in the present study is positive, a finding which agrees with the linear relationship of the industrialized populations presented by Mcintyre and Kacerosky, further supporting their conclusions.

5.3. Trends in Phenotypic Outcomes

5.3.1. The Differential Roles of Fat and Lean Mass in Morphological Variation

As was described above, it seems variation in the post-uterine environment, measured through variation in age at menarche, has stronger implications for adulthood variation in body composition, specifically % fat and % dry lean mass, at least under the condition of reduced variation in birth weight. It was found that the % fat mass was negatively associated with age at menarche, such that individuals with a lower age at menarche tended to have a higher % fat mass compared to lean mass. Percent fat mass was found to be positively correlated with chest, underbust, max-stomach, waist, hip, mid-thigh, calf, bicep, and forearm circumferences, while lean mass was not found to be correlated with any measures of body circumference. Furthermore, % fat mass was shown to have a negative correlation with trunk, leg, and arm SA:Vol relationships, likely driven by the positive relationship to segment circumference, and consequently, volume. In addition to the negative correlation to segment SA:Vol ratios, negative correlations between % fat mass and intra-segment SA and Vol ratios were observed, such that increases to % fat mass were associated with a decrease in Forearm:Upper Arm or Calf:Thigh SA and Vol. This negative relationship is likely a result of increases in the distal segment

circumferences in the absence of corresponding increases to distal segment lengths as no significant relationship between % fat mass and linear growth was observed in this sample.

A positive relationship was observed between % dry lean mass and intra-segment SA and Vol ratios such that increases in lean mass saw corresponding increases in Forearm:Upper Arm or Calf:Thigh SA and Vol. This relationship is likely a result of co-linearity of age at menarche, % dry lean mass, and linear growth. As age at menarche increases, there are corresponding increases in % dry lean mass and limb segment lengths. Increases in limb length, primarily in the distal limb segment, are likely what is driving the increase in intra-segment ratios of SA and Vol.

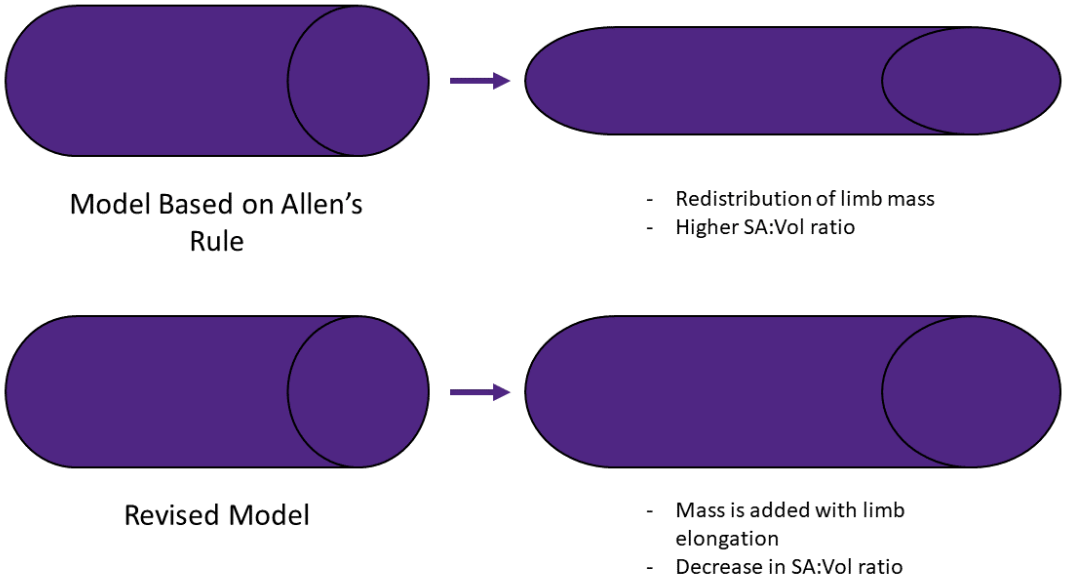
Percent dry lean mass was significantly correlated with femur, tibia, and humerus lengths, as well as crural indices; however, it was not found to be correlated with forearm length or brachial indices. This finding is unexpected given the relationship with other aspects of linear growth and may be the result of one of two things. First, this result may indicate that tibia length is more sensitive to energy availability than forearm length; however, this explanation is weakened by the existing positive correlations with both femur and humerus lengths, and it has previously been noted that zeugopod length is likely more plastic than stylopod lengths (Pomeroy et al., 2012). The second, and potentially more likely explanation, is that these results stem from differences in the protocol. When the sample used in this study was collected in 2016/2017, femur, tibia, and humerus lengths were measured using standard anthropometric measures; however, forearm length (measured as the distance between the Radiale® and stylion landmarks) was not collected (Norton, 2018). For the purposes of the present study, forearm length was recorded as the 3D distance between the Size Stream autogenerated elbow and wrist landmarks. This difference in methods introduces a degree of error in the measurement,

potentially explaining the discrepancy in the results between the arm and leg measurements and ratios.

5.3.2. Implications for Crural Index and Surface Area to Volume Ratio

Building on the above discussion of the limb proportion variation, one interesting finding was that the crural index had moderate, negative correlations with leg, arm, and trunk SA:Vol ratios, indicating that as the crural index increased, SA:Vol ratio in the leg decreased, (likely mediated through increases in tibial length). As previously mentioned, the crural index was found to be correlated with % dry lean mass and not % fat mass. Furthermore, % dry lean mass was not correlated with changes in limb circumference (positively or negatively). Applying a cylinder model to the limb, increases in limb length have a larger impact on segment volume when compared to the surface area when breadth is kept the same (Kasabova & Holliday, 2015). Therefore, based on the known plasticity of tibia length under energetic stress (Pomeroy et al., 2012), increases in distal limb length are likely driving the negative relationship between the crural index and limb SA:Vol. This observation contradicts commonly held assumptions connecting limb length and SA:Vol relationships purposed by Allen's rule, which suggests that as limbs become elongated, SA:Vol ratio increases (James, 2018). The implication is that elongated limbs should be observed in hot, arid environments, whereas shorter limbs should be observed in colder environments to promote thermoregulatory homeostasis (James, 2018). However, this ignores the fact that as you increase the length of a limb, mass is added in the form of soft tissue and bone, not simply redistributed throughout the limb segment (Figure 21).

Figure 21 *Comparison of SA:Vol Ratios Under Different Models of Limb Elongation*



Note. Under Allen's model, limb elongation leads to an increase in SA:Vol to promote heat dissipation; for this model to work, mass must be redistributed. Under the revised model for limb elongation proposed here, mass (in the form of boney and soft tissues) is added with the increases in length, leading to a decrease in the SA:Vol ratio. This model explains the negative correlation between crural index and leg SA:Vol observed in the UK female sample.

Historically, the estimation methods for human body surface area and volume, primarily for use in the discussion of thermoregulation, have treated the whole body as a cylinder (Ruff, 1994). Comparisons of Ruff's cylinder model to the "Tin Man" model (Kasabova & Holliday, 2015), which treats each body segment as an independent cylinder, have revealed that the latter method produces more accurate estimations of body surface area and volume. Additionally, a comparison of the use of cylinder and segmented body models for heat balance estimation during locomotion found significantly different results. Estimates for heat balance that used the segmented body model were significantly lower than those that used the cylinder model, showing that intersegmental differences have important implications for heat balance estimation (Cross et al., 2008).

Both Ruff (1994), and Kasabova and Holiday have shown that body breadth has a greater impact on overall body surface area to volume ratios when compared to limb lengths and crural indices (2015). Therefore, the lack of positive correlations between crural indices and segment SA:Vol ratios in this study is not unprecedented. The lack of correlation here between crural index and Arm:Trunk and Leg:Trunk SA and Vol ratios is supported by the conclusions of Kasabova and Holiday that variation in limb proportions is not necessarily tied to eco-geographical patterns (2015). The fact that body breadth was found to be associated with birth weight in this study further supports this conclusion.

Based on the results of this study, it can be suggested that limb proportion variation is not being driven by thermoregulatory adaption but is rather the result of either generalized stress affecting skeletal growth or is reflective of neutral processes. These findings call for further examination of the generalizability and applicability of Allen's rule to studies of morphological variation in humans. It also illustrates the need for more comparative studies incorporating the estimation technique developed by Cross and colleagues to determine if differences in limb proportions significantly affect heat balance during locomotion (2008).

5.3.3. Implications for Life History Reconstructions

This study found significant correlations between the early life variables birth weight and age at menarche, and multiple measures of body composition and linear growth. Canonical correlation analyses indicated that of the two early life variables, variation in age at menarche was contributing more to variation in body composition and linear growth than birth weight, but that birth weight was independently correlated with measures of body breadth (bi-iliac and shoulder breadths). Notably, these findings have important implications for the construction of life history estimation techniques based on skeletal metrics. Currently, population-based estimate

techniques for measures of size at birth are based on individuals that died at birth, and age at menarche is estimated based on the presence/absence of the fusion of the hook of hamate in females who died during adolescence (DeWitte & Lewis, 2020). A limitation of these methods, and any studies that investigate questions relating to life history schedule or resilience, is that they are based on the skeletal remains of people who did not, or just barely, survived those life history milestones, a component of the osteological paradox (Wood et al., 1992). However, the results of the present study suggest that it may be possible to estimate age at menarche and birth weight, based on variation in skeletal anatomy observed in adulthood. This would allow for population estimates of these characteristics to be based on those who survived the developmental period, rather than those who did not. The development of regression equations based on modern living humans using multiple skeletal elements to estimate aspects of life history and the soft tissue variation would aid in creating more accurate reconstructions of life in the past, namely, the social, behavioural, and environmental pressures that would have influenced the energetic trade-offs that shape the human life history trajectory. While sample size and breadth of the current study limit these possibilities, they present an avenue for further research.

5.3.4. The Female Phenotype: Energetic Investment Strategies

It has been suggested by Mcintyre and Kacerosky (2011) that some aspects of the human female phenotype as they relate to life history strategy may manifest as norms of reaction. They argue that this can be seen in the presentation of stature and menarche in industrial and agrarian populations as discussed previously. However, the variation in the multiple life history and morphological traits reported in this study may hint at the existence of more complex phenotypic interactions that lie on a continuum.

Canonical correlation analyses showed that of the two early life variables examined in this study, that age at menarche was contributing more to adult phenotypic variation than birth weight. Aside from bi-iliac and shoulder breadth, the correlations with birth weight were likely being driven by collinearity with age at menarche. However, this finding does not ignore the weak, but significant correlation between birth weight and age at menarche. This may point to energetic variability *in utero* shaping age at menarche, which in turn is connected to variation in other phenotypic traits.

Overall trends in the data suggest that there are associations between later age at menarche and "healthier" growth and body composition outcomes compared to individuals who had an earlier age at menarche. Cumulatively, age at menarche showed positive correlations with linear growth and % lean mass, and negative correlations with % fat mass. In turn, % lean mass was positively associated with crural indices, bi-iliac breadth, Waist:Hip ratio, and Zeugopod:Stylopod surface areas and volumes while % fat mass was associated with body-wide increases in volume, namely waist circumference, and a decrease in stature. Taken collectively, these results suggest that there is variation in tissue investment strategy relating to energetic trade-offs between reproduction, investment, maintenance, and defence. At one end of the spectrum, under the conditions of energetic favourability during the developmental period, energetic investment is focused on growth and maintenance and increasing later reproductive potential. This is reflected in a later age at menarche, a longer growth period, and increases in lean mass accumulation. Conversely, unfavourable energetic conditions during the developmental window may lead to a 'faster' life history schedule, with an earlier age at menarche, a shorter growth period, and accumulation of fat mass as an energy reserve over lean mass. Fat mass is more energetically available under prolonged stress and fuels both immune

response and reproduction-related activities like pregnancy and lactation (Rebuffé-Scrive et al., 1985; Urlacher et al., 2018). While this investigation did not identify a link between investment in gluteal-femoral fat depots and an earlier age at menarche, a future avenue of research would be to determine if age at menarche influences the speed of accumulation, distribution, and relative amassment of gluteal-femoral fat depots in pregnant women.

Another potential implication of these findings is that for the effects of energy availability *in utero* to influence aspects of the adult phenotype, birth weight needs to fall below a critical threshold dependent on gestational age. Should birth weight exceed this threshold, it is seemingly the energetic conditions experienced during infancy, childhood, and adolescence that have a stronger influence on other phenotypic outcomes. While this study fails to capture the energy environment mid-childhood, it captures the collective energetic landscape with age at menarche.

While not perfect, interpretation of these relationships in a life history context can be taken as evidence for the existence of different female phenotypes relating to investments in reproductive potential and timing. This interpretation, however, is geared toward an adaptionist view of female life history and physical phenotype (Gould & Lewontin, 1979). An alternative explanation is that the observations reported here are simply the cumulative results of sequential energetic trade-offs experienced over the life course and that the individuals making up the sample experienced similar environmental conditions during their developmental periods.

6. Conclusions: Contributions, Limitations, and Remaining Questions

This study aimed to clarify the role of early life environment in shaping the timing and tempo of the human life history strategy as it is expressed in females and how this contributes to variation in the presentation of the physical phenotype in later life. This was achieved through the investigation of a sample of females from Cambridge, UK. Through the analysis of a combination of novel 3D and traditional anthropometric measures, and a survey that queried birth weight and age at menarche, several questions relating to the manifestation of variation in adult phenotypic outcomes were addressed.

6.1. Findings and Contribution to Research Paradigm

The results of this study suggest the existence of a broad relationship between a slower life history tempo and more favourable phenotypic outcomes that, consequently, reduce risk factors for non-communicable diseases. Birth weight was weakly and positively correlated with some measures of linear growth and body breadth; however, age at menarche showed stronger positive correlations with linear growth and body composition variables in both univariate and canonical correlation analyses. This is taken as evidence of a stronger influence of the post uterine environment in shaping the life history strategy and schedule of human females, at least in this population where none of the participants fell under clinical thresholds for low birth weight. Moreover, the fact that birth weight did not have a strong correlation with body composition or other measures of tissue investment strategy, as noted in other studies, suggests the existence of a minimum birth weight threshold that must be breached in order to affect the adult phenotype.

Furthermore, segment surface area to volume ratios were not found to correlate positively with limb proportion ratios, namely curial indices. This unexpected, but interesting, finding

supports the notion that limb proportions do not necessarily relate to thermoregulatory adaptations, but rather are the result of energetic trade-offs during development, indicating the need for further research in this realm (Kasabova & Holliday, 2015).

6.1.1. Applications to Bioarchaeology

One of the major goals of bioarchaeologists is to produce reconstructions of life in the past that are as accurate as possible, despite the inherent limitations of working with skeletal remains. This field of study has historically been limited to examining aspects of health, such as the presence of identifiable pathologies, trauma history, and evidence of stress; and some perceived aspects of identity, such as social status, gender, and occupation. Assessing the life history of an individual, specifically their individual growth and developmental patterns, the timing of biological milestones, and how social and biological variation in early life contribute to skeletal and soft tissue variation, has been harder to evaluate. Interpretations are limited given that few aspects of life history are visible from skeletal remains and that children are under-represented in skeletal populations for multiple reasons, including differential mortuary treatment and the poor preservation of small and fragile bones in the archaeological record. Therefore, to understand the variation in life history trajectories of individuals and populations in the past, it is necessary to use modern human populations as a model system (Longman et al., 2020).

This study found that there were significant correlations between birth weight and measures of body breadth and stature. These characteristics can be estimated from skeletal remains and may be used to create birth weight estimation techniques. This would contribute to both our understanding of the life history of that individual and indicate, to some degree, the health and nutritional status of the mother during pregnancy. This information can be used to

inform the reconstruction of both biological and social realms surrounding the lived experiences of women and children in the archaeological record.

Age at menarche was correlated with measures of linear skeletal growth and body composition. Similar to birth weight, variation in skeletal growth has the potential to be used to estimate both variation in age at menarche, as well as patterns of tissue composition and distribution. As it stands, there are methods to estimate body mass using skeletal elements, but the variable contributions of lean mass and fat mass to body mass are not commonly included in such estimation techniques. As shown here, variation in age at menarche is linked to variation in both lean and fat masses, which, in turn, can be linked to disease risk in adulthood.

The negative correlations observed between the crural index and SA:Vol ratios of the limb and trunk segments indicate that the assumed relationship between limb proportions and thermoregulation summarized in Allen's rule needs to be revisited and interpreted with energetics and life history in mind. This may lead to new interpretations of variation in skeletal populations. Recent work by Longman and colleagues (2021), supports re-interpretation of eco-geographical patterning of limbs, showing that individuals with hot-adaptive phenotypes perform better in ultramarathon races under thermal stress (Longman et al., 2021). Furthermore, they report this pattern is more evident in females. These findings taken together exemplify the need for further study of the role of life history variability and sex-based differences in energetic allocation strategies in determining phenotypic outcomes and morphological adaptation.

While the current sample was limited in diversity and size, the observed relationships between phenotypic traits suggest the potential of developing techniques for the estimation of life history variables in bioarchaeological populations. Specifically, the correlations between phenotypic variation in skeletal elements (limb lengths and body breadths) and variation in age at

menarche and birth weight, respectively, indicate there is the potential to develop regression equations that estimate these life history variables, as well as aspects of body composition, based on skeletal remains. Acknowledging that such estimation techniques would not be precise, there is the opportunity to determine the rough manifestation of the life history schedule and strategy of specific human populations. This would aid in the reconstruction of both health and life in the past as it reflects the environmental and energetic conditions experienced during early life. Understanding sources of variation in human life history and how they manifest in skeletal remains (specifically as they relate to the female phenotype) is important. Variation in a population's life history schedule as a result of the local environment or cultural conditions has the potential to impact population demography, health, and social constructs relating to identity. For example, having an earlier age at menarche may inform the construction of an individual's social identity as it may relate to the transition to adulthood. Additionally, earlier age at menarche would allow for reproduction to start earlier, potentially affecting population demographics. Being able to identify, even broadly, variation in life history outcomes from skeletal remains would provide an additional line of evidence for the bio-archaeological reconstruction of life in the past.

6.2. Limitations and Recommendations

Chief among the limitations to this study were the restrictions placed on research activities due to the COVID-19 pandemic. This prohibited the expansion of the sample and the type of information collected for this study and thereby narrowed the degree of variation that the sample could capture. Restriction of the sample likely influenced statistical analyses and subsequent interpretations. Specifically, the small sample size may be causing a restriction of the

true population range, leading to a low correlation in some of the variable relationships examined (Bland & Altman, 2011).

6.2.1. Capturing Early Life

A shortcoming of this research is that the variables collected fail to capture the effects of environmental variation at the midpoint between birth and adolescence, and instead, only capture the cumulative result of that variation in age at menarche. Numerous studies have pointed to the fact that while birth weight FGA has some influence on the phenotype, the timing and pattern of post-natal growth experienced onward to mid-childhood (about age seven), is perhaps more influential on the adult phenotype (Sloboda et al., 2007; Tam et al., 2006; Terry et al., 2009). The inclusion of stature and body composition at mid-childhood alongside birth weight relative to gestational age and age at menarche may provide a more holistic view of the connection between life history and variation in later life phenotypic outcomes. Therefore, future longitudinal or retrospective research should incorporate these variables.

Another limitation of this study is the under-representation of the lower end of the birth weight spectrum in this sample, potentially reflecting low variability between mothers in the uterine environment. This is likely influenced by the existence of universal health care access in the UK. The National Health Service, NHS as it is commonly known, is available to all residents of the UK and includes free access to pre-natal care that encompasses access to midwives, doctors, dietitians, pediatricians, and a health visitor (a trained nurse) that is available until your child's fifth birthday (*Antenatal Support: Meet the Team*, 2020).

6.2.2. The Size Stream 3D Body Surface Scanner in Anthropometric Research

This study aimed, in part, to extract novel data from previously collected datasets to allow for phenotypic analyses not possible through conventional anthropometric approaches. The

volumetric and surface area data collected from 3D meshes allowed for direct comparison between variation in segmental surface area and volume to other phenotypic traits that have not previously been reported. Through this process, proof of principle and limitations for the use of the Size Stream 3D Body Scanner in anthropological studies of human variation were provided. One of the major limitations identified was the comparability of the measures autogenerated using the Size Stream to those taken using standard anthropometry methods, specifically, differences in stature due to foot placement and the inclusion of hair for the Size Stream. This informed interpretations of the data and limited comparability to other studies where standard measurement procedures are used. Despite this limitation, valuable information was extracted in terms of body volume and surface area, showing the potential of such methods. Furthermore, the majority of key analyses did not include data that were biased by the Size Stream auto-measurement protocol, and a significant amount of time was spent removing potential biases from the surface scans themselves. Another finding of this process was the importance of hand orientation (anatomical position) and clothing type (skin-tight/spandex) in the post-scanning digital manipulation of the meshes. These discoveries led to the development of a document outlining "best practices" for scanning and segmentation that will inform future studies that utilise 3D whole-body scanning technologies. Future directions include more systematic evaluation and validation of the accuracy of Version 12 of the Size Stream scanner and determining appropriate correction factors. This has been done for Version 14 of the Size Stream, however, the generalizability of this study has yet to be assessed as Version 14 and Version 12 of the Size Stream have not been compared (Tiwari & Anand, 2021).

6.2.3. A Note On Ratio Use

The employ of ratio variables is common in the biological sciences to attempt to remove sources of variation, for example, the division of a scaling variable by body size. However, in their 1976 paper, Atchley and colleagues report that ratio variables are often not normally distributed, and analyses that compare ratio variables with shared components (same numerator or denominator) can experience the artificial inflation of correlation coefficients. The present study employs the use of ratio variables in the statistical analyses, and these instances must be addressed.

Percent fat mass and % lean mass are both ratio variables employed in this study and share total body mass as a denominator. Results of the Shapiro-Wilk test for both variables show significant deviations from normality. However, the Pearson correlation between these two variables was not of interest to this study and not relevant to the interpretations presented in the discussion. Furthermore, it has been shown that when there are more than ten observations, the normality assumption for regression analyses are relaxed (Schmidt & Finan, 2018).

However, it is important to note that both % lean mass and % fat mass were used in the construction of the body composition variate for the CCA. As Atchley and colleagues point out (1976), the shared denominators of these variables may be contributing in excess to the construction of the synthetic variant. Therefore, related work in the future should consider the effects of ratio variables in analyses and attempt to mitigate the effects.

6.3. Remaining Questions and Next Steps

This research sought to answer questions regarding the relationship between experiences in early life and variation in phenotypic and life history outcomes. Ultimately, this study addressed these questions and highlighted new lines of questioning and opportunities for future research.

The foundation of future research lies in expanding the sample size and depth of information collected, and further validation and standardization of the use of 3D body scanners for use in anthropological studies of human variation. These steps will allow for more specific questions regarding the role of environmental variation in the manifestation of phenotypic variation to be addressed. Specifically, the negative relationship between the crural index and segment SA:Vol ratios observed here needs to be further explored within a life history context, moving past traditional clinal or thermoregulatory explanations. Another line of inquiry is the role age at menarche plays in determining bone morphology. Estrogen concentration controls the length of the developmental window afforded to skeletal growth. Presumably, variation in age at menarche has the potential then to affect the development of bone morphology, and ultimately the degree of bone functional adaptation achieved. This relationship needs to be further clarified as it has the potential to impact interpretations of activity from skeletal remains.

Overall, this study illustrates the effectiveness of using modern, living humans as a model system for understanding phenotypic variation, environmental adaptation, and the plasticity of the human life history strategy, and additionally serves to bridge the divide between the realms of human biology and bioarchaeology. It also demonstrates the necessity of the adoption of life history theory as an explanatory framework in bioarchaeology and biological anthropology.

7. References

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8. Appendix

Table 1 *Summary of Descriptive Statistics*

Characteristics	Pooled			Athletes			Non-Athletes		
	n	Mean	Std. Deviation	n	Mean	Std. Deviation	n	Mean	Std. Deviation
% Dry Lean Mass	97	23.96	2.49	63	24.43	2.28	34	23.09	2.66
% Fat Mass	97	22.51	5.81	63	21.03	4.93	34	25.23	6.40
Age (years)	104	22.94	3.55	67	22.79	3.59	37	23.22	3.51
Age at Menarche (years)	104	13.00	1.52	67	13.04	1.38	37	12.92	1.77
Arm SA/Vol Ratio	97	51.06	4.21	64	50.15	3.85	33	52.81	4.38
Arm/Trunk Surface Area (cm^2)	97	24.28	1.77	64	24.59	1.85	33	23.68	1.45
Arm/Trunk Volume (cm^3)	97	7.47	0.73	64	7.66	0.72	33	7.11	0.60
Axilla Chest Circumference Tape Measure (cm)	97	94.22	6.49	64	94.88	5.19	33	92.94	8.41
Bicep Circumference (cm)	97	28.12	2.93	64	28.63	2.60	33	27.12	3.31
Bi-iliac Breadth (cm)	104	27.73	1.75	67	27.81	1.88	37	27.59	1.51
Birth Weight (g)	87	3476.36	498.59	58	3473.98	556.04	29	3481.10	366.20
Body Mass (kg)	104	64.06	10.15	67	65.60	9.46	37	61.26	10.88
Brachial Index	103	68.72	5.07	66	68.89	5.23	37	68.41	4.81
Calf Circumference (cm)	97	37.55	2.65	64	37.58	2.52	33	37.49	2.92
Calf/Thigh Surface Area (cm^2)	97	57.98	5.83	64	57.31	5.70	33	59.30	5.94
Calf/Thigh Volume (cm^3)	96	58.32	4.42	63	58.66	4.55	33	57.68	4.17
Crural Index	102	84.71	3.79	65	85.31	3.71	37	83.65	3.75
Femur Length (cm)	102	43.83	2.21	65	43.94	2.25	37	43.64	2.17
Forearm Circumference (cm)	97	25.43	1.92	64	25.97	1.70	33	24.37	1.90
Forearm/Upper Arm Surface Area (cm^2)	96	80.30	6.12	63	81.20	6.48	33	78.58	5.04
Forearm/Upper Arm Volume (cm^3)	96	63.83	6.56	63	64.90	7.02	33	61.79	5.08
Hip Circumference (cm)	104	98.14	7.02	67	98.33	6.59	37	97.80	7.81
Humerus Length (cm)	102	31.71	1.74	65	32.02	1.66	37	31.16	1.77
Leg SA/Vol Ratio	97	28.14	2.24	64	27.88	2.02	33	28.65	2.57
Leg/Trunk Surface Area (cm^2)	97	66.67	5.11	64	67.01	5.05	33	66.02	5.25

Table 1 *Summary of Descriptive Statistics*

Characteristics	Pooled			Athletes			Non-Athletes		
Leg/Trunk Volume (cm^3)	97	37.22	3.63	64	37.55	3.51	33	36.58	3.83
Lower Arm Length (cm)	103	22.30	1.98	66	22.81	1.92	37	21.38	1.77
Max Stomach Circumference (cm)	98	86.76	7.59	64	87.19	7.20	34	85.94	8.33
Mid-thigh Circumference (cm)	97	48.38	4.60	64	49.32	4.14	33	46.55	4.96
Shoulder Breadth (cm)	98	35.08	3.44	64	35.85	2.95	34	33.63	3.86
Stature (cm)	104	169.89	7.42	67	171.27	7.15	37	167.38	7.32
Tibia Length (cm)	102	37.12	2.41	65	37.47	2.28	37	36.51	2.54
Trunk Length (cm)	96	145.35	6.62	63	145.50	5.97	33	145.05	7.81
Trunk SA/Vol Ratio	97	15.68	1.23	64	15.60	1.11	33	15.84	1.45
Underbust Circumference (cm)	97	82.32	6.31	64	83.65	5.50	33	79.74	7.04
Waist Circumference (cm)	104	71.86	6.12	67	72.14	4.85	37	71.35	7.98
Waist to Hip Ratio (cm)	104	0.73	0.03	67	0.73	0.03	37	0.73	0.04
Valid N (listwise)	**71**			**48**			**23**		

Table 2 *Tests of Normality and Difference of Means*

Characteristics	Shapiro-Wilk Normality Test			T-Test**	Mann-Whitney U-Test†
	Statistic	df	Sig.	Sig.	Sig.
Age (years)	0.904	104	0.000*	–	0.404
Stature (cm)	0.978	104	0.087	0.01*	–
Body Mass (kg)	0.969	104	0.015*	–	0.012*
Birth Weight (g)	0.992	87	0.897	0.950	–
Bi-iliac Breadth (cm)	0.988	104	0.488	0.546	–
Waist Circumference (cm)	0.95	104	0.001*	–	0.098
Hip Circumference (cm)	0.977	104	0.064	0.716	–
Waist to Hip Ratio (cm)	0.988	104	0.480	0.423	–
% Fat Mass	0.953	97	0.002*	–	0.001*
% Dry Lean Mass	0.949	97	0.001*	–	0.017*
Humerus Length (cm)	0.978	102	0.092	0.016*	–
Femur Length (cm)	0.992	102	0.808	0.509	–
Tibia Length (cm)	0.975	102	0.054	0.052	–
Crural Index	0.99	102	0.636	0.032	–
Age at Menarche (years)	0.952	104	0.001*	–	0.583
Lower Arm Length (cm)	0.927	103	0.000*	–	0.000*
Brachial Index	0.973	103	0.035	0.641	–
Forearm/Upper Arm Volume (cm^3)	0.987	96	0.481	0.027*	–
Calf/Thigh Volume (cm^3)	0.989	96	0.603	0.308	–
Forearm/Upper Arm Surface Area (cm^2)	0.984	96	0.289	0.045*	–
Calf/Thigh Surface Area (cm^2)	0.986	97	0.399	0.111	–
Arm/Trunk Volume (cm^3)	0.984	97	0.266	0.000*	–
Leg/Trunk Volume (cm^3)	0.994	97	0.942	0.215	–
Arm/Trunk Surface Area (cm^2)	0.989	97	0.604	0.016*	–
Leg/Trunk Surface Area (cm^2)	0.991	97	0.724	0.367	–
Arm SA/Vol Ratio	0.988	97	0.539	0.003*	–
Leg SA/Vol Ratio	0.993	97	0.871	0.106	–
Trunk SA/Vol Ratio	0.989	97	0.570	0.353	–
Chest Circumference(cm)	0.969	97	0.021*	–	0.023*
Trunk Length (cm)	0.968	96	0.020*	–	0.537
Underbust Circumference (cm)	0.975	97	0.066	0.003*	–
Calf Circumference (cm)	0.981	97	0.167	0.881	–
Forearm Circumference (cm)	0.993	97	0.922	0.000*	–
Mid-thigh Circumference (cm)	0.982	97	0.199	0.004*	–
Shoulder Breadth (cm)	0.948	98	0.001*	–	0.004*
Max Stomach Circumference (cm)	0.978	98	0.106	0.441	–
Bicep Circumference (cm)	0.985	97	0.324	0.016*	

*Significance at $\alpha = 0.05$

**Difference between Athlete and Non-Athlete sub-samples for normally distributed characteristics

†Difference between Athlete and Non-Athlete sub-samples for non-normally distributed characteristics

Table 3 *Summary of Pearson Correlation Analyses*

		Stature (cm)	Body Mass (kg)	Birth Weight (g)	Bi-iliac Breadth (cm)	Waist Circumference (cm)	Hip Circumference (cm)	Waist to Hip Ratio (cm)	% Fat Mass	% Dry Lean Mass	Humerus Length (cm)	Femur Length (cm)	Tibia Length (cm)	Crural Index	Age at Menarche (years)	Lower Arm Length (cm)	Brachial Index	Forearm/Upper Arm Volume (cm^3)	Calf/Thigh Volume (cm^3)	Forearm/Upper Arm Surface Area (cm^2)	Calf/Thigh Surface Area (cm^2)	Arm/Trunk Volume (cm^3)	Leg/Trunk Volume (cm^3)	Arm/Trunk Surface Area (cm^2)	Leg/Trunk Surface Area (cm^2)	Arm SA/Vol Ratio	Leg SA/Vol Ratio	Trunk SA/Vol Ratio	Chest Circumference (cm)	Trunk Length (cm)	Underbust Circumference (cm)	Calf Circumference (cm)	Forearm Circumference (cm)	Mid-thigh Circumference (cm)	Shoulder Breadth (cm)	Max Stomach Circumference (cm)
Body Mass (kg)	Pearson Correlation	.571**																																	
	Sig. (2-tailed)	0																																	
	N	104																																	
Birth Weight (g)		.301** 0.005 87	.219* 0.042 87																																
Bi-iliac Breadth (cm)		.467** 0 104	.502** 0 104	.248* 0.02 87																															
Waist Circumference (cm)		.215* 0.028 104	.840** 0 104	0.082 0.453 87	.418** 0 104																														
Hip Circumference (cm)		.379** 0 104	.919** 0 104	0.143 0.185 87	.391** 0 104	.836** 0 104																													
Waist to Hip Ratio (cm)		-0.187 0.057 104	0.11 0.268 104	-0.084 0.441 87	0.146 0.14 104	.527** 0 104	-0.024 0.808 104																													
% Fat Mass		-.297** 0.003 97	.413** 0 97	-0.056 0.62 80	0.013 0.899 97	.620** 0 97	.603** 0 97	.201* 0.048 97																												
% Dry Lean Mass		.749** 0 97	0.118 0.251 97	0.158 0.161 80	.287** 0.004 97	-0.193 0.059 97	-0.054 0.6 97	-.262** 0.01 97	-.653** 0 97																												
Humerus Length (cm)		.798** 0 102	.442** 0 102	0.165 0.13 85	.323** 0.001 102	.195* 0.049 102	.308** 0.002 102	-0.116 0.246 102	-0.194 0.059 95	.627** 0 95																										

Table 3 *Summary of Pearson Correlation Analyses*

	Stature (cm)	Body Mass (kg)	Birth Weight (g)	Bi-iliac Breadth (cm)	Waist Circumference (cm)	Hip Circumference (cm)	Waist to Hip Ratio (cm)	% Fat Mass	% Dry Lean Mass	Humerus Length (cm)	Femur Length (cm)	Tibia Length (cm)	Crural Index	Age at Menarche (years)	Lower Arm Length (cm)	Brachial Index	Forearm/Upper Arm Volume (cm^3)	Calf/Thigh Volume (cm^3)	Forearm/Upper Arm Surface Area (cm^2)	Calf/Thigh Surface Area (cm^2)	Arm/Trunk Volume (cm^3)	Leg/Trunk Volume (cm^3)	Arm/Trunk Surface Area (cm^2)	Leg/Trunk Surface Area (cm^2)	Arm SA/Vol Ratio	Leg SA/Vol Ratio	Trunk SA/Vol Ratio	Chest Circumference (cm)	Trunk Length (cm)	Underbust Circumference (cm)	Calf Circumference (cm)	Forearm Circumference (cm)	Mid-thigh Circumference (cm)	Shoulder Breadth (cm)	Max Stomach Circumference (cm)
Femur Length (cm)	.716**	.346**	.209	.203*	.091	.229*	-.184	-.145	.561**	.712**																									
	0	0	.055	.041	.361	.021	.064	.16	0	0																									
	102	102	85	102	102	102	102	95	95	102																									
Tibia Length (cm)	.889**	.529**	.224*	.382**	.226*	.371**	-.153	-.13	.602**	.784**	.724**																								
	0	0	.039	0	.023	0	.124	.211	0	0	0																								
	102	102	85	102	102	102	102	95	95	102	102																								
Crural Index	.478**	.372**	.091	.328**	.221*	.275**	-.014	-.029	.236*	.328**	-.085	.625**																							
	0	0	.409	.001	.026	.005	.886	.784	.021	.001	.394	0																							
	102	102	85	102	102	102	102	95	95	102	102	102																							
Age at Menarche (years)	.263**	-.064	.236*	-.041	-.206*	-.15	-.141	-.262**	.309**	.241*	.295**	.261**	.055																						
	.007	.517	.028	.676	.036	.128	.153	.01	.002	.015	.003	.008	.619																						
	104	104	87	104	104	104	104	97	97	102	102	102	102																						
Lower Arm Length (cm)	.449**	.267**	.018	.270**	.058	.143	-.112	-.210*	.396**	.562**	.365**	.461**	.246*	.176																					
	0	.006	.868	.006	.561	.151	.261	.04	0	0	0	0	.013	.075																					
	103	103	86	103	103	103	103	96	96	101	101	101	101	103																					
Brachial Index	.101	-.09	.045	.035	-.188	-.115	-.171	-.107	.1	.226*	.172	.089	-.068	.119	.707**																				
	.311	.368	.684	.729	.057	.249	.084	.297	.333	.023	.085	.374	.501	.23	0																				
	103	103	86	103	103	103	103	96	96	101	101	101	101	103	103																				
Forearm/ Upper Arm Volume (cm^3)	.126	-.181	-.002	.054	-.263**	-.304**	-.011	-.544**	.338**	.057	.034	.009	-.015	.065	.252*	.206*																			
	.223	.077	.986	.599	.001	.003	.915	0	.001	.582	.743	.928	.887	.531	.014	.046																			
	96	96	81	96	96	96	96	90	90	94	94	94	94	96	95	95																			
Calf/Thigh Volume (cm^3)	.007	-.222*	-.085	.136	-.280**	-.358**	.039	-.509**	.300**	.028	.048	.014	-.028	.015	.255*	.176	.819**																		
	.503	.030	.449	.185	.006	0	.703	0	.004	.791	.645	.897	.786	.881	.013	.089	0																		
	96	96	81	96	96	96	96	90	90	94	94	94	94	96	95	95	96																		

Table 3 *Summary of Pearson Correlation Analyses*

	Stature (cm)	Body Mass (kg)	Birth Weight (g)	Bi-iliac Breadth (cm)	Waist Circumference (cm)	Hip Circumference (cm)	Waist to Hip Ratio (cm)	% Fat Mass	% Dry Lean Mass	Humerus Length (cm)	Femur Length (cm)	Tibia Length (cm)	Crural Index	Age at Menarche (years)	Lower Arm Length (cm)	Brachial Index	Forearm/Upper Arm Volume (cm^3)	Calf/Thigh Volume (cm^3)	Forearm/Upper Arm Surface Area (cm^2)	Calf/Thigh Surface Area (cm^2)	Arm/Trunk Volume (cm^3)	Leg/Trunk Volume (cm^3)	Arm/Trunk Surface Area (cm^2)	Leg/Trunk Surface Area (cm^2)	Arm SA/Vol Ratio	Leg SA/Vol Ratio	Trunk SA/Vol Ratio	Chest Circumference (cm)	Trunk Length (cm)	Underbust Circumference (cm)	Calf Circumference (cm)	Forearm Circumference (cm)	Mid-thigh Circumference (cm)	Shoulder Breadth (cm)	Max Stomach Circumference (cm)
Forearm/ Upper Arm Surface Area (cm^2)	0.141 0.17 96	-0.056 0.587 96	-0.047 0.675 81	0.081 0.433 96	-0.128 0.214 96	-0.156 0.129 96	0.005 0.962 96	-.349** 0.001 90	.248* 0.018 90	0.068 0.518 94	0.032 0.761 94	0.087 0.404 94	0.096 0.359 94	0.018 0.861 96	.288** 0.005 95	0.171 0.098 95	.921** 0 96	.776** 0 96																
Calf/Thigh Surface Area (cm^2)	0.053 0.609 97	-.226* 0.026 97	-0.069 0.543 81	0.187 0.067 97	-.250* 0.014 97	-.340** 0.001 97	0.063 0.538 97	-.405** 0 91	.206* 0.05 91	0.005 0.965 95	0.049 0.636 95	0.017 0.867 95	-0.029 0.779 95	0.086 0.405 97	0.038 0.71 96	0.063 0.543 96	.215* 0.035 96	.551** 0 96	0.081 0.432 96														
Arm/Trunk Volume (cm^3)	0.197 0.053 97	0.131 0.2 97	-0.087 0.442 81	-0.155 0.129 97	-0.002 0.981 97	0.054 0.6 97	-0.091 0.377 97	-.234* 0.026 91	.294** 0.005 91	.322** 0.001 95	0.135 0.192 95	.212* 0.039 95	0.138 0.183 95	0.132 0.198 97	.421** 0 96	0.124 0.229 96	0.11 0.286 96	0.039 0.707 96	0.142 0.167 96	-0.137 0.18 97													
Leg/Trunk Volume (cm^3)	0.142 0.167 97	0.157 0.125 97	-0.001 0.9 81	-0.187 0.067 97	-0.033 0.747 97	.204* 0.05 97	-.374** 0 97	-0.023 0.829 91	0.149 0.158 91	.238* 0.02 95	.304** 0.003 95	.241* 0.019 95	0 1 95	0.079 0.443 97	.341** 0.001 96	0.176 0.087 96	0.156 0.128 96	0.047 0.65 96	.203* 0.048 96	-.216* 0.033 97	.553** 0 97													
Arm/Trunk Surface Area (cm^2)	0.14 0.171 97	0.131 0.201 97	-0.076 0.499 81	-.227* 0.025 97	0.047 0.644 97	0.102 0.321 97	-0.007 0.964 97	-0.075 0.478 91	0.199 0.059 91	.333** 0.001 95	.221* 0.032 95	.223* 0.03 95	0.058 0.577 95	0.103 0.314 97	.389** 0 96	0.165 0.108 96	-0.004 0.967 96	-0.091 0.378 96	0.036 0.727 96	-.245* 0.015 97	.907** 0 97	.607** 0 97												
Leg/Trunk Surface Area (cm^2)	0.142 0.164 97	0.162 0.113 97	0.006 0.956 81	-.215* 0.035 97	0.019 0.852 97	.208* 0.041 97	-.286** 0.005 97	0.048 0.653 91	0.127 0.23 91	.229* 0.026 95	.361** 0.003 95	.274** 0.007 95	-0.019 0.855 95	0.068 0.509 97	.279** 0.006 96	0.145 0.161 96	0.031 0.767 96	-0.073 0.479 96	0.092 0.372 96	-.287** 0.004 97	.531** 0.001 97	.920** 0 97	.716** 0 97											
Arm SA/Vol Ratio	-.384** 0 97	-.883** 0 97	-0.081 0.475 81	-.355** 0 97	-.786** 0 97	-.831** 0 97	-0.144 0.158 97	-.394** 0 91	-0.051 0.632 91	-.286** 0.005 95	-0.103 0.319 95	-.343** 0.001 95	-.360** 0 95	0.111 0.281 97	-.242* 0.017 96	0.126 0.222 96	.224* 0.028 96	.229* 0.025 96	0.077 0.457 96	-.250* 0.014 97	-.384** 0 97	-0.147 0.151 97	-.307** 0.002 97	-0.152 0.137 97									
Leg SA/Vol Ratio	-.334** 0.001 97	-.908** 0 97	-0.106 0.307 81	-.328** 0.001 97	-.797** 0 97	-.928** 0 97	-0.115 0.259 97	-.536** 0 91	0.055 0.603 91	-.288** 0.005 95	-0.179 0.082 95	-.346** 0.001 95	-.283** 0.005 95	0.14 0.17 97	-.247* 0.015 96	0.055 0.597 96	0.193 0.06 96	.224* 0.028 96	0.042 0.682 96	-.302** 0.003 97	-0.167 0.103 97	-.369** 0 97	-0.187 0.067 97	-.329** 0.001 97	.873** 0 97							

Table 3 *Summary of Pearson Correlation Analyses*

	Stature (cm)	Body Mass (kg)	Birth Weight (g)	Bi-iliac Breadth (cm)	Waist Circumference (cm)	Hip Circumference (cm)	Waist to Hip Ratio (cm)	% Fat Mass	% Dry Lean Mass	Humerus Length (cm)	Femur Length (cm)	Tibia Length (cm)	Crural Index	Age at Menarche (years)	Lower Arm Length (cm)	Brachial Index	Forearm/Upper Arm Volume (cm^3)	Calf/Thigh Volume (cm^3)	Forearm/Upper Arm Surface Area (cm^2)	Calf/Thigh Surface Area (cm^2)	Arm/Trunk Volume (cm^3)	Leg/Trunk Volume (cm^3)	Arm/Trunk Surface Area (cm^2)	Leg/Trunk Surface Area (cm^2)	Arm SA/Vol Ratio	Leg SA/Vol Ratio	Trunk SA/Vol Ratio	Chest Circumference (cm)	Trunk Length (cm)	Underbust Circumference (cm)	Calf Circumference (cm)	Forearm Circumference (cm)	Mid-thigh Circumference (cm)	Shoulder Breadth (cm)	Max Stomach Circumference (cm)
Trunk SA/Vol Ratio	-.289**	-.871**	0.123	-.346**	-.859**	-.884**	-.200*	.615**	0.121	-.208*	-.157	-.309**	-.255*	0.171	-0.095	0.131	.368**	.362**	.219*	.309**	0.003	-0.028	-0.137	-0.168	.839**	.863**									
	0.004	0	0.276	0.001	0	0	0.049	0	0.253	0.043	0.128	0.002	0.013	0.093	0.36	0.202	0	0	0.032	0.002	0.977	0.782	0.18	0.099	0	0									
	97	97	81	97	97	97	97	91	91	95	95	95	95	97	96	96	96	96	96	97	97	97	97	97	97	97									
Chest Circumference (cm)	.344**	.866**	0.082	.456**	.867**	.821**	.306**	.508**	-0.088	.300**	0.106	.335**	.350**	-0.156	0.163	-0.067	-.241*	-.250*	-0.115	-.246*	-0.004	0.081	0.026	-0.023	-.802**	-.777**	-.852**								
	0.001	0	0.469	0	0	0	0.002	0	0.407	0.003	0.308	0.001	0.001	0.127	0.113	0.518	0.018	0.014	0.266	0.015	0.968	0.433	0.801	0.823	0	0	0								
	97	97	81	97	97	97	97	91	91	95	95	95	95	97	96	96	96	96	96	97	97	97	97	97	97	97	97								
Trunk Length (cm)	.626**	.713**	.265*	.470**	.545**	.648**	-0.024	0.206	.232*	.436**	.297**	.505**	.382**	-0.071	.294**	0.138	-0.049	-0.046	0.029	-0.026	-0.007	-0.091	-0.103	-0.107	-.561**	-.569**	-.574**	.612**							
	0	0	0.017	0	0	0	0.816	0.051	0.028	0	0.004	0	0	0.489	0.004	0.184	0.636	0.66	0.784	0.802	0.501	0.379	0.316	0.298	0	0	0	0							
	96	96	80	96	96	96	96	90	90	94	94	94	94	96	95	95	95	95	95	96	96	96	96	96	96	96	96	96							
Underbust Circumference (cm)	.418**	.831**	0.165	.456**	.814**	.724**	.368**	.349**	0.038	.310**	0.154	.389**	.374**	-0.097	0.184	-0.109	-.203*	-0.167	-0.115	-0.152	-0.048	-0.189	-0.017	-0.096	-.769**	-.696**	-.839**	.846**	.604**						
	0	0	0.14	0	0	0	0	0.001	0.719	0.002	0.135	0	0	0.345	0.072	0.291	0.047	0.104	0.266	0.138	0.642	0.064	0.872	0.346	0	0	0	0	0						
	97	97	81	97	97	97	97	91	91	95	95	95	95	97	96	96	96	96	96	97	97	97	97	97	97	97	97	97	96						
Calf Circumference (cm)	0.18	.689**	0.09	.324**	.624**	.699**	0.046	.379**	-0.074	0.114	0.092	0.123	0.07	-0.136	0.198	0.041	-0.03	0.061	0.009	0.093	0.002	.297**	-0.022	0.129	-.640**	-.788**	-.613**	.588**	.453**	.508**					
	0.077	0	0.425	0.001	0	0	0.657	0	0.485	0.27	0.373	0.236	0.498	0.184	0.053	0.688	0.771	0.52	0.927	0.366	0.849	0.003	0.845	0.219	0	0	0	0	0	0					
	97	97	81	97	97	97	97	91	91	95	95	95	95	97	96	96	96	96	96	97	97	97	97	97	97	97	97	97	96	97					
Forearm Circumference (cm)	.438**	.841**	0.143	.381**	.714**	.748**	0.14	.220*	0.135	.340**	0.139	.339**	.318**	-0.083	.304**	-0.046	0.021	-0.033	0.074	-0.144	.413**	0.152	.304**	0.122	-.928**	-.798**	-.727**	.740**	.558**	.736**	.668**				
	0	0	0.203	0	0	0	0.172	0.036	0.201	0.001	0.178	0.001	0.002	0.421	0.003	0.658	0.837	0.75	0.474	0.166	0	0.136	0.002	0.235	0	0	0	0	0	0	0				
	97	97	81	97	97	97	97	91	91	95	95	95	95	97	96	96	96	96	96	97	97	97	97	97	97	97	97	97	96	97	97				
Mid-thigh Circumference (cm)	.358**	.909**	0.116	.349**	.779**	.879**	0.058	.471**	-0.042	.273**	0.165	.340**	.293**	-0.128	.227*	-0.084	-0.17	-.241*	-0.046	-.301**	0.167	.283**	0.163	.256*	-.869**	-.943**	-.838**	.771**	.526**	.727**	.759**	.825**			
	0	0	0.301	0	0	0	0.574	0	0.691	0.007	0.109	0.001	0.004	0.211	0.026	0.414	0.097	0.016	0.581	0.003	0.102	0.004	0.112	0.011	0	0	0	0	0	0	0	0			
	97	97	81	97	97	97	97	91	91	95	95	95	95	97	96	96	96	96	96	97	97	97	97	97	97	97	97	97	96	97	97	97			

Table 3 *Summary of Pearson Correlation Analyses*

	Stature (cm)	Body Mass (kg)	Birth Weight (g)	Bi-iliac Breadth (cm)	Waist Circumference (cm)	Hip Circumference (cm)	Waist to Hip Ratio (cm)	% Fat Mass	% Dry Lean Mass	Humerus Length (cm)	Femur Length (cm)	Tibia Length (cm)	Crural Index	Age at Menarche (years)	Lower Arm Length (cm)	Brachial Index	Forearm/Upper Arm Volume (cm^3)	Calf/Thigh Volume (cm^3)	Forearm/Upper Arm Surface Area (cm^2)	Calf/Thigh Surface Area (cm^2)	Arm/Trunk Volume (cm^3)	Leg/Trunk Volume (cm^3)	Arm/Trunk Surface Area (cm^2)	Leg/Trunk Surface Area (cm^2)	Arm SA/Vol Ratio	Leg SA/Vol Ratio	Trunk SA/Vol Ratio	Chest Circumference (cm)	Trunk Length (cm)	Underbust Circumference (cm)	Calf Circumference (cm)	Forearm Circumference (cm)	Mid-thigh Circumference (cm)	Shoulder Breadth (cm)	Max Stomach Circumference (cm)
Shoulder Breadth (cm)	.465**	.532**	.223*	.376**	.432**	.444**	.0115	.0173	.0154	.477**	.394**	.524**	.306**	.01	.258*	-.0001	-.0115	-.0099	-.0013	-.0011	.0095	.0112	.0077	.0119	-.411**	-.408**	-.389**	.482**	.327**	.418**	.280**	.339**	.405**		
	0	0	.0045	0	0	0	.0258	.01	.0143	0	0	0	.0002	.0327	.0011	.999	.0266	.0335	.9002	.9014	.0354	.0276	.0452	.0247	0	0	0	0	.0001	0	.0005	.0001	0		
	98	98	81	98	98	98	98	92	92	96	96	96	96	98	97	97	96	96	96	97	97	97	97	97	97	97	97	97	96	97	97	97	97		
Max Stomach Circumference (cm)	.344**	.869**	.167	.473**	.841**	.857**	.226*	.578**	-.0083	.295**	.237*	.364**	.245*	-.0177	.052	-.207*	-.322**	-.292**	.195	-.235*	-.0038	.02	.033	.092	-.805**	-.842**	-.904**	.817**	.551**	.813**	.623**	.730**	.819**	.447**	
	.001	0	.136	0	0	0	.025	0	.432	.004	.02	0	.016	.881	1	.042	.001	.004	.058	.02	.712	.844	.749	.473	0	0	0	0	0	0	0	0	0	0	
	98	98	81	98	98	98	98	92	92	96	96	96	96	98	97	97	96	96	96	97	97	97	97	97	97	97	97	97	96	97	97	97	97	98	
Bicep Circumference (cm)	.266**	.865**	.055	.340**	.833**	.858**	.187	.516**	-.0113	.194	.025	.247*	.312**	-.0162	.251	-.0081	-.265**	-.278**	-.0113	-.271**	.243*	.096	.201*	.102	-.943**	-.872**	-.855**	.836**	.568**	.780**	.680**	.876**	.882**	.328**	.801**
	.008	0	.659	.001	0	0	.067	0	.284	.06	.808	.016	.002	.114	.051	.433	.009	.006	.974	.007	.016	.348	.049	.321	0	0	0	0	0	0	0	0	0	.001	0
	97	97	81	97	97	97	97	91	91	95	95	95	95	97	96	96	96	96	96	97	97	97	97	97	97	97	97	97	96	97	97	97	97	97	97

** Correlation is significant at the 0.01 level (2-tailed).

* Correlation is significant at the 0.05 level (2-tailed).

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