The Influence of Developmental Parameters on Body Size and Composition: An Analysis of Sex Differences in Life History Trajectories

by

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Abstract

Previous research has indicated that markers of early life development (birth weight, age at first menstruation (menarche), and relative leg length) are predictors of adult body size and composition (adiposity and lean mass). The life history theory states that an individual distributes metabolic energy between four functions: reproduction, growth, maintenance, and immunity. The pace of growth and development and the way metabolic energy is allocated is significantly impacted by the markers of early life development. Females tend to allocate more energy into adiposity (fat mass) while males tend to allocate more into lean mass. My research looks at a sample of ultramarathon runners (n=71), including both males and females. I aim to determine (1) if there are relationships between markers of early life development and adult body size and composition; (2) if these relationships appear in ways predicted by the life history model; and (3) if males invest more or less into lean mass. I ran partial Pearson's correlation analyses and linear regression analyses to investigate any relationships. Furthermore, I assess the impact that early life predictors of adult body size and composition may have in predicting individuals who are at a heightened risk of developing metabolic and/or cardiovascular diseases. I also consider the influence that socioeconomic status has on an individual's access to preventative care and treatments.

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Introduction

The way our bodies grow and develop can have lifelong effects. Previous research has indicated that markers of early life growth in females, such as birth weight, age at menarche (first menstruation), and relative leg length predict adult body size and composition (Wells 2010, pg. 9; Macintosh et al. 2018, pg. 168). Females born with lower birth weights (<2,500 grams) tend to become smaller adults in stature, yet have higher adiposity (body fat percentage; Kim & Saada, 2013, pg. 2298; Macintosh et al. 2018, pg. 168). This is significant as adipose tissue is an important reproductive tissue in females (Wells 2012, pg. 417). While these trends are increasingly studied in maternal health studies, there is a lack of research conducted on how birth weight affects the pace of male growth and development and, thus, adult male body size and composition. Throughout my paper, I examine the relationship between early life markers of growth and development (birth weight, age at menarche, relative leg length) adult body size (height, weight) and composition (percentage of lean mass and adiposity). I also examine how these relationships compare and/or differ between adult males and females.

The Life History Model

The life history model, or life history theory, postulates that organisms, in this case humans, go through their lives making trade-offs in their metabolic energy distribution (Macintosh et al. 2018, pg. 168). There are four central metabolic functions: reproduction, growth, maintenance, and immunity (Macintosh et al. 2018, pg. 168). Throughout the lifespan, an organism differentially distributes metabolic energy towards these functions through a variety of levels (Hill 1993, pg. 79-80; Brumbach et al. 2009, pg. 26). These distributions “are made at the molecular, physiological, and behavioural levels,” (Hill 1993, pg. 80) meaning it is usually a metabolic decision rather than a conscious decision by the individual through their actions. Accordingly, the life history model is situated in evolutionary studies of metabolic energy distribution (Brumbach et al. 2009, pg. 26).

Reproduction is an important aspect of the life history model that is often focused on from an anthropological perspective. In females, if birth weight is low, reproduction is often prioritized over growth and maintenance and her pace of development will speed up (Macintosh et al. 2018, pg. 168; Wells et al. 2016, pg. 134; Hill 1993, pg. 80). This implies that the conditions of her environment are unfavourable in one or many ways, and, thus, she invests more into reproductive function to ensure she will reproduce (Macintosh et al. 2018, pg. 168; Wells et al. 2016, pg. 134). This is seen through earlier age at menarche and an increase in adiposity (Macintosh et al. 2018, pg. 172). In adulthood, she will likely have a shorter stature which is indicative of her investing more into reproductive development over growth and maintenance (Macintosh et al. 2018, pg. 168 & 172). She may achieve catch up growth if her mother is able to invest heavily into lactation (Macintosh et al. 2018, pg. 168). Catch-up growth is vital for improving an infant's chance of survival if their fetal conditions were unfavourable (Wells 2010, pg. 9). However, catch-up growth is correlated with an increase in adipose tissue and disease risk especially if the individual was born very small (Wells 2010, pg. 3). Thus, low birth weight may still carry implications for adult body composition and cardiovascular disease risk even if the individual was well-nourished as an infant and child (Wells 2010, pg. 11).

Alternatively, if a female is born with a mid-to-high birth weight, she will likely allocate more metabolic energy towards growth and maintenance and invest in reproduction later (Macintosh et al. 2018, pg. 168). This is indicated by a later age at menarche, taller stature, and less adiposity (Macintosh et al. 2018, pg. 173). Wells (2010, pg. 10) states that maternal height is indicative of fertility rates noting that “in western societies, women marginally shorter than average have [a] greater fertility rate,” likely because they invest more energy into reproduction than taller females do (Wells 2010, pg. 10). As far as I am aware, the life history model has not been as heavily studied in males, and thus, data on male strategies of metabolic energy distribution is lacking.

Significance of Birth Weight

Consideration of birth weight is imperative to understanding early life growth and it is often used as a proxy for fetal development and maternal environment (Macintosh et al. 2018, pg. 168). It is often an indicator of how well an infant will survive after birth and potentially how long they will live as an adult (Wells et al. 2016, pg. 134). In a 2013 study, 8.2% of infants born in the United States and 4.8-7.1% of infants born in Western European countries were of low birth weight (Kim & Saada 2013, pg. 2298). This study also states that infants that are born with a low birth weight “have a 20 times higher risk of death than heavier infants” (Kim & Saada 2013, pg. 2298).

Birth weight is an important marker of maternal investment of energy into a fetus (Wells 2010, pg. 8 & 10). Specifically, low birth weight often indicates lower investment of maternal “capital” into offspring (Wells 2010, pg. 10). Maternal capital, in this sense, is “defined as phenotypic resources enabling investment in the offspring” (Wells 2010, pg. 2). This will result in restricted fetal growth, even if the infant is delivered full-term (Merklinger-Gruchala, Jasienska, & Kapiszeqska 2017, pg. 71). Often, “poor fetal nutrition [during pregnancy…] induces substantial long-term costs” on offspring growth and development into adulthood (Wells 2010, pg. 9). Furthermore, Wells (2010) states that low birth weight infants may have developed their small size as a strategy that allows them to reduce their "energy burden" on their mother if she has limited energy resources to transfer to them in the womb (pg. 9).

Catch-up growth is one response that a low birth weight infant may have if they receive adequate nutrition in infancy (Wells 2010, pg. 9). This will cause a rapid increase in their adiposity to alleviate the stress of small body size and improve the infants' chances of survival (Well 2010, pg. 9). This high percentage of adiposity may persist into childhood and adolescence, leading to an increased risk of obesity and disease risk in adulthood (Wells 2010, pg. 3).

Previous research has found that individuals who experience a poor fetal developmental environment are "predispose[d] to stroke, type 2 diabetes, and cardiovascular disease" (Wells 2010, pg. 2). Additionally, low birth weight has also been correlated with an increased "risk of the metabolic syndrome (high blood pressure, glucose intolerance, dyslipidemia, central abdominal adiposity) and diseases such as […] hypertension and heart disease" (Wells 2010, pg. 2). Low birth weight carries implications for the rate of compositional growth of an individual (specifically in adipose tissue development), especially if they experience rapid catch-up growth in infancy and childhood to increase the individual's chances of survival (Wells 2010, pg. 3 & 9). This may also accelerate reproductive development in females (Wells 2010, pg. 9 & 10).

Significance of Age at Menarche

Menarche is defined as a female's first incidence of menstrual bleeding (Ziomkiewicz & Koziel 2015, pg. 169). It is an important marker in a female's reproductive development and a "complex physiological process of puberty" (Ziomkiewicz & Koziel 2015, pg. 169). The average age of menarche differs greatly between populations, usually depending on the typical growth conditions of that population (Ziomkiewicz & Koziel 2015, pg. 170). For example, females living in populations that experience frequent bouts of malnutrition will often have a later age at menarche whereas females with high-energy intake and higher levels of adiposity will often experience menarche earlier (Ziomkiewicz & Koziel 2015, pg. 170).

Age at menarche is also associated with the developmental pace of a female (Wronka 2013, pg. 319). For instance, Ziomkiewicz and Koziel (2015) state that age at menarche is dependent on the amount of adipose tissue a female has stored (pg. 170). Therefore, females with increased adiposity will often experience menarche earlier and, thus, be able to reproduce sooner than their leaner counterparts (Ziomkiewicz & Koziel 2015, pg. 170). Arguably, the catch-up growth experienced by low birth weight infants who receive adequate infancy and childhood nutrition may impact a females' age at menarche as she may develop an excess amount of adipose tissue (Ziomkiewicz & Koziel 2015, pg. 170; Wells 2010, pg. 3). Additionally, Macintosh and colleagues (2018) found in their study that "[a]ge at menarche exhibited stronger relationships with adult adiposity than did physical activity" (pg. 167).

Studies have shown that females who experience menarche later relative to their population counterparts will often grow to be taller adults with lower adiposity (Macintosh et al. 2018, pg. 173; Nieczuja-Dwojacka et al. 2017, pg. 142). An earlier age at menarche and low birth weight are both considered to be faster life history strategies associated with shorter stature and higher adiposity (Wells et al. 2016, pg. 140).

Significance of Relative Leg Length

Relative leg length is an indicator of the pace of growth that I look at in my participants. Bogin and Varela-Silva (2010, pg. 1060) state that relative leg length "is an indicator of the quality of the environment for growth during infancy, childhood and the juvenile years of development.” Relative leg length is calculated by dividing leg length by height (Bogin & Varela-Silva 2010, pg. 1047). Studies have shown that relative leg length is a more accurate depiction of nutritional status in infancy and childhood than total adult height or weight (Bogin & Varela-Silva 2010, pg. 1061). Furthermore, relative leg length can also be indicative of an individual’s socioeconomic status as an infant, child, and adolescent (Kinra et al. 2011, pg. 1023; Ashizawa et al. 2008, pg. 68). Various factors can influence relative leg length including “poor childhood health, insufficient diet, adverse family circumstances and maternal smoking during pregnancy” (Bogin & Varela-Silva 2010, pg. 1064).

The reason that relative leg length is examined and not simply an individuals’ height is because relative leg length is highly affected “by a shortage of nutrients, infection, parasites, physical or emotion trauma, and other adverse conditions (Bogin & Varela-Silva 2010, pg. 1060). Bogin and Varela-Silva (2010, pg. 1060) state that from birth to seven years old, an individuals’ legs, particularly the tibia in the lower leg, grow at a rate more rapid than any other body segment. The growth rate of leg length, particularly lower leg growth, is very prominent in infancy, childhood, and adolescence and is, therefore, a very good indicator of the nutritional environment of an individual while they were growing and may even be indicative of one’s socioeconomic status (Koziel, Gomula et al. 2016, pg. 405; Bogin & Varela-Silva 2010, pg. 1060-1061).

Leg length can also be an important indicator of health status and outcomes (Bogin & Varela-Silva 2010, pg. 1064). Research has shown that individuals with “relatively shorter legs and shorter stature […] may increase the risk for overweight (fatness), coronary heart disease and diabetes” and various cancers (Bogin & Varela-Silva 2010, pg. 1064; Nieczuja-Dwojacka et al. 2017, pg. 142). Additionally, individuals with shorter leg length, particularly lower leg length, are at an increased risk of "hypertension, obesity or liver dysfunction, and dementia" (Koziel et al. 2016, pg. 406). Fetal and infant malnourishment and disease are correlated with relatively shorter adult legs and increased risk of disease and early mortality though the reasons for this are not completely clear (Bogin & Varela-Silva 2010, pg. 1067).

Significance of Adipose Tissue

Adipose tissue is a very important reproductive tissue in females (Wells 2012, pg. 417). Wells found that "dimorphism in body composition is typically substantially greater than that in stature or BMI, averaging 25-55% for adiposity outcomes and 27% for lean mass," with females favouring adiposity and males favouring lean mass (Wells 2012, pg. 416-417). Factors that tend to increase adipose distribution include "early menarcheal age, increased number of pregnancies, high parity [number of previous births] and the event of menopause" (Debnath et al. 2020, pg. 210; Merklinger-Gruchala 2017, pg. 75). Debnath and colleagues (2020, pg. 210) state that increased abdominal adiposity and waist circumference is associated "with socioeconomic factors like age, educational status, occupational status, [and] food habit."

Females use adipose tissue as an energy reserve, particularly during and after pregnancy (Wells 2012, pg. 417). During pregnancy, a good supply of adipose tissue is crucial for mothers due to the high energetic costs of growing an infant (Wells 2010, pg. 5). After pregnancy, adiposity is still valuable as females use fat reserves to produce milk for her infant(s) (Wells 2012, pg. 417; Wells 2010, pg. 5). This is referred to as lactation or breastfeeding (Fewtrell, Shukri, & Wells 2020, pg. 2). The World Health Organization (WHO) suggests that infants be breastfed for at least 2 years; thus, mothers must be actively lactating for a significant period of time and thus need a sufficient supply of energy (Fewtrell et al. 2020, pg. 2; Wells 2012, pg. 417).

Adiposity in childhood and adolescence is a significant indicator of adulthood obesity in males (Sandhu et al. 2006, pg. 17). Sandhu and colleagues (2006) found that higher adiposity in childhood led to an earlier start of pubertal development in participants. Earlier puberty then led to epiphyseal plate, or growth plate, fusion earlier (Sandhu et al. 2006, pg. 17). Thus, the researchers found that participants experienced an "earlier cessation of growth, with consequent interruption to lean body mass acquisition" (pg. 17). Similar to what has been observed in females by Macintosh and colleagues (2018, pg. 168), the males who experienced a later puberty became taller adults with lower body mass index (BMI; Sandhu et al. 2006, pg. 17). This is significant because it suggests that there is a "critical period" for preventative measures to curve the effects of prepubertal childhood obesity on adult male bodies (Sandhu et al. 2006, pg. 19). Sandhu and colleagues (2006, pg. 19) state that research on childhood adiposity "is particularly important to determine whether the age at onset has differential effects on the persistence, morbidity and mortality effects of obesity."

Significance of Waist-to-Hip Ratio

Waist-to-hip ratio, for the purposes of my research, was a useful way to "describe abdominal or central adiposity" (Kabalin et al. 2012, pg. 364). This is calculated by dividing waist circumference by hip circumference (World Health Organization 2008, pg. 1). A low waist-to-hip ratio would be if an individual had a "small waist and large hips" (Cashdan 2008, pg. 1099). This value is significant for studies on body size and composition because previous research has found that as waist-to-hip ratio increases, so does the risk of cardiovascular disease

(Cashdan 2008, pg. 1099). Generally, as one ages, so does their waist-to-hip ratio (Cashdan 2008, pg. 1099).

Significance of Lean Mass

Lean mass is a combination of several components of human body composition. The components are: bone, muscle, vital organs (such as liver, brain, heart, and kidneys), extracellular fluid, and lipid in cellular membranes (Heymsfield et al. 2007, pg. 83; Yu et al. 2013, pg. 2). Together, fat mass (adipose tissue) and fat free mass (lean mass) make up an individual's total body weight (Yu et al. 2013, pg. 2). Wells states that organ growth (such as in the heart, liver, kidney, and pancreas) is closely aligned with both fetal and infant development and growth in stature (Wells 2010, pg. 2). Moreover, those born at a low birth weight often experience faster growth trajectories and rapid weight gain postnatally (Wells 2010, pg. 2). Additionally, low birth weight is also correlated with low percentage of lean mass in adulthood (Wells 2010, pg. 3). In turn, low lean mass has been found to be significantly correlated with an increased risk of "suffering from diabetes or cardiovascular disease" in adulthood (Wells 2010, pg. 2).

In males, lean mass is an especially important component of body composition (Wells 2012, pg. 416-417). Johnson and colleagues (2020) state that lean mass, particularly muscle mass, "promotes sexual signalling" to potential mates (pg. 205), with males having, on average, 61% greater muscle mass than females (Lassek & Gaulin 2009, pg. 322). Additionally, lean mass is an important male secondary sexual characteristic that is often indicative of the amount of "mating opportunities and the number of offspring fathered" (Wells 2012, pg. 417). In females, lean mass is often a significant indicator of offspring birth weight (Johnson et al. 2020 pg. 205).

As one ages, adipose tissue generally begins to increase as percentage of lean mass usually begins to decline (Wells 2012, pg. 417; Koster et al. 2011, pg. 889). Particularly in females, visceral adipose tissue has been shown to increase as they go through menopause (Demerath et al. 2011, pg. 247). Visceral tissue is primarily found within the abdominal region (Demerath et al. 2011, pg. 254). For both males and females, however, excessive adipose tissue paired with a significant decrease in muscle mass increases an individual's risk of developing sarcopenia (Koster et al. 2011, pg. 889). This disease is characterized by "a combination of low muscle mass with low muscle function" (Tournadre et al. 2019, pg. 309).

For males and females, investing energy into reproductive function is quite different. Females invest mostly into adipose tissue to support the metabolic costs of pregnancy and lactation, whereas males will generally invest more into lean mass as a secondary sexual characteristic that often indicates reproductive success (Wells 2012, pg. 417). Therefore, for my study, I was interested in seeing if low birth weight males invest more into reproductive development (lean mass), similar to how low birth weight females tend to invest more into adiposity.

The Social Determinants of Health

As outlined above, low birth weight, early age at menarche, and shorter relative leg length, all have shown correlations with various metabolic diseases related to a rapid life history trajectory (short stature, high adiposity, and low lean mass) including obesity, cardiovascular diseases, and type 2 diabetes (Bogin & Varela-Silva 2010, pg. 1064; Wells 2010, pg. 13; Wronka 2013, pg. 326). Poor early life conditions, for which low birth weight is a proxy of, are correlated with a rapid life history trajectory wherein reproductive development is prioritized and elevated adipose tissue and short stature are the typical outcomes (Macintosh et al. 2018, pg. 172). These relationships are significant for researchers who study how early life growth conditions may affect adult health, and also for understanding how low socioeconomic status may have long-term impacts on an individual's health outcomes (O'Campo & Urquia 2012, pg. 1871). For this reason, The Social Determinants of Health were developed.

The Social Determinants of Health were developed by the World Health Organization to critically analyze the social conditions of health outcomes and inequalities (Yates-Doerr 2020, pg. 378). Examples of factors that can influence health outcomes include "income, educational opportunities, access to housing, and food security" (Yates-Doerr 2020, pg. 378-379). Furthermore, an individual's access to healthcare and overall health status is also influenced by socioeconomic factors such as race, ethnicity, housing, gender, and "social capital/cohesion" (Kim & Saada 2013, pg. 2299-2300). Low socioeconomic status and heighted periods of stressors in early life has been correlated with an increase in adolescent and adult chronic diseases regardless of whether the individual increased their socioeconomic standing in their adolescent and adult years (Braveman & Gottlieb 2014, pg. 24). Braveman and Gottlieb (2014) define early life as being before the age of 5 (pg. 24).

As an example of how socioeconomic status may affect overall health outcomes, Braveman and Gottlied (2014, pg. 26) outline several ways that education can be linked to development of chronic diseases. Education facilitates literacy and overall understanding of health. Thus, educated individuals may be more likely to exercise regularly, make conscious choices in their diet, and avoid risky behaviours such as smoking (Braveman & Gottlieb 2014, pg. 26). So, while education may not directly impact an individual's health, it influences the decisions they make and the understanding of their bodies, which may positively affect their health outcomes later in life (Braveman & Gottlieb 2014, pg. 26). Additionally, education may improve an individual's job outcomes so they may have access to healthier foods and safer work conditions (Braveman & Gottlieb 2014, pg. 26).

Importance of Maternal Health

When looking at factors of developmental growth, the overall health of an individual's mother cannot be overlooked. Low birth weight is often indicative of lower maternal investment which has been shown to speed up her offspring's life history trajectory (Macintosh et al. 2018, pg. 172). Therefore, when a female is born with a lower birth weight, she will often speed up physical growth and achieve "shorter stature, smaller pelvises, and lower lean mass" (Macintosh et al. 2018, pg. 168). Adversely, a mother's ability to invest more in her infant through nourishment during and following pregnancy (via lactation) has been shown to slow her infants' pace of growth and allow them to invest more metabolically into growth and maintenance (Wells et al. 2016, pg. 134-135).

There are aspects of maternal body size and composition that impact a woman's ability to conceive a child. For example, Wells (2010, pg. 10) states that "women marginally shorter than average have greater fertility rate." Furthermore, Wells (2010, pg. 10) states that maternal weight has also been associated with increased fertility in higher socioeconomic status individuals in lower-income regions.

A mother's socioeconomic status can also significantly affect her offspring's fetal and infant development (Kim & Saada 2013, pg. 2299-2300). Various socioeconomic factors such as race, ethnicity, gender, residential segregation impact living and working conditions, stress levels and psychological wellbeing, and health outcomes (Kim & Saada 2013, pg. 2300). Financial factors also play a major role in blocking a mother's access to prenatal care in addition to inaccessible transportation services in some neighbourhoods (Kim & Saada 2013, pg. 2300). In turn, research has shown that these factors are significantly correlated with an increase in adverse birth outcomes, such as low birth weight, and infant mortality rates (Kim & Saada 2013, pg. 2300).

Intergenerational imprints may impact the signals that a mother gives to her fetus (Wells 2010, pg. 8). Specifically, the growth and developmental conditions that a mother experienced throughout her life trajectory may be "translate[d] into pay-offs in offspring birth weight" (Wells 2010, pg. 8). Thus, low socioeconomic status may impact a fetus' birth weight and infant weight gain if the mother experienced poor nutritional status during her development (Wells 2010, pg. 8). Specifically, if a mother is born at a low birth weight and experiences a rapid life history trajectory that prioritizes reproductive development, her offspring may receive signals to develop in a similar way (Wells 2010, pg. 11). This is often observed when mothers born at a low birth weight give birth to infants born at a low birth weight, even if they were not "malnourished throughout postnatal life" (Wells 2010, pg. 11). In turn, an intergenerational effect takes place which may be reversed if adequate nutrition and improved environmental conditions ensue for future generations (Wells 2010, pg. 11). Therefore, it is arguable that improving maternal health care and environmental conditions may have a positive impact on reducing chronic metabolic diseases in adults.

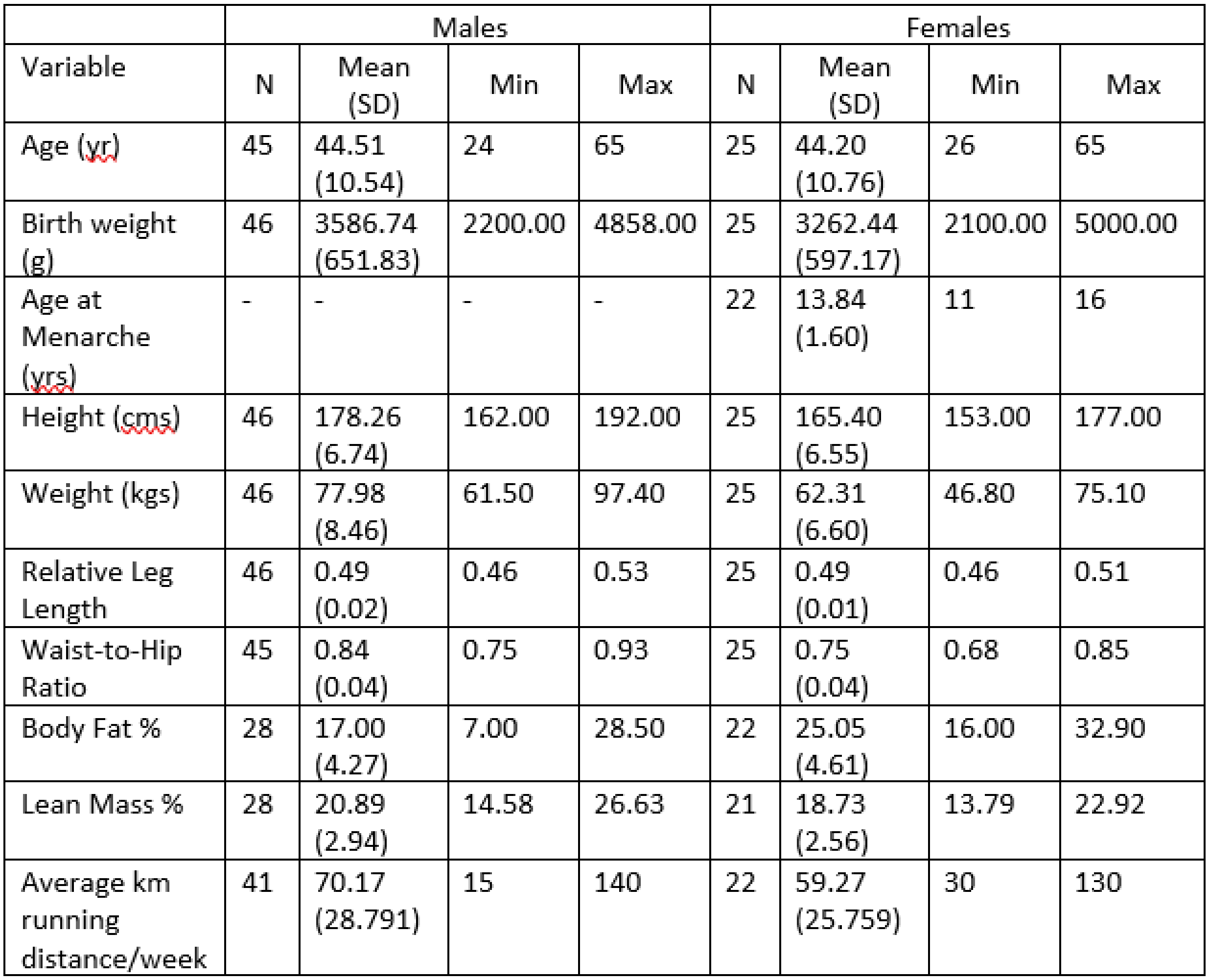
Research Questions

My aim in this research is to see how markers of early life development may affect body size and composition in both males and females. I also wanted to see if I could find similar trends to what has been observed through previous research, such as low birth weight leading to higher adiposity and shorter stature in females (Macintosh et al 2018, pg. 168). Thus, my primary research question is to determine if there are relationships between markers of early life growth, such as birth weight, relative leg length, and age at menarche, and adult body size and composition differences between males and females. If so, my secondary research question is: do they differ in ways predicted by life history theory? Specifically, does poor early life growth (as indicated by proxy measures) lead to differential investment in lean mass among males and fat mass among females? Do males with lower birth weight invest more or less energy into secondary sex characteristics like lean mass?

Research Sample

My research draws on a dataset initially comprised of 140 individuals. The dataset was generated for a study by Dr. Alison Murray and colleagues to examine the physical, hormonal, and metabolic impacts of ultramarathon training on both males and females. The participants were given questionnaires prior to the start of the study so they were given adequate time to retrieve their recorded birth weight information. My research focused on analyzing the data of those participants (n=71) who were able to recall their birth weights, resulting in a sample that included 25 females and 46 males. Relevant data included bodily measurements: height (cm); weight (kgs); sitting height (cm), which is the length of the head, neck, and torso; and relative leg length (cm), which is height minus sitting height. Body composition was assessed using waist and hip circumferences (cm) and percentage of fat and dry lean (an estimate of lean mass minus body water) in proportion to their body weight (Wells & Fewtrell 2006, pg. 615). This was collected by Dr. Murray and colleagues using bioelectrical impedance analysis with a Quadscan Bodystat 4000 (Grundmann, Yoon, & Williams 2015, pg. 1290). For females, relevant data included age at menarche which was also recalled on the questionnaires. Additionally, I consider the role that age may play in the distribution of adipose tissue and lean mass among both my male and female participants. I have included summary statistics for all of the variables of interest below in Table 1.

Table 1. Summary statistics for relevant body size, body composition, and activity levels by sex.



	Males				Females			
Variable	N	Mean (SD)	Min	Max	N	Mean (SD)	Min	Max
Age (yr)	45	44.51 (10.54)	24	65	25	44.20 (10.76)	26	65
Birth weight (g)	46	3586.74 (651.83)	2200.00	4858.00	25	3262.44 (597.17)	2100.00	5000.00
Age at Menarche (yrs)	-	-	-	-	22	13.84 (1.60)	11	16
Height (cms)	46	178.26 (6.74)	162.00	192.00	25	165.40 (6.55)	153.00	177.00
Weight (kgs)	46	77.98 (8.46)	61.50	97.40	25	62.31 (6.60)	46.80	75.10
Relative Leg Length	46	0.49 (0.02)	0.46	0.53	25	0.49 (0.01)	0.46	0.51
Waist-to-Hip Ratio	45	0.84 (0.04)	0.75	0.93	25	0.75 (0.04)	0.68	0.85
Body Fat %	28	17.00 (4.27)	7.00	28.50	22	25.05 (4.61)	16.00	32.90
Lean Mass %	28	20.89 (2.94)	14.58	26.63	21	18.73 (2.56)	13.79	22.92
Average km running distance/week	41	70.17 (28.791)	15	140	22	59.27 (25.759)	30	130

It is important to note that the ultramarathon runners in this study were all highly trained athletes at the time of data collection and were not living sedentary lifestyles by any means. During data collection, participants were asked to self-report their average kilometres of training per week via a questionnaire (Table 1). It is possible that participants exaggerated or misremembered their average weekly training kilometres, but I chose to not exclude the highest training numbers as I was not running statistical analyses on these numbers. They were mostly used as a way to understand the extent to which participants trained each week.

The length and locations of an ultramarathon can vary quite a bit, but on average, they are any distance longer than 42 kilometres and can be as long as 250 to 300 kilometres (Knoth et al. 2012, pg. 1; Gajda, Walasek, & Jarmuszewski 2020, pg. 1-2). While I do not have personal information on the lifestyles of my participants, such as socioeconomic status, race, or ethnicity, Dr. Murray has provided me with information regarding the location, distance, and climate in which my participants competed. The ultramarathons in which my participants took part included: the Rovaniemi150 in Arctic Finland (66-300 kilometres nonstop self-supported on foot, bike, or ski); the Beyond the Ultimate Jungle Ultra in the Peruvian Amazon (230 kilometres over 5 days); the Al Andalus Ultimate Trail in southern Spain (230 kilometres over 5 days); and the Everest Trail Race in the Himalayas of Nepal (170 kilometres over 6 days).

On average, ultramarathon participants are generally from developed nations and from mid-to-high socioeconomic backgrounds (Knoth et al. 2012, pg. 5). This is due to the fact that participants are required to devote dozens of hours a week into training and the race fees can be quite expensive with comparably low dollar amount prizes (Knoth et al. 2012, pg. 5). Furthermore, it is especially expensive for participants if the race takes place in a location outside of their country of residence (Knoth et al. 2012, pg. 5). All of the races in which the participants in my study competed were destination extreme ultras, so entry fees were quite high (several thousand euros in addition to the cost of flights). Some places were reserved for local participants in the Jungle Ultra and the Everest Trail Race, but these individuals did not take part in the study conducted by Dr. Murray and colleagues.

Conceptual Approach (Argument and Hypotheses)

Based on findings of previous research, I hypothesize that the females with lower birth weight will have an earlier age at menarche (Macintosh et al. 2018, pg. 173). This is because females with a lower birth weight must accelerate their pace of growth to ensure they will reach an age in which they may reproduce (Macintosh et al. 2018, pg. 172-173). I also hypothesize that the females with lower birth weight will also have shorter statures and a higher percentage of adiposity (Macintosh et al. 2018, pg. 172-173). I predict that age at menarche will be a strong predictor of body size and composition in females, consistent with the findings of Macintosh and colleagues (2018, pg. 172).

With respect to males, I hypothesize that participants with low birth weight will show similar trends to what are seen in females with low birth weight. Specifically, I hypothesize that they will experience rapid development of adipose tissue and shorter stature due to an earlier cessation of growth (Sandhu et al. 2006, pg. 17). The reason I predict that they will invest more into adipose tissue rather than lean mass, their main secondary sexual tissue, is because low birth weight infants who are well nourished in infancy and childhood will often experience accelerated catch-up growth through a rapid increase of adipose tissue (Wells 2010, pg. 3). In turn, males who have an excess of adipose tissue often experience lower levels of testosterone, particularly during puberty (Novak et al. 2020, pg. 1). This may lead to a decrease in muscle mass, a major component of lean mass, as high muscle mass requires high testosterone levels (Lassek & Gaulin 2009, pg. 326).

Relative leg length is another predictor of adult body size that I hypothesize will have an important impact on my participants. Due to the fact that my participants are highly trained athletes, I hypothesize that relative leg length will be the best indicator of their childhood and adolescent pace of development because this variable may not be as heavily influenced by physical activity as adiposity and lean mass (Bogin & Varela-Silva 2010, pg. 1060).

Additionally, I predict that age at menarche will show very strong correlations with female adult body size and composition. Specifically, I predict that there will be strong correlations with age at menarche and adult stature and adiposity. Finally, I also hypothesize that males will have stronger correlations with lean mass than females in my sample because lean mass is an important secondary sex characteristic in males (Wells 2012, pg. 417). Conversely, I hypothesize that females will show strong correlations with adiposity compared to male participants (Wells 2012, pg. 416-417).

Significance

Previous research has found significant correlations between poor fetal growth, often represented by low birth weight, and disease risk into adulthood (Li et al. 2015, pg. 6; Mahmoodi et al. 2017, pg. 306). Research on developmental outcomes in body size and composition is important because it helps to establish who is at risk of developing various health conditions. For example, type 2 diabetes is a major concern that is linked to low birth weight (Li et al. 2015, pg. 1; Wells et al. 2016, pg. 134). Previous research has found that low birth weight was "associated with glucose intolerance, a lesser insulin secretory capacity, and increased risk of type 2 diabetes" in adults (Li et al. 2015, pg. 1). Furthermore, shorter relative leg length has also been correlated with diabetes (Bogin & Varela-Silva 2010, pg. 1064). This is significant as "[t]ype 2 diabetes is one of the fastest growing disorders worldwide, with the largest increase in incidence in developing countries" (Penno et al. 2013, pg. 177).

Additionally, predicting who may be at risk of certain disorders and diseases may help health care professionals to determine ways to help prevent health conditions before they develop. There is recent evidence that regular physical activity may improve an individual's glucose intolerance, thus lowering their chances of developing type 2 diabetes (Li et al. 2015, pg. 1 & 4). This illustrates the importance of understanding how diseases develop and ways in which an individual can prevent them.

Another major health concern that is correlated with early life development is cardiovascular disease (Fall 2011, pg. 410). Cardiovascular disease includes both heart disease and stroke (Liebert et al. 2013, pg. 228), which Wells and colleagues' paper identify as the leading cause of death worldwide in 2016 (Wells et al. 2016, pg. 134). Fall (2011, pg. 410) states that low birth weight males and females are found to be at an increased risk of mortality due to cardiovascular disease. Additionally, waist-to-hip ratio has been found to be a "predictor for the development of hypertension[,]" a "leading cause of morbidity and mortality from cardiovascular disease" (Kabalin et al. 2012, pg. 363).

Obesity is another health condition that has been linked to various markers of early life development (Wells 2010, pg. 13; Bogin & Varela-Silva 2010, pg. 1064). Low birth weight and earlier age at menarche have both been linked to adulthood obesity (Wells 2010, pg. 13; Amigo et al. 2010, pg. 554). Additionally, shorter relative leg length has also been shown to increase the risk of becoming overweight (Bogin & Varela-Silva 2010, pg. 1064). Henneberg and Grantham (2013, pg. 1-2) state that while obesity on its own does not cause premature death, it has been linked to an increased risk of "hypertension, cardiovascular disease, hyperglycaemia and dyslipidaemia as well as a tendency towards cancer." Obesity is also linked to type 2 diabetes (Logue et al. 2011, pg. 3003).

Previous research has shown that metabolic diseases, such as type 2 diabetes, can develop differently in males and females (Paul et al 2012, pg. 1556). For example, Logue and colleagues (2011, pg. 3006) found that males of all ages are "diagnosed with diabetes at a lower BMI [body mass index] than women" which may explain why European males are diagnosed with type 2 diabetes at a higher rate than females of the same population. Males and females invest in their body composition differently and, thus, it is important to look at both males and females separately to recognize how metabolic diseases develop (Wells 2012, pg. 417).

Understanding how socioeconomic inequalities affect health brings insight to how healthcare professionals can anticipate health outcomes in various individuals. As indicated by the Social Determinants of Health, individuals of a lower socioeconomic status are predisposed to many health conditions such as cardiovascular diseases and type 2 diabetes (Braveman & Gottlieb 2014, pg. 129; Meier 2009, pg. 1255). Moreover, previous research has shown that lower socioeconomic status and lower levels of education in the United States was correlated with low birth weight, especially among racial minority groups (Martinson & Reichman 2016, pg. 751). There is evidence, however, that accessible and quality prenatal health care, may improve birth weight outcomes (Martinson & Reichman 2016, pg. 753-754). Furthermore, if low birth weight is associated with shorter stature and higher percentage of adiposity, then individuals born weighing 2,500 grams or less may benefit from being educated about preventative activities such as regular physical activity to lower their risk of developing many chronic diseases (Li et al. 2015, pg. 1 & 4).

For my research, I examined if low birth weight male participants experience the same accelerated growth patterns, such as high adiposity and short stature, that low birth weight females have in previous studies (Macintosh et al. 2018, pg. 177). I chose to include males because, as far as I am aware, there seems to be lacking data on male energy distribution under the life history model.

Methods

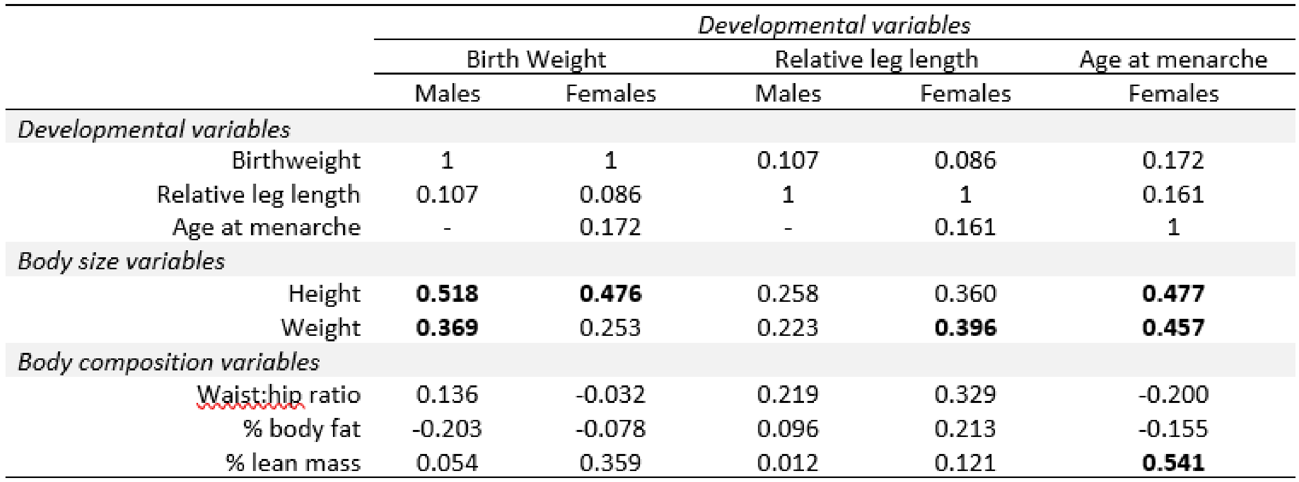
As described above, my working dataset totaled 72 participants (47 males and 25 females), which I coded in Statistical Package for the Social Sciences (SPSS) as “1” (males) and “2” (females). I first tested the variables in my dataset for outliers by running normality tests. To do this, I split my file by sex to run tests for males and females separately. I tested each variable individually (height, weight, birth weight, etc.) and examined their individual summaries (e.g., standard deviation, range, averages). In the summaries, I looked for various indicators that could tell me how the data was distributed. First, I calculated the skewness by dividing “statistic” by the “Std. Error.” If the number was between -3.29 and +3.29, I concluded that that trait was not skewed (Field 2009, pg. 26).

To further test the normality, I used a Kolmogorov-Smirnov test and looked at the probability value (p value) to assess whether the sample reflected a normal population distribution (Field 2009, pg. 548). A p value greater than 0.05 indicates that the data are not normally distributed when compared “to a normally distributed set of scores with the same mean and standard deviation” (Field 2009, pg. 144). Thus, if I found that the p value was less than 0.05, I concluded that the data was normally distributed. I also looked at each trait as a scatter plot and a box plot to visualize how my data were distributed. There was one male participant, ID #17, whose traits were not normally distributed in my normality tests. I chose to exclude this individual from further analysis because I could not be certain that there was not a mistake in the data collection. Therefore, my subsequent analyses were conducted with 71 participants (46 males and 25 females).

My next step was to compute relative leg length and waist-to-hip ratio. For relative leg length, I divided leg length (cm) by height (cm). For the waist-to-hip ratio, I divided waist circumference (cm) by hip circumference (cm). I looked at the waist-to-hip ratio in addition to the other components of body size and composition because it is generally a good marker of "central fat distribution" (Samanta et al. 2019, pg. 107).

After completing my normality testing and ensuring my data was distributed properly, I ran my partial Pearson's correlation analyses. Results for all partial Pearson's correlation analyses are presented in Table 2. I controlled for age because body composition normally changes with age and I wanted to examine how each of the variables related to one another without the influence of age. Partial Pearson's correlation analyses are intended to tell me how one of my selected variables relates to my other selected variables (Field 2009, pg. 167). Pearson's correlations show whether two components are positively correlated, not correlated at all, or negatively correlated (Field 2009 pg. 167). If they are positively correlated, as one value increases or decreases, the other value does as well (Field 2009, pg. 167). If they are not correlated, there is no relationship between the values; as one value increases or decreases, the constant stays the same (Field 2009, pg. 167). If they are negatively correlated, as one value decreases the other value increases (Field 2009, pg. 167). Significant p values (values between 0.00-<0.05, with values closer to 0.00 showing greater significance) are bolded in Tables 2. The p value, or probability, is the odds of a correlation being by chance (Field 2009, pg. 50). If the $p < 0.05$, then "there is only a 5% chance of something occurring by chance [and] we can accept that it is a genuine effect" (Field 2009, pg. 50). Thus, the closer the p value is to 0.00, the more significant it is (Field 2009, pg. 50-51). The p values that are deemed significant are bolded on my graphs. The developmental variables I used were birth weight (g), relative leg length (cms), and age at menarche (yrs) (in females only). The body size variables I used were height (cms) and weight (kgs). The body composition variables I used were waist-to-hip ratio, percentage of body fat, and percentage of lean mass.

Table 2. Partial correlation coefficients for relationships between developmental proxies and adult body size and body composition variables, after controlling for age.



	Developmental variables				
	Birth Weight		Relative leg length		Age at menarche
	Males	Females	Males	Females	Females
Developmental variables					
Birthweight	1	1	0.107	0.086	0.172
Relative leg length	0.107	0.086	1	1	0.161
Age at menarche	-	0.172	-	0.161	1
Body size variables					
Height	**0.518**	**0.476**	0.258	0.360	**0.477**
Weight	**0.369**	0.253	0.223	**0.396**	**0.457**
Body composition variables					
Waist:hip ratio	0.136	-0.032	0.219	0.329	-0.200
% body fat	-0.203	-0.078	0.096	0.213	-0.155
% lean mass	0.054	0.359	0.012	0.121	**0.541**

Significant partial correlations indicated in bold.

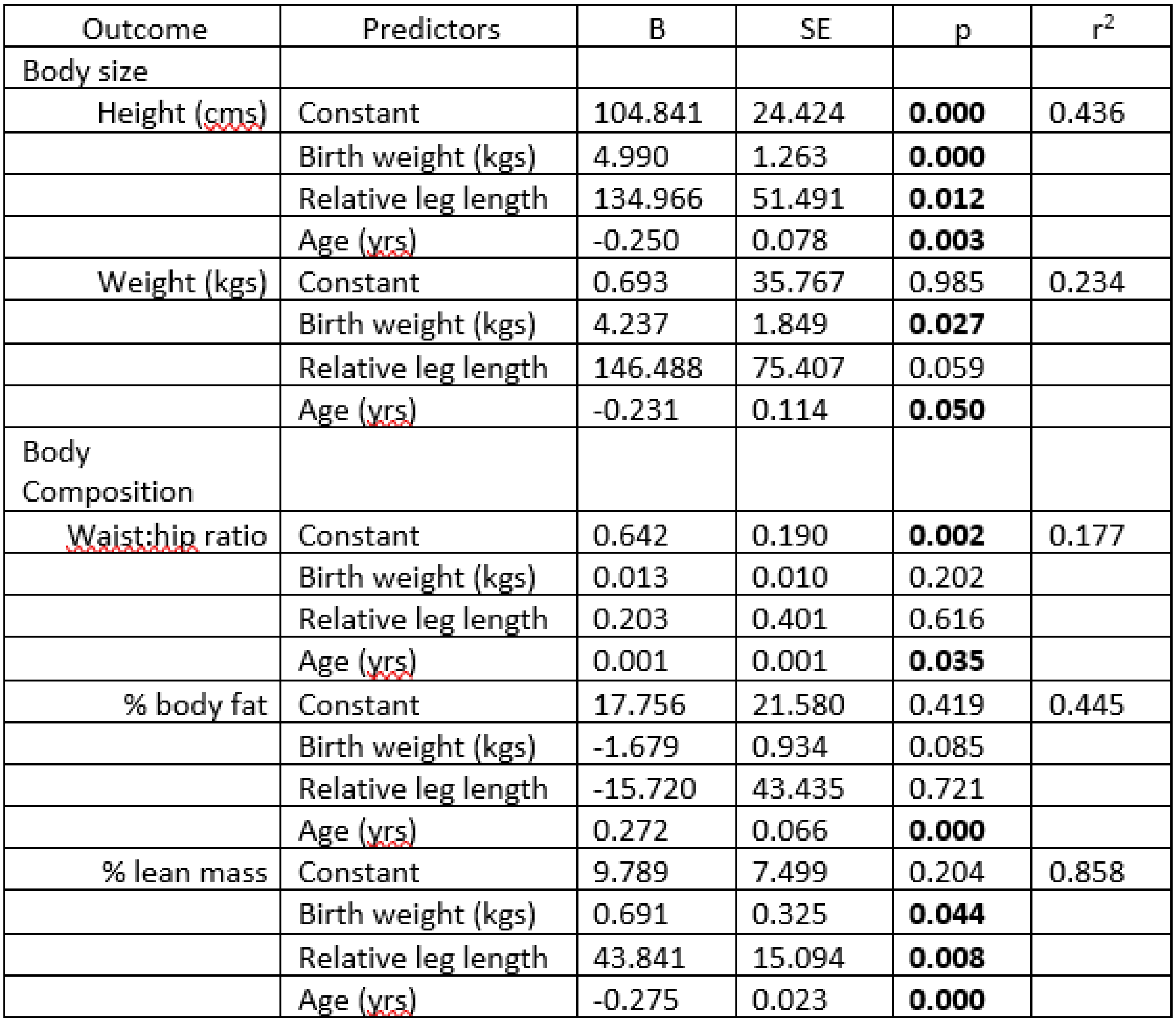
Next, I ran linear regression analyses on males and females separately (Tables 3 and 4). Before I discuss my findings, I first define some of the terms used in my charts. The B-value shows how the outcomes (body size and composition) and the predictors (early markers of development) are related to each other (Field 2009, pg. 238). The SE-value is the standard error of the mean. This value shows “how much variability there is in this statistic across samples from the same population” (Field 2009 pg. 794). When there are larger SE values, this indicates that the population may not be completely accurately represented by that statistic (Field 2009, pg. 794). The r^2-value is also known as the coefficient of determination (Field 2009, pg. 179). It “is a measure of the amount of variability in one variable that is shared by the other” (Field 2009, pg. 179).

Table 3. Developmental and age-related predictors of adult female body size and composition.

Outcome	Predictors	B	SE	p	r^2
Body size					
Height (cms)	Constant	94.974	42.078	**0.037**	0.450
	Birth weight (kgs)	3.745	1.956	0.073	
	Age at Menarche (yrs)	1.541	0.739	0.052	
	Relative leg length	69.740	88.572	0.442	
	Age (yrs)	0.077	0.115	0.510	
Weight (kgs)	Constant	-33.711	46.825	0.481	0.403
	Birth weight (kgs)	0.632	2.177	0.775	
	Age at Menarche (yrs)	1.706	0.822	0.054	
	Relative leg length	128.291	98.564	0.210	
	Age (yrs)	0.179	0.128	0.179	
Body Composition					
Waist:hip ratio	Constant	0.111	0.297	0.714	0.362
	Birth weight (kgs)	-0.007	0.014	0.633	
	Age at Menarche (yrs)	-0.007	0.005	0.205	
	Relative leg length	1.456	0.626	**0.033**	
	Age (yrs)	0.001	0.001	0.239	
% body fat	Constant	-21.588	32.152	0.512	0.402
	Birth weight (kgs)	-1.819	1.451	0.229	
	Age at Menarche (yrs)	-0.304	0.533	0.577	
	Relative leg length	95.825	66.444	0.170	
	Age (yrs)	0.227	0.088	**0.021**	
% lean mass	Constant	-0.335	13.224	0.980	0.726
	Birth weight (kgs)	2.277	0.641	**0.003**	
	Age at Menarche (yrs)	0.590	0.220	**0.018**	
	Relative leg length	21.789	27.329	0.439	
	Age (yrs)	-0.166	0.038	**0.001**	

Birth weight converted to kgs in order to increase coefficient size and improve ease of interpretation. Birth weight was converted to kilograms for regression analyses in order to increase the size of the coefficients for easier interpretation; P values in bold indicate significant contribution to predictions at an alpha of <0.05.

Table 4. Developmental and age-related predictors of adult male body size and composition



Outcome	Predictors	B	SE	p	r^2
Body size					
Height (cms)	Constant	104.841	24.424	**0.000**	0.436
	Birth weight (kgs)	4.990	1.263	**0.000**	
	Relative leg length	134.966	51.491	**0.012**	
	Age (yrs)	-0.250	0.078	**0.003**	
Weight (kgs)	Constant	0.693	35.767	0.985	0.234
	Birth weight (kgs)	4.237	1.849	**0.027**	
	Relative leg length	146.488	75.407	0.059	
	Age (yrs)	-0.231	0.114	**0.050**	
Body Composition					
Waist:hip ratio	Constant	0.642	0.190	**0.002**	0.177
	Birth weight (kgs)	0.013	0.010	0.202	
	Relative leg length	0.203	0.401	0.616	
	Age (yrs)	0.001	0.001	**0.035**	
% body fat	Constant	17.756	21.580	0.419	0.445
	Birth weight (kgs)	-1.679	0.934	0.085	
	Relative leg length	-15.720	43.435	0.721	
	Age (yrs)	0.272	0.066	**0.000**	
% lean mass	Constant	9.789	7.499	0.204	0.858
	Birth weight (kgs)	0.691	0.325	**0.044**	
	Relative leg length	43.841	15.094	**0.008**	
	Age (yrs)	-0.275	0.023	**0.000**	

Birth weight converted to kgs in order to increase coefficient size and improve ease of interpretation. Birth weight was converted to kilograms for regression analyses in order to increase the size of the coefficients for easier interpretation; P values in bold indicate significant contribution to predictions at an alpha of <0.05.

I chose linear regression as it would allow me to see how well my developmental variables and age would predict features of adult body size and composition. For males, the developmental and age-related predictors I used were birth weight, converted from grams to kilograms to increase the size of the coefficients for easier interpretation; relative leg length; and age. The elements of body size and composition that I used were height (cms), weight (kgs), waist-to-hip ratio, percent body fat, and percent lean mass. I used the same components for females, with age at menarche added to the developmental predictors. To determine what traits are significant, I looked at the p values. Any correlations that were less than 0.05 were considered to be significant.

In addition to measurements of body size and composition, I also examined data on the average kilometres per week each of the participants trained. This allowed me to have an idea of how much training they are doing on a weekly basis.

For my body composition statistics, I was limited by the fact that a significant portion of my male and female data was not recorded. Specifically, I only had data from 28 male participants for percentage of adipose tissue and lean mass, and data on 22 and 21 females for percentage of body fat and lean mass, respectively. This limited my findings as I had an even smaller sample to work with.

Results

Table 1 summarizes the descriptive statistics for age, markers of early life development (birth weight, age at menarche, relative leg length), body size and composition variables, and average kilometres per week spent training for both males and females for the analyzed samples. Table 2 shows the partial Pearson's correlation coefficients for relationships between developmental proxies and adult body size and composition variables. For this analysis, I controlled for age. I did this so I could look at how my variables correlate with one another without the influence of age. This is due to the fact that age has consistently been shown to increase adiposity and decrease lean mass in both males and females (Koster et al. 2011, pg. 889). There were no significant correlations between any of the developmental variables in the males or females (for example, birth weight was not correlated with age at menarche). In males, birth weight was significantly correlated with adult height and weight, indicated as Figures 1 and 2 respectively. In males, relative leg length was not significantly correlated with any of the body size and composition variables.

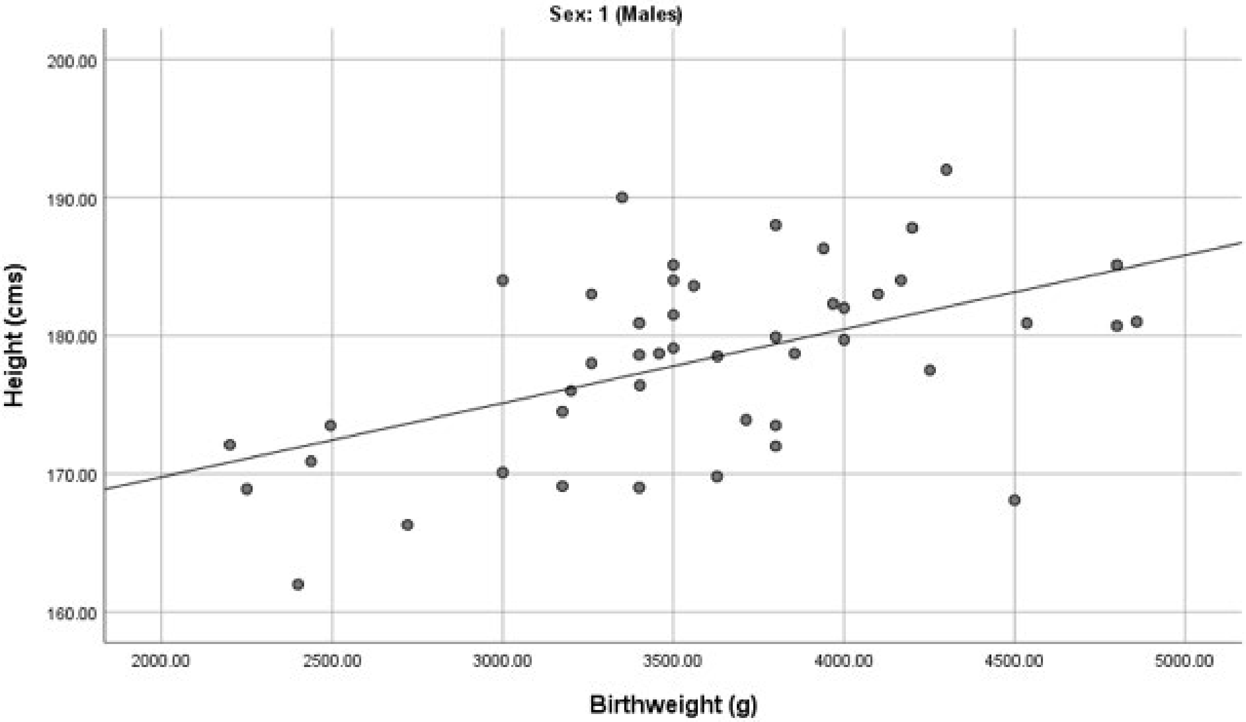


Figure 1. Scatter plot indicating positive correlation between birthweight (g) and height (cms) in males. Cut off for significance is an alpha of 0.05.

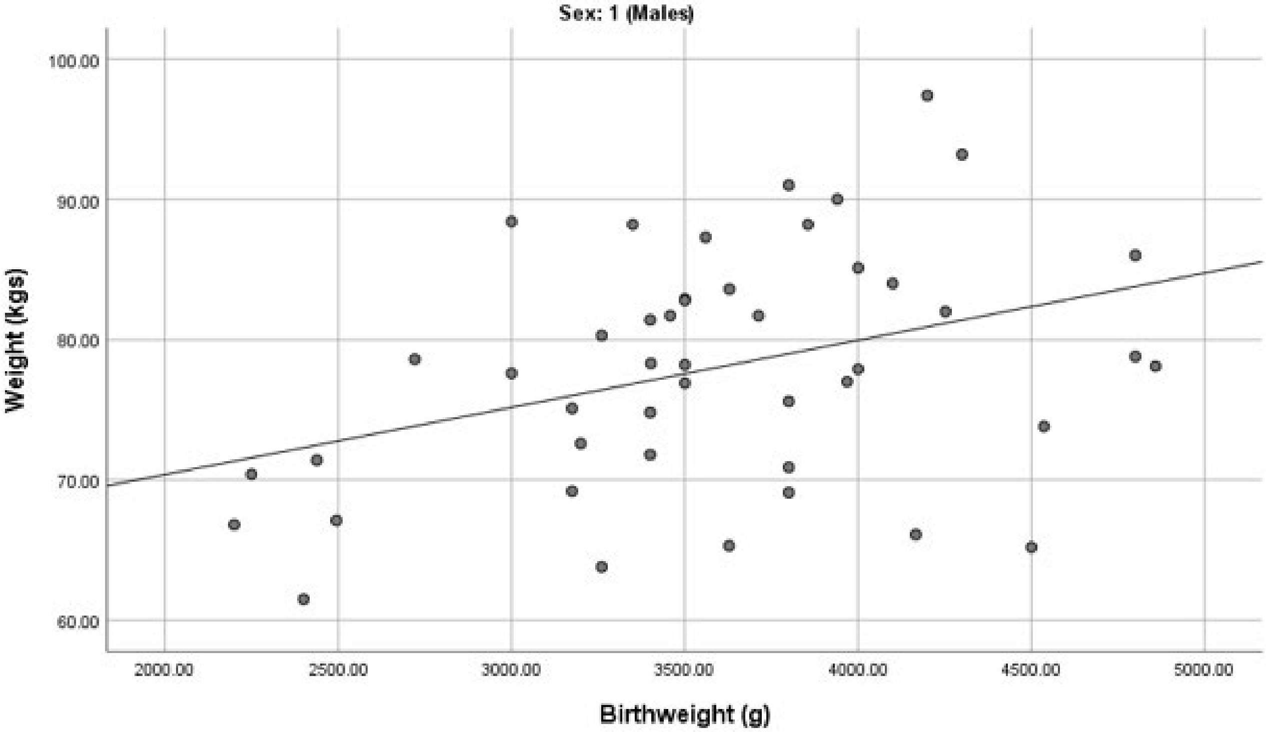


Figure 2. Scatter plot indicating positive correlation between birthweight (g) and weight (kgs) in males. Cut off for significance is an alpha of 0.05.

In females, however, birth weight was only significantly correlated with adult height, as indicated in Figure 3. In females, relative leg length was significantly correlated with weight but not height or any body composition variables (Figure 4). Age at menarche showed the highest number of significant correlations among the variables with adult height, weight, and percentage of lean mass in the female participants, indicated in Figures 5, 6, and 7, respectively.

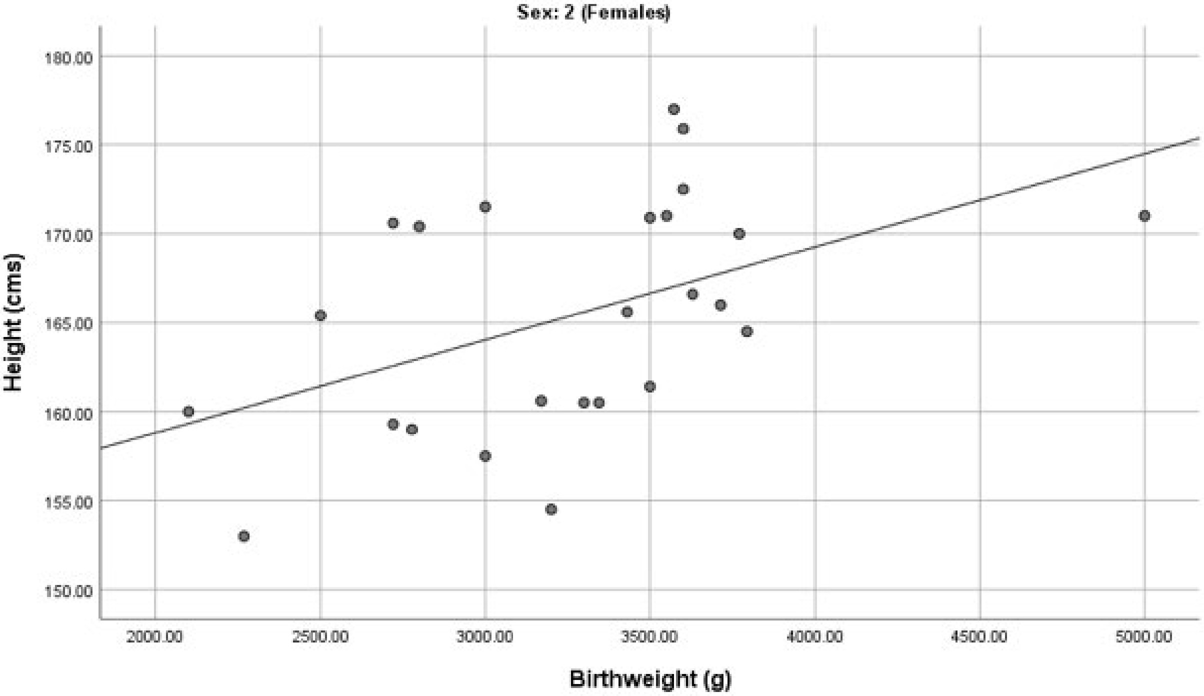


Figure 3. Scatter plot indicating positive correlation between birthweight (g) and height (cms) in females. Cut off for significance is an alpha of 0.05.

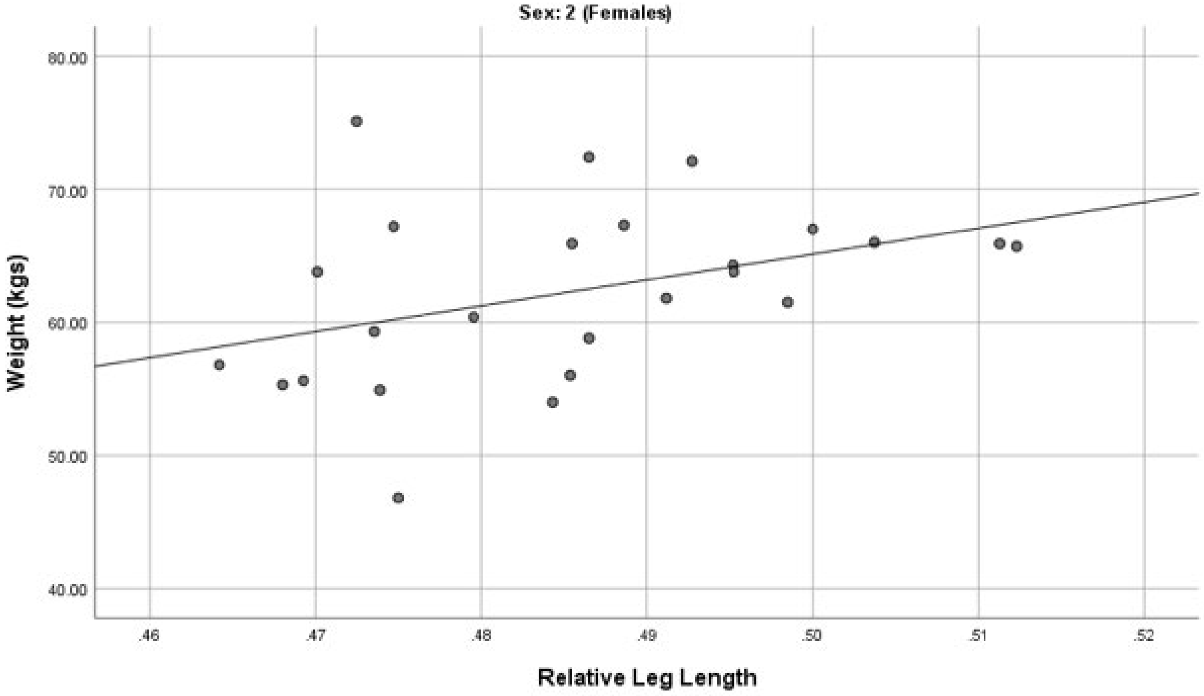


Figure 4. Scatter plot indicating positive correlation between relative leg length and weight (kgs) in females. Cut off for significance is an alpha of 0.05.

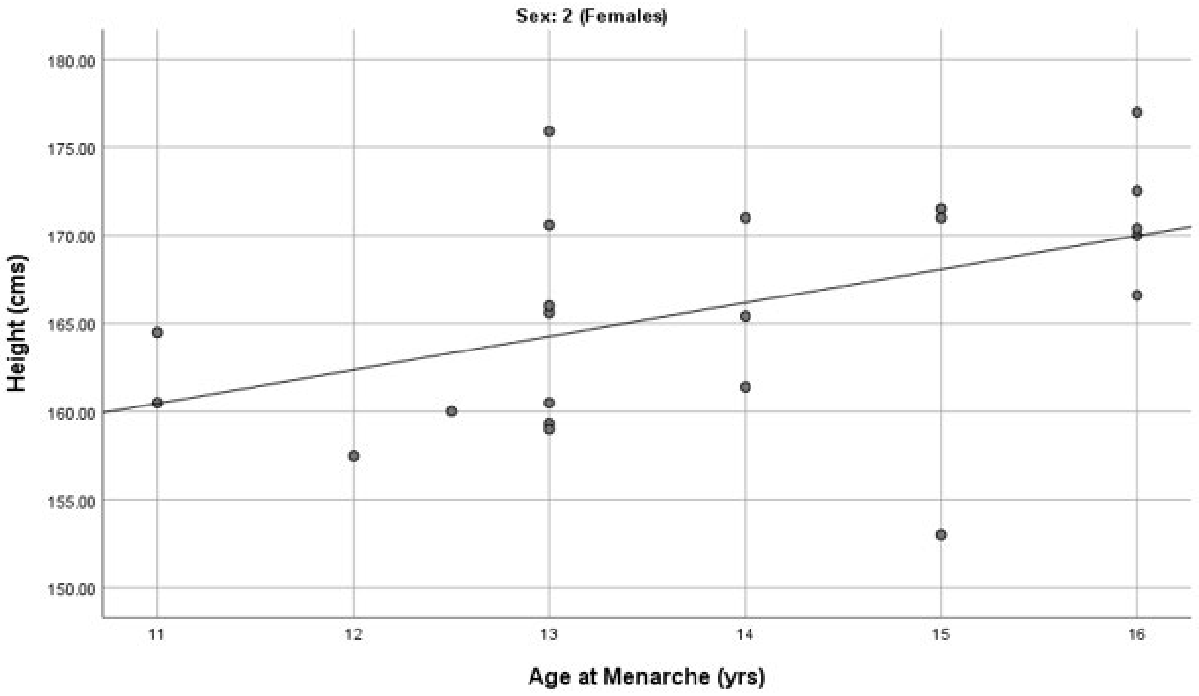


Figure 5. Scatter plot indicating positive correlation between age at menarche (yrs) and height (cms) in females. Cut off for significance is an alpha of 0.05.

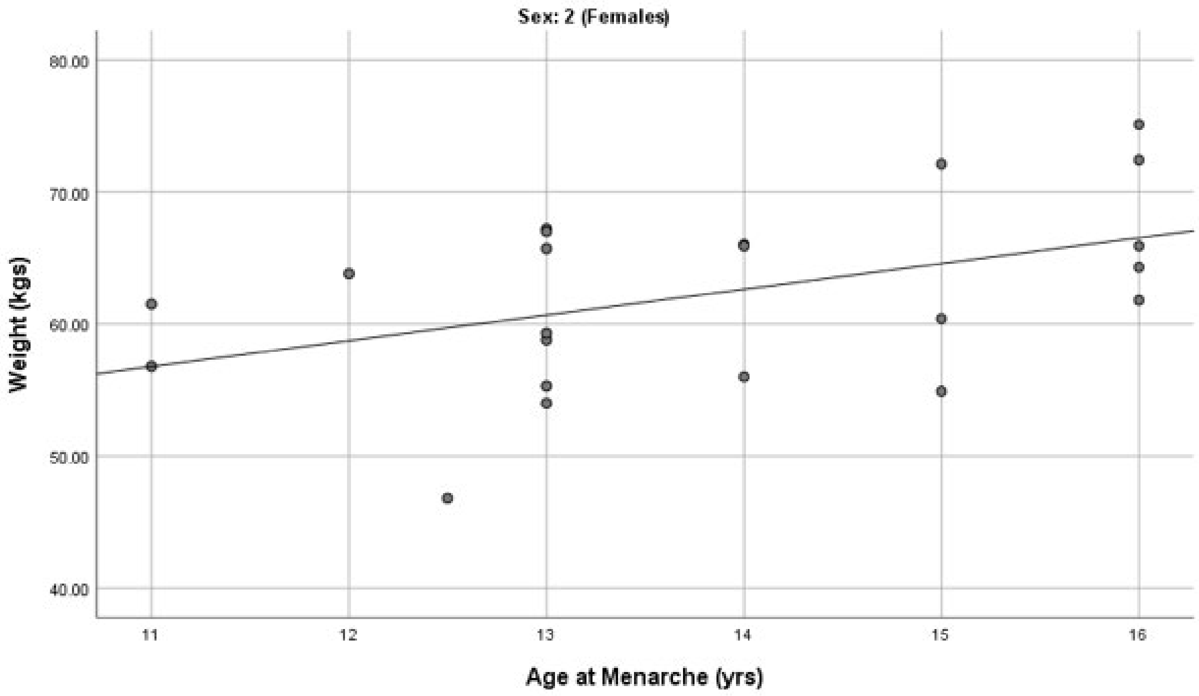


Figure 6. Scatter plot indicating positive correlation between age at menarche (yrs) and weight (kgs) in females. Cut off for significance is an alpha of 0.05.

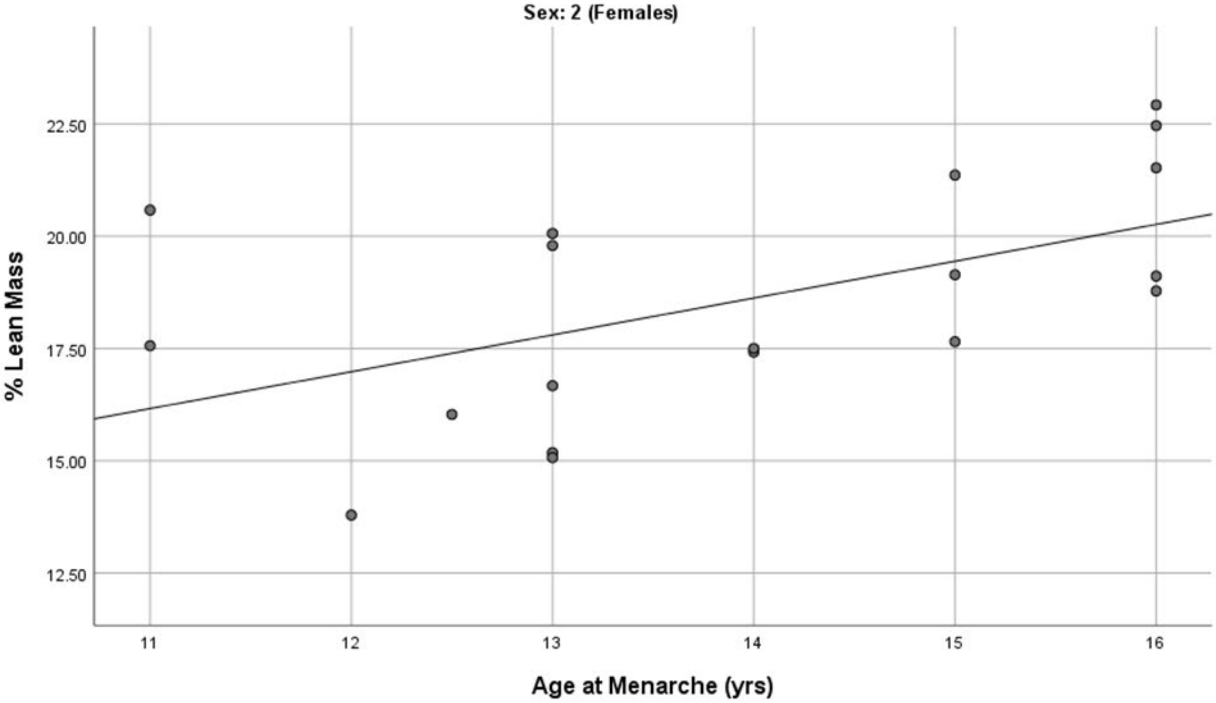


Figure 7. Scatter plot indicating positive correlation between age at menarche (yrs) and percentage of lean mass in females. Cut off for significance is an alpha of 0.05.

Linear regression analyses of males in my sample, show that birth weight was a significant predictor of adult height, adult weight, and adult percentage of lean mass. I also found that relative leg length was a significant predictor of adult height and percentage of lean mass. Additionally, age at the time of data collection was significantly correlated with adult height, weight, waist-to-hip ratio, percentage of body fat, and percentage of lean mass.

Linear regression analyses of female data, showed that relative leg length was a significant predictor of the waist-to-hip ratio. I also found that age was a significant predictor of percentage of body fat. Additionally, both birth weight, age at menarche, and age at the time of data collection were significant predictors of percentage of lean mass.

Discussion

This study anticipated finding significant correlations between of markers of early life development (birth weight, relative leg length, and age at menarche) and adult body size (weight and height) and composition (percentage of body fat and lean mass) variables in males and females. My results show that birth weight showed the most significant positive correlations with adult body size in males. This indicates that among males bigger babies became bigger adults, in both stature and weight. This suggests that the bigger birth weight babies had a slower pace of growth and development. I also found that birth weight was significantly correlated with height in females, indicating that bigger female babies became taller females. This partially aligns with previous research that states that bigger birth weight babies will exhibit a slow pace of growth (Macintosh et al., pg. 173), though it also suggests that there is plasticity with adult female weight potentially due to environmental factors like amount of regular physical activity.

My results also show that among females age at menarche had the most significant correlations, particularly with height, weight, and percentage of lean mass. These results suggest that the pace of growth and childhood environmental conditions (proxied by age at menarche) have a greater influence on adult height and weight than birth weight in the females. Interestingly, percentage of lean mass was positively correlated with age at menarche. This differed from initial hypothesis where I predicted that age at menarche would be correlated with adiposity. I suggest that the reason for this is that my participants were athletically training intensively at the time of data collection. The female participants were highly active and, on average, were training 59.27 kilometres per week. Some recorded training as few as 30 kilometres per week and others as many as 130 kilometres. Furthermore, I suggest that the positive correlation of age at menarche and weight reflects the fact that lean mass is more dense than adipose tissue and, thus, their weight may be increased due to their dense lean mass (Erlandson et al. 2017, pg. 1831).

For relative leg length in my partial correlation analyses, I found that only female weight was significantly correlated and that there were no correlations in the males. I found this to be very surprising, as research has shown that relative leg length is very strongly correlated with the pace of growth of an individual (Bogin & Varela-Silva 2010, pg. 1060). My results suggest that relative leg length was not a reliable indicator of the pace of growth of my male participants. It also indicates that the females with longer legs were heavier, though this could be attributed to their high prevalence of lean mass.

For males, my partial correlation analyses showed that age was negatively correlated with lean mass, indicating that as males age, they lose lean mass. Similarly, adiposity and lean mass were negatively correlated, indicating that as males increase in adipose tissue, they lose lean mass. I observed the same trend with adiposity and lean mass among females, for whom they exhibit a negative correlation. For age and dry lean, their p value was 0.051 and the correlation value was negative. What this means is that there was a 5.1% chance that age and dry lean (fat free mass minus water) were significantly correlated. While that p value is not technically statistically significant, I would argue that it is borderline significant and worthy of note. With a larger sample size of female participants, this value may have been more significant. These trends were something I had hypothesized that I would see, as aging individuals, both male and female, generally increase in percentage of adiposity as they decrease in percentage of lean mass (Koster et al. 2011, pg. 889).

I also wanted to look at how early markers of development were predictive of adult body size and composition. Specifically, I wanted to see not only how they were correlated, but how one variable was predicting another. In my linear regression analyses of male participants, I found that birth weight was a significant predictor of height, weight, and percentage of lean mass. Similar to the correlation analyses, this means that larger male babies will likely grow into larger adults in body size and will also invest more into lean mass than adiposity. This was an outcome I hypothesized as larger birth weight male babies generally are able to invest more into lean mass than low birth weight babies who invest into catch-up growth (Wells 2010, pg. 3). Because they likely do not experience an excess of adiposity in childhood, they will likely have greater levels of testosterone prior to puberty and, thus, invest more into lean mass (Novak et al. 2010, pg. 1; Lassek & Gaulin 2009, pg. 326). However, I argue that, similar to the female participants, physical activity may have increased the percentage of lean mass in my participants beyond what is normally observed since the male participants on average were training 70.17 kilometres per week. Some reported some training as few as 15 kilometres per week and others reported as many as 140 kilometres.

Relative leg length was also a significant predictor of height and percentage of lean mass in males. This indicates that participants who had longer legs were overall taller in stature. It also indicates that the participants with longer relative leg length were also leaner. This may be due to the fact that participants with longer relative leg length may have experienced slower life history strategies and invested less into adipose tissue development and more into lean mass. This may also reflect the fact that males generally invest more into lean mass, their major secondary sexual characteristic (Wells 2012, pg. 417). Thus, as per my third research question, it appears that the males of my study invested more into lean mass than adipose tissue.

Age was a significant predictor of height, weight, waist-to-hip ratio, percentage of body fat, and percentage of lean mass in males. Table 3 illustrates that as one ages, they decrease in height, weight, and percentage of lean mass. Adversely, as they age, they are increasing their waist-to-hip ratio and their percentage of body fat. This illustrates the complex effect of age on male body size and composition. This aligns with research that states that as they age, males begin to decrease in lean mass, increase in adipose tissue, and decrease in overall height (Koster et al. 2011, pg. 889).

For my female participants, my linear regression analyses revealed some interesting patterns. Birth weight and age at menarche were predictive variables of percentage of lean mass, rather than adiposity as I had initially hypothesized. I suggest that the reason for this may be because the participants were investing more into lean mass than adiposity due to their intensive physical training. The participants exhibited levels of lean mass that would likely differ quite a lot from recreationally active populations. Relative leg length was predictive of waist-to-hip ratio. This indicates that the participants with longer legs had a greater waist-to-hip ratio, although this could also reflect how waist-to-hip ratio generally increases as an individual ages (Cashdan 2008, pg. 1099).

Age was also found to be predictive of percentage of adiposity and percentage of lean mass. The B-value shows that adiposity was positive and lean mass was negative, indicating that as females age, similarly to males, they increase in adiposity and decrease in lean mass.

To look further at this relationship, I created a scatter plot to compare the average number of weekly kilometres in training with the percentage of lean mass of both males and females. Overall, I found that the plots did not show clear trends between the training averages and lean mass, though I did not run any statistical analyses on this data. Looking at the relationship between hours spent training and body compositional outcomes may be useful for future research.

These findings suggest that our start in life does not always predict our adult body size and composition. The activities that an individual does and the lifestyle they live may still have a significant impact on how their body grows and develops. For example, adiposity is generally associated with a female's birth weight and age at menarche (Macintosh et al. 2018, pg. 172-173). For my research, I found that lean mass was associated with age at menarche. This suggests that the female participants in my sample invested more into lean mass than into adipose tissue, likely in response to their physical activity.

I hypothesize that the main reason that my participants showed many trends that were not in line with previous research is due to the fact that they were all ultramarathon runners at the time of data collection. My male and female participants were training, on average 70.17 and 59.27 kilometres per week, respectively, which is an intensive training regimen. Due to previous research, I had predicted that females would still have a strong correlation with adipose tissue as they generally invest more into adiposity than males do (Wells 2012, pg. 417). This indicates that one's early life development does not always have an ultimate say on their adult body size and composition and that the activities they do later in life can still have a major impact on their bodies. This illustrates that even individuals with poor early life growth (specifically low birth weight) can still maintain a healthy lifestyle.

I suggest that there may be gendered differences in why the males were training, on average, over 10 kilometres more per week than the females. This may be due to the fact that my female participant sample is smaller than my male participant sample. However, it could also suggest that there were fewer females registering in ultramarathon races. There are a variety of social reasons for this. Firstly, muscularity in women is not a cultural norm in Western countries (Krane et al. 2004, pg. 316). There is a perception that being too toned and muscular is too masculine, and many women feel pressure to maintain a stereotypically feminine figure (Krane et al. 2004, pg. 317). Furthermore, there are additional stereotypes placed on female athletes who are also mothers. There is a societal expectation that a mother will selflessly devote their lives to their families. As discussed by McGannon and colleagues (2015, pg. 52), there are greater expectations placed on females who choose to divide their time between family and training and many females who have children are made to feel a sense of guilt for prioritizing athletic training.

While I did not have access to the participants' socioeconomic information, I would suggest that, due to the expense of the race(s) and travel in addition to the extensive hours spent training, the participants were likely of a mid-to-high socioeconomic bracket. Additionally, in order to maintain their health throughout their training, it is likely that the participants were conscious of their nutritional intake. Additionally, to meet the metabolic energy demands of ultramarathon running, they likely increased their nutrient intake substantially (Wardenaar et al. 2015, pg. 375). This also indicates that they may have access to healthier foods and, thus, may have better health outcomes because of this although there is some research that suggests ultramarathon competitions may lead to gastrointestinal (GI) distress (Wardenaar et al. 2015, pg. 375).

In addition to lack of information on socioeconomic status, I did not have access to health outcomes of my participants. However, I would suggest that, due to their intensive training regimen over the course of months and years, participants would be at a decreased risk of developing type 2 diabetes, regardless of their birth weight. Previous research has shown that regular physical activity has been shown to decrease the risk of type 2 diabetes in adults with low birth weight (Li et al. 2015, pg. 1 & 4). Additionally, due to their regular physical activity, none of my participants showed an excess of adiposity and certainly were not close to being in the obese range. Higher lean mass and lower adiposity would also likely lower the risk of cardiovascular and diabetic conditions related to obesity (Logue et al. 2011, pg. 3003; Wells 2010, pg. 2).

Conclusion

Our early life development can impact the way we grow and develop into adults. Birth weight was significantly correlated with adult body size in my male sample, indicating that bigger babies will grow into bigger adults. This supports previous findings that state that babies born at a weight greater than 2,500 grams will be leaner and experience a slower pace of growth and development (Wells 2010, pg. 2).

While previous research has indicated that these markers (birth weight, age at menarche, and relative leg length) show strong correlations with our adult body composition (Bogin & Varela-Silva 2010, pg. 1061; Macintosh et al. 2018, pg. 168; Wells 2010, pg. 9), my research suggests that external factors – such as physical activity – may still have an important influence on our adult bodies, despite our start in life. Specifically, my findings suggest that there is some degree of plasticity with how the human body grows and develops in relation to our external environments and activities.

My findings also suggest that ultramarathon running requires intensive training that influences female bodies in ways that are not commonly observed in recreationally active populations. For instance, they exhibited significant allocation of energy to lean mass. There were no significant correlations between adiposity and low birth weight, despite there being many findings by previous researchers who support this.

Body size and composition can also have major implications for health outcomes for an individual. Excess adiposity and shorter leg length can lead to obesity, type 2 diabetes, cardiovascular disease, and many other metabolic-related diseases (Wells 2010, pg. 13; Bogin & Varela-Silva 2010, pg. 1064; Logue et al. 2011, pg. 3003; Henneberg and Grantham 2013, pg. 1-2). Understanding how markers of early life development, such as low birth weight, can impact an individuals' growth and development into adulthood is important for developing systems of adequate preventative care for individuals at an increased risk of metabolic and cardiovascular diseases. Furthermore, understanding the varying ways in which males and females allocate energy can also aid in the understanding of preventative actions. Additionally, understanding the barriers that an individual faces in receiving health care due to socioeconomic inequalities will also help to reduce the occurrence of these diseases while improving the quality of life for individuals all over the world.

Limitations

Throughout my research process, I encountered many limitations. My primary limitation was that the dataset I used was collected for a purpose other than my research. Thus, many of the factors that I would have liked to explore in depth, such as the influence of socioeconomic status and timing of pubertal development in males, were not possible. Another limitation I faced was the small sample size of my participants. The conclusions I could draw from my data were limited by the fact that I simply did not have enough people for both males and females. Body composition data was not recorded for a number of participants, furthering the correlations and predictions I could find.

The specificity of the population I was studying was another limitation. I hypothesized that despite their high level of physical training, I would still see similar trends to previous research. I found, however, that physical activity had more of an impact than I had predicted. I feel my research would have benefited from including individuals who were recreationally active as opposed to the highly active participants on which my study was based.

Another major limitation I faced was not having access to any personal records of my participants. Had I collected the data myself for the purpose of this study, I would have asked questions about socioeconomic status, childhood environmental and developmental conditions, mother's health and socioeconomic status, race, ethnicity, country of origin and country of residence, weeks of gestation, reproductive success, health history. This information would have allowed me to hypothesize about how socioeconomic status in childhood and adulthood may impact health outcomes.

Additionally, another limitation is that my participants were asked to self report their birth weights and age at menarche. While they were given their questionnaires prior to the start of the study to ensure they had time to retrieve personal records such as their birth certificates, there is always the possibility that recall may be flawed.

Finally, a major limitation that impacted my ability to compare males and females completely was the absence of data on the timing of pubertal development in my male participants. For males, there is not often a clear marker of when puberty begins that is easy to recall in adulthood. Therefore, I was unable to use a proxy for age at menarche for my male participants.

Future Research

Future research should include a significantly larger and more diverse sample. Ideally, it would include a significant number of individuals who are recreationally active. With individuals who are highly active, it is arguable that a lot of the outcomes are impacted by their high levels of physical activity. Thus, having a recreationally active sample would allow for the researcher to analyze body size and compositional outcomes without the influence of intensive athletic training.

Further research would also benefit from examining the fetal and infant developmental conditions of participants. For example, how many weeks of gestation did they attain? This would be useful information as preterm births (<37 weeks gestation) are heavily correlated with low birth weight (Kim & Saada 2013, pg. 2298). This would also allow for implications to be made about the fetal development conditions in utero. Information on reproductive success for both the male and female participants may have also been useful especially when examining adiposity and lean mass percentages.

Additionally, future research could look into the impact of intensive physical activity on body composition. My females showed trends that did not align with what is commonly predicted by the life history model. This suggests that they were investing more into lean mass than adipose tissue. Further research could look into why some ultra-athletic females prioritize lean mass over reproductive tissues. Furthermore, it may be useful to examine the parity of the females to see if the amount of adipose tissue that they do have can be correlated with the number of births they have had.

Future research should also consider the impact of socioeconomic status on body size and composition outcomes. With the sample I used for this study, it is likely (though not proven) that many, if not all, of my participants were from a mid-to-high socioeconomic status. This is based on the fact that ultramarathons are generally not inexpensive activities. My research would have benefitted from having participants of various socioeconomic backgrounds, so that trends in relation to the Social Determinants of Health could be better observed.

Overall, a longitudinal study wherein participants are observed over a longer period of time for infant development, pubertal timing, and adult body composition and health outcomes would overall increase the understanding of how early life development may affect an individual's development and risk for metabolic diseases such as cardiovascular diseases and type 2 diabetes.

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