What makes girls participate in sport? An analysis of biological correlates of sport participation.

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What makes girls participate in sport? An analysis of biological correlates of sport participation.

by

Elizabeth Vandenborn

A Thesis
Submitted to the Faculty of Graduate Studies through the Department of Kinesiology in Partial Fulfillment of the Requirements for the Degree of Master of Human Kinetics at the University of Windsor

Windsor, Ontario, Canada

2017

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What makes girls participate in sport? An analysis of biological correlates of sport participation.

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ABSTRACT

Introduction: Females are currently participating in sport at a lower rate than males. It has been determined that girls who participate in sport gain many advantages (i.e. better bone health, greater cardio-respiratory fitness, a better quality of life). Therefore, it’s important to determine why some females choose to continue sport participation, while others do not. Objective: To determine if 2DR and salivary testosterone (sT) are correlates of sport participation. Methods: A cross-sectional analysis of resting and indirect prenatal androgen concentrations (i.e. second to fourth digit ratios) were obtained from a sample of 18-30y females. Participant demographic (via questionnaire), anthropometric, behavioural (via questionnaire), and retrospective sport participation (via questionnaire) information were collected on one occasion and saliva was collected on two occasions. Results: 2DR ratio ($r = -0.650, p = 0.538$) was not significantly correlated with total sport participation, nor was sT ($r = 0.094, p = 0.387$). Secondary analysis revealed significant correlations between sport participation and max hand grip ($r = -0.406, p = 0.000$), sport competitiveness (Sport Orientation Questionnaire) ($r = 0.475, p = 0.000$) and Sport Aggression (Scale of Children’s Action Tendencies in Sport) ($r = 0.240, p = 0.021$). Conclusion: It does not appear that androgens (whether prenatally or current) have an impact on female sport participation. Given that females participate in sport at lower rates than males, and that sport provides multiple social and health advantages, continuing to determine what factors influence sport participation is necessary.
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ABBREVIATIONS

AR – Androgen Receptor
DHEA - Dehydroepiandrosterone
DHEA-S - Dehydroepiandrosterone sulfate
DHT – Dihydrotestosterone
DNA – Deoxyribonucleic Acid
E – Epinephrine
ER – Estrogen Receptor
FSH - Follicle Stimulating Hormone
GPR30 – G-protein Coupled Receptor 30
HPG – Hypothalamus-Pituitary-Gonadal
LH - Luteinizing Hormone
MeApd – Medial Nucleus of the Amygdala
NE – Norepinephrine
NO – Nitric Oxide
pT – Prenatal Testosterone
RNA – Ribonucleic acid
ROS – Reactive Oxygen Species
SHBG – Sex Hormone Binding Globulin
StAR – Steroidogenic Acute Regulatory Protein
sT – Salivary Testosterone
TSP – Total Sport Participation
3β-HSD – 3Beta Hydroxysteroid Dehydrogenase
17β-HSD – 17Beta Hydroxysteroid Dehydrogenase
Chapter One

Introduction

The birth of modern sport was likely initiated by Pierre de Coubertin, the man responsible for the modern Olympic Games, held in Athens Greece in 1896. However, sport as we know it today is very different than in the late 1800’s. During the first modern games, women were not allowed to be competitors because de Coubertin thought that including women would be, “impractical, uninteresting, unaesthetic and incorrect,” (Edwards, 2012). The majority of the 241 participants at the Games of the I Olympiad were men. Today, it is easy to see that much has changed in the Olympics, and sport in general. There are more people than ever, from more diverse populations of both sexes, participating in sport today. For example, immigrants in Canada who arrived after 1990 participate in sport at approximately the same rate (29%) as Canadian-born citizens (27%) (Canadian Heritage Sport Participation Research Paper, 2010). At the 2012 London Olympics, women competed in every sport, could have represented every country involved in the games (i.e. no restriction), and there were a greater percentage of women athletes than any previous Summer Olympics (Donnelley & Donnelley, 2013). Reports from the 2016 games will likely surpass these milestones. Contrary to de Coubertin’s view, the participation of women in sport is practical, interesting, athletic, and important, not only for women’s health, but the health of the entire population. With respect to the latter, the benefits of female sport participation include, but are not limited to, the observations that girls and women who are physically active or engage in sport:

- exhibit better bone health than those who don’t at all ages (Deal, 2001);
• have greater cardiorespiratory and cardiovascular fitness across the lifespan (Yusuf et al., 2004);
• report greater quality of life and health in later years (Buman et al., 2010);
• are less likely to engage in risky sexual behaviours (e.g. multiple partners, unprotected sex, unwanted pregnancies) (Lehman & Koerner, 2004);
• are less likely to report anxious and depressive episodes (De Moor, Beem, Stubbe, Boomsma, & De Geus, 2006);
• tend to perform better in academics (Fox, Barr-Anderson, Neumark-Sztainer, & Wall, 2010).

It is not surprising, then, that greater than 90% of executive women reported in one survey that they had played organized sports during some level of schooling, over half at the university varsity level (female executives say participation in sports helps accelerate leadership and career potential, 2014). Nonetheless, males have and continue to participate in sport in significantly higher numbers than females. According to the Canadian Heritage Sport Participation 2010 Report, one-third of Canadian men participate in sport whereas only one-sixth of Canadian women are currently participating (Canadian Heritage Sport Participation Research Paper, 2010). This observation holds true today, even in the face of the multitude of physiological, psychological, and social benefits of female sport participation.

Understanding sport participation involves examining the motivations and barriers to the problem and this can take multiple approaches that culminate in addressing (or attempting to address) barriers in order to increase participation. However, while resultant programs show success at allowing more children to participate in sport
(Dishman & Buckworth, 1996), these programs are not 100% effective and the effects on lifetime physical activity are relatively unknown. For example, a meta-analysis of after-school programs focused on reducing the body mass index (BMI) of children and adolescents, found an overall effect size of only 0.068 (Vasques et al., 2014). Moreover, over the past decade, with significant government backed initiatives to increase physical activity, the percentage of Canadian men and women who engage in leisure time physical activity that meets recommended guidelines has remained relatively consistent (approximately half the population)(Canadian Heritage Sport Participation Research Paper, 2010). As well, there is discrimination in leisure time physical activity (LTPA) between athletes and non-athletes, with athletes having higher LTPA scores on average (Taylor et al., 1978). Consequently, even with the creation of these programs, there are still people, particularly women and girls, who do not undertake adequate physical activity. In fact, there is some evidence to suggest that there is a genetic basis for both sport participation and LTPA (Maia, Thomis, & Beunen, 2002). Both intuitively and observationally, leisure-time physical activity in young women is associated with sport participation (Mota & Esculcas, 2002, Mota, Santos, & Ribeiro, 2008). Trying to address barriers is a valid strategy, however, since structured physical activity is a more important component of a young girls physical activity than boys (Mota & Esculcas, 2002), it is also important to understand who are the females who choose to stay in sport. Knowledge of the success stories could help with strategies that encourage prolonged female sport participation.

Human behaviour is a complex subject and an action such as sport participation has many influences that change across the lifespan. Consequently, it is difficult to
imagine that a single factor influences this behaviour. As outlined in figure 1, biological, psychological and sociological factors likely impact sport participation together.
Figure 1: Depiction of the three realms associated with sport participation. Each realm contains examples which are representative of associated factors. This figure is not meant to be exhaustive, but merely a representation of the cohesive nature of factors which are involved in the decision of sport participation.
It is important to note that biological, psychological, and sociological determinants of behaviour are fields of study that possess their own inherent complexity. It is beyond the scope of this thesis to address themes that require entire University faculties to teach. However, as will become apparent later, it is hypothesized that, from a biological perspective, the major androgen, testosterone, may be responsible for female success in sport, and consequently drive young women to participate. For example, it is well known that testosterone has a ubiquitous effect on the body that is favourable to increased lean body mass, muscular strength and power, and many traits such as aggression, which would make an individual prone to success in athletic endeavours.

The classical theory of operant conditioning states that behaviours that are reinforced tend to be repeated (Skinner, 1953) and it appears that modifying behaviours, including reinforcement, is most effective (more effective than environmental and cognitive-behaviour modification) in increasing physical activity participation (Dishman & Buckworth, 1996). As such, greater success in sport, simply as a result of biological advantages and the consequent rewards, accolades, encouragement, social gain, etc. may reinforce participation. From an athlete perspective, this is exemplified by the premier league football player, Benoit Assou-Ekotto. He stated that although he did not ‘hate’ playing football (soccer), it was not his passion. Instead he viewed football simply as a job, however one that he continued because it afforded him significant material and social gains (David Hytner, 2010). There are likely many successful athletes who would state the same.
**Steroids**

A steroid is a lipophilic, low-molecular weight compound that is derived from cholesterol (McVeigh, 2013). There are many different steroids within the human body that can be classified by site of production (i.e. gonadal or adrenal steroids), biological function (i.e. glucocorticoids, mineralocorticoids, sex steroids), molecular action (i.e. an estrogen receptor agonist), and/or biochemical effects. The three main sites of production of steroids are the adrenal glands (produce androgens and corticosteroids), the ovaries (secrete estrogens and progestins) and the testes (secrete androgens) (Hu, Zhang, Shen, & Azhar, 2010).

The formation of steroids begins from the conversion of acetate to cholesterol that can occur in all steroid-producing tissues (Hu et al., 2010). Further, cholesterol can be derived from circulating low density and high density lipoproteins, hydrolyzed cholesterol esters or interiorized from the plasma membrane (Ruiz-Cortes, 2012). Steroidogenic acute regulatory protein (StAR) is responsible for the transfer of cholesterol into the inner mitochondrial membrane of steroid producing cells (Woods, Schwartz, Baskin, & Seeley, 2000). The transfer of cholesterol from the outer to the inner mitochondrial membrane via StAR is the rate limiting step in steroid formation (Stocco, 2001). Sex steroids, important to this literature review, are discussed in further detail in the following section.

**Sex Steroids**

There are three main classifications of sex steroids: estrogens, progestins and androgens (figure 2). Each family of hormones serve a specific function within the body though their actions are typically ubiquitous. In addition to the role of estrogen in the
development of primary and secondary sex characteristics, estrogen also plays a role in energy homeostasis (Qiu et al., 2006), metabolism (Clegg, 2012), prevention of bone loss (Michael, Härkönen, Väänänen, & Hentunen, 2005), and vasoprotection (Tostes, Nigro, Fortes, & Carvalho, 2003). Progesterone is referred to as the pregnancy hormone (Spencer & Bazer, 2002), although it also plays a role in the menstrual cycle (Jabbour, Kelly, Fraser, & Critchley, 2006). Progesterone is also present in males, although this role is less clear (Oettel & Mukhopadhyay, 2004). Reference ranges for estrogen and progesterone, in males and females, can be found in Appendices F and G. Androgens are responsible for male differentiation of the gonads (Sitteri & Wilson, 1974) and the male secondary sex characteristics (Hu et al., 2010). Androgen function will be described in more detail below.
Figure 2: Process of sex steroid synthesis in the body, initiating at cholesterol. The hormones highlighted by the boxes (progesterone, testosterone, estradiol/estrogen) are the primary sex hormones. Testosterone, the main hormone under consideration, was discussed in this literature review (adapted from (Bernstein, 2015)).
Mechanism of Action of Sex Steroids

Typically, steroids have two types of actions on target cells, genomic and non-genomic.

Genomic Actions

Genomic actions of sex steroids cause long term effects within target cells. Due to the lipophilic nature of sex steroids, they have the ability to cross cell membranes (Oren, Fleishman, Kessel, & Ben-Tal, 2004). Once inside the target cell, steroids modulate transcription through interactions with receptors in the nucleus or the cytoplasm (Lutz et al., 2003). Steroid hormone receptors are typically transcription factors that have the ability to regulate the expression of target genes (Simoncini & Genazzani, 2003b). As described by Michels & Hoppe (2008), the binding of a steroid to a steroid receptor creates a steroid-protein complex that can bind to steroid response elements (i.e. nucleotide sequences specifically recognized by steroid receptors) that are situated on the promoter region of target genes. This process regulates gene expression (i.e. the making of new proteins) in the target cell that ultimately dictates cellular function (Michels & Hoppe, 2008).

Non-Genomic Actions

Unlike genomic actions, non-genomic actions require the continued presence of the hormone in order for it to have an effect. It has been well established that sex steroids have non-genomic actions, although the specific molecular mechanisms are still under investigation (Simoncini & Genazzani, 2003). Nonetheless, it is known that sex steroids have the ability to influence membrane spanning ion channels. For example, all sex steroids can act as vasodilators (Ruehlmann & Mann, 2000), but estrogen particularly,
acts as a vasodilator of arterial walls in both sexes, although the increased concentration of estrogen receptors in females allows the effect to be more potent in women (R. E. White, 2002).

Simoncini & Genazzani (2003), state that sex steroids also have important non-genomic actions on the brain. For example, estrogen can influence neuron excitability by decreasing the rapid firing of some neurons. Similarly, administration of the androgen, dihydrotestosterone (DHT), is able to inhibit the accumulation of cellular cyclic-adenosine monophosphate (cAMP), a second messenger involved in various biological processes including hypothalamic hormone secretion, thereby decreasing the amount of GnRH released (Simoncini & Genazzani, 2003).

**Formation of Active Metabolites**

As described by Martini et al., (1990), for certain sex steroids and certain target tissues, steroids can be metabolized into different types causing different or altered potency of effects. One example is the conversion of testosterone to 5α-DHT at the prostrate that allows testosterone to influence prostate growth and function (Martini et al., 1990).

**The HPG Axis**

The function of the Hypothalamus-Pituitary-Gonadal (HPG) Axis and the role that sex hormones (i.e. estrogen, progesterone and testosterone) play in the body has been well established in several texts and reviews (Miller & Auchus, 2011, Simpson et al., 2005, Stocco, 2001, Burger, 2002).
The HPG axis is initiated at the paraventricular nuclei (PVN) of the hypothalamus (Hiller & Bartke (1998)). In the PVN, afferent nerves gather to convey stress and homeostatic signals from the body (Yin & Gore, 2010). Initiation of the HPG hormonal system occurs when homeostatic feedback is integrated by the hypothalamus causing gonadotropin releasing hormone (GnRH) to be secreted. GnRH travels through the hypophyseal portal vessels to the anterior pituitary, causing the release of the gonadotropins, luteinizing hormone (LH) and follicle stimulating hormone (FSH), into the systemic circulation in both males and females. LH and FSH travel through the systemic circulation until they reach the reproductive glands (i.e. the ovaries and testes). Steroids, specifically the sex steroids progesterone, testosterone and estrogen, are subsequently synthesized and/or released from the reproductive glands (Hiller-Sturmhofel & Bartke, 1998).

The HPG axis is self-governing (Hiller-Sturmhofel & Bartke (1998). The end products of the system (i.e. the sex hormones) typically, except in the case of estrogen at certain periods of the female menstrual cycle, have an inhibitory effect on upstream organs of the system (i.e. the pituitary and hypothalamus). For example, a high concentration of circulating testosterone will reduce or inhibit hypothalamic release of GnRH, thereby reducing the output of pituitary gonadotropins, and finally testosterone (Hiller-Sturmhofel & Bartke, 1998) in order to maintain a hormone concentration within a fairly consistent range.

In females, the feedback loop is more complicated. As reviewed by Gupta & Chia (2013), the function of the feedback loop changes throughout the menstrual cycle. In females, LH exerts its effects on the theca cells to produce androgens and progesterone
(figure 3). Progesterone, which is synthesized in theca cells in females, has a negative feedback control on the hypothalamus. The granulosa cells in the ovary release inhibin and synthesize estradiol primarily from theca cell produced androgens, including testosterone. Inhibin is responsible for the inhibition of FSH release from the anterior pituitary (Gupta & Chia, 2013).
Figure 3: Visual representation of an ovarian follicle (left), with an enlarged version of the theca and granulosa cells (right), along with the associated hormones secreted from each, as adapted from (Journal of Mid-Life Health).
Depending on the phase of the menstrual cycle, estrogen and progesterone can have a positive or negative feedback effect on the HPG axis (Gupta & Chia, 2013). Menstrual cycle hormone changes repeat over an approximately 28 day cycle (Heitz, Eisenman, Beck, & Walker, 1999) and are described in more detail in the legend to figure 4.

In males, the feedback loop is simpler. GnRH leads to LH and FSH release that subsequently cause testosterone to be released from the Leydig cells in the testes (Encyclopaedia Britannica, Smith & Walker, 2014). Testosterone and FSH-mediated inhibin secretion from the Sertoli cells in the testes exert negative feedback on the hypothalamus and anterior pituitary (Hiller-Sturmhofel & Bartke, 1998).
Figure 4: Depiction of the female hormone fluctuation across the menstrual cycle, as adapted from (Womack) (The woman’s health program, 2010). The luteal phase of the menstrual cycle is characterized by large amounts of progesterone and estrogen, released from the corpus luteum, which inhibit LH and FSH release. The late luteal stage is when LH and FSH begin to rise due to the degradation of the corpus luteum, concurrent with a decrease in circulating estrogen and progesterone concentrations. The decline in estrogen and progesterone begin the initiation of a new follicle. The new follicle subsequently begins to grow, releases estrogen which exerts an inhibitory effect on LH and FSH release (i.e. early follicular phase). During the pre-ovulatory stage (i.e. late follicular phase), there is a spike in LH and FSH, likely caused by a switch from estrogen-induced inhibition to stimulation, that is necessary for ovulation. Circulating testosterone concentrations in females are more consistent across the menstrual cycle, but tend to follow that of estrogen given that the stimulus for estrogen production results in steroidal synthesis of which androgens occur upstream (Gupta & Chia, 2013).
Androgens

Androgens play a major role in the development of male primary and secondary sexual characteristics. The primary circulating androgen is testosterone.

Formation

In males, LH is responsible for the production of pregnenolone in the Leydig cells of the testes (Miller & Auchus, 2011). Pregnenolone is converted to dehydroepiandrosterone (DHEA) and then, DHEA is converted to testosterone through the intermediates, androstenediol and androstenedione. This conversion to testosterone is catalyzed by 17β-hydroxysteroid dehydrogenase (17β-HSD) and 3β-HSD which are present in high concentrations in the testes. Therefore, the conversation of DHEA to testosterone occurs very quickly. In target tissues, testosterone is converted to DHT via the enzyme 5α-reductase. DHT is 2.5-10 times more potent than testosterone, and binds to androgen receptors (AR) with a 2-3-fold higher affinity (Mozayani & Raymon, 2012), however, serum DHT concentrations are 1/10th that of testosterone (Hay & Wass, 2008). The prostate is the major site of non-testicular production of DHT, therefore DHT concentrations are several times higher than the concentration of testosterone at this location (Hay, Wass, & Wiley InterScience, 2008).

In females, there are three forms of testosterone precursors including dehydroepiandrosterone sulfate (DHEA-S) (produced in the zona reticularis of the adrenal gland), DHEA (produced in the zona reticularis of the adrenal gland, ovarian theca cells, and peripherally from circulating DHEA-S) and androstenedione (produced from the zona fasciculata of the adrenal gland, the ovarian stroma, and peripherally from circulating DHEA) (Burger, 2002). Approximately 50% of testosterone production in
females is a result of peripheral conversion from androstenedione. The other 50% is produced in the zona reitcularis and the ovarian stroma. DHT is also produced in females, but the circulating concentration is very low (Burger, 2002). Consequently, in both males and females, circulating testosterone concentrations are the most common measure of androgen availability.

**Circulation**

Circulating testosterone is mainly (~70%) bound to SHBG (Dent, Fletcher, & McGuigan, 2012). In males, SHBG is relatively saturated due to the higher concentration of circulating testosterone, whereas it is significantly less saturated in females (Anderson, 1974). Although estrogen also has the ability to bind to SHBG, testosterone has a significantly increased affinity (Hammond, 2011). In fact, at one time, SHGB was referred to as androgen-binding protein. Therefore, if there is a significant change in SHBG concentrations, there will be a larger difference in free testosterone than estrogen concentrations. Testosterone can partially bind to albumin (30%), a non-specific plasma protein hormone carrier, although the affinity for albumin is relatively low (Anderson, 1974).

The androgens not bound to plasma proteins are referred to as “free” or bioavailable (0.5-3%) (Dent et al., 2012). Free androgens are able to have an effect on target cells through the mechanisms described above. Consequently, free hormone concentrations give a good indication of the likelihood of observing the actions of that hormone. In females, circulating free testosterone concentrations fluctuate during the menstrual cycle. There is a 30-40% increase mid cycle that coincides with the pre-
ovulatory spike in LH (Dent et al., 2012) (see Appendix H - reference ranges for total and free testosterone concentrations for males and females).

**Role of Testosterone in the Body**

**Secondary Sex Characteristics**

Androgens are primarily responsible for the development of secondary sex characteristics in males (Hu et al., 2010). During puberty, there is a significant spike in androgens, and it is this spike that leads to physical changes associated with puberty in males. These include, but are not limited to, the development of reproductive function, the growth of the genitals (including scrotum appearance, growth in testicular size, and penis enlargement), the growth of pubic hair, defined body proportions, and the achievement of the adult fat mass/muscle mass ratio (Huang et al., 2012, Bertelloni, Dati, & Baroncelli, 2008).

**Skeletal Muscle**

The major genomic action of testosterone on the musculature is to maintain and increase muscle mass, which typically leads to an increase in muscle strength. The increase in muscle hypertrophy occurs through an increase in whole body protein synthesis concomitant with an increase in myocyte size (Schoenfeld et al., 2016) and myonuclei number (Hanssen et al., 2013). Differences between satellite cell function between males and females are innate (Manzano et al., 2011). For example, stem cells in females exhibit a regenerative potential, although this gender difference only occurs in the presence of oxidative stress (Deasy et al., 2007). Satellite cells attach to muscle fibers under the basal lamina and are defined through a unique myogenic differentiation process.
Satellite cells are typically activated in response to exercise or injuries. Damage causes them to proliferate and differentiate into mature muscle cells (Kuang & Rudnicki, 2008). In a study completed by Sinha-Hikin, Cornforf, Gaytan, Lee & Bhasin (2006), a dose dependent response of testosterone administration was found that correlated with hypertrophy in type I and II skeletal muscle fibers, likely because of increased satellite cell muscle entry and myonuclear hypertrophy.

Supplemental evidence for the effect of androgens on skeletal muscle is evident in research on anabolic steroid users. The purpose of anabolic steroid use is usually to increase muscle size and strength, although this can only occur if accompanied by proper physical training and diet (do Carmo et al., 2011). Muscle mass increase is associated with muscle fiber hypertrophy (occurring in both type I and II fibers) and increases in myonuclei number (Sinha-Hikim et al., 2006). Steroid users also show increases in the muscle fibers expressing myosin heavy chain isoforms when compared to control groups (Yu et al., 2014). The presence of myosin heavy chain is essential for specialized muscle function and myofibril stability (Wells, Edwards, & Bernstein, 1996). Anabolic steroids in combination with strength training resulted in fiber hypertrophy and fiber hyperplasia (formation of new fibers), thought to be partially due to the activation of muscle satellite cells (Yu et al., 2014). Because the doses used by anabolic steroid users and in published reports are typically supra-physiological, it is unclear whether basal levels of testosterone work on muscles in a dose dependent manner.

Further, as proposed by Estrada, Espinosa, Muller et al., (2003), androgens could impact skeletal muscle size by activating a G protein-linked membrane receptor, increasing intramuscular calcium and consequently, muscle power and potentially
hypertrophy. Activation of this receptor would result in rapid actions independent of genomic androgen receptors, however, it is important to note that no study has directly investigated this non-genomic role in humans.

**Cardiovascular Physiology**

Testosterone can directly influence cardiac hypertrophy. Cardiac myocytes are muscle cells. This means that they can be affected by testosterone in the same manner as skeletal muscles. The influence of testosterone on the cardiovascular system can be seen in the difference in heart size between males and females. Males have hearts that weigh approximately 280g to 340g and females have hearts weighing 230 to 280g (Gray, 1918). The difference in heart size is thought to be partially due to the difference in testosterone concentrations between males and females.

This is supported by studies in which exogenous testosterone is given to cardiac failure patients. After administration, a significant increase in cardiac output and overall exercise capacity can be seen (Chung et al., 2007). However, it is unknown whether supra-physiological doses of testosterone are able to induce cardiac hypertrophy above normal ranges, although, there are anecdotal incidences of this occurring. In the study completed by Chung et al., (2007), nandrolone (200mg) and testosterone (200mg) was administered to 30 male participants (age 18-45 y) weekly over a 4-week duration, but this did not significantly increase chamber size in either group, including the placebo group. Although small changes in chamber size were seen, the results were within the normal limits for the age range (Chung et al., 2007). In other studies, testosterone concentrations were negatively correlated with common carotid artery intima-media thickness (Rai & Ramasamy, 2016). More studies on the long term effects of supra-
physiological doses of androgens need to be conducted in order to fully understand the effect that testosterone has on heart size.

A significant amount of research into the effects of testosterone on cardiac health has come from users of anabolic steroids. The use of anabolic steroids typically includes the exogenous supplementation of nandrolone, testosterone, and to a lesser extent other synthetic androgens. According to Chung et al., (2007), there are anecdotal cases of cardiomyopathy, myocardial infarction, arrhythmia, hypertension, cardiac failure, stroke, pulmonary embolism and sudden death which have been seen in anabolic steroid users. However, other indirect mechanisms of testosterone on cardiac health include a positive effect of testosterone on symptoms of angina, atherosclerosis, obesity, ventricular repolarization and intima-media thickness of the coronary artery (Oskui et al., 2013).

**Hematology (Blood)**

It has been well established that testosterone is important in the regulation of erythropoiesis (i.e. the production of red blood cells) in both males and females (Beggs et al., 2014). In the not so distant past, androgens were used as treatment for anemia associated with chronic diseases such as end stage renal failure (Snyder & Shoskes, 2016). It has been well established that testosterone has the ability to stimulate erythropoiesis, but the mechanisms by which this occurs are not well understood (Fried & Gurney, 1968). A study conducted by Guo et al., (2013) identified hepcidin (i.e. the iron regulating hormone) as the downstream target of androgen receptors. In both males and females, hepcidin is downregulated, which in turn increases the upregulation of Ferroportin, thereby increasing iron export from the spleen (Yu et al., 2014). This causes an increase in iron availability for hemoglobin synthesis, which ultimately leads to an
increase in red blood cells. Moreover, androgens can control erythropoiesis through erythropoietin (EPO), the hormone responsible for erythropoiesis (i.e. red blood cell production) in the bone marrow (Beggs et al., 2014).

**Testosterone and Metabolism**

Men with a testosterone deficiency typically show an increased fat mass associated with insulin resistance. This means that a decrease in testosterone concentration is usually associated with metabolic syndrome and other metabolic conditions, like type II diabetes (Kapoor, Malkin, Channer, & Jones, 2005). In a study completed by Usui et al., (2014), it was hypothesized that the cause of this reduction of fat mass was through a testosterone mediated elevated energy expenditure. An increase in mitochondrial biogenesis in skeletal muscle was observed in the male rats, a result the authors attributed to testosterone.

**Testosterone and the Brain**

Testosterone can influence the structure and physiology of the brain, which may impact cognitive function and behaviour. For example, differences in male and female behaviour are at least partially due to differences in sex hormones throughout development.

As previously discussed, testosterone is typically produced in the ovaries in females or in the testes in males. There is also evidence suggesting that testosterone can be synthesized directly in the brain, potentially *de novo* from cholesterol, or from other steroids (progesterone or deoxycorticosterone) which enter through the blood into the nervous system (Celec, Ostatnikova, & Hodosy, 2015).
More specifically, it has been shown that astrocytes have all of the cellular machinery necessary to make sex hormones from cholesterol (Rossetti, Cambiasso, Holschbach, & Cabrera, 2016), and there is a possibility that androgens and estrogens might be controlled near the local blood vessels (Krause, Duckles, & Gonzales, 2011). Therefore, cerebral blood concentrations of sex steroids might be different than what is observed in the circulation (Krause, Duckles, & Pelligrino, 2006). It is true that gonadal hormones also act on other cells within the neurovascular unit (e.g. astrocytes and neurons, circulating blood elements like platelets and leukocytes), and thus androgens can also have an indirect effect on the cerebrovascular function through these actions.

Gonadal sex steroids (testosterone for males and estrogen for females) peak in the middle trimester during pregnancy, and then drop in the perinatal period and peak a second time within the first 3 months of life (Knickmeyer, Woolson, Hamer, Konneker, & Gilmore, 2011). What is defined as the “pre and postnatal stages” are thought of as periods of permanent “organizational” sexual differentiation of the brain (Sinclair, Purves-Tyson, Allen, & Weickert, 2014). Specifically, androgen exposure before birth has the ability to alter the number of neurons in specific brain areas, as well as their connectivity and neurotransmitter content (Roselli & Klosterman, 1998). When a child reaches adolescence, there is another significant increase in sex hormones which have an altering effect on the child through the form of pubertal physical and behavioural changes (Sinclair et al., 2014). Therefore, testosterone has organizational, as well as activational, effects on the brain (Falter, Arroyo, & Davis, 2006). Even during adulthood, evidence suggests that sex steroids have the ability to remodel synapses, change the dendritic
structure and alter neurogenesis, discrediting the notion that the adult brain is unchanging (McEwen, 1994).

The sexually dimorphic characteristics of the brain may not necessarily be due to the immediate effects of testosterone on the brain. Testosterone, can be converted to estradiol by the aromatase enzyme. It is thought that it is actually the action of estradiol on the brain that is responsible for the sexually dimorphic characteristics between males and females (Purve et al., 2001). Therefore, if a fetus in vitro is exposed to high prenatal testosterone (pT), there is more opportunity for the conversion of testosterone to estradiol. This results in an increased opportunity for estradiol to modify the developing brain in males as opposed to females. However, it is important to note that results are still equivocal in terms of whether this difference manifests itself into a difference in behaviour (Manlove, Guillermo, & Gray, 2008).

One of the hypothesized effects of pT exposure is on brain lateralization. For example, males are significantly more lateralized for language and are often more left-handed than females. It is hypothesized that the high concentrations of testosterone prenatally, slow the development of the left hemisphere (Lust et al., 2010). Another theory developed by Witelson & Nowakowski (1991) (i.e. the Callosal hypothesis) postulates that pT increases axonal pruning in the corpus callosum, which would lead to a reduction in communication between the two brain hemispheres (Witelson & Nowakowski, 1991). Hines & Shipley (1984) put forward the sexual differentiation theory, which hypothesized that pT causes a more “masculine” pattern of lateralization (i.e. an increased degree of lateralization) (Lust et al., 2010). All of the above theories have been challenged and none are currently accepted unconditionally, but they all were
created to try and explain the difference in language lateralization present between males
and females.

Some of the most extensively studied dimorphic areas of the brain are the preoptic
nucleus, the ventromedial nucleus and the suprachiasmatic nucleus, which are all
divisions of the amygdala and the stria terminalis (Herrera Gutiérrez, Vergara Onofre,
Rosado-García, & Rosales Torres, 2005). The ventromedial nucleus of the hypothalamus
regulates sexual behaviour in females. The density of this nucleus is much greater in
males than it is in females. Further, the synaptic connections in females are known to
vary throughout the menstrual cycle, due to changes in estrogen concentrations
(Hagemann et al., 2011). Studies have indicated that the ventromedial nucleus showed an
increase in the nucleus, nucleoli, and cell soma cellular components (a result of increased
cell metabolism and protein synthesis) in response to estrogen administration,
demonstrating the susceptibility of this brain structure to estradiol as opposed to
testosterone (Herrera Gutiérrez et al., 2005).

The suprachiasmatic nucleus (SCN) controls the circadian (including hormonal,
physiological and behavioural) rhythms of the body. It has been hypothesized that the
SCN may be also related to sexual behaviour (Herrera Gutiérrez et al., 2005). Rodent
studies have demonstrated that the prenatal administration of anti-estrogens increases
mounting behaviours (Sodersten, Hansen, & Srebro, 1981). This change in behaviour
strengthens the hypothesis that the SCN is under the control of sex steroids.

The preoptic area of the hypothalamus is responsible in part for control of sexual
behaviour, the release of gonadotropins, and mechanisms involved in ovulation. In the
adult rat brain, there is no difference in the size of the preoptic area of the hypothalamus between sexes, but there is a significant difference in the growth rate. At birth, the volume of the preoptic area is approximately double in females. During the first 10 days of life, there is an increase in the volume of the area in males, whereas there is no postnatal growth in females. This difference in postnatal growth has been attributed to the pT exposure from the testes in males (Herrera Gutiérrez et al., 2005).

The amygdala has been identified as one area of the brain responsible for emotions associated with aggressiveness, fear, and anxiety (Herrera Gutiérrez et al., 2005). Animal studies have looked at the sexually dimorphic properties of the medial nucleus of the amygdala (MeApd) in rats, which plays a large role in social behaviour. The MeApd volume is greater in males than in females rats (Cooke & Woolley, 2005). This difference is thought to be partly due to circulating testosterone concentrations in male and female rats. There have been studies completed on young rats who exhibit rough and tumble play, which show that testosterone in the MeApd has a masculinizing effect on play whereas when the amygdala is bilaterally destructed, the rat takes on a more feminine social behaviours (i.e. care of infants is usually performed by females rats) (Meaney, Dodge, & Beatty, 1981). This means that the hormones exhibited in the adult rat could be influencing the MeApd, which has already be differentiated by prenatal sex hormones (Cooke & Woolley, 2005).

**Alternative Sources of Sex Steroids**

As previously discussed, one of the main alternative sites for sex steroid synthesis is the adrenal glands. In males, DHEA and androstenedione are produced in the zona reticulate and the zona fasciculate of the adrenal cortex. In females, DHEA is produced
from the zona reticularis, and androstenedione is produced in the zona fasciculata. Moreover, in females, about 50% of the circulating testosterone is produced in the zona reticularis (Burger, 2002).

Another important production site of sex steroids is adipose tissue (Kershaw & Flier, 2000) (Musi & Guardado-Mendoza, 2014). Specifically, the stromal cells of the adipose tissue are involved in the conversion, activation, and inactivation of sex steroids. The two most highly expressed enzymes in adipose tissue responsible for steroid conversion are cytochrome p450-dependent aromatase and 17β-hydroxysteroid dehydrogenase (17βHSD). Specifically, 17βHSD mediates the conversion of androstenedione to testosterone and estrogen to estradiol. The ratio of 17βHSD to aromatase is correlated in a positive manner with central obesity, which implicates increased local androgen production in visceral adipose tissue (Kershaw & Flier, 2000) (Musi & Guardado-Mendoza, 2014).

Another less prominent site of sex hormone conversion is the bone. The conversion of testosterone to estrogen occurs using the enzyme p450 aromatase. The conversion to estrogen is responsible for aiding in the maturation of the epiphysis of the bone, as well as implications in increasing bone density and reducing rates of bone turnover (Sinnesael, Boonen, Claessens, Gielen, & Vanderschueren, 2011). Therefore, the conversion of testosterone to estrogen occurring in the bone has important local effects on this production site of sex hormones.

The active biosynthesis of testosterone can also take place in the brain (Celec et al., 2015). Specifically, astrocytes have all the necessary enzymes for synthesis of
progesterone, testosterone and estrogen from cholesterol (Diana N. Krause et al., 2006). As many (if not all) areas of the brain exhibit estrogen and androgen receptors, sex steroids are able to have an important influence on behaviour as previously discussed.

**Female Sport Participation**

In 2013, the Canadian government released a Sport Participation Research Paper based on the census 2012 data. In accordance with the previous reports, sport participation in Canada is continuing to decline, although the rate of decline does seem to be slowing. Fewer Canadians ages 15 and younger are participating in sport than before. In 2005, only 28% of the population regularly participated in sport, and in the 2010 report, this number has dropped to 26% of the population.

In accordance with the results from the Sport Participation 2010 report, Canada has recently been given the grade of D- in overall physical activity on the 2014 ParticiPACTION Report Card on Physical Activity for Children and Youth (Active Healthy Kids Canada, 2015). The report card is based on 11 different indicators which are grouped into three categories including Strategies and Investments (i.e. Government and non-government), Settings and Sources of Influence (i.e. Family and peers, school, community and environment) and Behaviours that Contribute to Overall Physical Activity (i.e. organized sport and physical activity participation, sedentary behaviours, active transportation, active play). The grade given is reflective of the balance between age groups which are meeting the physical activity standards (3 to 4 year olds), and those who are doing poorly (age 5-11 year olds and 12-17 year olds).
In both the Sport Participation 2010 report, and the 2014 ParticiPACTION report, gender differences in physical activity levels are clear. The ParticiPACTION report highlights the difference in overall physical activity through total steps taken per day. In 2011-2014, Canadian boys took an average of 11604 steps, whereas Canadian girls took only 10443 steps, emphasizing the difference in activity levels between the two groups. As stated in the Sport Participation 2010 report, “In Canada, men are more likely to participate in sport than women, although the participation rates have declined for both genders over the years.” However, between 2005 and 2010, the participation rate for males has remained stable at around 35%. Noteworthy, is the significant drop in participation rate for women aged 15-19 y with a 13% drop, and for females aged 20-24 y with a 14% drop in sport participation. Visual depictions highlighting male and female sport participation in Canada can be viewed in figure 5.
Figure 5: Sport participation rates for 2005 and 2010 in Canada in A, males and B, females. Black box is indicative of the largest change between groups for females, as adapted from (Canadian Heritage Sport Participation Research Paper, 2010).
A specific recommendation from the ParticiPACTION report card stated the need to support children and youth in adding bouts of physical activity throughout their daily lives. In order to have the most impact on sport participation, it is important to target the gender and age range that is at most risk of dropout. As highlighted by the black box in figure 8 above, the largest drop off rate for sport participation between males and females occurs between the age ranges of 15-19 and 20-24 y in the female group. Therefore, in order to have the most impact on sport participation rates, this would be the ideal age range to target.

**Potential Biological Correlates of Participation**

If a child is able to experience success in sport, they are more likely to continue participation. The specific ways in which testosterone can influence sport are discussed in the following sections.

**Testosterone and Skeletal Muscle**

As previously discussed, it is well known that testosterone has the ability to act as an anabolic agent in order to increase muscle size through genomic actions (i.e. an increase in myocyte size and number). There is a relationship which exists between the size of the muscle and the ability for it to produce power (3). Therefore, the larger the muscle, the more capable it is of moving weight (resistance). This combined with the fact that testosterone has the ability to reduce body fat in both men and women when combined with exercise training (Bescos et al., 2009), demonstrates that testosterone not only has the ability to positively impact power output in explosive activities like jumping (Markovic, Mirkov, Nedeljkovic, & Jaric, 2014) and sprinting, but it can also have a significant impact on absolute strength (Frontera, Meredith, O’Reilly, Knutgen, &
Evans, 1988). Coincidentally, it is not hard to imagine that there has been a direct correlation found between testosterone concentrations and muscle strength as well as muscle power. For example, in a study which looked at basal testosterone levels in relationship to performance within male soccer players (n=32, age = 24.6 ± 4.1 y), testosterone was positively correlated with counter-movement jump height and running speed, but negatively associated with results of the Cooper’s test (i.e. a test in which participants are asked to run as far as they can on a track, within a 12 minute time frame). Therefore, athletes that were better at explosive and sprinting performance had higher levels of basal testosterone (Bosco, Tihanyi, & Viru, 1996). This suggested a relationship between testosterone and the development of fast twitch muscle fibers in athletes.

Further, in a study on testosterone and squat performance on elite male athletes (age = 23.6 ± 1.6 y), participants clearly separated themselves into two groups, good squatters and average squatters, based on the maximum weight that they could squat. The good squatters revealed a strong predictive relationship of pre-testing salivary free testosterone concentration, whereas this relationship was much weaker in the average squatters (B Crewther, Cook, Gaviglio, Kilduff, & Drawer, 2012). It would be informative to explore the possibility that a cross over point in strength exists, at which point the correlations become strong or if this is some sort of threshold effect.

Nonetheless, as noted by Bosco, Tihanyi & Viru (1995), “Well-trained athletes do not constitute a homogenous group by their hormonal profile. Therefore, it is justified to ask whether individual differences in basal hormone levels in the blood are related to an athlete’s performance capacities” (Bosco et al., 1996). Further, the majority of studies which correlate basal testosterone concentrations and performance measures were done
on males. This is a major gap in the literature. Further research needs to be conducted in order to explore if a similar relationship exists in females where basal testosterone concentrations are considerably lower.

For the female athlete specifically, there is a fluctuation of testosterone across the menstrual cycle. Testosterone peaks around ovulation in a female and the peak lasts for about 2-3 days. There is currently no evidence in the literature that proves this fluctuation in testosterone is beneficial to female athletes during the peak in concentrations. However, Dent et al. (2012), suggest that the use of oral contraceptives (which eliminate this testosterone peak) might reduce the optimal hormone physiology for elite female performance. Therefore, Dent et al., (2012) suggested that athletes whose events require strength or power, might benefit from using a contraceptive device that is not hormonally based.

**Testosterone and Cardiorespiratory Fitness**

The anabolic properties of androgens impact cardiac muscle such that at all ages, male hearts are typically larger than female hearts even when myocardial mass is normalized to body surface area. Specifically, in young adult elite athletes (18-39 y), the difference in left ventricular mass/body surface area approaches a 20g difference between males and females (Prakken et al., 2010). A larger heart with corresponding increase in left ventricular size is beneficial for endurance type exercise because of the greater stroke volume supplied with each contraction of the heart. This observation alone could help to explain the sex-based advantage in maximal oxygen consumption (VO2max) between males and females.
Further, testosterone has the ability to stimulate the formation of red blood cells (Mooradian, Morley, & Korenman, 1987). It is well known that mean hemoglobin content of the blood is greater in males (although this may not be an androgen mediated effect alone and is likely more a function of menstrual cycle related blood loss) (Harbour & Miller, 2001). Both an increase in stroke volume and oxygen carrying capacity of the blood would result in increased maximal oxygen consumption (VO$_{2\text{max}}$). For example, elite female distance runners can reach VO$_{2\text{max}}$ values in the range of 70+ ml/kg/min (Pate, Sparling, Wilson, Cureton, & Miller, 1987) whereas males can reach values as high as 80+ ml/kg/min (Pollock, 1977). Consequently, females competing with genetic and gonadally intact males, particularly through the pubertal years, may be placed at a disadvantage in those sports requiring high levels of muscular strength, power and/or cardiorespiratory endurance.

To look specifically at females with increased basal testosterone concentrations, Bermon, Vilain, Fenichel & Ritzen (2015), compared females with PCOS to a body-mass index matched control group. The endurance athletes with PCOS demonstrated the most anabolic body composition, highest VO$_{2\text{max}}$ and highest performance values (Bermon et al., 2015), thought to be at least partially attributed to the difference in resting testosterone concentrations.

An important study which looked at the influence of basal testosterone concentrations in female athletes was performed by Rickenlund et al., (2003). In this study, 39 female athletes who regularly participated in endurance sports (minimum of 70km of running a week) were included. Blood samples were taken after an overnight fast to determine serum T and SHBG, and in a subgroup of the athletes, high androgen
levels were found in accordance with low levels of SHBG and an increased LH:FSH ratio (Rickenlund et al., 2003). The suspected cause of the characteristics of this subgroup was mild hyperandrogenism. The findings of this study were unusual because hyperandrogenism is most often seen in female athletes where strength is a benefit. This was the first observation of a role of testosterone in endurance athletes who rely more heavily on aerobic metabolism. Although the mechanisms are not clear, this study would suggest that hyperandrogenism might have a positive impact on elite female endurance athletes.

Given that testosterone, both pre and post-natal, has been associated with more aggressive type behaviours, it is possible that testosterone induced aggression could be a trait that self-selects certain individuals toward sport. Aggression inserts itself into the sporting world when the goal of the competition is to win, which typically occurs in a travel or competitive league, but can be found in less typically competitive leagues (i.e. house-leagues). However, although the use of aggression in sport could include the intent to injure, that is not typical of its use. In this sense, athletic aggression is viewed in a positive light, and seen as a positive attribute when used to describe a player or team. As stated by Makarowski (2013), many sport journalists and coaches believe that aggression in sport is a positive behavior, and contributes positively to the achievement of success. Particularly at young ages, the most aggressive children will stand out as they run after a ball or skate after a puck, and as such, these children are more likely to be selected for higher level teams that bring more practice and competition experience. Consequently, it is possible that testosterone would exert some influence on continued and higher level sport participation.
Alternative Influences on Sport Participation

As previously discussed, the decision to participate in sport is complex. Biological correlates of sport participation as a possible influence of behavior, is an area of research which has not been studied extensively. Conversely, sociological correlates of sport participation are have been the primary areas of focus. Specifically for adolescents, familial influence has a great impact on whether a child participates in sport. The Canadian Sport Heritage report, released by the government of Canada in 2010, outlines several sociological determinants of sport participation.

For example, the 2010 report states that the higher the level of education a person has, the more likely they are to participate in sport (Canadian Heritage Sport Participation Research Paper, 2010). Approximately one-third of University graduates, 25% of people who have a post-secondary diploma and/or some university, and 22% of people who have a college, trade or high school diploma are currently participating in sport. This is supported through data presented by the Canadian Fitness and Lifestyle Research Institute in 2006, which concurs that Canadians who have received a higher level of education (greater than secondary school) are more likely than those who only have a secondary school diploma, to be active (Lifestyle, 2006).

For youth and adolescents, if the parent currently participates or participated in sport during their youth, it is more likely that the child is going to participate in sport. More specifically, the influence of the mother’s sport participation is greater than the father’s (Ruseski, Humphreys, Hallmann, & Breuer, 2011). Parents are important to the positive values, attitudes and behaviours toward sport, and consequently, the child is more likely to behave in a similar way (Côté, 1999).
It is well known that children who participate in youth sport generally come from mid-high income families (Canadian Heritage Sport Participation Research Paper, 2010). Socioeconomic status (SES) has a positive correlation with sport participation, meaning that the more income a family brings in, the more likely their children are to participate in sport. An important statistic to consider is the relationship between childhood sport participation and participation into adulthood. People who participate in sport during their youth are more likely to continue to participate into adulthood. During childhood is when SES has the biggest impact on sport participation because it is generally under the discretion of the parent whether the child participates in sport or not. White & McTeer (2012) examined the relationship between differing levels of SES and sport participation at different stages of development in children. In this study, it was found that SES was a significant predictor of sport participation for children aged 6-9 y, but there was no effect for children aged 10-15 y. One possible explanation is that sport opportunities might increase within the school setting as children get older. For example, organized sports teams are initiated around grade 6 when a child is around 12 y, thereby providing more opportunities for children of all social status’ to participate in sport. The findings of this paper, suggest that the relationship between SES and participation in sport might be more complicated than previously thought because the influence of SES on sport participation varies depending on stage of life.

Similarly, the use of competitiveness in sport has also fostered positive connotations in terms of success. Competitiveness in sport can be defined as, “the desire to enter, and strive for success in, sport situations or the desire to win in interpersonal situations,” (Gill & Deeter, 1988). As was noted by Gill & Dzewaltowski (1988), it
should make sense that an individual’s innate competitiveness would be related to sport achievement and success (Gill & Dzewaltowski, 1988). Gill & Dzewaltowski (1998), also commented on the fact that when a person is highly oriented towards achievement, they are more likely to approach achievement situations, try hard, strive for success and persist in the face of failure. All of these attributes of a competitive person would be beneficial in leading an athlete towards sporting success (Gill & Dzewaltowski, 1988).

Both aggression (briefly described earlier) and competiveness have the ability to aid in the achievement of success in sport. The achievement of success could bring about an external motivation for continued sport participation and therefore drive their internal motivation to further compete. Joy is found through success, therefore motivation could be found through admiration of their abilities from others. Further, joy brought from winning combined with internal and external motivations may will athletes to train and play harder, creating a cycle of success from the inherent traits of aggression and competiveness.

The Problem of Sex in Sport

In the development of males and females, there is no requirement that phenotypic sex occur in a one-to-one ratio with chromosomal sex. In an extreme example, presence of the transcription factor, SRY (sex determining region of the Y-chromosome), in an XX (two X chromosome) individual, could result in that chromosomal female individual undergoing normal male phenotypic development and sex assignment (Rajender et al., 2006). In a more common occurrence, genetic females with excess androgen production either by the gonads or by the adrenal glands, could develop under the influence of excess (to a typical female) circulating androgen concentrations, thereby expressing phenotypes
that range from typical females to ambiguous or male-like development (Azziz et al., 2004). Although the International Olympic Committee (IOC) has provided regulations on the processes involved with reporting, investigating, and adjudicating cases of female hyperandrogenism (*IOC Regulations on Female Hyperandrogenism*, 2012), and both the IOC and International Association of Athletics Federations (IAAF) have provided policies on sex reassignment through their Hyperandrogenism Regulations; Explanatory Notes, released in 2009, there is still much grey area when considering natural androgen levels in athletic women. This is even more of a problem because of the issue with exogenous androgen administration to many elite athletes.

This poses problems with respect to the categorical placement of intersex athletes in competition. Previously, the policy used by IAAF, the IOC and the World Anti-Doping Association in gender verification of females focuses on the phenotypic expression of external genitalia in the competing athlete (IOC et al., 2012). The underlying premise in these policies is that males are at a competitive advantage in many athletic endeavours, primarily because pubertal and post pubertal males have higher circulating concentrations of gonadal androgens (i.e. testosterone, and its more active metabolite, dihydrotestosterone (DHT), in particular) than females.

Consequently, while the benefits of exogenous, supraphysiological doses of androgens may be advantageous to the competing female athlete, *it is unknown whether naturally elevated testosterone is advantageous to athletic females.*

Currently, there is data to suggest that athletic females have high androgen concentrations. In a study completed by Hagmar, Berglund, Brismar & Linden
Hirschberg (2009), 90 of Sweden’s elite female athletes (mean age of 24.0 ± 3.6 y) representing 27 different sports and who were considered to be representative of the Swedish participation in the upcoming Summer and Winter Olympic Games were included in this study. These women underwent various tests including measurements of body composition, a gynecologic examination and peripheral venous blood samples in order to determine hormonal status. It was determined that 27% of these athletes had menstrual disturbances, and that the most common mechanism underlying this disturbance was the hyperandrogenic condition, Poly-Cystic Ovary Syndrome (PCOS). The instance of PCOS in this athletic population was 37% as compared to the instance in a normative population of 20%. Therefore, it was suggested specifically that “PCOS may reflect an anabolic state that could be advantageous for physical performance and may thereby play a key role in the achievement of a high competitive standard by female athletes,” (Hagmar et al., 2009).

In a study by Coste et al., (2011), younger females (n=36) with a mean age of 15.4 y were investigated. These adolescent girls were all a part of a competitive swimming program, and similarly underwent testing to determine menstrual status and blood sampling to determine their hormonal status. A total of 72% of the swimmers tested had testosterone concentrations great than 0.5ng/mL, characterizing them as hyperandrogenic, where 50% of the females showed signs of hyperandrogenism and 8 of the 36 females showed signs of PCOS as determined through a pelvic ultrasonography. Again, it was suggested that the PCO-like syndrome in these elite-adolescent female athletes was perhaps not a coincidence and specifically that, “A predisposition to
hyperandrogenism might orient girls towards sports, such as swimming, where strength is a performance criterion,” (Coste et al., 2011).

Bescos et al., (2009) found that female athletes exhibit lower 2D:4D (2DR) ratios (index to ring finger ratios), a known correlate with exposure to high prenatal testosterone, than control populations. Therefore, there is the potential for both prenatal and basal testosterone concentrations to have an effect on female athletic participation through physiological and psychological advantages.

**Pilot Data**

Pilot data extends from a study conducted in the Physical Activity and Cardiovascular Research (PACR) lab examining resting salivary and urine testosterone concentrations of female varsity athletes compared with self-identified non-athletes. Although there was not a significant difference between the two populations, it was noteworthy that within the cumulative sample population, there were noticeably higher salivary testosterone (sT) levels (39 – 148 pg/mL) than reported in an average population (i.e. 7.7 ± 2.6 pg/mL) in a separate study from another lab (Karkazis, Jordan-Young, Davis, & Camporesi, 2012). However, the data was more consistent with those reported by other labs in reference to female athletic populations (65.3 ± 3.2 pg/ml and ~55-100 pg/ml in 2 separate studies of athletic females) (Cook & Beaven, 2013, Crewther et al., 2012). It was theorized that the findings were the result of the sample population; many of the “non-athlete” participants scored high on Godin’s leisure time exercise questionnaire (LTEQ) suggesting that even though these individuals did not play varsity sport, they were more active than typical females. It is believed that this data, combined
with the aforementioned studies, is sufficient to warrant a larger scale study in this regard.
Chapter Two

Introduction

Why do females participate in and continue to participate in sport? Unfortunately, when it comes to human behaviour, the answer to this question is anything but simple. Human behaviour has different influences at different times of life, and under different environmental conditions. Nonetheless, lifetime sport and physical activity participation provide many physiological and sociological benefits to the participant, and consequently, understanding this behaviour is important to the health and well-being of women and girls.

Although often used interchangeably, physical activity and exercise (of which sport is a component) represent different concepts. The most basic difference is that sport and exercise is typically structured physical exertion with the intent of improving physical fitness and most often occurs in the leisure-time of an individual versus the activities of daily living (sleep, chores, work, etc. as well as leisure time physical activity, LTPA) that would encompass physical activity more generally (Caspersen, Powell, & Christenson, 1985). Nonetheless, sport and exercise are generally, what is taught in physical education programs to adolescents, and the difference between North American youth sport (with its emphasis on winning, increasing time and financial commitment, etc.) and unstructured play is a concrete example of this difference. However, individuals who compete in youth sport are more likely to meet moderate to vigorous physical activity (MVPA) guidelines as youth and throughout life (Silva et al., 2013).
Unfortunately, there is a discrepancy between the LTPA and sport participation rates of males and females worldwide. As a population, North Americans do not meet the guidelines of daily recommended MVPA standards in both sexes, but males are more likely to participate in LTPA, including sport participation, than females (Stephens, Jacobs, & White, 1985). Moreover, while there is a general decline in physical activity and sport participation starting in the early teens, the largest drop in participation rates occur between the mid to late teens for young adult females. This problem is well-known and multifaceted, and generally, the implementation of programs to overcome barriers such as a lack of opportunity or deemphasizing “winning” and encouraging “play” are encouraging, but not 100% effective. Youth sport participation in Canada for both boys and girls have been fairly consistent over the past decade with 75% of youth between 5 and 17 participating in sport. However, it is important to note that more boys (81%) than girls (70%) participate in sport.

The focus of this research study is to examine the relationship between sport participation and biological factors. In particular, it is possible that the circulating androgen, testosterone, may predispose girls toward sport participation during these periods. Testosterone is a steroid that may impact various traits that are beneficial in sport. For example, testosterone is known to increase skeletal muscle mass (Yu et al., 2014), a major determinant of strength and power, and it is able to increase cardiac size (Gray, 1918) resulting in a larger stroke volume, manifesting into increases in VO$_{2\text{max}}$. Testosterone also regulates erythropoiesis which increases the oxygen carrying capacity of the blood (Beggs et al., 2014). Testosterone’s effect on energy metabolism relate directly with fat loss (Usui et al., 2014), which is highly beneficial in most sport settings.
Moreover, while equivocal, prenatal (Coyne, Manning, Ringer, & Bailey, 2007) and circulating testosterone are correlated with competitiveness and aggressiveness (Book, Starzyk, & Quinsey, 2001), traits that may also predispose individuals to success in sport. Success in sport may subsequently lead to more physical practice, more extrinsic motivation (awards, praise, etc. (Vallerand & Losier, 1999)), more intrinsic motivation (fun, winning, etc. (Vallerand, Deci, & Ryan, 1987)), and a greater belief in physical abilities (self-efficacy (McAuley, Wraith, & Duncan, 1991)), that may increase success in sport in a cyclical fashion. In fact, exogenous anabolic androgen administration is one of the biggest doping concerns for male and female athletic competitions because of the effectiveness of this hormone (Sjöqvist, Garle, & Rane, 2008).

It is important to note that very few studies have addressed the potential for biology to be a component of sport participation. It is likely that this has not been omitted in error, but is more due to the difficulties of performing large scale studies that include costly biological analyses. Consequently, while it is obvious that biology is important to athletic success, there is a gap in the literature as to the extent biology plays in sport participation, while sometimes, it is the role of biology that can have non-obvious forms. For example, research on the relative age effect has shown that children born early in their respective competitive age group are more likely to make higher level teams and play longer in youth (Andronikos, Elumaro, Westbury, & Martindale, 2015). Also, genetics has been found to explain approximately 30% of the variability in young adult female LTPA (Maia et al., 2002). Pilot data from our lab has found a positive relationship between LTPA and salivary testosterone (sT) in young adult females (Vandenborn and Milne, unpublished results). Combined with the potential sport-
related benefits of testosterone on performance, this forms the basis for the main hypothesis of the current study:

Hypothesis 1: 2DR ratio, a marker of prenatal testosterone, and salivary testosterone will correlate with sport participation through adolescence (9-18 y) as well as current sport participation.

An understanding of whether such a biological relationship exists with sport participation in females will give researchers a better idea of the type of female who chooses to continue sport participation into adulthood. As such, a secondary objective of this study is to examine whether athletic females exhibit higher circulating levels of testosterone that may subsequently inform policy decisions regarding female athlete endogenous hormone levels and competition.

Design

To tests the hypotheses above, a cross-sectional analysis of resting and indirect pre-natal androgen concentrations using second to fourth digit (2DR) ratios of the hand were obtained from a sample of 18-30y females recruited from the University of Windsor and the Windsor-Essex community as a convenience sample. Participant demographic (via questionnaire), anthropometric, behavioural (via questionnaire), and retrospective sport participation (via questionnaire) information were collected on 1 occasion and saliva was collected on 2 occasions. All information was collected in the Physical Activity and Cardiovascular Research Laboratory (PACR Lab) in the Department of Kinesiology at the University of Windsor.
Methods

Definition of Sport and Sport Participation

According to the Sport Participation 2010 research paper produced by the government of Canada:

Sport involves formal rules, procedures, requires tactics and strategies, specialized neuromuscular skills, and a high degree of difficulty and effort. The competitive nature of sport implies the development of trained coaching personnel. It does not include activities in which the performance of a motorized vehicle is the primary determinant of the competitive outcome (Canadian Heritage Sport Participation Research Paper, 2010).

Active participation was defined as “any individual who engages in sport for the purpose of competition with others, under a set of rules, or to improve their personal sporting performance,” (Bloom, Grant, & Watt, 2005). A list of those events which qualified as a sport under the government of Canada’s guidelines was used in the study (Appendix A).

Sport Participation Score

In the determination of sport participation, only those sports recognized by the government of Canada (Appendix A) were included. Participants were asked to remember as best as they could all of the sports which they had participated in between the ages of 9 to 18y. Subsequently, participants were asked to start in the current year, then work backwards from 18y to indicate sport participation at a given age that included
the 4 dimensions of number of sports played, highest level of achievement in any sport, the frequency of participation per week (in hours), and the number of the months of the year in which the sport season(s) took place (Appendix B).

The strong positive intercorrelations among the 4 dimensions fell in the range of $r = 0.296$ to $r = 0.631$ (Table 17, in Appendix L), indicating that they could be used to produce a single composite sport participation variable. The composite sport participation score was calculated using a rectified summation of individual $z$-scores for correlated variables of the sport participation questionnaire and is represented in equation 1:

Eq. 1:  
$$cSP_i = z_{Ni} + z_{Mi} + z_{fi} + z_{HLi}$$

Where $cSP$ is the composite sport participation score for a specific individual ($i$), $z$ are the individual $z$-scores for number of sports played in a given year ($N$), months of sport participation in a given year ($M$), frequency of sport participation per week in a given year ($f$), and highest level attained for any sport in a given year ($HL$). Using $z$-scores resulted in all domains being equally weighted in the calculation of sport participation. In order to generate a participation score without negative values, each individual’s composite sport participation score was transformed using equation 2:

Eq. 2:  
$$tSP_i = cSP_i + |cSP_{min}|$$

49
Where $tSP_i$ is the transformed sport participation score for individual ($i$), $cSP$ is the composite sport participation score for a given individual, and $cSP_{min}$ is the minimum sport participation composite score across the sample. Total sport participation ($TSP_{9\text{to}18}$) in youth was initially determined as the sum of $tSP_i$ scores across and inclusive of the ages 9-18 y (equation 3).

\[
TSP_{9\text{to}18} = \sum_{y=9}^{18} tSP_{(y)}
\]

The TSP score exhibited intercorrelations of $r = 0.296$ to $r = 0.631$, $p=0.000$ (table 17). The pattern of TSP across the ages of 9y to 18y (figure 6) revealed an inverted ‘U’ shaped curve suggesting a peak of TSP around the ages of 13. This was confirmed by one-way repeated measures ANOVA with the independent repeated variable of age. At 13y, TSP was greater than at all other ages (figure 6).
Figure 6: Age versus average sport participation. Sport participation, expressed as arbitrary units, for each year is the sample mean of tSPi of the number of sports played in each year, number of months playing sports in each year, highest sport level attained in each year, and the weekly frequency of sport participation in hours for each participant. Points represent mean scores across that age ± SD, N=92. A one-way repeated measures ANOVA revealed a significant main effect for age. Post hoc analysis revealed several differences between ages, however, for clarity, significance is only shown at 13y which was the only age observed to significantly differ from all other ages (*, p<0.05).
Consequently, TSP was divided into two separate scores that encompassed sport participation across 2 equal age ranges: 9y to 13y (TSP\textsubscript{early}: equation 4), and 14y to 18y (TSP\textsubscript{late}: equation 5).

Eq. 4:
\[
\text{TSP\textsubscript{early}} = \sum_{9 \leq y \leq 13} \text{tSP}\textsubscript{y(y)}
\]

Eq. 5:
\[
\text{TSP\textsubscript{late}} = \sum_{14 \leq y \leq 18} \text{tSP}\textsubscript{y(y)}
\]

Given that one individual indicated that she had not participated in sport across the retrospective timeline, all sport participation scores were truly 0 bounded at the lower end (i.e. a score of 0 is representative of no sport participation).

2D:4D Collection

The 2D:4D ratio (2DR) was measured as an indicator of pre-natal androgens (Lutchmaya, Baron-Cohen, Raggatt, Knickmeyer, & Manning, 2004), however the 2DR ratio does not appear to be predictive of sT in adult females (Crewther, Cook, Kilduff, & Manning, 2015). The 2DR data was collected using a flatbed scanner (Cannon CanoScan LiDE 110 Flatbed Scanner), in which the participants were asked to place both of their hands, with their fingers spread out onto the scanner bed. Subsequently, a black towel was placed over the hands of the participants to increase the contrast of the final scan. Prior to collection, participants were asked to remove all rings and jewelry, which might have been detrimental to the later analysis. Scans were performed in colour at a...
resolution 2400 dpi and the images were subsequently saved with only their unique identifiers as filenames.

Analysis of the 2DR ratio was performed by 3 separate raters using the Image J program (available for download at https://imagej.nih.gov/ij/, developed by Wayne Rasband at the Research Services Branch of the National Institute of Mental Health), which offers freeware picture image processing and analysis in a java format. Pictures were uploaded into the Image J program. Images were subsequently set to an 8-bit image type (gray scale), and rotated 90 degrees to the left so that the hands were in the upright position. Second digit and fourth digit finger lengths were determined using the line and measure function on the Image J program. Land marks for each finger consisted of the distal, center apex of each finger, and the middle of the basal, proximal crease of the finger (figure 7).
Figure 7: Representative hand scan image. Second digit (2D) and fourth digit (4D) finger lengths were determined using the line and measure function on the Image J program. Landmarks for each finger consisted of the distal, center apex of each finger (upper white lines). BC – basal crease.
Intra and inter rater reliability were determined and a mean of the 3 raters was used in statistical analyses.

Intra-rater reliability was assessed with intraclass correlation coefficients. An ICC two-way mixed effects model with absolute-agreement definition was used. The intra-rater reliability test was performed a minimum of 2 weeks after the final analysis on 3 random hands (Table 1).
Table 1: Intra-rater reliability for 2DR measurements.

<table>
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<tr>
<th>Examiner</th>
<th>ICC Agreement</th>
<th>95% CI</th>
</tr>
</thead>
<tbody>
<tr>
<td>Examiner A</td>
<td>0.988</td>
<td>0.825 - 1.000</td>
</tr>
<tr>
<td>Examiner B</td>
<td>0.930</td>
<td>0.760 - 1.000</td>
</tr>
<tr>
<td>Examiner C</td>
<td>0.996</td>
<td>0.883 - 1.000</td>
</tr>
</tbody>
</table>

*Note. ICC – Intraclass correlation, CI – Confidence Interval*
Inter-rater reliability was assessed with intraclass correlation coefficients. An ICC two-way mixed-effects model with absolute-agreement definition was used (Bescos et al., 2009) and turned out to be excellent (Average Measures ICC = 0.917, Confidence Interval 0.878-0.944, p=0.000).

Salivary Testosterone (sT)

Saliva was collected using the SalivaBio Oral Swab SOS from Salimetrics (product # 5001.02). Saliva samples were taken between 9-12am in order to account for the circadian fluctuation in androgen concentrations. Participants were instructed not to brush, floss or have dental work done at least 30 minutes before testing. Further, they were asked to avoid food for one hour before collection as food particles may skew the results. Participants were asked to rinse their mouth in order to ensure no food particles are present. After waiting 10 minutes (to avoid sample dilution) the swab was placed under the participant’s tongue by the researcher for exactly two minutes. Participants were sitting in a chair during collection and were instructed not to talk while the swab is in their mouth. After collection, the Oral Swab was placed in the swab collection tube from Salimetrics (product # 5001.05) and subsequently centrifuged for 15 minutes at 1000xg. The saliva sample was immediately stored at -70°C until analysis was performed.

A Testosterone Salivary Immunoassay Kit (ELISA) was used for analysis of the saliva samples (Salimetrics, product #1-2312). The correlation between serum and sT, as determined by Salimetrics, is r=0.61.
The hormones which show cross reactivity for the ELISA Kit greater than 1% are: Androstenedione (1.157%), Dihydrotestosterone (36.4%), 11-Hydroxytestosterone (1.90%) and 19-nortestosterone (21.02%). However, salivary DHT concentrations in normal, healthy adults, are less than 10pg/mL (Wang, Wakelin, White, & Wood, 1986) (i.e. well below the concentration of 500ng/ml used to test cross reactivity) and 19-nortestosterone is absent in healthy males and females (Reznik, Herrou, Dehennin, Lemaire, & Leymarie, 1987).

Analysis of sT was conducted according the kit instructions. Briefly, 25μL of each saliva sample were pipetted into antibody coated wells of a 96-well microplate along with standards and controls. After incubation with a conjugate solution (contains: testosterone conjugated to Horseradish Peroxidase, preservative) for 1 hour, the plate was washed and subsequently incubated with 200μL of TMB substrate for 30 minutes in the dark. After stopping the enzyme reaction, the plate was read on a plate reader at 450 nm. These methods for sT measurement are in accordance with the Testosterone Salivary Immunoassay kit (ELISA/EIA), and have been performed in our lab previously.

Physical Testing

Subsequent to anthropometric measurements of height and weight, muscular power was assessed using a hand dynamometer (Cardio Grip IBX H-101, Zona Health). The hand grip protocol was adapted from the Tufts, Brown and John Hopkins Hand Grip Strength Protocol. While standing, participants were asked to grip the dynamometer with their non-dominant hand so that the valley between their thumb and pointer finger was directly centered over the force gauge, with their other fingers wrapped around the device. Participants were asked to stand with their hands at their sides, arms not touching
their body. Participants were instructed to perform a maximal voluntary contraction for 3-5 seconds in duration. After a 2 minute rest, this procedure was performed an additional 3 times. An average of all 3 scores was used for the final analysis. The hand dynamometer was chosen due to the efficiency and practicality of the measure. Further, hand dynamometer scores have been used previously in order to characterize overall strength (Bohannon, 2001).

**Godin Leisure Time Exercise Questionnaire**

The LTEQ is a simple, self-explanatory questionnaire that estimates leisure time physical activity, and can be viewed in Appendix C. It has been validated and proven reliable in a number of studies (G Godin, 1997). The LTEQ required that participants report on how many days in the previous 7 days they had engaged in strenuous physical activity (e.g. jogging, running, basketball), moderate physical activity (fast walking, baseball, volleyball) and mild exercise (e.g. yoga, golf, fishing) for more than 15 minutes of their free time. Weekly frequencies of strenuous, moderate, and light activities were multiplied by 9, 5, and 3 METs, respectively. Total weekly leisure activity was calculated in arbitrary units by summing the products of the separate components, the results of which correlate strongly with overall fitness levels (G. Godin & Shephard, 1985). Total leisure activity is correlated with maximal oxygen consumption, although the time spent in vigorous exercise appears to be even more predictive of maximal oxygen consumption (G Godin & Shephard, 1985).

**The Scale of Children’s Action Tendencies in Sport**

The Scale of Children’s Action Tendencies in Sport (SCATS) was used to measure self-reported aggression tendencies in a sport specific context (Appendix D).
Although the scale was primarily used for children, and the participants in the study will be aged 18-30. The SCATS aggression score was divided into physical and non-physical responses. Scoring of the SCATS involved summing the number of times the aggressive responses were chosen. On this version of the SCATS, the possible aggression scores ranged from 0-12. The validity of the SCATS was tested by its relation to the CATS subscale, which yielded medium to high correlations including Assertion (r=0.53, p >0.001), Physical, (r=0.74, p< 0.001), and Non-Physical Aggression (r=0.68, p<0.001), and Submission (r=0.58, p<0.001). The validity of SCATS during its creation was also tested against a behavioral index provided by teacher ratings. Correlations between the average teacher behavioral ratings and SCATS subscales were significant for all but the submission subscale and included Assertion (r=.24, p<0.01), Physical Aggression (r=0.33, p<0.01), Verbal Aggression (r=0.22, p<0.01), and Submission (r=0.06, p<0.01).

The internal consistency reliability of the SCATS as a whole was 0.85.

**The Buss-Perry Aggression Questionnaire**

The Buss-Perry Aggression Questionnaire measures aggression by separating it into four subcategories: physical aggression, verbal aggression, anger and hostility. The Bus-Perry Aggression Questionnaire can be viewed in Appendix E. It has been validated and proven reliable in a number of studies (Buss & Perry, 1992). There are 29 total statements in which each participant was asked to give a rating of 1-5 as a rating of how either uncharacteristic (1) or characteristic (5) each statement best describes themselves. Questions 9 and 16, as indicated by an asterisk on the questionnaire, was scored in reverse. The total of all of the responses are summed and this gives an overall rating of aggression as a score out of 145 (29 questions x 5 possible marks per question).
The Sport Orientation Questionnaire

The sport orientation questionnaire (SOQ) measured three factors in regards to the competitiveness in sport, which was used to discriminate those who are competitive in a sport specific setting and those who are not (Appendix I). The SOQ contains 25 items and uses a 5-point Likert scale in order to measure the participant’s agreement to each item. The items were divided into three subscales including competitiveness (13 items), win orientation (6 items) and goal orientation (6 items) which are all separate, but related factors in the questionnaire. Competitiveness was defined as the desire to enter and strive for success in sport competition. Goal orientation is an orientation towards personal standards, regardless of the situation. Lastly, win orientation is the desire to win in a sport specific situation although it was not related to general individual achievement orientation. The internal consistency reliability of the subscales are as follows: competitiveness 0.94, win orientation 0.86 and goal orientation 0.80 (Petroczi, 2007).

Statistical Analyses

All statistical analyses were performed using IBM SPSS Statistics (Version 24). In all analyses, a p-value of less than 0.05 was set for statistical significance. Prior to statistical analyses, the raw data was assessed to make sure entry methods were congruent across all participants. For example, The LTEQ asked participants to indicate the number of times per week that they participated in Strenuous Physical Activity for bout of 15mins or more. If the participant entered the number in word form, the entry was transformed to a numerical entry. For example, the entry “two” would be transformed to 2. Further, if the participant entered a range into the same question, the average of the range was used
for analysis. For example, if the participant entered “4-5” as a response, the value of 4.5 was used.

During the data assessment, incidences of missing data also needed to be accounted for. For example, one participant did not have a hand scan done in order to analyze 2D:4D. Due to the missing data, this person was excluded from all analyses where 2D:4D was included. The number of participants (N) was reported for each analysis. There were no other issues concerning missing data within the thesis.

The following question was asked during the demographic questionnaire “Are you currently on any supplements, medications (prescribed or not) that could potentially alter your testosterone concentrations?” There were 5 individuals who answered YES to this question. Upon further analysis, none of these participants had testosterone values which were outside of the mean (32.53, 42.60, 23.75, 93.31 and 79.26 pg/ml, respectively). Therefore, the decision was made to include these participants in the analyses which included salivary testosterone.

An Analysis of Variance (ANOVA) was performed in order to determine the difference between TSP across all age groups (9-18 years old). Prior to performing the ANOVA, the following assumptions were tested.

1. Assumption 1: The normality of the dependent variable for each age group was tested using the Shapiro-Wilks test.

2. Assumption 2: The homogeneity of variances was tested using the Levene’s Test for Homogeneity of Variances
3. Assumption 3: The assumption of independence of observations was met based on the fact that the TSP of one person would have no impact on the TSP of another person.

Salivary testosterone concentrations were taken on 2 separate occasions, approximately 2 weeks apart. Of the 92 total participants, there were 15 people who did not return for a second saliva sample. However, for the 77 participants who did have 2 saliva samples, there was a significant correlation found between the first and second sample (r=0.630, p=0.000). As such, the decision was made to use the first saliva sample for the 92 participants in the primary data analyses.

A bivariate Pearson correlation (p<0.005) was computed between the independent variables (IV), salivary testosterone concentration and 2D:4D ratio (2DR), and the dependent variable (DV), sport participation in each of its 4 dimensions, TSP_{9to18}, TSP_{early}, TSP_{late}, and TSP_{NOW}. All variables were continuous and there were no outliers (defined as 2.5 standard deviations above or below the mean). The assumption of linearity was checked using a scatter plot of the data.

Subsequent to the initial correlations, Bivariate Pearson correlations were performed on continuous supplemental data collected, including alternate biological (max hand grip, body mass index (BMI), age of first menarche, and birth month) and psychological (SOQ, Buss-Perry AQ, SCATS) IVs with the main outcome variables of TSP_{9to18}, TSP_{early}, TSP_{late}, and TSP_{NOW}. All IVs that were found to be significantly correlated (p<0.005) with the outcome variables were included in an enter-method multiple linear regression analysis. Further, the following potential social confounding
variables were dummy coded into the following dichotomous (0,1) variables: use of birth control (none = 0), presence of older brothers (none = 0), ethnicity (Caucasian = 0), and mother’s highest education level (above high school = 0). Prior to performing linear regression, the following assumptions were tested:

1. Assumption 1: A linear relationship between the independent and dependent variables was tested by performing correlations (above) and analyzing scatter plots.

2. Assumption 2: Normality between residuals in the regression was tested by reviewing Q-Q plots (Appendix M).

3. Assumption 3: To determine if there is multicollinearity in the data, the Variance Inflation Factors (VIFs) were analyzed. Any VIFs over 10 were deemed unsatisfactory for a multiple linear regression. VIFs for the regressions in the present study ranged from 1.030–1.338.

4. Assumption 4: To test for autocorrelation, the Durbin-Watson test in SPSS was used. The acceptable range for the Durbin–Watson test is 1.5-2.5, Durbin–Watson values in the current study ranged from 1.776 – 1.847.

5. Assumption 5: To test for homoscedasticity, scatter plots of the IVs vs. the DV were inspected to make sure the error terms along the regression line were equal (Appendix N).

Since a regression allows the possibility for a set of variables to have joint predictive capabilities even when individually, they may not, and because 2DR and testosterone formed the main hypotheses of this thesis, in all regression analysis, 2DR and testosterone were included in the multiple linear regressions. Power analysis was
performed post-hoc using SPSS.

Results

Participants

Ninety-two participants (females aged 18-30 y) were recruited from the University of Windsor and surrounding area following University of Windsor research ethics board clearance (REB # 33486, Appendix K). Anthropometric and demographic data of the participants are outlined in Table 2. All participants had, or were in the completion of a post-secondary education. Only 1 participant indicated that she had not participated in sport in any year in the retrospective timeline.
Table 2: Anthropometric and demographic data of participants

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<th>Anthropometric Data</th>
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<td>Age</td>
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<td></td>
</tr>
<tr>
<td>BMI (kg/m²)</td>
<td>24.40 ± 4.56</td>
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</tr>
<tr>
<td>Height (cm)</td>
<td>168.29 ± 6.93</td>
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</tr>
<tr>
<td>Weight (kg)</td>
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<th>Ethnicity (%)</th>
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<td>Caucasian</td>
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<td></td>
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*Note: Anthropometric data is presented as means ±SD. Ethnicity is presented as percent of the sample (number of participants, N=92). University major and contraceptive use are presented as number of participants.*
Primary Analyses

Salivary testosterone (sT) and 2DR were analyzed against the 4 dimensions of sport participation (i.e. TSP_{9\to18}, TSP_{early}, TSP_{late} and TSP_{current}) using a bivariate Pearson correlation. None of the correlations were significant. Results of the correlation are in Table 3. 2DR (figure 8) and sT are shown graphically in plots against TSP_{9\to18} (figure 9).
Table 3: *Pearson Correlation between TSP (4 dimensions), 2DR, and salivary testosterone (sT)*

<table>
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<tr>
<th></th>
<th>TSP&lt;sub&gt;9to18&lt;/sub&gt;</th>
<th>TSP&lt;sub&gt;NOW&lt;/sub&gt;</th>
<th>TSP&lt;sub&gt;early&lt;/sub&gt;</th>
<th>TSP&lt;sub&gt;late&lt;/sub&gt;</th>
<th>sT</th>
<th>2DR</th>
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<td>0.736**</td>
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*Note:* **Correlation is significant at the 0.01 level (2-tailed).*
Figure 8: Scatterplot of adolescent sport participation ($TSP_{9to18}$) and 2DR. AU = arbitrary units. Line of best fit and $r$ value are displayed ($p>0.05$).
Figure 9: Scatterplot of salivary testosterone and total sport participation ($TSP_{9018}$). AU = arbitrary units. Line of best fit and r value are displayed ($p>0.05$).
Secondary Analysis

Results of Bivariate Pearson Correlations between TSP$_{9\text{to}18}$, TSP$_{\text{early}}$, and TSP$_{\text{late}}$ revealed significant correlations between all three dimensions and maximum hand grip (HG$_{\text{max}}$), SOQ total, and SCATS total. For TSP$_{9\text{to}18}$, significant correlations were: HG$_{\text{max}}$ (r=0.406, p = 0.000), SOQ (r=0.475, p = 0.000) and SCATS (r=0.240, p = 0.021). For TSP$_{\text{early}}$, significant correlations were: HG$_{\text{max}}$ (r=0.272, p = 0.009), SOQ (r=0.362, p = 0.000) and SCATS (r=0.200, p = 0.055). For TSP$_{\text{late}}$, significant correlations were: HG$_{\text{max}}$ (r=0.518, p = 0.000), SOQ (r=0.545, p = 0.000) and SCATS (r=0.251, p = 0.016). Results of all correlations are in Tables 4 and 5.
Table 4: Alternative Biological Correlates of $TSP_{90-18}$, $TSP_{early}$ and $TSP_{late}$

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<th>$TSP_{early}$</th>
<th>$TSP_{late}$</th>
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<th>$sT$</th>
<th>BMI</th>
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<td>.896**</td>
<td>.406*</td>
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Note: ** Correlation is significant at the 0.01 level. * Correlation is significant at the 0.05 level.
Table 5: Alternative Behavioural Correlates of TSP9to18, TSP early and TSP late

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<th>2DR</th>
<th>SOQ SUM</th>
<th>Buss-Perry AQ SUM</th>
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** Correlation is significant at the 0.01 level. * Correlation is significant at the 0.05 level.
Regression Analyses

Factors significantly correlated with TSP$_{9to18}$, TSP$_{\text{early}}$ and TSP$_{\text{late}}$ were included into a standard enter method multiple linear regression as predictor variables (i.e. HG$_{max}$, SOQ and SCATS). The main biological variables of study (sT and 2DR) were also included. Potential confounders of testosterone (i.e. ethnicity and age of first menarche) as well as known sociological confounds (mother’s education level and older brothers) were included as control factors into the regression. Results of the initial regression (tables 6, 7 and 8) were as follows: TSP$_{9to18}$ (F(9, 81) = 4.609, p = 0.000), TSP$_{\text{early}}$ (F(9, 81) = 2.482, p = 0.015), and TSP$_{\text{late}}$ (F(9, 81) = 7.192, p = 0.000.). After initial regression analyses, due to no change in the relationships between 2DR and salivary testosterone, a backwards multiple linear regression was performed (tables 9, 10 and 11). Final and best predictor models were as follows for TSP$_{9to18}$ (F(9, 81) = 13.252, p = 0.000), TSP$_{\text{early}}$ (F(9, 81) = 7.373, p = 0.000), and TSP$_{\text{late}}$ (F(9, 81) = 21.512, p = 0.000).
Table 6: Model Equation 1 for TSP\textsubscript{90-18}

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<td>0.305</td>
<td>1.198</td>
<td>0.025</td>
</tr>
<tr>
<td>SOQ SUM</td>
<td>0.530</td>
<td>0.150</td>
<td>0.385</td>
</tr>
<tr>
<td>Ethnicity</td>
<td>-13.141</td>
<td>5.934</td>
<td>-0.206</td>
</tr>
<tr>
<td>Birth Control</td>
<td>1.388</td>
<td>5.020</td>
<td>0.026</td>
</tr>
<tr>
<td>Older Brothers</td>
<td>-8.343</td>
<td>5.407</td>
<td>-0.145</td>
</tr>
<tr>
<td>Mother’s Education</td>
<td>-0.927</td>
<td>7.073</td>
<td>-0.013</td>
</tr>
</tbody>
</table>

Note: Dependent Variable: TSP\textsubscript{90-18}. F(9, 81) = 4.609, p = 0.000.

Table 7: Model Equation 1 for TSP\textsubscript{Early}

<table>
<thead>
<tr>
<th>Model</th>
<th>Unstandardized Coefficients</th>
<th>Standardized Coefficients</th>
<th>Collinearity Statistics</th>
</tr>
</thead>
<tbody>
<tr>
<td></td>
<td>B</td>
<td>Std. Error</td>
<td>Beta</td>
</tr>
<tr>
<td>(Constant)</td>
<td>20.483</td>
<td>59.733</td>
<td></td>
</tr>
<tr>
<td>2DR</td>
<td>-0.212</td>
<td>0.601</td>
<td>-0.036</td>
</tr>
<tr>
<td>sT</td>
<td>-0.019</td>
<td>0.077</td>
<td>-0.026</td>
</tr>
<tr>
<td>HG\textsubscript{max}</td>
<td>0.145</td>
<td>0.170</td>
<td>0.097</td>
</tr>
<tr>
<td>SCATS SUM</td>
<td>0.245</td>
<td>0.813</td>
<td>0.033</td>
</tr>
<tr>
<td>SOQ SUM</td>
<td>0.283</td>
<td>0.102</td>
<td>0.330</td>
</tr>
<tr>
<td>Ethnicity</td>
<td>-7.985</td>
<td>4.028</td>
<td>-0.201</td>
</tr>
<tr>
<td>Birth Control</td>
<td>0.479</td>
<td>3.408</td>
<td>0.015</td>
</tr>
<tr>
<td>Older Brothers</td>
<td>-5.784</td>
<td>3.671</td>
<td>-0.161</td>
</tr>
<tr>
<td>Mother’s Education</td>
<td>1.698</td>
<td>4.802</td>
<td>0.037</td>
</tr>
</tbody>
</table>

Note: Dependent Variable: TSP Early. F(9, 81) = 2.482, p = 0.015.

Table 8: Model Equation 1 for TSP\textsubscript{Late}

<table>
<thead>
<tr>
<th>Model</th>
<th>Unstandardized Coefficients</th>
<th>Standardized Coefficients</th>
<th>Collinearity Statistics</th>
</tr>
</thead>
<tbody>
<tr>
<td></td>
<td>B</td>
<td>Std. Error</td>
<td>Beta</td>
</tr>
<tr>
<td>(Constant)</td>
<td>2.325</td>
<td>36.812</td>
<td></td>
</tr>
<tr>
<td>2DR</td>
<td>-0.196</td>
<td>0.370</td>
<td>-0.045</td>
</tr>
<tr>
<td>sT</td>
<td>-0.013</td>
<td>0.047</td>
<td>-0.023</td>
</tr>
<tr>
<td>HG\textsubscript{max}</td>
<td>0.351</td>
<td>0.105</td>
<td>0.319</td>
</tr>
<tr>
<td>SCATS SUM</td>
<td>0.060</td>
<td>0.501</td>
<td>0.011</td>
</tr>
<tr>
<td>SOQ SUM</td>
<td>0.247</td>
<td>0.063</td>
<td>0.393</td>
</tr>
<tr>
<td>Ethnicity</td>
<td>-5.157</td>
<td>2.483</td>
<td>-0.178</td>
</tr>
<tr>
<td>Birth Control</td>
<td>0.911</td>
<td>2.100</td>
<td>0.038</td>
</tr>
<tr>
<td>Older Brothers</td>
<td>-2.559</td>
<td>2.262</td>
<td>-0.097</td>
</tr>
<tr>
<td>Mother’s Education</td>
<td>-2.623</td>
<td>2.959</td>
<td>-0.079</td>
</tr>
</tbody>
</table>

Note: Dependent Variable: TSP Late. F(9, 81) = 7.192, p = 0.000.
### Table 9: Model Equation 2 for TSP \(9_{to18}\)

<table>
<thead>
<tr>
<th>Model</th>
<th>Unstandardized Coefficients</th>
<th>Standardized Coefficients</th>
<th>Collinearity Statistics</th>
</tr>
</thead>
<tbody>
<tr>
<td></td>
<td>B</td>
<td>Std. Error</td>
<td>Beta</td>
</tr>
<tr>
<td>(Constant)</td>
<td>-19.767</td>
<td>13.536</td>
<td>.1460</td>
</tr>
<tr>
<td>(HG_{max})</td>
<td>.514</td>
<td>.242</td>
<td>.213</td>
</tr>
<tr>
<td>SOQ SUM</td>
<td>.516</td>
<td>.137</td>
<td>.375</td>
</tr>
<tr>
<td>Ethnicity</td>
<td>-12.969</td>
<td>5.697</td>
<td>-.204</td>
</tr>
</tbody>
</table>

Note: Dependent Variable: TSP \(9_{to18}\). \(F(9,81) = 13.252, p = 0.000.\)

### Table 10: Model Equation 2 for TSP \(early\)

<table>
<thead>
<tr>
<th>Model</th>
<th>Unstandardized Coefficients</th>
<th>Standardized Coefficients</th>
<th>Collinearity Statistics</th>
</tr>
</thead>
<tbody>
<tr>
<td>(Constant)</td>
<td>5.104</td>
<td>8.214</td>
<td>.536</td>
</tr>
<tr>
<td>SOQ SUM</td>
<td>.325</td>
<td>.083</td>
<td>.378</td>
</tr>
<tr>
<td>Ethnicity</td>
<td>-8.631</td>
<td>3.806</td>
<td>-.217</td>
</tr>
<tr>
<td>Older Brothers</td>
<td>-6.164</td>
<td>3.479</td>
<td>-.171</td>
</tr>
</tbody>
</table>

Note: Dependent Variable: TSP Early. \(F(9,81) = 7.373, p = 0.000.\)

### Table 11: Model Equation 2 for TSP \(late\)

<table>
<thead>
<tr>
<th>Model</th>
<th>Unstandardized Coefficients</th>
<th>Standardized Coefficients</th>
<th>Collinearity Statistics</th>
</tr>
</thead>
<tbody>
<tr>
<td>(Constant)</td>
<td>-19.845</td>
<td>5.649</td>
<td>-3.513</td>
</tr>
<tr>
<td>(HG_{max})</td>
<td>.349</td>
<td>.101</td>
<td>.317</td>
</tr>
<tr>
<td>SOQ SUM</td>
<td>.253</td>
<td>.057</td>
<td>.403</td>
</tr>
<tr>
<td>Ethnicity</td>
<td>-5.158</td>
<td>2.378</td>
<td>-.178</td>
</tr>
</tbody>
</table>

Note: Dependent Variable: TSP late. \(F(9,81) = 21.512, p = 0.000\)
Discussion

The primary aim of the current study was to determine whether biological androgen and/or androgen exposure could predict female sport participation. It was hypothesized that early exposure to testosterone, as inferred from the 2DR, may predispose girls toward sport participation. In fact, this has been shown to some extent in males and females, where lower ratios appear to relate to fitness and level of sport (Bescos et al., 2009; Hönekopp, T. Manning, & Müller, 2006; Ranson, Stratton, & Taylor, 2015, Golby & Meggs, 2011). Testosterone is known to impact many traits that are beneficial to sport success, such as an increase in skeletal muscle mass (a determinant of strength and power), increase in cardiac size (resulting in a larger stroke volume), and a regulation of erythropoiesis which could potentially increase the oxygen carrying capacity of the blood.

In contrast to previous studies, neither testosterone nor 2DR was related to TSP_{90:18} alone (r = 0.032, p = 0.765 and r = -0.065, p=0.538, respectively) or when the potential for covariates and confounders were added in a linear regression. Previous research has shown an association between the digit ratios (2DR) of elite female fencers (n=87) and current and highest past rankings (Bescos et al., 2009). However, the athletes in the Bescos et al. study (2009) had all achieved world class ranking at some point in their past and this relation was true only when ethnicity and the rankings were accounted for (Bescos et al., 2009). Similarly, Paul et al., (2006) analyzed the 2DR ratio of 607 females (53.8 ± 8.5 y). Participants were asked to rate their highest level achieved (on a scale of 1 – 5 ranging from social participation only, to national level) for a total of 12 sports. When adjusted for age, the highest level achieved in any sport was significantly,
negatively associated with average 2DR ($b = -4.93$, $p = 0.01$). Consequently, it is possible that the relation between 2DR and sport success might only manifest in elite level athletes. Moreover, the average age of participants in this study were $22.32 \pm 2.68$ y, much younger than those in the aforementioned study (Paul et al., 2006). As such, some of the participants in the present study may not have reached their full potential and achieved to their highest level within the timeframe analyzed. Indeed, the average age of female Olympians is in the mid to late 20’s in many sports (Arti Patel, 2012). Nonetheless, only a small fraction of the population and present sample, specifically, would be expected to reach Olympic level at any point in their lives, and further, one of the central aims of the current study was to determine the relationship between androgens and sport participation, not simply highest level. Even though no significant relationship was found, it is interesting to note that the direction of the relationship was negative ($TSP_{9018}$: $r = 0.065$, $TSP_{early}$: $r = -0.047$, $TSP_{late}$: $r = -0.079$) which is suggestive of a lower 2DR ratio (i.e. indicative of higher prenatal testosterone) negatively influencing female sport participation.

Moreover, Paul et al., (2006) suggested that the performance of childhood athletes may not correlate well with best adult performance due to variability in development. Therefore, it may not be valid to look at adolescent sport participation as a sum, due to ranges in age of development (Paul et al., 2006). With respect to testosterone, although pubertal testosterone and adult testosterone appear to correlate (Apter & Vihko, 1990), the Mayo Clinic indicates that total testosterone values can range from 7-44ng/dl at the age of 10-11 y to 8-60ng/dl at 19 y. In order to control for this, TSP was divided into $TSP_{early}$ (9-13) and $TSP_{late}$ (14-18) and age of menarche was included as
a covariate in regression analyses in order to determine whether biological influences may have had a greater impact at later stages or for girls who began development later. Nonetheless, in all cases, neither testosterone nor 2DR was significantly related to sport participation. Further, neither sT nor 2DR was significantly correlated with TSP\textsubscript{NOW} ($r = 0.116$, $p = 0.317$, and $r = -0.162$, $p = 0.125$, respectively), though it should be noted that for both, the r values were the strongest and p values the lowest when analyzed against TSP\textsubscript{NOW}. Interestingly, traits that would otherwise correlate with sport participation such as youth fitness have been shown to correlate with 2DR ratio in young boys and girls (Hönekopp et al., 2006), however, that and recent evidence suggests that this effect appears much stronger in boys (Ranson et al., 2015).

The mean sT values of the participant sample (60.18 ± 23.44 pg/ml) were within the reference ranges for females provided by the Salimetrics ELISA Kit (48.47 ±38.44 pg/ml). Although the sT concentrations are slightly higher than average, they were similar to previous studies which have analyzed sT in athletes (Cook & Beaven, 2013)(Blair Crewther et al., 2015), and the pilot data collected for this thesis. This supports the validity of the sT concentrations found. To the knowledge of the researcher, this is the only study thus far which has analyzed sT and sport participation in a sample that included recreational athletes, as most studies have been completed on elite athletes or sport participation has not been recorded (Cook & Beaven, 2013)(B. T. Crewther, Hamilton, Casto, Kilduff, & Cook, 2015). Moreover, only one participant in the sample reported that they had not participated in sports at any time during their life. The mean moderate-strenuous leisure time physical activity score of the group (as determined by the Godin Leisure-Time Physical Activity Questionnaire), was 40 ± 24.2 units. Godin,
(2011) developed three categories to group activity levels based on the subtotal of moderate and strenuous activity: active (24 units or more) moderately active (14-23 units) and insufficiently active (less than 14 units). On average, participants from the current sample showed ranges in the active category. Approximately 72% of the participants in the study were either currently or had been previously enrolled in a university Kinesiology program. Results from the pilot data are in accordance with the current study, which found that students in Kinesiology exhibit greater activity levels than other majors. Further, those who participate in organized sport during adolescence, have on average, higher weekly hours of moderate-to-vigorous physical activity into young adulthood (Walters, Barr-Anderson, Wall, & Neumark-Sztainer, 2009). Therefore, the active categorization of the current study sample is not surprising, considering all participants excluding one, had participated in sport at some point in their life. However, the variation in total sport participation across all participants, does allow for some speculation as to the influences of sport participation on athletes through adolescence.

Potential confounders of the current participant sample were indicative of a higher sport participation rate. For example, ethnicity was a significant predictor of TSP$_{9018}$, TSP$_{early}$ and TSP$_{late}$. Interestingly, European females who are a part of an ethnic minority, participate in sport less frequently than females who belong to an ethnic majority (Elling & Knoppers, 2005). In the present sample, approximately 77% of participants in the current sample self-identified as Caucasian.

Also, socio-economic status (SES) is known to influence sport participation such that the higher the SES, the greater the sport participation (White & McTeer, 2012). A proxy for SES of children and adolescents is parental education (Walters et al., 2009)
however, maternal education appears to be the strongest predictor of childhood SES (Hoff & Tian, 2005). In the current sample, approximately 72% of participants had mothers who had attended post-secondary education (ranging from a college to a PhD degree). This is greater than the national average of Canadians who have acquired a post-secondary degree at 64.1% (Statistics Canada). The higher maternal education level of this sample could have had a positive effect on the total sport participation of the current females.

One of the strongest correlations found was the association of TSP<sub>9to18</sub>, TSP<sub>early</sub> and TSP<sub>late</sub> with a marker of upper body strength [i.e. maximum hand grip (HG<sub>max</sub>)]. Not only was HG<sub>max</sub> correlated on its own, but according to structure coefficients, it contributed 35% and 43.3% to the predicted sport participation score in the regression models for TSP<sub>9to18</sub> and TSP<sub>late</sub>, respectively. However, causality is unknown. It is possible that by participating in sport, an athlete is exposed to environments and situations in which strength may increase. Conversely, a person who is stronger initially might do better in a sport situation, thereby increasing their sport success. Indeed, it has been proposed that androgens help to develop grip strength in men and women, although this relation may be subdued in females (Isen, McGue, & Iacono, 2014). Interestingly, HG<sub>max</sub> was removed from the final model in the prediction for TSP<sub>early</sub>. This may suggest that strength is a result of sport participation given that TSP<sub>late</sub> would have been much closer in time to participation in this study. Nonetheless, the relationship between HG<sub>max</sub> and sport participation was clear even without a categorization of sport, which suggests that, strength is an important component of sport participation and achievement.
Noteworthy, is the correlation between \( TSP_{9to18} \) \((r = 0.240, p = 0.021)\) and \( TSP_{late} \) \((r = 0.251, p = 0.016)\) and the Scale of Children’s Action Tendencies in Sport (SCATS). As discussed previously, the SCATS is a questionnaire used to determine sport specific aggression tendencies. A significant amount of research has studied aggressive tendencies in athletes, and whether the aggressive tendencies are viewed as being inherent to the athlete, or are developed through the nature of the sporting environment (Kimble, Russo, Bergman, & Galindo, 2010). Results of this correlation were not surprising, considering aggression has previously been defined as a positive factor of performance (Rascale, Coulomb, & Pfister, 1998, Widmeyer & Birch, 1984). However, drawing conclusions across studies including sport aggression and sport participation/success should be taken with caution, as many studies define aggression in different ways. For example, a study assessing aggression in ice hockey players defined aggression as “an overt verbal or physical act that has the potential to physically or psychologically injure a person,” (Visek & Watson, 2005). However, some studies have chosen to segregate aggression into two forms: hostile and instrumental, (which defines the intent of the aggressive act as trying to gain a competitive advantage, rather than for malicious intent). Therefore, even though this classic definition still involves the intent to injure, based on the differentiation between competitive and malicious intent, it can still be viewed in a positive light, leading a player towards sport success. The SCATS score was also significantly correlated with the total score of the Buss-Perry Aggression Questionnaire (AQ), however, the AQ was not correlated with any range of sport participation and when the SCATS was added into regression analyses, it was not found to be a significant component of the final model, unlike the SOQ score. This suggests
that aggression is not as important as competitiveness to sport participation even though aggression can be used to gain a competitive advantage.

It has been suggested in the literature that prenatal and basal testosterone may be associated with aggressive tendencies. The majority of literature in this field uses the definition of aggression which implies malicious behaviour and intent to injure. Moreover, historically, males were thought to be more aggressive than females (Giammanco, Tabacchi, Giammanco, Di Majo, & La Guardia, 2005). The increased concentration of testosterone in males is taken as evidence for the link between testosterone and aggression (Book et al., 2001). There has been a positive relationship between testosterone and aggression established in non-humans, but this relationship is significantly less established in humans. In contrast, prenatal testosterone has been linked to aggressive tendencies, although these results have primarily been found in males (Bailey & Hurd, 2005, Dogan, Barut, Konuk, & Bilge, 2007, Kuepper & Hennig, 2007). However, in the current study, there were no correlations found between standard measures of aggression (Buss-Perry AQ) and sports aggression (SCATS) and either sT concentrations or 2DR (although once again, the negative direction of the relationship between 2DR and the SCATS should be noted).

As noted above, a strong relationship between competitiveness in sport situations (SOQ) and TSP_{9018}, TSP_{early}, and TSP_{late} was found (r=0.473, p=0.000, r = 0.362, p=0.000, and r = 0.545, p = 0.000, respectively), suggesting that the higher a female’s measure of competitiveness, the higher their sport participation score. There are three subscales (i.e. Win Orientation, Goal Orientation and Competitiveness) associated with the SOQ and the correlations of the subscales with TSP_{9018} were as follows: Win (r =
0.324, p = 0.002), Goal (r = 0.331, p = 0.001) and Competitiveness (r = 0.544, p = 0.000).

To the knowledge of the author, the SOQ has not been used as a measure to predict sport participation. The majority of studies have used the SOQ to determine the competitiveness levels in existing groups of athletes (Jamshidi, Hossien, Sajadi, Safari, & Zare, 2011, Manouchehri & Tojari, 2013). The original purpose of the SOQ in the current study was to aid in predicting sport participation, but due to the fact that all participants (excluding one) had or currently were participating in sport, comparisons to previous studies can be made.

For example, a study conducted by Finkenberg & Moode (1998), compared the sport orientation of a group of collegiate athletes (N=40) and non-athletes (N=36). The author found that collegiate athletes scored higher on the SOQ than non-athletes. When compared to the current study, the average scores on the subscales although slightly higher, were quite similar. The differences are defined as follows:

<table>
<thead>
<tr>
<th></th>
<th>Competitiveness</th>
<th>Goal Orientation</th>
<th>Win Orientation</th>
</tr>
</thead>
<tbody>
<tr>
<td>Finkenberg et al.,</td>
<td>48.2 ± 10.03</td>
<td>24.6 ± 3.7</td>
<td>20.6 ± 4.9</td>
</tr>
<tr>
<td>(1998)</td>
<td></td>
<td></td>
<td></td>
</tr>
<tr>
<td>Current Study</td>
<td>49.8 ± 12.0</td>
<td>21.8 ± 5.3</td>
<td>25.8 ± 4.0</td>
</tr>
</tbody>
</table>

It was interesting to note that of the psychological questionnaires used as supplementary data in this study, sport participation was highly correlated with the positive attributes (sport aggression and competitiveness) and not correlated with the
negative attribute (standard aggression). This result helps to support the notion that participating in sports contributes to fostering positive attributes in females, which has been shown in many previous studies (Fraser-thomas, Côté, Deakin, & Co, 2007, Troutman & Dufur, 2008, Holt, Tamminen, Tink, & Black, 2009).

Post-hoc power analyses resulted in power values ranging from 0.842 – 0.999 for all regression models. The power analyses were performed including all 92 participants, although a study utilizing a larger sample size would result in a stronger overall power. Moreover, a participant pool which included more females who did not participate in sport at any time in their life would add to the strength of the study.

Limitations

92 females were recruited for the present study. For standard multiple linear regression, a sample size of 15 individuals per variable entered in a prediction model is normally accepted. In our secondary analyses, 6 predictors and 3 dummy coded confounding variables were entered into regression analyses. However, using backward regression, the final model consisted of only 3 predictors and measures of statistical validity were deemed acceptable. Further, in retrospect, one aspect of the study design that might have skewed the type of individual who volunteered for the study was the study title. Due to the fact that the title included the words “sport participation” this might have deterred females who had not ever participated in sport from volunteering, thereby attracting only females with a sporting history. Nonetheless, a greater sample size and one that included more women who had never participated in sport would provide greater insights into female adolescent sport participation.
Another limitation of this study is the fact that our sport participation scores were primarily retrospective, while the questionnaires and sT measure were current. However, participants were asked to fill out the sport participation questionnaire for the current year, and therefore, TSP_{NOW} could be compared with the independent variables. In the majority of outcomes, correlations were similar (i.e. between variables) and final regression models included the same variables.

Measuring any hormone in humans comes with its own limitations. Testosterone varies throughout the day as well as throughout the menstrual cycle in females (although these changes are small in comparison to the changes in estrogen across the menstrual cycle). Therefore, testosterone samples were taken between 9-12am when testosterone concentrations were the highest to take into account its circadian fluctuation. Moreover, sT is not 100% correlated with free testosterone in serum (r = 0.61) and is less correlated to serum concentrations in females than males (Shirtcliff, Granger, & Likos, 2002). The saliva sample used to determine testosterone was only indicative of free testosterone within the body. A more accurate measure could have been determined through serum blood draws (which could have measured total testosterone), although this method may have negatively impacted the total number of participant volunteers. Further, free testosterone concentrations may not be indicative of testosterone’s impact on tissue due to potential ranges in testosterone receptor sensitivity or SHBG concentration (see literature review). Nonetheless, the use of sT in females has been used previously in sport literature (Cook & Beaven, 2013, B Crewther et al., 2012, Cook & Beaven, 2013, Gatti & De Palo, 2011).
The cSP developed for this thesis also presents limitations. The aim of the cSPi was to try to present an arbitrary value to sport participation. There were four dimensions chosen for this thesis, but it is possible that more than four factors could be relevant to a person’s sport participation (e.g. multiple leagues or overlap of different sports). It is a challenge of research to try and put an arbitrary number to a complex phenomenon. However, the current cSPi was modelled after a published report with similar dimensions.

Lastly, a limitation of this study was the use of simple proxies in order to try and quantify complex subjects. For example, 2DR was used as a proxy for prenatal androgen exposure, although this relationship has only been established in a few high impact, broad scientific journals (Voracek & Loibl, 2009), which is suggestive that this measure is not yet widely accepted (Lutchmaya et al., 2004). Another example of simple proxies is the use of single questionnaires to try and determine complex behavioural aspects (i.e. standard aggression, competitiveness, sport aggression). This is common practice in literature, and all questionnaires used had high validity and reliability, although to capture the true behavioural nature of any human is most likely impossible.

Conclusion

In summary, it does not appear that androgens, whether prenatally or current, impact female sport participation. However, it is interesting to note that the relationship between 2DR ratio and sport participation were negative in all relations though not significant. Participation in sport is a result of a complex relationship including but not limited to biological advantages, familial and peer influences, socioeconomic status, positive and negative experiences within sport, and chance. Given that women and girls participate in sport at lower rates than men and boys, and given that sport participation
provides social and health advantages throughout life for those who participate, attempting to determine what factors most influence sport participation is a necessary field of study.

**Future Directions**

Future studies should include a greater number of participants who have never been involved in sport. This would contribute to a better understanding of whether the correlates of sport participation found in this thesis are due to sport participation itself, or simply due to chance. Moreover, the current study was retrospective, a study with a cross-sectional design across the ages of 9 to 18 may provide better insights into the biological correlates of female sport participation.
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Appendix A

ELIGIBLE SPORTS

Snow sports
- Biathlon
- Bobsleigh
- Broomball
- Curling
- Dog Sledding
- Figure Skating
- Hockey
- Ice Sailing
- Luge
- Ringette
- Skeleton
- Ski Jumping
- Skiing
- Downhill/Alpine
- Skiing, Cross
- Country/Nordic
- Skiing, Nordic
- Combined
- Snowboarding
- Snowshoeing
- Speed Skating

Combat Sports
- Boxing
- Judo
- Karate
- Taw Kwon Do
- Wrestling
- Fencing

Water sports
- Canoeing
- Diving
- Kayaking

Track and Field
- Shot, Discuss, Hammer Throw
- High Jump, Long Jump, Javelin, Pole Vault
- Steeple Chase, Hurdles

Large Team Sports
- Ball Hockey
- Baseball
- Basketball
- Broomball
- Cheerleading
- Cricket
- Field Hockey
- Football, touch, flag
- In-line hockey
- Lacrosse
- Ringette
- Rugby
- Softball

Artic sports
- Traditional Aboriginal Sports

Skill Sports
- Archery
- Bowling (5 pin)
- Golf
- Lawn bowling
- Badminton
- Handball (4 walls)
- Netball
- Racquetball
- Squash
- Table Tennis
- Team Handball

Endurance Sports
- Adventure racing
- Canoeing
- Kayaking
- Skiing

Non-Traditional Sports
- BMX
- Cheerleading
- Climbing
- Cricket
- Orienteering
- Skateboarding
- Mountain Boarding
- Wakeboarding

In-line skating
- Kayaking
- Modern Pentathlon
- Mountain Biking
- Orienteering
- Running
- Cross Country
- Running, Road
- Triathlon
- Race Walking

Equestrian
- Power Lifting

Urban Road Sports
- BMX
- Cycling
- Inline Skating
- Running
- Road
- Skateboarding
- Race Walking

Multiple Sport
- Adventure Racing
- Modern Pentathlon

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Appendix B

Sport Participation Questionnaire

Please fill out the following questionnaire for ALL SPORTS that you participated in between the ages of 9-18 years of age. If you have any questions, please do not hesitate to ask the researcher for clarification.

**Highest level of participation:** please indicate the HIGHEST LEVEL ONLY of sport you participated in taking into consideration ALL SPORTS played during each year.

**Frequency of Activity:** please list the average number of hours you played a game or practiced (including official practice and/or team workout/lifting sessions) per week, on average during the months you participated in sport.

**Months of Sports Season:** please list the accumulated number of months in which you played sports during the year. Please round up to the nearest month.

2015/2016

Please fill out the following section for the past year.

<table>
<thead>
<tr>
<th>Number of Sports</th>
<th>Type here</th>
</tr>
</thead>
<tbody>
<tr>
<td>Highest Level of Participation</td>
<td>---</td>
</tr>
<tr>
<td>Weekly Frequency of Activity (in hours)</td>
<td>Type here</td>
</tr>
<tr>
<td>Months of Sport Season</td>
<td>---</td>
</tr>
</tbody>
</table>
Appendix C

Godin Leisure-Time Exercise Questionnaire

1. During a typical 7-Day period (a week), how many times on average do you do the following kinds of exercises for more than 15 minutes during your free time (write on each line the appropriate number).

Times Per Week

a. Strenuous Exercise
(HEART BEATS RAPIDLY)
(e.g. running, jogging, hockey, football, soccer, squash, basketball, cross country, skiing, judo, roller skating, vigorous swimming, vigorous long distance bicycling).

b. Moderate Exercise
(NOT EXHAUSTING)
(e.g. fast walking, baseball, tennis, easy bicycling, volleyball, badminton, easy swimming, alpine skiing, popular and folk dancing)

c. Mild Exercise
(MINIMAL EFFORT)
(e.g. yoga, archery, fishing from river bank, bowling Horseshoes, golf, snow-mobiling, easy walking)

During a typical 7-Day period (a week), in your leisure time, how often do you engage in any regular activity long enough to work up a sweat (heart beats rapidly)?

Often

Sometimes

Never/rarely
Appendix D

Scale of Children’s Action Tendencies in Sport

Imagine that …

1. You're playing basketball with some friends and a kid on the other team keeps shoving you to get the ball. What would you do?

*(Pick one choice from each pair of choices below)*

<table>
<thead>
<tr>
<th>A. Shove the kid back</th>
<th>Or</th>
<th>B. Tell the kid to stop</th>
</tr>
</thead>
<tbody>
<tr>
<td>(circle A or B)</td>
<td></td>
<td></td>
</tr>
</tbody>
</table>

<table>
<thead>
<tr>
<th>C. Pretend it didn't bother me</th>
<th>Or</th>
<th>D. Shove the kid back</th>
</tr>
</thead>
<tbody>
<tr>
<td>(circle C or D)</td>
<td></td>
<td></td>
</tr>
</tbody>
</table>

<table>
<thead>
<tr>
<th>E. Tell the kid to stop</th>
<th>Or</th>
<th>F. Pretend it didn't bother me</th>
</tr>
</thead>
<tbody>
<tr>
<td>(circle E or F)</td>
<td></td>
<td></td>
</tr>
</tbody>
</table>

2. You're playing soccer and a player from the other team trips you on purpose. What would you do?

*(Pick one choice from each pair of choices below)*

<table>
<thead>
<tr>
<th>A. Tell the player not to do that again</th>
<th>Or</th>
<th>B. Stay away from that player the rest of the game</th>
</tr>
</thead>
<tbody>
<tr>
<td>(circle A or B)</td>
<td></td>
<td></td>
</tr>
</tbody>
</table>

<table>
<thead>
<tr>
<th>C. Get even by tripping the player later</th>
<th>Or</th>
<th>D. Tell the player not to do that again</th>
</tr>
</thead>
<tbody>
<tr>
<td>(circle C or D)</td>
<td></td>
<td></td>
</tr>
</tbody>
</table>

<table>
<thead>
<tr>
<th>E. Tell the player not to do that again</th>
<th>Or</th>
<th>F. Stay away from that player the rest of the game</th>
</tr>
</thead>
<tbody>
<tr>
<td>(circle E or F)</td>
<td></td>
<td></td>
</tr>
</tbody>
</table>

3. You are playing hockey and the kids on your team really want to win. Some kids on your team want to try to hurt the best player on the other team. They ask you to help them. What would you do?
(Pick one choice from each pair of choices below)

<table>
<thead>
<tr>
<th>A. Help the kids on my team hurt the other player</th>
<th>Or</th>
<th>B. Tell the kids on my team I don't hurt people</th>
</tr>
</thead>
<tbody>
<tr>
<td>(circle A or B)</td>
<td></td>
<td></td>
</tr>
</tbody>
</table>

<table>
<thead>
<tr>
<th>C. Just pretend to help, but not really try to hurt the player</th>
<th>Or</th>
<th>D. Help the kids on my team hurt the other player</th>
</tr>
</thead>
<tbody>
<tr>
<td>(circle C or D)</td>
<td></td>
<td></td>
</tr>
</tbody>
</table>

<table>
<thead>
<tr>
<th>E. Tell the kids on my team I don't hurt people</th>
<th>Or</th>
<th>F. Just pretend to help, but not really try to hurt the player</th>
</tr>
</thead>
<tbody>
<tr>
<td>(circle E or F)</td>
<td></td>
<td></td>
</tr>
</tbody>
</table>

4. You are playing hockey and a fight starts. Some kids from your team are fighting kids from the other team. What would you do?

(Pick one choice from each pair of choices below)

<table>
<thead>
<tr>
<th>A. Stay far away from the fight</th>
<th>Or</th>
<th>B. Try to stop the kids from fighting</th>
</tr>
</thead>
<tbody>
<tr>
<td>(circle A or B)</td>
<td></td>
<td></td>
</tr>
</tbody>
</table>

<table>
<thead>
<tr>
<th>C. Join the fight</th>
<th>Or</th>
<th>D. Stay far away from the fight</th>
</tr>
</thead>
<tbody>
<tr>
<td>(circle C or D)</td>
<td></td>
<td></td>
</tr>
</tbody>
</table>

<table>
<thead>
<tr>
<th>E. Try to stop the kids from fighting</th>
<th>Or</th>
<th>F. Join the fight</th>
</tr>
</thead>
<tbody>
<tr>
<td>(circle E or F)</td>
<td></td>
<td></td>
</tr>
</tbody>
</table>
Appendix E

**Buss-Perry Aggression Questionnaire**

Using the 5-point scale shown below, indicate how uncharacteristic or characteristic each of the following statements is in describing you. Check off the box which corresponds with your rating in the box to the right of the statement.

1 = extremely uncharacteristic of me  
2 = somewhat uncharacteristic of me  
3 = neither uncharacteristic nor characteristic of me  
4 = somewhat characteristic of me  
5 = extremely characteristic of me

<table>
<thead>
<tr>
<th></th>
<th>1</th>
<th>2</th>
<th>3</th>
<th>4</th>
<th>5</th>
</tr>
</thead>
<tbody>
<tr>
<td>1</td>
<td></td>
<td></td>
<td></td>
<td></td>
<td></td>
</tr>
<tr>
<td>2</td>
<td></td>
<td></td>
<td></td>
<td></td>
<td></td>
</tr>
<tr>
<td>3</td>
<td></td>
<td></td>
<td></td>
<td></td>
<td></td>
</tr>
<tr>
<td>4</td>
<td></td>
<td></td>
<td></td>
<td></td>
<td></td>
</tr>
<tr>
<td>5</td>
<td></td>
<td></td>
<td></td>
<td></td>
<td></td>
</tr>
</tbody>
</table>

1. Some of my friends think I am a hothead.
2. If I have to resort to violence to protect my rights, I will.
3. When people are especially nice to me, I wonder what they want.
4. I tell my friends openly when I disagree with them.
5. I have become so mad that I have broken things.
6. I can’t help getting into arguments when people disagree with me.
7. I wonder why sometimes I feel so bitter about things.
8. Once in a while, I can’t control the urge to strike another person.
9. I am an even-tempered person.
10. I am suspicious of overly friendly strangers.
11. I have threatened people I know.
12. I flare up quickly, but get over it quickly.
13. Given enough provocation, I may hit another person.
14. When people annoy me, I may tell them what I think of
<p>| | |</p>
<table>
<thead>
<tr>
<th></th>
<th></th>
</tr>
</thead>
<tbody>
<tr>
<td>15.</td>
<td>I am sometimes eaten up with jealousy.</td>
</tr>
<tr>
<td>16.</td>
<td>I can think of no good reason for ever hitting a person.</td>
</tr>
<tr>
<td>17.</td>
<td>At times I feel I have gotten a raw deal out of life.</td>
</tr>
<tr>
<td>18.</td>
<td>I have trouble controlling my temper.</td>
</tr>
<tr>
<td>19.</td>
<td>When frustrated, I let my irritation show.</td>
</tr>
<tr>
<td>20.</td>
<td>I sometimes feel that people are laughing at me behind my back.</td>
</tr>
<tr>
<td>21.</td>
<td>I often find myself disagreeing with people.</td>
</tr>
<tr>
<td>22.</td>
<td>If somebody hits me, I hit back.</td>
</tr>
<tr>
<td>23.</td>
<td>I sometimes feel like a powder keg ready to explode.</td>
</tr>
<tr>
<td>24.</td>
<td>Other people always seem to get the breaks.</td>
</tr>
<tr>
<td>25.</td>
<td>There are people who pushed me so far that we came to blows.</td>
</tr>
<tr>
<td>26.</td>
<td>I know that “friends” talk about me behind my back.</td>
</tr>
<tr>
<td>27.</td>
<td>My friends say that I’m somewhat argumentative.</td>
</tr>
<tr>
<td>28.</td>
<td>Sometimes I fly off the handle for no good reason.</td>
</tr>
<tr>
<td>29.</td>
<td>I get into fights a little more than the average person.</td>
</tr>
</tbody>
</table>
Appendix F

Table 12: Progesterone Reference Ranges

<table>
<thead>
<tr>
<th></th>
<th>Males</th>
<th>Females</th>
</tr>
</thead>
<tbody>
<tr>
<td>0-23 months</td>
<td>0.87-3.37 ng/mL</td>
<td>0.87-3.37 ng/mL</td>
</tr>
<tr>
<td>2-9 y</td>
<td>&lt;0.15 ng/mL</td>
<td>0.20-0.24 ng/mL</td>
</tr>
<tr>
<td>10-17 y</td>
<td>Adult levels are</td>
<td>Values increase</td>
</tr>
<tr>
<td></td>
<td>attained by puberty</td>
<td>through puberty and</td>
</tr>
<tr>
<td></td>
<td></td>
<td>adolescence</td>
</tr>
<tr>
<td>≥ 18 y</td>
<td>0.20-1.40 ng/mL</td>
<td>Levels vary</td>
</tr>
<tr>
<td></td>
<td></td>
<td>throughout the</td>
</tr>
<tr>
<td></td>
<td></td>
<td>menstrual cycle, see</td>
</tr>
<tr>
<td></td>
<td></td>
<td>table 2.</td>
</tr>
</tbody>
</table>

*Adapted from Lippe, LaFranchi, Lavin et al., (1974).

Table 13: Pre-Menopausal Progesterone Reference Ranges

<table>
<thead>
<tr>
<th>Phase</th>
<th>Range</th>
</tr>
</thead>
<tbody>
<tr>
<td>Follicular Phase</td>
<td>0.20-1.50 ng/mL</td>
</tr>
<tr>
<td>Ovulation Phase</td>
<td>0.80-3.00 ng/mL</td>
</tr>
<tr>
<td>Luteal Phase</td>
<td>1.70-27.00 ng/mL</td>
</tr>
<tr>
<td>Postmenopausal</td>
<td>&lt;0.15-0.80 ng/mL</td>
</tr>
</tbody>
</table>

*Adapted from Lippe, LaFranchi, Lavin et al., (1974).
## Appendix G

### Table 14: Estradiol Concentrations

#### Free Estradiol – Serum

<p>| | |</p>
<table>
<thead>
<tr>
<th></th>
<th></th>
</tr>
</thead>
<tbody>
<tr>
<td>Adult Males</td>
<td>0.2 – 1.5pg/mL</td>
</tr>
<tr>
<td>Adult Females</td>
<td>0.6 – 7.1pg/mL</td>
</tr>
</tbody>
</table>

#### Total Estradiol – Serum

<p>| | |</p>
<table>
<thead>
<tr>
<th></th>
<th></th>
</tr>
</thead>
<tbody>
<tr>
<td>Newborns (Males + females)</td>
<td>Concentrations are elevated at birth and fall rapidly during the first week to values &lt; 15pg/mL</td>
</tr>
<tr>
<td>Males &lt; 6m</td>
<td>Concentrations increase to 10-32pg/mL between days 3-60, with a subsequent decline to pre-pubertal levels of &lt;15pg/mL during the first year</td>
</tr>
<tr>
<td>Females &lt; 1 year</td>
<td>Concentrations increase to 5.0 – 50pg/mL between days 30-60, then decline to pre-pubertal values of &lt; 15pg/mL during the first year.</td>
</tr>
</tbody>
</table>

<table>
<thead>
<tr>
<th></th>
<th>Males</th>
<th>Females</th>
</tr>
</thead>
<tbody>
<tr>
<td>Adult Values</td>
<td>8.0</td>
<td>30 – 100pg/mL</td>
</tr>
<tr>
<td></td>
<td></td>
<td>Luteal</td>
</tr>
<tr>
<td></td>
<td></td>
<td>70 – 30pg/mL</td>
</tr>
<tr>
<td></td>
<td></td>
<td>Postmenopausal</td>
</tr>
<tr>
<td></td>
<td></td>
<td>&lt;15pg/mL</td>
</tr>
</tbody>
</table>

*Adapted from Elmlinger, Kuhnel & Ranke (2002).*
### Appendix H

#### Table H.1: Free Testosterone Reference Values

<table>
<thead>
<tr>
<th>Age Group</th>
<th>Male Reference Values</th>
<th>Female Reference Values</th>
</tr>
</thead>
<tbody>
<tr>
<td>20-25 years</td>
<td>5.25 - 2.07 ng/dl</td>
<td>0.66 - 1.09 ng/dl</td>
</tr>
<tr>
<td>25-&lt; 30 years</td>
<td>5.05 - 19.80 ng/dl</td>
<td>0.06 - 1.06 ng/dl</td>
</tr>
<tr>
<td>30-&lt; 35 years</td>
<td>4.85 - 19.00 ng/dl</td>
<td>0.06 - 1.03 ng/dl</td>
</tr>
<tr>
<td>35-&lt; 40 years</td>
<td>4.65 - 18.10 ng/dl</td>
<td>0.06 - 0.98 ng/dl</td>
</tr>
<tr>
<td>40-&lt; 45 years</td>
<td>4.46 - 17.10 ng/dl</td>
<td>0.06 - 0.92 ng/dl</td>
</tr>
<tr>
<td>45-&lt; 50 years</td>
<td>4.26 - 16.40 ng/dl</td>
<td>0.06 - 0.90 ng/dl</td>
</tr>
<tr>
<td>50-&lt;60 years</td>
<td>4.06 - 15.60 ng/dl</td>
<td>0.06 - 0.87 ng/dl</td>
</tr>
<tr>
<td>55-&lt;60 years</td>
<td>3.87 - 14.70 ng/dl</td>
<td>0.06 - 0.84 ng/dl</td>
</tr>
<tr>
<td>60-&lt; 65 years</td>
<td>3.67 - 13.90 ng/dl</td>
<td>0.06 - 0.82 ng/dl</td>
</tr>
<tr>
<td>65-&lt; 70 years</td>
<td>3.47 - 13.00 ng/dl</td>
<td>0.06 - 0.79 ng/dl</td>
</tr>
<tr>
<td>70-&lt; 75 years</td>
<td>3.28 - 12.20 ng/dl</td>
<td>0.06 - 0.76 ng/dl</td>
</tr>
<tr>
<td>75-&lt; 80 years</td>
<td>3.08 - 11.30 ng/dl</td>
<td>0.06 - 0.73 ng/dl</td>
</tr>
<tr>
<td>80-&lt; 85 years</td>
<td>2.88 - 10.50 ng/dl</td>
<td>0.06 - 0.71 ng/dl</td>
</tr>
<tr>
<td>85-&lt; 90 years</td>
<td>2.69 - 9.61 ng/dl</td>
<td>0.06 - 0.68 ng/dl</td>
</tr>
<tr>
<td>90-&lt; 95 years</td>
<td>2.49 - 8.76 ng/dl</td>
<td>0.06 - 0.65 ng/dl</td>
</tr>
<tr>
<td>95-100+ years</td>
<td>2.29 - 7.91 ng/dl</td>
<td>0.06 - 0.62 ng/dl</td>
</tr>
</tbody>
</table>

#### Table H.2: Total Testosterone Reference Values

<table>
<thead>
<tr>
<th>Age Group</th>
<th>Male Reference Values</th>
<th>Female Reference Values</th>
</tr>
</thead>
<tbody>
<tr>
<td>0-5 months</td>
<td>75 - 400 ng/dl</td>
<td>20 - 80 ng/dl</td>
</tr>
<tr>
<td>6 months - 9 years</td>
<td>&lt;7 - 20 ng/dl</td>
<td>&lt;7 - 44 ng/dl</td>
</tr>
<tr>
<td>10-11 years</td>
<td>&lt;7 - 130 ng/dl</td>
<td>&lt;7 - 75 ng/dl</td>
</tr>
<tr>
<td>12-13 years</td>
<td>&lt;7 - 800 ng/dl</td>
<td>20 - 75 ng/dl</td>
</tr>
<tr>
<td>14 years</td>
<td>&lt;7 - 1200 ng/dl</td>
<td>8 - 60 ng/dl</td>
</tr>
<tr>
<td>15-16 years</td>
<td>100 - 1200 ng/dl</td>
<td>8 - 60 ng/dl</td>
</tr>
<tr>
<td>17-18 years</td>
<td>300 - 1200 ng/dl</td>
<td>8 - 60 ng/dl</td>
</tr>
<tr>
<td>≥ 19 years</td>
<td>240 - 950 ng/dl</td>
<td>8 - 60 ng/dl</td>
</tr>
</tbody>
</table>
Appendix I

Name: ________________________________________________

Current School Year: ________________    Major:________________________

____________________________________

Date of Birth (month and year only):

_______________________________________________

Email: _________________________________________

(For project use only – ID # _____________________________)

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1. Demographic Information

Date of Birth (MM/YY) ___/___

Today’s Date (DD/MM/YY) ___/___/____

Age: _____

Race/Ethnic Background (please circle)
- Aboriginal
- Asian or Asian Descent
- Hispanic/Latino
- Non-Hispanic Black or African Descent
- Non-Hispanic White or Caucasian
- Other/Mixed (please describe) _________________
- Prefer not to answer

Are you currently pregnant? (please circle) NO

YES

Do you have any children? (please circle) NO

YES

Are you left handed or right handed? (please circle) LEFT

RIGHT

2. House Hold/Family Information

Parent Information:

<table>
<thead>
<tr>
<th>Parent</th>
<th>Highest Level of Education Completed</th>
</tr>
</thead>
<tbody>
<tr>
<td></td>
<td></td>
</tr>
<tr>
<td></td>
<td></td>
</tr>
<tr>
<td></td>
<td></td>
</tr>
</tbody>
</table>
Sibling Information:

*Please list the gender, age and order of your BIOLOGICAL siblings only (if any) including yourself in the list. For example, if you have one older and one younger brother you would put …. Brother (24 years old), Me (22 years old), Brother (19 years old), on the lines below.*

________________________________________________
________________________________________________
________________________________________________
________________________________________________
________________________________________________

Are you a twin or part of a multiple birth? (please check one box)  
YES  ☐ NO

If so, please indicate whether you are fraternal or paternal, further as the gender of your siblings.

________________________________________________
________________________________________________
________________________________________________
________________________________________________

3. Pubertal Information

Age of Menarche
(As best as you can remember, indicate how old you were when you first started menstruating on the line below)
Date of last menarche
(As best as you can remember, please indicate the last day of menstruation (i.e. bleeding) of your most previous period. If you aren’t sure of the last day, please guess the approximate day)

YES NO

Are you currently on birth control? YES NO
(please circle)

If yes, please indicate what brand:

Do you have any pre-existing conditions which may alter your testosterone concentrations?
(please circle) YES NO

Are you currently on any supplements, medications (prescribed or not) that could potentially alter your testosterone concentrations?

YES NO

If yes, please indicate the specific medication ______________________________.

If yes to the above question, please indicate the number of years you have been taking the above supplement, medication or drug since the age of 9.
Appendix J

Sport Orientation Questionnaire

The following statements describe reactions to sport situations. We want to know how you usually feel about sports and competition. Read each statement and circle the letter that indicates how much you agree or disagree with each statement on the scale: A, B, C, D, or E. There are no right or wrong answers; simply answer as you honestly feel. Do not spend too much time on any one statement. Remember, choose the letter that describes how you usually feel about sports and competition.

<table>
<thead>
<tr>
<th></th>
<th></th>
<th>Strongly agree</th>
<th>Slightly agree</th>
<th>Neither agree nor disagree</th>
<th>Slightly disagree</th>
<th>Strongly disagree</th>
</tr>
</thead>
<tbody>
<tr>
<td>1.</td>
<td>I am a determined competitor.</td>
<td>A</td>
<td>B</td>
<td>C</td>
<td>D</td>
<td>E</td>
</tr>
<tr>
<td>2.</td>
<td>Winning is important.</td>
<td>A</td>
<td>B</td>
<td>C</td>
<td>D</td>
<td>E</td>
</tr>
<tr>
<td>3.</td>
<td>I am a competitive person.</td>
<td>A</td>
<td>B</td>
<td>C</td>
<td>D</td>
<td>E</td>
</tr>
<tr>
<td>4.</td>
<td>I set goals for myself when I compete.</td>
<td>A</td>
<td>B</td>
<td>C</td>
<td>D</td>
<td>E</td>
</tr>
<tr>
<td>5.</td>
<td>I try my hardest to win.</td>
<td>A</td>
<td>B</td>
<td>C</td>
<td>D</td>
<td>E</td>
</tr>
<tr>
<td>6.</td>
<td>Scoring more points than my opponent is very important to me.</td>
<td>A</td>
<td>B</td>
<td>C</td>
<td>D</td>
<td>E</td>
</tr>
<tr>
<td>7.</td>
<td>I look forward to competing.</td>
<td>A</td>
<td>B</td>
<td>C</td>
<td>D</td>
<td>E</td>
</tr>
<tr>
<td>8.</td>
<td>I am most competitive when I try to achieve personal goals.</td>
<td>A</td>
<td>B</td>
<td>C</td>
<td>D</td>
<td>E</td>
</tr>
<tr>
<td>9.</td>
<td>I enjoy competing against others.</td>
<td>A</td>
<td>B</td>
<td>C</td>
<td>D</td>
<td>E</td>
</tr>
<tr>
<td>10.</td>
<td>I hate to lose.</td>
<td>A</td>
<td>B</td>
<td>C</td>
<td>D</td>
<td>E</td>
</tr>
<tr>
<td>11.</td>
<td>I thrive on competition.</td>
<td>A</td>
<td>B</td>
<td>C</td>
<td>D</td>
<td>E</td>
</tr>
<tr>
<td>12.</td>
<td>I try hardest when I have a specific goal.</td>
<td>A</td>
<td>B</td>
<td>C</td>
<td>D</td>
<td>E</td>
</tr>
<tr>
<td>13.</td>
<td>My goal is to be the best athlete possible.</td>
<td>A</td>
<td>B</td>
<td>C</td>
<td>D</td>
<td>E</td>
</tr>
<tr>
<td></td>
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<td></td>
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<tr>
<td>---</td>
<td>----------------------------------------------------------------</td>
<td>---</td>
<td>---</td>
<td>---</td>
<td>---</td>
<td></td>
</tr>
<tr>
<td>14.</td>
<td>The only time I am satisfied is when I win.</td>
<td>A</td>
<td>B</td>
<td>C</td>
<td>D</td>
<td>E</td>
</tr>
<tr>
<td>15.</td>
<td>I want to be successful in sports.</td>
<td>A</td>
<td>B</td>
<td>C</td>
<td>D</td>
<td>E</td>
</tr>
<tr>
<td>16.</td>
<td>Performing to the best of my ability is very important to me.</td>
<td>A</td>
<td>B</td>
<td>C</td>
<td>D</td>
<td>E</td>
</tr>
<tr>
<td>17.</td>
<td>I work hard to be successful in sports.</td>
<td>A</td>
<td>B</td>
<td>C</td>
<td>D</td>
<td>E</td>
</tr>
<tr>
<td>18.</td>
<td>Losing upsets me.</td>
<td>A</td>
<td>B</td>
<td>C</td>
<td>D</td>
<td>E</td>
</tr>
<tr>
<td>19.</td>
<td>The best test of my ability is competing against others.</td>
<td>A</td>
<td>B</td>
<td>C</td>
<td>D</td>
<td>E</td>
</tr>
<tr>
<td>20.</td>
<td>Reaching personal performance goals is very important to me.</td>
<td>A</td>
<td>B</td>
<td>C</td>
<td>D</td>
<td>E</td>
</tr>
<tr>
<td>21.</td>
<td>I look forward to the opportunity to test my skills in competition.</td>
<td>A</td>
<td>B</td>
<td>C</td>
<td>D</td>
<td>E</td>
</tr>
<tr>
<td>22.</td>
<td>I have the most fun when I win.</td>
<td>A</td>
<td>B</td>
<td>C</td>
<td>D</td>
<td>E</td>
</tr>
<tr>
<td>23.</td>
<td>I perform my best when I am competing against an opponent.</td>
<td>A</td>
<td>B</td>
<td>C</td>
<td>D</td>
<td>E</td>
</tr>
<tr>
<td>24.</td>
<td>The best way to determine my ability is to set a goal and try to reach it.</td>
<td>A</td>
<td>B</td>
<td>C</td>
<td>D</td>
<td>E</td>
</tr>
<tr>
<td>25.</td>
<td>I want to be the best every time I compete.</td>
<td>A</td>
<td>B</td>
<td>C</td>
<td>D</td>
<td>E</td>
</tr>
</tbody>
</table>
Appendix K

Today's Date: November 03, 2016
Principal Investigator: Ms. Elizabeth Vandenborn
REB Number: 33486
Research Project Title: REB# 16-195: "Why do girls play? Can circulating and prenatal testosterone predict sport participation in adolescent females?"
Clearance Date: November 3, 2016
Project End Date: August 31, 2017
Milestones:
Renewal Due-2017/08/31(Pending)

This is to inform you that the University of Windsor Research Ethics Board (REB), which is organized and operated according to the Tri-Council Policy Statement and the University of Windsor Guidelines for Research Involving Human Subjects, has granted approval to your research project on the date noted above. This approval is valid only until the Project End Date.

A Progress Report or Final Report is due by the date noted above. The REB may ask for monitoring information at some time during the project’s approval period.

During the course of the research, no deviations from, or changes to, the protocol or consent form may be initiated without prior written approval from the REB. Minor change(s) in ongoing studies will be considered when submitted on the Request to Revise form.

Investigators must also report promptly to the REB:
a) changes increasing the risk to the participant(s) and/or affecting significantly the conduct of the study;
b) all adverse and unexpected experiences or events that are both serious and unexpected;
c) new information that may adversely affect the safety of the subjects or the conduct of the study.

Forms for submissions, notifications, or changes are available on the REB website: www.uwindsor.ca/reb. If your data is going to be used for another project, it is necessary to submit another application to the REB.

We wish you every success in your research.

Dr. Suzanne McMurphy, Ph.D.
Chair, Research Ethics Board
2146 Chrysler Hall North
University of Windsor
519-253-3000 ext. 3948
Email: ethics@uwindsor.ca

The information contained in this e-mail message is confidential and protected by law. The information is intended only for the person or organization addressed in this e-mail. If you share or copy the information you may be breaking the law. If you have received this e-mail by mistake, please notify the sender of the e-mail by the telephone number listed on this e-mail. Please destroy the original; do not e-mail back the information or keep the original.
### Appendix L

Table 15: Correlation between 4 components of Composite Sport Participation Score

<table>
<thead>
<tr>
<th></th>
<th>Number of Sports</th>
<th>Highest Level</th>
<th>Weekly Frequency</th>
<th>Months of Sport Season</th>
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<tr>
<td><strong>Pearson Correlation</strong></td>
<td>1</td>
<td>.296**</td>
<td>.484**</td>
<td>.619**</td>
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<tr>
<td><strong>Sig. (2-tailed)</strong></td>
<td>0.000</td>
<td>0.000</td>
<td>0.000</td>
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<tr>
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<td>1012</td>
<td>1012</td>
<td>1012</td>
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</tr>
<tr>
<td><strong>Pearson Correlation</strong></td>
<td>.296**</td>
<td>1</td>
<td>.541**</td>
<td>.481**</td>
</tr>
<tr>
<td><strong>Sig. (2-tailed)</strong></td>
<td>0.000</td>
<td>0.000</td>
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</tr>
<tr>
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<td>1012</td>
<td>1012</td>
<td>1012</td>
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</tr>
<tr>
<td><strong>Pearson Correlation</strong></td>
<td>.484**</td>
<td>.541**</td>
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<td>.631**</td>
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<tr>
<td><strong>Sig. (2-tailed)</strong></td>
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<td>0.000</td>
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</tr>
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<td><strong>Pearson Correlation</strong></td>
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<td>.481**</td>
<td>.631**</td>
<td>1</td>
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<tr>
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<td>0.000</td>
<td>0.000</td>
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</tr>
<tr>
<td><strong>N</strong></td>
<td>1012</td>
<td>1012</td>
<td>1012</td>
<td>1012</td>
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</tbody>
</table>

*Note:* **. Correlation is significant at the 0.01 level (2-tailed).
Appendix M

Q-Q Plots for Regression Model 1

TSP\textsubscript{9to18}  

Normal P-P Plot of Regression Standardized Residual  
Dependent Variable: NN.SportPart\textsubscript{total}  

TSP\textsubscript{early}  

Normal P-P Plot of Regression Standardized Residual  
Dependent Variable: NN.SportPart\textsubscript{Pre14}  

TSP\textsubscript{late}  

Normal P-P Plot of Regression Standardized Residual  
Dependent Variable: NN.SportPart\textsubscript{Post15}
Q-Q Plots for Regression Model 2

TSP_{9to18}

Normal P-P Plot of Regression Standardized Residual
Dependent Variable: NN.Sport.Part.total

TSP_{early}

Normal P-P Plot of Regression Standardized Residual
Dependent Variable: NN.SportPart.Pre14

TSP_{late}

Normal P-P Plot of Regression Standardized Residual
Dependent Variable: NN.Sport.Part.Post15
Appendix N

Regression Model 1 Standardized Predicted Value vs. Standardized Residual

TSP\textsubscript{9to18}

![Scatterplot of TSP\textsubscript{9to18}](image)

TSP\textsubscript{early}

![Scatterplot of TSP\textsubscript{early}](image)
Regression Model 2 Standardized Predicted Value vs. Standardized Residual

TSP\textsubscript{late}

TSP\textsubscript{9to18}
$TSP_{\text{early}}$

Scatterplot
Dependent Variable: NN.SportPart.Pre14

![Scatterplot](image)

$TSP_{\text{late}}$

Scatterplot
Dependent Variable: NN.SportPart.Post15

![Scatterplot](image)
Vita Auctoris

<table>
<thead>
<tr>
<th>NAME</th>
<th>Elizabeth Vandenborn</th>
</tr>
</thead>
<tbody>
<tr>
<td>PLACE OF BIRTH</td>
<td>Chatham, ON</td>
</tr>
<tr>
<td>YEAR OF BIRTH</td>
<td>1993</td>
</tr>
<tr>
<td>EDUCATION</td>
<td>Chatham-Kent Secondary School, Chatham, ON, 2011</td>
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<tr>
<td></td>
<td>University of Windsor, BHK, Windsor, ON, 2015</td>
</tr>
<tr>
<td></td>
<td>University of Windsor, MHK., Windsor, ON, 2017</td>
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