COMPARISON OF ANABOLIC HORMONE RESPONSES TO AEROBIC AND RESISTANCE EXERCISE IN PHYSICALLY ACTIVE PREMENOPAUSAL FEMALES

by

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B.Sc. University of Waterloo, 1998

A Thesis Submitted in Partial Fulfilment of the Requirements for the Degree of Master of Science in Exercise and Sport Science in the Graduate Academic Unit of Kinesiology

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THE UNIVERSITY OF NEW BRUNSWICK

September, 2000

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ABSTRACT

Previous research has indicated the association between physical activity and improved health benefits. However, recent statistics estimate as many as 67% of the Canadian population are not active enough to receive these benefits. With the advancing age of the baby boom generation increasing strain is being placed on the health care system. Knowledge that certain steroid hormones decrease with age and are linked to healthy living has led to the proliferation in the sale of hormone supplements. Unfortunately, these supplements are not always safe and can become quite costly. It would, therefore, be beneficial to find a way to increase these hormones naturally. Exercise is believed to be a possible answer. Currently, little data is available comparing female hormonal responses between the two main types of exercise: aerobic and resistance exercise.

Sixteen females between the ages of 19 and 47 were recruited for the study. Each participant completed the Canadian Physical Activity, Fitness and Lifestyle Appraisal, and a familiarization session to become orientated with each of the exercise protocols. Three testing sessions took place during the luteal phase of each subject’s menstrual cycle. These randomized sessions included a resistance session, an aerobic session, and a control session. The resistance exercise session consisted of three sets of 10 repetitions of eight exercises using Universal equipment. The intensity equaled the previously determined 10 RM. The endurance session consisted of 40 minutes of pedaling on a Monarch cycle ergometer at 75% of the subject’s maximum heart rate.
The hormones under investigation included testosterone, estradiol, growth hormone, dehydroepiandrosterone (DHEA), and insulin-like growth factor I (IGF-I). Samples were taken 10 minutes before the beginning of each testing session, immediately following each session, and 30 minutes after the session had been completed. Lactate levels, heart rate and the Borg Rating of Perceived Exertion (RPE) Scale were used to monitor each subject’s intensity level. Lactate was measured before each testing session and again immediately following each session.

After all blood samples were corrected for plasma volume change, an analysis of variance (ANOVA) was used to analyze the data. Post-hoc comparisons used Tukey analyses when necessary. Area under the curve for each hormone was also analyzed for each session. Statistical significance was set at P < 0.05.

Lactate and RPE values were significantly higher (P < 0.001) in the resistance session than the aerobic and control sessions, however, average heart rate was highest during the aerobic session. No significant differences in hormonal response were observed between the aerobic and resistance session. Growth hormone increased in both the resistance and aerobic sessions over the control session (P < 0.001 and P < 0.02 respectively). Estradiol and testosterone increased significantly in only the aerobic session over the control session (P < 0.003 and P < 0.04 respectively). No exercise related changes were observed for DHEA or IGF-I. Despite differences in the intensity variables, hormone responses were similar between the aerobic and resistance sessions.
ACKNOWLEDGMENTS

The author would like to acknowledge the following individuals who assisted in this thesis:

- My supervisor, Dr. Mark Tremblay for his support and guidance.
- Jennifer Copeland for her invaluable assistance and knowledge.
- All the subjects who volunteered their time, effort, and blood for this study.
- Dr. Jim Sexsmith for his helpful review of this thesis.
- My family for their continued support, encouragement and long distance phone calls.
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CHAPTER 1
INTRODUCTION

There is strong research evidence documenting that regular physical activity is associated with a lower risk of premature development of many health problems including atherosclerosis, colon cancer, osteoporosis, obesity, depression, diabetes, and coronary heart disease (U.S. Department of Health and Human Services, 1996). Physical activity is also associated with prolonged quality of life (Shephard, 1996) and independent living in the elderly population (Chodzko-Zajko, 1996). The most widely appreciated long-term benefits of regular exercise include an improvement in cardiovascular function, improved muscular strength and endurance, enhanced flexibility, and a reduction in body fat (Howley and Franks, 1997). Other benefits include enhanced performance of metabolic, endocrine, and immune systems (U.S. Department of Health and Human Services, 1996).

Two main components of physical fitness are cardiovascular endurance and muscular strength. Workouts that help improve these two fitness components include aerobic and resistance training, respectively.

Aerobic exercise is known to maintain and improve various aspects of the cardiovascular system such as increased stroke volume and decreased resting heart rate (Franklin, 1998). Aerobic training has been linked to reductions in risk factors associated with diseases such as heart disease and diabetes (Franklin, 1998). It also improves health status and increases life expectancy (Franklin, 1998). In addition, aerobic training has
been associated with physiological adaptations including increased respiratory capacity, improved aerobic enzyme activity, and increased mitochondrial and capillary densities (Kraemer, 1994).

Resistance exercise can also confer substantial health benefits. Eighty percent of low-back problems are muscular in nature (Westcott, 1991). Since four out of five Americans experience low-back pain (Westcott, 1991), strengthening exercises for the lower back and abdominal regions are important. Resistance exercises have also been shown to reduce the risk of joint and muscle injuries that may occur during other types of physical activity (Stone, 1990). The loss of muscle strength and bone density that is associated with aging can be delayed with resistive exercise (Frontera et al., 1991). Osteoarthritis, the most common form of arthritis, is associated with the gradual loss of articular cartilage around a joint (Howley and Franks, 1997). Resistance exercise is believed to improve movement and reduce arthritis pain by improving the strength and function of the surrounding connective tissue, which is also damaged by the disease (Pothier and Allen, 1991).

Without regular resistance exercise, up to one kilogram of muscle may be lost every five years after the age of 25 (Westcott, 1991). One of the primary results of resistance exercise is the increase of muscle mass. Since skeletal muscle is more metabolically active than adipose tissue, resistance exercise is an important factor in the prevention and treatment of obesity.

Although exercise may not be the "fountain of youth," regular physical activity can retard the decline of functional capacity that is associated with aging (McArdle et al., 1994; U.S. Department of Health and Human Services, 1996). Paffenbarger et al. (1986)
determined that mortality rates were 21% lower for men who walked nine or more miles per week compared to their male counterparts who walked three or less miles per week. From this same study it was determined that men who exercised in light sport activities increased their life expectancies 24% over their sedentary counterparts. Similar exercise induced health benefits in females have been documented elsewhere (U.S. Department of Health and Human Services, 1996).

As the population ages, increased pressures are being felt on our health care system. It is estimated that chronic disease conditions cost as much as one trillion dollars annually in health care expenses and lost productivity in the United States (Booth et al., 2000). Included in this cost is approximately $300 billion and $6 billion that is spent on sarcopenia related disabilities (Carlson et al., 1999) and osteoporosis (Wolff et al., 1999) respectively. Booth et al. (2000) suggest that current research efforts are incomplete since the majority of work focuses on secondary and tertiary prevention of disease. In contrast to the conventional methods of treating the ill, health care providers today must think in terms of primary prevention. The idea is to prevent people from becoming sick and consequently dependent on others.

Recently, prevention strategies for osteoporosis have targeted young women with the notion of increasing peak bone mass and reducing age-related bone loss in the premenopausal years (Snow, 1996). It is important to remember that the lower levels of bone density observed in the elderly are not due to sudden losses. Instead, it is the result of the gradual loss of bone density throughout their lifetime. Bone density begins to decline before menopause and is correlated with decreases in estrogen production also seen at this time (Johnston et al., 1985; Steinberg et al., 1989). Evidence shows that low
levels of reproductive hormones in premenopausal women may influence their current bone mass (Sowers et al., 1998) and be predictive of bone mass levels as they age (Newton-John and Morgan, 1968).

Levels of gonadal steroids are related with peak bone mass levels in premenopausal women and measurement of these hormone levels are used to help identify women who are at increased risk of developing osteoporosis in the future (Sowers et al., 1998). Research is needed to determine methods that could raise these low hormone levels and decelerate their decline that starts in the premenopausal years. By taking proactive approaches women will be at a decreased risk of developing osteoporosis as they age.

As the baby-boomers increase in age, attempts to preserve their youth have become omnipresent. Each year people spend millions of dollars on anti-aging products. Recently, one of the most marketed products has been dehydroepiandrosterone (DHEA). In 1996, the Food and Drug Administration (FDA) banned the sale and distribution of DHEA for therapeutic use until further scientific knowledge on its safety could be established (Armsey and Green, 1997). The media attention that followed this decision popularized this supplement so much that manufacturers began selling DHEA as a nutritional aid rather than a therapeutic drug (Armsey and Green, 1997). As a precursor to androgenic steroids, DHEA may increase the production of testosterone, which has well known anabolic effects.

Endogenous DHEA declines with age (Orentreich et al., 1984). This decline in DHEA is associated with an increased frequency of disability and disease. Studies indicate that low levels of DHEA are associated with increased risk of cardiovascular
disease in men over 50 years of age (Barrett-Connor et al., 1986) and breast cancer in premenopausal women (Helzsouer et al., 1992). Experiments using animals indicate that DHEA supplementation reduces the severity of a variety of diseases (Coleman et al., 1982; Schwartz, 1979). Unfortunately, because none of the animals used in these studies synthesize their own DHEA, as humans do, this work must be interpreted with caution.

In contrast to the public attention DHEA has received, the scientific community has paid little attention. In one of the few studies conducted involving DHEA supplementation, Morales et al. (1994) examined men and women between the ages of 40 and 70 years who received 50 mg of DHEA supplementation for three months. Within two weeks of supplementation serum concentrations of DHEA had risen between threefold and fivefold. In the women taking the DHEA, serum concentrations of androstenediol, testosterone, and dihydrotestosterone rose, however only androstenediol rose in the men. Serum concentrations of insulin-like growth factor 1 (IGF-1) rose approximately 10%. Psychological benefits were also observed. Eighty two percent of women and 67% of men on the DHEA supplement reported an improved sense of “well-being” (improved quality of sleep, greater energy, and increased ability to handle stress). In comparison, less then 10% of those taking the placebo reported similar results.

Hormones play a role in many diseases and disorders such as diabetes, hypothyroidism, obesity, and infertility. Recently, research has been directed at the problems associated with decreased hormone levels in women. In younger women, issues such as amenorrhea have been investigated. Although, the exact cause of this disorder is still under question, many believe an endocrine disturbance may play a critical role. In older women, negative postmenopausal side effects are quite common. These
problems have been linked to the decline in reproductive hormones that occur with increasing age. This in turn has led to an increase in the prescription of hormone replacement therapies. Unfortunately, these supplements and hormone replacement therapies are not always safe. Potentially harmful side effects and the excessive cost of these products make them undesirable.

Researchers today are trying to find ways to naturally increase these desirable hormones in the body. Increasing anabolic hormone levels naturally in young women could be beneficial in delaying and/or decelerating the age-related decline of these hormones. This could prevent the use of potentially dangerous and expensive synthetic hormone therapies. In addition, maintaining or even raising steroid hormone levels naturally to optimal levels in young women will decrease their risk of obtaining diseases that are related to declining hormone levels. One proposed method of augmenting these hormones is through exercise. This would be beneficial because not only would these hormone levels increase naturally but individuals would also receive other benefits that have been traditionally related to exercise such as improved cardiovascular and muscular fitness.

Clearly physical activity has a role to play in containing health care costs. A recent report from the Centers for Disease Control and Prevention (1998a) confirmed this by stating "physical inactivity is one of the underlying causes of premature mortality in the United States". Unfortunately, it appears the public has overlooked the benefits of regular physical activity. Research from the Canadian Fitness and Lifestyle Research Institute (CFLRI) demonstrated that 67% of Canadians are not sufficiently active to derive substantial health benefits (Craig et al., 1998). Evidently most Canadians do not
engage in regular exercise. Consequently, it is important, especially with an aging population, to determine what kind of exercise yields the most desirable health benefits.

To date, there have been no studies directly comparing the anabolic hormone response to aerobic versus resistance exercise in adult females.

**Purpose**

Numerous health benefits have been associated with physical activity. However, the majority of the population is not active enough to obtain these benefits. Clearly both aerobic and resistance exercises are important to an individual's state of health, however, it is important to identify the specific benefits related to each type of exercise. This might allow for more accurate exercise prescriptions to be offered while also attracting an increase in participation. Therefore, the purpose of this research was to compare the anabolic hormone response to resistance versus aerobic exercise in healthy premenopausal females. Due to the paucity of research identifying the anabolic hormone response to exercise in females, a modest age range was selected for this study. It is imperative that initial efforts be made to recognize generalizations within this group. It has been well documented that low anabolic hormone levels in premenopausal women have been associated with increased risk of disease. Research involving premenopausal women is crucial for the implementation of proactive approaches in preventive medicine. In addition, if healthy physical activity habits can be created while females are still young, it is more probable these healthy habits will continue as they age. The lack of previous research in this area, specifically the response of steroid hormones to exercise in females, has left a clear gap in academic and medical fields.
Hypotheses

1. Testosterone, DHEA, growth hormone, IGF-I, and estradiol would be significantly elevated immediately after the aerobic exercise session.

2. DHEA, estradiol, growth hormone, and IGF-I, would be significantly elevated immediately after the resistance exercise session.

3. There would not be a significant difference in anabolic hormone responses between the aerobic and resistance exercise sessions.

Limitations and Delimitations

Limitations

1. It was assumed that subjects followed pre-testing guidelines.

2. It was assumed that all subjects entered in the study completed their health and medical screening form accurately.

3. It was assumed that all subjects entered in the study had been actively cross-training for at least four months.

4. It was assumed that the intensity determined for the resistance session was the subject’s true 10 RM for each exercise.

5. It was assumed that each subject’s true maximum heart rate was determined during the familiarization session. Therefore, it was assumed that each subject worked at the intensity of 75% of her maximum heart rate during the aerobic session.
Delimitations

1. Generalizations are limited due to the small sample size \( (n = 16) \) of participants in the study from the same area.

2. The hormones evaluated in this study were limited to those that were believed to be the most significant anabolic hormones in female exercise endocrinology.

3. Venous blood sampling was the only source of hormone measurements.
CHAPTER 2
LITERATURE REVIEW

Background on Hormones
There are two major control systems that help maintain homeostasis in the human body: the nervous system and the endocrine system. The endocrine system consists of endocrine glands, which secrete chemical messengers called hormones. Hormones play a vital role in regulating growth and development, metabolism, and reproduction (Bunt, 1986). They are also essential for helping the body deal with both physical and psychological stress (McArdle et al., 1994). Hormonal actions in the target cell can include alterations in the cell membrane permeability, changes in enzyme activity, or alterations in the rate of enzyme-protein synthesis (Bunt, 1986). Hormones are generally classified into three categories based on their chemical structure and mechanisms of action: (a) steroids, (b) peptides and glycoproteins, and (c) amines and thyroid hormones (Vander et al., 1994).

Health Effects of Hormones
Hormones play an important role in many disease processes. Disorders such as hypothyroidism and diabetes can be directly related to disruptions of the endocrine system. By the early 1970s, it had become evident that many female athletes were experiencing training-associated disruptions in their menstrual cycle (Bunt, 1986). Complaints of oligomenorrhea or amenorrhea in athletes, in particular distance runners.
gymnasts, and ballet dancers, led researchers to believe that low bodyweights and fat content led to these disturbances. The findings of Loucks and Horvath (1985) that amenorrheic athletes had low estradiol levels led investigators to speculate that hormonal disruptions, particularly to the anterior pituitary and the ovary glands, may play a critical role. No differences in the levels of DHEA, dehydroepiandrosterone sulfate (DHEAS), androstenedione, testosterone, cortisol, follicle stimulating hormone (FSH), or prolactin between amenorrheic runners, eumenorrheic runners, and eumenorrhic nonrunners have been found (Baker et al., 1981). However, Schwartz et al. (1981) observed that the ratio of estrone to estradiol was significantly higher in runners than nonrunners.

Hormones have also been implicated in the development of osteoporosis. During the first two decades of life, skeletal mass increases with adequate sources of calcium and osteoblast domination (Watson et al., 1996). However, beginning in the third decade, osteoclast activity begins to dominate, leading to a decrease in skeletal mass (Watson et al., 1996). The human skeleton stores 99% of the body's calcium, therefore, sufficient sources of calcium are also important in the maintenance of skeletal integrity (McArdle et al., 1994). When dietary intake of calcium is not sufficient, the body is forced to draw upon this storage site as a form of compensation. Osteoporosis (literally meaning "porous bones") occurs if this process of osteoclast domination and calcium deficiency is prolonged as the bones begin to lose their mineral mass and become brittle (McArdle et al., 1994). It is estimated that approximately 25% of women over the age of 65 experience spontaneous bone fractures related to osteoporosis (McArdle et al., 1994).

Osteoporosis is often associated with chronically low levels of estradiol, an important hormone for the maintenance of bone mineral content (Bunt, 1986). Estradiol
acts indirectly on bone by suppressing parathyroid hormone mediated bone resorption (Steinberg et al., 1989). It also acts indirectly on calcium absorption by the intestines and renal tubular reabsorption of calcium by stimulating the synthesis of 1,25-dihydroxyvitamin D in the kidney (Steinberg et al., 1989). It is believed that lower levels of estradiol reduce the ability for calcium to be absorbed by these mechanisms and as a result, calcium is drawn from bones. This action causes a decrease in bone density and consequently an increase in the occurrence of fractures. With the finding of estradiol receptors in human osteoblast-like cells, Eriksen et al. (1988) have speculated that estradiol may have direct actions on bone. Further research is needed in this area.

While much research demonstrates that estradiol plays a primary role in bone density in females, discrepancies exist concerning the importance of other steroid hormones. Steinberg et al. (1989) observed no correlation between free estradiol and bone density in premenopausal women. They did, however, report a positive relationship between free testosterone and bone density. Supporting the importance of testosterone in bone remodeling, Francis (1999) reported that testosterone replacement therapy could improve bone density in men suffering from hypogonadal osteoporosis. However, it was not evident if the increase in bone density was the direct action of testosterone or mediated by the aromatization of testosterone to estradiol. In addition, Chesnut et al. (1983) showed that women with postmenopausal osteoporosis respond to stanozolol with increased bone formation. Previous research by Komm et al. (1988) suggests that testosterone may compete with estradiol for receptor sites and therefore, may have a direct effect at the level of the osteoblast. This finding reinforces the notion of Steinberg et al. (1989) that testosterone may play a key role in bone density in premenopausal
women. Steinberg et al. (1989) also found a negative association between sex hormone-binding globulin (SHBG) and bone density in premenopausal women. Based on knowledge that low SHBG is associated with increased availability of free estradiol and free testosterone, SHBG may influence bone density by regulating the availability of these hormones (Steinberg et al., 1989).

It is believed that sex steroids play an integral role in maintaining skeletal integrity (Steinberg et al., 1989). Evidence exists that women with osteoporosis have lower free and total levels of sex steroids than their control counterparts (Aloia et al., 1985). A crucial finding has been that bone density begins to decline before menopause (Johnston et al., 1985; Steinberg et al., 1989). This belief has been supported by Prior et al. (1990) who observed a mean one-year bone loss of 2% in premenopausal women. In addition to traditional reactive methods of treating older women already suffering from osteoporosis, proactive methods involving young women need to be investigated.

The association between low steroid levels and bone mineral density (BMD) has also been observed in younger women. Research by Sowers et al. (1990) demonstrated that low luteal phase estrogen and testosterone levels were associated with lower premenopausal bone mass measured at the femoral neck. In a follow-up study, Sowers et al. (1998) reported that premenopausal women with BMD at the lowest 10\textsuperscript{th} percentile had significantly lower urinary excretion levels of both estrogen and progesterone metabolites during the luteal phase compared with hormone levels in women with BMD between the 50\textsuperscript{th} and 75\textsuperscript{th} percentile. These studies show a definite link between BMD and steroid hormones in young women. Ideally, if these hormone levels could be raised
and/or have their age-related decline decelerated, BMD would follow a similar pattern, and subsequently lower their risk of developing osteoporosis.

An additional area of research has studied the relationship between exercise, sex steroids and the incidence of coronary heart disease (CHD). Due to the beneficial effects of estrogen, premenopausal women with normal estrogen levels appear to be protected against CHD, and are at a lower risk for myocardial infarction compared to males or postmenopausal women (Hanke et al., 1997). A number of findings have supported the beneficial effects of estrogen on CHD. Hanke et al. (1997) reported that estradiol levels were lower in premenopausal women with CHD than their healthy counterparts. In addition, after menopause, when estrogen levels are known to decrease, the incidence of CHD in women not receiving estrogen replacement therapy increases (Wingard et al., 1989). In contrast, those women receiving estrogen replacement after menopause, decrease their risk of cardiovascular mortality by 50% compared to those women not receiving hormonal therapy (Stampfer et al., 1991). It has been suggested that estrogen may exert a beneficial effect directly on arterial vessel walls and by its effect on cholesterol metabolism (Hanke et al., 1997). However, the exact mechanism relating estradiol levels and CHD remains unclear and requires further investigation (Haskell, 1984).

Another hormone that may have implications with CHD due to its well-documented decline with age is DHEA. Research indicates that both DHEA and DHEAS appear to be associated with the reduction of cardiovascular disease risk factors (Haffner and Valdez, 1995). DHEA is also associated with the reduction of nicotinamide adenine dinucleotide phosphate (NADPH) activity, which will reduce fatty acid synthesis and
subsequently lower the amount of low density lipoproteins (LDL) available to be oxidized (Watson et al., 1996). In addition, Jesse et al. (1995) have reported that DHEA is capable of retarding platelet aggregation, which may contribute to the cardioprotective effect of this hormone.

Hormones also play an important role in the preservation of muscle. Although women appear to lose less total muscle mass than men with aging, relative (%) reductions in muscle mass are similar (Proctor et al., 1998). Cross-sectional data reports that 35-40% of skeletal muscle mass is lost between the ages of 20 and 80 years (Evans, 1995; Fleg and Lakatta, 1988). Age related losses in muscle mass and function appear to be related to diminished levels of anabolic hormones (Proctor et al., 1998). This is supported by the findings of Butterfield et al. (1997) who reported that growth hormone administration in elderly women could significantly increase muscle protein synthesis. Although exercise cannot completely stop the loss of muscle mass as one ages, it can decrease the rate of muscle loss. In support of this belief, Tseng et al. (1995) reported that physically active older individuals maintain higher levels of muscle mass and function with aging compared to their sedentary peers. The evidence presented here suggests that several endogenous anabolic hormones confer substantial health benefits, and in general, enhancing levels of these hormones improves health.

**Steroid Hormones**

Steroid hormones are produced by the adrenal cortex, the gonads (testes and ovaries), and the placenta during pregnancy (Vander et al., 1994). The adrenal cortex is responsible for the secretion of approximately 40 steroid hormones, which are classified
as mineralocorticoids, glucocorticoids, or androgens (Fox et al., 1993). Androgens promote the development of male secondary sex characteristics (Fox et al., 1993). Although androgens are primarily secreted by the testes in males, some production does take place in the adrenal glands in both sexes (Fox et al., 1993). The five main steroid hormones produced from the adrenal cortex are DHEA (androgen), aldosterone (mineralocorticoid), cortisol (glucocorticoid), corticosterone (glucocorticoid), and androstenedione (androgen) (Vander et al., 1994). DHEA is the most abundant steroid hormone in the body (Ebeling and Koivisto, 1994).

The other site for steroid hormone production in humans is the gonads, which includes the ovaries in females and the testes in males. Testosterone is the main androgen secreted by the testes. Although DHEA is the most abundant androgen in the body (Ebeling and Koivisto, 1994), testosterone is considered the most potent androgen because of its androgenic and anabolic effects (Fox et al., 1993; Vander et al., 1994). In females, the ovarian cells have a high concentration of the enzyme aromatase, which converts testosterone to the major female sex hormone, estradiol (Vander et al., 1994). The ovaries are also responsible for the secretion of a second major steroid hormone called progesterone. It should be noted that the ovaries do release a small amount of testosterone in females, and a very minor amount of estradiol is produced from testosterone in the testes of males.

The ovaries are the main source of estradiol and luteal phase progesterone, while the adrenal glands are the primary source of DHEA and DHEAS (Shangold, 1984). Both the ovaries and the adrenal glands are the main sources of androstenedione and
testosterone in females with both contributing approximately equal amounts of each hormone (Shangold, 1984).

Cholesterol is the precursor of all steroid hormones (McArdle et al., 1994). Although many of the steroid-producing endocrine glands synthesize some of their own cholesterol, the major source of cholesterol is delivered to the cells by plasma lipoproteins that are produced in the liver and circulate in the blood (Vander et al., 1994). Steroids are highly lipid-soluble, and once synthesized they diffuse easily across the plasma membrane of the steroid producing cell and enter the interstitial fluid and subsequently the bloodstream (Vander et al., 1994).

**Testosterone**

Testosterone is a steroid hormone that has an anabolic effect on the muscle tissue (Deschenes et al., 1991). In males, the hypothalamus secretes gonadotrophin-releasing hormone (GnRH) which stimulates the anterior pituitary to release luteinizing hormone (LH) and FSH into the bloodstream (Deschenes et al., 1991). Luteinizing hormone stimulates the production of testosterone in the Leydig cells of the testes (Kraemer, 1992). In females, LH stimulates the production of small amounts of testosterone from the ovaries (Deschenes et al., 1991).

Testosterone in plasma exists in three forms: bound to albumin (53-55%), bound to SHBG (43-45%), and free (~2%) (Sodegard et al., 1982). Testosterone that is SHBG-bound is not biologically active (Sodegard et al., 1982). Normal blood testosterone (total) levels for males are 19.85 ± 4.68 nmol/L (Tietz and Logan, 1986), and in females testosterone levels can range anywhere between one-tenth to one-half as much as males.
The primary source of testosterone in females comes from the peripheral conversion of androstenedione and DHEA (Kirschner and Bardin, 1972). Androstenedione accounts for approximately 50-70% of plasma testosterone in normal women, while DHEA serves as a precursor for 15% of testosterone (Kirschner and Bardin, 1972). The remainder of testosterone in females is secreted from the ovaries (5-20%), and the adrenal glands (0-30%) (Kirschner and Bardin, 1972). Fluctuations in the levels of testosterone are minimal during the menstrual cycle (Ross et al., 1981). It has been demonstrated that plasma total testosterone declines steeply with age in premenopausal women (Zumoff et al., 1995). By the age 40, females have approximately half the level of testosterone in circulation as they did when they were 21 (Zumoff et al., 1995). This decline is presumably linked to the decline in prehormones, DHEA and DHEAS, from which some sources of testosterone are derived (Zumoff et al., 1995). In contrast to this finding, others have reported that testosterone levels in women do not decline significantly throughout their lifetime (Labrie et al., 1997a).

Testosterone's anabolic effects are realized through an increase in protein synthesis and decrease in the rate of protein catabolism in the muscle fibre (Hedge et al., 1987). Testosterone is freely permeable to the cell's plasma membrane because it is lipid-soluble and cellular membranes consist of a lipid bilayer (Hedge et al., 1987). Once inside the cell, testosterone is capable of binding with receptors in the cytosol (Deschenes et al., 1991). It is these hormone-receptor complexes that are responsible for the increase in transcription of genes located in the nuclear deoxyribonucleic acid (DNA) that code for the synthesis of contractile proteins (Hedge et al., 1987). The messenger RNA
(mRNA) that results from this transcription is translocated from the nucleus to the cytosol where protein synthesis (e.g., translation) occurs (Hedge et al., 1987).

**Estrogens**

Estrogens are a group of 18-carbon steroids which are secreted primarily from the ovaries, and to a lesser extent from the adrenal glands in females (Martin, 1978). In males, estrogens are secreted by the adrenal glands and the testes (Martin, 1978). Estradiol (E₂) is considered the major estrogen and has received most of the focus in exercise physiology. Other less potent estrogens include estrone (E₁) and estriol (E₃) (Bunt, 1990).

Peripheral conversion of androgens to estrogens in adipose and muscle tissue have also been reported (Loucks and Horvath, 1985). Androstenedione and testosterone are both substrates for estrogen production (Ojeda, 1996). Testosterone can be converted to estradiol, while androstenedione can be metabolized into estrone (Ojeda, 1996). In both of these instances the androgens are converted to estrogens through an aromatase system (Martin, 1985). The aromatase reaction involves hydroxylation, oxidation, removal of the C-19 carbon, and the aromatization of the A ring of the androgen (Ojeda, 1996).

One of the largest problems associated with studying the effects of exercise on estrogen has been the problem with defining an individual’s “estrogen status” (Bunt, 1990). While estradiol reference ranges for males are 29-132 pmol/L, reference ranges for females vary dramatically over the course of the menstrual cycle (Tietz and Logan, 1986). Estradiol concentrations are at their highest midcycle (367-1,835 pmol/L),
followed by the luteal phase (184-881 pmol/L) and are at their lowest during the follicular phase (37-330 pmol/L) (Tietz and Logan, 1986).

As with other hormones, the effect of estrogen is dependent not only on plasma concentrations but also on the concentration of binding globulin, the receptor site and other hormone levels (Bunt, 1986). Concentrations of estradiol for instance, are modified by circulating levels of progesterone (P₄), another ovarian steroid that produces "anti-estrogenic" effects (Jensen et al., 1987).

Among the many hormones the pituitary gland produces are the two glycoproteins, FSH and LH. These two hormones are regulated by gonadotropin-releasing hormone (GnRH), which is a decapeptide produced in the arcuate nucleus of the hypothalamus (Shangold, 1984). Androgen synthesis by the ovarian follicle and stroma is initiated by LH (Tsang et al., 1979). FSH on the other hand is responsible for follicular maturation and estrogen production (from androgen precursors) (Moon et al., 1978).

**DHEA and DHEAS**

In the adrenal cortex, DHEA(S) is synthesized from cholesterol, through a series of enzymatic reactions (McIntosh, 1999). The synthesis of DHEA in females proceeds through the Δ⁷-3-β-hydroxysteroids pathway with dominant production arising from the adrenal glands (90%) and to a lesser extent the ovaries (10%) (Speroff et al., 1989). As a result of the minimal contribution of the ovaries in the production of DHEA, fluctuations in this hormone are minimal during the menstrual cycle (Ross et al., 1981).

DHEAS is the predominant adrenal steroid for both sexes (Orentreich et al., 1984). Plasma DHEAS concentration is approximately 1,000-fold higher than total
estrogens in young adult females and approximately 200-fold higher than testosterone and DHEA in young adult males (Beer et al., 1994). DHEA(S) production in the adrenal glands is under the control of adrenocorticotropic hormone (ACTH) and possibly other tropic hormones (Berdanier et al., 1993). Peak levels of DHEAS occur at age 19-20 in females and age 20-24 in males, thereafter these levels decline in both sexes (Orentreich et al., 1984). Labrie et al. (1997a) reported that serum DHEA levels fall 44.5% in women between the ages of 20 to 50. This decline could explain the bone loss that precedes any detectable decrease in ovarian steroidogenesis in premenopausal women (Labrie et al., 1997a). The decline in DHEA(S) with age may be attributed to a decreased responsiveness of 17, 20 lyase and 3-β-hydroxysteroid sulfotransferase genes to ACTH or other hormones that stimulate adrenal steroidogenesis (Mortola and Yen, 1990). In contrast, other adrenocortical hormones remain relatively unchanged with advancing age (Hinson and Raven, 1999).

The metabolic clearance rate (MCR) for DHEA and DHEAS differ greatly. The MCR of DHEA is much quicker, in the range of 2,000 L/day compared to its sulfur conjugate, DHEAS, which has the lowest MCR of any steroid at 13 L/day (Longcope, 1996). There appears to be no significant differences in MCR between males and females (Longcope, 1996). The difference in MCR between these two steroids may be explained, at least in part, by their different binding strengths to albumin (Longcope, 1996). In contrast to the weak binding of DHEA to albumin, DHEAS is strongly bound to albumin. It is believed the binding of DHEAS to albumin is powerful enough to affect the metabolism of this steroid (Longcope, 1996).
It is apparent that DHEA and DHEAS interconvert, with the metabolism of DHEAS to DHEA being the more dominant pathway. Target tissues such as the gonads, skin, and adipose tissue possess the enzyme DHEAS sulfatase which is capable of converting DHEAS to DHEA (McIntosh, 1999). The magnitude of this conversion is disputed. Bird et al. (1984) reported that approximately 60% of DHEAS that enters the bloodstream will be converted to DHEA. Haning et al. (1989) reported a much smaller conversion of 32%. The discrepancy between these two studies is likely due to differences in methodology. The percentage of DHEA that re-enters the bloodstream as DHEAS is much smaller at approximately 6% (Bird et al., 1978).

Both DHEA and DHEAS are used as precursors for the synthesis of androgens and to a lesser extent estrogen synthesis (Berdanier et al., 1993). Both DHEAS and DHEA can be converted to androstenedione and testosterone, however, DHEAS appears to be the more dominant precursor for these two products (Longcope, 1996). Small amounts of estrogen can also be aromatized from both DHEA and DHEAS in women although the quantity is too small to be considered a significant source (Longcope, 1996).

The importance of DHEAS to women may be related to its conversion to testosterone. In females, approximately 65% of circulating testosterone is derived from the metabolism of peripheral precursors (Kirschner and Bardin, 1972). Haning et al. (1993) demonstrated that DHEAS could be taken up by the ovarian follicle, converted to testosterone, and consequently secreted into the bloodstream.

The precise biological function of DHEA and its sulfate ester, DHEAS, remain unclear at present (Ebeling and Koivisto, 1994). Previous research suggests that DHEA could act to preserve an increased bone mass, maintain a favourable lipid profile, and
prevent the problems often associated with menopause (Labrie et al., 1997b). Numerous reports also indicate the association between low DHEA(S) levels and CHD (Barrett-Connor et al., 1986; Feldman et al., 1998; Jesse et al., 1995). It has also been speculated that DHEA may have antitumoral and antiobesity effects (Bernadier et al., 1993). Caution should be used when interpreting these findings, however, due to the limited number of studies using humans and with the majority of these using only male subjects.

Nordin et al. (1985) reported that low plasma concentrations of DHEA may be a predictor of osteoporosis (Nordin et al., 1985). In support of this finding, Labrie et al. (1997b) reported an increase in bone density when DHEA was administered topically as a skin cream in healthy, postmenopausal women. However, to date, it has not been clarified if this association is due to its role as an estrogen precursor, or if DHEA has a direct role in bone formation. Clearly, more research on the role of DHEA(S) in health and disease needs to be performed.

**Growth Hormone (Somatotrophin)**

Growth hormone is the most abundant hormone synthesized in the anterior segment of the pituitary gland (Chattoraj and Watts, 1986). This peptide hormone is synthesized in somatotropic cells of the anterior pituitary and is stored in intracellular granules (Chattoraj and Watts, 1986). The secretion of growth hormone is under hypothalamic control, and is stimulated by the release of growth hormone-releasing hormone (GHRH) and inhibited by the release of somatostatin (Borst et al., 1994). Growth hormone is released in an episodic manner, with six to eight pulses per day (Borst et al., 1994). Growth hormone concentrations in the blood of normal adults
remain relatively low and stable throughout the day (Chattoraj and Watts, 1986). A marked rise in this hormone occurs 60-90 minutes into sleep with peak values occurring during the period of deepest sleep (stages III and IV) (Chattoraj and Watts, 1986). Other smaller peaks can also be noted 3-4 hours after meals (Chattoraj and Watts, 1986). Plasma concentrations of growth hormone are at their lowest levels in the morning (Corpas et al., 1993).

The degradation of growth hormone proceeds quickly (Borst et al., 1994). When growth hormone is attached to one of its two binding proteins the plasma half-life is approximately 20 minutes (Baumann et al., 1987). In contrast, the uncomplexed form has a half-life of less than 5 minutes (Baumann et al., 1987).

Growth hormone has been reported to play a critical role in the growth and development of bone, connective, visceral, adipose and muscle tissue (Hedge et al., 1987). It also promotes protein synthesis by increasing amino acid transport through cell membranes, stimulating ribonucleic acid (RNA) formation, or activating cellular ribosomes (McArdle et al., 1994). The release of growth hormone also influences metabolism. Increased concentrations of this hormone are associated with decreased carbohydrate utilization and a subsequent increase in lipid metabolism (McArdle et al., 1994).

The effects of growth hormone can either be direct or indirect. While some research suggests that growth hormone may exert its anabolic effect by binding directly to receptors on the muscle tissue, most data indicates that the anabolic effects are indirect and carried out by somatomedians (Hedge et al., 1987; Kraemer, 1988). Growth hormone stimulates the liver and other cells in the body to secrete growth-promoting
peptide hormones called somatomedians, which are more commonly referred to as insulin-like growth factors (Kraemer, 1988).

It has been well documented that growth hormone decreases with age (Rudman et al., 1981). Progressive alterations in body composition are also associated with aging including an increase in adipose tissue and a decline in lean body mass (Rudman, 1985). Recently, studies have involved the administration of recombinant growth hormone to the elderly population with the prospect of reversing these changes. Positive body composition changes were reported by Rudman et al. (1990) who administered synthetic human growth hormone to men 61 to 81 years old for 6 months. Results indicated a 14.4% decrease in adipose-tissue mass, an 8.8% increase in lean body mass, and a 1.6% increase in average lumbar vertebral bone density. As with other hormone replacement therapies, several negative consequences are associated with the use of recombinant growth hormone including costly expenses, repeated injections, and adverse side effects including reduced insulin sensitivity (Marcus et al., 1990).

In contrast to the clinical use of administering growth hormone, evidence exists that some athletes are administering growth hormone anticipating ergogenic effects such as increased strength. Recent seizures of athlete’s luggage containing synthetic growth hormone has raised concerns (Jenkins, 1999). The number of studies involving supraphysiological doses of growth hormone are small, but suggest no ergogenic properties and potential adverse reactions. Yarasheski et al. (1992) administered recombinant growth hormone for 12 weeks to healthy young untrained men involved in resistance training. Despite increases in serum growth hormone and IGF-I levels of 6-fold and 3-fold respectively, no differences in strength were observed between the treated
and placebo group. Similar findings were reported by Deyssig et al. (1993), who used power athletes. In both studies, approximately 25% of participants were forced to withdrawal due to the development of carpel tunnel syndrome (Deyssig et al., 1993; Yarasheski et al., 1992). Unfortunately, due to the sparse number of studies conducted, it is unknown how many and with what severity the side effects associated with chronic, supraphysiological doses of growth hormone exist.

**Insulin-Like Growth Factors (Somatomedians)**

Somatomedians, commonly referred to as Insulin-Like Growth Factors (IGFs) are a group of polypeptides that are synthesized primarily in the liver and are released into the blood after stimulation of the liver by growth hormone (Deschenes et al., 1991). It was originally thought that IGF of hepatic origin was the only pathway to mediate the actions of growth hormone (Borst et al., 1994). However, it is now recognized that many of the actions of growth hormone are mediated by locally produced IGF-I acting in a paracrine or autocrine manner (LeRoith and Roberts, 1993). Extrahepatic sites of IGF-I production include muscle, bone and adipose tissue (LeRoith and Roberts, 1993). The local production of IGF-I is under the influence of different regulators dependent on the tissue type (Muller et al., 1999). For example, in bone, sex steroids, growth hormone, and parathyroid hormone regulate IGF-I production, whereas in the reproductive system, sex steroids appear to be the main regulators of this hormone (Muller et al., 1999).

IGFs travel in the blood attached to binding proteins (Kraemer, 1994). Among the six binding proteins that have been documented (Jones and Clemmons, 1995), over 95% of circulating IGF-I and IGF-II is bound to binding protein three (Muller et al.,
IGFs are released as free hormones to bind to receptors on the muscle cell and carry out the growth promoting properties attributed to growth hormone (Deschenes et al., 1991).

IGF-I is a 70-amino acid polypeptide essential for growth postnatally (Kraemer, 1994). Insulin-like growth factor II (IGF-II) is a 67-amino acid polypeptide, which is suspected to be a growth factor prenatally although its physiological significance remains unknown (Bang et al., 1990). Age, sex, nutritional status, and growth hormone all appear to affect serum concentrations of IGF-I (Muller et al., 1999). At birth, plasma levels of IGF-I are low, but rise substantially during childhood and puberty (Muller et al., 1999). During the third decade of life, IGF-I levels begin to decline linearly with age (Bennett et al., 1984). Research by Rudman et al. (1981) indicating that growth hormone also decreases with age, has led to the speculation that the decline of these two hormones are linked.

Evidence exists that IGF concentrations may play an important role in skeletal growth and development (Bennett et al., 1984). Research indicates that IGF-I levels are low in individuals suffering from growth hormone deficiency, which is associated with childhood osteopenia (Bennett et al., 1984). As well, it has been reported that IGF-I levels are elevated in acromegaly, which is associated with increased bone density (Bennett et al., 1984). Based on knowledge that IGF-I can stimulate both DNA and protein synthesis (Jones and Clemmons, 1995), the decline of this hormone with age could contribute to the reduction in bone density that is associated with osteoporosis.
Oral Contraceptives

In 1995, it was reported that 27% of American women between the ages of 15-44 used oral contraceptives (OC) as a form of birth control (Centers for Disease Control and Prevention, 1998b). Oral contraceptives are composed of the synthetic hormones estrogen and progestin. The administration of these hormones often leads to altered endocrine and metabolic function that needs to be considered with exercise.

Bemben and coworkers (1992) demonstrated the effects of chronic OC use on growth hormone responses during prolonged submaximal exercise in moderately active women. The exercise protocol involved 90 minutes of treadmill walking at 50% maximal oxygen uptake. Testing was performed between days 10 to 21 of the pill cycle for the OC group and during the luteal phase of the menstrual cycle in controls. Results indicated that chronic OC use was associated with enhanced growth hormone responses during the early portion of exercise. This increase in growth hormone due to OC use has been reported by others (Bernardes and Radomski, 1998; Bonen et al., 1991; Davidson and Holzman, 1973). In addition, Bemben et al. (1992) reported that energy substrate utilization appeared to be influenced by chronic OC use. Oral contraceptive users had lower blood glucose levels throughout the exercise session, and diminished carbohydrate oxidation.

The speculation is that the exogenous estrogen contained in OC incites the accelerated release of growth hormone. Earlier work by Frantz and Rabkin (1965) indicated that exogenous estrogens have a modulatory role in the release of growth hormone. Evidence that estrogen can act as a physiological stimulant toward growth hormone release includes sex differences in growth hormone responses to exercise (Ho et
The enhanced growth hormone response to exercise in the OC group may also be a result of lower blood glucose levels compared with their eumenorrheic counterparts (Bemben et al., 1992). Low glucose levels can increase growth hormone secretion by stimulating hypothalamic glucoreceptors (Galbo, 1983).

Increases in growth hormone can promote carbohydrate-sparing through its stimulation of lipolysis (Bemben, 1993). Consistent with the carbohydrate-sparing effect of growth hormone, Bonen et al. (1991) found higher serum levels of free fatty acid in OC users as a consequence of increases in growth hormone. In contrast, Bemben et al. (1992) found that although carbohydrate oxidation was diminished in OC users, no difference in fat utilization was noted. The authors cited that a possible explanation for the apparent discrepancy in energy substrate utilization might be related to changes in protein utilization that was not accounted for in the study. Based on previous work indicating that during exercise luteal levels of endogenous estrogen and progesterone are associated with protein catabolism (Lamont et al., 1987), speculation exists that use of OC may enhance protein utilization.

Bemben (1993) also suggested that the interaction between female sex steroids and endogenous opioids could modulate the growth hormone response. Previous research indicates that OC use has been associated with enhanced hypothalamic opioid activity (Casper et al., 1984) and that beta-endorphins are physiological stimulators of growth hormone release (Morley, 1981).

Bonen and coworkers (1991) demonstrated that in contrast to controls, no increases in estradiol were observed in OC users during a 30-minute walk at ~40% VO2max, followed by a 30-minute walk at 85% VO2max. Suppression of estradiol in OC
users during exercise may be related to reduced FSH levels that are also observed in these individuals (Bonen et al., 1991). Deficient levels of FSH lower the synthesis of estradiol, which likely limits the pool of estradiol available for secretion during exercise (Bonen et al., 1991).

Resting endocrine levels have also been reported to be affected by OC use (Davidson and Holzman, 1973; Kasdorf and Kalkhoff, 1988). Contrary to Bonen and colleagues (1991), who did not observe significantly lower levels of estradiol at rest in OC users, Kasdorf and Kalkhoff (1988) reported that individuals had significantly lower basal estradiol levels after both 3 and 6 months of OC treatment. In addition, Kasdorf and Kalkhoff (1988) observed that resting serum concentrations of both free and total testosterone, as well as androstenedione, were lower after treatment sessions of OCs, whereas, DHEAS did not change (Kasdorf and Kalkhoff, 1988). It is proposed that OC use is involved in the suppression of ovarian secretion of these androgen hormones. Basal levels of growth hormone may be OC dose dependent. Increases in basal growth hormone levels have been associated with higher (50 μg mestranol, 1 mg norethindrone) dose OC (Davidson and Holzman, 1973) while lower dosage (35 μg estrogen, ≤ 1 mg progestin) has not affected basal levels of this hormone (Bemben et al., 1992).

**Aerobic Exercise**

Aerobic exercise can help maintain and improve various aspects of cardiovascular function (Franklin, 1998). Aerobic training can also lead to metabolic changes including increased respiratory capacity, lower blood lactate concentrations at a given submaximal exercise intensity, improved aerobic enzyme activity, and increased mitochondrial and
capillary densities (Kraemer, 1994). In addition, aerobic exercise has been linked to reductions in risk factors associated with disease states (heart disease, diabetes, etc.), improved health status, and increased life expectancy (Franklin, 1998). While these are some of the accepted benefits associated with aerobic exercise, an increasing amount of interest has been directed toward the endocrinology of aerobic exercise. Previous findings testify that intense exercise may play a factor in the high incidence of menstrual cycle irregularities in female athletes (Baker et al., 1982). This has led to investigations on the role of androgens and estrogens in exercise. Also of interest has been the influence of exercise on androgens in postmenopausal women. It is the belief of some that if androgen levels could be raised with regular exercise, the prevalence and severity of postmenopausal health problems would decrease (Johnson et al., 1997).

**Testosterone**

While the research is sparse on women, some studies have indicated that plasma testosterone, androstenedione, and DHEA increase with aerobic exercise in females (Baker et al., 1982; Cumming and Rebar, 1985; Shangold et al., 1981; Webb et al., 1984).

In a study by Shangold et al. (1981), healthy female runners between the ages of 25 and 47 had their testosterone levels measured after 30 minutes of running at their customary pace. Results indicated that peripheral testosterone concentrations increased acutely following the run. Further analysis indicated that the mean increment was significantly greater for women during their follicular phase (19.7 ± 2.4) than their luteal phase (8.0 ± 2.3) despite similar baseline testosterone levels.

Bonen and Keizer (1987) demonstrated that testosterone levels could increase significantly in trained females during a marathon race at a running intensity between
60% and 85% VO$_2$max. This study also indicated that within two hours post-marathon, testosterone levels returned to baseline levels. These findings are in agreement with Baker et al. (1982) who found an increase in testosterone levels in females after a 10-mile run, but levels of this hormone returned to baseline 12 to 24 hours after the race. All subjects in the Bonen and Keizer (1987) study also showed an increase in testosterone before the race began, indicating that testosterone levels may show an anticipatory effect.

There appears to be a difference in the pattern of increase of hormones such as testosterone and DHEAS depending on the training status of the individual (Cumming and Rebar, 1985; Keizer et al., 1987). Keizer et al. (1987) reported that although the basal levels of testosterone and DHEAS were significantly higher in untrained compared to trained females, during acute exercise the increase in these hormones was more pronounced in the trained individuals. While muscle hypertrophy is not a typical response to aerobic exercise, aerobic exercise that is performed at high intensities has been known to increase anabolic hormones such as testosterone to counterbalance the catabolic conditions that occur. Testosterone levels may be increased to maintain protein synthesis to keep up with the protein loss (Tapperman, 1980; Terjung, 1979).

**DHEA and DHEAS**

The impact of exercise on DHEA and DHEAS levels in females has been relatively understudied. Baker et al. (1982) reported that after a 10 mile run, females had elevated plasma levels of both DHEA and DHEAS. In contrast to DHEA, which returned to baseline 12 to 24 hours after the run, DHEAS remained elevated. Bonen and Keizer (1987) later supported this continued elevation of DHEAS after a marathon run. It was reported that DHEAS levels became significantly elevated in the last 10 km of a 40 km
marathon, and that these levels remained elevated for at least 2 hours after the run. These results suggest that after heavy aerobic exercise there is an increased steroidogenic activity of the adrenal cortex, although a decreased rate of desulfation in the liver after exercise cannot be ruled out (Bonen and Keizer, 1987). Generalizations from Bonen and Keizer (1987) are limited due the small sample size (n = 5).

Limited research does indicate that DHEAS can be elevated with aerobic exercise, although the training status of the individual appears to play a critical role (Bonen and Keizer, 1987; Keizer et al., 1987). Keizer and coworkers (1987) indicated that trained females significantly increased their DHEAS levels after prolonged treadmill exercise while DHEAS levels remained unchanged in untrained subjects. In contrast, Cumming and Rebar (1983) observed an increase in DHEA after an incremental cycling exercise to maximum in both trained and untrained females.

How DHEA and DHEAS levels adapt with aerobic training remains unclear. The few studies that have been reported have found inconsistent and contradictory results. Resting levels of DHEA have been reported to be higher in sportswomen after 16 weeks of training for handball or volleyball compared to sedentary individuals (Filaire et al., 1998). Keizer et al. (1987) reported that basal levels of DHEAS were significantly higher in untrained compared to marathon-trained females. Contrary to these findings Ronkainen et al. (1986) reported that there were no differences in basal serum concentrations of DHEAS between trained runners and untrained females. The findings of Keizer et al. (1987), however, may be explained by the younger age of the untrained group since DHEAS levels are reported to decline with age (Orentreich et al., 1984). Unfortunately, none of these studies measured both DHEA and DHEAS levels in the
same subject group, and, therefore, it has been difficult to determine if exercise has a
different effect on the two hormone pools.

While the abovementioned studies investigated the effects of aerobic exercise on
DHEA and DHEAS levels in a younger population (premenopausal), perhaps a more
important issue is whether these hormones can be affected by exercise as age increases.
This is of interest because resting levels of both of these hormones have been reported to
decrease after the age of 25 (Nestler et al., 1988). DHEA and DHEAS decline
progressively and considerably with age unlike another adrenal steroid, cortisol whose
serum levels remain unchanged with aging (Johnson et al., 1997).

Previous research indicates that decreasing levels of DHEA coincide with the
adverse effects of aging such as increased mortality, decline in immune system function,
increased incidence of cancer, atherosclerosis, osteoporosis, and a declined sense of well-
being (Watson et al., 1996). These results have led researchers to believe that if levels of
DHEA and DHEAS could be raised, it could offset some of these negative effects of
aging.

Johnson et al. (1997) reported that both DHEA and DHEAS levels are elevated
after 30 minutes of treadmill exercise (at 80% VO_{2}\text{max}) in postmenopausal women. An
interesting finding from this study was that estrogen therapy appeared to enhance DHEA
response to the exercise while DHEAS was unaffected. Milani et al. (1995) indicated
that DHEAS levels in males undergoing cardiac rehabilitation were unaffected by
exercise. It should be noted, however, that the Milani et al. (1995) study involved cardiac
rehabilitation patients that were required to exercise at lower intensities (70-85% of
maximum heart rate). Therefore, an upper intensity threshold may have to be reached before DHEAS levels increase.

It is evident that more research is needed to determine the effects of aerobic exercise on DHEAS and especially DHEA. Whereas, the training status of an individual may be a factor in the response of DHEA and DHEAS to aerobic exercise, other contributing factors such as intensity and duration of the exercise bout have not been reported in previous research.

**Growth Hormone**

Circulating growth hormone levels have been reported to be elevated both during and following aerobic exercise and these elevations appear to be positively correlated with exercise intensity (Bloom et al., 1976; Galbo, 1985). VanHelder et al. (1986), using male subjects, investigated the response of growth hormone to two different 40 minute continuous aerobic cycling protocols of differing pedaling frequency and load but with similar VO₂ (40% of their maximum) and lactate levels. Their results indicated that although the higher pedaling frequency (90 rev/min) increased growth hormone levels by 200%, and the lower pedaling frequency group (50 rev/min) increased growth hormone levels by only 145%, these levels were not significantly different from each other. Both groups reached their maximum levels at 8 minutes into recovery. This finding lends support to the hypothesis that oxygen availability may be one of the regulators of growth hormone release during exercise (VanHelder et al., 1986). Farrell et al. (1986), also using the cycle ergometer, but using a higher percentage of aerobic capacity (70% VO₂max), reported a 166% increase in serum growth hormone after exercise.
Bunt et al. (1986) examined sex and training differences in growth hormone release during a 1-hour treadmill run at 60% of VO_{2}max. The study was based on previous research that indicated that the increased growth hormone response observed in females may be related to estradiol levels, which were thought to stimulate growth hormone releasing factor (Frantz and Rabkin, 1965). It was not known however, if this greater response of growth hormone in females was due to lower fitness levels. Previous research has indicated that training slows the exercise response of growth hormone (Galbo, 1985). Bunt et al. (1986) reported that females exhibited higher resting levels of growth hormone than males, regardless of training status. These higher levels in females persisted during the first half (30 minutes) of the run at 60% VO_{2}max. While females showed no statistically significant training differences, trained male runners achieved higher growth hormone levels throughout exercise and into recovery.

Studies examining the response of growth hormone during recovery from exercise have been inconsistent. Bunt et al. (1986) and Farrell et al. (1986) reported that levels of growth hormone began to decline immediately after exercise, whereas VanHelder et al. (1986) demonstrated that growth hormone levels did not peak until eight minutes into recovery. A possible explanation for these contradictory findings may be the different exercise intensities that were used. Higher intensities were used by Bunt et al. (1986) and Farrell et al. (1986), (60% and 70% of VO_{2}max respectively) compared to VanHelder et al. (1986) who used a lower intensity (40% of maximal aerobic power). Clearly, more research is needed in this area to determine the direct effects of exercise intensity and duration on growth hormone response to aerobic exercise in women.
Insulin-Like Growth Factor I (IGF-I)

Kelly et al. (1990) and Poehlman and Copeland (1990) both reported significant correlations between IGF-I and physical fitness (determined as the subject’s VO₂max). Cappon et al. (1994) demonstrated that brief high-intensity aerobic exercise could increase serum concentration of IGF-I. In the first part of their study, 11 healthy young adults were used (10 male, 1 female) to compare the growth hormone and IGF-I response to differing diets while cycling for 10 minutes at approximately 72% of their VO₂max. A meal high in fat significantly attenuated the growth hormone response to exercise compared to the high glucose or placebo diet. Insulin-like growth factor I increased equally in all protocols. IGF-I levels peaked at the end of the 10 minute exercise protocol (mean 14% above pre-exercise levels) and remained significantly elevated for 20 minutes into recovery. Contrary to previous beliefs, no substantial differences in the time course of the growth hormone and IGF-I responses were observed. The increases in IGF-I appeared to be independent of circulating levels of growth hormone. These findings were verified by Bang et al. (1990) who found that significant increases in serum concentrations of IGF-I (26 ± 5%) after 10 minutes of moderate exercise (60% of the subject’s VO₂max) were independent of the growth hormone response. Wilson and Horowitz (1987) observed significant increases in growth hormone in short children (below the fifth percentile) after 15 minutes of cycling at an intensity ≥ 60% above their resting heart rate, however. IGF-I levels remained unchanged.

Estrogen (Estradiol)

Jurkowski et al. (1978) were the first to demonstrate that estradiol could increase with exercise. Using healthy females aged 20-24, 20 minutes of exercise at 30-35% of
maximum power output (light), 20 minutes at 60-66% (heavy), and exercise to exhaustion at 85-95% was performed. These subjects were tested at both midfollicular and midluteal phases of their menstrual cycle to determine if menstrual cycle phase was a factor in estradiol response. In the luteal phase, nonsignificant increases in estradiol were observed with light exercise. Significant increases were reported with heavy exercise and exhaustive exercise when compared to resting levels. In the follicular phase, exercise-induced increases in estradiol were only significant at exhaustion. These results indicate that both exercise intensity and menstrual cycle phase play an important role in the response of estradiol to aerobic exercise. Increased levels of this hormone during the luteal phase may indicate that the ovary is more sensitive to stimuli such as exercise during this time (Jurkowski et al., 1978). Jurkowski et al. (1978) concluded that the increases in estradiol during exercise were ovarian in origin, based on the assumption that the contribution the adrenal gland makes to plasma levels of estradiol is relatively small.

As with other plasma hormones, the increased levels of estradiol may not be solely due to increased production. Increased hormone levels may also occur from decreased metabolic clearance due to the decrease in hepatic blood flow during exercise (Rowell, 1974). Metabolic clearance rate is inversely related to exercise intensity (Astrand and Rodahl, 1986). Mild to moderate exercise generally enhances the clearance rate due to increased blood flow, but intensive exercise decreases this rate due to the shift of blood flow to working muscles (Montagnani et al., 1992). Bonen et al. (1991), in a study comparing the exercise responses of young women taking OCs to a control group, discussed this factor. In this study it was concluded that the absence of an increase in estradiol during exercise in women taking OC indicated the secretion of estradiol was
impaired in this group. In comparison, the exercise-induced increase in estradiol seen in the control group was believed to be due to an increase in secretion rather than a decrease in hepatic clearance.

Whereas short aerobic exercise bouts appear to increase the plasma concentration of estradiol, it appears that prolonged aerobic activity may decrease estradiol levels. Montagnani et al. (1992) examined the effects of a 2-hour continuous treadmill run on the plasma concentration of estradiol and the MCR of this hormone in young untrained women. The running intensity in this study corresponded to 70% of their running speed at anaerobic threshold. Results indicated that estradiol decreased by 30% and the MCR of this hormone increased significantly by the end of the test. Caution should be used when generalizing these findings however, due to the small sample size used (n=10). Another important consideration is that these were untrained women. It is unknown if training status may affect these findings.

Previous studies have indicated increased human growth hormone levels and exercise responses in females on estradiol therapy (Chakmakjian and Bethune, 1968), women taking OC (Davidson and Holzman, 1973), and during ovulation (Hansen and Weeke, 1974). These findings have led some to believe that increased levels of estradiol may enhance growth hormone secretion. Hornum et al. (1997) measured the response of growth hormone concentrations after a 10 minute high-intensity cycling exercise during both high estradiol (periovulatory phase) and low estradiol (follicular phase) conditions. While growth hormone increased significantly in response to exercise for both estradiol conditions, growth hormone levels before and during exercise were greater with higher estradiol levels. Contrary to these findings, Hansen and Weeke (1974) found no
differences in growth hormone response between users and nonusers of OC, and Bonen et al. (1983) reported that menstrual cycle phase had no significant effects on growth hormone secretion.

It should be noted that although estradiol may play a role in augmenting growth hormone levels, other factors are certainly involved. Bunt (1986) reported that resting levels of estradiol only accounted for a small amount of variability (11.4%) observed in the growth hormone response compared to training-related variables such as absolute workload (36.3%) and training status (32.2%).

**Resistance Exercise**

Heavy resistance exercise has proven to be an effective stimulus for both strength and muscle fiber hypertrophy (Kraemer et al., 1991). Two of the primary anabolic hormones that have been linked to muscle growth and remodeling are testosterone and growth hormone (McArdle et al., 1994).

Resistance exercise helps offset the loss in muscle mass and strength typically associated with normal aging. Additional benefits from regular resistance exercise include improved bone health and a reduction in risk for osteoporosis; improved postural stability, thereby reducing the risk of falling and associated injuries and fractures; increased flexibility and range of motion; an extension of independent living in the elderly; and an increase in recreational opportunities.

Previous research has indicated that under normal conditions human muscle strength decreases with increasing age after middle-age and especially at the onset of the sixth decade in both sexes (Frontera et al., 1991). This decline in strength with aging is
related to a reduction in muscle mass (i.e. sarcopenia) which is observable in both men and women. Sarcopenia is often associated with the decreased frequency and intensity of daily physical activities. A decline in muscle strength may also be a result of alterations in hormone concentrations, since androgen levels have been reported to decrease with age in both sexes (Hakkinen and Pakarinen, 1993; Jaffe, 1986).

The remodeling of muscle tissue after exercise is a complex process involving a multitude of events ranging from cell receptors interacting with various hormones to the DNA production of new contractile proteins (Kraemer, 1992). The endocrine system plays an important role in remodeling muscle cells through a link of complex cellular processes (Florini, 1987). It is these hormones that form a vital link between exercise and the adaptive response of the muscle and energy systems. Anabolic drugs are often used as a substitute to the body’s natural release of hormones. In this instance, cells respond to a pharmacological level of a substance rather than a physiologically timed response and release of the body’s natural hormones (Kraemer, 1992).

**Testosterone**

Hakkinen and Pakarinen (1993) reported that the decrease in testosterone in females with aging was positively correlated with cross-sectional area of the quadriceps femoris muscle and maximal voluntary force production of the leg extensor muscles. Celotti and Cesi (1992) observed that testosterone influences androgen receptors found in the myofibrils resulting in protein synthesis. This knowledge led some researchers to believe that decreasing basal levels of testosterone may lead to a decrease in muscle anabolism, eventually contributing to muscle atrophy and progressive weakness (Hakkinen and Pakarinen, 1993).
Unfortunately, most studies to date have used young or middle-aged males as subjects leaving a gap in research dealing with females and the elderly. Most studies that have included women have shown no significant changes in testosterone levels as a consequence of acute heavy resistance exercise (Fahey et al., 1976; Hakkinen and Pakarinen, 1995; Kraemer et al., 1991; Kraemer et al., 1993; Kraemer et al., 1998; Weiss et al., 1983). In the only study showing positive exercise-induced increases in testosterone, Cumming et al. (1987) observed a small but significant increase in total testosterone concentration in females after isokinetic resistance exercises.

Kraemer et al. (1998) investigated the hormonal adaptations to an 8 week heavy resistance exercise program. At both 6 weeks and 8 weeks after the commencement of the program basal testosterone levels were increased for both untrained males and females. This observed increase in basal levels of testosterone is in conflict with other studies (Hakkinen et al., 1990, 1992; Staron et al., 1994). In another study, Stoessel et al. (1991) observed no significant differences in basal levels of testosterone between untrained women and members of the United States women's weightlifting team. It should be noted that even though Kraemer et al. (1998) found significant increases in basal levels of testosterone in females due to training, no significant exercise-induced increases in testosterone were detected after each session. These studies indicate a lack of resistance exercise-induced increase in testosterone concentration (Fahey et al., 1976; Hakkinen and Pakarinen, 1995; Kraemer et al., 1991; Kraemer et al., 1993; Kraemer et al., 1998; Weiss et al., 1983) and have led researchers to believe that another anabolic hormone, such as the growth hormone, may be the direct contributor to anabolic adaptations in female muscles (Kraemer et al., 1993).
DHEA and DHEAS

There is a paucity of information investigating the levels of DHEA and DHEAS in response to resistance exercise, especially in women. Those studies that have been conducted have found inconsistent results. Copeland (1998) investigated both DHEA and DHEAS levels in post-menopausal women after resistance exercise sessions of varying volume. No changes in these two hormones were observed after any of the sessions. In contrast, Tremblay (1994) demonstrated a rise in DHEAS levels in males performing a resistance exercise session and that these levels remained elevated during recovery.

Alen et al. (1988) observed a decrease in DHEA concentrations over a 24-week strength training period in males. However, DHEAS levels remained statistically unchanged throughout the training period. Tremblay (1994) reported higher basal levels of DHEAS in resistance trained males than endurance trained males.

Growth Hormone

Previous research indicates that strenuous resistance exercise can raise growth hormone levels in both sexes, although this response may be diminished in the elderly (Pyka et al., 1992). Using two distinctly different heavy resistance protocols, Kraemer et al. (1991) demonstrated that serum concentrations of growth hormone, but not testosterone, were affected in females during an acute bout of exercise. Although both protocols used the same eight resistance exercises, one protocol utilized a 5 RM load, and 3 minute rest periods. The second protocol consisted of a 10 RM load, 1 minute rest periods, and had the higher total work of the two protocols. This second mentioned protocol was considered the more anaerobic workout of the two. Female subjects
produced a distinct and sustained elevation in growth hormone throughout the more anaerobic protocol but the other protocol had virtually no effect on growth hormone concentration.

In a follow-up study by Kraemer et al. (1993) it was determined that extending the length of the rest period (10 RM with 3 minutes rest), or when the duration of the set was decreased by increasing the resistance (5 RM with 1 minute rest), growth hormone showed no significant increase above resting concentration. Therefore, it appears that exercise that produces the greatest demand on anaerobic glycolysis will have the greatest effect on elevating serum growth hormone (Kraemer et al., 1993). Pyka et al. (1992) investigated the effect of exercise intensity on growth hormone response in young adults. The protocol consisted of 13 exercises with varying loads of 60%, 70%, and 85% of the individual’s previously determined 1 RM. Three sets of 8 repetitions with 30 seconds rest between sets was used. Results indicated that growth hormone response was minimal at 60% and increased progressively at 70% and 85% of 1 RM. These results lend support that growth hormone response is intensity dependent.

VanHelder et al. (1984a) compared two different types of exercises of nearly equal duration and work output. Results indicated that although growth hormone was significantly higher at the end of the 20 minute anaerobic exercise and up to 30 minutes into recovery compared to basal levels, no significant changes occurred during the aerobic exercise. Contrary to these findings, Kindermann et al. (1982) reported that subjects had significantly higher levels of growth hormone after an aerobic treadmill run, than after an anaerobic run. These conflicting findings may be explained by the differing duration and energy expenditure in the two protocols.
VanHelder et al. (1984b) studied the response of growth hormone in males to different repetition number and load magnitude, while intensity, total work output and duration of exercise was equal. The authors found that seven sets of seven vertical leg lifts at 85% of 7-RM elicited a significant increase in growth hormone concentration, whereas seven sets of 21 repetitions at 28% of 7-RM did not achieve significant changes in the hormone.

A study by Mulligan et al. (1996) using a constant rest period (1 minute) and different volumes of resistance exercise (1 set vs. 3 sets) compared hormonal responses in a group of young eumenorrheic females (age 24.1 ± 4.3). Results indicated that serum growth hormone in the multiple-set protocol was significantly higher than the single-set protocol immediately following and up to 15 minutes after the exercise. While the multiple-set protocol showed serum growth hormone was significantly higher than baseline values immediately following exercise and at all post-exercise times (up to 30 minutes), the single-set protocol only showed a significant increase over baseline levels at the 15 minute post-exercise time period.

What causes changes in growth hormone with exercise remains unclear. Throughout the years a number of possible stimuli have been suggested including hypoxia, hypoglycemia, decreased insulin levels, and increased lactate levels (Grossman et al., 1984; Miller et al., 1984; VanHelder et al., 1986). Although VanHelder et al. (1984b) found significant correlations during exercise between lactate and growth hormone levels for both anaerobic (r = 0.87) and aerobic exercises (r = 0.93), other investigators (Karagiorgos et al., 1979) have found no such correlations. Sutton et al. (1976) suggested that blood lactate levels may not influence growth hormone release by
demonstrating that artificial manipulation of lactate levels did not affect growth hormone levels.

The concentration of growth hormone maybe influenced by somatostatin, a hormone that inhibits the secretion of growth hormone from the pituitary gland (Deschenes et al., 1991). With age it has been suggested that the pituitary gland undergoes increasing sensitivity to somatostatin releasing factor (SRIF) (Deschenes et al., 1991). This increased sensitivity could be a factor in the decreased growth hormone response to exercise in the elderly. Levels of SRIF have been found to be at their highest at the same time that growth hormone levels are at their maximum levels (Cuttler et al., 1986). This indicates that growth hormone may be regulated by SRIF levels through a feedback loop (Deschenes et al., 1991).

Growth hormone release may also be affected by β-endorphins (Deschenes et al., 1991). During intense exercise it has been shown that β-endorphins are released into the blood and that these hormones may be able to offset the inhibitory effect of SRIF on growth hormone (Borer et al., 1986). In addition to these factors, another mechanism that may increase growth hormone secretion is related to the exercise-induced mobilization of calcium (Deschenes et al., 1991). It has been previously reported (Ljunhall et al., 1984; Vora et al., 1983) that aerobic exercise of moderate intensity results in an elevation of calcium and is related to exercise intensity. The early phase of growth hormone release is elicited by growth hormone-releasing factor (GRF) which is dependent on calcium mobilization (Login et al., 1986). Therefore, increased amounts of calcium in the blood may increase calcium uptake by the pituitary cells upon GRF binding. This intracellular calcium can then be used as a secondary messenger increasing the rate of DNA
transcription within the pituitary gland. This will lead to an increase in growth hormone synthesis and secretion from the pituitary gland (Login et al., 1986).

**Insulin-Like Growth Factor (IGF-I)**

Kraemer et al. (1990) and Kraemer et al. (1991) found that IGF-I increased after heavy-resistance exercise with no associated changes in growth hormone over a 1-2 hour recovery period. However, the acute responses of IGF-I still remain unclear. No changes in serum IGF-I were recorded 23 hours after a moderate-intensity and low-volume heavy resistance exercise session (Kraemer et al., 1992).

Kraemer et al. (1995b), using young male subjects, investigated the response of IGF-I and growth hormone to a high-intensity bout of heavy-resistance exercise consisting of three sets of eight exercises at 10 RM and 1 minute rest between sets and exercises. In this study, the acute 24-hour recovery period was observed, based on previous research that indicated that serum IGF-I concentrations increased within 24 hours after recombinant human growth hormone was administered. Post-exercise levels of both growth hormone and lactate were found to be significantly greater than baseline values, with the greatest increase in growth hormone found immediately after the heavy-resistance exercise. However, the most notable finding in this study was that no significant increases in IGF-I were observed either post-exercise or during recovery. These results indicate that there may be a difference in IGF-I synthesis between growth hormone administered acutely and endogenous growth hormone. This study, along with the aerobic exercise results of Cappon et al. (1994), demonstrate that IGF-I concentrations can function independently of the changes in acute growth hormone concentrations. Other investigators have suggested that the magnitude of resting IGF-I
may play an important role in the responsiveness to an exercise-induced growth hormone increase (Marcus et al., 1990). Marcus et al. (1990) observed a blunted response of IGF-I on the last day of exogenous growth hormone administration, indicating the possibility of an upper limit of responsiveness. If an upper limit does exist, this would help explain the lack of IGF-I response found by Kraemer et al. (1995b). Subjects used in the study by Kraemer et al. (1995b) were weight trained and may have already reached their upper limit of IGF-I concentrations. Further analysis did indicate that these subjects had higher resting values of IGF-I than those subjects in previous studies where acute increases in IGF-I were noted (Kraemer et al., 1990; Kraemer et al., 1991).

**Estrogen (Estradiol)**

The response of estrogen to resistance exercise has been relatively neglected in previous research. Kraemer et al. (1995a) demonstrated that young, untrained females could elicit increases in estradiol after three sets of four resistive exercises at 10 RM. This response was observed during both follicular and luteal phases with a more pronounced increase seen in the latter.

Walberg-Rankin et al. (1992) investigated the response of estradiol to resistance exercise during energy balance and energy restriction. The subjects used in this study were young (mean age 24.4) females who were familiar with weightlifting. The exercise protocol consisted of four sets of eight exercises at approximately 85% of the estimated 1 RM with one and one half minutes rest between sets. Estradiol increased from baseline to immediately post-exercise under both energy balance and negative energy balance conditions, but this increase was only significant during the hypocaloric diet. These findings suggest that diet may play a role in the release of this hormone during exercise.
Walberg-Rankin et al. (1992) cautioned that because estradiol is known to have a negative feedback effect on the hypothalamus to reduce gonadotropin releasing hormone (GnRH), it may be possible that repeated elevations of this hormone during weightlifting, along with energy restriction, could contribute to disruptions in the menstrual cycle. However, there has been no research to date to validate this suggestion.

Clearly there are contradictions and inconsistencies throughout the exercise endocrinology literature. In many cases these inconsistencies arise from variations in methodological protocols (Tremblay et al., 1995). Despite the equivocal nature of the literature, Table 1 attempts to summarize the anabolic hormone responses to aerobic versus resistance exercise.

### Table 1: Summary of anabolic hormone responses to aerobic and resistance exercise.

<table>
<thead>
<tr>
<th>VARIABLE</th>
<th>AEROBIC EXERCISE</th>
<th>RESISTANCE EXERCISE</th>
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<tr>
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<td>Male</td>
<td>Female</td>
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<tr>
<td>Testosterone</td>
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<tr>
<td>DHEA</td>
<td>↑</td>
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<tr>
<td>Estradiol</td>
<td>↑</td>
<td>↑↓</td>
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<tr>
<td>Growth hormone</td>
<td>↑</td>
<td>↑</td>
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<tr>
<td>IGF-I</td>
<td>↔↑</td>
<td>↔↑</td>
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</table>

Note: ↑ = increase  ↔ = no change  ↓ = decrease  ? = unknown
Aerobic vs. Resistance Exercise

There are large differences in the adaptations that take place at the muscular level when comparing aerobic to resistance training. For example, training performed at 80% of 1 RM is known to increase muscle mass (McDonagh and Davies, 1984), while endurance training is considered an inadequate stimulus for muscle hypertrophy (Henriksson and Reitman, 1976; Ingjer, 1979; Howard et al., 1985). Snow-Harter et al. (1992) confirmed this with an eight-month resistance and endurance training study involving young healthy females. Results indicated that weight training significantly increased muscle strength in all muscle groups trained, while no significant muscular changes were noted in either the running or control groups. In contrast, endurance performance improved only in the running group. Of interest, was that the lumbar BMD increased significantly in both exercise groups but did not differ significantly. Therefore, it is apparent that although resistance and endurance exercises may overload different physiological systems, some similar adaptations may occur. Future research is needed to investigate these differences and similarities.

To date, Jensen et al. (1991) and Tremblay (1994) are the only known authors to investigate same sample comparisons of hormonal responses to endurance versus resistance exercise. Jensen et al. (1991) compared the changes in testosterone concentration in males after both a single 90 minute bout of resistance exercise and a 90 minute bout of jogging at approximately 70% of their VO$_2$max. Results indicated that while testosterone levels had increased significantly immediately following both the resistance and endurance exercises (27% and 37% respectively), no significant differences were identified between the two types of exercises.
Tremblay (1994) compared anabolic steroid hormone responses between endurance and resistance trained males. Testing sessions included a resistance exercise session, a 40-minute run session at 50-55% of VO₂ max, and a resting session. Results indicated that levels of DHEAS, LH, total and free testosterone were higher in resistance trained individuals compared to endurance trained males during all three sessions. In contrast to Jensen et al. (1991), Tremblay (1994) reported that resistance exercise elicited a greater testosterone response than endurance exercise (13.3% vs. 9.4%). It should be noted that Tremblay (1994) controlled energy expenditure and exercise volume between the endurance and resistance sessions while Jensen et al. (1991) did not. These differences in controlled variables may explain the contradictory results. Unfortunately, no known studies on females have been conducted using the same subject pool when comparing the two types of exercise.

Nevill et al. (1996) studied the response of growth hormone to a 30 second treadmill sprint in both sprint and endurance-trained athletes. Results indicated that serum growth hormone was greater in the sprint-trained athletes than the endurance-trained athletes, but there were no significant differences between males and females. A possible contributing factor to this increased response in the sprint-trained athletes may be the fact that they were working at a higher peak and mean power level than the endurance-trained athletes. This may indicate that growth hormone is related to the absolute workload of an individual.
Summary and Rationale of the Study

Research indicates that aerobic exercise in females can elicit an increase in both testosterone and DHEA levels. Training status of the individual appears to be an important factor in the response of these hormones to the exercise. Growth hormone in females also appears to increase during aerobic exercise in females. Current research indicates that exercise intensity is positively correlated with this hormone’s response. IGF-I levels also rise during aerobic exercise in females. Recent research indicates that IGF-I increases may be independent of growth hormone responses. Estradiol also appears to rise during aerobic exercise in females.

While limited, current research indicates that testosterone levels do not increase during resistance exercise. To date, no known studies have investigated the response of DHEA to resistance exercise in young, adult females. Growth hormone response in females appears to be related to the anaerobic response the resistance exercise produces. Both IGF-I and estradiol responses to resistance exercise in females have been neglected to date.

Currently, there is little research examining the differences in steroid hormone responses to aerobic versus resistance exercise, particularly in females. The purpose of this study was to determine the anabolic hormone response in the same group of female subjects for each of an acute bout of resistance and aerobic exercise. By using females, this study was able to determine if exercise leads to an elevation of certain hormones that are known to decrease with age and whose decrease is associated with some of the health problems associated with aging.
Hormones play a role in numerous disorders affecting females. Current research indicates that hormones, specifically steroid hormones, decline with age. Today millions of dollars are spent on attempts to replenish these hormones through pharmaceuticals. However, these methods can become quite costly, and are not always safe. It would be beneficial to know if these hormones could be raised naturally through methods such as exercise. Unfortunately, no studies, prior to the present study, have been conducted that indicate which type of exercise (aerobic or resistance), if any, can achieve beneficial increases in these hormones.
CHAPTER 3
METHODOLOGY

Subject Recruitment and Screening

Sixteen healthy, premenopausal female subjects, between the ages of 19 and 47 volunteered for this study. Recruitment took place through local advertisement in Fredericton, New Brunswick. Potential subjects had to be currently cross-training (both endurance and resistance training) for a minimum of four months preceding this study to be included in the study. Cross-training was considered an average of two sessions each of aerobic and resistance exercise per week. All subjects were non-smokers.

All subjects completed a Physical Activity Readiness Questionnaire (PAR-Q) (Appendix B) and a medical/health questionnaire (Appendix B). Subjects that answered “yes” to one or more of the questions on the PAR-Q were referred to a physician where a Physical Activity Readiness Medical Examination (PARmed-X) (Appendix B) was completed by the physician before further participation in the study was permitted. Use of hormone replacement therapy (HRT) and oral contraceptives (OC) were identified on the medical questionnaire. Positive identification of these variables resulted in exclusion from the study.

Consent forms (Appendix B) outlining the experimental procedures and potential risks and benefits of the study were completed by each subject. Any questions posed by the subjects regarding the study were answered. Prior to any testing sessions resting heart rate and blood pressure were required to be below 100 beats per minute and 145/95.
mm Hg respectively as a safety precaution. This study received ethical approval from the University of New Brunswick Ethics Review Board (Appendix E).

**Experimental Procedures**

Subjects attended six separate laboratory sessions. These included: a medical screening and consent session, a fitness appraisal session, a familiarization session, an endurance training session, a resistance training session, and a resting control session (Figure 1).

<table>
<thead>
<tr>
<th>Session</th>
<th>1</th>
<th>2</th>
<th>3</th>
<th>4</th>
<th>5</th>
<th>6</th>
</tr>
</thead>
<tbody>
<tr>
<td>Screening Assessment Session</td>
<td>Fitness Assessment Session</td>
<td>Familiarization Session</td>
<td>Aerobic Session</td>
<td>Resistance Session</td>
<td>Control Session</td>
<td></td>
</tr>
</tbody>
</table>

Randomized Sessions
Luteal Phase (Days 14-21)
Same Time of Day

**Figure 1. Schematic diagram of experimental design.**

Pre-testing guidelines (Appendix C) were given to the subjects to be followed before each of their sessions. These guidelines included the avoidance of eating for at least two hours before the fitness appraisal and familiarization sessions. Before the exercise testing sessions subjects were requested to follow a standardized breakfast meal that included juice, milk, cereal, toast, and jam. In addition, the subjects were asked to
abstain from the consumption of alcohol or caffeine for at least six hours before each session and refrain from heavy exercise for twenty-four hours before testing.

**Fitness Appraisal Session**

Following the health and medical screening, subjects attended an information session in which the experimental procedures of the study were explained. The potential risks and benefits of the study were clarified and any questions posed by the subjects regarding the study were answered. During this session, subjects from whom informed consent was received completed the Canadian Physical Activity, Fitness and Lifestyle Appraisal (CPAFLA) (Canadian Society for Exercise Physiology, 1996a). The CPAFLA evaluated health-related fitness components such as body composition, aerobic fitness, flexibility, strength, muscular endurance, and leg power. The body composition component included the anthropometric measurements of height, weight, waist girth, and five skinfolds (triceps, biceps, subscapular, iliac crest, and medial calf). Aerobic fitness was calculated using the modified Canadian Aerobic Fitness Test. Trunk forward flexion was used to evaluate flexibility. Grip strength and vertical jump measures were used as indicators of muscular strength and power. Push-ups and partial curl-up were used to evaluate muscular endurance. In addition, percent body fat was calculated according to the three-site (triceps, suprailiac and abdomen) Jackson and Pollock method (Canadian Society for Exercise Physiology, 1996b).

**Familiarization Session**

During this orientation session, subjects were familiarized with both the endurance and resistance exercise protocols. Each subject had their 10 repetition maximum (10 RM) determined for each of the eight resistance exercises that were used
for this study. The procedure that was used to determine the subject’s 10 RM is explained by Wathen (1994). For each exercise, the subject performed one set of 10 repetitions using a light weight. Depending on the ease with which this was completed additional weight was added and another set of 10 repetitions was attempted. A one minute rest was given between subsequent sets. The previously described procedure was repeated until a weight was found that allowed 10 repetitions to be completed to exhaustion. This weight was used as the subject’s 10 RM for that exercise. This process was completed for each of the eight exercises performed in the study.

During this session, subjects were also familiarized with the cycling protocol. A submaximal Sjostrand cycling test was performed (three, four minute stages) following which, the resistance on the bike was increased by 0.5 kilopound every minute until exhaustion for each subject (Figure 2).

![Graph](image)

**Figure 2.** Illustration of an individual cycling test during the familiarization session.
This was performed to directly establish the maximal heart rate for each subject and assess aerobic fitness. A five minute active cool-down followed the test. An appropriate resistance was determined on the Monarch cycle ergometer to elicit 75% of the subject's previously determined maximum heart rate. In both the resistance and aerobic familiarization sessions, subjects were familiarized with the Borg Rating of Perceived Exertion Scale (Borg, 1982).

**Testing Sessions**

The testing sessions consisted of one endurance exercise session, one resistance exercise session, and one control session, and all took place during day 14-21 of each subject's menstrual cycle to control for hormonal variations due to the menstrual cycle phase. To control for diurnal variations in the hormones being measured, all testing sessions were performed at the same time of day. These three sessions were randomized, with a minimum of one day separating each session.

Prior to each of these three sessions an intravenous catheter was inserted into a forearm vein by a fully trained individual. Subjects had blood samples taken in a seated position in a climate controlled environment. Blood samples were taken 10 minutes before the beginning of each session, immediately following each session and a final sample was taken 30 minutes after the session had been completed. In total, 36 ml of blood was drawn per session. All samples were measured for serum levels of DHEA, testosterone, growth hormone, IGF-I and estradiol. The samples taken pre-session and immediately after the session were also analyzed for lactate. Blood samples were analyzed in duplicate for hematocrit using a Readacrit Centrifuge (Becton, Dickinson and Company) and the microhematocrit method.
Before each of the exercise sessions the subjects performed a standardized five minute warm-up on a cycle ergometer. This was followed by appropriate stretching exercises. The resistance session consisted of three sets of 10 repetitions of eight exercises using Universal equipment. The intensity equaled the previously determined 10 RM for each exercise and up to one minute rest was given between sets. The resistance exercises included: supine chest press, latissimus pull-down, leg press, biceps curl, triceps push-down, shoulder press, leg extension, and leg curl. The order in which these exercises were performed was standardized. The endurance session consisted of 40 minutes of pedaling on a Monarch cycle ergometer at 75% of the subject’s previously determined maximum heart rate. Pedal cadence remained between 50 and 80 revolutions per minute.

During these two exercise sessions, ratings of perceived exertion (RPE) and heart rate using the Polar XL were measured. Perceived exertion was measured using the Borg Rating of Perceived Exertion Scale (Borg, 1982). Subjects were asked to subjectively rate their intensity level based on this scale at five minute intervals for the aerobic session and at the end of each of the eight resistance exercises. As well, pre- and post-exercise blood pressure was measured as a safety precaution.

During the control session, subjects were asked to sit quietly for 35 minutes. Blood samples were taken at the same time intervals as with the exercise sessions.

**Biochemical Analyses**

Serum blood samples were analyzed in duplicate for the hormones DHEA, testosterone, growth hormone, IGF-1, and estradiol. Estradiol and DHEA levels were
measured by commercial radioimmunoassay procedures (Appendix D). Serum growth hormone and IGF-I levels were measured by commercial immunoradiometric assay procedures (Appendix D). Testosterone was measured by chemiluminescent procedures (Appendix D). Plasma lactate levels were also measured in duplicate before and immediately after each session using spectrophotometric procedures (Appendix D). Individual blood samples from all sessions were assayed together to eliminate inter-assay variation. High and low controls were included in each assay to evaluate inter-assay variability and the validity of the assay procedures. The acceptable coefficient of variation between duplicate samples was set at 15% for DHEA, estradiol, growth hormone and IGF-I assays. Acceptable variation between duplicate testosterone and lactate samples was set at 0.2 nmol/L and 0.5 mmol/L respectively. Samples were stored at -40 °C until analyzed.

**Statistical Analyses**

Descriptive data were expressed as means and standard deviations. Hormone levels were expressed as absolute change (post-exercise minus pre-exercise) for each individual. All post-exercise blood samples were corrected for changes in plasma volume according to the procedures explained by van Beaumont (1972).

Changes in hormone levels were assessed using an analysis of variance (ANOVA) with session and time as main effects. Area under the curve (AUC) for each hormone was also analyzed for each session using an ANOVA. In all instances, statistical significance was set at P < 0.05. *Post-hoc* comparisons used Tukey HSD analyses when necessary.
Average heart rate and changes in lactate throughout the three sessions were analyzed using an ANOVA to determine if the type of session performed had an effect on these intensity variables. Average RPE throughout the two exercise sessions was analyzed by an ANOVA to determine if differences existed.

Pearson correlation coefficients were performed between resting hormone levels and descriptive fitness variables. In addition, correlation coefficients were also performed to determine if changes in hormone levels were related to intensity variables for the two exercise sessions.
CHAPTER 4
RESULTS

Subject Characteristics

All subjects were classified as premenopausal based on self-reported regular menstrual cycles. Participants ranged in age from 19-47 years (33 ± 8, mean ± SD). Results from the CPAFLA are reported in Tables 2-4. Table 2 presents a summary of anthropometric values for all participants in the study. The mean and range of aerobic and musculoskeletal fitness scores are reported in Table 3. Results from the Physical Activity Participation and Lifestyle Questionnaires are reported in Table 4.

Table 2. Anthropometric scores (n = 16).

<table>
<thead>
<tr>
<th>Measurement</th>
<th>Overall Mean</th>
<th>Rating</th>
<th>Overall Range</th>
<th>Measurement</th>
<th>Rating</th>
</tr>
</thead>
<tbody>
<tr>
<td>Height (cm)</td>
<td>163.5</td>
<td>NA</td>
<td>157 – 176</td>
<td>NA</td>
<td></td>
</tr>
<tr>
<td>Weight (kg)</td>
<td>62.9</td>
<td>NA</td>
<td>49.9 – 78.6</td>
<td>NA</td>
<td></td>
</tr>
<tr>
<td>BMI (kg/m²)</td>
<td>23.4</td>
<td>H</td>
<td>20.2 – 27.1</td>
<td>UH – H</td>
<td></td>
</tr>
<tr>
<td>Waist Girth (cm)</td>
<td>73</td>
<td>H</td>
<td>64 – 82</td>
<td>H</td>
<td></td>
</tr>
<tr>
<td>SO5S (mm)</td>
<td>81.3</td>
<td>H</td>
<td>56.7 – 127.4</td>
<td>UH – H</td>
<td></td>
</tr>
<tr>
<td>SO2S (mm)</td>
<td>35.3</td>
<td>H</td>
<td>20.2 – 52.3</td>
<td>UH – H</td>
<td></td>
</tr>
<tr>
<td>Body Composition Score</td>
<td>12.1</td>
<td>E</td>
<td>4 – 16</td>
<td>F – E</td>
<td></td>
</tr>
<tr>
<td>Body Fat (%)</td>
<td>25.7</td>
<td>NA</td>
<td>18.8 – 33.3</td>
<td>NA</td>
<td></td>
</tr>
</tbody>
</table>

Note: BMI = body mass index, SO5S = sum of 5 skinfolds, SO2S = sum of 2 skinfolds, NA = not applicable, H = healthy, UH = unhealthy, F = fair, E = excellent.
Table 3. Aerobic and musculoskeletal fitness scores (n = 16).

<table>
<thead>
<tr>
<th></th>
<th>Overall Mean</th>
<th>Overall Range</th>
</tr>
</thead>
<tbody>
<tr>
<td></td>
<td>Measurement</td>
<td>Rating</td>
</tr>
<tr>
<td>Aerobic Fitness (score)</td>
<td>439</td>
<td>VG</td>
</tr>
<tr>
<td>Grip Strength (kg)</td>
<td>63</td>
<td>G</td>
</tr>
<tr>
<td>Push-Ups (#) a</td>
<td>39</td>
<td>E</td>
</tr>
<tr>
<td>Trunk Forward Flexion (cm)</td>
<td>34.5</td>
<td>G</td>
</tr>
<tr>
<td>Leg Power (kg-m/sec)</td>
<td>75.6</td>
<td>E</td>
</tr>
</tbody>
</table>

Note: E = excellent, VG = very good, G = good, NI = needs improvement.

a Data obtained from 15 of 16 participants.

Table 4. Physical Activity Participation and Lifestyle Questionnaires (n = 16).

<table>
<thead>
<tr>
<th></th>
<th>Overall Mean</th>
<th>Overall Range</th>
</tr>
</thead>
<tbody>
<tr>
<td></td>
<td>Score</td>
<td>Rating</td>
</tr>
<tr>
<td>Healthy Physical Activity</td>
<td>10</td>
<td>E</td>
</tr>
<tr>
<td>Participation Questionnaire</td>
<td></td>
<td></td>
</tr>
<tr>
<td>Fantastic Lifestyle</td>
<td>79</td>
<td>VG</td>
</tr>
<tr>
<td>Questionnaire</td>
<td></td>
<td></td>
</tr>
</tbody>
</table>

Note: E = excellent, VG = very good, G = good.

Resting Hormone Levels

Table 5 lists resting hormone levels based on the mean of the three pre-exercise samples for all participants in the study. Mean resting testosterone, estradiol, growth hormone and IGF-I were all within the clinical and expected reference ranges. The mean resting DHEA was 2 nmol/L above the level expected by the assay manufacturer (Diagnostic Systems Laboratories, Inc., Webster, Texas USA). Individual data indicated that six individuals had DHEA levels above those expected by the assay manufacturer. One individual had resting testosterone levels below the clinical reference range. Five
participants had low estradiol, and two had low growth hormone levels. All individuals had resting IGF-I levels within the expected range.

Pearson correlation coefficients were calculated between the resting hormone levels to determine if any relationships existed. Only DHEA and testosterone levels were significantly correlated \((r = 0.60, P < 0.05)\).
### Table 5. Average resting hormone levels based on three separate day samples.

<table>
<thead>
<tr>
<th>Subject</th>
<th>DHEA (nmol/L)</th>
<th>Testosterone (nmol/L)</th>
<th>Estradiol (pmol/L)</th>
<th>Growth Hormone (μg/L)</th>
<th>IGF-I (μg/L)</th>
</tr>
</thead>
<tbody>
<tr>
<td>1</td>
<td>79 ± 10</td>
<td>1.2 ± 0.2</td>
<td>93 ± 8</td>
<td>4.02 ± 3.35</td>
<td>397 ± 54</td>
</tr>
<tr>
<td>2</td>
<td>53 ± 30</td>
<td>1.3 ± 0.5</td>
<td>102 ± 10</td>
<td>0.17 ± 0.11</td>
<td>313 ± 16</td>
</tr>
<tr>
<td>3</td>
<td>30 ± 10</td>
<td>1.1 ± 0.2</td>
<td>107 ± 26</td>
<td>2.63 ± 1.14</td>
<td>430 ± 39</td>
</tr>
<tr>
<td>4</td>
<td>67 ± 11</td>
<td>1.8 ± 0.1</td>
<td>367 ± 263</td>
<td>0.15 ± 0.13</td>
<td>330 ± 2</td>
</tr>
<tr>
<td>5</td>
<td>33 ± 6</td>
<td>0.8 ± 0.2</td>
<td>211 ± 104</td>
<td>0.13 ± 0.02</td>
<td>323 ± 9</td>
</tr>
<tr>
<td>6</td>
<td>44 ± 8</td>
<td>1.7 ± 0.2</td>
<td>563 ± 475</td>
<td>1.66 ± 1.92</td>
<td>266 ± 45</td>
</tr>
<tr>
<td>7</td>
<td>36 ± 17</td>
<td>1.7 ± 0.2</td>
<td>165 ± 27</td>
<td>0.17 ± 0.06</td>
<td>369 ± 57</td>
</tr>
<tr>
<td>8</td>
<td>22 ± 10</td>
<td>0.9 ± 0.4</td>
<td>163 ± 38</td>
<td>0.06 ± 0.03</td>
<td>257 ± 22</td>
</tr>
<tr>
<td>9</td>
<td>71 ± 9</td>
<td>1.5 ± 0.3</td>
<td>218 ± 108</td>
<td>0.07 ± 0.01</td>
<td>206 ± 21</td>
</tr>
<tr>
<td>10</td>
<td>21 ± 5</td>
<td>1.4 ± 0.3</td>
<td>183 ± 77</td>
<td>1.10 ± 0.30</td>
<td>212 ± 50</td>
</tr>
<tr>
<td>11</td>
<td>24 ± 4</td>
<td>0.5 ± 0.1</td>
<td>275 ± 24</td>
<td>0.79 ± 0.85</td>
<td>258 ± 28</td>
</tr>
<tr>
<td>12</td>
<td>38 ± 6</td>
<td>1.3 ± 0.0</td>
<td>155 ± 48</td>
<td>0.73 ± 1.11</td>
<td>207 ± 24</td>
</tr>
<tr>
<td>13</td>
<td>34 ± 6</td>
<td>1.1 ± 0.1</td>
<td>264 ± 103</td>
<td>1.75 ± 2.58</td>
<td>112 ± 22</td>
</tr>
<tr>
<td>14</td>
<td>20 ± 8</td>
<td>0.9 ± 0.4</td>
<td>111 ± 159</td>
<td>1.68 ± 2.52</td>
<td>145 ± 18</td>
</tr>
<tr>
<td>15</td>
<td>11 ± 1</td>
<td>0.6 ± 0.2</td>
<td>155 ± 13</td>
<td>3.11 ± 2.60</td>
<td>188 ± 31</td>
</tr>
<tr>
<td>16</td>
<td>24 ± 16</td>
<td>0.4 ± 0.2</td>
<td>36 ± 4</td>
<td>1.02 ± 0.53</td>
<td>126 ± 5</td>
</tr>
<tr>
<td>Mean ± SD</td>
<td>38 ± 20</td>
<td>1.1 ± 0.4</td>
<td>201 ± 125</td>
<td>1.20 ± 1.20</td>
<td>259 ± 96</td>
</tr>
</tbody>
</table>

**Clinical Reference Range**

- 7-18
- 1.28 ± .35
- 184-881
- < 10
- NA

**Expected Values**

- 3 - 36
- 0.5 - 2.6
- 128 - 1373
- 0.10 - 7.02
- 96 - 575

*Note: NA = not available.*

a Tietz and Logan (1986).

b Supplied by Diagnostic Systems Laboratories, Inc. (2000).

c Supplied by Chiron Diagnostics Corporation (1996).
Resting Hormone Levels and CPAFLA Results

Pearson correlation coefficients were calculated between the five resting hormone levels and selected fitness scores. Fitness variables selected included percent body fat, the aerobic fitness score, push-ups, trunk forward flexion, and leg power. The only significant correlation was between aerobic fitness and resting IGF-I (r = 0.58, P < 0.05).

Exercise Effect on Anabolic Hormones

Figures 3-7 summarize the absolute changes (mean ± SE) in anabolic hormone levels for each of the three testing sessions across sample time. Figures 8-12 illustrate the individual changes for each hormone for each of the three sessions across sample time. All hormone responses were analyzed with a 3 x 3 analysis of variance (ANOVA) using session and sample time as main effects. Estradiol and growth hormone were the only hormones that revealed significant interactions. Non-significant interactions for DHEA, testosterone, and IGF-I were excluded from their respective models.

DHEA

Figure 3 illustrates the mean response of DHEA to the three sessions across sampling time. Analysis of variance revealed no significant interaction. Further analysis revealed no session effect for absolute changes in DHEA, however, a significant sample time effect (P < 0.001) was illustrated. Tukey's HSD post hoc analysis revealed that the last sample taken (recovery) was significantly lower than the pre-session sample (P < 0.003) and the post-session sample (P < 0.002).

Individual data for changes in DHEA for each of the three sessions across time are illustrated in Figure 8. Immediately after the aerobic exercise, 8 of the 16 participants
had increased their DHEA levels above baseline. In comparison, seven and four individuals showed increases at the same time point in the resistance and control session respectively.

**Testosterone**

Serum testosterone changes for the three sessions across sample time are shown in Figure 4. No significant interaction between session type and sample time effect was observed. A significant session effect \((P < 0.05)\) was observed for the absolute change in testosterone. Tukey’s HSD post hoc comparison revealed that the mean absolute change in testosterone after the aerobic session was significantly higher \((P < 0.04)\) than the corresponding change in the control session. No significant changes were observed between the aerobic and resistance session or between the resistance and control session. In addition, no overall sample time effect was observed.

Individual data for each session across time are reported for testosterone in Figure 9. Immediately after the aerobic exercise, eight individuals raised their testosterone levels above baseline. Six and five individuals raised their testosterone levels above baseline at the same corresponding time in the resistance and control sessions respectively.

**Estradiol**

Changes in serum estradiol for the three sessions across sample time are illustrated in Figure 5. A significant \((P < 0.008)\) session x sample interaction was noted. It is apparent from Figure 5 that the change in estradiol that was reported at the second sampling time depends on which session has taken place. A significant session effect was observed for estradiol \((P < 0.004)\). Tukey’s HSD post hoc analysis revealed
significant higher (P < 0.003) mean changes in estradiol levels after the aerobic session than the control session. No significant differences were observed between the aerobic and resistance session or between the resistance and control session. A significant sample time effect was observed (P < 0.01) for estradiol. Tukey’s HSD post hoc analysis indicated that the post-session estradiol levels were significantly elevated over the pre-session levels (P < 0.02) and the recovery levels (P < 0.05). No significant differences were observed between the pre-session and recovery samples.

Individual changes in estradiol for each session, across sample time are reported in Figure 10. Immediately after the aerobic exercise, 15 of the 16 participants had increased estradiol levels above baseline values. Twelve and seven participants showed increases at the same time point in the resistance and control session respectively.

**Growth Hormone**

The changes observed in serum growth hormone for each of the three sessions across sample time are shown in Figure 6. A significant (P < 0.002) session x sample interaction was reported. Similar to estradiol, it is apparent from Figure 6 that the change that occurs to growth hormone at the second sample point depends on which session has occurred. A significant session effect (P < 0.001) was observed for mean absolute changes in growth hormone. Tukey’s HSD post hoc analysis indicated significant increases for both the aerobic and resistance session over the control session. Mean absolute changes in growth hormone after the resistance session were significantly higher (P < 0.001) than the corresponding control values. Mean absolute changes in growth hormone after the aerobic session were significantly higher (P < 0.02) than the
corresponding control session. No significant differences were observed between the aerobic and resistance sessions.

A significant sample time effect (P < 0.001) was observed for growth hormone. Tukey's HSD post hoc analysis indicated that the sample taken immediately after the session was significantly higher (P < 0.001) than both the pre-session and the 30 minute recovery sample. There was no significant difference between the pre-sample and recovery sample.

Individual data for each session across time are reported for growth hormone in Figure 11. Immediately after the resistance exercise, 13 of the 16 participants had increased their growth hormone levels above baseline. Twelve and nine participants showed increases at the same time point in the aerobic and control session respectively.

**IGF-I**

No significant interaction or main effects were noted for changes in IGF-I using session and sample time as independent variables. Mean changes in IGF-I for the three sessions across sample time are illustrated in Figure 7. Individual data for each session across time are reported for IGF-I in Figure 12. Immediately after the aerobic exercise, nine participants showed increases from baseline for this hormone. Six individuals after the resistance exercise showed increases, while eight showed a similar trend after the corresponding time in the control session.

Area under the curve (AUC) analyses confirmed the above session effects for estradiol and growth hormone. AUC analyses revealed no session effects for the remaining three hormones.
Figure 3. Mean (± SE) absolute change in serum DHEA levels during the aerobic (top), resistance (middle), and control (bottom) session.
Figure 4. Mean (± SE) absolute change in serum testosterone levels during the aerobic (top), resistance (middle), and control (bottom) session. * P < 0.04 compared with the corresponding mean control session level.
Figure 5. Mean (± SE) absolute change in serum estradiol levels during the aerobic (top), resistance (middle), and control (bottom) session. * P < 0.003 compared with corresponding mean control session level.
Figure 6. Mean (± SE) absolute change in serum growth hormone levels during the aerobic (top), resistance (middle), and control (bottom) session. * $P < 0.02$ compared with the corresponding mean control session level. ** $P < 0.001$ compared with the corresponding mean control session level.
Figure 7. Mean (± SE) absolute change in serum IGF-I levels during the aerobic (top), resistance (middle), and control (bottom) session.
Figure 8. Individual absolute change in serum DHEA levels during the aerobic (top), resistance (middle), and the control (bottom) session.
Figure 9. Individual absolute change in serum testosterone levels during the aerobic (top), resistance (middle), and control (bottom) session.
Figure 10. Individual absolute change in serum estradiol levels during the aerobic (top), resistance (middle), and the control (bottom) session.
Figure 11. Individual absolute change in serum growth hormone levels during the aerobic (top), resistance (middle), and control (bottom) session.
Figure 12. Individual absolute change in serum IGF-I levels during the aerobic (top), resistance (middle), and control (bottom) session.
The order in which the three sessions took place was randomized. No order effect was observed for the absolute change in DHEA, testosterone, estradiol or growth hormone. However, a significant order effect (P < 0.01) was observed for IGF-I. Tukey’s HSD post hoc analysis revealed that the last session the participants attended resulted in a significantly lower (P < 0.03) mean absolute change in IGF-I when compared to the first and second sessions attended. In addition, there were no significant differences in initial levels of each of the hormones across the three session types.

**Intensity Variables**

**Lactate**

Individual lactate levels for the aerobic, resistance, and control session are reported in Table 6. A one-way ANOVA was performed to determine if there was a session effect for the change in lactate from pre-session to post-session. Results indicated a significant session effect (P < 0.001). Tukey’s HSD post hoc analysis revealed that mean absolute changes in lactate levels were significantly higher (P < 0.001) immediately after the resistance session compared to both the aerobic and control sessions. There were no significant differences between the aerobic and control sessions when comparing changes in lactate values between pre- and post-session.
Table 6. Lactate levels for the aerobic, resistance, and control sessions.

<table>
<thead>
<tr>
<th>Subject</th>
<th>Aerobic Lactate Levels (mmol/L)</th>
<th>Resistance Lactate Levels (mmol/L)</th>
<th>Control Lactate Levels (mmol/L)</th>
</tr>
</thead>
<tbody>
<tr>
<td></td>
<td>Pre</td>
<td>Post</td>
<td>Pre</td>
</tr>
<tr>
<td>1</td>
<td>1.81</td>
<td>3.35</td>
<td>2.01</td>
</tr>
<tr>
<td>2</td>
<td>1.29</td>
<td>1.08</td>
<td>0.99</td>
</tr>
<tr>
<td>3</td>
<td>1.31</td>
<td>1.96</td>
<td>0.89</td>
</tr>
<tr>
<td>4</td>
<td>2.04</td>
<td>1.58</td>
<td>1.75</td>
</tr>
<tr>
<td>5</td>
<td>1.50</td>
<td>0.96</td>
<td>1.33</td>
</tr>
<tr>
<td>6</td>
<td>1.27</td>
<td>1.31</td>
<td>2.23</td>
</tr>
<tr>
<td>7</td>
<td>1.21</td>
<td>1.17</td>
<td>1.77</td>
</tr>
<tr>
<td>8</td>
<td>1.27</td>
<td>1.11</td>
<td>1.27</td>
</tr>
<tr>
<td>9</td>
<td>2.57</td>
<td>1.29</td>
<td>1.77</td>
</tr>
<tr>
<td>10</td>
<td>1.49</td>
<td>1.22</td>
<td>1.44</td>
</tr>
<tr>
<td>11</td>
<td>1.31</td>
<td>1.35</td>
<td>0.97</td>
</tr>
<tr>
<td>12</td>
<td>1.14</td>
<td>1.78</td>
<td>1.58</td>
</tr>
<tr>
<td>13</td>
<td>1.07</td>
<td>1.66</td>
<td>1.02</td>
</tr>
<tr>
<td>14</td>
<td>0.95</td>
<td>1.89</td>
<td>1.48</td>
</tr>
<tr>
<td>15</td>
<td>0.66</td>
<td>1.56</td>
<td>1.10</td>
</tr>
<tr>
<td>16</td>
<td>1.10</td>
<td>2.13</td>
<td>1.32</td>
</tr>
</tbody>
</table>

Mean ± SD: 1.37 ± 1.59 ± 1.43 ± 5.57 ± 1.39 ± 1.43 ± 1.36 ± 0.49

* Significantly different (p < 0.001) from the corresponding aerobic and control mean.

Heart Rate and RPE

Individual summary heart rate and RPE data for the two exercise sessions are reported in Table 7. A one-way ANOVA indicated a significant (P < 0.001) session effect for average heart rate during the sessions. Tukey's HSD post hoc analysis revealed that average heart rate was significantly higher (P < 0.001) during the aerobic session.
than the resistance session. However, average heart rate was significantly higher 
(P < 0.001) during the resistance session compared to the control session.

Ratings of perceived exertion were also compared between the two exercise 
sessions by ANOVA. Results indicated the average RPE rating was significantly higher 
(P < 0.001) during the resistance session than the aerobic session.

Table 7. Average heart rate and RPE for aerobic and resistance sessions.

<table>
<thead>
<tr>
<th>Subject</th>
<th>Average Heart Rate (Range)</th>
<th>Average RPE (Range)</th>
</tr>
</thead>
<tbody>
<tr>
<td></td>
<td>Aerobic</td>
<td>Resistance</td>
</tr>
<tr>
<td>1</td>
<td>142 (134-153)</td>
<td>117 (70-161)</td>
</tr>
<tr>
<td>2</td>
<td>136 (118-149)</td>
<td>96 (67-135)</td>
</tr>
<tr>
<td>3</td>
<td>140 (135-152)</td>
<td>94 (72-144)</td>
</tr>
<tr>
<td>4</td>
<td>136 (123-147)</td>
<td>128 (109-159)</td>
</tr>
<tr>
<td>5</td>
<td>133 (122-152)</td>
<td>NA</td>
</tr>
<tr>
<td>6</td>
<td>144 (131-158)</td>
<td>150 (112-187)</td>
</tr>
<tr>
<td>7</td>
<td>139 (134-149)</td>
<td>115 (86-149)</td>
</tr>
<tr>
<td>8</td>
<td>139 (128-152)</td>
<td>121 (75-159)</td>
</tr>
<tr>
<td>9</td>
<td>144 (132-154)</td>
<td>113 (86-148)</td>
</tr>
<tr>
<td>10</td>
<td>131 (127-141)</td>
<td>92 (62-134)</td>
</tr>
<tr>
<td>11</td>
<td>148 (145-157)</td>
<td>119 (86-161)</td>
</tr>
<tr>
<td>12</td>
<td>134 (131-146)</td>
<td>96 (70-146)</td>
</tr>
<tr>
<td>13</td>
<td>136 (130-149)</td>
<td>88 (61-132)</td>
</tr>
<tr>
<td>14</td>
<td>140 (125-155)</td>
<td>123 (89-157)</td>
</tr>
<tr>
<td>15</td>
<td>124 (119-136)</td>
<td>107 (78-143)</td>
</tr>
<tr>
<td>16</td>
<td>135 (124-148)</td>
<td>116 (92-158)</td>
</tr>
<tr>
<td>Mean ± SD</td>
<td>138 ± 6</td>
<td>112 ± 17*</td>
</tr>
</tbody>
</table>

Note: RPE = rating of perceived exertion, NA = data not available. 
* Significantly different (p < 0.001) from corresponding average aerobic value.
Intensity and Changes in Hormone Levels

Pearson correlation coefficients were calculated to determine if there were relationships between intensity variables and changes in hormone levels for both the aerobic session (Table 8) and the resistance session (Table 9). Changes in DHEA and corresponding changes in lactate levels immediately after the aerobic session were positively correlated ($r = 0.64$, $P < 0.01$). Changes in growth hormone were negatively associated with average RPE ($r = -0.61$, $P < 0.05$).

<table>
<thead>
<tr>
<th></th>
<th>$\Delta$ Lactate</th>
<th>Average Heart Rate</th>
<th>Average RPE</th>
</tr>
</thead>
<tbody>
<tr>
<td>$\Delta$ DHEA</td>
<td>0.64**</td>
<td>0.03</td>
<td>0.15</td>
</tr>
<tr>
<td>$\Delta$ Testosterone</td>
<td>0.08</td>
<td>0.39</td>
<td>-0.38</td>
</tr>
<tr>
<td>$\Delta$ Estradiol</td>
<td>-0.42</td>
<td>-0.24</td>
<td>0.10</td>
</tr>
<tr>
<td>$\Delta$ Growth Hormone</td>
<td>0.09</td>
<td>0.24</td>
<td>-0.61*</td>
</tr>
<tr>
<td>$\Delta$ IGF-I</td>
<td>-0.28</td>
<td>0.12</td>
<td>0.04</td>
</tr>
</tbody>
</table>

Note: $\Delta =$ change from baseline to immediately after exercise session.

** $P < 0.01$  * $P < 0.05$.

A similar relationship as that reported for the aerobic session was noted between changes in DHEA and lactate levels in the resistance session ($r = 0.50$, $P < 0.05$). Changes in testosterone were positively correlated with changes in lactate ($r = 0.58$, $P < 0.05$). Both the changes in estradiol and growth hormone were positively correlated with average heart rate during the resistance session ($r = 0.58$, $P < 0.05$ and $r = 0.51$, $P < 0.05$). The absolute change in growth hormone in the resistance session was positively correlated with the average RPE reported ($r = 0.52$, $P < 0.05$).
Table 9. Pearson correlation coefficients between intensity variables and changes in hormone levels after the resistance session.

<table>
<thead>
<tr>
<th></th>
<th>Δ Lactate</th>
<th>Average Heart Rate</th>
<th>Average RPE</th>
</tr>
</thead>
<tbody>
<tr>
<td>Δ DHEA</td>
<td>0.50*</td>
<td>0.43</td>
<td>0.18</td>
</tr>
<tr>
<td>Δ Testosterone</td>
<td>0.58*</td>
<td>0.39</td>
<td>0.41</td>
</tr>
<tr>
<td>Δ Estradiol</td>
<td>0.24</td>
<td>0.58*</td>
<td>0.09</td>
</tr>
<tr>
<td>Δ Growth Hormone</td>
<td>0.09</td>
<td>0.51*</td>
<td>0.52*</td>
</tr>
<tr>
<td>Δ IGF-I</td>
<td>0.30</td>
<td>-0.17</td>
<td>-0.20</td>
</tr>
</tbody>
</table>

Note: Δ = change from baseline to immediately after exercise session.
* P < 0.05

Pearson correlation coefficients were calculated between the changes in the five anabolic hormones for each type of exercise. No significant findings were observed with the exception of the positive relationship between changes in testosterone and changes in DHEA in the resistance session (r = 0.68, P < 0.005).

**Hormone Assays**

Table 10 reports the acceptable and average intra-assay coefficient of variation for each hormone and lactate. Also, reported are the inter-assay coefficient of variation for both high and low controls, and the sensitivity of each assay procedure.
### Table 10. Intra- and inter-assay coefficient of variations and sensitivity of assays.

<table>
<thead>
<tr>
<th></th>
<th>Acceptable Intra-assay Coefficient of Variation</th>
<th>Average Intra-assay Coefficient of Variation</th>
<th>Average Inter-assay Coefficient of Variation</th>
<th>Sensitivity of Assay</th>
</tr>
</thead>
<tbody>
<tr>
<td>DHEA</td>
<td>≤ 15%</td>
<td>6%</td>
<td>18%</td>
<td>5%</td>
</tr>
<tr>
<td>Estradiol</td>
<td>≤ 15%</td>
<td>5%</td>
<td>10%</td>
<td>16%</td>
</tr>
<tr>
<td>Growth Hormone</td>
<td>≤ 15% for all samples above 0.10 μg/L</td>
<td>6%</td>
<td>22%</td>
<td>30%</td>
</tr>
<tr>
<td>IGF-1</td>
<td>≤ 10%</td>
<td>3%</td>
<td>8%</td>
<td>6%</td>
</tr>
<tr>
<td>Testosterone</td>
<td>≤ 0.2 nmol/L difference between duplicate samples</td>
<td>7%</td>
<td>5%</td>
<td>2%</td>
</tr>
<tr>
<td>Lactate</td>
<td>≤ 0.5 mmol/L difference between duplicate samples</td>
<td>7%</td>
<td>28%</td>
<td>34%</td>
</tr>
</tbody>
</table>

Note: Sensitivity of the assay indicates the minimum detection limit.

* With the exception of one sample with a coefficient of variation of 21%.
CHAPTER 5
DISCUSSION

Main Findings of the Study

The main purpose of the present study was to compare the anabolic hormone response to resistance versus aerobic exercise in healthy, premenopausal women. As was originally hypothesized, no significant differences in anabolic hormone responses were observed between the two types of exercise. There were however, significant differences between each type of exercise when compared to a control session. Testosterone, estradiol, and growth hormone were significantly elevated after the aerobic exercise compared to the control session. In contrast, only growth hormone showed significant increases after the resistance exercise compared to the control session.

Subject Fitness Levels

Subjects included in this study were healthy, premenopausal women who had been actively cross-training (endurance and resistance exercise) at least twice a week for the past 4 months. All individuals completed physical activity records to make sure minimum requirements were met. All subjects were considered eumenorrheic based on self-report. The Canadian Physical Activity, Fitness, and Lifestyle Appraisal (CPAFLA) (CSEP, 1996a) was completed to obtain physical fitness scores on all individuals. The CPAFLA provides scores and ratings on different fitness components including, physical activity and lifestyle assessment, body composition, aerobic fitness, and musculoskeletal
fitness. Scores and ratings are based on normative values obtained from the Canadian population of similar age and gender. Mean results indicate that individuals in this study had “healthy” anthropometric measurements (Table 2), and “very good” aerobic fitness scores (Table 3). Muscular endurance, which was evaluated by push-ups and partial curl-ups, was given a mean rating of “excellent”. Both grip strength and leg power are identified as indicators of muscular strength and were rated as “good” and “excellent” respectively (Table 3). Flexibility was rated as ‘good’ (Table 3). Self-reported physical activity participation and lifestyle assessment were rated as “excellent” and “very good” respectively (Table 4). Therefore, it is apparent from these ratings that the individuals selected for this study were both healthy and physically fit based on Canadian normative values.

**Resting Hormone Levels in Subjects**

Resting hormone levels were determined based on the average between three separate morning samples. Mean testosterone, estradiol, growth hormone, and IGF-I levels were all within the clinical and expected reference ranges (Table 5). Mean resting serum levels of DHEA were 2 nmol/L higher than expected. Individual data indicated that a total of 6 of the 16 subjects had resting DHEA levels higher than expected. A number of explanations may account for this. Research indicates a well defined circadian rhythm for DHEA with the acrophase occurring at approximately 0800 h in both sexes (Lejeune-Lenain et al., 1987; Liu et al., 1990). Therefore, because testing sessions took place during the morning, DHEA levels were likely at their highest. Another important factor is the large variation of this hormone. Our data is in agreement with Sulcova et al.
who also reported large variations in this hormone. In addition, because the adrenals are the main source of DHEA (Shangold et al., 1984) psychological stress can not be ruled out as playing a role in elevating this hormone. Training status may have also affected resting DHEA levels. It was noted that Subject #1 who had the highest resting DHEA levels was also a competitive long-distance runner in the midst of training. Although all individuals in the study were considered physically fit and met minimum training criteria, individual training variation did occur. Conflicting results have been reported as to the influence of training on resting DHEA levels. Filaire et al. (1998) has indicated that an intensive training program of both aerobic and resistance exercise in female athletes has proven to significantly increase resting saliva DHEA levels. In contrast, Tsai et al. (1991) found no significant increases in DHEA after 9 months of endurance training in elite women endurance athletes. Finally, the specificity of the DHEA assay kits must be considered. Although both high and low controls were in the appropriate ranges, and the percent cross-reactivity with various compounds was minimal according to the manufacturer, specificity can not be ruled out as a cause for the high DHEA levels.

The only significant inter-hormone correlation was between resting DHEA and testosterone levels. A similar relationship has been reported by others (Lejeune-Lenain et al., 1987). This association is not surprising based on the knowledge that 65% of plasma testosterone is derived from the prehormones DHEA and androstenedione in women (Kirschner and Bardin, 1972).
Resting Hormones and Fitness Results

There does not appear to be any clear relationship between resting hormone levels and fitness performance scores in the present study. These results support research by Fahey et al. (1976) that found no significant relationships between resting serum testosterone levels and static strength or body composition in college women. The only significant correlation observed in the present study was a weak, positive relationship between aerobic fitness scores and resting IGF-1 levels. These results were in agreement with Kelly et al. (1990) and Poehlman and Copeland (1990) who both found significant relationships between aerobic capacity measures and resting IGF-1 levels. To date, the mechanisms behind this relationship, if any, have not been explained.

It is speculated that training activity (e.g., speed, distance, and duration) may be a better predictor of resting hormone levels than fitness scores. For example, Schwartz et al. (1981) found a significant correlation between resting LH and the number of miles run per week in amenorrheic runners.

Exercise Effect on DHEA

There were no significant differences between the DHEA response to aerobic vs resistance exercise. In addition, neither the DHEA response to aerobic nor resistance exercise showed significant changes over the control group.

Aerobic Exercise

The lack of DHEA change after aerobic exercise in the present study is in conflict with results from other studies (Baker et al., 1982; Cumming and Rebar, 1983; Johnson et al., 1997). However, because of the few studies conducted on this hormone, it is difficult
to compare studies due to methodological differences. Baker et al. (1982) observed significant increases in DHEA levels after a 10-mile race in premenopausal women. Exercise intensity and duration were not reported, and therefore, it is difficult to compare these results to our own. It is assumed nonetheless, that both the intensity and duration of this activity were different than that of the present study.

Pearson correlation coefficients from the present study indicate that changes in DHEA are positively related to changes in lactate. Therefore, it appears that the DHEA response to aerobic exercise may be related to intensity. This notion is supported by previous work by Cumming and Rebar (1983) that indicated that vigorous cycling to exhaustion caused a significant increase in DHEA levels. In addition, Johnson et al. (1997) reported that 30 minutes of treadmill running at 90% of the subject's heart rate maximum could increase DHEA levels in postmenopausal women. These results indicate that perhaps there is an exercise intensity threshold that needs to be reached before DHEA levels can increase and that this threshold may not have been reached in the present study. Another factor that should be considered is that the subjects in the Johnson et al. (1997) study were postmenopausal women. Based on the knowledge that resting DHEA levels decrease with age (Labrie et al., 1997a) these women would have had lower resting DHEA levels than the subjects in the present study. Therefore, perhaps another factor that needs to be considered is the initial resting levels of DHEA.

Resistance Exercise

To date, this has been the only study involving the measurement of DHEA in response to a resistance session in premenopausal women. The lack of DHEA response after the resistance session is however, in agreement with the findings of Copeland
(1998) who reported that DHEA levels did not change after varying resistance exercise volumes in postmenopausal women. Results from the Pearson correlation coefficients in the present study indicate that changes in lactate are positively associated with changes in DHEA in the resistance session. Therefore, it is possible that our subjects were not working at a high enough intensity during this session to elicit a DHEA response.

**DHEA Circadian Rhythm**

A significant sample time effect was observed for DHEA. Previous research indicates a circadian rhythm for DHEA with the acrophase occurring at approximately 0800 h in both sexes (Lejeune-Lenain et al., 1987; Liu et al., 1990) and the nadir occurring at 2200 h in males (Lejeune-Lenain et al., 1987). Due to the fact that the acrophase occurred during our testing time it could account for the higher than expected resting DHEA levels. ACTH which is known to stimulate both DHEA and cortisol has been reported to be highest in the morning (Reilly et al., 2000) and is presumably the reason why both of these hormones have marked circadian rhythms. However, while it is well documented that ACTH plays a role in stimulating DHEA, the differences between cortisol and DHEA during a variety of physiological conditions has led researchers to believe that other regulators are involved in adrenal androgen secretion (Hinson and Raven, 1999). To date, these factors still remain unclear but should be further investigated to help understand the effects of exercise on DHEA.

**Exercise Effect on Testosterone**

No significant difference was observed between the testosterone response to aerobic and resistance exercise. However, while testosterone levels rose significantly
over control levels after the aerobic session, no differences were observed between the resistance and control session.

**Aerobic Exercise**

Significant increases in testosterone after aerobic exercise have been supported by others (Baker et al., 1982; Bonen and Keizer, 1987; Shangold et al., 1981). Baker et al., (1982) observed elevated testosterone levels in healthy, premenopausal runners after a 10-mile run. While the present study used females in their luteal phase, Baker et al. (1982) used a majority of subjects in their follicular phase (5 follicular phase, 1 luteal phase). This indicates that increases in testosterone may be independent of menstrual cycle phase. The reasoning behind this is likely due to the relatively low and unvarying contribution of the ovary in secreting testosterone throughout the menstrual cycle (Bonen and Keizer, 1987). Bonen and Keizer (1987) also observed rises in testosterone levels in young women runners of varying gynecological status. Testosterone levels increased linearly throughout a marathon race, reaching their highest levels at the end of the race. Bonen and Keizer (1987) found a much more pronounced increase in testosterone than the present study. This may be due to the longer duration and differing intensity of the marathon run compared to our aerobic session. This explanation is supported by Wilkerson et al. (1980) who indicated that exercise intensity affects plasma testosterone.

Results from this study indicate that there were no significant differences between initial hormone levels between sessions. Therefore, it appears that there was no pre-exercise increase in testosterone in anticipation of exercise as has been observed by others (Cumming and Rebar, 1983; Wilkerson et al., 1980).
A factor that must be considered when discussing increases in testosterone levels during aerobic exercise is the changes in MCR due to the decrease in hepatic blood flow that is associated with exercise (Rowell, 1974). A major route for steroid degradation is through hepatic clearance (Webb et al., 1984). Therefore, a decrease in hepatic blood flow could account for apparent increases in steroid hormone concentrations (Webb et al., 1984).

**Resistance Exercise**

No significant increases in testosterone were observed between the resistance and control session. A lack of change in testosterone after an acute bout of resistance exercise has been supported by other researchers (Fahey et al., 1976; Kraemer et al., 1991; Kraemer et al., 1993; Kraemer et al., 1995a).

Using a similar protocol as our own (8 exercises, 3 sets of 10 RM, and 1 minute rest) Kraemer et al. (1991) did not observe increases in testosterone values in females. These results indicate that other endogenous anabolic responses other than circulating testosterone levels may be of importance for strength gains that are associated with resistance exercise in females. The levels of the anabolic hormone androstenedione are approximately 10-fold higher than testosterone in females (Kraemer et al., 1991). Research indicates that resistance exercise is a strong enough stimulus to elevate levels of this hormone in females (Kraemer et al., 1995a). However, testosterone is still considered a much more potent anabolic hormone than androstenedione (Kraemer et al., 1991). Similar to the DHEA response, Pearson correlation coefficients indicate that changes in this hormone are related to changes in lactate.
Although not significant, a declining trend in testosterone was observed during the control session. Lejeune-Lenain et al. (1987) have reported a circadian rhythm in testosterone in males. Testosterone levels show a general decline throughout the morning with an acrophase occurring at approximately 0500 h in men (Lejeune-Lenain et al., 1987).

**Exercise Effect on Estradiol**

No significant differences were observed between the serum estradiol response to aerobic and resistance exercise. However, estradiol levels after the aerobic exercise rose significantly over control levels. In contrast, no significant changes were observed between the resistance and control session estradiol levels.

**Aerobic Exercise**

The present study showed an increase in serum estradiol during the luteal phase of the menstrual cycle. These findings are similar to Jurkowski et al. (1978) who found significant increases in estradiol during the luteal phase of females cycling for 20 minutes at 63% of their aerobic capacity. It is apparent that both exercise intensity and menstrual cycle phase play an important role in the response of estradiol to aerobic exercise. Jurkowski et al. (1978) found that females in their follicular phase had to cycle at a higher intensity to achieve the same increases in estradiol as those females in their luteal phase. It is believed that the increases in estradiol observed with aerobic exercise are ovarian in origin. This is based on the fact that the majority of estradiol is produced from the ovary, with minimal amounts being produced by peripheral conversion (Bonen and Keizer, 1987).
Duration of exercise may also play a critical role in estradiol response. In contrast to the 40 minutes of aerobic exercise in the present study, Montagnani et al. (1992) observed a decrease in estradiol with females running on a treadmill for 2 hours.

It is believed that the MCR plays a vital role in the reported values of estradiol and other steroid hormones. At greater exercise intensities, the MCR decreases as blood flow is diverted from the splanchnic area to working muscles and consequently steroid hormones become elevated (Keizer et al., 1980). Keizer et al., (1980) reported that the MCR of estradiol decreased by 36% by the end of a 10-minute cycling protocol at 70% VO₂max. However, Bonen et al. (1991) has indicated that increases in estradiol due to exercise are a consequence of increased secretion rather than a decrease in the hepatic clearance.

**Resistance Exercise**

The results from the present study indicate that although a definite increasing trend was observed in serum estradiol after the resistance session, these increases were not significantly different from the control session. The results from this study contradict the findings of Kraemer et al. (1995a) who observed an increase in estradiol after low-volume resistance exercise in females during both follicular and luteal phases. It is important to note that these subjects were untrained and therefore, increases in estradiol may have occurred because of initial training status.

The present results were comparable to those of Walberg-Rankin et al. (1992) who, similar to the present study, observed a non-significant increase in estradiol after resistance exercise. In this study all females were already currently involved in a resistance program so the initial adaptation period was not a factor. Subjects from the
same study who were consuming a hypocaloric diet did achieve significant increases in estradiol. It is apparent that more research is needed to study the effects of resistance exercise on estradiol levels. Results from these preliminary studies indicate that both training and nutritional status may play key roles.

**Exercise Effect on Growth Hormone**

Similar to the findings with the other hormones, there was no significant difference between the serum growth hormone response between the aerobic and resistance session. However, unlike the other hormones, serum growth hormone levels rose significantly in both the aerobic and resistance session compared to the control session.

**Aerobic Exercise**

Results from this study indicate that 40 minutes of cycling at 75% of an individual's maximal heart rate can elevate serum growth hormone levels significantly. Exercise intensity may be the main determinant of growth hormone response during aerobic activity. It was recently reported that a linear dose-response relationship existed between exercise intensity and the growth hormone response in men (Pritzlaff et al., 1999). This relationship involved escalating growth hormone responses across the full range (sublactate to supralactate threshold) of exercise intensities (Pritzlaff et al., 1999).

It is still not known what specific mechanisms are involved in the stimulation of growth hormone. Chwalbinska-Moneta et al. (1996) found that in endurance athletes cycling to exhaustion, growth hormone shows an exponential increase, similar to lactate, epinephrine and norepinephrine. In addition, Kozlowski et al. (1983) reported significant
correlations between growth hormone and lactate during constant rate exercise. From these results it would be apparent that the combination of decreasing blood pH from lactate accumulation and the increment of catecholamine concentrations may be considered as signals for growth hormone increase. However, results from the present study indicate that growth hormone levels can increase despite no significant increases in lactate.

A recent study by Weltman et al. (2000) examined the relationship between sympathetic outflow and the subsequent growth hormone response to acute exercise in men. Results indicated that norepinephrine and epinephrine always preceded peak growth hormone response. This response was observed over varying levels of intensities, including exercise producing the same lactate response as our own aerobic session. Although catecholamine response was not measured in the present study it may have played a role in the growth hormone response.

Although Pearson correlation coefficients from the present study indicate a significant negative relationship between changes in growth hormone and average reported RPE, these results must be viewed with caution. The aerobic exercise used in this study was steady-state exercise. Workload on the cycle ergometer was adjusted to maintain 75% of the subject's maximal heart rate. Therefore, changes in intensity variables during this session were minimal and consequently the importance of these relationships should not be overstated.

Individual data for growth hormone response to aerobic exercise indicated that 12 of the 16 individuals showed absolute increases in the hormone between resting levels and levels immediately after the cycling protocol. An interesting finding was that the
remaining four individuals that showed absolute decreases in this hormone tended to have higher initial growth hormone levels than the other individuals. All four of these individuals were among the top 6 individuals with the highest resting growth hormone levels.

Similar findings were observed with the resistance session. Only 3 of the 16 individuals showed absolute decreases in growth hormone between the initial and immediately post resistance exercise sample. These 3 individuals were within the top 5 individuals with the highest initial levels. Therefore, growth hormone auto-negative feedback may play a role in blunting the growth hormone response to exercise.

**Resistance Exercise**

Significant absolute increases were observed in growth hormone after resistance exercise. These results are in agreement with those observed by Kraemer et al. (1991) who using a similar protocol (10 RM, 1 minute rest) observed significant increases in growth hormone after the exercise session and these levels remained elevated up to 15 minutes into recovery. An interesting note was that in the Kraemer et al. (1991) study when 5 RM with 3 minutes rest in between sets was used no significant increases in this hormone were observed. Therefore, the degree of anaerobic involvement appears to be a determinant in the degree of growth hormone response. In agreement with this finding, Mulligan et al. (1996) reported that a multiple set heavy-resistance exercise protocol could produce significantly higher growth hormone responses over a single set protocol in females.

Pearson correlation coefficients indicate that average heart rate and self-reported values of RPE were significantly correlated with changes in growth hormone during the
resistance session. This also supports the assumption that the degree of intensity is related to changes in this hormone.

Individual dietary intake did not play a role in growth hormone response during this study. Previous literature has indicated that growth hormone response to exercise can be increased by fat intake (Jenkins et al., 1999). Therefore, all individuals in the present study were required to eat a standardized light meal before each testing session to control for this variable. In addition, food logs were kept for the 24 hours preceding each session.

**Exercise Effect on IGF-I**

There was no significant difference between the IGF-I response to aerobic vs resistance exercise. In addition, neither the IGF-I response to aerobic nor resistance exercise showed significant changes over the control group.

**Aerobic Exercise**

In contrast to our findings, Cappon et al. (1994) reported that brief (10 minutes), high intensity (~ 72% VO2max) cycling could significantly increase IGF-I levels in adults. IGF-I peaked immediately after the 10 minute protocol and remained elevated for 20 minutes into recovery. This discrepancy may be because the subjects in the Cappon et al. (1994) study exercised at a different intensity and the duration was shorter than the present study. The results of Cappon et al. (1994) indicate that the response of IGF-I to exercise can be very quick (within 10 minutes), therefore, the longer sampling period in the present study (40 minutes) may not have detected any initial elevations. In addition, Cappon et al. (1994) had their subjects cycle above the lactate threshold, while the
subjects in the present study did not have lactate levels significantly different from the lactate levels in the control session.

The findings of Cappon et al. (1994) are consistent with those of Bang et al. (1990) and Hormum et al. (1997) who observed significant increases in IGF-I after 10 minutes of cycling. Each subject in the Bang et al. (1990) study cycled for 30 minutes at approximately 83% of his or her maximum heart rate. Again, it is evident that these subjects were exercising at a higher intensity than our own, and this was supported by higher lactate values than the present study. The important result from this study indicates that IGF-I levels increased significantly by 10 minutes into the exercise, but then returned to resting levels within the exercise period. Therefore, it is quite possible that any initial increases in IGF-I that occurred at the onset of exercise in the present study were missed. By only drawing blood samples at the completion of the exercise period, temporary increases in IGF-I may have been missed. However, if transient increases in IGF-I did occur, it is difficult to interpret the biological significance of such a short event.

In agreement with the present study, Bang et al. (1990) and Cappon et al. (1994) found no relationship between IGF-I and growth hormone changes. It is believed that changes in IGF-I due to increases in growth hormone were not achieved during the recovery hours in the present study. This assumption was based on the results of Cappon et al. (1994) who reported that increases in growth hormone had no post-exercise effect on serum IGF-I levels over the subsequent 21 hours of recovery.

While the present study may have been of too long a duration to detect brief increases in IGF-I, it may also have been too short to observe decreases in this hormone previously observed during prolonged aerobic exercise. Suikkari et al. (1989), studying
the effects of prolonged endurance exercise in males, reported that IGF-1 concentrations decreased after 7.5 hours of a 75-km cross-country ski race. The significance of IGFs in aerobic exercise may be related to their insulin-like effects on both adipose and muscle tissues (Froesch et al., 1985). This includes lipid and protein synthesis, the stimulation of glucose transport, and the inhibition of lipolysis (Froesch et al., 1985). During prolonged exercise, declining insulin levels are a major determinant of increased lipolysis (Galbo, 1983). Therefore, if IGF-1 exhibits insulin-like effects then an inhibition of this hormone may be advantageous for the enhancement of lipolysis during prolonged aerobic exercise (Suikkari et al., 1989).

Although no increases in circulating IGF-1 were noted, the fact that local muscular production of IGF-1 existed cannot be discounted. Recent work by Eliakim et al. (1997) indicated that five days of endurance training caused different responses between circulating IGF-1 and muscle protein IGF-1. In this instance, circulating IGF-1 showed no response while muscle IGF-1 protein increased significantly. Another possible mechanism that may account for the absence of detectable increases in circulating IGF-1 may be due to muscular uptake of this hormone. Therefore, increases in secretion of IGF-1 may occur but are masked by the increased muscular uptake.

It is clear that more research is needed to investigate the role of IGF binding proteins (IGFBPs) during exercise. Over 95% of circulating IGF-1 is bound to IGFBP-3 (Muller et al., 1999). Schwarz et al. (1996) reported that 10 minutes of high-intensity cycling caused both IGF-I and IGFBP-3 to increase. However, the interesting finding in this study was that IGFBP-3 proteolytic activity also increased over baseline. If
proteolysis of IGFBP-3 could be increased during exercise then this could lead to increased bioavailability of IGF-I.

**Resistance Exercise**

The results from the present study indicate that a resistance exercise session does not produce significant changes in serum IGF-I levels when compared to a control session. Kraemer et al. (1993) investigating premenopausal women under different heavy resistance protocols found similar results to our own. Comparable to our own data, individuals in the Kraemer et al. (1993) study worked at intensities high enough to elicit significant increases in lactate. However, also similar to the present study, both IGF-I and testosterone levels remained unchanged after the resistance session while growth hormone levels increased.

The lack of a consistent IGF-I response to resistance exercise was validated by the findings of Kraemer et al. (1991). In this instance, a protocol similar to our own did produce significant increases in IGF-I immediately after the resistance session. In contrast, in the same study, a protocol using a lighter workload produced elevated IGF-I levels only after 60 minutes of recovery. These inconsistent findings, were evident in our own data. Despite tight control our subjects showed large inter-individual variation in their IGF-I response to resistance exercise.

Kraemer et al. (1993) suggested these inconsistent findings may be due to a number of different physiological factors. These include concentrating mechanisms in the blood (e.g., differing metabolic clearance rates), increases in transporter proteins, or the release of IGF-I from other nonhepatic cells (e.g., fat, muscle and connective tissue cells) due to tissue disruption from exercise (Kraemer et al., 1993).
The present study was not able to determine if local muscular production of IGF-I was influenced by resistance exercise.

**Conclusion**

The present study confirmed the original hypothesis that there would be no differences in anabolic hormone response between aerobic and resistance exercise in premenopausal females. To date, this has been the only study using same sample comparisons of hormonal responses to endurance versus resistance exercise in females. These results are in agreement with Jensen et al. (1991) that indicated that the anabolic hormone response of testosterone is similar between the two types of exercise in males.

While a difference in anabolic hormone response between the two types of exercise was not observed in this study, each individual exercise did show unique responses when compared to the control session. Aerobic exercise was responsible for testosterone, estradiol, and growth hormone responses above those observed in the control session. In contrast, growth hormone was the only hormone that was significantly elevated after the resistance exercise compared to the control session.

There are a number of different variables that influence hormonal responses in females. These factors include menstrual cycle phase and status, fitness levels, nutritional status, stress levels, and substance use (e.g., medication or supplementation) (Tremblay et al., 1995). The previously discussed study attempted to control for these variables. Nonetheless, large inter-subject variations were observed in hormonal response.
**Recommendations for Future Work**

1. More information is needed on the biological significance of exercise-induced hormone changes. If hormone concentrations can be increased with exercise, research is needed to specify what specific health related benefits are associated with these changes. For example, if increases in anabolic hormones do occur in premenopausal women, then more research is needed to identify if these changes have an impact on current and/or future bone density.

2. A better understanding of the factors that influence the growth hormone-IGF-I axis is needed such as somatostatin and the IGFBPs. Research is needed to clarify the concentration changes and proteolysis activity of the binding proteins as a consequence of exercise.

3. More research is needed concerning the local production of IGF-I. Specifically, research is needed to determine what events are occurring at the muscular level with respect to IGF-I production and muscular uptake of circulating IGF-I.

4. A better understanding of the intensity threshold and the duration of exercise needed before significant hormonal changes occur is needed.

5. More information is needed on the individual variability in resting hormone levels in premenopausal women. Additional research is needed to determine if exercise (aerobic and/or resistance) training has an effect on resting anabolic hormone levels in females. Subsequently, it needs to be determined if these differing resting levels have consequences on the hormonal response to exercise.
REFERENCES


APPENDIX A

ABBREVIATIONS
APPENDIX A

Abbreviations

ACTH – adrenocorticotropic hormone
ANOVA – analysis of variance
AUC – area under the curve
BMD – bone mineral density
CFLRI – Canadian Fitness and Lifestyle Research Institute
CHD – coronary heart disease
CPAFLA – Canadian Physical Activity, Fitness and Lifestyle Appraisal
CSEP – Canadian Society for Exercise Physiology
DHEA – dehydroepiandrosterone
DHEAS – dehydroepiandrosterone sulfate
DNA – deoxyribonucleic acid
E₁ – estrone
E₂ – estradiol
E₃ – estriol
FDA – Food and Drug Administration
FSH – follicle stimulating hormone
GHRH – growth hormone-releasing hormone
GnRH – gonadotrophin-releasing hormone
GRF – growth releasing factor
HRT – hormone replacement therapy
IGF-I – insulin-like growth factor I
IGF-II – insulin-like growth factor II
IGFBPs – insulin-like growth factor binding proteins
IGFs – insulin-like growth factors
LDL – low density lipoproteins
LH – luteinizing hormone
MCR – metabolic clearance rate
mRNA – messenger ribonucleic acid
NADPH – nicotinamide adenine dinucleotide phosphate
OC – oral contraceptive
PARmed-X – Physical Activity Readiness Medical Examination
PAR-Q – Physical Activity Readiness Questionnaire
P₄ – progesterone
RM – repetition maximum
RNA – ribonucleic acid
RPE – rating of perceived exertion
SHBG – sex hormone-binding globulin
SRIF – somatostatin releasing factor
APPENDIX B

SUBJECT FORMS

Information and Consent Form

PAR-Q

PARmed-X

Medical and Health Screening Form

Resistance Training Log

Aerobic Training Log
INFORMATION AND CONSENT FORM

A study comparing the effect of age on the endocrine response to endurance and resistance exercise in women.

Name: ___________________________ Age: ___________

The purpose of this study is to compare the effect of age on hormone levels after an acute bout of endurance and resistance exercise. As an experimental subject you will be required to make a total of five visits to the UNB Exercise Physiology Lab. The first session will involve a standardized, health-related fitness appraisal to measure both aerobic and musculoskeletal fitness and will last approximately one hour. The second session will be a familiarization session where you will be introduced to the cycling and weightlifting exercises. Also, during this session the approximate weight that you are able to lift for 10 repetitions will be determined for 8 different weightlifting exercises. The final three sessions will be the testing sessions. These will include an endurance exercise session, a resistance exercise session and a resting session. Each of the sessions will last approximately 2 hours. The order in which you perform each of these sessions will be chosen randomly. Three blood samples will be taken through a small tube that will be placed in an arm vein by a fully trained and certified individual. Before each of these sessions resting blood pressure and heart rate will be measured.

The testing will involve the following procedures:
- heart rate and blood pressure measurement
- heart rate monitoring
- Canadian Physical Activity, Fitness and Lifestyle Appraisal
- familiarization cycling and weight training session
- cycling, weight training and resting sessions with three blood samples (before, after, and one hour after)

A short description of each procedure will follow.

HEART RATE AND BLOOD PRESSURE MEASUREMENT
Both heart rate and blood pressure measurements are taken as a precaution before exercise testing. These measurements assist in the evaluation of an individual’s health and fitness status.

HEART RATE MONITORING
Heart rate will be measured during the fitness appraisal and through each of the exercise sessions using a Polar heart rate monitor.

CANADIAN PHYSICAL ACTIVITY, FITNESS AND LIFESTYLE APPRAISAL
The CPAFLA is a health-related fitness appraisal which includes measurements of standing height, weight, girths, and skinfolds, and tests of grip strength, push-ups, trunk forward flexion, curl-ups, and vertical jump.
SJOSTRAND CYCLE TEST
The Sjostrand is a submaximal cycle test used to estimate an individual’s VO2max. The test involves three, four minute stages of varying intensities. Corresponding heart rates for each stage are used to predict your VO2max. Subsequently, the resistance on the bike will be increased every minute until you reach exhaustion. This will be done to establish your maximal heart rate.

FAMILIARIZATION WEIGHT TRAINING AND CYCLING SESSION
During this session you will be familiarized with eight resistance exercises. The eight exercises will be performed using Universal equipment and they will include the supine chest press, latissimus pull down, leg press, biceps curl, triceps pushdown, shoulder press, leg extension and leg curl. You will be instructed on proper technique and your 10 repetition maximum (10 RM) will be established. To determine your 10 RM, you will perform one set of 10 repetitions with a light weight. A two to four minute recovery period will then be given. Depending on the difficulty of the first set, additional weight will be added and another set will be completed. This process will be continued until a weight is found that will allow only 10 repetitions to be completed.

At this time you will also be familiarized with the stationary cycle and the protocol that will be used. Your subjective rating of perceived effort will be evaluated at your target heart rate.

TESTING SESSIONS
The testing sessions include an endurance and resistance exercise session as well as a control session where no exercise will be performed. Blood samples will be collected through a small tube that is designed to remove small amounts of blood. The tubing allows easier sampling and requires only ONE initial “needle prick”. A fully certified individual will place the tube in an arm vein. Three blood samples will be taken for each session. The baseline sample will be taken 10 minutes before you exercise, the second sample will be taken immediately post-exercise and a final sample will be taken 30 minutes post-exercise.

Prior to the endurance and weight training sessions you will be given a 10 minute warm up including light aerobic activity and stretching. Then you will complete a session of either eight weight lifting exercises or 40 minutes of cycling. The resistance exercise session will consist of 3 sets of each exercise with one minute of rest given between each set. The aerobic session will consist of 40 minutes of cycling at 70% of your age-predicted maximum heart rate. The order in which these sessions are performed will be randomized and all sessions will be performed at the same time of day.

POTENTIAL RISKS
The fitness appraisal, and endurance and resistance training sessions may involve some degree of risk. You may experience periods of transient lightheadedness, fainting, elevated blood pressure, leg cramps, or nausea. You may also experience muscular
soreness and fatigue for a day or two following each session. These are normal sensations associated with physical exertion.

The measurement of blood variables using the above method is only performed by a fully trained individual, and extreme care is taken to ensure that all blood sampling is done under sterile conditions. You may feel slight discomfort when the small tube is inserted into your arm. While there is the potential risk of complications such as infection at the insertion site no such complications as a result of blood sampling have occurred in this lab.

BENEFITS
The Canadian Physical Activity, Fitness and Lifestyle Appraisal will be performed by a certified individual and will provide you with valuable information regarding your health and fitness relative to the Canadian population. The body composition analysis will provide you with an estimate of lean body mass and percentage of body fat. In addition, you will be contributing to research which may be important to females suffering from age-related problems. Also, a free fitness re-appraisal will be offered to you within one year of the study.

CONSENT
I acknowledge that:
- based on my knowledge and the advice of my physician, there is no medical reason why I cannot perform the testing as described
- I have completed a PAR-Q and none of my responses were "yes"
- I have reported any medication that I am taking
- the testers have answered all my questions
- I understand the potential risks and benefits of this experiment
- my results will be provided to me upon completion of all testing, if I desire
- all of my results, both medical and performance, will be kept strictly confidential and if used for publication, my identity will remain anonymous
- I am aware that blood samples may be taken to the Dr. Everett Chalmers Hospital to be analyzed
- I recognize that my involvement is voluntary and I may discontinue the testing at any time
- I agree to inform the researcher if my health condition changes or if I begin taking any medication while I am involved with this study.

Signature: _______________________________ Date: ______________________

Witness: _______________________________ Date: ______________________
PAR - Q & YOU

(A Questionnaire for People Aged 15 to 69)

Regular physical activity is fun and healthy, and increasingly more people are starting to become more active every day. Being more active is very safe for most people. However, some people should check with their doctor before they start becoming much more physically active.

If you are planning to become much more physically active than you are now, start by answering the seven questions in the box below. If you are between the ages of 15 and 69 the PAR-Q will tell you if you should check with your doctor before you start. If you are over 69 years of age and you are not used to being very active, check with your doctor.

Common sense is your best guide when you answer these questions. Please read the questions carefully and answer each one honestly.

1. Has your doctor ever said that you have a heart condition and that you should only do physical activity recommended by a doctor?

2. Do you feel pain in your chest when you do physical activity?

3. In the past month, have you had chest pain when you were not doing physical activity?

4. Do you lose your balance because of dizziness or do you ever lose consciousness?

5. Do you have a bone or joint problem that could be made worse by a change in your physical activity?

6. Is your doctor currently prescribing drugs (for example, water pills) for your blood pressure or heart condition?

7. Do you know of any other reason why you should not do physical activity?

YES to one or more questions:

If you answer "YES" to any one or more of questions 1 to 7, talk with your doctor by phone or in person before you start becoming much more physically active. You may be able to do any activity you want — as long as you start slowly and build up gradually. Or you may need to restrict your activities to those which are safe for you. Talk with your doctor about the kinds of activities you wish to participate in and follow his advice.

Find out which community programs are safe and helpful for you.

NO to all questions:

If you answered "NO" honestly to all PAR-Q questions, you can be reasonably sure that you can:

- Start becoming much more physically active — begin slowly and build up gradually. This is the safest and easiest way to go.

- Take a "health appraisal" — it is an excellent way to determine your basic fitness so that you can plan the best way for you to live actively. It is also strongly recommended that you have your blood pressure evaluated. If your reading is over 144/94, talk with your doctor before you start becoming much more physically active.

Please note: If your health changes so that you then answer "YES" to any of the above questions tell your fitness or health professional. Ask whether you should change your physical activity plan.

You are encouraged to copy the PAR-Q but only if you use the entire form.

NOTE: The PAR-Q is being given to a person before he or she participates in a physical activity program or a fitness appraisal. This section may be used for legal or administrative purposes.

I have read, understood, and completed this questionnaire. Any questions I had were answered to my full satisfaction.

NAME

SIGNATURE __________________________________________________________________________

DATE _____________________________________________________________________

SIGNATURE OF PARENT ___________________________________________________________________

DATE _____________________________________________________________________

GUARDIAN for participants under the age of majority

DATE _____________________________________________________________________

WITNESS _____________________________

continued on other side
We know that being physically active can provide numerous benefits for our health. Not only does active living help to maintain a healthy heart and stroke risk factors, but it also helps to increase our quality of life and overall well-being. The key is to find activities that you enjoy and that fit into your daily routine. This might mean incorporating physical activity into your daily commute or using downtime for exercise. Whatever you choose to do, make sure it is something that you enjoy and that you can sustain long-term.

Physical activity can also be a great way to socialize and connect with others. Joining a group fitness class or participating in a sports team can be a fun and fulfilling way to stay active. It can also be a great way to meet new people and make new friends.

Incorporating physical activity into your daily routine can be as simple as taking a walk during your lunch break, doing some stretching exercises at work, or even taking the stairs instead of the elevator. Even small changes can make a big difference in your overall health.

Physical activity is especially important during pregnancy. Regular exercise can help to improve your mood, reduce the risk of gestational diabetes, and improve labor and delivery outcomes. It is important to consult with your healthcare provider before starting any new exercise program during pregnancy.

In summary, physical activity is an essential part of living a healthy lifestyle. By incorporating physical activity into your daily routine, you can improve your health, increase your energy levels, and boost your mood. So, let's get moving and enjoy the many benefits that physical activity can bring to our lives.
The PARmed-X is a physical activity-specific checklist to be used by a physician with patients who have had positive responses to the Physical Activity Readiness Questionnaire (PAR-Q). In addition, the Conveyance/Referral Form in the PARmed-X can be used to convey clearance for physical activity participation, or to make a referral to a medically-supervised exercise program.

Regular physical activity is fun and healthy, and increasingly more people are starting to become more active every day. Being more active is very safe for most people. The PAR-Q by itself provides adequate screening for the majority of people. However, some individuals may require a medical evaluation and specific advice (exercise prescription) due to one or more positive responses to the PAR-Q.

Following the participant's evaluation by a physician, a physical activity plan should be devised in consultation with a physical activity professional (CSEP: Professional Fitness and Lifestyle Consultant). To assist in this, the following instructions are provided:

PAGE 1: Sections A, B, C, and D should be completed by the participant BEFORE the examination by the physician. The bottom section is to be completed by the examining physician.

PAGES 2 & 3: A checklist of medical conditions requiring special consideration and management.

PAGE 4: Physical Activity & Lifestyle Advice for people who do not require specific instructions or prescribed exercise.

Physical Activity Readiness Conveyance/Referral Form - an optional tear-off tab for the physician to convey clearance for physical activity participation, or to make a referral to a medically-supervised exercise program.

<table>
<thead>
<tr>
<th>Section</th>
<th>Description</th>
</tr>
</thead>
<tbody>
<tr>
<td>A</td>
<td>PERSONAL INFORMATION:</td>
</tr>
<tr>
<td></td>
<td>NAME</td>
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<tr>
<td></td>
<td>ADDRESS</td>
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<tr>
<td></td>
<td>TELEPHONE</td>
</tr>
<tr>
<td></td>
<td>BIRTHDATE</td>
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<tr>
<td></td>
<td>MEDICAL No.</td>
</tr>
<tr>
<td>B</td>
<td>PAR-Q: Please indicate the PAR-Q questions to which you answered YES</td>
</tr>
<tr>
<td></td>
<td>Q 1 Heart condition</td>
</tr>
<tr>
<td></td>
<td>Q 2 Chest pain during activity</td>
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<tr>
<td></td>
<td>Q 3 Chest pain at rest</td>
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<td></td>
<td>Q 4 Loss of balance, dizziness</td>
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<td></td>
<td>Q 5 Bone or joint problem</td>
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<td></td>
<td>Q 6 Blood pressure or heart drugs</td>
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<tr>
<td>C</td>
<td>RISK FACTORS FOR CARDIOVASCULAR DISEASE: Check all that apply</td>
</tr>
<tr>
<td></td>
<td>Q Less than 30 minutes of moderate physical activity most days of the week</td>
</tr>
<tr>
<td></td>
<td>Q Currently smoker (tobacco smoking 1 or more times per week)</td>
</tr>
<tr>
<td></td>
<td>Q High blood pressure reported by physician after repeated measurements</td>
</tr>
<tr>
<td></td>
<td>Q High cholesterol level reported by physician</td>
</tr>
<tr>
<td>D</td>
<td>PHYSICAL ACTIVITY INTENTIONS:</td>
</tr>
<tr>
<td></td>
<td>Q Excessive accumulation of fat around waist</td>
</tr>
<tr>
<td></td>
<td>Q Family history of heart disease</td>
</tr>
<tr>
<td></td>
<td>Please note: Many of these risk factors are modifiable. Please refer to page 4 and discuss with your physician</td>
</tr>
<tr>
<td>E</td>
<td>Physical Exam:</td>
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<td></td>
<td>Ht</td>
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<td></td>
<td>Conditions limiting physical activity:</td>
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<tr>
<td></td>
<td>Q Cardiovascular</td>
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<td></td>
<td>Q Respiratory</td>
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<tr>
<td></td>
<td>Q Musculoskeletal</td>
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<tr>
<td></td>
<td>Q Abdominal</td>
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<tr>
<td>F</td>
<td>Tests required:</td>
</tr>
<tr>
<td></td>
<td>Q Resting ECG</td>
</tr>
<tr>
<td></td>
<td>Q Exercise Stress Test</td>
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<tr>
<td></td>
<td>Q X-Ray</td>
</tr>
<tr>
<td></td>
<td>Q Blood</td>
</tr>
<tr>
<td></td>
<td>Q Urinalysis</td>
</tr>
</tbody>
</table>

Physical Activity Readiness Conveyance/Referral: Based upon a current review of health status, I recommend:

| Q No physical activity |
| Q Only a medically-supervised exercise program until further medical clearance |

Further information:

| Q Unrestricted physical activity - start slowly and build up gradually |
| Q Limited physical activity |

Supported by Canadian Society of Exercise Physiology (CSEP-Professionals in Fitness and Lifestyle Consultancy).
Following is a checklist of medical conditions for which a degree of precaution and/or special advice should be considered for those who answered "YES" to one or more questions on the PAR-Q, and people over the age of 69. Conditions are grouped by system. Three categories of precautions are provided. Comments under Advice are general, since details and alternatives require clinical judgement in each individual instance.

<table>
<thead>
<tr>
<th>Absolute Contraindications</th>
<th>Relative Contraindications</th>
<th>Special Prescriptive Conditions</th>
</tr>
</thead>
<tbody>
<tr>
<td>Permanent restriction or temporary restriction until condition is treated, stable, and/or past acute phase.</td>
<td>Highly variable. Value of exercise testing and/or program may exceed risk. Activity may be restricted.</td>
<td>Individualized prescriptive advice generally inappropriate.</td>
</tr>
<tr>
<td>Cardiovascular</td>
<td></td>
<td></td>
</tr>
<tr>
<td>1. aortic aneurysm (assessing)</td>
<td>1. aortic stenosis (moderate)</td>
<td>1. aortic stenosis—mild angina patients and other manifestations of coronary insufficiency (e.g. post-acute infarct)</td>
</tr>
<tr>
<td>2. aortic stenosis (severe)</td>
<td>2. subacute stenosis (severe)</td>
<td>2. chronic heart disease</td>
</tr>
<tr>
<td>3. congestive heart failure</td>
<td>3. marked cardiac enlargement</td>
<td>3. conduction disturbances</td>
</tr>
<tr>
<td>4. crescendo angina</td>
<td>4. supraventricular dysrhythmias (uncontrolled or high rate)</td>
<td>4. complete AV block</td>
</tr>
<tr>
<td>5. myocardial infarction (acute)</td>
<td>5. ventricular ectopic activity (irrespective of frequency)</td>
<td>5. left BBB</td>
</tr>
<tr>
<td>6. myocardial infarction (active or recent)</td>
<td>6. ventricular aneurysm</td>
<td>6. Wolff-Parkinson-White syndrome</td>
</tr>
<tr>
<td>7. pulmonary or systemic embolism—acute</td>
<td>7. hypertension—untreated or uncontrolled severe (systemic or pulmonary)</td>
<td>7. syncope (intermittent or fixed)</td>
</tr>
<tr>
<td>8. thrombophlebitis</td>
<td>8. hyperthyroidism</td>
<td>8. gastroschisis—corrected</td>
</tr>
<tr>
<td>9. &quot;proximal&quot; tachycardia and other dangerous dysrhythmias (e.g. &quot;high-rate ventricular activity&quot;)</td>
<td>9. compensated congestive heart failure</td>
<td>9. rapid atrial fibrillation</td>
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<td>10. intermittant claudication</td>
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<tr>
<td>Infections</td>
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<tr>
<td>1. acute infectious disease regardless of etiology</td>
<td>2. subacute, chronic, recurrent infectious disease (e.g. malaria, others)</td>
<td>1. chronic infections</td>
</tr>
<tr>
<td>3. HIV</td>
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<tr>
<td>Metabolic</td>
<td></td>
<td></td>
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<tr>
<td>1. uncontrolled metabolic disorder (diabetes mellitus, thyrotoxicosis, myxedema)</td>
<td>2. renal, hepatic and other metabolic insufficiency</td>
<td>2. obesity</td>
</tr>
<tr>
<td>3. single kidney</td>
<td></td>
<td>3. dietary moderation, and initial light exercises with slow progression (walking, swimming, cycling)</td>
</tr>
<tr>
<td>Pregnancy</td>
<td></td>
<td></td>
</tr>
<tr>
<td>1. comaplicated pregnancy (e.g. toxemia, premature, incompentent cervix, etc.)</td>
<td>2. advanced pregnancy (late 3rd trimester)</td>
<td>3. refer to the &quot;PARmed-X for PREGNANCY&quot;</td>
</tr>
</tbody>
</table>

ADVICE

- Clinical exercise test may be warranted in selected cases, for specific determination of functional capacity and limitations and precautions (if any).
- Slow progression of exercise to levels based on test performance and individual tolerance.
- Consider individual need for initial conditioning program under medical supervision (indirect or direct).

References:


The PAR-Q and PARmed-X were developed by the British Columbia Ministry of Health. They have been reviewed by an Expert Advisory Committee assembled by the Canadian Society for Exercise Physiology and the Fitness Program, Health Canada (1995).

You are encouraged to copy the PARmed-X, but only if you use the entire form.

Disponible en français sous le titre "Évaluation médicale de l'aptitude à l'activité physique (X-AAP)"
| Physical Activity Readiness
<table>
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<tbody>
<tr>
<td>Medical Examination (revised 1995)</td>
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</tbody>
</table>

## Special Prescriptive Conditions

<table>
<thead>
<tr>
<th>Lung</th>
<th>ADVICE</th>
</tr>
</thead>
<tbody>
<tr>
<td>1. Chronic pulmonary disorders</td>
<td>Special relaxation and breathing exercises</td>
</tr>
<tr>
<td>2. Obstructive lung disease</td>
<td>Breath control during endurance exercises to tolerance; avoid polluted air</td>
</tr>
<tr>
<td>3. Asthma</td>
<td>Avoid hyperventilation during exercise; avoid extremely cold conditions, warm up adequately, utilize appropriate medication</td>
</tr>
</tbody>
</table>

<table>
<thead>
<tr>
<th>Musculoskeletal</th>
<th></th>
</tr>
</thead>
<tbody>
<tr>
<td>1. Low back conditions (pathological, functional)</td>
<td>Avoid or minimize exercise that precipitates or exacerbates (e.g., forced extreme flexion, extension, and violent twisting, correct posture, proper back exercises)</td>
</tr>
<tr>
<td>2. Arthritis—acute infective, traumatic (gout)</td>
<td>Treatment, plus judicious blend of rest, stretching, and gentle movement</td>
</tr>
<tr>
<td>3. Arthritis—subacute</td>
<td>Progressive increase of active exercise therapy</td>
</tr>
<tr>
<td>4. Arthritis—chronic osteoarthritis and above conditions</td>
<td>Maintenance of mobility and strength; non-weight-bearing exercises to minimize joint trauma (e.g., cycling, aquatic activity, etc.)</td>
</tr>
<tr>
<td>5. Orthopedic</td>
<td>Highly variable and individualized</td>
</tr>
<tr>
<td>6. Scoliosis</td>
<td>Physiotherapy exercises, postural, and therapeutic exercises</td>
</tr>
<tr>
<td>7. Osteoporosis or low bone density</td>
<td>Avoid exercise with high risk for fracture such as push-ups, curl-ups, vertical jump and trunk forward flexion</td>
</tr>
</tbody>
</table>

<table>
<thead>
<tr>
<th>CNS</th>
<th></th>
</tr>
</thead>
<tbody>
<tr>
<td>1. Convulsive disorder not completely controlled by medication</td>
<td>Monitor or avoid exercise in hazardous environments and/or exercising alone (e.g., swimming, mountaineering, etc.)</td>
</tr>
<tr>
<td>2. Recent concussion</td>
<td>Thorough examination; history of two concussions, review for discontinuation of contact sport if three concussions, depending on duration of unconsciousness, retrograde amnesia, persistent headaches, other objective evidence of cerebral damage</td>
</tr>
</tbody>
</table>

<table>
<thead>
<tr>
<th>Blood</th>
<th></th>
</tr>
</thead>
<tbody>
<tr>
<td>1. Jaundice—severe (10 Grit)</td>
<td>Control preferred exercise as tolerated</td>
</tr>
<tr>
<td>2. Exercise insufficiencies</td>
<td></td>
</tr>
</tbody>
</table>

<table>
<thead>
<tr>
<th>Medications</th>
<th></th>
</tr>
</thead>
<tbody>
<tr>
<td>1. Antihypertensives</td>
<td></td>
</tr>
<tr>
<td>2. Diuretics</td>
<td></td>
</tr>
<tr>
<td>3. Antidiabetic</td>
<td></td>
</tr>
<tr>
<td>4. Anticoagulants</td>
<td></td>
</tr>
<tr>
<td>5. Anticonvulsants</td>
<td></td>
</tr>
<tr>
<td>6. Antihistamines</td>
<td></td>
</tr>
<tr>
<td>7. Antispasmodics</td>
<td></td>
</tr>
<tr>
<td>8. Antimalarials</td>
<td></td>
</tr>
<tr>
<td>9. Corticosteroids</td>
<td></td>
</tr>
<tr>
<td>10. Adrenergics</td>
<td></td>
</tr>
</tbody>
</table>

<table>
<thead>
<tr>
<th>Other</th>
<th></th>
</tr>
</thead>
<tbody>
<tr>
<td>1. Post-exercise syndrome</td>
<td>Moderate program</td>
</tr>
<tr>
<td>2. Heat intolerance</td>
<td>Prolong cool-down with light activities; avoid exercise in extreme heat</td>
</tr>
<tr>
<td>3. Temporary minor illness</td>
<td>Postpone until recovered</td>
</tr>
<tr>
<td>4. Cancer</td>
<td>If potential metastases; test by cycle ergometry, consider non-weight-bearing exercises at lower end of prescriptive range (40-65% of heart rate reserve); depending on condition and recent treatment: radiation chemotherapy, monitor hemoglobin and lymphocyte counts; add dynamic/fitting exercise to strengthen muscles using machines rather than weights</td>
</tr>
</tbody>
</table>

| Refer to special publications for elaboration as required |

The following companion forms are available by contacting the Canadian Society for Exercise Physiology (address below): The Physical Activity Readiness Questionnaire (PAR-Q) - a questionnaire for people aged 15-69 to complete before becoming much more physically active The Physical Activity Readiness Medical Examination for Pregnancy (PARmed-X for PREGNANCY) - to be used by physicians with pregnant patients who wish to become more physically active

To order multiple printed copies of the PARmed-X and/or any of the companion forms for a nominal charge, please contact the Canadian Society for Exercise Physiology 185 Somerset St. West, Suite 202 Ottawa Ontario Canada K2P 0J2 Tel (613) 234-3758 FAX (613) 234-3565

**Note to physical activity professionals...**

It is a prudent practice to retain the completed Physical Activity Readiness Conveyance Referral Form in the participant's file.
Physical Activity & Lifestyle Advice

We know that being physically active provides benefits for all of us. Physical inactivity is recognized by the Heart and Stroke Foundation of Canada as one of the four modifiable primary risk factors for coronary heart disease (along with high blood pressure, high blood cholesterol, and smoking). Physical activity has also been shown to reduce the incidence of hypertension, colon cancer, maturity onset diabetes mellitus, and osteoporosis. It can also reduce stress and anxiety, relieve depression, and improve self-esteem.

People are physically active for many reasons — play, work, competition, health, creativity, enjoying the outdoors, being with friends. There are also as many ways of being active as there are reasons. What we choose to do depends on our own abilities and desires. No matter what the reason or type of activity, physical activity can improve our well-being and quality of life. Well-being can also be enhanced by integrating physical activity with enjoyable healthy eating and positive self and body image. Together, all three equal VITALITY. So take a fresh approach to living. Check out the VITALITY tips below!

Active Living:
- make meaningful and satisfying physical activities a valued and integral part of daily living
- accumulate 30 minutes or more of moderate physical activity most days of the week
- choose from an endless range of opportunities to be active according to your own abilities and desires:
  - take the stairs instead of an elevator
  - get off the bus early and walk home
  - join friends in a sport activity
  - take the dog for a walk with the family
  - follow a fitness program

Healthy Eating:
- follow Canada’s Food Guide to Healthy Eating
- enjoy a variety of foods
  - emphasize cereals, breads, other grain products, vegetables and fruit
  - choose lower-fat dairy products, leaner meats and foods prepared with little or no fat
  - achieve and maintain a healthy weight by enjoying regular physical activity and healthy eating
  - limit salt, alcohol and caffeine
  - don’t give up foods you enjoy — aim for moderation and variety

Positive Self and Body Image:
- accept who you are and how you look
- remember, a healthy weight range is one that is realistic for your own body make-up (body fat levels should neither be too high nor too low)
- try a new challenge
- complement yourself
- reflect positively on your abilities
- laugh a lot

Enjoy eating well, being active and feeling good about yourself. That’s VITALITY.

Physical Activity Readiness Conveyance/Referral Form

Based upon a current review of the health status of _________________________________, I recommend:
- No physical activity
- Only a medically-supervised exercise program until further medical clearance
- Progressive physical activity
  - with avoidance of: _________________________________________________________
  - with inclusion of: _______________________________________________________
  - with Physical Therapy ____________________________________________________
- Unrestricted physical activity — start slowly and build up gradually

Further Information:
- Attached
- To be forwarded
- Available on request

Physician's clinic stamp: __________________________ M.D.

_________________________ 19 __________
(date)
MEDICAL AND HEALTH SCREENING FORM

The purpose of this form is to get some information about your medical and health history. This is just to ensure that you do not have any medical or health problems that are contraindications to the testing.

Name: ___________________________ Date: ________________

Address:
________________________________________________________________________
________________________________________________________________________
________________________________________________________________________

Phone (home): ________________ (work): ________________

Birth date: ________________ Medicare: ________________

Family Physician: ________________ Phone: ________________

MENSTRUAL STATUS

1. To the best of your knowledge, what was the date of your last menstrual period?

2. How regular is your menstrual cycle?

3. Have you ever been prescribed any supplemental hormones for any reason (including birth control pills and hormone replacement therapy)? If so, when and for how long? What kind?
**GENERAL SCREENING**

4. Have you answered "yes" to any of the questions on the PAR-Q?

5. Do you have any allergies? If so, to what?

**MEDICAL HISTORY**

6. Please identify if you have had any of the following health problems:

<table>
<thead>
<tr>
<th>Problem</th>
<th>Yes</th>
<th>No</th>
</tr>
</thead>
<tbody>
<tr>
<td>a) high blood pressure</td>
<td>yes</td>
<td>no</td>
</tr>
<tr>
<td>b) heart trouble</td>
<td>yes</td>
<td>no</td>
</tr>
<tr>
<td>c) diabetes</td>
<td>yes</td>
<td>no</td>
</tr>
<tr>
<td>d) asthma</td>
<td>yes</td>
<td>no</td>
</tr>
<tr>
<td>e) epilepsy</td>
<td>yes</td>
<td>no</td>
</tr>
<tr>
<td>f) thyroid disorder</td>
<td>yes</td>
<td>no</td>
</tr>
<tr>
<td>g) any disease of the glands</td>
<td>yes</td>
<td>no</td>
</tr>
<tr>
<td>h) rheumatic fever</td>
<td>yes</td>
<td>no</td>
</tr>
<tr>
<td>i) arthritis, rheumatism</td>
<td>yes</td>
<td>no</td>
</tr>
<tr>
<td>j) kidney disease</td>
<td>yes</td>
<td>no</td>
</tr>
<tr>
<td>k) fainting syncope</td>
<td>yes</td>
<td>no</td>
</tr>
<tr>
<td>l) other ________________</td>
<td>yes</td>
<td>no</td>
</tr>
</tbody>
</table>
7. Have you ever had any musculoskeletal problems (injuries to bones, muscles, joints)? If so, describe the problem. Are you currently having any musculoskeletal problems? If so, what?

8. Have you ever been injured in an accident? If so, how?

9. List any surgery you have had.

10. Have you ever been advised for medical reasons not to participate in vigorous activity? If so, when? Why?

FAMILY HISTORY

11. Is there a history of sudden death in your family?

12. Is there a history of cardiac disease in your family?

DRUGS, FOOD SUPPLEMENTS AND MISCELLANEOUS AGENTS

13. Are you taking any medications at present? If so, what? Why?
14. Are you taking any vitamins at present? If so, what?

15. Do you regularly consume sports bars or sports drinks as a part of your training?
   How often, how many, and what brand?

16. Are you taking any stimulants (benzedrine, amphetamines)? If so, what?

17. Are you taking any anabolic steroids or agents? If so, what?

18. Are you taking any other prescription drugs? If so, what?

19. Are you taking any herbal preparations or non-prescription medications not listed above? If so, what?

20. Do you smoke? If so, how much?
OTHER

21. Is there any other medical information that you think may be important to your participation in this study?

22. Is there any reason why you think you may not be able to actively participate in this study?
# RESISTANCE TRAINING LOG

<table>
<thead>
<tr>
<th>DATE</th>
<th>Comments</th>
</tr>
</thead>
</table>

<table>
<thead>
<tr>
<th>Exercise</th>
<th># of reps</th>
<th># of sets</th>
<th>weight</th>
<th># of reps</th>
<th># of sets</th>
<th>weight</th>
<th># of reps</th>
<th># of sets</th>
<th>weight</th>
<th># of reps</th>
<th># of sets</th>
<th>weight</th>
</tr>
</thead>
<tbody>
<tr>
<td>DATE</td>
<td>EXERCISE</td>
<td>DURATION</td>
<td>INTENSITY (incl. speed, resistance)</td>
<td>COMMENTS</td>
<td></td>
<td></td>
<td></td>
<td></td>
<td></td>
<td></td>
<td></td>
<td></td>
</tr>
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</tr>
</tbody>
</table>

**AEROBIC TRAINING LOG**
APPENDIX C

PRE-TESTING GUIDELINES
PRE-TESTING GUIDELINES

Name: ___________________________  Age: ____________

Session Date: ______________________  Time: ____________

Location: _______________________________________

Please adhere to the following conditions:

**Dress Requirements:** Shorts or sweat pants and short-sleeved shirt should be worn. Sneakers are also required.

**Food and Beverages:** Eat your standardized meal two hours before each of your sessions. No other food is to be consumed within two hours of your appointment. No caffeine or alcohol should be consumed for at least six hours prior to your appointment.

**Physical Activity:** Physical activity should be avoided for twenty-four hours prior to your appointment. This is very important.
APPENDIX D

BIOCHEMICAL ANALYSIS PROCEDURES
Table 11: Two-site immunoradiometric assay (IRMA) procedures for serum growth hormone and serum insulin-like growth factor I

<table>
<thead>
<tr>
<th>GROWTH HORMONE</th>
<th>INSULIN-LIKE GROWTH FACTOR I</th>
</tr>
</thead>
<tbody>
<tr>
<td>Label Anti-hGH-Coated Tubes in duplicate for Standards, Controls and unknowns. Label 2 tubes (uncoated) for Total Count tubes.</td>
<td>Label Anti-IGF-I-Coated Tubes in duplicate for Standards, Controls and unknowns. Label 2 tubes (uncoated) for Total Count tubes.</td>
</tr>
<tr>
<td>Pipet 50 μL of Standards, Controls, and unknowns to bottom of appropriate tubes.</td>
<td>Add 50 μL of reconstituted Standards, Controls and prediluted unknowns to bottom of appropriate tubes.</td>
</tr>
<tr>
<td>Mix tubes by shaking test tube rack gently and incubate all tubes at room temperature (~25°C) on a shaker set at 180 rpm for 4 hours.</td>
<td>Mix tubes by shaking test tube rack gently and incubate all tubes at room temperature (~25°C) on a shaker set at 180 rpm for 2 hours.</td>
</tr>
<tr>
<td>Decant all tubes, (except Total Count tubes), by simultaneous inversion with a sponge rack. Strike tubes on absorbent material and then allow them to drain for 1-2 minutes. Blot tubes to remove droplets.</td>
<td></td>
</tr>
<tr>
<td>Add 3 mL of deionized water to all tubes (except Total Count tubes). Decant these tubes.</td>
<td>Add 3 mL of Wash Solution to each tube (except Total Count tubes). Decant these tubes.</td>
</tr>
<tr>
<td>Repeat previous step twice for a total of three washings. Count all tubes in gamma counter for one minute.</td>
<td></td>
</tr>
<tr>
<td>Calculate the net counts per minute (net CPM) by subtracting the mean CPM of the 0 ng/mL Standard from the mean CPM of each Standard, Control and unknown.</td>
<td></td>
</tr>
<tr>
<td>Calculate the % Bound/Total (B/T) for each Standard, Control and unknown.</td>
<td>%B/T = ( \frac{\text{Net CPM}}{\text{Mean Total CPM}} \times 100 )</td>
</tr>
<tr>
<td>Plot a curve of %B/T for each standard against the hGH concentration on log-log graph paper and draw a standard curve.</td>
<td>Plot a curve of %B/T for each standard against the IGF-I concentration on log-log graph paper and draw a standard curve.</td>
</tr>
<tr>
<td>Based on the standard curve, determine the hGH concentration from the means of the duplicate counts of each Control and the unknown.</td>
<td>Based on the standard curve, determine the IGF-I concentration from the means of the duplicate counts of each Control and the unknown.</td>
</tr>
<tr>
<td>Any sample reading greater than the highest Standard should be diluted with 0 ng/mL hGH Standard and reassayed.</td>
<td>Any sample reading greater than the highest Standard should be diluted with the Sample Diluent and reassayed.</td>
</tr>
<tr>
<td>Any sample reading less than the lowest Standard should be reported as such.</td>
<td></td>
</tr>
</tbody>
</table>
Table 12: Radioimmunoassay procedures for serum estradiol and serum dehydroepiandrosterone

<table>
<thead>
<tr>
<th>ESTRADIOL (ultra-sensitive)</th>
<th>DEHYDROEPIANDROSTERONE</th>
</tr>
</thead>
<tbody>
<tr>
<td>Label tubes in duplicate for Total Counts, Non-Specific Binding (NSB), Standards, Controls and unknowns.</td>
<td>Label Anti-DHEA-Coated Tubes in duplicate for Standards, Controls and unknowns. Label two tubes (uncoated) for Total Counts.</td>
</tr>
<tr>
<td>Add 200 µL of the Standards, Controls or unknowns to appropriate tubes. Add 300 µL of the 0 pg/mL Estradiol Standard to the NSB tubes.</td>
<td>Add 100 µL of the Standards, Controls and unknowns to the appropriate tubes.</td>
</tr>
<tr>
<td>Add 100 µL of Estradiol Antiserum to all tubes except NSB and Total Count tubes. Vortex all tubes, then cover and incubate at room temperature (~25°C) for 1 hour.</td>
<td>Add 500 µL of the DHEA [I-125] Reagent to all tubes. Vortex all tubes for 1-3 seconds.</td>
</tr>
<tr>
<td>Add 100 µL of Estradiol [I-125] Reagent to each tube. Vortex all tubes then cover and incubate at room temperature for 2 hours.</td>
<td>Incubate all tubes at 37°C for 2 hours.</td>
</tr>
<tr>
<td>Add 1 mL of Precipitating Reagent to all tubes except Total Count tubes. Vortex and let stand at room temperature for 15-20 minutes.</td>
<td>Decant all tubes (except Total Count tubes), by simultaneous inversion with a sponge rack. Strike tubes, then allow them to drain onto absorbent material for a minimum of 2 minutes. Blot tubes to remove droplets.</td>
</tr>
<tr>
<td>Centrifuge all tubes, except the Total Count tubes, for 15-20 minutes at 1500 x g.</td>
<td>Count all tubes in a gamma counter for one minute.</td>
</tr>
<tr>
<td>Calculate the mean counts per minute (cpm) for each Standard, Control and unknown. Calculate % Bound/Total (% B/T) for each Standard, Control and unknown. % B/T = ( \frac{\text{Mean Sample Counts}}{\text{Mean Total Counts}} ) x 100</td>
<td>Plot a curve for % B/T for the DHEA Standards against the DHEA concentration on a log-linear graph paper. Draw a standard curve.</td>
</tr>
<tr>
<td>Decant all tubes except Total Count tubes, by simultaneous inversion with a sponge rack. Allow tubes to drain on absorbent material for 15-30 seconds, then gently blot tubes.</td>
<td>Determine the DHEA concentration using the standard curve from the means of the duplicate counts of each Control and unknown.</td>
</tr>
<tr>
<td>Count all tubes in gamma counter for one minute.</td>
<td></td>
</tr>
</tbody>
</table>
Table 12: Radioimmunoassay (RIA) procedures for estradiol and dehydroepiandrosterone (continued)

<table>
<thead>
<tr>
<th>ESTRADIOL (ultra-sensitive)</th>
<th>DEHYDROEPIANDROSTERONE</th>
</tr>
</thead>
<tbody>
<tr>
<td>Calculate the mean counts per minute (cpm) for each Standard, Control and unknown.</td>
<td>Any sample reading greater than the highest Standard should be diluted with 0 ng/mL DHEA Standard and reassayed. Any sample reading less than the lowest Standard should be reported as such.</td>
</tr>
</tbody>
</table>
| Calculate the % Bound/Total (% B/T) for each Standard, Control and unknown. % B/T = \[
\frac{\text{Mean Sample Counts} - \text{NSB Counts}}{\text{Mean Total Counts}} \times 100
\] | |
| Plot % B/T for the Standards against the estradiol concentration on semi-log graph paper. Draw a standard curve. | |
| Determine the estradiol concentration using the standard curve from the means of the duplicate counts of each Control and unknown. | |
| Any sample reading greater than the highest Standard should be diluted with 0 ng/mL Standard and reassayed. Any sample reading less than the lowest Standard should be reported as such. | |
### Table 13: Chemiluminescent procedure for serum total testosterone

<table>
<thead>
<tr>
<th>Step</th>
<th>Description</th>
</tr>
</thead>
<tbody>
<tr>
<td>Wash sample probe with 225 μL of Wash Reagent 2.</td>
<td></td>
</tr>
<tr>
<td>Dispense 15 μL of sample and 50 μL of Releasing Agent into cuvette.</td>
<td></td>
</tr>
<tr>
<td>Dispense 50 μL of Lite Reagent and 300 μL of Solid Phase into cuvette.</td>
<td></td>
</tr>
<tr>
<td>Incubate at 37°C for 5 minutes.</td>
<td></td>
</tr>
<tr>
<td>Separate, aspirate, and wash cuvette.</td>
<td></td>
</tr>
<tr>
<td>Dispense 300 μL each of Reagent 1 and Reagent 2 to start chemiluminescent reaction.</td>
<td></td>
</tr>
<tr>
<td>Measure the amount of light being emitted by sample.</td>
<td></td>
</tr>
<tr>
<td>Calculate the amount of testosterone in the sample based on an inverse relationship between the amount of testosterone and the RLUs (relative light units) detected.</td>
<td></td>
</tr>
</tbody>
</table>
Table 14: Spectrophotometric procedures for lactate analysis

| LACTATE |
|------------------|------------------|------------------|
| Label cuvets in duplicate for Standards, Blank and unknowns. |
| Pipet 1.0 mL of the Lactate Reagent Solution into the Blank, Standard and unknown cuvets. |
| To the Standard cuvets add 10 µL of Lactate Standard Solution. |
| To the test cuvets add 10 µL of sample plasma. |
| Incubate the cuvets for 5-10 minutes. |
| Read and record the absorbance (A) of the Standard and unknowns versus the Blank as a reference at 540 nm. Complete all readings within 10 minutes following the incubation period. |
| Calculate Lactate concentration of Standards and unknowns. |
| Lactate (mg/dL) = \( \frac{\text{Absorbance Test}}{\text{Absorbance Standard}} \times 40^* \) |
| * Concentration of lactate in standard. |
| To convert mg/dL to mmol/L multiply mg/dL by 0.111. |