A COMPARATIVE STUDY OF THE EFFECT OF ACUTE EXERCISE ON THE HYPOTHALAMIC-PITUITARY-GONADAL AXIS BETWEEN TRAINED AND NON-TRAINED HUMAN MALE SUBJECTS

PRESENTED BY

JEFFREY MOORE

MASTERS THESIS PRESENTED TO

THE SCHOOL OF GRADUATE STUDIES

UNIVERSITY OF OTTAWA

ISBN 0-315-85792-7

UNIVERSITÉ D'OTTAWA
UNIVERSITY OF OTTAWA

TABLE OF CONTENTS

List of Figures

List of Tables

ABSTRACT

The purpose of this study was to compare the effects of acute exercise on plasma testosterone, LH, FSH and prolactin. In addition, the effects of LH, FSH, Prolactin and physical fitness on testosterone levels were investigated. The serum levels of testosterone, LH, FSH and prolactin were measured at rest, immediately after, 30 minutes and 60 minutes after acute exercise, which comprised of running on a treadmill at 70% of the subjects' previously determined MVO_2 for 20 minutes. Trained (n=12) and untrained (n=9) male subjects were tested. Mean testosterone levels increased significantly ($p<0.01$) in both groups immediately after cessation of exercise, independent of the effects of LH, FSH and prolactin. Testosterone responses to exercise were significantly ($p<0.01$) greater in trained (45% increase) than in the untrained (19.5% increase) group. In addition, basal levels of testosterone were significantly lower ($p<0.01$) in the trained group compared to the untrained group.

INTRODUCTION

Some research has been done on the effects of training and exercise on such hormones as testosterone, luteinizing hormone (LH), follicle stimulating hormone (FSH), and prolactin in males. However, this area is still not well understood and there is much confusion and contradiction when comparing the conclusions of various researchers (Lamb et al., 1975; Hackney et al., 1986; Keizer et al., 1989; Jezová et al., 1985).

One of the reasons why attention is being focused on these hormones is because they play a vital role in relation to exercise. This role includes testosterone's involvement in protein synthesis, muscle strength and aggressiveness (Hackney et al., 1989a; Diamond et al., 1989; Wilkerson et al., 1980; Kuoppasalmi et al., 1980; Catt et al., 1978). These factors are very important, especially in competitive events. Another reason why attention was being focused on these hormones (especially testosterone) was the fact that it was thought endurance exercise could possibly cause infertility in males (Nash et al., 1987; Wheeler et al., 1984; Ayers et al., 1985; Aakvaag et al., 1978; Cumming et al., 1983).

Responses of testosterone, LH, FSH and prolactin to exercise (in males) have varied quite extensively from study to study (see table 1). Studies have shown that testosterone levels can increase, decrease or remain unchanged as a result of exercise.

There seems to be conflicting results in the literature concerning exercise and testosterone response. However, in the studies where testosterone levels decreased, endurance exercise was performed by the subjects (Opstad et al., 1982; Urhausen et al., 1987a; De Lignieres et al., 1976; Cumming et al., 1987). In a study by Semple et al. (1985), subjects ran a marathon and it was found that testosterone levels in the volunteers were lower than at the beginning of the race. In studies

where testosterone increased, the exercises performed were of short to moderate duration and more intense (Cumming et al., 1986; Sutton et al., 1973; Galbo et al., 1977; Métivier et al., 1980; Kindermann et al., 1982). However, the aforementioned type of exercise does not necessarily lead to an increase in testosterone levels. In the case of Bottecchia et al. (1987), short-term, intense exercise lead to no changes in testosterone levels.

Long-term training or training programs have also been seen to have an effect on reproductive hormone levels in males. Training can decrease testosterone levels, especially basal levels (Hackney et al., 1988; McColl et al., 1989; Hakkinen et al., 1988; Hakkinen et al., 1989; Rowland et al., 1987). In addition, it has been shown that trained subjects have a larger testosterone response to exercise than non-trained subjects (Dessypris et al., 1976; Fellmann et al., 1985; Remes et al., 1979). In one case, however, it was found that non-trained subjects had a larger response than trained subjects (Mathur et al., 1986).

Studies have also shown that LH levels may increase, decrease or remain unchanged due to exercise (Kuoppasalmi et al., 1976; MacConnie et al., 1986; Dessypris et al., 1976; Elias et al., 1989). The majority of the studies seem to indicate that LH levels are unaffected by exercise (see table 2). In addition, it has been shown that FSH levels remain unchanged (Gawel et al., 1979; Vogol et al., 1985) or decrease (Brisson et al., 1979) with exercise but most of the studies indicate that FSH levels remain unchanged due to exercise (see table 3). Most researchers have found that prolactin increases with exercise (Christensen et al., 1985; Kaufman et al., 1985; Delitala et al., 1987) but some researchers have found no change (Toriola et al., 1986; Rolandi et al., 1985).

Thus, there seems to be conflicting results in the literature. This

is due to the fact that these hormonal responses, especially those of testosterone, are dependent on the type and duration of the exercise, the workload and the physical fitness of the subject (Galbo et al., 1981; Weiss et al., 1983; Cadoux-Hudson et al., 1985).

Table 1

Review of Literature on Testosterone Response to Exercise

Testosterone increases	Testosterone decreases	Testosterone N.C.
Weiss et al., 1983	Aakvaag et al., 1978	Lamb et al., 1975
Métivier et al., 1980	Opstad et al., 1982	
Kuoppasalmi et al., 1980	Ayers et al., 1985	
MacConnie et al., 1986	Semple et al., 1985	
Vogol et al., 1985	Urhausen et al., 1987a	
Diamond et al., 1989	Nash et al., 1987	
Remes et al., 1979	De Lignieres et al., 1976	
Cumming et al., 1986	Dessypris et al., 1976	
Jezová et al., 1985		
Wilkerson et al., 1980		
Sutton et al., 1973		
Galbo et al., 1977		
Kindermann et al., 1982		
Gawel et al., 1979		
Cadoux-Hudson et al., 1985		

N.C. = no change

Table 2

Literature Review of LH Responses to Exercise

LH increases	LH decreases	LH (N.C.)
Kuoppasalmi, 1976	Dessypris et al., 1976	Sutton et al., 1973
Cumming et al., 1986	Elias et al, 1989	Métivier et al., 1980
Gawel et al., 1979	Brisson et al., 1979	Galbo et al., 1977
		Toriola et al., 1986
		MacConnie et al., 1986
		McColl et al., 1989

N.C. = no change

Table 3

Literature Review of FSH Responses to Exercise

FSH increases	FSH decreases	FSH (N.C.)
Cumming et al., 1986	Brisson et al., 1979	Aakvaag et al., 1978
		Gawel et al., 1979
		Vogol et al., 1985
		Toriola et al., 1986
		Rolandi et al., 1985

N.C. = no change

Purpose

The purpose of this study was to compare the effects of an exercise intensity equivalent to 70% of maximal oxygen consumption (MVO_2) on plasma testosterone, LH, FSH and prolactin levels between trained and untrained subjects. Another purpose was to investigate the effects of LH, FSH, Prolactin and physical fitness on testosterone levels.

Hypothesis

It was hypothesized that an exercise intensity corresponding to 70% of the subject's MVO_2 would elevate plasma levels of testosterone and that this elevation would be independent of the influences of LH, FSH and prolactin. It was also hypothesized that increased physical fitness would lower basal levels of testosterone and also increase the testosterone response to exercise.

METHODS

Subjects

Twenty-one healthy white male subjects between the ages of 19 and 28 years (mean age of 22.5), who were all students of the University of Ottawa, agreed to participate in this study and signed a consent form to that effect. Nine of these students were considered untrained (i.e. their MVO_2 was less or equal to 50 ml O_2/kg/min). The other 12 were considered to be trained (i.e. their MVO_2 was greater than or equal to 55 ml O_2/kg/min) and were members of the University of Ottawa cross-country running team.

Anthropometric measurements

Before the maximum oxygen consumption test, anthropometric variables were measured, including weight and height. The percentage of fat in the body was estimated according to the measurement of skinfold thickness (Fahey et al., 1976).

Maximum oxygen consumption

A continuous progressive treadmill test (see table 4) was used to determine each subject's maximum oxygen consumption (MVO_2) approximately one week before the study. Blood pressure and heart rate were measured before and after the maximal exercise, heart rate being measured via a sport-tester heart rate monitor. The sport-tester was also used to measure heart rate throughout the entire exercise.

After a two-minute warm-up (at 5 km/hr, 0% grade for untrained individuals or 10km/hr, 0% grade for trained subjects), the treadmill speed was increased by 1 km/hr and the treadmill elevated to a 5% grade. For the remainder of the test, the speed was increased by 1 km/hr every 2 minutes but the grade remained constant.

Expired air samples were collected and analyzed by a Beckman Metabolic Measurements Cart (BMMC) at each minute of the protocol using

a high-speed respiratory valve mounted on an adjustable head harness. Minute sampling was continued until either the subject proclaimed fatigue or MVO_2 values failed to increase in the face of increased workload.

Experimental design

The experimental study protocol consisted of a 20 minute rest period, 20 minutes of exercise and a 60 minute recovery period conducted in the morning following a 12-hour fast. The exercise was performed on a motor-driven treadmill at a workload designed to elicit an oxygen uptake of 70% of the previously determined MVO_2. The workload was appropriately readjusted during the test to maintain the desired oxygen uptake. During the exercise, the BMMC directly measured 1-minute expired air samples for oxygen uptake, and these measurements were used as an indication as to whether the workload had to be changed. Heart rate was also monitored continuously through the use of a sport-tester watch. Blood samples were taken immediately before exercise, immediately after the exercise, and then 30 and 60 minutes following cessation of exercise.

Table 4

Protocol used for Treadmill Running

Stage (#)	Time (min)	Grade (%)	Speed (km/h)
warm-up *	2	0	5
1	2	5.0	6
2	2	5.0	7
3	2	5.0	8
4	2	5.0	9
5**	2	0	10
6	2	5.0	11
7	2	5.0	12
8	2	5.0	13
9	2	5.0	14
10	2	5.0	15
11	2	5.0	16
12	2	5.0	17
13	2	5.0	18

* First stage for untrained subjects

** First stage for trained subjects

Analytical Methods

Blood samples for the determinations of serum hormones were drawn by venipuncture from the antecubital vein of each subject. For each sample of blood, a volume of 10cc was drawn from the vein. The blood was placed

in pre-chilled (2-8°C) heparinized glass tubes. The plasma was then separated from the blood as quickly as possible, using a refridgerated centrifuge (2-8°C). The samples were then stored at -20°C until assayed.

Concentrations of testosterone were determined using the Coat-A-Count Total Testosterone kit (Diagnostic Products Corp., 1988), which consisted of a solid phase radioimmunoassay, based on testosterone-specific antibody immobilized to the wall of a propylene tube. ^{125}I-labelled testosterone competed for a fixed time with testosterone in the subject sample for antibody sites. The tube was then decanted, to separate "bound" from "free", and counted in a gamma counter.

Concentrations of FSH were determined using a solution of FSH which was marked with ^{125}I. The Amerlex-M FSH RIA kit (Amersham, 1989) was used for this purpose. In addition, the Amerlex-M LH RIA kit was used to determine concentrations of LH. Prolactin was measured using the Tandem-R Prolactin Immunoradiometric assay kit which was developed by Hybritech (1989).

For the evaluation of changes in plasma volume, hematocrit determinations were made according to the microhematocrit technique. Once the hematocrit values were determined, the change in plasma volume was found using the following equation:

$$\%\Delta\ PV = (100/(100-H_1)) \times ((100 \times (H_1 - H_2))/H_2)\ ^*$$

where PV = plasma volume

H_1 = pre-exercise hematocrit value

H_2 = post-exercise hematocrit value

* taken from Van Beaumont (1972).

Statistical analysis

Measures were analyzed for significant differences and/or

interactions utilizing a 2 (subject group) x 4 (time) factorial ANOVA (i.e. a two-way analysis of variance) with repeated measures on the last factor. Where applicable, significant differences were determined by Tukey HSD post-hoc procedures.

RESULTS

Subjects

Table 5 and Table 6 give a description of the subjects used in this study.

Table 5

Characterization of trained subjects

Descriptor	n	Mean	Standard Error	Minimum	Maximum
Age (yrs)	12	22.25	0.55	19.00	26.00
Height (cm)	12	179.1	1.7	170.2	189.6
Weight (kg)	12	71.3	1.9	62.3	85.2
Body Fat (%)	12	12.28	0.68	8.26	16.28
MVO2 *	12	65.28	1.79	57.00	79.90
Resting H.R.	12	54	3	38	75
Max. H.R.	12	185	3	168	207

* MVO_2 units are ml O_2/kg/min

H.R. = heart rate (measured in beats/minute)

Table 6

Characterization of untrained subjects

Descriptor	n	Mean	Standard Error	Minimum	Maximum
Age (yrs)	9	23	0.73	20	28
Height (cm)	9	180.8	1.9	173.7	190.3
Weight (kg)	9	87.2	4.4	69.0	111.3
Body Fat (%)	9	19.90	1.35	15.27	28.18
MVO2	9	45.29	1.02	41.59	49.60
Resting H.R.	9	66	3	54	82
Max. H.R.	9	195	2	186	201

Hematocrit and hormones

Table 7 gives the values for hematocrit, testosterone, LH, FSH and prolactin.

Table 7

Group means of hormones (± SE) and hematocrit.

Variable	Trained Subjects (n=12)				Untrained Subjects (n=9)			
	PRE	POST	P30	P60	PRE	POST	P30	P60
Testosterone	19.8*	28.7	20.7	19.3	25.3	30.2	23.2	22.1
(nmol/L)	±0.7	±0.9	±0.9	±0.9	±0.4	±0.9	±0.7	±1.1
Prolactin	6.4	6.8	5.6	5.3	4.4	4.8	4.6	4.1
(ng/ml)	±0.7	±0.7	±0.6	±0.5	±0.5	±0.6	±0.4	±0.5
LH	5.3	5.8	5.1	5.1	6.8	6.7	7.1	6.7
(mlU/ml)	±0.4	±0.3	±0.3	±0.4	±0.6	±0.5	±0.6	±0.6
FSH	4.7	4.5	4.5	4.2	5.7	5.5	5.3	5.2
(mlU/ml)	±0.3	±0.3	±0.3	±0.3	±0.7	±0.7	±0.6	±0.6
Hematocrit	46.3	48.8	45.3	45.9	45.9	48.4	45.4	45.4
(Vol. %)	±0.5	±0.6	±0.6	±0.5	±0.8	±0.7	±0.8	±0.7

PRE = before exercise
Post = immediately after exercise
P30 =30 minutes after exercise
P60 =60 minutes after exercise

*Pre-exercise value of testosterone is significanty lower ($p<0.01$) in trained subjects than untrained. Differences in other paired pre-exercise values were insignificant.

Hematocrit values were elevated significantly ($p<0.01$) by 5.4% in both trained and untrained subjects after cessation of exercise. According to the equation developed by Van Beaumont (1972), this translates to a 9.5 % decrease in plasma volume. The values returned to resting levels after 30 minutes of recovery (see table 7 and figure 1).

The pre-exercise values (basal level) of plasma testosterone were within a normal range for all subjects. Plasma testosterone levels were elevated significantly ($p<0.01$) at the end of exercise for both groups, but then returned to basal levels after 30 minutes of recovery (see table 7 and figure 2). Testosterone levels increased by 45% in the trained group whereas an increase of 19.5% was observed in the untrained group. The difference in these increases between the two subject groups was found to be significant ($p<0.01$).

Prolactin rose slightly in both groups after exercise but was found to be statistically insignificant. Levels returned back to pre-exercise values in the untrained group whereas in the trained group the recovery values fell below basal levels (see figure 5). None of the recovery values were significantly different (statistically) from pre-exercise values.

FSH levels declined slightly (nonsignificant) in both groups after cessation of exercise and this decline continued into the 60 minute recovery period (see figure 4). In addition, none of the recovery levels of FSH were significantly different from pre-exercise values.

LH responses to exercise were unique for each group. LH levels increased after cessation of exercise in the trained group and returned back to resting levels after 30 minutes of recovery (see figure 3). However, none of these post-exercise levels were significantly different from the resting level of LH. In the untrained group, LH levels

decreased slightly after cessation of exercise. Also, after 30 minutes of recovery, LH levels were found to increase above the resting level. After 60 minutes of recovery, LH levels were back to basal levels in the untrained group. None of the post-exercise measurements of LH in the untrained group were significantly different from basal level.

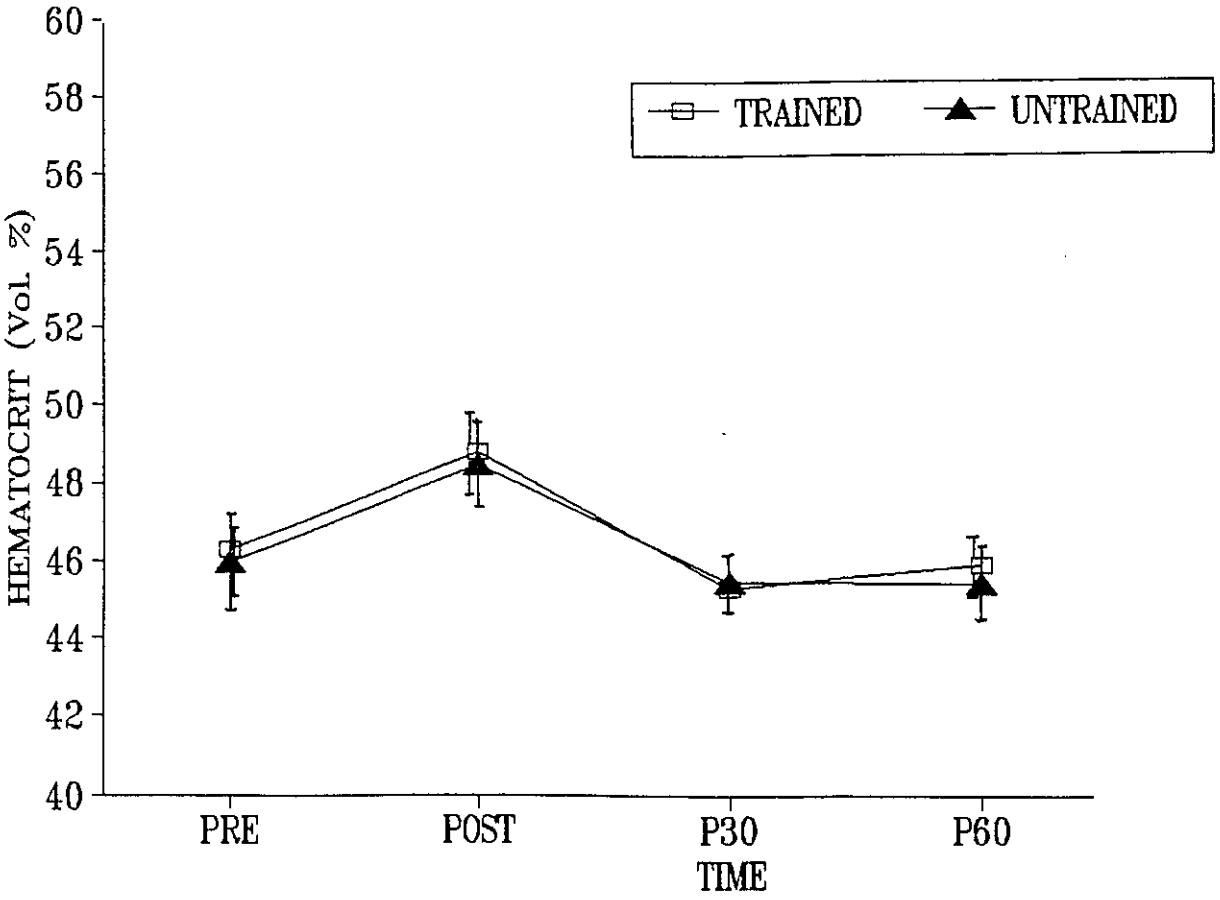

Figure 1: Hematocrit values in trained and untrained subjects.

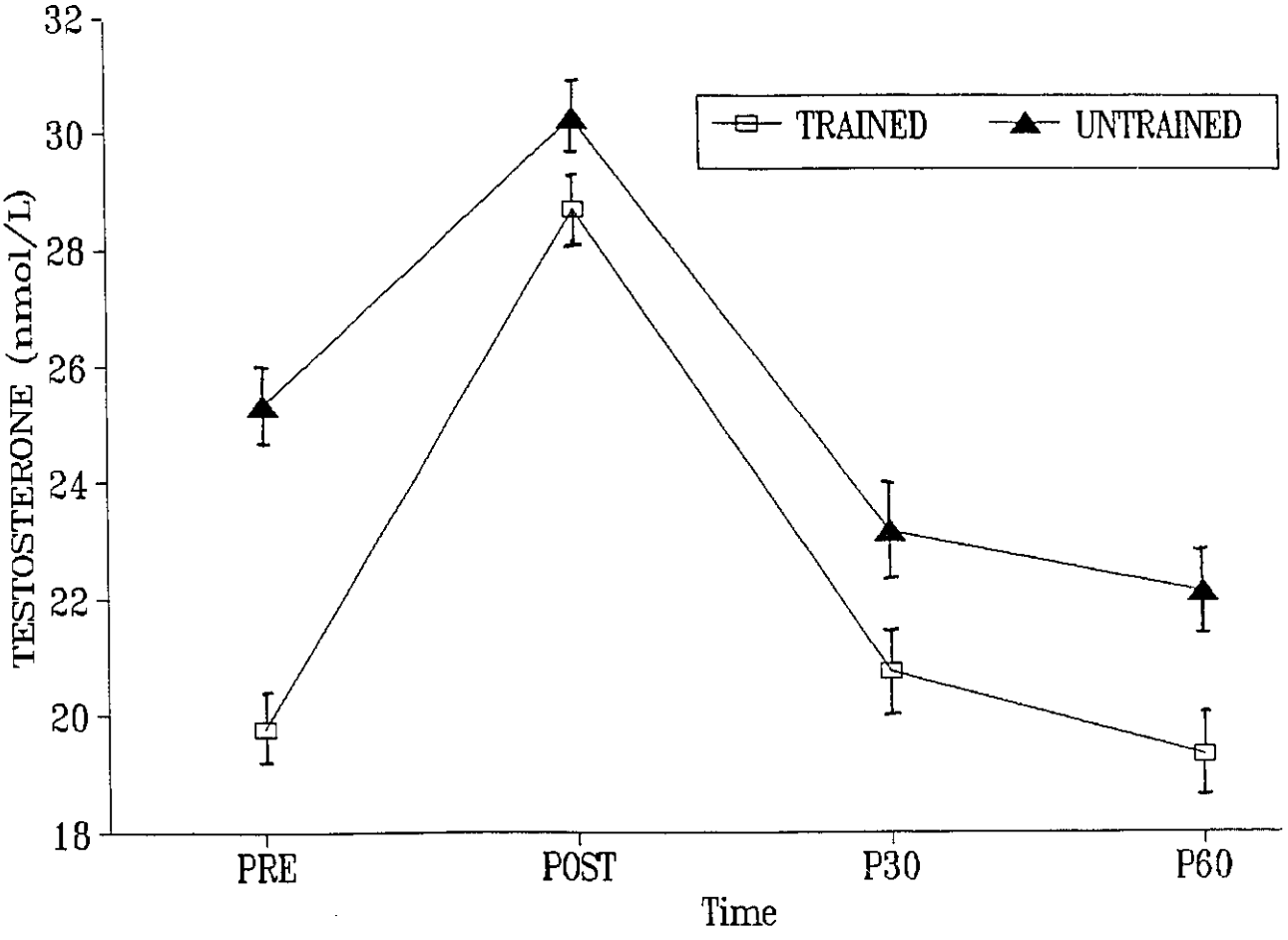

Figure 2: Testosterone response to exercise in trained and untrained subjects.

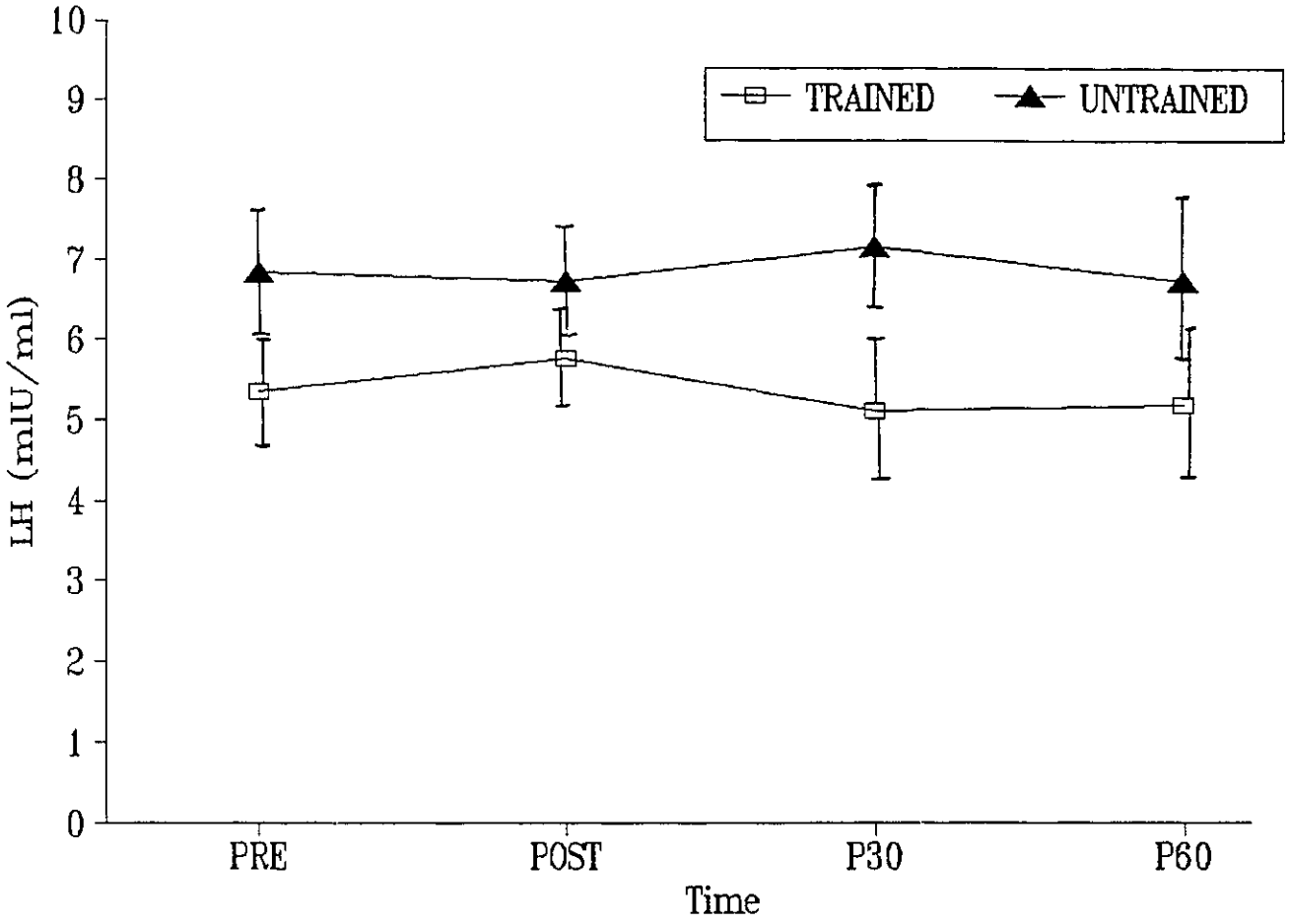

Figure 3: LH response to exercise in trained and untrained subjects.

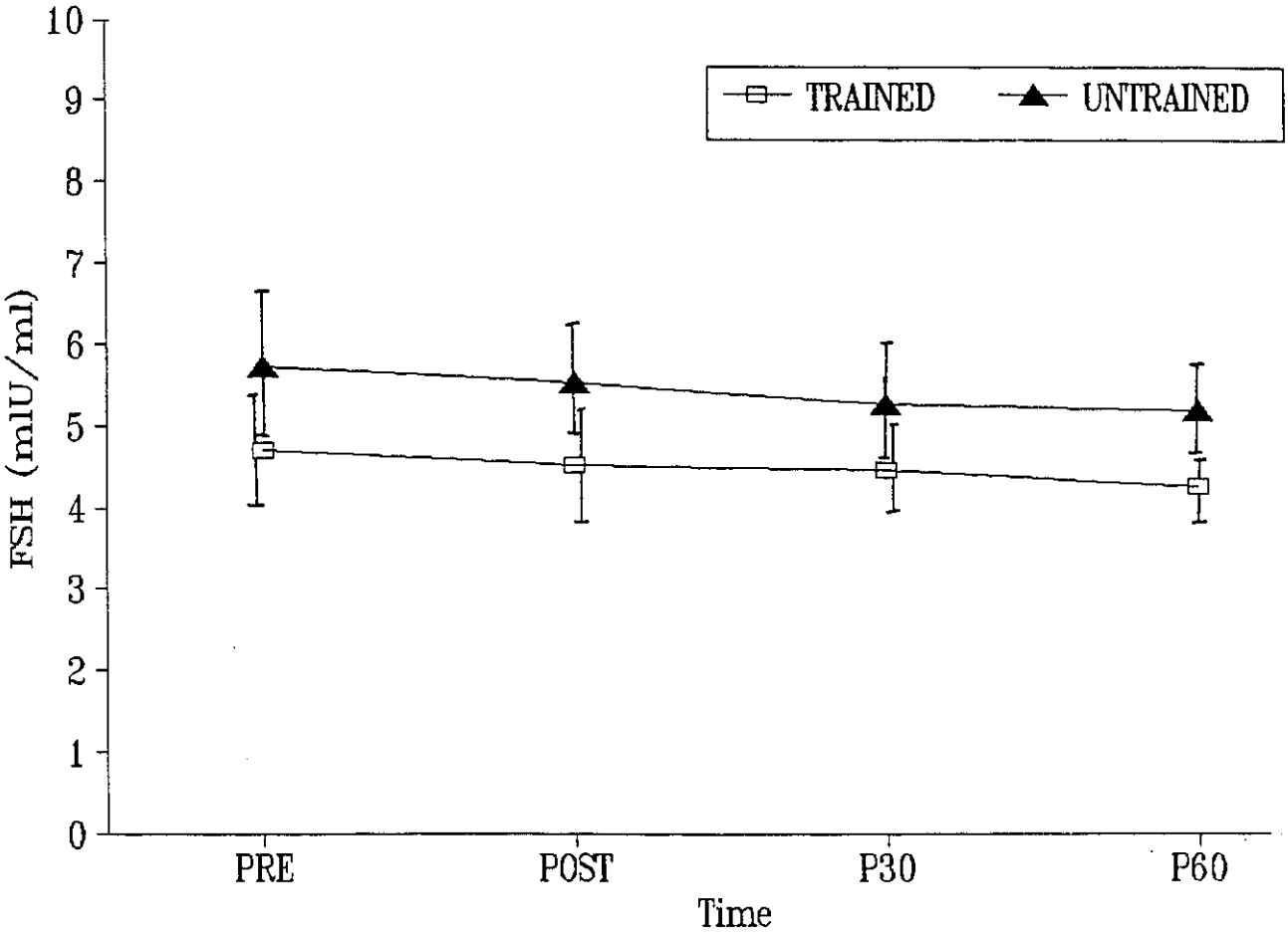

Figure 4: FSH response to exercise in trained and untrained subjects.

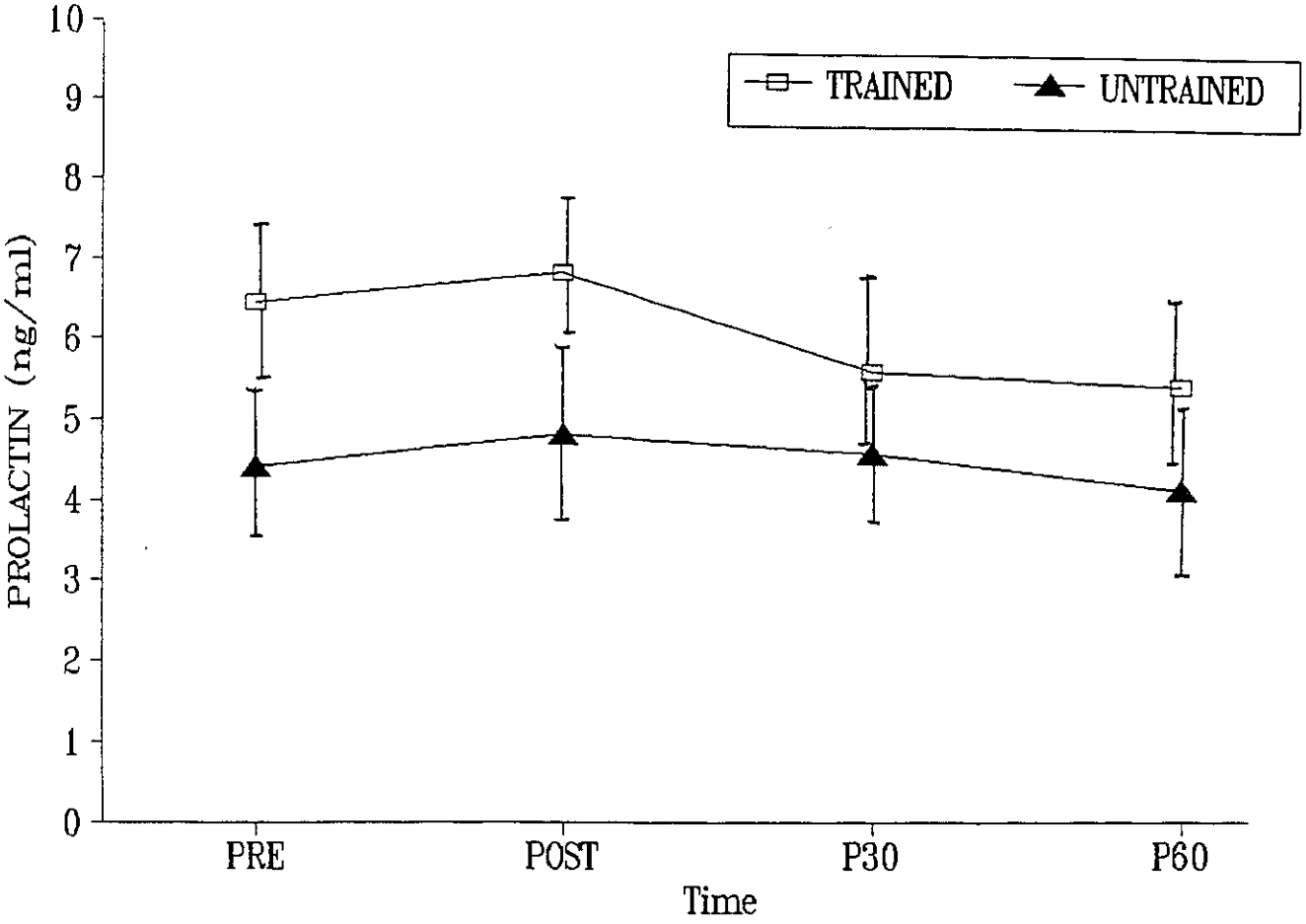

Figure 5: Prolactin response to exercise in trained and untrained subjects.

DISCUSSION

The metabolic response to exercise in trained and untrained males is determined by many variables, including physical fitness status, the work load imposed, and the duration of exercise. In this study, care was taken to directly measure MVO_2 as an expression of physical fitness and to maintain exercise intensity sufficient to elicit 70% of MVO_2 for 20 minutes. This work load varied from individual to individual, depending mainly upon the magnitude of MVO_2. However, the relative exercise performed in terms of percent MVO_2 was nearly the same for all individuals.

A rise in serum testosterone levels immediately after exercise in both trained and untrained groups was observed in this study. However, the increase was more pronounced in the trained group. In addition, plasma volume decreased by 9.5% in both subject groups as a result of exercise, due probably to a combination of perspiration and increased interstitial fluid. Thus, it is possible that hemoconcentration caused the increase in testosterone levels after exercise or at least played a small role in the augmented testosterone levels. Studies such as the one performed by Wilkerson et al. (1980) and Bottechia et al. (1987) illustrated that a loss in plasma volume was the sole reason for raised plasma testosterone levels. In this case, however, the change in plasma volume was not great enough to explain the large increases in testosterone levels. If hemoconcentration would have affected testosterone levels, then other hormones would also have been affected in the same way. But, as can be seen from the results, none of the other hormone levels increased significantly. In fact, FSH levels in both subject groups decreased with exercise, as well as LH levels for the untrained group. Results such as these support the findings of other authors (eg. Mathur et al., 1986; Remes et al., 1979), who believe that

elevated levels of testosterone after exercise are not due to hemoconcentration but are due to increased testosterone production.

A major factor to consider with regards to this study is the fact that a trained group (athletes) was compared to an untrained group (sedentary). These subjects were performing an exercise at 70% of their MVO_2. The problem with using this intensity is that untrained subjects usually reach the anaerobic threshold at approximately 60% of their MVO_2 but trained distance runners reach the anaerobic threshold at approximately 75% of their MVO_2.

Below the anaerobic threshold (during exercise), there is a redistribution of blood flow so that the active muscles receive the greatest proportion of the cardiac output. This redistribution results from (1) reflex vasoconstriction of the arterioles supplying the inactive areas of the body, especially those of the visceral organs and skin; (2) reflex vasodilation of the arterioles supplying the active skeletal muscles and (3) vasodilation in the active muscles caused by increases in local temperature, CO_2 and lactic acid levels and by a decrease in oxygen, particularly as the exercise continues. When a person reaches anaerobic threshold and goes beyond it, blood flow to the skin increases greatly and the amount of blood available to the working muscles is correspondingly reduced. In addition, Christensen et al. (1979) and Galbo et al. (1979) found that catecholamines (vasoconstrictors) were secreted in greater quantities at anaerobic threshold. This was attributed to circulatory needs resulting from thermoregulation (i.e. cutaneous vasodilation).

Thus, as previously mentioned, untrained subjects would be exercising at the anaerobic threshold. Hence, this subject group would have higher levels of catecholamines to offset the effects of thermoregulation. These catecholamines could have impaired gonadal blood flow (i.e.

through increased testicular vascular resistance), therefore resulting in reduced testosterone release. This could explain why testosterone levels increased more dramatically in trained subjects, since they would have lesser amounts of circulating catecholamines. Jezová et al. (1985) found that adrenergic blocking agents could modify the testosterone response to exercise, which would imply that during exercise plasma testosterone was influenced by the sympathetic nervous system or circulating catecholamines. The conclusion from this study was that catecholamines could be involved in the control of plasma testosterone through their effects on circulation or directly on testicular adrenergic receptors. Unfortunately, the only way to study whether the testes are being stimulated to produce more testosterone by catecholamines is to canulate the vessel; set up an intravenous tube and remove blood directly from the testes before, during and after exercise; and that procedure cannot ethically be done in humans.

It is possible that higher circulating concentrations of catecholamines in untrained subjects, due to anxiety, may have impaired gonadal blood flow, thus resulting in a reduced testosterone release (Wuttke et al., 1976; Levin et al., 1967; Jezová et al., 1981; Cartensen et al., 1972). Diamond et al. (1989) showed that the period preceding strenuous exercise, as well as the period of exercise itself, may be stressful to anxious subjects, especially if these subjects are not accustomed to being in a laboratory environment and also if they are unaccustomed to performing strenuous exercise. Diamond et al. also observed that testosterone appeared sensitive to levels of anxiety. Thus, when addressing the hypophysial-testicular endocrine axis, it is important to assess the psychological characteristics, especially for untrained subjects, who, according to their anxiety level, could have a stressful perception of strenuous exercise whereas trained subjects are

accustomed to exercise. This could explain the drop in testosterone levels 30 and 60 minutes post-exercise. Perhaps the concentration of testosterone at 60 minutes post-exercise is the actual basal concentration of testosterone, whereas the concentration of testosterone measured pre-exercise was elevated due to anxiety.

An important cardiovascular adjustment which occurs with exercise is the shunting of blood from the visceral organs (eg. liver, kidney) to the active muscles. This in turn reduces hepatic blood flow. Hence, testosterone will not travel to the liver but will instead travel to the working muscles, where it is needed most. In other words, the metabolic clearance rate (MCR) of testosterone will decrease, leading to higher plasma levels. This is probably the most likely explanation for the increased testosterone levels seen in both subject groups after exercise. This is in agreement with the conclusions of Cadoux-Hudson et al. (1985), as well as those of Kuoppasalmi et al. (1980), who has shown that 50% increases in plasma testosterone over the basal level may be caused by decreased hepatic MCR in exercise. However, this is not in agreement with the findings of Jezová et al. (1981). In this study, beta-adrenergic receptors were blocked, leading to a decrease in cardiac output. The decreased cardiac output caused a decrease in hepatic blood flow, but the levels of testosterone were unaffected.

Thus, a variety of alternate control pathways have been suggested to explain the testosterone increase. These include catecholamine stimulation as mentioned previously (Galbo et al., 1977; Mueller et al., 1976; Moretti et al., 1983; Elias et al., 1983), contribution of adrenal androgens (Dessypris et al., 1984; Kuoppasalmi et al., 1980b; Judd et al, 1978), decreased metabolic clearance as a result of decreased hepatic blood flow (Kuoppasalmi et al., 1980a; Métivier et al., 1980; Rowell et al, 1974; Eik-Nes et al., 1964; Eik-Nes et al., 1969),

increased available substrate such as cholesterol, and apparent increase due to hemoconcentration (Galbo et al., 1977; Bliss et al., 1972; Adlercreutz et al., 1976; Bunt et al., 1986). The rise in testosterone levels could also be due to increased stimulation of production in the testis (Bardin et al., 1978, Cook et al., 1986; Guezennec et al., 1982). Adrenal androgens could only make a very small contribution and there is no absolute evidence, as least from this study, which indicates that increased production of testosterone resulted from exercise.

Testosterone levels can also be augmented through the actions of LH, FSH, or prolactin, where LH has a direct effect on testosterone secretion and FSH, along with prolactin, have an indirect effect (Genuth et al., 1983; Williams et al., 1974; Schmid et al., 1982; Baker et al., 1975; Faiman et al., 1971). However, this study showed that neither LH, FSH nor prolactin levels changed significantly after exercise. This is in agreement with several studies (Rogol et al., 1984; Métivier, 1983; Métivier, 1985; Veldhuis et al., 1986), which observed that concentrations of LH, FSH and prolactin were insignificantly altered due to exercise. Similar results were reported by Rolandi et al. (1985). The numerous discrepancies reported in the literature on the response of gonadotrophins to exercise may be related not only to the difference in type, intensity and duration of physical activity, but also to other extraneous factors (i.e. drugs).

Thus, the exercise-associated increase in testosterone is not accompanied by increases in LH, FSH nor prolactin. However, even if the increase in LH for trained subjects (see figure 3) immediately after exercise (POST) were significant, the exercise-associated increase in serum testosterone levels could not result from gonadotropin stimulation since the increases in serum LH and testosterone were synchronous. The explanation behind this is that it has been reported that the time

interval between a spontaneous pulse of LH and the succeeding increment in testosterone exceeds 45 minutes (Naftolin et al., 1973).

Earlier it was mentioned that basal levels of testosterone were found to be lower in trained compared to untrained subjects ($p<0.01$) in this study. Several studies have reported reduced levels of testosterone in trained compared to untrained subjects (Ayers et al., 1985; Wheeler et al., 1984). Hackney et al. (1989b) also showed that basal levels of testosterone were lower in trained subjects. If this is due to a training effect, then the mechanism behind this effect is not known. On the other hand, the high basal levels of testosterone in untrained subjects could possibly be stress-related (Young et al., 1979; McGrady et al., 1984; Aono et al., 1976; Aakvaag et al., 1978).

The reduction of basal levels of testosterone in trained subjects could result in suppressed bodily functions, as this androgen plays a vital role in key physiological processes. Specifically, spermatogenesis and protein synthesis rely upon adequate levels of androgens to function properly (Bartsch et al., 1984; Coppage et al., 1965; Lipsett et al., 1978; McConnell et al., 1984). Whether these low, but within the normal range, androgen concentrations found in trained subjects has an effect on the androgen-dependent processes of the body is not certain. However, Ayers et al. (1985) found oligospermic conditions in two trained subjects having very low testosterone concentrations. In addition, the fact that LH and FSH levels are normal in this study indicate that low testosterone levels are most probably due to gonadal impairment, not a disruption of the hypothalamic-pituitary axis.

There is also a training effect involved in testosterone responses to exercise (Adlercreutz et al., 1986; Frey et al., 1983; Hakkinen et al., 1985; Hakkinen et al., 1987). For instance, Fellmann et al. (1985) observed that testosterone response to exercise increased with greater

training. They concluded that the more "trained" someone is (indicated by MVO_2), the greater the exercise-induced testosterone response. Similar findings were made by Dessypris et al. (1976), using marathon runners. This finding raises two questions: 1) What are the mechanisms involved in the increase of the testosterone response to exercise and 2) Does this hormonal change have any effect on energy metabolism in relation to the bioenergenic changes that are observed at the same time.

To reiterate, an increase in testosterone response (level) could be secondary either to an increase in testicular production of testosterone or to a decrease in testosterone catabolism (i.e. a decrease in blood flow through the liver and an increase through skeletal musculature (shunting of blood) could lead to decreased testosterone catabolism). This last possibility is probably not the case in Fellmann's study because the training program used in that study had been shown to reduce the epinephrine response to exercise and not to modify the norepinephrine response. In addition, a change in vascular resistance in the testis could explain a relative increase in testosterone release in circulating blood.

The role of androgens in exercise, although extensively discussed, is still unclear. However, the increase in the exercise testosterone response induced by training should lead to another look at the actual role of this hormone. The increase in MVO_2, lactic anaerobic threshold, and end exercise circulating free fatty acids that was observed in Fellmann's study (1985) are in relation to an increase in muscular oxidative capacity lipid utilization. The increase in serum testosterone may play a role in these bioenergetics and metabolic phenomena (Kuoppasalmi et al., 1985). Since testosterone increases muscle glycogen synthesis by stimulating glycogen synthase, the increase in serum testosterone might reduce the glycogenolysis induced by cortisol during

exercise (Newsholme et al., 1983). On the other hand, the increase in serum testosterone could act against the proteolysis effect of glucocorticoids since testosterone increases muscle protein synthesis.

Thus, it is evident that the effects of strenuous physical activity on testosterone levels are controversial. Instances have been quoted where serum testosterone levels decreased after short bouts of activity (Lamb et al., 1975; Dessypris et al., 1976). However, the findings of this study support those of several other authors who observed increases in the levels of testosterone after varying degrees of physical exercise (Galbo et al., 1977; Sutton et al., 1978).

CONCLUSIONS

It is concluded that exercise induces an increase in serum testosterone levels in both trained and untrained subjects. Also, since it was evident that exercise had no effect on LH, FSH or prolactin levels, it was concluded that the aforementioned hormones did not play a role in the increased levels of testosterone.. The increased levels were either due to hemoconcentration, reduced metabolic clearance rate, increased catecholamine levels, actual stimulation of testicular production or a combination of some or all of the above. In addition, trained subjects had lower resting levels of testosterone and a greater testosterone response to exercise as compared to untrained subjects. This is possibly due to both psychological and endocrinological reasons.

BIBLIOGRAPHY

Aakvaag, A., & Sand, T. (1978). Hormonal changes in serum in young men during prolonged physical strain. *European Journal of Applied Physiology, 39*, 283-291.

Adlercreutz, H., & Harkonen, M. (1986). Effects of training on plasma anabolic and catabolic steroid hormones and their response during physical exercise. *International Journal of Sports Medicine, 7*, 27-28.

Adlercreutz, H., & Kosunen, K. (1976). Physical activity and hormones. *Advances in Cardiology, 18*, 144-157.

Amersham International plc, (1989). Amerlex-M FSH RIA Kit. Great Britian: Amersham.

Amersham International plc, (1989). Amerlex-M LH RIA Kit. Great Britian: Amersham.

Aono, T., & Kurachi, K. (1976). Influence of surgical stress under general anaesthesia on serum gonadotrophin levels in male and female patients. *Journal of Clinical Endocrinology and Metabolism, 42*, 144-149.

Ayers, J.W.T., & Komesu, Y. (1985). Anthropomorphic, hormonal and psychologic correlates of serum quality in endurance-trained male athletes. *Fertility and Sterility, 43*, 915-921.

Baker, H.W.G., & Romani, T. (1975). Rhythms in the secretion of gonadotropin and gonadal steroids. *Journal of Steroid Biochemistry, 6*, 793-801.

Bardin, C.W., & Ansbacher, R. (1978). Pituitary-testicular axis. *In Yen and Jaffe (eds.) Reproductive Endocrinology*, 110-125.

Bartsch, W., & Voigt, K.D. (1984). Endocrine aspects of ageing in the male. *Maturitas, 6*, 243-251.

Bliss, E.L., & Frischat, A. (1972). Brain and testicular function. *Life Sciences, 11*, 231-232.

Bottecchia, D., & Bordin, D. (1987). Effects of different kinds of physical exercise on the plasmatic testosterone level of normal adult males. *Journal of Sports Medicine, 27*, 1-5.

Brisson, G.R., & Volle, M.A. (1979). Blood testosterone (bound and free), Delta^4-androstenedione, FSH, LH and PRL in trained male athletes submitted to light (55%), medium (70%) and heavy (85% of MVO_2) bicycle work loads. *Fourth International Symposium of Biochemistry on Exercise*, 5.

Bunt, J.C., & Martino, R. (1986). Hormonal alterations due to exercise. *Sports Medicine, 3*, 331-345.

Cadoux-Hudson, T.A., & Few, J.D. (1985). The effect of exercise on the production and clearance of testosterone in well trained young men. *European Journal of Applied Physiology, 54*, 321-325.

Cartensen, H., & Tarner, N. (1972). Testosterone, luteinizing hormone and growth hormone in blood following surgical trauma. *Acta Chiropractica Scandinavia, 138*, 1-5.

Catt, K.J., & Pierce, J.G. (1978). Gonadotropic hormones of the adenohypophysis. In *Yen and Jaffe (eds.) Reproductive Endocrinology*, 34-62.

Christensen, S.E., & Imms, F.J. (1985). Body temperature elevation, exercise and serum prolactin concentrations. *Acta Endocrinologica, 109*, 458-462.

Cook, N.J., & Read, G.F. (1986). Changes in adrenal and testicular activity monitored by salivary sampling in males throughout marathon runs. *European Journal of Applied Physiology, 55*, 634-638.

Coppage, D., & Cooner, T. (1965). Testosterone in human plasma. *New England Journal of Medicine, 273*, 902-905.

Cumming, D.C., & Quigley, M.E. (1983). Acute suppression of circulating testosterone levels by cortisol in men. *Journal of Clinical Endocrinology and Metabolism, 57*, 671-673.

Cumming, D.C., & Yen, S.C. (1986). Reproductive hormone increases in response to acute exercise in men. *Medicine and Science in Sports and Exercise 18*, 369-373.

Cumming, D.C., & Yokama, T. (1987). Reproductive hormone responses to resistance exercise. *Medicine and Science in Sports and Exercise, 19*, 234-238.

De Lignieres, B., & Plas, J.N. (1976). Sécrétion testiculaire d'androgènes après effort physique prolongé chez l'homme. *Nouveau Press de Medecine, 5*, 2060-2064.

Delitala, G., & Tomasi, P. (1987). Prolactin, growth hormone and thyrotropin-thyroid hormone secretion during stress states in man. *Baillière's Clinical Endocrinology and Metabolism, 1*, 391-404.

Dessypris, A., & Adlercreutz, H. (1976). Plasma cortisol, testosterone, androstenedione and LH in a non-competitive marathon run. *Journal of Steroid Biochemistry, 6*, 33-37.

Dessypris, A., & Adlercreutz, H. (1984). Serum, total/free testosterone and sex hormone binding globulin binding capacity in a non-competitive marathon run. *Acta Endocrinologica, 265*, 18-19.

Diagnostic Products Corporation (1988). Coat-A-Count Total Testosterone. Diagnostic Products Corporation.

Diamond, P., & Brisson, G.R. (1989). Trait anxiety, submaximal physical exercise and blood androgens. *European Journal of Applied Physiology, 58*, 699-704.

Eik-Nes, K.B., & Candas, B. (1964). On the relationship between testicular blood flow and the secretion of testosterone. *Canadian Journal of Physiology and Pharmacology, 42*, 671-677.

Eik-Nes, K.B., & Peronnet, F. (1969). An effect of isopreterenol on rates of synthesis and secretion of testosterone. *American Journal of Physiology, 217*, 1764-1770.

Elias, A.N., & Szekeres, A. (1983). GABAergic and dopaminergic mechanisms in gonadotropin secretion in males-effects of baclofen and metoclopramide. *Acta Endocrinologica, 103*, 451-456.

Elias, A.N., & Fairshter, R. (1989). B-endorphin/B-lipoprotein release and gonadotropin secretion after acute exercise in physically conditioned males. *European Journal of Applied Physiology, 58*, 522-527.

Fahey, T., & Rolph, R. (1976). Serum testosterone, body composition and strength of young adults. *Medicine and Science in Sports and Exercise, 8*, 31-34.

Faiman, C.J., & Winter, J.S.D. (1971). Diurnal cycles in plasma FSH, testosterone and cortisol in men. *Journal of Clinical Endocrinology and Metabolism, 33*, 186-192.

Fellmann, N., & Coudert, J. (1985). Effects of endurance training on the androgenic response to exercise in man. *International Journal of Sports Medicine, 6*, 215-219.

Frey, M.A., & Doerr, B.M. (1983). Exercise training, sex hormones and lipoprotein relationships in men. *Journal of Applied Physiology, 54*, 757-762.

Galbo, H., & Hummer, L. (1977). Thyroid and testicular hormone responses to graded and prolonged exercise in man. *European Journal of Applied Physiology, 36*, 101-106.

Galbo, H., & Petersen, I.B. (1981). Endocrinology and metabolism in exercise. *International Journal of Sports Medicine, 2*, 203-211.

Gawel, M.J., & Park, D.M. (1979). Exercise and hormonal secretion. *Postgraduate Medical Journal, 55*, 373-376.

Genuth, S., & Rose, C. (1983). Endocrinology. In *Berne and Levy (eds.) Physiology*, 893-1114.

Guezennec, C.Y., & Ferre, P. (1982). Effect of prolonged exercise and fasting upon plasma testosterone levels in rats. *European Journal of Applied Physiology, 49*, 159-168.

Hackney, A.C., & Sinning, W.E. (1986). The effects of wrestling training on reproductive hormones. *Medicine and Science in Sports and Exercise, 18*, 196.

Hackney, A.C., & Sharp, R.L. (1988). Reproductive hormonal profiles of endurance-trained and untrained males. *Medicine and Science in Sports and Exercise, 20*, 60-65.

Hackney, A.C., & Runyan, W.S. (1989a). Endurance training and testosterone levels. *Sports Medicine, 8*, 117-127.

Hackney, A.C., & Ness, R.J. (1989b). Relationship of resting prolactin and testosterone in males during intensive training. *British Journal of Specialist Medicine, 23*, 194.

Hakkinen, K., & Pakarinen, A. (1985). Serum hormones during prolonged training of neuromuscular performance. *European Journal of Applied Physiology, 53*, 287-293.

Hakkinen, K., & Pakarinen, A. (1987). Relationships between training volume, physical performance capacity and serum hormone concentrations during prolonged training in elite weight lifters. *International Journal of Sports Medicine, 8*, 61-65.

Hakkinen, K., & Pakarinen, A. (1988). Daily hormonal and neuromuscular responses to intensive strength training in 1 week. *International Journal of Sports Medicine, 9*, 422-428.

Hakkinen, K., & Keskinen, K.L. (1989). Serum hormone concentrations during prolonged training in elite endurance-trained and strength-trained athletes. *European Journal of Applied Physiology, 59,* 233-238.

Hybritech Incorporated (1989). Tandem-R Prolactin Immunoradiometric assay. San Diego: Hybritech.

Jezová, D., & Tatar, P. (1981). Testosterone response to exercise during blockade and stimulation of adrenergic receptors in man. *Hormone Research, 15,* 141-147.

Jezová, D., & Vigas, M. (1985). Plasma testosterone and catecholamine responses to physical exercise of different intensities in men. *European Journal of Applied Physiology, 54,* 62-66.

Judd, S.J., Rakoff, J.S., & Yen, S.S. (1978). Inhibition of gonadotropin and prolactin release by dopamine: effects of endogenous levels. *Journal of Clinical Endocrinology and Metabolism, 47,* 494-498.

Kaufman, F.L., & Mills, D.E. (1985). Prolactin responses to chronic exercise in males. *Proceedings of the Society for Experimental Biology and Medicine, 179,* 546-548.

Keizer, H., & Janssen, G.M.E. (1989). Changes in basal plasma testosterone, cortisol, and dehydroepiandrosterone sulfate in previously untrained males and females preparing for a marathon. *International Journal of Sports Medicine, 10,* 139-145.

Kindermann, W., & Schnabel, A. (1982). Catecholamines, growth hormone, cortisol, insulin and sex hormones in anaerobic and aerobic exercise. *European Journal of Applied Physiology, 49,* 389-399.

Kuoppasalmi, K., & Naveri, H. (1976). Effect of strennuous anaerobic running exercise on plasma GH, cortisol, LH, prolactin, testosterone, estrone and estradiol. *Journal of Steroid Biochemistry, 7,* 823-829.

Kuoppasalmi, K., & Adlercreutz, H. (1980a). Plasma cortisol, androstenedione, testosterone and luteinizing hormone in running exercise of different intensities. *Scandinavian Journal of Clinical Laboratory Investigations, 40*, 403-409.

Kuoppasalmi, K., & Naveri, H. (1980b). Plasma testosterone and sex hormone binding globulin capacity in physical exercise. *Scandinavian Journal of Clinical Laboratory Investigations, 40*, 411-418.

Kuoppasalmi, K., & Adlercreutz, H. (1985). Interaction between catabolic and anabolic steroid hormones in muscular exercise. In *Fotherly, K. and Pal, S.B. (eds.) Exercise Endocrinology*, 65-156.

Lamb, D.R., & Beitins, I.Z. (1975). Androgens and exercise. *Medicine and Science in Sports, 7*, 1-5.

Levin, J., & Lloyd, C.W. (1967). The effect of epinephrine on testosterone production. *Acta Endocrinologica, 55*, 184-192.

Lipsett, M.B., & Schork, M.A. (1978). Steroid hormones. In *Yen and Jeffe (eds.) Reproductive Endocrinology*, 80-92.

MacConnie, S.E., & Barkan, A. (1986). Decreased hypothalamic gonadotropin-releasing hormone secretion in male marathon runners. *New England Journal of Medicine, 315*, 411-417.

Mathur, D.N., Toriola, A.L., & Dada, O.A. (1986). Serum cortisol and testosterone levels in conditioned male distance runners and non-athletes after maximal exercise. *Journal of Sports Medicine, 26*, 245-250.

McColl, E.M., & Wheeler, G.D. (1989). The effects of acute exercise on pulsatile LH release in high-mileage male runners. *Clinical Endocrinology, 31*, 617-621.

McConnell, T.R., & Sinning, W.E. (1984). Exercise and temperature effects on human sperm production and testosterone levels. *Medicine and Science in Sports and Exercise, 16*, 51-55.

McGrady, A.V., & Bittel, J. (1984). Effects of psychological stress on male reproduction. *Archives of Andrology, 13*, 1-7.

Métivier, G. (1983). The effects of an acute exercise bout on the serum level of testosterone and luteinizing hormone in male subjects above forty years of age. *Biochemistry of Exercise*, 667-671.

Métivier, G. (1985). Pituitary and gonadal secretory variations and control mechanism during physical exercise. *Journal of Sports Medicine, 25*, 18-26.

Métivier, G., Gauthier, R., & Grymala, D. (1980). The effect of acute exercise on the serum levels of testosterone and luteinizing hormone in human male athletes. *Journal of Sports Medicine and Physical Fitness, 20*, 235-238.

Moretti, C., & Fabbri, A. (1983). Naloxone inhibits exercise-induced release of PRL and GH in athletes. *Clinical Endocrinology, 18*, 135-138.

Mueller, G.P., & Simpkins, J. (1976). Differential effects of dopamine agonists and haloperidol on release of prolactin, thyroid stimulating hormone, growth hormone and luteinizing hormone in rats. *Neuroendocrinology, 20*, 121-135.

Naftolin, F., & Judd, H.L. (1973). Pulsatile patterns of gonadotropins and testosterone in man; the effects of clomiphene with and without testosterone. *Journal of Clinical Endocrinology and Metabolism, 36*, 285-288.

Nash, H.L., & Casey, J. (1987). Can exercise suppress reproductive hormones in men? *The Physician and Sportsmedicine, 15*, 180-189.

Newsholme, E.A., & Leech, A.R. (1983). Biochemistry of Exercise. J. Wiley and Sons inc., New York, 1983.

Opstad, P.K., & Aakvaag, A. (1982). Decreased serum levels of oestradiol, testosterone and prolactin during prolonged physical strain and sleep deprivation, and the influence of a high calory diet. *European Journal of Applied Physiology, 49*, 343-348.

Remes, K., & Kuoppasalmi, K. (1979). Effect of long-term physical training on plasma testosterone, androstenedione, luteinizing hormone and sex hormone binding globulin capacity. *Scandinavian Journal of Clinical and Laboratory Investigations, 39*, 743-749.

Rogol, A.D., & Veldhuis, J.D. (1984). Pulsatile secretion of gonadotrophins and prolactin in male marathon runners. *Journal of Andrology, 5*, 21-27.

Rolandi, E., & Reggiani, E. (1985). Comparison of pituitary responses to physical exercise in athletes and sedentary subjects. *Hormone Research, 21*, 209-213.

Rowell, L.B., & Copeland, T. (1974). Human cardiovascular adjustments to exercise and thermal stress. *Physiological Reviews, 54*, 75-159.

Rowland, T.W., & Mayer, L. (1987). Serum testosterone response to training in adolescent runners. *Sports Medicine, 141*, 881-883.

Schmid, P., & Pusch, H.H. (1982). Serum FSH, LH and testosterone in humans after physical exercise. *International Journal of Sports Medicine, 3*, 84-89.

Semple, C.G., & Thomson, J.A. (1985). Endocrine responses to marathon running. *British Journal of Sports Medicine, 19*, 148-151.

Skierska, E., & Ustupska, J. (1976). Effect of physical exercise on plasma cortisol, testosterone and growth hormone levels in weight lifters. *Endokrynol Pol, 27*, 159-165.

Strauss, R.H., & Lanese, R.L. (1985). Weight loss in amateur wrestlers and its effect on serum testosterone levels. *JAMA, 254*, 3337-3338.

Sutton, J.R., & Coleman, M.J. (1973). Androgen responses during physical exercise. *British Medical Journal, 165*, 520.

Sutton, J.R., & Coleman, M.J. (1978). Testosterone production rate during exercise. In *Landry F., Orban, W.A.R. (eds.) Proceedings of the third Symposium on the Biochemistry of Exercise, 3*, 227-234.

Toriola, A.L., & Mathur, D.N. (1986). Serum electrolytes and sex hormones in male sprinters after submaximal exercise. *Journal of Sports Medicine, 26*, 5-7.

Urhausen, A., & Kindermann, W. (1987a). A 7-week follow-up study of the behavior of testosterone and cortisol during the competition period in rowers. *European Journal of Applied Physiology, 56*, 528-533.

Urhausen, A., & Kullmer, T. (1987b). Behavior of testosterone, sex hormone binding globulin, and cortisol before and after a triathlon competition. *International Journal of Sports Medicine, 8*, 305-308.

Van Beaumont, W. (1972). Evaluation of hemoconcentration from hematocrit measurements. *Journal of Applied Physiology, 32*, 712-713.

Veldhuis, J.D., & Rogol, A.D. (1986). Spectrum of the pulsatile characteristics of LH release in normal men. *Journal of Andrology, 7*, 83-92.

Vogol, R.B., & Books, C.A. (1985). Increase of free and total testosterone during submaximal exercise in normal males. *Medicine and Science in Sports and Exercise, 17*, 119-123.

Weiss, L.W., & Cureton, K.J. (1983). Comparison of serum testosterone and androstenedione responses to weight lifting in men and women. *European Journal of Applied Physiology, 50*, 413-419.

Wheeler, G.D., & Wall, S.R. (1984). Reduced serum testosterone and prolactin levels in male distance runners. *JAMA, 252*, 514-516.

Wilkerson, J.E., & Horvath, S.M. (1980). Plasma testosterone during treadmill exercise. *Journal of Applied Physiology, 49*, 249-253.

Williams, R.H., & Gutin, B. (1974). Textbook of Endocrinology, WB Saunders co., Philadelphia.

Wuttke, W., & Bradley, A. (1976). Neuroendocrine mechanisms in reproductive physiology. *Review of Physiology, Biochemistry and Pharmacology, 76*, 59.

Young, R.J., & Ismail, A.H. (1979). Prediction of serum testosterone before and after an exercise program using physiological and personality variables. *Journal of Human Ergology, 8*, 29-38.

APPENDIX A

CHAPTER 1

INTRODUCTION

With the growing popularity of fitness and mass participation in physical activity in today's society, it is becoming clear that exercise is a facet of life which must be understood more clearly.

Some research has been done on the effects of training and exercise on such hormones as testosterone, luteinizing hormone (LH), follicle stimulating hormone (FSH), and prolactin in males. However, this area is still not well understood and there is still much confusion and contradiction when comparing the conclusions of various researchers (Lamb et al., 1975; Hackney et al., 1986).

One of the reasons why attention is being focused on these hormones is because they play a vital role in relation to exercise. This role includes testosterone's involvement in protein synthesis, muscle strength and aggressiveness (Hackney et al., 1989a). These factors are very important, especially in competitive events. Another reason why attention was being focused on these hormones (especially testosterone) was the fact that it was thought that endurance exercise could possibly cause infertility in males (Nash et al., 1987; Wheeler et al., 1984).

Responses of testosterone, LH, FSH and prolactin to exercise (in males) have varied quite extensively from study to study (see table 1). Some studies have shown that testosterone levels decrease as a result of exercise (Aakvaag et al., 1978; Opstad et al., 1982; Ayers et al., 1985; Semple et al., 1985; Urhausen et al., 1987a; Nash et al., 1987). Other studies have shown an increase in testosterone levels due to exercise (Cadoux-Hudson et al., 1985; Métivier et al., 1980; Kuoppasalmi et al., 1980a; MacConnie et al., 1986; Vogel et al., 1985; Diamond et al.,

1989). Others, on the other hand, have observed no changes at all (Lamb et al., 1975; Bottechia et al., 1987).

There seems to be conflicting results in the literature concerning exercise and testosterone response. However, in the studies where testosterone levels decreased, endurance exercise was performed by the subjects. In the study by Semple et al. (1985), subjects ran a marathon and it was found that testosterone levels in the subjects were lower than at the beginning of the race. In studies where testosterone increased, the exercises performed were of short to moderate duration and more intense. However, the aforementioned type of exercise does not necessarily lead to an increase in testosterone levels. In the case of Bottecchia et al. (1987), short-term, intense exercise lead to no changes in testosterone levels.

Studies have shown that LH levels may remain unchanged (Sutton et al., 1973; Metivier et al., 1980; Galbo et al., 1977), or increase (Kuoppasalmi et al., 1976) or decrease (Dessypris et al., 1976). The majority of the studies seem to indicate that LH levels are unaffected by exercise. In addition, it has been shown that FSH levels remain unchanged (Gawel et al., 1979; Vogel et al., 1985) or decrease (Brisson et al., 1979) but most of the studies indicate that FSH levels remain unchanged due to exercise. Most researchers have found that prolactin increases with exercise (Christensen et al., 1985; Kaufman et al., 1985; Delitala et al., 1987) but some researchers have found no change (Toriola et al., 1986; Rolandi et al., 1985).

Long-term training or training programs have also been seen to have an effect on reproductive hormone levels in males. It has been found that training can decrease testosterone levels, especially basal levels (Hackney et al., 1988; McColl et al., 1989; Hakkinen et al., 1988). In addition, it has been shown that trained subjects have a larger

testosterone response to exercise than non-trained subjects (Dessypris et al., 1976; Fellmann et al., 1985; Remes et al., 1979). In one case, however, it was found that non-trained subjects had a larger response than trained subjects (Mathur et al., 1986).

Thus, there seems to be conflicting results in the literature. This is due to the fact that these hormonal responses, especially those of testosterone, are dependent on the type and duration of the exercise, the workload and the physical fitness of the subject (Galbo et al., 1981).

Purpose

The purpose of this study is to compare the effects of an exercise intensity equivalent to 70% of maximal oxygen consumption (MVO_2) on plasma testosterone, LH, FSH and prolactin levels. Another purpose is to investigate the effects of LH, FSH, Prolactin and physical fitness on testosterone levels.

Hypothesis

It is hypothesized that an exercise intensity corresponding to 70% of the subject's MVO_2 will elevate plasma levels of testosterone and that this elevation will be independent of the influences of LH, FSH and prolactin. It is also hypothesized that increased physical fitness will lower basal levels of testosterone and also increase the testosterone response to exercise.

Definitions

Acute exercise : Running on a treadmill at 70% of MVO_2

Plasma levels of hormones : Measured concentrations of hormones taken from blood samples

Untrained : Maximal oxygen consumption is less or equal to 45 ml O_2/kg/min and subjects are sedentary.

Trained : Maximal oxygen consumption is greater or equal to 55 ml O_2/kg/min and are amateur athletes.

Limitations

Limitations of this study include the accuracy and/or reliability of the protocol used to find MVO_2 and also of the gas analysers. In addition, the kits used to determine the concentrations of testosterone, LH, FSH and prolactin are accurate but there is some error involved (approximately 5%). The accuracy of the sport-tester (heart rate monitor) also has to be questioned since outside signals can interfere with it.

In terms of the whole study, the results will only be able to be extrapolated to a similar population of subjects (i.e. white males, between the ages of 19 and 28).

APPENDIX B

CHAPTER 2

REVIEW OF LITERATURE

Hypothalamic-pituitary-testicular axis

In males, blood testosterone levels normally range from 3.0 to 11.0 ug/L (Bartsch et al, 1984). The majority of the circulating testosterone comes from production in the interstitial cells of Leydig at the testes. In addition, small amounts (less than 5%) are produced by the zonas reticularis and fasciculata of the adrenal gland and also in the peripheral tissue of the body (Hackney et al., 1989a).

Regulation of testicular production occurs through a negative feedback loop mechanism which involves the anterior pituitary gland and the hypothalamus; this is referred to as the "hypothalamic-pituitary-testicular axis" (Williams et al., 1974).

The hypothalamus periodically releases pulses of gonadotrophin-releasing hormone (GnRH) into the hypophyseal circulation which supplies the hypothalamus and the anterior pituitary gland. The GnRH stimulates the anterior pituitary to produce and release luteinizing hormone (LH) and follicle stimulating hormone (FSH). Because of the GnRH pulsatile release, LH and FSH are also released into the systemic circulation in a pulsatile fashion. In a healthy male, 2 to 4 LH and FSH pulses are observed over a 6 to 8 hour period, but the amplitude of the LH pulses are much greater than FSH (Baker et al., 1975; Veldhuis et al., 1986). At the testes these circulating hormones interact with their target tissue (LH, Leydig cells; FSH, Sertoli cells) receptors located on the respective cell membranes. Once the hormone-receptor complex is formed, there is an adenyl cyclase-mediated increase of cyclic AMP which results in phosphorylation of intracellular proteins by activation of a protein

kinase mechanism. At the Leydig cells this leads to a mobilization of steroid precursors, in particular the activation of pregnenolone (the parent compound of testosterone) synthesis from cholesterol. Another pituitary hormone, prolactin, acts in a synergistic way to potentiate the effects of LH at the Leydig cells. Conversely, the FSH receptor-hormone formation at the Sertoli cells results in an initiation of the spermatogenesis process (Catt et al., 1978).

Newly synthesized testosterone diffuses from the Leydig cells into the testicular vascular system and/or into adjacent testicle compartments containing the Sertoli cells. In the Sertoli cells, testosterone plays an essential role in the facilitation of the spermatogenesis process. The pulsatile release of LH results in some fluctuations of testosterone levels in the circulation. Furthermore, there appears to be a circadian cycle imposed where large nocturnal elevations in testosterone are observed (Bardin et al., 1978).

The majority (approximately 97%) of circulating testosterone is transported bound to carrier or binding proteins. The principal binding protein is sex steroid-binding globulin (SSBG). The remaining 3% of the circulating testosterone exists in an unbound form referred to as free testosterone. The free testosterone is considered to be the biologically active form of the hormone (Bardin et al., 1978; Lispett et al., 1978). Circulating bound and free testosterone are collectively known as "total testosterone".

Role of testosterone

Testosterone performs several functions, including : stimulation of protein anabolism, somatic growth and ossification; induction and maintenance of differentiation of somatic tissues; induction of development of secondary sexual characteristics; aid in development and maintenance of accessory sex organs; support of testicular

spermatogenesis; influence on both sexual and aggressive behavior; and control of mass and strength of skeletal muscle (Hackney et al., 1989a). Testosterone also aids in increased glycogen synthesis and storage (Guezennec et al., 1982).

Production and secretion of testosterone

Testicular testosterone production in the average male is approximately 7000 ug/day (Hackney et al., 1989a). As stated, LH and testosterone levels are directly related, and as elevations in LH occur, testosterone production increases. Reductions in substrate (i.e. cholesterol or pregnenolone) availability, as with any chemical reaction, can produce a lower synthesis rate of testosterone (Newsholme et al., 1983). Eik-Nes et al. (1964 and 1969) also demonstrated in animals that elevations in circulating catecholamines can lead to increases in testosterone levels independent of LH changes. However, catecholamine concentrations in excess of physiological ranges may be necessary to produce this effect.

Testosterone secretion is principally affected by testicular blood flow since testosterone is essentially fat soluble (i.e. freely diffusable) and the testes has little or no storage capacity for testosterone. Testicular blood flow is a function of the levels of vascular vasoconstriction or vasodilation. Thus, circulating catecholamine or other vascular effectors, as well as direct sympathetic-parasympathetic tone, interact to affect testosterone secretion (Eik-Nes et al., 1964; Levin et al., 1967).

Metabolic clearance of testosterone

The clearance rate of testosterone has been estimated at being approximately 1100 L/day (Hackney et al., 1989a). The degradation process involves the conversion of testosterone into 17-ketosteroid and glucuronide which are excreted in the urine (Genuth et al., 1983).

Changes in hepatic blood flow result in the same changes in the removal rate of testosterone (Lispett et al., 1978).

The observed changes in blood levels of a hormone may not necessarily reflect alterations in production, secretion or metabolic clearance rates. Due to the binding of testosterone to carrier proteins, increases or decreases in plasma volume leading to haemoconcentration alterations can also produce significant changes in hormone concentrations which are not representative of changes in turnover rate.

Effect of exercise on LH, FSH and prolactin

Prolactin, which previously has been said to increase testosterone production through the potentiation of LH, has been shown to increase due to exercise (Gawel et al., 1979). In this study, eleven healthy male volunteers were exercised on a Monarch bicycle ergometer at 15, 30 and 40% of their maximum work load, while pedalling at 20 km/hr for successive periods of 10 minutes. This resulted in a significant rise in prolactin levels in the volunteers. In a study done by Kaufman et al. (1985), eight healthy male subjects, age 21 to 26 years, performed cycle ergometer exercise at 50% of their maximal oxygen uptake, on ten consecutive days in an environmental chamber. On days 1, 5, and 10 the exercise was 45 minutes in duration. On the remaining days subjects exercised for 90 minutes. It was found that acute exercise on day 1 significantly increased plasma prolactin from 8.6 ng/ml to 14.1 ng/ml. However, chronic exercise at a similar work load failed to elicit prolactin increases (i.e. with repeated exercise at a constant intensity, the plasma prolactin response attenuated within 5 days).

In the study by Kaufman et al., the intensity of the exercise was constant, with only the duration varying on the test days. In addition, environmental conditions were held constant throughout the study. In other studies, the presence of stressful stimuli were not completely

removed thus providing a stimulus to prolactin secretion above that of the chronic exercise. For example, Moretti et al. (1983) reported a 13.4 ng/ml rise in plasma prolactin in professional athletes following 20 minutes of cycle ergometer exercise at 80% of their maximal heart rate. This intensity and duration of exercise is very stressful and was more demanding than the regular exercise program of the subjects. Thus, the stress of an unusually intensive exercise session may have accounted for the prolactin response in this study. Similarly, Rogol et al. (1984) examined post-exercise serum prolactin levels in marathon runners and found that prolactin tended to be higher post-exercise than in untrained controls. Those subjects who did have increased prolactin levels had exercised by running at a fast pace in adverse weather. Thus, the environmental stress rather than chronic exercise was most likely responsible for any prolactin changes.

Christensen et al. (1985), put 10 healthy males on bicycle ergometers at 450 kpm/min for 40 minutes and found that prolactin levels increased significantly. This is in contrast to a study done by Toriola et al. (1986), where subjects (nine top male sprinters) participated in a submaximal exercise which involved cycling a bicycle ergometer at a rate of 600 kpm/min for 15 minutes. In this case, prolactin plasma levels were insignificantly altered due to exercise.

LH and FSH, which have also been discussed as being involved in stimulating the production of testosterone, are affected by exercise in several ways. For instance, nine spinters (22-26 years old) from the University of Ife volunteered for a study done by Toriola et al. (1986). Subjects participated in a submaximal exercise which involved cycling a bicycle ergometer at a rate of 600 kpm/min for 15 minutes. The exercise was performed at about 75% of the subject's heart rate. Blood samples were taken under basal conditions and one minute after the end of the

exercise. It was found that concentrations of FSH and LH (also prolactin) were insignificantly altered due to exercise. However, slight reductions in serum levels of LH (-1.7%) and an increase of FSH (+3%) were noted. Serum prolactin showed a near significant decline of 12.5%. This decrease in prolactin concentration to near significant level is in contrast with the increase described by Galbo et al. (1981). The underlying mechanism for this reduction in prolactin level is uncertain, but increased levels of dopamine during stress may be a cause of reduced prolactin levels (Mueller et al., 1976).

In a study done by MacConnie et al. (1986), subjects included 6 highly trained male runners who usually ran 125 to 200 km per week for at least five years and a control group of 13 healthy age-matched men who ran no more than five km per week. The exercise used in this study consisted of a five minute warm-up on a treadmill at 6.5 km/hr, 0% grade. Subjects then ran for two hours while the speed and grade of the treadmill were adjusted to elicit a heart rate and oxygen consumption equivalent to 70-75% of the subject's maximal oxygen consumption (MVO_2). Comparison of hormonal levels before and after exercise showed no significant difference in luteinizing hormone pulse frequency or amplitude.

Métivier et al. (1983) found results similar to those of MacConnie et al. (1986) but using a different protocol. Metivier had 17 male volunteers with a mean age of 50.3 years perform an exercise which consisted of walking on a motor-driven treadmill according to the protocol developed by Bruce et al. (1981) until 90% of predicted maximal heart rate was reached. Luteinizing hormone increased from 10.7 miu/ml to 11.4 miu/ml after the work bout. This change, however, was not statistically significant. This was found to be the same case as in younger males (Métivier et al., 1980).

Studies performed by McColl et al. (1989) included 6 runners (28-32 years) training at least 80 km per week and 6 age-matched sedentary controls. Subjects ran on a treadmill at 5% below their ventilatory threshold for 60 minutes. This strenuous acute exercise was unable to induce significant changes in the amount of LH released or the frequency at which LH is released. Similar results were found in a study by Kuoppasalmi et al. (1980a). Ten male subjects (5 sprinters and 5 long-distance runners) between the ages of 20 and 26 years old volunteered for this study. The sprinters performed short-term exercises (i.e. three subjects ran 20m, 40m and 60m, resting 30 seconds between each run, and two subjects ran 3 x 40m, resting five minutes between each run) while the long-distance runners performed long-term exercises (i.e. running 21km in approximately 90 minutes and running 13-14 km in 45 minutes). Mean plasma LH did not alter significantly during short-term exercise and, in addition, no significant changes occurred during long-term exercise. However, 30 minutes after long-term exercise the mean plasma LH level decreased significantly (42%) from the preceding levels. One hour after the runs the mean plasma LH had returned to the pre-exercise levels. Thus, no significant changes in plasma LH during running exercise were found in this study. However, some decrease in LH secretion would be expected, for the longer and more intense the exercise the greater the decrease in the blood flow to the liver and kidneys, which are the main sites of LH catabolism. Deceased plasma LH 30 minutes after both long-term runs could be due to negative feedback caused by the increased plasma levels of either oestradiol or testosterone during the exercise (Sutton et al., 1973). The decrease in LH was, as mentioned previously, followed by a compensatory rise during the second half hour. This return of LH to the pre-exercise level may be explained in the intense run (13-14 km for 45 minutes) by the withdrawal

of the factors inhibiting LH release, but the same finding after the moderate run (21 km for 90 minutes) is harder to explain. As GnRH release is probably regulated by catecholamine-containing nerve endings in the hypothalamus (Wuttke et al., 1976), functional changes in these nerve endings may be partly responsible for the changes in LH. However, there does not seem to be much information available in the literature concerning hypothalamic catecholamine levels in physical strain.

A similar decrease in LH levels was observed by Elias et al. (1989). Six male volunteers between the ages of 22 and 26 were exercised at 70% of their maximal oxygen consumption for 15 minutes on a treadmill. Following exercise there was a significant decrease in serum LH concentrations. Although it is possible that an increase in central opioidergic tone (catecholamines) following exercise could account for the post-exercise depression of LH concentrations, changes in the central concentrations of other neurotransmitters such as dopamine (Judd et al., 1978) and gamma-aminobutyric acid (Elias et al., 1983) must also be considered.

In contrast, Cumming et al. (1986) showed conflicting data. Five active but untrained male volunteers between the ages of 24 and 26 cycled at 60 revolutions/min on an electrically braked cycle ergometer. The load was incremental following the first three minutes at zero load. The load was increased by 25 W/min until the subject was no longer able to continue. A small but consistent rise in LH was seen immediately before exercise in all five subjects. There was a further increase during exercise to reach a mean level 50% above baseline value at 25-30 minutes. This increase in LH is in contrast to the decreases seen with McColl et al. (1989) and Elias et al. (1989). In addition, Cumming also observed significantly elevated levels of serum FSH, but only at five minutes after the beginning of the exercise.

Gawel et al. (1979) obtained results similar to those of Cumming regarding LH levels, but not FSH levels. Gawel obtained 11 healthy male volunteers between the ages of 18 and 38. The subjects were exercised on a Monark bicycle ergometer at 15, 30 and 40% of their maximal oxygen consumption, while pedalling at 20 km/hr for successive periods of 10 minutes. A small rise in plasma LH levels was observed towards the end of the exercise and no change was observed in plasma levels of FSH.

Schmid et al. (1982), observed different hormonal responses due to different types of exercise. For instance, in a 800m run performed by male sprinters, it was observed that FSH levels at the end of the run rose by 28.8% compared to basal levels. However, exhaustive bicycle ergometer and treadmill tests which were on average 13 minutes in duration caused significant decreases in plasma FSH levels. In addition, long-term exercise such as cross-country skiing events and bicycle ergometry tests which were about 90 minutes in duration caused no significant changes in FSH levels. Schmid also witnessed increases of plasma LH levels in the order of 24% and 49% in 800m runs and 300m sprints, respectively. Galbo et al. (1977) did not see any LH changes in eight students after 10 minutes of a maximal and submaximal bicycle ergometer test. In contrast, Brisson et al. (1979) found a decrease of the LH levels after 20 minutes of bicycle ergometry tests of varying intensity. In a long-term test in 14 highly trained rowers, Sutton et al. (1973) found nonsignificant decreases in LH levels by about 10% both after maximal and submaximal exertion of 60 minute duration. In contrast, 11 moderately trained cross-country skiers showed a nonsignificant increase in plasma LH levels by about 9% after a marathon run (Dessypris et al., 1976).

Rolandi et al. (1985) studied eight competitive volleyball players and eight sedentary healthy males, all between the ages of 21 and 31.

Exhaustive exercise testing was done using a bicycle ergometer, with work load starting at 50 W with 50 W increases every three minutes until exhaustion. No significant response was found in LH or FSH in either group. Similarly, Aakvaag et al. (1978) found LH and FSH levels in military recruits to be unaffected (i.e. responses were insignificant) by a rigorous 5 day training course. In addition, Vogel et al. (1985), exercised 10 healthy untrained males on a bicycle ergometer at 50% of their maximal oxygen consumption for 45 minutes and concluded that serum LH levels did not change significantly during the exercise or recovery periods. In addition, no significant changes were witnessed in FSH levels.

Thus, the numerous discrepancies reported in the literature on the response of gonadotrophins to exercise may be related not only to the differences in type, intensity and duration of exercise, but also to the interference by other extraneous factors such as drugs and anaesthesia (Toriola et al., 1986). In addition, it is important to realize that because of the pulsatile release of LH and FSH, it is difficult to evaluate exercise responses of gonadotropins.

Effect of training on testosterone levels and response

One of the first studies investigating testosterone responses to training was done by Remes et al. (1979). Remes' test group consisted of 39 army recruits which were examined during the first six months of their military service (ages ranged from 19 to 28 years old). The recruits took part in the physical training required of them during their military service. The average time spent at various physical activities increased from about 20 hours/week during the first ten weeks to 30-40 hours/week during the last months of the study. During the 6-month study period the mean plasma testosterone level increased, highly significantly, by 21%. The mean increase was found to be higher in the

well-conditioned group (28%) than in the poorly conditioned group. In the ten best conditioned subjects the mean increase was 43% which contrasted with the 13% increase in the ten worst conditioned subjects. Thus, the recruits who were better trained had a larger testosterone response to exercise.

Basal (resting) levels of testosterone are also affected by training, which was observed by Hackney et al. (1988). Two groups of subjects volunteered for this study; eleven subjects comprising the trained group (participants of endurance exercise training programs for over five years) and eleven in the untrained group (sedentary, except for normal daily activity). Resting levels of testosterone were measured over a span of four hours and it was discovered that the testosterone concentration was lower in trained subjects, being only 68.8% of the concentration found in the untrained group. There were no significant changes over time in testosterone concentrations for either of the groups; however, the mean concentrations of the trained group were less than those of the untrained group at each time point (every hour). It is important to realize that reduction of testosterone concentrations could result in suppressed bodily functions, as these androgens play vital roles in key physiological processes. Specifically, spermatogenesis and protein synthesis rely upon adequate levels of androgens to proceed appropriately (Genuth et al., 1983). Whether these low, but within normal range, androgen concentrations found in the trained athletes has an effect on the androgen-dependent process of the body is not certain (Coppage et al., 1965). However, in a study by Ayers et al. (1985), oligospermic conditions were found in two trained subjects having very low testosterone concentrations.

Unlike Hackney, Mathur et al. (1986) was unable to detect differences in testosterone resting levels between trained and untrained

subjects. Seven healthy and actively training male long distance runners (24 to 28 years) and five male non-athletes (22 to 26 years) volunteered to participate in Mathur's study. Subjects were exercised on a bicycle ergometer until exhaustion. The resting values of serum testosterone were within normal limits for all the participants. Also, in distance runners, the testosterone level increased significantly by 19.6% during the post-exercise period, but decreased by 3.9% after one hour. A similar pattern of changes was noted in non-athletes. Thus basal levels of testosterone and testosterone responses to exercise were quite similar between trained and untrained subjects in this study.

Fellmann et al. (1985) had results comparable to those of Mathur. Six healthy male volunteers between the ages of 31 and 39, were put on a training program that was 40 weeks in duration. This program consisted of pedalling, three sessions per week, on a mechanically braked Monark bicycle ergometer. Each session lasted 60 minutes and the exercises were performed at 80% to 85% of MVO_2. During the training course, the work load was regularly adjusted to this constant relative power. Training did not significantly change the resting level of plasma testosterone. In contrast, the values at the end of exercise were increased, passing from 18.8 nmol/L the week before training began to 23.8 nmol/L at the end of the program. The amplitude of the exercise response was thus increased, reaching its highest value at week 40 (4.8 nmol/L compared to 1.0 nmol/L before). The lack of significant change in the resting plasma levels of testosterone in the course of the training agrees with previous findings by Kuoppasalmi et al. (1980b) but conflicts with the results of Remes et al. (1979). However, the measurements taken by Remes et al. were performed during the first six months of the military service of the subjects, and the reported increases in basal levels of testosterone can be ascribed to an adaptation to military life.

Inversely, Frey et al. (1983) reported a significant fall in the testosterone level after the tenth week of training in subjects, but these authors speculated that this "testosterone variation may be a transient reduction which would be reversed after a longer training program".

A study by Keizer et al. (1989) took 25 untrained male volunteers and put them through an 18-20 month training program during which training distance was gradually increased. The investigators observed that endurance training augmented basal testosterone levels, which is in line with the results of Kuoppasalmi and Adlercreutz (1985) and Remes et al. (1979) but disagrees with those of Fellmannn et al. (1985). This discrepancy may be caused by the small number of subjects in Fellmann's study and different experimental designs as well.

Hakkinen et al. performed two studies (1985 and 1989) where he found no training effects on testosterone. In his first study in 1985, he used 21 males between the ages of 22 and 28. The subjects went through a training period of 24 weeks, followed by a 12 week detraining period. The subjects were divided into two groups, where group A participated in heavy resistance strength training and group B participated three times a week in controlled strength training. In group A, the mean serum testosterone concentration was 25.9 nmol/L before the 24-week training period and 25.7 nmol/L after it, and no significant changes were noted at any time. In group B, the testosterone concentration was 28.9 nmol/L before the training program and 29.5 nmol/L at the end.

Hakkinen's other study in 1989 consisted of nine elite male swimmers, who had been training regularly for the past 8-10 years, and eight elite male weightlifters, who had been training for about the same duration. The study was one year in duration and the athletes followed their own personalized training program. At the end of the year, it was

observed that there were no statistically significant changes between the two groups in the concentrations of serum testosterone during the course of the year.

Wheeler et al. (1984), however, had results which conflicted with those of Hakkinen above. Wheeler's subjects consisted of 31 men running at least 64 km each week and 18 sedentary controls (18-46 years old). Resting levels of testosterone were found to be significantly lower in runners than in controls. The mean value for total testosterone in runners was 83.3% of that in the control group, but the non-sex hormone-binding globulin-bound and free testosterone values were 69.5% and 68.1% of mean values in control subjects, respectively. The lower testosterone levels in runners could reflect differences in clearance or production of the hormone because the non-specifically bound portions were reduced with no significant difference in serum binding.

Similarly, Ayers et al. (1985), noted a decrease in basal testosterone levels (4.2 ng/ml versus 6.9 ng/ml) before and after an endurance training program of one year duration. Twenty male subjects were used and their training consisted of running 40-80 miles per week. Hakkinen et al. (1988) also found decreases in basal testosterone levels, which contradicts his other studies (1985 and 1989). For this investigation he selected eight elite weightlifters who had been training for five years to participate in a one week weight-lifting training camp, which was described as a last stressful preparatory training week with high training loads and with a high overall training volume. It was observed that significant decreases took place in the mean morning values for serum testosterone during the training period and one of the possible reasons for this decrease in serum testosterone during the stressful training period could be its increased consumption during prolonged intensive muscular work.

Hackney et al. (1988) compared resting testosterone levels in 10 km runners, wrestlers, weight-lifters and sedentary subjects. All the athletes in the study had been training in their sport for at least five years. Not only was testosterone lower in the endurance runners than the controls, but it was also lower than that observed in the weight-lifters. Interestingly, the testosterone levels of the wrestlers, while not different from the endurance runners, was also lower than the other groups. This reduced testosterone in wrestlers is not an uncommon finding, and considering the extensive training and weight loss practices involved in wrestling training, agrees with the data for distance runners (Hackney et al., 1986; Strauss et al., 1985). Hackney et al. (1988) had ten moderately trained males perform eight weeks of intensive training and evaluated testosterone levels at two week intervals. These subjects exercised 90 minutes per day (cycle ergometry) performing continuous aerobic sessions (75% of MVO_2) one day per week and interval anaerobic sessions (85-200% MVO_2) four days per week. From weeks 2 through 6 testosterone was significantly (20-40%) reduced from pretraining values. By week 8 of training, however, testosterone levels had begun to return to the pretraining values.

Kuoppasalmi et al. (1985) found no differences in the resting testosterone levels of 16 long distance runners and 7 untrained subjects evaluated every 4-6 weeks over a nine month period corresponding to the athlete's training season. MacConnie et al. (1986) collected blood samples for eight hours at 30 minute intervals in six marathon runners who ran 125-200 km/week for at least five years and six marginally active controls (recreational exercisers). No difference was found in resting testosterone levels. Hackney et al. (1988) evaluated blood samples every 20 minutes for four hours in groups of distance runners (110 km/week, longer than 12 years) and sedentary controls. In contrast

to MacConnie et al.'s results, testosterone levels of the runners were approximately 70% of those in the control subjects throughout the sampling period.

The contradiction between the findings of these training studies may be a factor of the training stimulus and volume employed. That is, the studies finding significantly lowered testosterone levels used subjects who had been training for approximately 5 years or more at high volumes and were competitive endurance athletes. Another problem with these studies has been that the individuals in question have subjected themselves to the rigours of the training program for years and the investigator is observing the end result. Because of the wide variations in interindividual response to training, as well as forms of training, a great deal of variance is observed in the testosterone responses of these subjects. A final problem is the principal assumption made within these studies, that the observed effects on testosterone levels are primarily a function of the endurance training. Obviously, this assumption may not be completely valid unless such things as psychological stress, sleep loss, diet, weight loss, and hereditary factors, all of which affect testosterone levels, are controlled (McGrady et al., 1984; Opstad et al., 1983).

Effects of exercise on testosterone levels

To this day, there are not all that many studies that have been done on the effects of exercise on plasma testosterone levels. The studies that have been done range greatly in the methods (i.e. exercise, blood sampling, etc.) used and results.

A study done by Cumming et al. (1986) consisted of five active but untrained male volunteers who exercised at 60 revolutions per minute on an electrically braked cycle ergometer (acute exercise). The load was increased by 25 W/min until the subject was no longer able to continue.

A statistically significant increase in serum testosterone was seen in the sample taken immediately before exercise compared with baseline values. The pre-exercise rise represented a mean increase of 15% above baseline values and continued to a peak value of 30% above baseline at 20 minutes from the start of exercise. Levels then fell, returning quickly to baseline within 10 minutes following peak levels. LH was found to increase synchronously with testosterone but the exercise-associated increase in serum testosterone levels could not result from LH stimulation since it has been reported that the time interval between a spontaneous pulse of LH and the succeeding increment in testosterone exceeds 45 minutes (Naftolin et al., 1973).

Somewhat similar results were reported by Vogel et al. (1985). Ten healthy males were exercised for 45 minutes on a bicycle ergometer at a work load designed to elicit 50% of MVO_2 and it was found that the mean testosterone level increased significantly during exercise but the subjects demonstrated peak testosterone levels at different time intervals. Also, plasma testosterone levels decreased during recovery. In this case, Vogel was unable to attribute the testosterone increase to LH, FSH or prolactin since levels of these hormones did not change.

Using a different protocol, Métivier et al. (1980) drew the same conclusions. His protocol consisted of giving twelve subjects (average age was 19 years) an MVO_2 test on a treadmill, using five minute constant speed intervals while the elevation was set at 8.6% grade. In addition, the speed was increased by 2 MPH after each time interval. The major finding from this research was that there were significant differences in plasma testosterone levels before and immediately after the acute exercise. Also, there were no changes in serum LH levels. Hence, it was thought that the high testosterone level seen after the exercise was the result of increased secretion by gladular tissue and

the hemoconcentration effect of acute strenuous exercise. However, if the high testosterone level was solely the result of simple hemoconcentration, a similar non-significant increase would have been observed for LH levels. Thus, this is considered as evidence that secretion of testosterone increased during exercise.

These results support those of Sutton et al. (1973), who showed that during exercise the testosterone level increase was independent of serum LH. It also agrees with the results of Faiman et al. (1971) and Cartensen et al. (1972) that there is no relationship between testosterone secretion and LH in males engaged in muscular activity.

Métivier followed with another study in 1983 which agreed with the results of his 1980 study, but using different subjects and a different protocol. Seventeen male sujects with a mean age of 50.3 years volunteered for the study. The experimental protocol consisted of walking on a motor-driven treadmill according to the protocol developed by Bruce et al. (1981). The exercise continued until the subjects reached 90% of their maximal heart rate. Metivier found that testosterone levels rose significantly from 290.6 mg/100 ml to 360 mg/100 ml after the exercise, to fall significantly to 276.6 mg/100 ml sixty minutes later. The significant increase in testosterone during exercise represents a ninefold increase of normal daily output, or a 24% change which could not be explained entirely by a plasma volume change, as the hematocrit changed by only 8.8%. In addition, the drop in the plasma level of testosterone following exercise could have been the result of increased metabolic degradation or rapidly reversible binding to carrier protein (Plager et al., 1965; Sandberg et al., 1957).

Bottecchia et al. (1987) had results which contrasted with those of Metivier. In Bottecchia's study, five normal male medical students performed strenuous physical exercise on a treadmill that produced

exhaustion in 2-4 minutes and also performed an exercise which required 75% of their MVO_2 for 15, 30 and 60 minutes each. It was observed that an intense, rapidly exhausting (2-4 minutes) physical exercise produced an increase in the plasma level of testosterone. However, this increment was not statistically significant. Also, physical exertion corresponding to 75% of MVO_2 and prolonged over different time periods also caused an increase in testosterone levels but these changes were also not statistically significant. In addition, no changes were found in the plasmatic concentration of LH, which is consistent with the results of Métivier.

In a study done by Cadoux-Hudson et al. (1985), four oarsmen who were training for international events were tested in pairs. One subject of the pair would remain seated and the other exercised on a bicycle ergometer for one hour. During the first 10 minutes of exercise the work load was gradually increased until a heart rate of 160-165 beats/minute was reached which corresponded to 60% of their MVO_2. In all subjects the exercise caused a significant increase in the plasma concentration of testosterone. At the same time, metabolic clearance rate of testosterone, hepatic plasma flow and plasma volume were significantly decreased. The finding that a one hour period of vigorous exercise led to a modest (27%) increase in plasma testosterone concentration is consistent with the findings of others (Sutton et al., 1973; Métivier et al., 1980). However, this increase was due solely to the reduction in the rate of clearance of testosterone and not to any increase in the rate of production of testosterone.

Thus, the study by Cadoux-Hudson et al. seems to indicate a lack of increase, or even a slight fall in testosterone production. The authors hypothesized it was possible that there could have been a greater decrease in testosterone production if the exercise had been extended.

Therefore, exercise tends to lead to a decrease in testosterone production but would not become apparent unless it was continued for a considerable period of time. It was also hypothesized that during shorter periods of exercise any tendency to a reduction in testosterone production would be obscured by the correspondingly greater decrease in the metabolic clearance rate of testosterone, leading to an increase in plasma testosterone levels. Also, the reduced rate of clearance of testosterone could be accounted for by the simultaneous decrease in liver (hepatic) blood flow.

Conclusions similar to those of Cadoux-Hudson were found by Diamond et al. (1989). Diamond exercised twelve male college students on an electrically-braked ergocycle for twenty minutes at 80% of their MVO_2. It was observed that plasma levels of testosterone increased in response to the exercise. However, the testosterone increase could not be attributed to hemoconcentration, since the data was corrected for reduction in plasma volume. The authors hypothesized that it was possible that the response in serum androgens could be explained, at least partly, through a decrease in the metabolic clearance of the hormones rather than from an augmented production (Sutton et al., 1978).

A study performed by Galbo et al. (1977) consisted of eight healthy male students (20-28 years) who, on the first morning of the study, ran three times separated by two 15 minute resting periods. The first and second run lasted for ten minutes each and the work loads were designed to approximate 50% (mild) and 75% (moderate), respectively, of individual MVO_2s. The last run was initiated with two minutes of the 75% load but then the load was increased every 25 seconds until complete exhaustion was reached within a further 4-5 minutes. On the second morning, the subjects exercised at a moderate workload during repeated 20-minute bouts separated by 10-minute rest intervals (this sequence was

continued until exhaustion).

Thus, it was observed in this study that testosterone concentrations increased by 13% after maximal exercise and 31% after 40 minutes of prolonged exercise. During prolonged exercise testosterone concentrations then declined gradually and were at exhaustion significantly less than concentrations obtained after 40 minutes of running. Galbo observed that serum testosterone concentrations were not definitely changed during short exercise bouts of mild to maximal intensity. A significant increase, however, was found after 40 minutes of prolonged moderate exercise. The rise in testosterone concentrations was found to be independent of serum LH, since the concentration of this hormone, in accordance with other findings (Sutton et al., 1973; Métivier et al., 1980), was not raised by exercise.

The authors hypothesized that increased testicular stimulation by the high catecholamine concentrations possibly contributed to the rise in testosterone levels. In addition, decreased hepatic blood flow during exercise may have reduced hepatic testosterone clearance (Cadoux-Hudson et al., 1985). But, although catecholamine levels further increased during continued exercise, and hepatic blood flow possibly further decreased, testosterone concentrations nevertheless declined significantly. In addition, Galbo thought that since the rate of secretion of testosterone depends on the rate of testicular blood flow, the decline in testosterone concentrations during the latter part of the run possibly was due to a catecholamine mediated increase in testicular vascular resistance, similar to the increasing splanchnic vascular resistance leading to decreasing hepatic blood flow during continued exercise (Rowell et al., 1974).

A study by Jezova et al. (1985) showed both significant and insignificant increases in testosterone levels due to different

intensities of exercise. Jezova had seven healthy untrained male students participating in three bicycle ergometer exercise tests performed at intervals of one week, in randomized order. Exercise A consisted of three 6-minute periods of exercise with increasing work loads of 1.5, 2.0 and 2.5 W/kg, separated by a one minute rest, and with a 60 minute recovery period. Exercise B consisted of three 6-minute periods of exercise, each with work loads of 2.0 W/kg, separated by a one minute rest, and also with a 60 minute recovery period. Exercise was almost identical except that it consisted of two 4.5-minute periods of exercise with work loads of 5 W/kg. Plasma testosterone changes during exercises A and B were found to be insignificant while in test C the plasma testosterone concentrations were significantly elevated at the end of both exercise bouts, as well as 10 minutes after cessation of effort.

This study by Jezova is one of the few studies which examines the relationship between work intensity and plasma testosterone concentration. Sutton et al. (1973) reported that, in highly trained athletes, a significant rise in serum androgens occurs only in response to maximal and not to submaximal exercise. However, in their experiments, the maximal exercise was performed in the mornings and the submaximal exercise in the afternoons. Thus, diurnal variations could account for the described differences in serum androgen response to exercise.

Decreased hepatic blood flow, reduced metabolic clearance rate and hemoconcentration are supposed to be the most important factors for the exercise-induced rise in serum androgens (Galbo et al., 1983; Weiss et al., 1983). The slight increases in hematocrit values observed in Jezova's study were similar in all three exercise tests, whereas significant increases in plasma testosterone occurred only during

exercise C. Thus, in these experiments, hemoconcentration does not seem to be the main factor responsible for exercise-induced elevations of plasma testosterone levels.

Kuoppasalmi et al. (1980a) also studied the relationship between intensity of exercise and plasma testosterone levels. Seventy-eight male volunteers participated, of which 31 of them were short and long-distance runners who had trained regularly for many years and the other 47 were untrained men (controls). The exercises consisted of a 15 second, 2 minute, 45 minute (high intensity), and 90 minute (moderate intensity) running tests. No significant changes were noticed in plasma testosterone during the 15 second test. During the 2 minute running test, plasma testosterone levels increased by 13% and six hours after the test the mean plasma testosterone levels decreased as compared with pre-exercise levels. No changes in testosterone were observed during the 45 minute test, although a tendency to increased levels was observed in plasma testosterone. Finally, no changes in plasma testosterone were observed during the 90 minute running test. Kuoppasalmi thought that the main reason for the tendency of plasma testosterone to rise during the intense exercise was a decrease in the hepatic metabolic clearance rate of testosterone. It had been previously shown that with the decrease in hepatic metabolic clearance rate during exercise plasma testosterone may rise by as much as 50% above the initial level (Mean et al., 1977).

Kuoppasalmi performed another study in 1980, again studying the relationship between intensity of exercise and plasma testosterone levels. This time he had 10 male volunteers, of which five were sprinters and the other five were long-distance runners.

This particular study consisted of a short-term running test and a long-term running test. For the short-term running test, three subjects ran 20, 40 and 60m, resting for 30 seconds between each run, and two

subjects ran 3x40m, resting five minutes between each run (done at maximal speed). Plasma testosterone levels for this test did not change significantly.

The long-term running test consisted of the five long-distance runners running 21 km in 90 minutes (intense) and 13-14 km in 45 minutes (moderate). Both the moderate and the intense long-term runs tended to increase plasma testosterone levels (7% and 21% respectively) from pre-exercise levels. But, 30 minutes after the intense run, plasma testosterone levels were decreased as compared with the pre-exercise levels or immediate post-exercise levels. Kuoppasalmi theorized that this low plasma testosterone level after intense exercise was probably associated with decreased testicular secretion, since it had been shown in rats that swimming for one hour decreases testosterone levels in both plasma and testis to 1/3 of control levels (Bliss et al., 1972). Although the mechanism leading to the depression of testosterone levels during exercise is not known, a shift in the biosynthetic pathway of androgens (Aono et al., 1976) as well as a decrease in the activities of the enzymes in the testis due to stress may contribute to the lowered plasma testosterone.

In addition to Kuoppasalmi, Wilkerson et al. (1980) also studied the potential relationship between exercise intensity and plasma testosterone levels. Five males volunteered to walk (100 m/min) on five separate occasions at 30, 45, 60, and 90% of their MVO_2. There was a significant increase in plasma total testosterone concentration above resting control levels at all work intensities except 45%. However, these increases were similar to the hemoconcentration observed, yielding no statistically significant net change from resting control levels in total plasma content of testosterone at any submaximal work level and max. These results are apparently in contradiction to those reported by

investigators who had found increases (Galbo et al., 1977; Kuoppasalmi et al., 1976; Sutton et al., 1973) and those who had found decreases (De Lignieres et al., 1976; Dessypris et al., 1976; Lamb et al., 1975) in plasma testosterone concentration levels.

Kindermann et al. (1982), instead of looking at intensities, focussed on different types of exercise and their effects on testosterone levels. In this study, 17 healthy male college students were examined. Within one week every subject performed three types of exercise tests on a motor-driven treadmill. Initially, a progressive exercise test was performed. The treadmill was set at a constant slope of 5%, the initial speed at 6 km/hr. Every three minutes the speed was increased by 2 km/hr until exhaustion. At least three days after the progressive test a prolonged test was performed, consisting of steady-state exercise at the anaerobic threshold of 4 mmol/l blood lactate for 50 minutes. One day after this test a short-term anaerobic exercise test was carried out. For this test, the subjects had to perform an all-out run at a treadmill slope of 5% and a speed of 20 km/hr.

Kindermann observed plasma testosterone level increases of 14% and 16% in the short-term anaerobic exercise test and the prolonged test, respectively. In this study the percent changes of testosterone exceeded only slightly the percent changes in plasma volume. Consequently, secretion of these hormones appeared to be essentially unaffected by a single bout of anaerobic exercise. In addition to hemoconcentration, decreased metabolic clearance due to reduced hepatic blood flow had to be considered. Thus, according to other studies in this area, the increase in testosterone by 20% during prolonged exercise in this study could be explained entirely by decreased clearance (Sutton et al., 1976).

Gawel et al. (1979) observed significant increases in

testosterone levels due to exercise and these results were quite similar to those of Métivier et al. (1980) and Vogel et al. (1985). Gawel exercised eleven healthy male volenteers on a Monark bicycle ergometer at a constant speed of 20 km/hr at 15, 30 and 40% of their maximum loads for successive periods of 10 minutes. It was observed that plasma testosterone levels rose at the end of the maximal exercise and began to fall during rest whereas the small rise in LH is found to be insignificant. Thus, the rise in testosterone in this experiment is independent of LH.

A study performed by Urhausen et al. (1987b) found results which were slightly different from those of Gawel et al. Urhausen studied eight male subjects who were participating in 1/4 triathalon. Testosterone was found to have a biphasic behavior during the event. After an initial increase, testosterone levels decreased after exercise durations exceeding three hours. This decrease in testosterone and in particular of free testosterone was attributed to several factors such as reduced secretion of LH and FSH (Aakvaag et al., 1978).

Weiss et al. (1983) took a different approach from most investigators and looked at the effects of weight-lifting on plasma testosterone levels. Thus, twenty males performed heavy resistance weight-lifting for 30 minutes. Immediately following exercise, the mean plasma level of testosterone for subjects was significantly higher than at rest by 22%. The values then returned to resting levels by 30 minutes post-exercise. These findings are in agreement with those described by Fahey et al. (1976), who reported a significant increase (19%) in serum testosterone immediately following a strenuous bout of weight-lifting in college men. In contrast to the findings of Weiss, Skierska et al. (1976) did not find a significant increase in serum testosterone in well-trained male weight-lifters following 30 minutes of weight-lifting.

Although the duration of exercise in that study was the same as in this study, the intensity was not reported. Therefore, a difference in work intensity could be the basis for the contrasting results.

The effects of marathon runs on plasma testosterone levels have been studied extensively by several researchers and the majority of them have come to the same conclusion: that marathon runs cause a decrease in serum testosterone concentrations. For instance, Dessypris et al. (1984) studied nine male participants (all in regular training) in a marathon run and observed that testosterone levels deceased by about 20% as compared to pre-race levels.

Similarly, Kuusi et al. (1984) studied 20 men who participated in a marathon and found that the mean serum testosterone level decreased significantly by 20% during the marathon. In addition, Semple et al. (1985) observed 10 healthy adult males during a marathon run and witnessed a decrease in serum testosterone from 18.5 to 14.9 nmol/l. LH levels were also reduced which might have suggested a hypothalamic-pituitary impairment following marathon running. Cook et al. (1986) also had similar results when studying eight men in successive marathons. Their testosterone levels were significantly lower than their pre-race levels.

Thus, the type, duration and intensity of exercise, from study to study, varies from one extreme to the other. These differences in exercise protocols have a great influence on serum testosterone levels. The pattern seems to be that exercise of short duration leads to an increase in testosterone levels, while long-term exercise leads to a decrease in testosterone levels. However, there are exceptions to this pattern. In addition, the changes in testosterone levels documented in these studies are not necessarily due to changes in actual testosterone production but could be due to changes in clearance rate, plasma volume

or hemoconcentration.

APPENDIX C

CHAPTER 3

METHODOLOGY

Subjects

Twenty-one healthy white male subjects between the ages of 19 and 28 years (mean age of 22.5), who are all students of the University of Ottawa, agreed to participate in this study. Nine of these students were considered untrained (i.e. their MVO_2 is less or equal to 50 ml O_2/kg/min). The other 12 were considered to be trained (i.e. their MVO_2 is greater than or equal to 55 ml O_2/kg/min) and were members of the University of Ottawa cross-country running team.

Procedures

For the first part of the experiment, each subject's arrival was scheduled in the morning or afternoon. As soon as a subject arrived, he was given a consent form and health screening form which needed to be signed before any testing could be done. Afterwards, the subject's height, weight and skinfold measurements were taken according to the method by Durnin et al. (1974). In addition, resting heart rate and blood pressure were also taken before any exercise, just as a safety measure. A test of maximal oxygen consumption (MVO_2) was then performed on a treadmill using direct gas open circuit measurements. Each subject was carefully monitored at all times, especially his heart rate, via a sports-tester. Details of the protocol for the MVO_2 test are presented in table 1.

For the second part of the experiment, the subjects arrivals' in the laboratory were scheduled in the morning, approximately one week after the maximal test. The subject, who was fasting since midnight, laid in bed for 20 minutes. At the end of this period (immediately before

exercise), a sample of blood (10 cc) was taken from the antecubital vein. The subject then ran on the treadmill at 70% of his MVO_2 for 20 minutes, heart rate being monitored throughout the exercise. Immediately after the exercise and 60 minutes after, blood samples of 10 cc were again taken from the antecubital vein.

Table 1

Protocol used for Treadmill Running

Stage (#)	Time (min)	Grade (%)	Speed (km/h
w-up*	2	0	5
1	2	5.0	6
2	2	5.0	7
3	2	5.0	8
4	2	5.0	9
5**	2	0	10
6	2	5.0	11
7	2	5.0	12
8	2	5.0	13
9	2	5.0	14
10	2	5.0	15
11	2	5.0	16
12	2	5.0	17
13	2	5.0	18

* First stage for untrained subjects

** First stage for trained subjects

Blood analysis

Blood samples taken from the antecubital vein by a qualified technician were placed in glass tubes which contained 10.5 mg of EDTA. The tubes were then pre-chilled in an ice bath at a temperature of 2 to

8°C. Then, the plasma was separated from the blood as soon as possible. This was done by centrifugation in a refridgerated centrifuge (2 to 8°C). Afterwards, the samples were stored at -20°C until the actual analysis.

Concentrations of testosterone were determined using the Coat-A-Count Total Testosterone kit, which consisted of a solid phase radioimmunoassay, based on testosterone-specific antibody immobilized to the wall of a propylene tube. ^{125}I-labelled testosterone competed for a fixed time with testosterone in the subject sample for antibody sites. The tube was then decanted, to separate "bound" from "free", and counted in a gamma counter.

Concentrations of FSH were determined using a solution of FSH which was marked with ^{125}I. The Amerlex-M FSH RIA kit (1988) was used for this purpose. In addition, the Amerlex-M LH RIA kit (1988) was used to determine concentrations of LH. Prolactin was measured using the Tandem-R Prolactin Immunoradiometric assay kit which was developed by Hybritech (1988).

Statistical analysis

A two-way analysis of variance with repeated measurements was used to determine differences for testosterone, prolactin, LH and FSH immediately following exercise, 30 minutes post and 60 minutes post-exercise as compared to levels before exercise in both subject groups. Post hocs were done, using Tukey's test.

APPENDIX D

Table A

Description of trained subjects.

Subject #	Weight (kg)	Height (cm)	Body fat %	Age (yrs)
2	75.7	189.6	14.74	22
3	64.0	170.2	13.89	23
4	78.4	179.4	12.74	21
5	73.5	187.7	11.52	26
6	67.3	175.8	11.96	23
7	69.1	176.1	13.40	22
9	62.3	172.3	8.87	24
11	69.8	179.4	9.87	21
14	67.0	183.1	8.26	22
17	85.2	182.7	16.28	24
19	68.2	178.7	12.50	20
21	74.9	174.7	13.29	19

Table B

Description of untrained subjects

Subject #	Weight (kg)	Height (cm)	Body fat %	Age
13	75.2	177.6	18.29	22
20	90.4	190.3	22.07	20
24	96.1	187.1	19.10	23
26	83.5	175.2	19.38	28
28	111.3	177.2	28.18	22
29	69.0	183.3	15.30	24
31	74.5	178.1	15.27	23
32	96.6	185.2	18.57	22
33	88.7	173.7	22.94	23

APPENDIX E

Table C

Physiological responses of trained subjects

Subject #	Resting Heart Rate (beats/min)	Maximum Heart Rate (beats/min)	MVO_2 ml O_2/kg/min	Mean MVO_2 at 70% MVO_2 ml O_2/kg/min	HeartRate @ 70%MVO_2 (beat/min)
2	50	185	57.0	41.45	149
3	60	168	63.75	45.79	168
4	55	207	59.0	42.17	155
5	51	183	63.76	43.20	155
6	49	187	70.15	49.58	153
7	38	184	69.0	45.72	149
9	39	181	79.9	55.45	148
11	53	197	64.68	44.34	134
14	46	179	70.09	49.15	152
17	75	178	60.80	45.05	155
19	67	184	62.27	41.51	152
21	55	190	62.90	41.91	148

Table D

Physiological responses of untrained subjects.

Subject #	Resting Heart Rate (beats/min)	Maximum Heart Rate (beats/min)	MVO_2 ml O_2/kg/min	Mean MVO_2 @ 70% MVO_2 ml O_2/kg/min	Heart Rate @ 70%MVO_2 (beat/min)
13	67	200	48.80	35.65	168
20	82	187	43.93	31.76	174
24	58	192	44.40	30.70	153
26	67	200	41.59	29.91	174
28	81	197	42.20	27.33	163
29	60	193	49.60	32.88	151
31	59	198	48.56	31.41	160
32	64	186	45.80	31.54	168
33	54	201	42.70	29.65	167

APPENDIX F

Table E

Hematocrit values (expressed in vol. %) before, immediately after, 30 minutes after and 60 minutes after an acute exercise in trained subjects.

Subject #	PRE	POST	POST30	POST60
2	46.6	49.9	45.9	45.8
3	44.6	49.1	45.2	44.5
4	48.4	51.1	48.1	49.3
5	48.2	49.8	46.4	46.4
6	46.4	48.8	45.9	45.3
7	44.0	45.2	40.7	42.5
9	47.7	50.6	45.8	47.0
11	48.6	50.3	48.1	48.2
14	46.2	47.8	43.7	46.0
17	45.5	48.0	47.0	47.0
19	44.0	--	44.5	44.0
21	45.2	46.2	42.4	44.7

Table F

Hematocrit (expressed as vol. %) values before, immediately after, 30 minutes after and 60 minutes after acute exercise in untrained subjects.

Subject #	PRE	POST	POST30	POST60
13	45.2	46.2	43.6	43.0
20	48.9	51.2	45.3	47.2
24	41.4	46.6	41.9	43.9
26	46.9	51.5	45.8	47.5
28	46.9	48.7	45.7	45.5
29	45.2	47.2	47.2	44.5
31	44.2	46.1	41.7	43.8
32	48.2	49.7	48.5	46.7
33	46.7	48.8	46.1	44.9

PRE = before exercise

POST = immediately after exercise

P30 = 30 minutes after exercise

P60 = 60 minutes after exercise

APPENDIX G

Table G

Testosterone levels (expressed in nmol/L) before, immediately after, 30 minutes after and 60 minutes after acute exercise in trained subjects.

Subject #	PRE	POST	P30	P60
2	15.8	23.9	15.5	15.4
3	22.9	32.8	21.2	21.8
4	20.0	28.6	20.7	18.1
5	15.7	21.7	16.5	16.7
6	21.4	32.1	27.3	20.7
7	20.2	28.6	18.8	16.4
9	17.8	27.1	17.6	16.4
11	21.0	28.5	22.4	21.4
14	21.8	30.6	20.2	17.8
17	17.9	27.5	23.9	17.9
19	20.3	31.0	23.5	26.5
21	22.3	31.6	21.3	22.8

PRE = before exercise

POST = immediately after exercise

P30 = 30 minutes after exercise

P60 = 60 minutes after exercise

Table H

Testosterone levels (expressed in nmol/L) before, immediately after, 30 minutes after and 60 minutes after acute exercise in untrained subjects.

Subject #	PRE	POST	P30	P60
13	25.0	27.2	22.4	18.5
20	25.5	33.8	23.6	23.1
24	23.1	27.1	22.7	22.2
26	26.2	33.1	23.7	19.8
28	25.0	29.0	24.0	25.1
29	24.8	32.7	18.7	17.2
31	27.2	28.7	24.1	21.4
32	26.2	31.8	26.2	26.7
33	24.7	28.8	23.0	24.9

PRE = before exercise

POST = immediately after exercise

P30 = 30 minutes after exercise

P60 = 60 minutes after exercise

Table I

Prolactin levels (expressed in ng/ml) before, immediately after, 30 minutes after and 60 minutes after acute exercise in trained subjects.

Subject #	PRE	POST	P30	P60
2	4.0	5.4	4.0	3.2
3	7.0	7.5	5.4	5.3
4	10.9	11.0	11.4	9.8
5	4.3	4.2	3.5	3.7
6	4.9	5.4	4.7	4.8
7	4.2	5.2	4.0	4.2
9	4.8	4.9	3.8	4.1
11	6.9	6.4	5.8	5.3
14	4.6	5.3	4.2	4.0
17	7.5	8.2	6.1	7.3
19	6.8	6.9	6.2	6.5
21	11.4	11.0	7.6	6.0

PRE = before exercise

POST = immediately after exercise

P30 = 30 minutes after exercise

P60 = 60 minutes after exercise

Table J

Prolactin levels (expressed in ng/ml) before, immediately after, 30 minutes after and 60 minutes after acute exercise in untrained subjects.

Subject #	PRE	POST	P30	P60
13	4.1	4.4	4.3	3.4
20	4.7	3.9	4.4	4.4
24	3.4	5.0	3.0	3.7
26	2.8	2.5	3.8	2.7
28	6.8	6.9	6.1	6.5
29	5.2	8.3	5.7	4.3
31	3.7	3.6	4.3	3.6
32	6.3	6.0	6.6	5.9
33	2.7	2.5	2.8	2.3

PRE = before exercise

POST = immediately after exercise

P30 = 30 minutes after exercise

P60 = 60 minutes after exercise

Table K

Luteinizing hormone (LH) levels (expressed in mlU/ml) after, immediately after, 30 minutes after and 60 minutes after acute exercise in trained subjects.

Subject #	PRE	POST	P30	P60
2	5.2	4.8	4.6	3.3
3	6.4	6.2	6.3	6.4
4	3.1	4.4	2.5	3.0
5	4.5	6.3	5.2	5.2
6	5.8	7.4	5.9	5.1
7	6.2	6.6	5.9	5.6
9	7.8	6.3	4.3	4.9
11	5.4	5.1	5.4	5.1
14	3.4	4.1	4.7	6.0
17	5.2	4.8	4.7	4.0
19	3.9	6.1	4.3	5.7
21	7.2	6.9	7.2	7.2

PRE = before exercise

POST = immediately after exercise

P30 = 30 minutes after exercise

P60 = 60 minutes after exercise

Table L

LH levels (expressed as mlU/ml) before, immediately after, 30 minutes after and 60 minutes after acute exercise in untrained subjects.

Subject #	PRE	POST	P30	P60
13	6.8	6.9	6.5	6.6
20	5.9	6.7	7.7	5.0
24	7.4	7.2	6.2	8.3
26	6.2	5.1	3.5	6.3
28	10.3	9.4	9.5	9.7
29	6.0	5.4	6.3	4.5
31	9.1	8.5	8.1	8.3
32	6.0	6.5	8.1	6.0
33	3.8	4.6	8.2	5.3

PRE = before exercise

POST = immediately after exercise

P30 = 30 minutes after exercise

P60 = 60 minutes after exercise

Table M

Follicle-stimulating hormone (FSH) levels (expressed in mlU/ml) before, immediately after, 30 minutes after and 60 minutes after acute exercise in trained subjects.

Subject #	PRE	POST	P30	P60
2	5.6	4.8	4.3	4.1
3	4.6	4.5	4.7	4.4
4	4.3	3.8	4.3	3.1
5	3.9	2.8	3.1	2.4
6	5.4	4.8	4.9	4.8
7	5.0	5.1	4.8	4.3
9	5.7	5.2	4.8	4.9
11	4.5	5.0	4.0	4.6
14	4.8	4.4	6.0	6.1
17	6.0	5.9	6.0	5.7
19	2.8	2.9	3.6	3.0
21	3.9	4.9	3.1	3.4

PRE = before exercise

POST = immediately after exercise

P30 = 30 minutes after exercise

P60 = 60 minutes after exercise

Table N

FSH levels (expressed in mlU/ml) before, immediately after, 30 minutes after and 60 minutes after acute exercise in untrained subjects.

Subject #	PRE	POST	P30	P60
13	7.6	7.5	6.4	6.5
20	7.1	6.6	6.9	7.1
24	6.5	6.6	5.6	5.9
26	4.6	4.2	3.8	3.8
28	7.5	6.5	7.5	7.0
29	3.3	2.9	2.4	2.6
31	8.2	8.7	6.2	6.7
32	3.0	2.9	4.0	2.7
33	3.7	3.8	4.6	4.2

PRE = before exercise

POST = immediately after exercise

P30 = 30 minutes after exercise

P60 = 60 minutes after exercise

APPENDIX H

Direct analysis of gases

The open circuit method for gas analysis was used to determine the aerobic capacity (MVO_2) of the subjects. To accomplish this, each subject had to breath into a valve which was connected via a hose to a metabolic analysis system (i.e. a metabolic cart). The system is composed of an 8 litre box, which receives the subject's air. A small portion of this air (0.6 litre/minute) flows into a tube which contains calcium sulfate. It is at this point that oxygen and carbon dioxide analyses are done directly the Amtek S-3A and Amtek CD-3A analysers, respectively. These analyses were done 30 seconds before the end of each stage in the Leger protocol (see fig.1).

APPENDIX I

Analysis of hormone levels in the blood

Luteinizing Hormone

The technique used to analyse this hormone in blood is known as a radioimmunoassay method. This method depends on the competition for a limited number of binding sites on an LH specific antibody between LH in serum and ^{125}I-labeled LH. The proportion of ^{125}I-labeled LH bound to antibody is inversely related to the concentration of unlabeled LH present in the sample. The antibody-bound LH is then reacted with the Amerlex-M second antibody reagent which contains second antibody that is bound to magnetic polymer particles. Separation of the antibody-bound fraction is effected by magnetic separation. Following decantation of the supernatant the proportion of ^{125}I-labeled LH bound in the presence of reference standard solutions containing known amounts of LH is determined by the use of a gamma counter to measure radiation. The concentration of LH present in the unknown samples can then be interpolated. The concentration of LH is given in units of mIU/ml or IU/L (international units per litre) . These are units developed by WHO (World Health Organization). This means that the standards used in this kit were calibrated against the 1$_{st}$ IRP (68/40). The same principles apply for the determination of FSH, except that the antibodies are FSH specific and the standards were calibrated against the 2$_{nd}$ IRP 78/549.

Prolactin

The prolactin kit is basically a solid phase two-site immunoradiometric assay. Samples containing prolactin are reacted with a plastic bead (solid phase) coated with a monoclonal antibody directed toward a unique site on the prolactin molecule and with a radiolabeled monoclonal antibody directed against a distinctly different antigenic site on the same prolactin molecule. Following the formation of the

solid phase/prolactin/labeled antibody sandwich, the bead is washed to remove unbound labeled antibody. The radioactivity bound to the solid phase is measured with a gamma counter. The amount of radioactivity measured is directly proportional to the concentration of prolactin present in the sample.

Testosterone

The testosterone kit determines total testosterone (i.e. protein-bound and free testosterone) in serum. This kit is a solid-phase ^{125}I radioimmunoassay designed for the quantitative measurement of testosterone in serum (or plasma). It is based on testosterone-specific antibody immobilized to the wall of a polypropylene tube. $_{125}I$-labeled testosterone competes for a fixed time with testosterone in the subject's sample for antibody sites. The tube is then decanted, to separate bound from free, and counted in a gamma counter. The amount of testosterone present in the subject's sample is then determined from a standard curve.

APPENDIX J

SCHOOL OF HUMAN KINETICS

THESIS RESEARCH CONSENT FORM

Studies involving human subjects require consent of the participants. I, _____ authorize Jeff Moore (564-5718 or 231-5396) of the School of Human Kinetics, University of Ottawa, to administer and conduct exercise tests designed to measure my maximal aerobic capacity (MVO_2) and adaptation to exercise through blood hormones (testosterone, prolactin, LH and FSH). This study will be conducted under the supervision of Dr. Guy Métivier (554-5718).

I understand that I will perform a gradual maximum test on a treadmill which involves progressively increased workloads every two minutes until exhaustion in order to establish my MVO_2. The test will be stopped when I would not be ableto maintain the pace due to fatigue or if I become distressed in any way or develop any abnormal response.

In order to establish my adaptation to exercise through hormone levels, a second test will be conducted in the following weeks and will involve performing a treadmill test at an intensity equal to 70% of my MVO_2 for 20 minutes. Blood pressure, height, weight and skinfold measurements will also be taken.

During all the tests, I will breathe through a mouthpiece and my heart rate will be monitored by a sport tester strap attached around my chest. Blood samples will be drawn (two tubes of 7 cc) from the vein of

my arm before, immediately after, 30 minutes and 60 minutes after the exercise on the treadmill at 70% of my MVO_2. A qualified technician will collect the blood samples. The tests will be conducted by qualified examiners (First Aid and CPR certified). I understand that I may experience some local muscular fatigue, dryness of the mouth and throat and an increased resistance to breathing while exercising. There might also be transient lightheadedness, fainting, chest discomfort and leg cramping associated with the exercise.

I recognize that a physical examination prior to performing maximal exercise testing is necessary and a medical certificate is required (for an adult < 35 years old and moderately active).

I understand that all data collectyed in this study will be kept confidential and presented in an anonymous form in the final report. I also understand that I will be informed of the results of the study upon its completion.

I understand that I have the right to withdraw from this study at any time and that I am free to ask any questions during the test.

Date: _____ Subject: _____

Witness: _____