Hormone Responses to Resistance vs. Endurance Exercise in Premenopausal Females

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Catalog Data

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Mots clés: déhydroépiandrostérone, testostérone, oestradiol, hormone de croissance, IGF-I

Abstract/Résumé
Sixteen, cross-trained, premenopausal women participated in an endurance, resistance, and control session to compare hormone responses. The resistance session included 3 sets of eight exercises at 10 RM intensity. The endurance session consisted of a 40-min cycling protocol at 75% of maximal heart rate. During the control session, subjects rested for 35 min. Serum DHEA, estradiol, testosterone, growth hormone, IGF-I, cortisol, and plasma lactate concentrations were measured pre-exercise, post-exercise, and 30 min into recovery. Differences in intensity variables existed between the three sessions. Endurance exercise elicited increases in growth hormone, estradiol, and testosterone compared to the control session, and growth hormone increased after the resistance compared to the control session. The exercise protocols used in this study indicate that an acute bout of exercise can stimulate the endocrine system in premenopausal females. In addition, these results indicate that differences exist between these two exercise protocols when compared to a control session.

Les adaptations hormonales de seize femmes préménopausées participant à trois activités (force, endurance et contrôle) sont comparées. La séance d’exercices de force consiste en 3

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Introduction

Previous research has indicated that anabolic hormones have important roles in growth and development (Bennett et al., 1984; Florini, 1987). For example, evidence shows that levels of reproductive hormones in premenopausal women may influence their current bone mass (Sowers et al., 1998) and be predictive of bone mass levels as they age (Newton-John and Morgan, 1968). Therefore, research on young females is needed to assess potential influences that could alter these hormone concentrations.

Recently, many people have turned to the use of hormone supplements to enhance physiological function and wellbeing. To maximize health benefits and reduce the financial and safety considerations associated with the use of supplements, it is more prudent to find ways to naturally increase these desirable hormones in the body. One proposed method of augmenting hormone concentrations is through exercise. Previous research indicates that exercise is capable of stimulating the endocrine system (Kraemer et al., 1991; Kraemer et al., 1995).

Regular physical activity is associated with lower risk of premature development of many health problems (U.S. Department of Health and Human Services, 1996). Exercise, an organized form of physical activity, may be conveniently categorized as endurance or resistance. While both of these types of exercise promote health, the physiological adaptations are specific to the exercise type. Benefits of endurance exercise include improved cardiovascular function, while resistance exercise has been associated with the development and preservation of muscle mass.

Currently, there is a paucity of information documenting the anabolic hormone response to exercise in females. The majority of previous endocrine research has focused on the hormone response to endurance and, to a lesser extent, resistance exercise in males. Endurance studies in females have documented acute increases in growth hormone (Bunt et al., 1986; Kraemer et al., 1993), testosterone (Baker et al., 1982; Shangold et al., 1981; Webb et al., 1984), estradiol (Jurkowski et al., 1978; Nicklas et al., 1989) and dehydroepiandrosterone (DHEA; Baker et al., 1982). However, quite often these studies have used protocols that are not typical of an exercise session of a recreational exerciser. The few female studies that have used resistance protocols have reported acute increases in growth hormone (Kraemer et al., 1991) but no change in testosterone concentrations (Fahey et al., 1976; Kraemer et al., 1991, 1993).
To our knowledge, no data exist that compare the acute hormone response to endurance versus resistance exercise using the same group of females. Although, it is evident that prolonged training of either type of exercise produces separate physiological adaptations, it is not known if the acute endocrine response differs between the two types of exercises. Therefore, the purpose of this study was to compare the acute anabolic hormone response to a typical endurance versus resistance exercise session in premenopausal females.

Methods

SUBJECTS

Sixteen, healthy, premenopausal females (aged 33 ± 8 years) were recruited for this study. All subjects had been actively cross-training (endurance and resistance exercise) at least twice a week for at least 4 months. All subjects were considered eumenorrheic based on (self-reported) regular menstrual cycles for the previous 12 months. None of the subjects were smokers or had used hormonal contraceptives within the previous 4 months. All participants provided written informed consent, and the institutional Research Ethics Board approved the study protocol and the use of human subjects.

EXPERIMENTAL DESIGN

Subjects were required to participate in six sessions. At the first session, informed consent and a medical health history were obtained. The remaining sessions are described below.

Fitness appraisal session. All subjects completed the Canadian Physical Activity, Fitness and Lifestyle Appraisal (CPAFLA; Canadian Society for Exercise Physiology, 1996). The CPAFLA evaluated health-related fitness components including body composition, aerobic fitness, flexibility, strength, muscular endurance, leg power, and physical activity behaviors. Percent body fat was also calculated employing the three-site (tricep, suprailiac, and abdomen) Jackson and Pollock (Jackson and Pollock, 1985) and Siri equations (Siri, 1956).

Familiarization session. During this orientation session, subjects were familiarized with both the endurance and resistance exercise protocols. All subjects had their 10 repetition maximum (10 RM) determined for each of the eight exercises that were used for this study according to procedures explained by Wathen (1994). During this session, subjects were also familiarized with the endurance (cycling) protocol. A graded maximal cycling test was performed to exhaustion to directly determine the maximal heart rate for each individual. An appropriate workrate (to elicit 75% of the subject's previously determined maximal heart rate) for the endurance session was determined. In both the resistance and endurance portions of the familiarization session, subjects were familiarized with the Borg Rating of Perceived Exertion (RPE) Scale (Borg, 1982).

Testing sessions. The testing sessions consisted of one endurance exercise session, one resistance exercise session, and one control session, and all took place during the early luteal phase (days 14–21) of each subject's menstrual cycle. To control for diurnal variations in the hormones being measured, all testing sessions were performed at the same time of day (0700h–1000h). Before each of these
testing sessions, subjects were instructed to follow a standardized breakfast meal, abstin from the consumption of alcohol or caffeine for at least 6 hours, and refrain from physical activity for 24 hours before testing. These three sessions were randomized, with a minimum of 1 day separating each session.

The resistance session consisted of three sets of 10 repetitions of eight exercises using Universal equipment. The intensity equaled the previously determined 10 RM for each exercise, and 1 min of rest was given between sets and exercises. The resistance exercises included supine chest press, latissimus pull-down, leg press, biceps curl, leg extension, triceps push-down, leg curl, and shoulder press. The order in which these exercises were performed was as listed above.

The endurance session consisted of 40 min of pedaling on a Monarch cycle ergometer at 75% of the subject’s previously determined maximum heart rate. Pedal cadence was maintained between 60 and 70 revolutions per minute. These exercise protocols were selected to represent typical recreational exercise sessions. During the control session, subjects sat quietly for 35 min. Every 5 min during these testing sessions, RPE values were obtained and heart rate was monitored continuously using the Polar XL, telemetric heart rate monitor.

BIOCHEMICAL PROCEDURES

Prior to each of the three testing sessions, an intravenous catheter was inserted into a forearm vein and maintained with a saline lock. Subjects had blood samples taken in a seated position in a climate controlled environment. Blood samples were taken 10 min before the beginning of each session, immediately following the session, and a third sample was taken 30 min after the session had been completed. All samples were measured for serum concentrations of DHEA, testosterone, estradiol, growth hormone, insulin-like growth factor I (IGF-I), and cortisol. The samples taken pre-session and immediately after the session were analyzed for plasma lactate. Blood samples were analyzed for hematocrit using the microhematocrit method.

All hormone and lactate measurements were competed in duplicate. Serum estradiol and DHEA concentrations were assayed using $^{125}$I-radioimmunoassay techniques (Diagnostic Systems Laboratories, Inc., Webster, TX). Growth hormone and IGF-I were measured using $^{125}$I-immunoradiometric assay procedures (Diagnostic Systems Laboratories, Inc., Webster, TX). Serum testosterone and cortisol were measured by an automated chemiluminescence technique (Chiron Diagnostics, East Walpole, MA). Plasma lactate was analyzed using a quantitative, enzymatic spectrophotometric method at 540 nm (Sigma Diagnostics, St. Louis, MO).

Average intra-assay variability, measured as coefficient of variation, for DHEA, estradiol, growth hormone, IGF-I, testosterone, cortisol, and lactate was 6%, 5%, 6%, 3%, 7%, 2%, and 7%, respectively. Due to the within-subject design of this study, all samples from each subject were analyzed in the same assay to avoid inter-assay variance.

STATISTICAL ANALYSES

Hormone concentrations were expressed as absolute change (post-exercise minus pre-exercise) for each subject to control for variation in pre-exercise hormone levels. All post-exercise blood samples were corrected for changes in plasma volume
according to van Beaumont (1972). A 2 x 3 analysis of variance (ANOVA) was used to examine the changes in hormone concentrations with respect to time (pre-post, pre-recovery) and condition (endurance, resistance, control). Area under the curve (AUC) was determined for each hormone after the pre-exercise concentration sample was subtracted. The AUC for each hormone was then analyzed by a one-way ANOVA to determine if either exercise session affected the hormone concentration. Average heart rate, changes in lactate, and RPE for each session were analyzed using a one-way ANOVA. When necessary, Tukey post-hoc comparisons were used. In all cases, statistical significance was set at $P < .05$.

### Results

The mean results and ratings from the fitness appraisal are reported in Table 1. Ratings are based on normative data collected from a representative sample of Canadians of the same gender and age, and can range from “needs improvement” to “excellent.” Table 2 lists the mean resting hormone concentrations for all participants involved in the study. Individual resting concentrations were determined as the mean of their three pre-exercise samples.

#### Table 1  Scores and Ratings for Selected CPAFLA variables (Mean ± SD)

<table>
<thead>
<tr>
<th>Measurement ± SD</th>
<th>Rating</th>
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<tbody>
<tr>
<td>Body mass index (kg/m²)</td>
<td>23.4 ± 2.0</td>
</tr>
<tr>
<td>Percent body fat (%)</td>
<td>25.7 ± 5.1</td>
</tr>
<tr>
<td>Aerobic fitness score</td>
<td>439 ± 72</td>
</tr>
<tr>
<td>Grip strength (kg)</td>
<td>63 ± 10</td>
</tr>
<tr>
<td>Leg power (kg-m/sec)</td>
<td>75.6 ± 11.3</td>
</tr>
<tr>
<td>Push-ups (#)</td>
<td>39 ± 10</td>
</tr>
<tr>
<td>Partial curl-ups (#)</td>
<td>24 ± 2</td>
</tr>
<tr>
<td>Trunk forward flexion (cm)</td>
<td>34.5 ± 8.9</td>
</tr>
<tr>
<td>Healthy Physical Activity Participation Questionnaire</td>
<td>10 ± 1</td>
</tr>
</tbody>
</table>

#### Table 2  Hormone Concentrations at Rest (Mean ± SD)

<table>
<thead>
<tr>
<th>Hormone</th>
<th>Concentration ± SD</th>
</tr>
</thead>
<tbody>
<tr>
<td>DHEA (nmol/L)</td>
<td>38 ± 20</td>
</tr>
<tr>
<td>Testosterone (nmol/L)</td>
<td>1.1 ± 0.4</td>
</tr>
<tr>
<td>Estradiol (pmol/L)</td>
<td>201 ± 125</td>
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<tr>
<td>Growth hormone (μg/L)</td>
<td>1.2 ± 1.2</td>
</tr>
<tr>
<td>IGF-1 (μg/L)</td>
<td>259 ± 96</td>
</tr>
<tr>
<td>Cortisol (nmol/L)</td>
<td>463 ± 118</td>
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</table>
EXERCISE-INDUCED CHANGES IN HORMONES

Differences were found in the hormone responses between endurance and resistance exercise compared to rest. Significant time x session interactions were observed for both estradiol and growth hormone responses. The mean absolute change in growth hormone was greater after both the resistance ($P < .01$) and endurance ($P < .05$) exercise compared to the control session (Figure 1). The mean estradiol

**Figure 1.** Mean ($\pm$ SE) absolute change in serum levels of growth hormone (upper panel), estradiol (middle panel), and testosterone (lower panel) during the endurance exercise (solid), resistance exercise (shaded), and a control session (open). *Indicates significant session difference ($P < .05$) compared to the control session; **indicates significant session difference ($P < .01$) compared to the control session.
and testosterone responses to endurance exercise were significantly higher ($P < .01$ and $P < .05$, respectively) than the control session (Figure 1). No significant exercise-induced changes were observed for DHEA, IGF-I, or cortisol (Figure 2). Area under the curve (AUC) analyses confirmed the above session effects for estradiol and growth hormone. AUC analyses revealed no session effects for the remaining hormones.

**Figure 2.** Mean ($\pm$ SE) absolute change in serum levels of IGF-I (lower panel), DHEA (upper panel), and cortisol (middle panel) during the endurance exercise (solid), resistance exercise (shaded), and a control session (open). * Indicates significant difference in absolute change between sample times ($P < 0.01$; effect on all sessions combined).
The results indicate a significant sample time effect for both DHEA and cortisol. Tukey's post hoc analyses indicated that the change in DHEA and cortisol samples between the pre-exercise and recovery samples were significantly greater ($P < .01$) than the change between the pre-exercise and post-exercise samples (Figure 2).

**INTENSITY VARIABLES**

Results of the intensity variables for the three sessions are reported in Table 3. Results indicate that mean absolute changes in lactate after the resistance session were significantly higher ($P < .001$) than the endurance and control sessions. There were no significant differences between the endurance and control sessions when comparing changes in lactate values between pre- and post-session. Average heart rate was significantly higher ($P < .001$) throughout the endurance session than the resistance session, and mean heart rate values were significantly higher ($P < .001$) in the resistance session compared to the control session. Average RPE scores were highest ($P < .001$) during the resistance session, and the endurance session reported mean RPE scores significantly higher ($P < .001$) than the control session.

**Discussion**

This study demonstrated that a typical acute bout of exercise is capable of increasing circulating anabolic hormones in premenopausal women. Results from the present investigation indicate that 40 min of moderate endurance exercise is capable of increasing serum levels of testosterone and estradiol compared to a resting session. Despite an increasing trend that was observed in these two hormones during resistance exercise compared to rest, these increases did not reach statistical significance. Similarly, no statistical difference was observed between growth hormone concentrations after a 40-min endurance session and a typical resistance session; however, these two exercise responses were greater compared to resting concentrations.

Significant increases in testosterone after endurance exercise in females have been previously reported (Baker et al., 1982; Bonen and Keizer, 1987; Shangold et

<table>
<thead>
<tr>
<th>Table 3 Intensity Variables for the Three Testing Sessions (Mean ± SD)</th>
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<tbody>
<tr>
<td><strong>Δ in Lactate (mmol/L)</strong></td>
</tr>
<tr>
<td>--------------------------------</td>
</tr>
<tr>
<td>Aerobic session</td>
</tr>
<tr>
<td>Resistance session</td>
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<tr>
<td>Control session</td>
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</table>

*Significantly greater ($P < .001$) than corresponding control session.

*Significantly different ($P < .001$) than corresponding endurance session.
These increases appear to be independent of menstrual cycle phase due to the low amount of testosterone that is produced from the ovary (Van der Molen, 1969). In contrast to the possible anabolic significance during resistance exercise, increases in testosterone during endurance exercise may be important for glycogen compensation (Guezenneec et al., 1984).

The mechanism(s) responsible for increases in testosterone are still under debate. Endurance exercise is responsible for a decrease in metabolic clearance rate (MCR; Rowell, 1974). Cadoux-Hudson et al. (1985) determined that increases in testosterone were attributed solely to a reduction in clearance rate, mainly due to decreases in hepatic plasma flow. Others have refuted the claim that increases in free testosterone are caused by a change in protein binding, instead reporting that sympathetic stimulation of endocrine glands may promote testosterone synthesis (Fahrner and Hackney, 1998).

Despite apparent increases in testosterone after an endurance session, it is well documented that an acute bout of resistance exercise is not capable of producing the same response in females (Fahey et al., 1976; Kraemer et al., 1993; Kraemer et al., 1995). The results from the present study confirm these findings. However, females are capable of obtaining significant strength and muscle mass gains from resistance training (Hakkinen et al., 1992). A number of possibilities could account for this. Although non-significant, an increasing trend in testosterone was observed after the resistance session. It is unclear what physiological importance these small, temporary increases may have during repeated sessions such as chronic training. Other anabolic responses such as increases in growth hormone cannot be ruled out as being responsible for the hypertrophic events that occur at the muscular level after repeated resistance sessions. In addition, delayed increases in testosterone have been reported hours after completion of a resistance session in males (McMurray et al., 1995). Since it has been suggested that for muscle anabolism to occur, an increase in anabolic hormones must be maintained (McMurray et al., 1995), further research is needed documenting these hormones during recovery and during repeated exercise sessions.

The present study showed an increase in serum estradiol in response to endurance exercise during the luteal phase of the menstrual cycle compared to the control session response. Similar increases in estradiol have been observed with both submaximal and exhaustive exercise in females (Jurkowski et al., 1978; Nicklas et al., 1989). Similar to testosterone, it is possible that a decrease in MCR may have contributed to these increases. It is apparent that during endurance exercise both intensity and menstrual cycle phase play an important role in the response of estradiol. Previous research indicates that at a given exercise intensity the ovary may be more sensitive to the release of estradiol during the luteal compared to the follicular phase of the menstrual cycle (Jurkowski et al., 1978; Nicklas et al., 1989).

The results from the present study indicate that although a definite increasing trend was observed in serum estradiol after the resistance session, these increases were not significantly different from the control session. These results are comparable to those of Walberg-Rankin et al. (1992), who also observed a nonsignificant increase in estradiol after resistance exercise in females. In contrast, others have reported significant increases in untrained females (Kraemer et al., 1995) and females on a hypocaloric diet (Walberg-Rankin et al., 1992). It is apparent
that more research is needed to confirm the effects of resistance exercise on estradiol levels.

Estradiol is capable of stimulating lipolysis (Hansen et al., 1980) and inhibiting gluconeogenesis and glycogenolysis (Mandour et al., 1977). During endurance exercise, lipid metabolism plays a critical role in preserving glycogen stores. Therefore, it is not surprising that endurance exercise elicited increases in this hormone. In contrast, during more anaerobic exercise such as resistance exercise, lipid metabolism is less pronounced. Instead the main source of energy comes from the breakdown of glycogen as confirmed by the significantly higher lactate concentrations in the present study. Therefore, the different metabolic demands between the two types of exercise may have influenced these different estradiol responses.

Unlike the other hormones under investigation in this study, growth hormone increased significantly after both endurance and resistance exercise compared to the control session. It has been previously reported that both exercise intensity and duration determine the magnitude of the growth hormone response (Sutton and Lazarus, 1974). In agreement with these findings, Pritzlaff et al. (1999) recently reported a positive linear dose-response relationship between exercise intensity and growth hormone response.

Significant increases in growth hormone were observed after resistance exercise. These results are in agreement with Kraemer et al. (1991) who, using a similar protocol, observed significant increases in growth hormone after the exercise session in female subjects. Similar to endurance exercise, the degree of anaerobic involvement appears to be a determinant in the magnitude of the growth hormone response (Kraemer et al., 1991; Mulligan et al., 1996). The resistance session in the present study was more anaerobic in nature than the endurance session as indicated by the differences in lactate concentrations; however, it is unlikely that lactate itself regulates the release of growth hormone. The results from the endurance session in the present study indicate that growth hormone levels can increase without changes in circulating lactate and are supported by a lack of growth hormone response during lactate infusion (Luger et al., 1992). These findings have led investigators to speculate that other physiological factors, such as central nervous system responses, may be promoting exercise-induced changes in growth hormone (Weltman et al., 2000).

The data from the present study indicates that neither an acute bout of 40 min of cycling or a resistance exercise session is capable of eliciting a significant change in IGF-I response in premenopausal females. Prior research has indicated that circulating IGF-I has increased after endurance exercise of shorter duration (Bang et al., 1990; Cappon et al., 1994; Hornum et al., 1997). Therefore, it is possible that any initial increases in IGF-I that occurred at the onset of exercise in the present study were missed. However, if transient increases in IGF-I did occur, it is difficult to interpret the biological significance of such a short event.

Although no increases in circulating IGF-I were noted in this study, augmented local muscular production of IGF-I cannot be ruled out and has been reported elsewhere (Eliakim et al., 1997). Muscular uptake of IGF-I may also account for the absence of detectable increases in this hormone. Therefore, an increase in secretion of IGF-I may occur but is masked by an increased muscular uptake.
The results from the present study indicate that a resistance exercise session does not produce significant changes in serum IGF-I concentrations when compared to a control session. Previous research of the IGF-I response to resistance exercise has been inconsistent showing increases (Kraemer et al., 1991) and no change (Kraemer et al., 1993). Kraemer et al. (1993) suggested these inconsistent findings may be due to a number of different physiological factors, including concentrating mechanisms in the blood (e.g., differing MCRs), increases in transporter proteins, or the release of IGF-I from other nonhepatic cells (e.g., fat, muscle, and connective tissue cells) due to tissue disruption from exercise. These inconsistent findings were evident in our own data as subjects showed large inter-individual variation in their IGF-I responses.

The lack of DHEA response to endurance exercise in the present study is in conflict with results from the other studies (Baker et al., 1982; Cumming and Rebar, 1983; Johnson et al., 1997). It appears that the DHEA response may be intensity related. Previous studies involving cycling to exhaustion and running at 90% of maximum heart rate have demonstrated increases in DHEA concentrations in females after exercise (Cumming and Rebar, 1983; Johnson et al., 1997). These studies utilized higher exercise intensity protocols than the present study, which may account for the discrepancy in the findings.

To date, we are unaware of any studies investigating the DHEA response to resistance exercise in premenopausal females. The lack of DHEA response, however, is in agreement with the findings of Copeland (1998), who reported that DHEA concentrations did not change after varying exercise volumes in postmenopausal females. More research is needed to clarify the DHEA response to both endurance and resistance exercise.

The circadian variations of both DHEA and cortisol were evident in the present study and have been reported elsewhere (Lejeune-Lenain et al., 1987; Liu et al., 1990). Adrenocorticotropic hormone, which is known to stimulate DHEA and cortisol, has been reported to be highest in the morning (Reilly et al., 2000) and is presumably the reason why both of these hormones have marked circadian rhythms.

A number of different variables influence hormonal responses to exercise in females including menstrual cycle phase and status, fitness levels, nutritional status, stress levels, and substance use (e.g., medication or supplementation; Tremblay et al., 1995). Although the present study attempted to control for these variables, large inter-subject variations were observed in hormonal responses.

In summary, endurance exercise appears to be capable of eliciting testosterone, estradiol, and growth hormone responses above those observed in a resting session. Only growth hormone demonstrated a significant increase in response to resistance exercise compared to rest. Clearly, more research is needed to elucidate the biological importance of these transient increases and to further clarify differences in hormone responses between endurance and resistance exercise.

References


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