Effect of training status and exercise mode on endogenous steroid hormones in men

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Tremblay, Mark S., Jennifer L. Copeland, and Walter Van Helder. Effect of training status and exercise mode on endogenous steroid hormones in men. J Appl Physiol 96: 531–539, 2004.—The purpose of this study was to determine the acute anabolic and catabolic hormone response to endurance and resistance exercise bouts of equal volume in subjects with differing training status. Twenty-two healthy men were recruited who were either resistance trained (n = 7), endurance trained (n = 8), or sedentary (n = 7). Three sessions were completed: a resting session, a 40-min run at 50–55% maximal oxygen consumption, and a resistance exercise session. Expired gases were monitored continuously during exercise, and the endurance and resistance exercise sessions were individually matched for caloric expenditure. Blood samples were drawn before exercise and 1, 2, 3, and 4 h after the start of the exercise. Plasma was analyzed for luteinizing hormone, dehydroepiandrosterone sulfate, cortisol, and free and total testosterone. Androgens increased in response to exercise, particularly resistance exercise, whereas cortisol only increased after resistance exercise. Dehydroepiandrosterone sulfate levels increased during the resistance exercise session and remained elevated during recovery in the resistance-trained subjects. Endurance-trained subjects displayed less pronounced changes in hormone concentrations in response to exercise than resistance-trained subjects. After an initial postexercise increase, there was a significant decline in free and total testosterone during recovery from resistance exercise (P < 0.05), particularly in resistance-trained subjects. On the basis of the results of this study, it appears that the endogenous hormone profile of men is more dependent on exercise mode or intensity than exercise volume as measured by caloric expenditure. The relatively catabolic environment observed during the resistance session may indicate an intensity- rather than a mode-dependent response.

In light of these potential benefits, exogenous anabolic-androgenic steroids may be prescribed to elderly or ill individuals (3, 33) or abused by athletes (22). Unfortunately, there are risks associated with the use of exogenous steroids, such as renal and hepatic toxicity, gynecomastia, and adverse effects on blood lipids, that may outweigh the benefits (22). The goals of optimal health and performance may be better served by determining what type of exercise should be practiced, or avoided, to maximize the availability of naturally occurring anabolic hormones.

Endurance-trained men tend to have lower levels of testosterone compared with sedentary controls (11, 32), whereas resistance-trained subjects have been shown to have higher basal testosterone levels (13, 20). Cross-sectional work by Arce et al. (2) found that both endurance- and resistance-trained subjects had lower testosterone levels than sedentary control subjects. The main signaling peptide of testosterone, luteinizing hormone (LH), may also be altered by training, with significantly elevated levels reported in endurance-trained men (11). Other research has focused on the ratio of testosterone to cortisol, which has been reported to both increase (1) and decrease (12) during resistance training.

Testosterone concentrations have been shown to increase after an acute bout of resistance (18, 21, 25) or endurance exercise (10, 16). In response to prolonged endurance exercise (e.g., a marathon), testosterone levels will typically decline (9, 17). Others have reported no change in testosterone after resistance exercise (27). There has been considerably less research looking at adrenal androgens; however, DHEAS has been shown to increase in response to endurance exercise (17).

In contrast, Hakkinen et al. (14) found no change in dehydroepiandrosterone after resistance exercise in men.

In many cases, conflicting results can be attributed to differences in the mode or volume of exercise or in the training status of the subjects. There are few studies that have looked at the interaction between training status and mode of exercise in determining the hormonal response to exercise. Previous work comparing hormone responses between endurance and resistance exercise in men and women used exercise bouts of equal duration, which does not necessarily reflect equal energy expenditure or work output (6, 16), and the total volume of exercise may contribute to differences observed in the hormone response to various exercise protocols (27). Hackney et al. (10) compared the hormone response between bouts of continuous and interval cycling exercise of equal work outputs, but they used only endurance-trained subjects. It is known that training status can influence the hormone response to exercise, but it is

There are a number of reasons why it could be beneficial to manipulate the concentrations of circulating anabolic hormones and the anabolic-to-catabolic hormone ratio in men. From the perspective of an athlete, an increase in anabolic-androgenic hormones can improve performance by decreasing body fat and increasing lean body mass and muscular strength (22). During aging, there is a decline in anabolic-androgenic hormones, namely testosterone and dehydroepiandrosterone sulfate (DHEAS), that may have significant negative effects on body composition, physical function, and libido in men (31). Increased anabolic hormone concentrations could slow the aging process and maintain quality of life in older individuals. Certain age-related disease states that are catabolic in nature, such as osteoporosis or sarcopenia, could be attenuated by an increase in anabolic hormone concentrations (31).

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not clear whether the mode of training can affect the hormone response to different modes of exercise. This information could be useful in designing training programs that will result in the most favorable ratio of anabolic and catabolic hormones. Therefore, the objective of this study was to determine the acute steroid hormone response to endurance and resistance exercise bouts of equivalent volume in subjects with differing training status. We hypothesized that androgens would increase with exercise and that resistance exercise would result in a more anabolic hormonal environment than endurance exercise. We also expected that sedentary subjects would have a greater hormone response than either resistance- or endurance-trained subjects in response to both modes of exercise.

METHODS

Subjects

Twenty-two healthy men between the ages of 18 and 55 volunteered for this research. Subjects were either resistance trained (n = 7), endurance trained (n = 8), or sedentary (n = 7). The resistance-trained subjects were weight training in excess of 7 h/wk, whereas the endurance-trained subjects were running a minimum of 75 km/wk at the time of testing. Control subjects were sedentary and had not been involved in any regular exercise for at least the previous 12 mo, and they remained sedentary for the duration of the study. The training schedule of all trained subjects was unchanged during the study. Subjects kept a training diary, beginning 4 wk before the start of testing, and were asked to record changes in body mass or any stressful life events during the study period. Body mass was also recorded at the start of each session.

All subjects were screened for contraindicating health problems or pharmaceutical use and were cleared for unrestricted physical activity by a physician. The experimental protocol was approved by the Institutional Review Board, and all subjects gave written informed consent.

Experimental Design

Figure 1 illustrates the experimental design of the study. A specified start time between 1500 and 1800 was determined for each subject, and all four sessions began at their predetermined start time. Late afternoon was chosen because it best represented the typical time period during which the subjects trained and because the diurnal variation of testosterone is minimized during this time (26). During session 1, baseline anthropometric and fitness measurements were obtained. During session 2, subjects rested quietly. A resting blood sample was drawn at 0.5 h, and subsequent blood samples were drawn each hour for the next 4 h (Fig. 1). This provided individual baseline hormone levels and diurnal variation patterns across the testing period. Endurance and resistance exercise bouts were completed during sessions 3 and 4 respectively. The exercise sessions were matched according to caloric expenditure (calculated from expired gases), and blood samples were drawn at the same times as during the control session (session 2). Each testing session was separated by at least 1 wk.

Baseline Anthropometric and Fitness Measurements

Height, body mass, and skinfold thicknesses were taken following the protocols outlined in the Canadian Physical Activity, Fitness and Lifestyle Appraisal (5). Strength measurements were performed by using a modified Hydra-Gym series HI-311 apparatus (Hydra-Fitness Industries, Belton, TX). Peak force for chest press-pull and knee extension-flexion were determined by using a resistance setting of six, which corresponds to an angular velocity of 40°/s (28).

### Table

<table>
<thead>
<tr>
<th>Session 1</th>
<th>Anthropometric measurements, strength assessment, VO₂max</th>
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<tbody>
<tr>
<td>0</td>
<td>0.5 hrs</td>
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<tr>
<td>rest</td>
<td>time 0</td>
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<table>
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<tr>
<th>Session 2</th>
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<tr>
<td>↓1 week</td>
<td>40 minute run</td>
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<table>
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<tr>
<th>Session 3</th>
<th>40 minute run</th>
<th>RECOVERY</th>
</tr>
</thead>
<tbody>
<tr>
<td>↓1 week</td>
<td>Resistance exercise</td>
<td></td>
</tr>
</tbody>
</table>

= BLOOD SAMPLE

Fig. 1. Summary of the experimental design. VO₂max, maximal oxygen consumption.

Maximal aerobic power was determined by using a progressive, incremental treadmill protocol. Speed was held constant at each subject’s self-determined pace, and the grade of the treadmill (model 24-72, Quinton Instruments, Seattle, WA) was increased 2% every 2 min for 8 min and then 1% every minute until volitional fatigue. Minute ventilation (VE) was determined by a Rayfield airflow meter (Rayfield Equipment, Waitsfield, VT) located on the inspired side. Gas analysis was done by using Applied Electrochemistry analyzers (S-3A1 oxygen analyzer, CD-3A carbon dioxide analyzer, Ametek, Pittsburgh, PA). VE, oxygen consumption (VO₂), carbon dioxide production (VCO₂), and respiratory exchange ratio (RER) were monitored, as well as cumulative caloric expenditure based on RER and VO₂ values.

Endurance Exercise Session

During session 3, subjects completed a 40-min jog at 50–55% of maximal VO₂ (VO₂max). The intensity was achieved by increasing level running speed or grade. Expired gases were monitored continuously, and cardiorespiratory and metabolic calculations were based on a 60-s sampling period. Heart rate was monitored by using a telemetric heart rate monitor (Polar, Kempele, Finland). The first postexercise blood sample was collected within 10 min of exercise cessation.

Resistance Exercise Session

In session 4, subjects completed a bout of resistance exercise. The volume of the resistance exercise was matched according to the total caloric expenditure of the run session. Caloric expenditure was monitored by the continuous measurement of expired gases. The resistance
exercise session consisted of a circuit of seven exercises. Knee extension-flexion, chest press-pull, and shoulder press-pull were all performed on the modified Hydra-Gym at a resistance setting of six. In addition, biceps curls, weighted abdominal crunches, dead-lift calf-raises, and triceps dumbbell presses were performed with free weights that corresponded to each subject’s 10-repetition maximum. Subjects cycled through the circuit until they matched their caloric expenditure from session 3. The number of sets, repetitions, and mass used were recorded for each subject. The length of the resistance session varied between subjects so that the first postexercise blood sample occurred shortly after exercise cessation in some subjects and in other subjects it occurred during the final portion of their resistance exercise. The time required for the subjects to complete the volume-matched resistance exercise session did not differ significantly between groups (sedentary = 66.3 ± 12.2, endurance trained = 64.9 ± 11.0; resistance trained = 62.9 ± 7.0 min).

Blood Collection and Analysis

Blood samples were collected through an indwelling venous catheter inserted in an arm vein, and 10 ml of blood were drawn for each sample. The catheter was maintained with a heparinized saline lock. Initial blood samples were drawn 30 min after catherer insertion. All samples were taken with subjects in a seated position in a climate-controlled environment (21°C). Samples were analyzed in duplicate for hematocrit, and then the samples were centrifuged and the plasma was stored at −80°C until assayed.

All plasma samples were analyzed in duplicate. Commercial radioimmunoassays (RIA) were used to analyze total testosterone (ICN Biomedicals, Aurora, OH), free testosterone (Diagnostic Products, Los Angeles, CA), DHEAS (ICN Biomedicals), and cortisol (Kallestad Laboratories, Chaska, MN). LH was determined by immunoradiometric (IRMA) assay (Diagnostic Products). Duplicate samples with a coefficient of variation >5% for the RIAs or >10% for the IRMA were reanalyzed. Interassay variation, for the low and high controls, respectively, was 5.9 and 0.3% for LH, 10.7 and 9.8% for DHEAS, 6.9 and 5.7% for cortisol, 13.3 and 5.5% for total testosterone, and 27.3 and 5.7% for free testosterone. To minimize the effects of interassay variation, all samples from one subject were analyzed in the same assay.

Statistical Analyses

All statistical analyses were performed by using SAS (version 6, SAS Institute, Cary, NC). All data are presented as means ± SD, and statistical significance was set at P < 0.05. Repeated-measures ANOVA was used to determine whether there were significant differences among groups and sessions across time. Total testosterone-to-cortisol, free testosterone-to-cortisol, and DHEAS-to-cortisol ratios were calculated and compared in the same manner as individual hormones. The area under the hormone-time curve (AUC) was calculated for each subject in each session. A three × three (group × session) ANOVA was used to look for differences in AUC results. Wherever significant main effects were found a Tukey’s post hoc analysis was used.

RESULTS

Subject Characteristics

Table 1 shows the mean anthropometric and fitness data by group. The resistance-trained subjects were significantly heavier and stronger than the endurance trained or sedentary subjects. The endurance-trained subjects had significantly greater maximal aerobic power. Body mass did not change before or during the study, and the diaries of stressful life events did not reveal any unusual changes in stress levels during the study.

Exercise Sessions

Cardiorespiratory data were collected every minute during the exercise sessions, and the individual means for each session were averaged by group. The group data are presented in Table 2. The 40-min run resulted in greater VO2 and VCO2 and lower RER than the resistance exercise. Endurance-trained subjects had significantly higher relative VO2 values and significantly lower RER during the run compared with resistance-trained and sedentary subjects. Resistance-trained subjects had a higher mean heart rate during resistance exercise compared with endurance-trained subjects.

Table 1. Subject characteristics

<table>
<thead>
<tr>
<th>Variable</th>
<th>Sed (n=7)</th>
<th>Res (n=7)</th>
<th>End (n=8)</th>
</tr>
</thead>
<tbody>
<tr>
<td>Age, yr</td>
<td>27.2±7.3</td>
<td>22.9±3.5</td>
<td>31.0±11.0</td>
</tr>
<tr>
<td>Height, cm</td>
<td>174.0±8.1</td>
<td>180.5±10.3</td>
<td>171.9±3.7</td>
</tr>
<tr>
<td>Body mass, kg</td>
<td>70.8±12.5</td>
<td>86.3±10.5*</td>
<td>67.4±9.0</td>
</tr>
<tr>
<td>Sum of skinfolds, mm</td>
<td>50.0±13.8</td>
<td>55.2±23.7</td>
<td>41.6±16.2</td>
</tr>
<tr>
<td>V̇O2max, ml/kg · min⁻¹</td>
<td>51.2±4.5</td>
<td>53.5±6.6</td>
<td>69.4±5.0‡</td>
</tr>
<tr>
<td>Knee extension peak torque, Nm</td>
<td>314±61</td>
<td>439±72*</td>
<td>254±7.4</td>
</tr>
<tr>
<td>Knee flexion peak torque, Nm</td>
<td>137±49</td>
<td>203±52*</td>
<td>139±3.5</td>
</tr>
<tr>
<td>Elbow extension peak force, N</td>
<td>899±206</td>
<td>1,247±289†</td>
<td>1,061±236</td>
</tr>
<tr>
<td>Elbow flexion peak force, N</td>
<td>620±154</td>
<td>942±283†</td>
<td>735±150</td>
</tr>
</tbody>
</table>

Values are means ± SD; n, no. of subjects. Sed, sedentary subjects; Res, resistance-trained subjects; End, endurance-trained subjects; V̇O2max, maximal oxygen consumption. *Res > Sed and End; P < 0.05. †Res > Sed; P < 0.05. ‡End > Sed and Res; P < 0.05.

Table 2. Average values for cardiorespiratory data during the 40-min run and resistance exercise session

<table>
<thead>
<tr>
<th>Variable</th>
<th>Sed (n=7)</th>
<th>Res (n=7)</th>
<th>End (n=8)</th>
<th>Total (n=22)</th>
</tr>
</thead>
<tbody>
<tr>
<td>VO2, l/min</td>
<td>1.8±0.3</td>
<td>2.4±0.4*</td>
<td>2.4±0.3*</td>
<td>2.2±0.4*</td>
</tr>
<tr>
<td>VCO2, l/min</td>
<td>1.7±0.2</td>
<td>2.2±0.4*</td>
<td>2.0±0.2</td>
<td>2.0±0.4*</td>
</tr>
<tr>
<td>ml/kg · min⁻¹</td>
<td>26.0±2.9</td>
<td>28.4±3.6</td>
<td>35.3±1.5c</td>
<td>30.1±4.9c</td>
</tr>
<tr>
<td>RER</td>
<td>0.92±0.04d</td>
<td>0.91±0.04d</td>
<td>0.85±0.04</td>
<td>0.89±0.05</td>
</tr>
<tr>
<td>HR, beats/min</td>
<td>144±17</td>
<td>139±10</td>
<td>130±12</td>
<td>137±14</td>
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<tr>
<td>VO2, l/min</td>
<td>36.1±10.3</td>
<td>53.2±13.8‡</td>
<td>41.0±8.1</td>
<td>43.3±12.6</td>
</tr>
<tr>
<td>VCO2, l/min</td>
<td>1.1±0.3</td>
<td>1.6±0.4‡</td>
<td>1.5±0.3</td>
<td>1.4±0.4</td>
</tr>
<tr>
<td>ml/kg · min⁻¹</td>
<td>16.1±3.2</td>
<td>18.4±3.4</td>
<td>21.7±2.7</td>
<td>18.9±3.8</td>
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<tr>
<td>RER</td>
<td>1.0±0.08</td>
<td>0.95±0.10</td>
<td>0.91±0.07</td>
<td>0.95±0.09b</td>
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<tr>
<td>HR, beats/min</td>
<td>140±10</td>
<td>148±12‡</td>
<td>124±20</td>
<td>137±18</td>
</tr>
</tbody>
</table>

Values are means ± SD; n, no. of subjects. VO2, minute ventilation; VCO2, carbon dioxide production; RER, respiratory exchange ratio; HR, heart rate. a Run > resistance, P < 0.0001. bResistance > Run, P < 0.002. cRes and End > Sed, P < 0.005. dRes > Sed, P < 0.005. eEnd > Res and Sed, P < 0.001. fRes and Sed > End, P < 0.05. gEnd > Sed, P < 0.01. hRes > End, P > 0.005.
Hormonal Responses

Hormone concentrations for all subjects were within the clinical reference range for all hormones. To compare the hormone responses between exercise sessions and groups, the hormone data were normalized and are presented as absolute change from the preexercise sample.

**LH.** Figure 2 shows the changes in LH, by group and across time, for the three sessions. When all subjects were combined, there was a significant main effect for session, with the resistance exercise session resulting in greater LH concentrations than the rest or the run. In the resistance-trained subjects there was a significant increase in LH during recovery from the run.

**DHEAS.** When all subjects were analyzed together, the resistance exercise session resulted in significantly greater DHEAS concentrations than rest or the run (Fig. 3). The levels of DHEAS during the resistance exercise session were significantly greater in resistance-trained subjects compared with sedentary or endurance trained. During the run session, endurance-trained subjects showed greater DHEAS concentrations than resistance-trained subjects. DHEAS levels remained elevated in recovery after resistance exercise in resistance-trained subjects.

**Cortisol.** The concentrations of cortisol tended to decline across time, particularly in the resting session, which is consistent with the typical diurnal pattern of cortisol. However, when all subjects were analyzed together, cortisol concentrations were significantly higher in the resistance exercise session compared with the rest or the run, and the concentration was higher in the run session compared with rest (Fig. 4). There were no significant group differences in the cortisol concentrations, although there was a significant group \( \times \) session interaction that indicated a dampened response to resistance exercise in endurance-trained subjects.

**Total testosterone.** There was an increase in total testosterone after exercise, particularly after resistance exercise (Fig. 5). There was a significant session \( \times \) time interaction due to the pronounced decline in total testosterone during recovery from resistance exercise. AUC results indicated that the sedentary subjects had significantly greater total testosterone concentrations compared with endurance-trained or resistance-trained subjects.

**Free testosterone.** The changes in free testosterone across sessions closely matched the changes in total testosterone. When all subjects were analyzed together, free testosterone was significantly greater during the resting session than during the run or resistance exercise session (Fig. 6). As seen with total testosterone, there was a significant decline in free testosterone during recovery from resistance exercise, despite an initial increase after exercise. Testosterone increased back to baseline levels by time 4 (4.5 h) after resistance exercise.

**Ratios.** The total and free testosterone-to-cortisol ratios were significantly higher during the resting session and the run compared with the resistance exercise (Fig. 7). The DHEAS-to-cortisol ratio was significantly greater during rest than either exercise session and was also greater during the run compared with the resistance session. There were no significant differences between groups for any of the ratios.

**Plasma Volume Changes**

Hematocrit levels were significantly greater during the resistance exercise session than the resting session and were higher at time 1 than time 0, 2, 3, or 4 \( (P < 0.05; \text{see Fig. 1}) \) in
the resistance exercise session. All groups showed similar changes in hematocrit after exercise, but there was a significant group × session interaction, indicating that resistance trained subjects had greater increases in hematocrit after resistance

Fig. 3. Absolute change in dehydroepiandrosterone sulfate (DHEAS; normalized to a baseline of 0) across time in sedentary subjects (A), endurance-trained subjects (B), and resistance-trained subjects (C), during rest (○), a 40-min run (●), and an equal volume of resistance exercise (▲). Values are means ± SE. Letters indicate significant effects (P < 0.05): a = ▲ time 1 > time 0; b = ▲ time 1 > time 0, 2, 3, 4; c = ▲ time 1 > time 0, 2; d = ▲ time 3, 4 > time 0; e = ▲ time 3 < time 0, 1.

Fig. 4. Absolute change in cortisol (normalized to a baseline of 0) across time in sedentary subjects (A), endurance-trained subjects (B), and resistance-trained subjects (C) during rest (○), a 40-min run (●), and an equal volume of resistance exercise (▲). Values are means ± SE. Letters indicate significant effects (P < 0.05): a = ▲ time 1 > time 0, 3, 4; b = ▲ time 2 > time 0, 4; c = ● time 0 > time 2, 3, 4; d = ▲ time 0 > time 3, 4.
Fig. 5. Absolute change in total testosterone (normalized to a baseline of 0) across time in sedentary subjects (A), endurance-trained subjects (B), and resistance-trained subjects (C), during rest (●), a 40-min run (■), and an equal volume of resistance exercise (▲). Values are means ± SE. Letters indicate significant effects (P < 0.05): a = time 1 > time 2, 3; b = time 1 > time 0; c = time 2 < time 0, 1, 4.

Fig. 6. Absolute change in free testosterone (normalized to a baseline of 0) across time in sedentary subjects (A), endurance-trained subjects (B), and resistance-trained subjects (C), during rest (●), a 40-min run (■), and an equal volume of resistance exercise (▲). Values are means ± SE. Letters indicate significant effects (P < 0.05): a = time 1 > time 2; b = time 1 > time 0, 2, 3, 4; c = time 2 < time 0.
exercise. When hormone data were analyzed after correction for changes in plasma volume (29), it resulted in only small changes in the results, which suggests the majority of hormone responses to exercise could not be explained by hemoconcentration. Because we believe it is important to know the concentrations of hormone that the target tissues are exposed to regardless of the mechanism (10), only uncorrected results are presented.

**AUC**

The results of the AUC analyses generally supported the results of the repeated-measures ANOVA. Cortisol AUC was significantly greater during the resistance exercise session than the rest or the run, whereas total and free testosterone AUC was significantly greater in the run session than the resistance or rest session. \(P < 0.05\). There were no significant group differences found in the AUC for any hormone.

**DISCUSSION**

Sedentary, resistance-trained, and endurance-trained subjects were used in this study to identify differences in testosterone, LH, DHEAS, and cortisol, as well as the ratios of testosterone and DHEAS to cortisol. These hormones were evaluated both at rest and during recovery from a 40-min run and a resistance exercise session. Resistance-trained subjects tended to have higher androgen levels and slightly higher LH levels. Androgens increased in response to exercise, particularly resistance exercise, whereas cortisol only increased after resistance exercise. DHEAS levels increased during the resistance exercise session and remained elevated during recovery in the resistance-trained subjects. LH showed a delayed increase during recovery from the run in resistance trained subjects. Endurance-trained subjects displayed less pronounced changes in hormone concentrations in response to exercise. There was a significant decline in free and total testosterone during recovery from resistance exercise, and the ratios of anabolic hormones (free and total testosterone and DHEAS) to cortisol were lower during resistance exercise, which, paradoxically, suggests a less anabolic environment.

This study was unique in that it compared the hormonal response to different modes of exercise in subjects who had very different training histories. To our knowledge, this is also the first study to compare the endocrine response to a bout of resistance and endurance exercise that were equated on the basis of caloric expenditure. Previous studies that have compared endurance and resistance exercise matched the exercise protocols for duration and perceived exertion (16). The sampling procedure used in the present study was designed to provide a “snapshot” of the hormone exposure after a bout of exercise.

Jensen et al. (16) found that testosterone increased significantly in men after both resistance and endurance exercise and returned to the resting level within 2 h. The magnitude and pattern of the change in testosterone were almost identical between the resting and endurance sessions. This is in contrast to the present results where the testosterone response to resistance exercise was greater than the response to the endurance exercise session. This suggests that exercise intensity may be more predictive of the testosterone response to exercise than total energy expenditure, because Jensen et al. (16) attempted to equate the two exercise sessions on duration and intensity. In the present study, the two sessions were of equal caloric expenditure but not necessarily of equal intensity. In fact, the intensity of the run session \((50–55\% \text{ of } \dot{V} \text{O}_2 \text{ max})\) may have been unusually easy for the endurance-trained sub-

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**Fig. 7.** Values, for all subjects combined, for total testosterone-to-cortisol (A), free testosterone-to-cortisol (B), and DHEAS-to-cortisol (C) ratios across time, during rest (●), a 40-min run (■), and an equal volume of resistance exercise (▲). Values are means ± SE. Letters indicate significant effects \((P < 0.05)\):

- a = ● time 4 > time 0, 1, 2, and 3; b = ■ time 4 > time 0, 1, 2; c = ● time 3 > time 0, 1, 2; d = ▲ time 3 > time 1.
Hackney et al. (10) used endurance-trained subjects (runners and tennis players). In this study, we used subjects that were specifically resistance trained or endurance trained, and they represented relative extremes with regard to training routines. The results presented in Table 1 illustrate the very different physical fitness characteristics of the three groups. In general, the endurance-trained subjects demonstrated less hormonal responsiveness to the exercise bouts, but as discussed previously, this may be explained by a relatively light exercise intensity, particularly in the run session. The greater testosterone response in the resistance-trained subjects during resistance exercise may reflect a training adaptation or a biological difference in these subjects that enhances the anabolic stimulus from resistance exercise.

After the initial postexercise increase in total and free testosterone, the recovery levels declined below the resting levels, particularly after initial exercise in resistance-trained subjects. This is similar to Kraemer et al. (19), who found increased testosterone levels during, and up to 15 min after, resistance exercise, after which testosterone began to decline back to preexercise levels. In the present study, cortisol levels increased sharply after resistance exercise and did not return to baseline until the time 4 sample. Nindl et al. (23) suggest that decreased testosterone and elevated cortisol levels will enhance lipolysis and protein catabolism to mobilize fuels for recovery and regeneration after exercise. The present results support this theory because the resistance exercise would likely result in more muscular stress and damage than the run session, which would explain why the testosterone and cortisol responses are more pronounced in the resistance session. By the end of the session (time 4), both total and free testosterone and cortisol had returned to baseline levels.

The changes in LH levels did not appear to correspond to the testosterone changes, as would be expected. However, these results should be interpreted cautiously because of the pulsatile nature of LH secretion. Nindl et al. (23) monitored hormone levels during the night after a bout of resistance exercise and found that both testosterone levels and LH levels were lower during the night after exercise. We cannot rule out similar changes in our subjects because the sampling protocol used here did not monitor nocturnal hormone levels. The subjects used by Nindl et al. were trained, physically fit, subjects. It would be interesting to know whether the same nocturnal hormone response would occur in endurance-trained or sedentary subjects. Our results suggest that resistance-trained subjects have a greater testosterone and cortisol response to resistance exercise. It is possible that one adaptation to training is more pronounced hormone changes that will enhance the mobilization of fuel for recovery.

DHEAS levels increased in response to exercise, although the response was greater after resistance exercise than the run. Levels of DHEAS remained elevated after resistance exercise in resistance-trained subjects. This is consistent with the results of other investigations (7, 8, 30). Hakkinen et al. (14) found no change in DHEAS after resistance exercise in middle-aged or elderly men. Our subjects were significantly younger than those used by Hakkinen et al., which may explain the difference in response. DHEAS is known to decline dramatically with age in men (24).

The ratio of androgens to cortisol was significantly smaller during the resistance exercise session than the other sessions, mainly as a result of the large postexercise increase in cortisol and also the decline in testosterone levels during recovery. This result is somewhat unexpected because we typically associate resistance exercise with anabolism. However, as discussed previously, the lower androgen-to-cortisol ratio may be beneficial from the perspective of fuel mobilization (23). It is possible that the action of glucocorticoids, such as cortisol, is inhibited by either anabolic-androgenic hormones or by exercise (15). Both resistance and endurance training have been shown to attenuate the muscle catabolism associated with high levels of glucocorticoids (15). The mechanism of this effect is not clearly understood, but it may be that regular exercise diminishes the gene expression typically induced by glucocorticoids (15). If this is the case, then the increased cortisol observed after resistance exercise may not have the expected negative effects on muscle in trained subjects.

It is also important to note that the androgen-to-cortisol ratio increased during recovery from exercise in all subjects, and it is possible the hormonal milieu became more anabolic at times beyond what were observed with our sampling protocol. Also, because other important anabolic hormones were not measured (growth hormone, insulin-like growth factor I), we cannot conclude that exercise resulted in a predominantly catabolic environment. This study design is also unable to assess biological changes outside the circulation (e.g., autocrine or paracrine activity).

On the basis of the results of this study, it appears that the circulating endogenous hormone profile is more dependent on exercise mode or intensity than exercise volume as measured by caloric expenditure. The relatively catabolic environment observed during the resistance session may indicate an intensity rather than mode dependent response. This study also provides evidence that hormone levels and exercise-induced hormone changes are different in subjects of different training status. Endurance-trained subjects displayed a less pronounced hormone response to exercise, whereas resistance-trained subjects demonstrated a more pronounced hormone response to exercise. These differences may be related to specific exercise intensity characteristics or training adaptations. Of course, it is not known whether training per se alters hormone profiles or whether athletes are self-selected into certain activities because their physical or physiological characteristics predispose them to success (4). More longitudinal investigations are necessary before we can begin to answer these questions.

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