20-Hour Physiological Responses to a Single Weight-Training Session

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Reference Data

ABSTRACT
This study compared trained and untrained subjects' responses to a single bout of weight-training exercise of the same relative intensity. Young men (N=21) were divided into three similar groups based on their 1-RM dead-stop squat. Group 1 (n=8) was made up of well-trained lifters (TWL). Group 2 (n=7) were untrained subjects who took part in the exercise session (UTWL). Group 3 (n=6) served as untrained controls (C). Food and drink were controlled and measured. Heart rate, profile-of-moods states, and blood samples were taken three times before and five times after exercise. Blood was analyzed for hematocrit (Hct), lactate (HLa), blood glucose (glu), free fatty acids (FFA), insulin (I), glucagon (G), cortisol (Cort), growth hormone (hGH), epinephrine (E), and norepinephrine (NE) using standard methods. Data were analyzed using ANOVA with a repeated-measures design. The alpha level was p<0.05. The single exercise session elicited significantly higher HR, HLa, glu, FFA, hGH, E, and NE values in the TWL and UTWL 5 min postexercise than in the C group. UTWL Cort values were significantly higher than the TWL or C groups at 5 min postexercise. At 1-1/2 hrs postexercise the UTWL had significantly higher values for HR, HLa, and Cort than the other groups. The UTWL had significantly higher FFA concentrations compared to the Controls at 20 hrs postexercise. Shifts in plasma volume did not adequately account for the observed changes in HLa, glu, or hormones. These data suggest that the magnitude of the response to weight-training exercise is related to the subject's trained state. Additionally, these data indicate that weight training can stimulate increased FFA concentrations at various times during recovery.

Key Words: weight training, substrates, hormones

Introduction
Energy substrate and hormonal responses to aerobic exercise and training have been researched extensively (1, 20, 21, 45, 61). However, relatively few studies have examined energy substrate and hormonal responses to weight training (16, 22, 31, 55, 66). This is especially true for postexercise recovery periods. Some data (58) and review of the literature suggest (59) that highly weight-trained subjects respond to absolute workloads and intensities with a diminished stress response compared to untrained subjects. It may be possible that trained subjects would show a diminished stress response to similar relative intensities (% 1-RM). It has also been suggested (59) that the volume of training can influence physiological adaptations and exercise response; physiological adaptations at rest or exercise responses may be more pronounced among athletes training with high loads (load = repetitions x mass lifted) (59). The purpose of this study was to examine the effects of weight-training experience on resting, exercise, and recovery hormonal and substrate responses.

Methods
Subjects
Written informed consent was obtained from all subjects. Twenty-two males volunteered for the study and were divided into three groups. Group 1 consisted of 8 well-trained subjects (TWL) averaging 3.75 years experience as weightlifters. They ranged from 18 to 36 years of age and had followed the same organized weight-training program (a periodization based workout) (51, 57, 60) for an average of 1 year, including participating in state and national weightlifting competitions. None had used any ergogenic aids for at least 2 years prior to the study. Unlike subjects in other studies (e.g., 28), these weightlifters represented a
more homogeneous group in terms of training. They had just completed a 3-week preparation phase (high loading) of training.

The remaining 14 volunteers were college-age males who had never trained as weightlifters, had not been involved in any regular physical activity, including resistance training programs, for at least 6 weeks prior to the study, and were considered untrained. They were divided into two equal groups based on their one-repetition maximum (1-RM) dead-stop squat (DSS) (defined as a concentric phase-only squat beginning with the bottom of the thigh approximately 2 cm below parallel to the floor). The squat was chosen because it is a commonly used large muscle mass exercise and should produce marked physiological stress (51). This method of squat exercise was performed in an effort to control for technique differences between the groups and is commonly used by weightlifters in training.

Seven subjects formed Group 2, the untrained weightlifters (UTWL), and participated in the weight-training workout. The remaining 7 subjects served as controls (C). One member of the C group became ill and was dropped from the study, leaving 6 control subjects. Efforts were made to match the groups on age, body mass, body composition, and height. The mean age and height of the groups were (±SEM), TWL = 25.5 ±5.0 yrs, 169.0 ±6.9 cm; UTWL = 20.0 ±2.2 yrs, 176.0 ±4.6 cm; C = 19.9 ±1.5 yrs, 172.2 ±5.5 cm; additional physical characteristics are shown in Table 1. There were no significant differences between groups for weight, percent body fat, fat mass, or lean body mass (Table 1). Thus the groups were matched on these variables.

Experimental Protocol
Three or 4 days prior to the study, all subjects were measured for body weight and height. Measurement of body composition employed the sum of skinfolds method (23). Body density (BD) was calculated using the Jackson-Pollock (23) sum of skinfolds equation (No. 4) for determining body density. Percentage of body fat was then determined by the Brozek and Keys formula (3). After a familiarization period the previous day, each subject's 1-RM DSS was determined using methods for 1-RM determination as described by Stone and O'Bryant (57). All subjects were given a list of guidelines and were asked to abstain from physical activity and caffeine beginning at noon on Day 1 and continuing for the length of the study.

On Day 1 at 5 p.m. all subjects and experimenters were assembled to review the experimental protocol (see Figure 1). Pulse rates were taken via radial palpation before each blood sample and the Profile of Mood States (POMS) (39) was completed after each blood sample. This procedure was repeated at each sample time during the study. On Day 2, after an 8 a.m. blood sample followed by breakfast, groups TWL and UTWL completed a workout (see Figure 2) based on their 1-RM DSS. Members of both groups were paired with Controls who observed the workout; this served as sham activity for the C (see Figure 1).

Meals
The four meals consumed during the study were prepared at a school cafeteria and were supervised and quantified by a registered dietitian. The menu for each meal was limited so that all subjects would

<table>
<thead>
<tr>
<th>DAY</th>
<th>TIME</th>
</tr>
</thead>
<tbody>
<tr>
<td>1</td>
<td>5:30 p.m.  Protocol</td>
</tr>
<tr>
<td></td>
<td>6:00 p.m.  Discussion</td>
</tr>
<tr>
<td></td>
<td>8:00 p.m.  Dinner</td>
</tr>
<tr>
<td>2</td>
<td>8:00 a.m.  Fasting</td>
</tr>
<tr>
<td></td>
<td>8:30 a.m.  Preexercise</td>
</tr>
<tr>
<td></td>
<td>10:00 a.m.  Exercise session</td>
</tr>
<tr>
<td>3</td>
<td>11:00 a.m.  5-min postexercise</td>
</tr>
<tr>
<td></td>
<td>12:30 p.m.  1.5-hr postexercise</td>
</tr>
<tr>
<td></td>
<td>1:00 p.m.  Lunch</td>
</tr>
<tr>
<td>6</td>
<td>5:00 p.m.  Fasting</td>
</tr>
<tr>
<td></td>
<td>5:30 p.m.  Dinner</td>
</tr>
<tr>
<td>7</td>
<td>8:00 p.m.  Fasting</td>
</tr>
<tr>
<td></td>
<td>8:00 a.m.  Blood collection: 1–8</td>
</tr>
</tbody>
</table>

Figure 1. Experimental protocol, showing the 3-day progression of the experiment. Blood was collected eight times (1–8) over a 36-hr period.
Stretch — 5 minutes

Please note all percentages below are based on the Dead-Stop Squat 1-RM determined earlier

<table>
<thead>
<tr>
<th>Dead-Stop Squats</th>
<th>Fri</th>
<th>Sat</th>
<th>Sat</th>
<th>Sat</th>
</tr>
</thead>
<tbody>
<tr>
<td>1 set of 10 reps at 40%</td>
<td>6 pm</td>
<td>8:30 am</td>
<td>1 pm</td>
<td>5:30 pm</td>
</tr>
<tr>
<td>1 set of 10 reps at 50%</td>
<td>2 - 1/2 min</td>
<td>rest between</td>
<td>20 ± 4</td>
<td></td>
</tr>
<tr>
<td>3 sets of 10 reps at 60%</td>
<td>1 min</td>
<td>rest between</td>
<td>20 ± 6</td>
<td></td>
</tr>
<tr>
<td>Each set</td>
<td></td>
<td></td>
<td>17 ± 1</td>
<td>28 ± 5</td>
</tr>
<tr>
<td>2 Minutes rest — seated</td>
<td></td>
<td></td>
<td>17 ± 1</td>
<td>36 ± 2</td>
</tr>
<tr>
<td>1/4 squats</td>
<td></td>
<td></td>
<td>17 ± 1</td>
<td>49 ± 4</td>
</tr>
<tr>
<td>1 set of 10 reps at 60%</td>
<td></td>
<td></td>
<td>31 ± 3</td>
<td>31 ± 3</td>
</tr>
<tr>
<td>3 sets of 10 reps at 75%</td>
<td></td>
<td></td>
<td>47 ± 2</td>
<td>49 ± 4</td>
</tr>
<tr>
<td>Each set</td>
<td></td>
<td></td>
<td>36 ± 1</td>
<td>31 ± 3</td>
</tr>
<tr>
<td>2 Minutes rest — seated</td>
<td></td>
<td></td>
<td>36 ± 1</td>
<td>31 ± 3</td>
</tr>
<tr>
<td>vertical jumps</td>
<td>1 min rest</td>
<td>between each set</td>
<td>1348</td>
<td></td>
</tr>
<tr>
<td>3 sets of 10 at body weight</td>
<td></td>
<td></td>
<td>1348</td>
<td></td>
</tr>
<tr>
<td>Each vertical jump is to be made from full squat to full extension with a maximum effort</td>
<td></td>
<td></td>
<td></td>
<td></td>
</tr>
</tbody>
</table>

Table 2

<p>| Percent Protein, Carbohydrate, and Fat Kcals and Total Kcal Consumed |
|----------------------------------------------------------|----------------|</p>
<table>
<thead>
<tr>
<th>Group</th>
<th>Fri</th>
<th>Sat</th>
<th>Sat</th>
<th>Sat</th>
</tr>
</thead>
<tbody>
<tr>
<td>Trained Pro.</td>
<td>11 ± 1</td>
<td>20 ± 1</td>
<td>17 ± 1</td>
<td>20 ± 4</td>
</tr>
<tr>
<td>weight-lifters</td>
<td>35 ± 5</td>
<td>45 ± 2</td>
<td>52 ± 6</td>
<td></td>
</tr>
<tr>
<td>Kcal</td>
<td>1678</td>
<td>366</td>
<td>1747</td>
<td>1568</td>
</tr>
<tr>
<td>± 85</td>
<td>± 43</td>
<td>± 217</td>
<td>± 155</td>
<td></td>
</tr>
<tr>
<td>Untrained Pro.</td>
<td>13 ± 2</td>
<td>19 ± 1</td>
<td>17 ± 1</td>
<td>20 ± 2</td>
</tr>
<tr>
<td>weight-lifters</td>
<td>48 ± 5</td>
<td>31 ± 3</td>
<td>47 ± 2</td>
<td>49 ± 4</td>
</tr>
<tr>
<td>Kcal</td>
<td>1354</td>
<td>364</td>
<td>1261</td>
<td>1348</td>
</tr>
<tr>
<td>±158</td>
<td>±40</td>
<td>±134</td>
<td>±131</td>
<td></td>
</tr>
<tr>
<td>Control Pro.</td>
<td>11 ± 2</td>
<td>16 ± 2</td>
<td>17 ± 1</td>
<td>17 ± 1</td>
</tr>
<tr>
<td>CHO</td>
<td>51 ± 6</td>
<td>34 ± 6</td>
<td>47 ± 3</td>
<td>54 ± 5</td>
</tr>
<tr>
<td>Fats</td>
<td>38 ± 5</td>
<td>50 ± 6</td>
<td>37 ± 2</td>
<td>29 ± 3</td>
</tr>
<tr>
<td>Kcal</td>
<td>1652</td>
<td>459</td>
<td>1648</td>
<td>1480</td>
</tr>
<tr>
<td>± 83</td>
<td>± 58</td>
<td>± 181</td>
<td>± 125</td>
<td></td>
</tr>
</tbody>
</table>

Figure 2. Weight-training exercise sessions.

consume the same types of food in approximately the same proportions of kilocalories from protein, carbohydrates, and fat (see Table 2). Plates were weighed using an Ohaus scale (Florham Park, NJ) before and after eating. Thus exact portions were recorded. Liquid amounts excluding water were also recorded. From these data, total kilocalories and percentage of kilocalories from fat, carbohydrate, and protein were determined via the Nutrition II software program (N-Squared Computing, Silverton, OR).

Psychological and Physiological Measurements

Profile of Mood States. The Profile of Mood States (POMS) was administered according to instrument instructions (39). Subjects were instructed to respond based on how they felt about each question “right now.”

Heart Rate (Pulse). Pulse rate was determined by radial artery palpation. Except for the immediate postexercise data collection, subjects were allowed to sit for 5 minutes before having their pulse rate taken.

Biochemical Analyses. Approximately 18 mL of blood was drawn from each subject at each sample time. Immediately after sampling, four microhematocrit capillary tubes were filled from the syringe (after removal of 20-g needle) to facilitate hematocrit (Hct) and lactate (HLa) determination. The remaining blood was then divided into two cooled centrifuge tubes. A tube with 12 mL of blood contained 60 μL EDTA and 120 μL of an antiprotease Traysol (Aprotinin, 500 KIU, Mobay Chemical Co., New York) and the 6 mL tube contained 60 μL of a pH 7.0 mixture of EGTA (12.4%) and glutathione (7.52%). The tubes were then centrifuged at 1500 x g for 20 min or 3000 x g for 10 min in a refrigerated centrifuge (4°C).

All of the plasma from the 6-mL tube was pipetted into one storage tube and immediately frozen (−10 to −15°C). The day after the experiment, the plasma was placed in ultralow temperature storage (−80°C) until assayed for catecholamines. The plasma from the 12-mL tube was divided among several small storage tubes and immediately frozen (−10 to −15°C). Estimated percent change in plasma volume was determined via previously described formula (65). Analyses were performed in duplicate. Lactate (HLa) was measured using a Yellow Springs Model 23L Lactate Analyzer (YSI, Yellow Springs, OH) according to the manual’s instructions (73). The intraassay coefficient of variation (CV) was <5%.

Nonesterified (or free) fatty acids (FFA) were determined using the NEFA in vitro enzymatic colorimetric method for the quantitative determination of nonesterified (or free) fatty acids (NEFA or FFA) (Wako Pure Chemical Industries, Dallas). The method was modified to use one-half of the reagents (2). Incubation at 37°C was accomplished via a Dubnoff Metabolic Incubator (Precision Scientific, Chicago). Absorbance at 550 nm was determined using a Perkin Elmer Lambda 3A UV-VIS Spectrophotometer (Oak Brook Instrument Div., Oak Brook, IL). Blood glucose (glu) was measured using a glucose colorimetric procedure from Sigma Diagnostics (St. Louis) and a Beckman DU-20 spectrophotometer (Beckman Instruments, Irvine, CA). Interassay coefficient of variation was 5.0% and intraassay CV was 4.1%.
Hormones were measured using standard radioimmunoassay (RIA) techniques (19). Measurement of human growth hormone (HGH) was made using a liquid phase RIA (Allegro HGH) from Nichols Institute (San Juan Capistrano, CA). Interassay CV was 3.4% and intraassay CV was 7.7%. The assay sensitivity was 0.06 μg/L. Dilutions of plasma displaced 125I-peg-HGH in parallel to the standard curve. Recovery of known amounts of hGH added to plasma was greater than 98%.

Cortisol (Cort) was measured using a solid phase RIA (Coat-a-Count, Diagnostic Products Corp., Los Angeles). The assay sensitivity was 0.55 nmol/L. Dilutions of plasma displaced 125I-cortisol in parallel to the standard curve. Recovery of known amounts of cortisol added to plasma was greater than 95%. Interassay CV was 4.4% and intraassay CV was 2.0%.

Insulin (I) was measured using the liquid phase double-antibody RIA method. The antibody was obtained from Dr. R.C. Adelman (University of Michigan), 125I-Insulin from New England Nuclear (Boston), and a porcine insulin standard from Novo Research Laboratories (Copenhagen, Denmark). Interassay CV was 3.1% and intraassay CV was 1.6%. Assay sensitivity was 30.85 pmol/L. Dilutions of plasma displaced 125I-insulin in parallel with the standard curve. Recovery of known amounts of insulin added to plasma was greater than 93%.

Glucagon (G) was measured using the single antibody liquid phase RIA method (19). The 30K antibody was obtained from Dr. R.H. Unger (Southeastern Medical School, Dallas), 125I-glucagon from New England Nuclear (Boston), and a porcine glucagon standard from Novo Research Laboratories (Copenhagen, Denmark). Interassay CV was 3.3% and intraassay CV was 2.3%. The assay sensitivity was 20 ng/L. Dilutions of plasma displaced 125I-glucagon in parallel to the standard curve. Recovery of known amounts of glucagon added to plasma was greater than 92%. Immunoreactivity values for hGH, Cort, I, and G were assessed via a micromedic 4/200 Automatic Gamma Counter (Horsham, PA) with a computer data reduction program.

Catecholamines were measured using high performance liquid chromatography (HPLC) with electrochemical detection (8). Determination of catecholamine levels was made using a Waters WISP 710B sample injector and Model 590 pump and a Coulòchem Model 5100A electrochemical detector and Model 5011 Analytical Cell from ESA (Bedford, MA). An IBM octadecyl type column (4.5 x 50 mm) filled with three micron particles was used for separation. The percent recovery for norepinephrine and epinephrine was 80% and 82%, respectively. Interassay CV was 13.5% and intraassay CV was 6.8%. All hormone assays were performed in the same endocrine laboratory that routinely makes these measurements.

**Statistical Analysis**

An ANOVA with repeated measures (Group \times Trials) procedure was used to compare all data for statistical purposes. A student Neuman-Keuls follow-up was used to test for significant group differences at specific times. Additionally, selected interactions were examined by testing gain scores across specific times using paired t tests. Significance was chosen at an alpha level of \( p \leq 0.05 \).

**Results**

**Physical Characteristics**

There was a significant age difference, with the TWL being older than the UTWL and C groups (M = 25.5 ±5.0 yrs for TWL; 20.0 ±2.2 yrs for UTWL; 19.9 ±1.5 yrs for C). The TWL group also was significantly shorter than the UTWL (TWL = 169.0 ±6.9 cm; UTWL = 176.0 ±4.6 cm) but was not significantly different from the C (172.2 ±5.5 cm). The TWL’s 1-RM squat (165.4 ±30.0 kg) was significantly greater than for the UTWL (102.5 ±20.0 kg) and the C (110.4 ±17.0 kg).

The differences between physical characteristics were not considered to be among possible reasons for differences in physiological or hormonal responses. Also, strength differences were not considered because, although total work was different, a weight training routine based on a percentage of each individual’s 1-RM DSS was used and non-significant 5-min postexercise lactate concentrations between TWL and UTWL groups seem to indicate equal relative workloads.

**Diet**

There were no significant differences between groups for kilocalories consumed or for percent of kilocalories coming from fat, carbohydrate, or protein (Table 2). Therefore diet was not considered one of the possible reasons for physiological or hormonal differences.

**Nonhormonal Responses**

Heart rate (HR) at the 11 a.m. sample period 5 min postexercise (Figure 3) was significantly lower for the TWL (112 ±3 bpm) than for the UTWL (123 ±3 bpm). At 71 ±4 bpm for the Controls, it was significantly lower than for both exercise groups at this time period. At the 12:30 p.m. sample period the UTWL heart rate (85 ±4 beats-min\(^{-1}\)) was still significantly higher than for the C (69 ±2 beats-min\(^{-1}\)), but the TWL (78 ±3 beats-min\(^{-1}\)) was not different from the C. At the 8 a.m. sample period on Day 3, the TWL (64 ±2 beats-min\(^{-1}\)) and UTWL (67 ±2 beats-min\(^{-1}\)) heart rates were significantly lower than for the C (74 ±2 beats-min\(^{-1}\)).

**Plasma Volume**

Plasma volume showed expected shifts as a result of weight-training exercise. No significant differences were noted between exercise groups at any time period, but the exercise groups showed significant differences compared to the C group at 10 a.m. and 12:30 p.m. (Table 3).
Figure 3. Heart rate response to meals and exercise (M ±SEM). Significant differences, P<0.05. Day 2, 11 a.m.: TWL < UTWL, C < TWL, UTWL; 12:30 p.m.: C < UTWL. Day 3, 8 a.m.: TWL, UTWL < C.

Table 3
Plasma Volume Changes (%)

<table>
<thead>
<tr>
<th>Group</th>
<th>3-4</th>
<th>4-5</th>
<th>5-6</th>
<th>6-7</th>
</tr>
</thead>
<tbody>
<tr>
<td>Trained weightlifters</td>
<td>±10</td>
<td>±17</td>
<td>±3</td>
<td>±4</td>
</tr>
<tr>
<td>Untrained weightlifters</td>
<td>±2</td>
<td>±1</td>
<td>±1</td>
<td>±1</td>
</tr>
<tr>
<td>Control*</td>
<td>±1</td>
<td>±2</td>
<td>±2</td>
<td>±2</td>
</tr>
</tbody>
</table>

Note. 3-4 = 10–11 a.m., 4-5 = 11 a.m.–12:30 p.m., 5-6 = 12:30–5 p.m., 6-7 = 5:30–8 p.m.

*Lactate. Lactate (HLa) at the 11 a.m. Day 2 sample period 5 min postexercise (Figure 4) was different between groups. The TWL (M = 9.20 ±0.66 mmol/L) and the UTWL (9.30 ±0.78 mmol/L) had significantly greater HLa concentrations than the C (0.8 ±0.14 mmol/L). At the 12:30 p.m. sample period, the TWL (0.96 ±0.12 mmol/L) and the C (0.55 ±0.07 mmol/L) had significantly lower HLa values than the UTWL (1.5 ±0.22 mmol/L).

*Hematocrit. No significant intergroup differences for hematocrit occurred during any of the sampling times.

Blood Glucose. Intergroup differences were found only at 11 a.m. on Day 2 for blood glucose (glu). The UTWL (M = 5.7 ±0.2 mmol/L) and the TWL (5.5 ±0.2 mmol/L) values were significantly higher than for the C (4.5 ±0.1 mmol/L) (see Figure 5).

Free Fatty Acids. Group means of the TWL and UTWL were significantly greater than those of the controls. Intergroup differences at specific times also occurred. For the preworkout sample at 10 a.m. on Day 2 (Figure 6), groups TWL (M =

Figure 4. Lactate response to meals and exercise (M ±SEM). Significant differences, P<0.05. Day 2, 11 a.m.: C < TWL, UTWL; 12:30 p.m.: C < TWL, UTWL.

Figure 5. Blood glucose response to meals and exercise (M ±SEM). Significant differences, P<0.05. Day 2, 11 a.m.: C < TWL, UTWL.

Figure 6. Free fatty acid response to meals and exercise (M ±SEM). Significant differences, P<0.05. Group difference: C < TWL, UTWL. Day 2, 10 a.m.: C < TWL, UTWL; 11 a.m.: C < TWL, UTWL. Selected interaction: 10 a.m. < 11 a.m. for UTWL. Day 3, 8 a.m.: C < UTWL.
46 ±1 mg/L and UTWL (43 ±3 mg/L) were significantly higher than the C (39 ±1 mg/L). For the postworkout 11 a.m. Day 2 sample, TWL (M = 50 ±3 mg/L) and UTWL (53 ±3 mg/L) were again significantly higher than the C (39 ±2 mg/L). The UTWL (71 ±6 mg/L) had significantly higher FFA concentrations than the C (49 ±3 mg/L) for the 8 a.m. Day 3 sample. The TWL (58 ±6 mg/L) was not different from either the UTWL or C groups. Additionally, a significant increase occurred in the UTWL group from 10 a.m. (43 ±2 mg/L) to 11 a.m. on Day 1 (53 ±3 mg/L); significant increases were not found for the other two groups.

Hormonal Responses

Insulin. On Day 2 at 5 p.m. (Figure 7), the TWL (M = 58 ±18 pmol/L) and the UTWL (85 ±13 pmol/L) had significantly lower concentrations than the C (253 ±16 pmol/L).

Glucagon. At the 5-min postexercise 11 a.m. sample (Figure 8), the C (M = 75 ±7 ng/L) was significantly higher than the TWL (57 ±6 ng/L). One TWL subject had G concentrations three times higher than any other subject at all time periods. TWL means ±SEM are shown without this subject (Figure 8). Additionally, a significant increase occurred for the TWL from 11 a.m. on Day 1 (57 ±6 ng/L) to 5 p.m. (95 ±10 ng/L); significant increases were not found for the other two groups.

Insulin/Glucagon Ratio. For the 5 p.m. Day 2 sample (Figure 9), the C group (M = 10.3 ±1.8) had a significantly higher value when compared to the TWL (1.7 ±0.6) and UTWL (3.7 ±0.9) groups.

Cortisol. At 8 a.m. on Day 2 (Figure 10), the TWL (M = 445 ±26 nmol/L) had significantly lower concentrations than the UTWL (582 ±46 nmol/L) but not the C (523 ±29 nmol/L). At the 11 a.m. sample period (5 min postexercise) the TWL (486 ±57 nmol/L) were not significantly different from either of the other groups, but the UTWL (600 ±58
nnmol/L) was significantly higher than the C (317 ±42 nmol/L). At the 12:30 p.m. sample period the UTWL group mean (614 ±55 nmol/L) was significantly greater than the TWL (432 ±65 nmol/L) and the C groups (267 ±31 nmol/L).

Growth Hormone. For the 11 a.m. sample period (5 min postexercise) (Figure 11), the TWL (M = 23.9 ±3.4 μg/L) was significantly lower than the UTWL (40.7 ±7.4 μg/L). The C group (6.3 ±2.7 μg/L) was significantly lower than both exercise groups.

Norepinephrine. At 8 p.m. on Day 1 (Figure 12), TWL (M = 3.6 ±0.3 nmol/L) was significantly higher than the C group (2.2 ±0.4 nmol/L), but neither were different from the UTWL (2.8 ±0.3 nmol/L). At 11 a.m. on Day 2 immediately postexercise, the TWL (17.5 ±1.6 nmol/L) and the UTWL group (15.8 ±2.7 nmol/L) were both significantly higher than the C (3.2 ±0.5 nmol/L). At 8 p.m. on Day 2 the C (3.3 ±0.3 nmol/L) was significantly higher than the UTWL (2.7 ±0.2 nmol/L). Neither was different from the TWL group (4.2 ±0.5 nmol/L).

Epinephrine. The 8 p.m. Day 1 sample (Figure 13) for the TWL (M = 6762 ±721 pmol/L) was significantly higher than either the UTWL group (4519 ±610 pmol/L) or the C (4497 ±352 pmol/L). For the 8 a.m. Day 2 sample the C (12870 ±1464 pmol/L) was significantly higher than the TWL (6304 ±906 pmol/L). The UTWL (9808 ±976 pmol/L) was not different from either TWL or C. At 10 a.m. Day 2 preexercise, the C (M = 10441 ±1784 pmol/L) was significantly higher than the TWL (4726 ±320 pmol/L), but neither were different from the UTWL (7892 ±875 pmol/L). At the 11 a.m. Day 2 postexercise sample both the TWL (10337 ±894 pmol/L) and the UTWL (9027 ±1270 pmol/L) were significantly higher than the C (5310 ±1220 pmol/L).

POMS. The TWL group (M = 14.0 ±5.6) had a significantly higher value for tension than the C (5.8 ±4.7) at the 5-min postexercise sample. The UTWL (9.1 ±5.3) was not different from the other groups. The 5-min postexercise sample for fatigue revealed significantly higher values for the TWL (11.1 ±6.6) and UTWL (12.4 ±5.7) groups when compared to the C (1.0 ±1.0). The TWL (6.7 ±5.8) and UTWL (6.3 ±3.3) fatigue values for the 12:30 p.m. Day 2 sample were also significantly higher than the C (1.0 ±1.4). The 5-min postexercise sample for total mood disturbance revealed significantly higher values for the TWL (33.0 ±11.3) and UTWL (39.4 ±16.9) groups when compared to the C group (13.5 ±17.9). No significant difference occurred between groups for confusion/bewilderment, depression/anxiety, anger/hostility, or vigor/activity.

Discussion

The single bout of weight training produced qualitatively similar changes in physiological and hormonal parameters among the TWL and UTWL
subjects. Measurements taken during the post-exercise recovery period provided useful information concerning adjustments following weight training. The control (C) group showed normal baseline responses over time.

Heart rates of the TWL were generally lower than for the other two groups, which agrees with the observations of other researchers (42, 51, 58) and reviews of the literature (57, 59) concerning resting heart rate of strength-power athletes. Weight training of the type (60) used by the TWL has been shown to result in a lower submaximal exercise heart rate (47) and a faster recovery heart rate (58), and is in general agreement with the heart rate results of this study.

Lactate (HLa) concentrations postexercise were within values expected for high intensity exercise (15, 27, 50, 53). Plasma volume changes did not differ between groups. Although the difference was small, it is possible that the lower HLa concentration of the TWL group compared to the UTWL group postexercise (12:30 p.m.) represents a training adaptation resulting in a faster metabolism/clearance of HLa from 5 min postexercise to 12:30 p.m. (24, 57, 58). This is in agreement with previous longitudinal observations suggesting that weight training can enhance the postexercise lactate reduction rate (47).

The weight-training exercise-induced increases in blood glucose were similar to those observed after high intensity (80 to 100% VO2 max) aerobic exercise (9, 13), anaerobic cycle exercise (29), and weight training (66). Catecholamines and Cort can increase glucose concentrations (5, 44, 52, 53). These hormones increased as a result of weight-training exercise and may have contributed to the increased glucose concentrations in the TWL and UTWL. The increases in glucose 5 min post-exercise, quickly followed by a return toward baseline concentrations, may reflect a biphasic mismatch between glucose production and use by the tissues (4). After cessation of exercise, a reduction in glucose production would lag behind the decrease in utilization, creating an increase in glucose concentration. Thereafter, production would be reduced to a level below utilization, bringing glucose concentrations back toward baseline (4).

Considerable evidence suggests that muscle contraction enhances the uptake of glucose by muscle even in the absence of insulin, and the increase in glucose transport may persist for several hours after exercise (10, 43). This increased uptake associated with glycogen synthesis may have contributed to the reduced glucose concentrations postexercise. However, it is possible that changes in plasma volume contributed to the decreases in glucose postexercise (11 a.m. to 12:30 p.m.). The average percent decrease in glucose for the two exercise groups was about 21%, but the average change for the plasma volume during that time was a 16% increase.

Both the UTWL and TWL had FFA concentrations that were significantly higher than the control immediately before exercise and remained significantly higher (and increased significantly 22% in the UTWL) compared to controls immediately after exercise (11 a.m.), and a third significant difference between exercise groups and the controls was observed 20 hours later, at 8 a.m. on Day 2. In this study the effects of weight-training exercise on FFA concentrations contrast with other studies using weight training. Guezennec et al. (16), using moderately trained male subjects, reported little change in FFA but did not sample during recovery. Vanhelder et al. (67), using untrained male subjects, reported a depression in FFA through only 10 min of recovery. Neither study (16, 67) used as high a volume of weight-training exercise as in the present study.

It has been speculated that chronic weight training could cause prolonged or chronic elevation of FFA concentrations (28, 57). This prolonged/chronic elevation of FFA as a result of weight training may explain the observation by Keul et al. (28) of elevated serum FFA concentrations among trained male weightlifters at rest compared to untrained control subjects.

In the present study, support for this contention is suggested by several things: First, free fatty acids did not follow the typical daily patterns noted by Hansen and Johansen in the exercised groups (18). Significantly higher group means were shown for both exercise groups compared to the control. Diet is not likely a factor because little difference was observed among groups. These group differences are largely a result of the responses within the exercise groups as a result of weight-training exercise and responses during the 20-hr recovery period. Second, a trend seems evident in that the TWL had higher concentrations of FFA at all time periods compared to the C group and at most time periods compared to the UTWL, and this may reflect regular weight-training exercise in the TWL. Third, several hormones including NE, E, G, Cort, and hGH can mobilize FFA (21, 44, 53, 63). The increased concentration of these hormones during or postexercise may support a rise (mobilization) in FFA at some point during recovery. The elevation of FFA following weight-training exercise may result in a shift in the use of energy substrate toward FFA and is consistent with 1 antagonism and the effects of GH, G, and Cort on FFA mobilization (13, 44, 45, 52, 53, 54, 63).

It may be that chronic weight-training exercise, especially multiple sessions per day, could result in
a chronically elevated blood FFA concentration and a shift toward FFA use for energy production during recovery. Support for a change in substrate use toward FFA after weight training has been noted in the unpublished observation in our laboratory of a shift in RER after weight training in trained subjects (n=4). Immediate preexercise respiratory exchange ratio (RER) was 0.90 in these subjects. The RER increased to 1.3 within 5 min after exercise (consisting of two warm-up sets and 3 x 10 repetitions at 65% of the 1-RM squat), then decreased to between 0.70 and 0.75 at 11 min postexercise. The RER remained at this (0.70-0.75) value until sampling was discontinued at 30 min postexercise.

A shift in substrate utilization would be important for metabolic support of the recovery process, considering the marked glycogenolysis during exercise and decreased muscle glycogen concentrations that can occur as a result of weight-training sessions (36, 49). Although not noted in this study because of matching subjects on body composition, this shift in substrate may be part of the reason for reduced body fat among many weight trainers, especially those training with high volumes/high loads such as elite bodybuilders and weightlifters (58).

The 5-min and 1.5-hr postexercise insulin (I) concentrations were lower than the immediate preexercise sample in all three groups. This suggests that exercise was not the reason for the drop. The lack of an exercise effect is similar to that observed by Bloom et al. (1) and Guezennec et al. (16), but it contrasts with Galbo and Gollnick (12), Kindermann et al. (29), Naveri et al. (45), and Wolfe et al. (70). Why the exercise-induced increase in glucose did not elicit an increase in I concentration is unknown. Depressed I concentration for the two exercise groups, compared to the control group which showed a normal I increase after the 12:30 p.m. meal, persisted until 5 p.m. on Day 2. This suggests that weight-training exercise may have a relatively long-lasting depressant effect on serum insulin concentrations.

The observation of a long-lasting (several hours) weight-training exercise-induced effect suggests an increased sensitivity to I and agrees with previous observations in aerobically trained athletes (37). Several longitudinal studies suggest that weight training can cause increased I sensitivity as a result of regular exercise and increased muscle mass (30, 41, 71, 72).

The weight-training exercise-induced delayed postexercise increase in serum glucagon (G) concentration in the TWL was similar to that observed by Vanhilder et al. (67) in weight trainers, and with that observed in other short-term, high-intensity exercise protocols (12). It is possible that this delayed rise in G (11 a.m. to 5 p.m. on Day 2) concentration was in part stimulated by the drop in glucose postexercise (11 a.m. to 12:30 p.m. on Day 2). Lower G values as a result of exercise are consistent with those noted in other trained groups (1, 17).

Compared to I or G concentrations alone, the I/G may be a better indicator of BG control (64, 69). The changes in the I/G over time better reflect changes in I, because of its greater fluctuations across time, than in G. The two exercise groups, TWL and UTWL, had significantly lower I/G postexercise (5 p.m. Day 2) compared to controls. Low I/Gs have been observed, for at least an hour postexercise, as the result of other forms of exercise (40, 41). For the TWL and UTWL, low I/G values persisted from postexercise until 5 p.m. on Day 2; the control group increased from 12:30 to 5 p.m. This resulted primarily from a rise in G concentration during the same time period in the TWL and UTWL groups compared to the control group, which showed a sharp rise in I concentration as well as an increase in G. A reduction in I/G postexercise appears to be necessary for maintaining blood glucose homeostasis as a result of decreasing glucose concentrations (10, 43, 70).

Cortisol concentrations followed expected circadian rhythms, with higher values for morning than for evening samples (34, 69). Cortisol responses to exercise have shown ambivalent results when comparing untrained versus trained subjects (1, 9, 47, 56, 67, 68). Aerobically trained subjects typically show a more rapid return to baseline concentrations during recovery (1, 61, 63). In this study the TWL had lower Cort concentrations as a result of exercise and exhibited a faster recovery rate (11 a.m. to 5 p.m.) compared to the UTWL. This agrees with previous research using aerobically trained subjects (1, 61, 63). A longitudinal study of high volume weight training has also shown reductions in serum Cort concentrations 20 min postquatting with absolute resistances (47). These results are not in agreement with the observations of Guezennec et al. (16), who observed no significant or consistent changes in recovery Cort concentrations in weight trainers over a 4-month period. However, it should be noted that in Guezennec et al.'s study the subjects used the bench press (small muscle mass), which does not raise metabolism as high as large muscle mass exercises such as squats (51). Thus, using the bench press for testing may not have been a sufficient stimulus for eliciting marked changes in Cort concentrations.

Cortisol responses to exercise are variable and depend, in part, on the intensity and duration of exercise as well as the novelty of exercise, with more intense (>60% VO2max) exercise and/or longer duration exercise producing higher and more prolonged Cort values in less trained subjects (7, 52, 54, 62, 63). It is possible that the
higher concentrations and prolonged elevation of Cort were due to the lesser trained state of the UTWL. It is also possible that the lower Cort concentrations for the TWL may be the result of subjects perceiving the workout as less stressful even though they were using relatively similar workloads. This agrees with previous longitudinal research with untrained subjects (47) and may be considered an adaptation to weight training of sufficient volume and intensity.

Exercise can increase hGH concentrations 20 to 40 times (52), depending on the subject’s “fitness” and the duration and intensity of exercise (54, 61). Serum concentrations of hGH may rise to greater levels following intermittent anaerobic exercise compared to other forms of exercise (66). The response to weight-training exercise for hGH, as well as other hormones, may depend upon protocol and the type of exercise used (small vs. large muscle mass). The leg press is a relatively smaller muscle mass exercise compared to the squat (57). Although the protocols were somewhat different, multiple sets of the leg press did not elicit significant increases in hGH (26); however, the squat did cause a significant response (6).

In this study, large increases in hGH occurred in both exercise groups. The UTWL had a 59% greater peak increase in hGH concentrations than did the TWL, agreeing with previous observations using trained versus untrained subjects (1, 46, 61). Postexercise elevations in hGH may persist for at least 15 to 30 min after a weight-training session (11, 32). Growth hormone has direct effects on carbohydrate and lipid metabolism (21) and stimulates the production of adipolytic lipase (52, 53). Growth hormone opposes the action of I by decreasing glucose uptake, possibly through changing cell membrane permeability or indirectly inhibiting glycolysis by mobilizing FFA (54). The overall effect is an increase in glucose and FFA that favors a shift toward fatty acid use as an energy substrate (21, 54). This shift may have occurred in the present study. The rise of FFA concentration was somewhat larger in the TWL compared to the UTWL. If hGH was a major factor in the increased serum FFA, then the TWL response may represent increased hormone sensitivity since the TWL hGH response was muted.

The serum NE concentrations rose markedly in the TWL and UTWL as a result of exercise. Norepinephrine concentrations before and after the 5-min postexercise sample were similar to resting values. The rise in catecholamines, especially NE, as a result of exercise may occur to support cardiovascular adjustments (13, 14, 62). Additionally, catecholamines may inhibit I release through alpha receptor stimulation (5, 25, 48). Through beta receptor stimulation, catecholamines can stimulate liver glycogenolysis and adipose tissue lipolysis, thus increasing blood glucose and FFA concentrations (5, 13, 62). In this study the exercise-induced rise in catecholamines may have been partially responsible for the exercise-induced rise in glucose and FFA concentrations observed in the TWL and UTWL groups.

It should also be noted that E showed much greater variability across time than did NE. This suggests at least a partial separation of the stimulation of the adrenal medulla with respect to the sympathetic nervous system.

The significantly higher E concentrations for the C group at 8 a.m. on Day 2 are difficult to explain. Perhaps it resulted from emotional stress (53); however, this was not reflected by the POMS. Although not significant, the UTWL also had higher E values at 8 a.m. on Day 2, supporting the suggestion of emotional stress/anxiety; the POMS, although not significant, showed a higher total mood disturbance in the UTWL compared to the other two groups. These observations are also consistent with the elevated cortisol concentrations of the TWL and UTWL at the same time period and again suggest increased anxiety on the part of the untrained subjects. Large increases in E concentrations occurred as a result of exercise in the TWL. Similar increases in epinephrine have been observed after other forms of anaerobic exercise (29, 38) and high intensity resistance training (31). It is possible that the increases in E are directly related to the absolute exercise intensity. If this is correct, then the greater rise of E in the TWL in response to exercise may reflect the higher absolute intensities used compared to the UTWL.

The lower cortisol values at the same time (11 a.m.) and at 12:30 p.m. may reflect less stress among the TWL. Thus they were working at a higher absolute workload but experiencing less stress. It is also possible that weight training may optimize blood catecholamine accumulation, producing higher catecholamine concentrations at maximum or near maximum intensities but not at submaximal relative values. Optimization of catecholamine response to exercise has been noted among trained runners (33).

Of concern to us was the possibility that the TWL group might be exhibiting symptoms of overtraining after the 3 weeks of high volume weight training immediately preceding the experiment. Raising the volume of training can cause symptoms of overtraining to occur in relatively short periods of time (35, 56). Overtraining can result in a variety of symptoms including increased resting and recovery HR, blood pressure, HLA, and hormone concentrations (56). Lower HR and faster recovery of HLA, hGH, and Cort were observed. Additionally, blood pressure (not reported) was
checked in some subjects pre- and postexercise, indicating lower blood pressure responses during and postexercise for the TWLs. Furthermore, the POMS inventory revealed no aberrant measures for the TWL. Thus there was no indication of overtraining in this group.

This study examined the changes in concentrations of blood glucose, serum FFA, and various hormones associated with energy substrate mobilization and use, consequent to a high-volume weight-training exercise session. Also compared were the responses of trained and untrained subjects. High volume weight training is supported by increases in glucose with a shift toward fat mobilization and use during recovery. The changes in glucose and FFA concentrations may be a direct or indirect effect of changes in various hormones, especially hGH, Cort, and NE. Based on lower resting, exercise, and recovery concentrations, trained weightlifters may be more hormone sensitive. This is especially apparent for I, Cort, and hGH. The HR and muted hormonal responses observed, especially for Cort and hGH, may reflect less physiological stress for the TWL as a result of weight-training exercise. The generally lower HRs and faster return toward resting values of HLA, hGH, and Cort suggest faster recovery capabilities of the TWL after weight-training exercise.

This study has described acute metabolic and hormonal responses to weight training in trained and untrained subjects. Further experiments, especially with trained subjects over longer periods of time with different protocols, would help explain these phenomena.

Practical Significance

These data indicate that weight-training experience of sufficient volume may alter acute physiological response to weight-training exercise in a manner suggesting reduced physiologic stress. This reduction in stress may contribute to enhanced performance for resistive training and other high intensity activities in a manner similar to that of the contribution of aerobic training adaptations to various endurance activities.

Additionally, these data indicate that the recovery process from weight training may enhance the use of FFA. Thus, weight training, particularly high-volume weight training, can be useful in causing body mass/body composition alterations, including fat mass and percent fat reductions.

References


