Serum leptin responses after acute resistance exercise protocols

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Zafeiridis, A., I. Smilos, R. V. Considine, and S. P. Tokmakidis. Serum leptin responses after acute resistance exercise protocols. J Appl Physiol 94: 591–597, 2003. This study examined the acute effects of maximum strength (MS), muscular hypertrophy (MH), and strength endurance (SE) resistance exercise protocols on serum leptin. Ten young lean men (age = 23 ± 4 yr; body weight = 79.6 ± 5.2 kg; body fat = 10.2 ± 3.9%) participated in MS [4 sets × 5 repetitions (reps) at 88% of 1 repetition maximum (1 RM) with 3 min of rest between sets], MH (4 sets × 10 reps at 75% of 1 RM with 2 min of rest between sets), SE (4 sets × 15 reps at 60% of 1 RM with 1 min of rest between sets), and control (C) sessions. Blood samples were collected before and immediately after exercise and after 30 min of recovery. Serum leptin at 30 min of recovery exhibited similar reductions from baseline after the MS (−20 ± 5%), MH (−20 ± 4%), and SE (−15 ± 6%) protocols that were comparable to fasting-induced reduction in the C session (−12 ± 3%) (P < 0.05). Furthermore, no differences were found in serum leptin among the MS, MH, SE, and C sessions immediately after exercise and at 30 min of recovery (P > 0.05). Cortisol was higher (P < 0.05) after the MH and SE protocols than after the MS and C sessions. Glucose and growth hormone were higher (P < 0.05) after exercise in the MS, MH, and SE protocols than after the C session. In conclusion, typical resistance exercise protocols designed for development of MS, MH, and SE did not result in serum leptin changes when sampled immediately or 30 min postexercise.

maximum strength; muscular hypertrophy; strength endurance; hormones; glucose

LEPTIN IS A HORMONE SECRETED by adipose tissue cells to regulate body weight (6). Although the precise mechanisms that underlie leptin secretion are not fully understood, a link with negative energy balance, sympathethic activation, other hormones, and metabolites has been observed (1, 32). Furthermore, recent findings suggest that leptin responds to low carbohydrate and energy availability, constituting a link between energy intake and storage (12). Therefore, it has become of interest to examine whether physical activity, through its disruptive effects on energy balance, sympathoadrenal drive, and hormonal and metabolic homeostasis, may affect serum leptin concentration. In the past 5 yr, almost all studies have been directed toward examining the effects of aerobic activity on serum leptin by the utilization of continuous running regimens (7, 11, 16, 20, 30, 31, 35, 36). It is agreed that, after a single bout of running exercise at moderate intensity, serum leptin remains relatively unchanged (30, 31, 35, 41), whereas extreme exercise conditions may reduce serum leptin (7, 20, 36). Information regarding the response of serum leptin to a single bout of resistance exercise is limited. In contrast to continuous running of moderate intensity, heavy resistance exercise is a potent nonoxidative stimulus that produces differential neural, metabolic, and neuroendocrine responses. More specifically, nonoxidative and resistance exercise elicit greater lactate, glucagon, cortisol, and growth hormone (GH) levels and may induce higher postexercise oxygen consumption and sympathoadrenal activation compared with moderate-intensity aerobic training exercise (4, 15, 38, 39). Furthermore, the ATP production during heavy resistance exercise relies primarily on creatine phosphate, glucose, and glycogen utilization (21, 29, 33) producing higher rates of glucose utilization-production cycles compared with submaximal aerobic regimens that rely on lipid mobilization. Previous studies have pointed out that glycogen depletion (36), inhibition of glycolysis (25), elevated glucose uptake in the presence of lactate (26), and state of acidosis (34) may decrease serum leptin. In contrast, acute administration of glucocorticoids (22) and GH (9), glucose infusion (2, 17, 24), as well as an increase in intracellular products of glucose metabolism (23, 40) have been shown to potentiate leptin secretion by adipocytes. Little has been established regarding the effects of a single bout of acute resistance exercise on serum leptin concentration. One study reports reduced 24-h serum leptin in diabetic but not in healthy individuals (14), and the other reports reduction of serum leptin levels 9–13 h postexercise in healthy lean men (27). The

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aforementioned studies had utilized a single resistance exercise protocol, and one of the two studies (14) examined only the 24-h leptin response. However, several resistance-training protocols have been routinely utilized by fitness and rehabilitation centers for the development of different aspects of strength, such as maximum strength (MS), muscular hypertrophy (MH), and strength endurance (SE). These protocols differ in the configuration of the acute program variables, such as intensity, number of repetitions, total work, and rest interval, placing a specific physiological stress on the body and eliciting distinct acute neuroendocrine and metabolic responses. An increase in total work or a decrease in rest interval favors higher growth hormone and cortisol responses (18). For example, the MH protocol elicits higher lactate, GH, and cortisol responses compared with MS protocol (10, 18). Thus the different resistance exercise protocols may induce distinct leptin responses as well.

With the limited information on the acute effects of heavy resistance exercise on serum leptin, the specific aims of the present study were to explore 1) whether a single bout of MS, MH, and SE resistance exercise protocols would acutely modulate serum leptin and 2) whether the configuration of the resistance exercise program variables would affect serum leptin.

MATERIALS AND METHODS

Subjects

Ten healthy young men (age 22.8 ± 4.1 yr, height 181 ± 7 cm, body weight 79.6 ± 5.2 kg, and body fat 10.2 ± 3.9%) with recreational experience in weight lifting volunteered to participate in this investigation. Before initiation of the study, the Institutional Review Board Committee of Democritus University of Thrace approved the experimental protocol and the subjects signed a written, informed consent. All subjects completed a medical questionnaire to ensure that they were not taking any medication, were free of cardiac, respiratory, renal, or metabolic diseases, and were not using steroids.

Study Design

After an overnight fast, each subject participated in one control session and three resistance exercise protocols: MS [4 sets × 5 repetitions (reps) at 88% of 1 repetition maximum (1 RM) with 3 min of rest between sets], MH [4 sets × 10 reps at 75% of 1 RM with 2 min of rest between sets], and SE [4 sets × 15 reps at 60% of 1 RM with 1 min of rest between sets]. All resistance exercise protocols were completed by using four exercises (bench press, latissimus dorsi pull-downs, squat, and overhead press) with 6 min of recovery between exercises. In the control session, the subjects were seated comfortably and rested for the entire study period to account for circadian rhythm variations. All sessions were scheduled 1 wk apart and performed in random order to offset any training or sequencing effects. Blood samples were collected before, immediately after, and 30 min after resistance exercise. The samples were analyzed for serum leptin, glucose, free fatty acid (FFA), lactate, cortisol, and GH concentrations.

Exercise Testing Procedures

On the first day of the study, each subject reported to the exercise laboratory for the assessments of resting energy expenditure (REE), body fat, and maximum voluntary contraction (1 RM) for bench press, latissimus dorsi pull-downs, squat, and overhead press exercises. Briefly, for the 1-RM measurements, the subjects completed a warm-up by using light weights (~50% of predicted 1 RM) and then performed single attempts on incremental weights, beginning at 90% of predicted 1 RM, until failure. The last lifted weight was considered as the 1 RM. After the assessment of 1 RM for each resistance exercise, the subjects reported to the laboratory four times to perform the MS, MH, and SE resistance exercise protocols and the control session in random order.

The resistance exercise sessions as well as the control session were conducted at the same time of the day (9:00 AM to 11:30 AM) for each subject to avoid the effects of fasting and circadian rhythm. All exercise sessions were conducted after an overnight fast. The time each exercise session started was adjusted so that the blood sample drawn at 30 min of recovery was obtained at the same time of the day for all sessions. Before each session, a 16-gauge intravenous catheter was inserted into an antecubital vein for serial blood collections, and the subjects performed a standardized warm-up. In the three experimental sessions, the four exercises were performed in the following order: bench press, latissimus dorsi pull-downs, squat, and overhead shoulder press. All exercises were executed with the use of free weights except the latissimus dorsi pull-downs, which were performed with a Universal weight machine. The subjects were instructed to follow a normal lifestyle maintaining daily habits, to avoid any medications, and to refrain from alcohol, caffeine, and exercise 3 days before each session.

Body Composition, Total Work, Energy Expenditure, and Biochemical Analyses

Body composition. Body density was predicted from the skinfolds measurements taken on the right side of the body using Harpenden calipers at the chest, abdominal, and thigh sites (13), and the percent body fat was then calculated by using the formula of Brozek et al. (3).

Total work. All exercises were structured according to the anatomic characteristics of each subject with grip widths and positions marked and kept constant for each exercise throughout the study. Lifting work was calculated as the weight load × the vertical distance moved per repetition × the number of repetitions. The weights of the body segments of the subjects × the vertical distance of the center of gravity of the body segments, which were moved, were also included in the calculations. The location of body segment centers of gravity and the estimation of body segment weights from the total body weight were assessed with the use of anthropometric tables (43). The distances were obtained from measurements with the subjects and the equipment in the starting and ending exercise positions.

Energy expenditure. Total energy expenditure of resistance exercise protocols was calculated as the sum of REE and the net caloric cost of resistance exercise protocol. REE was assessed over the last 25 min of a 30-min period with the subjects breathing into a mouthpiece in supine position by using the breath-by-breath technique with on-line-data processing and display (Oxycon Champion, Minhardt, Netherlands). The net energy expenditure (kcal) of resistance exercises was calculated by utilizing the following regression equations for predicting the caloric expenditure during multistation resistance exercise.

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Bench press:

\[ \text{kcal} = 0.56 + 0.006 \times \text{weight lifted in lb.} \]

Latissimus dorsi pull-downs:

\[ \text{kcal} = -0.40 + 0.008 \times \text{weight lifted in lb.} \]

Overhead press:

\[ \text{kcal} = 0.05 + 0.008 \times \text{weight lifted in lb.} \]

Squat:

\[ \text{kcal} = 2.41 + 0.071 \times \text{weight lifted in kg} \]

**Biochemical analyses.** Blood samples (10 ml) were obtained before resistance exercise, immediately after resistance exercise, and at 30 min of recovery. Two hundred microliters of blood were immediately added to 400 µl of trichloroacetic acid and centrifuged at 2,500 rpm for 15 min. The supernatant was removed and frozen at -80°C until later analysis of lactate concentration with an enzymatic method (procedure no. 826 UV, Sigma Chemical, St. Louis, MO). The remaining blood was centrifuged at 2,500 rpm for 25 min, and the serum was removed, separated into aliquots of 500 µl, and stored at -80°C for later analysis.

Serum leptin was measured by radioimmunoassay with a commercially available kit (Linco Research, St. Charles, MO). The intra- and interassay coefficients and the sensitivity of measurement for leptin were 6.2%, 8.3%, and 0.5 ng/ml respectively. Serum FFAs were assayed by using the NEFA-C kit (Wako Chemicals, Richmond, VA), and blood glucose was measured by the glucose oxidase method with a Hitachi U-2001 spectrophotometer (Sigma Chemical). Luminescence immunoassay technique was used to analyze the serum concentrations of cortisol (Ortho-Clinical Diagnostics, Johnson & Johnson, Rochester, NY; intra- and interassay coefficients and sensitivity = 3.8%, 5.9%, and <3 nmol/l, respectively). Serum GH concentration was analyzed by immunoradiometric assay (Medicorp, Montreal, Canada; intra- and interassay coefficients and sensitivity = 3.9%, 5.2%, and 0.09 µg/l, respectively).

**Statistical Analysis**

Statistical analyses were performed by using SPSS software (Chicago, IL). ANOVA (4 × 3) with repeated measures on resistance exercise protocol (4 levels) and time (3 levels) factors were used to determine the effects of exercise protocol, time, and exercise protocol by blood sampling time interaction on serum leptin, lactate, glucose, FFA, cortisol, and GH concentrations. Tukey’s post hoc multiple comparisons were performed to locate the statistical significant differences between exercise protocols and within each protocol. Data are presented as means ± SE with a value of \( P < 0.05 \) considered statistically significant.

**RESULTS**

The acute effects of the three resistance exercise regimens and the control session on serum leptin levels are presented in Fig. 1. Serum leptin concentrations significantly decreased (\( P < 0.05 \)) at the end of exercise compared with the respective baseline values after the MS (from 2.62 ± 0.26 to 2.14 ± 0.21 ng/ml; \(-17 \pm 2\%\)) and MH protocols (from 2.66 ± 0.31 to 2.18 ± 0.24 ng/ml; \(-16 \pm 1\%\)). However, leptin levels did not change significantly (\( P > 0.05 \)) during the corresponding time interval after the SE (from 2.26 ± 0.20 to 2.07 ± 0.18 ng/ml; \(-5 \pm 3\%)\) and the control sessions (from 2.49 ± 0.33 to 2.24 ± 0.30 ng/ml; \(-6 \pm 2\%)\).

During recovery, serum leptin concentrations continued to decline and were significantly lower in all sessions (MS: 2.06 ± 0.18; MH: 2.08 ± 0.22; SE: 1.87 ± 0.16; control: 2.16 ± 0.30 ng/ml) at 30 min of recovery compared with the respective baseline values (\( P < 0.05 \)). Pairwise multiple comparisons between groups at the end of exercise and at 30 min of recovery did not reveal statistical significant differences in serum leptin concentrations among the three exercise programs and the control session (see Fig. 1; \( P > 0.05 \)).

Table 1 depicts the concentrations of serum FFAs and blood glucose and lactate in the three resistance exercise and control sessions. Blood glucose was significantly higher immediately after the completion of all resistance exercise protocols compared with the respective baseline values (\( P < 0.05 \)). In the control session, serum glucose remained unchanged throughout the experiment (\( P > 0.05 \)). Pairwise comparisons indicated significantly higher glucose levels immediately after the MS, MH, and SE exercise protocols compared with the respective control value. During the 30 min of recovery, glucose concentrations were not different (\( P > 0.05 \)) among the three resistance exercise protocols and the control session. FFA concentrations did not change significantly (\( P > 0.05 \)) at the end of exercise and at 30 min of recovery compared with the respective baseline in the three resistance exercise protocols and the control session. Lactate concentrations were significantly higher (\( P < 0.05 \)) immediately after exercise and at 30 min of recovery in the MS, MH, and SE sessions compared with the respective baseline and control values (\( P < 0.05 \)). Post hoc comparisons between resistance exercise protocols revealed that, at the end of exercise and at 30 min of recovery, lactate levels were significantly higher in the SE and MH protocols compared with the respective values in the MS protocol (\( P < 0.05 \)).

Cortisol concentrations (Table 1) significantly increased immediately after the MH and SE resistance exercise protocols (\( P < 0.05 \)) and remained significantly elevated at 30 min of recovery compared with the baseline values (\( P < 0.05 \)). Cortisol levels did not
Table 1. Glucose free fatty acids, lactate, cortisol, and growth hormone concentrations in maximum strength, muscular hypertrophy, strength endurance, and control sessions

<table>
<thead>
<tr>
<th>Variable</th>
<th>Baseline</th>
<th>End of Exercise</th>
<th>30 min of Recovery</th>
</tr>
</thead>
<tbody>
<tr>
<td>Glucose, mmol/l</td>
<td></td>
<td></td>
<td></td>
</tr>
<tr>
<td>MS</td>
<td>4.42 ± 0.15</td>
<td>5.08 ± 0.17†</td>
<td>4.80 ± 0.15</td>
</tr>
<tr>
<td>MH</td>
<td>4.76 ± 0.12</td>
<td>5.55 ± 0.30†</td>
<td>5.12 ± 0.26</td>
</tr>
<tr>
<td>SE</td>
<td>4.59 ± 0.15</td>
<td>5.27 ± 0.24†</td>
<td>4.70 ± 0.20</td>
</tr>
<tr>
<td>C</td>
<td>4.70 ± 0.20</td>
<td>4.52 ± 0.16</td>
<td>4.71 ± 0.15</td>
</tr>
<tr>
<td>Free fatty acids, mmol/l</td>
<td></td>
<td></td>
<td></td>
</tr>
<tr>
<td>MS</td>
<td>0.79 ± 0.19</td>
<td>0.48 ± 0.09</td>
<td>0.67 ± 0.15</td>
</tr>
<tr>
<td>MH</td>
<td>0.73 ± 0.17</td>
<td>0.64 ± 0.16</td>
<td>0.58 ± 0.08</td>
</tr>
<tr>
<td>SE</td>
<td>0.51 ± 0.17</td>
<td>0.37 ± 0.03</td>
<td>0.57 ± 0.12</td>
</tr>
<tr>
<td>C</td>
<td>0.74 ± 0.16</td>
<td>0.68 ± 0.11</td>
<td>0.86 ± 0.20</td>
</tr>
<tr>
<td>Lactate, mmol/l</td>
<td></td>
<td></td>
<td></td>
</tr>
<tr>
<td>MS</td>
<td>1.22 ± 0.06</td>
<td>5.11 ± 0.32†</td>
<td>2.10 ± 0.16</td>
</tr>
<tr>
<td>MH</td>
<td>1.17 ± 0.09</td>
<td>9.87 ± 0.80††</td>
<td>3.97 ± 0.41††</td>
</tr>
<tr>
<td>SE</td>
<td>1.25 ± 0.03</td>
<td>11.08 ± 0.99††‡</td>
<td>4.30 ± 0.31††‡</td>
</tr>
<tr>
<td>C</td>
<td>1.14 ± 0.05</td>
<td>1.36 ± 0.13</td>
<td>1.16 ± 0.05</td>
</tr>
<tr>
<td>Cortisol, nmol/l</td>
<td></td>
<td></td>
<td></td>
</tr>
<tr>
<td>MS</td>
<td>307 ± 28</td>
<td>253 ± 31</td>
<td>262 ± 33</td>
</tr>
<tr>
<td>MH</td>
<td>325 ± 29</td>
<td>431 ± 59†</td>
<td>449 ± 78†</td>
</tr>
<tr>
<td>SE</td>
<td>335 ± 28</td>
<td>439 ± 37††‡</td>
<td>495 ± 54††‡</td>
</tr>
<tr>
<td>C</td>
<td>354 ± 47</td>
<td>301 ± 49</td>
<td>260 ± 29</td>
</tr>
<tr>
<td>Growth hormone, μg/l</td>
<td></td>
<td></td>
<td></td>
</tr>
<tr>
<td>MS</td>
<td>0.75 ± 0.54</td>
<td>3.75 ± 1.09*</td>
<td>1.38 ± 0.41</td>
</tr>
<tr>
<td>MH</td>
<td>0.99 ± 0.50</td>
<td>13.74 ± 4.83††‡</td>
<td>5.18 ± 2.28††‡</td>
</tr>
<tr>
<td>SE</td>
<td>0.73 ± 0.45</td>
<td>21.90 ± 6.40††‡</td>
<td>9.43 ± 2.64††‡</td>
</tr>
<tr>
<td>C</td>
<td>0.55 ± 0.35</td>
<td>0.76 ± 0.34</td>
<td>0.25 ± 0.03</td>
</tr>
</tbody>
</table>

Values are means ± SE. MS, maximum strength; MH muscular hypertrophy; SE, strength endurance; C, control. *P < 0.05 vs. respective baseline value. †P < 0.05 vs. respective control value. ‡P < 0.05 vs. respective MS value. §P < 0.05 vs. respective MH value.

change significantly after the MS exercise protocol (P > 0.05) and significantly declined in the control session (P < 0.05). Comparisons between protocols showed that cortisol levels were significantly higher (P < 0.05) at the end of recovery and at 30 min of recovery in the MH and SE protocols compared with the MS and the control protocols. GH concentrations (Table 1) significantly increased (P < 0.05) at the end of exercise and remained significantly elevated at 30 min of recovery in the MH and SE protocols compared with the respective baseline values. In the MS session, serum GH significantly increased (P < 0.05) at the end of exercise but returned to baseline value at 30 min of recovery (P > 0.05). In the control session, GH concentrations remained unchanged (P > 0.05). Pairwise comparisons showed that, at the end of exercise and at 30 min of recovery, serum GH was significantly higher (P < 0.05) in the MH and SE protocols compared with the MS and control protocols.

Total work performed in the MS protocol was lower compared with that in the MH and SE protocols, by 43 and 52%, respectively, and was 16% lower in MH than in SE protocol. Similarly, the energy expenditure in the MS protocol was lower by 27% compared with the MH protocol and by 30% compared with the SE protocol, and it was 4% lower in the MH than in the SE protocol. The calculated total work and estimates of energy expenditure during the MS, MH, and SE resistance exercise protocols are presented in Table 2.

**DISCUSSION**

The present study contributes to the limited information on the acute effects of resistance exercise on serum leptin. Furthermore, this is the first investigation to explore whether the configuration of the resistance exercise program variables may affect serum leptin. The main findings of the present study indicated that, in normal individuals, the MS, MH, and SE resistance exercise protocols elicit comparable serum leptin responses and that a single bout of heavy resistance exercise protocols has no significant acute effects on serum leptin compared with resting session. In agreement with previous studies (20, 35, 41), the decline in serum leptin was related to overnight and morning fasting rather than to resistance exercise because no differences were observed between resistance exercise and control sessions. This observation underlines the importance of using a resting session when evaluating the effects of exercise on serum leptin to control for fasting- and/or circadian rhythm-induced leptin reductions. The results of our study support the results of Nindl et al. (27), who also failed to document significant differences between resistance exercise and control session at 30 min after heavy resistance exercise.

Previous studies (7, 20, 36) that utilized aerobic regimens had shown that serum leptin responds mainly to extreme exercise conditions; therefore, the resistance exercise protocols employed in the present study were designed toward producing a great magnitude of hormonal and metabolic responses. It is evident by the significant rises in glucose, lactate, cortisol, and GH at the end of the resistance exercise programs that we achieved these goals. The SE and MH protocols produced significantly greater hormonal and metabolic responses. More specifically, lactate, cortisol, and GH changes were more pronounced in the MH and SE protocols compared with the MS and control protocols. Furthermore, the SE protocol was the most stressful protocol, as indicated by the highest lactate levels. Thus it was expected that the most pronounced serum leptin responses would be observed after the SE exercise protocol. Nevertheless, leptin response was virtually identical between the MS and MH protocols and very close between the MS and SE protocols that differed significantly in the magnitude of hormonal and metabolic responses (SE > MH > MS). Furthermore,

Table 2. Estimated total work and energy expenditure during maximum strength, muscular hypertrophy, strength endurance sessions

<table>
<thead>
<tr>
<th>Variable</th>
<th>Total Work, J</th>
<th>Estimated Energy Expenditure, kcal</th>
</tr>
</thead>
<tbody>
<tr>
<td>MS</td>
<td>33,271 ± 2,209</td>
<td>231.19 ± 6.8</td>
</tr>
<tr>
<td>MH</td>
<td>58,199 ± 2,209</td>
<td>314.86 ± 11.1</td>
</tr>
<tr>
<td>SE</td>
<td>69,534 ± 2,457</td>
<td>327.58 ± 12.8</td>
</tr>
</tbody>
</table>

Values are means ± SE.
the differences in serum leptin concentrations between the MS, MH, SE, and control sessions immediately after exercise and at 30 min of recovery were within the 19% day-to-day biovariability of leptin (42). It appears that differences in the configuration of the intensity, total work and rest interval among resistance training protocols do not affect acute serum leptin responses as they affect other hormonal responses.

In the present study, the decline of serum leptin in control group at the end of experiment is in accordance with diurnal variation and fasting (12 h) that have been previously reported (17, 20, 35, 41). Heavy resistance exercise performed in fasting state was expected to augment the decline in serum leptin compared with that observed in the control session, because studies have reported that elevated glucose uptake by peripheral tissues in the presence of lactate (26), state of acidosis (34), increased sympathoadrenal drive and energy expenditure, and inhibition of glycolysis in isolated cultured adipocytes (25) decrease serum leptin. In this study, blood lactate rose by 5-, 9-, and 11-fold in the MS, MH, and SE protocols, respectively, suggesting increased glucose uptake and utilization, increased rate of glycogenolysis and glycolysis and decreased pH levels in the contracting skeletal muscle. Furthermore, it is possible that glucose utilization by adipocytes was decreased because exercise shifts glucose utilization from the adipose tissue to muscle cells. Despite the aforementioned changes (rise in lactate and so forth), we did not observe an augmented decline in serum leptin after resistance exercise compared with that in the control session. It may be that 60 min of altered glucose transport and utilization in presence of lactate and the rise in lactate secondary to resistance exercise were not of sufficient stimulus to change serum leptin. Another explanation may be that, during heavy resistance exercise, glucose transport and metabolism of adipose tissue were not significantly altered to induce changes in leptin amount. In addition, Tuominen et al. (36) have suggested that serum leptin concentrations decrease by glycogen-depleting exercise. More specifically, the authors observed that the reduction in muscle glycogen content (~32%) after prolonged, intense exercise paralleled that of serum leptin. In the present study, we did not measure muscle glycogen content. However, the significant rise of blood lactate by 5- to 11-fold after resistance exercise protocols points toward an increased rate of glycolysis and consequently to reduction in muscle glycogen stores. In addition, protocols similar to those performed in the present study have consistently demonstrated reduction in muscle glycogen stores by 20–40% (21, 29, 33). Despite the suggested increase in glycogen utilization and a reduction in glycogen stores after resistance exercises, serum leptin concentrations decreased in similar manner after the three resistance exercise protocols and the control session. Therefore, in light of the results of the present study, the primary contribution of acute glycogen reduction or depletion to leptin concentration is not supported.

One possible explanation for the lack of an augmented leptin decline after resistance exercise compared with that in control trial is the recent findings that intracellular products of glucose metabolism (hexosamines) stimulate leptin mRNA expression and that infusion of small amounts of glucose (100 kcal) may prevent leptin reduction (2, 17, 23, 24, 40). Therefore, it is possible that the small but significant 15% increase in glucose after the MS, MH, and SE resistance exercise and a greater availability of hexosamines, because of higher glycolysis in contracting muscles, may have counteracted (attenuated) the reducing effects of resistance exercise trials on leptin such as increased negative balance and sympathetic stimulation, reduced glycogen stores, reduced pH, and increased glucose uptake with concomitant lactate production. Thus the significant increase in blood glucose after resistance exercise appears as a possible explanation to attenuated (lack of an augmented) leptin decline after resistance exercise compared with control.

Another possible explanation for the lack of differences in serum leptin decline after resistance exercise vs. the control trial is the prevailing theory of delayed leptin responses after physical activity. Previous studies that utilized aerobic running exercise in lean healthy men have reported delayed leptin reduction at 24 and/or 48 h after exercise (8, 28, 37). It appears from the literature that delayed serum leptin reductions after aerobic exercise were observed after energy cost of >800 kcal. This is in context with the results of Nindl et al. (27), who reported delayed leptin reduction 9–13 h postexercise in postabsorptive healthy lean men after a heavy resistance exercise protocol with total work during exercise of 313,000 J and an estimated total energy cost of 856 kcal. However, in the study of Kanaley et al. (14), the 24-h serum leptin after single bout of resistance exercise was reduced only in diabetic but not in healthy individuals. The aforementioned study, however, does not provide measures of energy cost, and speculation as to whether exercise was stressful enough to produce delayed leptin responses is not possible. The present study utilized three typical resistance exercise regimens with total work during exercises of 33,271 ± 3,891 J in MS, 58,199 ± 6,986 J in MH, and 69,534 ± 7,865 J in SE protocols, which are four to nine times below those used in the study of Nindl et al. (27). Despite the twofold greater energy cost in SE compared with MS, leptin responses were similar. Interestingly, aerobic exercises with total work ranging from 150 to 529 kcal, which includes the estimates of total energy cost of our exercise protocols (Table 2), did not result in different serum leptin responses 3.5 h postexercise (41). It is difficult to speculate as to whether we would observe delayed reduction in serum leptin if four facts are considered collectively. First, the typical resistance exercise protocols in our study were unlikely to produce energy cost >800 kcal. Second, observations regarding the “critical level” of energy cost (>800 kcal) and serum leptin decline are based on the results of aerobic exercise. Third, neural, endocrine, and metabolic responses
LENPEI RESPONSES TO ACUTE RESISTANCE EXERCISE PROTOCOLS

that may modulate leptin are different in resistance compared with aerobic exercise protocols. Fourth, it remains unknown how energy availability, sympathetic stimulation, and metabolites and hormones that down- and upregulate leptin concentration may interact to modulate leptin concentrations.

Similar to other studies, GH levels increased by 4-, 12-, and 20-fold after the MS, MH, and SE sessions, respectively, compared with control values, and cortisol levels were ~75% higher in the MH and SE trials than in the control (10, 18). Previous studies in subjects with GH deficiency and Cushing’s syndrome have shown that acute administrations of cortisol and GH within physiological range may increase leptin production at 6–7 and 24 h after cortisol and GH administrations, respectively (9, 22). Some investigators suggest that increase in cortisol levels during exercise may also counteract the reducing effects of exercise on serum leptin (16).

In summary, MS, MH, and SE resistance exercise protocols did not significantly affect serum leptin immediately after exercise and at 30 min of recovery. Serum leptin levels at 30 min of recovery exhibited similar reductions from baseline after MS (~20 ± 5%), MH (~20 ± 4%), and SE (~15 ± 6%) protocols. These reductions, however, were independent of exercise because comparable fasting-induced decline was documented in the control session (~12 ± 3%). There were no differences in serum leptin among the MS, MH, SE and control sessions immediately after exercise and at 30 min of recovery. The present study demonstrated that typical resistance exercise protocols did not result in acute serum leptin changes. However, the delayed effects of a single bout of MS, MH, and SE resistance exercises might be considered.

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REFERENCES


